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Delay discounting in youth at clinical high-risk for psychosis and adults with schizophrenia

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Original Article

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Abstract

Background. Schizophrenia (SZ) is typically preceded by a prodromal (i.e. pre-illness) period characterized by attenuated positive symptoms and declining functional outcome. Negative symptoms are prominent among individuals at clinical high-risk (CHR) for psychosis (i.e. those with prodromal syndromes) and highly predictive of conversion to illness. Mechanisms underlying negative symptoms in the CHR population are unclear. Two studies were conducted to evaluate whether abnormalities in a reward processing mechanism thought to be core to negative symptoms in SZ, value representation, also exist in CHR individuals and whether they are associated with negative symptoms transphasically.

Methods. Study 1 included 33 individuals in the chronic phase of illness who have been diagnosed with schizophrenia or schizoaffective disorder (SZ) and 40 healthy controls (CN). Study 2 included 37 CHR participants and 45 CN. In both studies, participants completed the delay discounting (DD) task as a measure of value representation and the Brief Negative Symptom Scale was rated to measure negative symptoms.

Results. Results indicated that patients with SZ had steeper discounting rates than CN, indicating impairments in value representation. However, CHR participants were unimpaired on the DD task. In both studies, steeper discounting was associated with greater severity of negative symptoms.

Conclusions. These findings suggest that deficits in value representation are associated with negative symptoms transphasically.

Introduction

Schizophrenia (SZ) is the leading cause of functional disability worldwide. Negative symptoms are the strongest predictor of functional disability. They also predict several other important clinical outcomes associated with poor prognosis, such as liability for illness, reduced subjective well-being, and lower rates of recovery (Pelletier-Baldelli, Strauss, Visser, & Mittal, 2017; Piskulic et al., 2012; Strauss, Harrow, Grossman, & Rosen, 2010; Strauss, Sandt, Catalano, & Allen, 2012). Unfortunately, currently available pharmacological and psychosocial treatments for negative symptoms have proven largely ineffective (Fusar-Poli et al., 2015).

To develop novel mechanistic targets for the treatment of negative symptoms, several theoretical accounts have been proposed, which focus on reward processing mechanisms resulting from disrupted cortico-striatal circuitry (Barch & Dowd, 2010; Kring & Barch, 2014). These accounts all have the fundamental assumption of hedonic normality in SZ, and propose that other aspects of reward processing (e.g. value representation, reinforcement learning, effort-cost computation) that rely on cortico-striatal interactions prevent intact hedonic responses from influencing decision-making processes needed to motivate goal-directed behaviors that are deficient in those with negative symptoms (Strauss, Waltz, & Gold, 2014).

Although such conceptual models of negative symptoms have been well-validated in adults with SZ, there is a lack of research on whether models developed for SZ also apply to youth at clinical high-risk (CHR) for psychosis. However, focusing on negative symptoms during this phase of illness may be beneficial, as negative symptoms are highly prevalent in CHR youth and one of the earliest markers of psychosis risk that often lead to initial contact with the treatment system (Addington & Heinssen, 2012; Carrión et al., 2016; Häfner, Löffler, Maurer, Hambrecht, & Heiden, 1999; Piskulic et al., 2012). Identifying mechanisms underlying negative symptoms in CHR youth may therefore be critical for achieving the goals of identifying individuals in the earliest phase of the prodrome when treatment efforts may be most effective at delaying or preventing illness onset (McGorry, Hickie, Yung, Pantelis, & Jackson, 2006; McGorry, Nelson, Goldstone, & Yung, 2010).

The reward processing construct of 'value representation' has been proposed to be perhaps the most critical deficit underlying negative symptoms in adults with SZ, suggesting that it is an important domain to be explored in CHR youth. Value representation refers to the ability to generate, update, or maintain mental representations of reward value and use them to guide decision-making. The orbitofrontal cortex plays a critical role in value representation, enabling

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the calculation of an outcome's value, whether an outcome fulfills motivational needs, and making comparisons of one outcome's value to another's (Wallis, 2007). Individuals with SZ display impairments on tasks where value representations must be updated (e.g. probabilistic reversal learning, intradimensional/extradimensional set-shifting tasks, Iowa Gambling task) (Ceaser et al., 2008; Elliott, McKenna, Robbins, & Sahakian, 1995; Lee et al., 2007; Pantelis et al., 1999; Sevy et al., 2007; Shurman, Horan, & Nuechterlein, 2005; Tyson, Laws, Roberts, & Mortimer, 2004; Waltz, Frank, Wiecki, & Gold, 2011), generated [e.g. delay discounting (DD) and preference judgment tasks (Elliott, Agnew, & Deakin, 2010; Heerey, Matveeva, & Gold, 2011; Heerey, Robinson, McMahon, & Gold, 2007; Strauss et al., 2011], and maintained (e.g. hedonic reactivity and maintenance tasks) (Gard et al., 2011; Kring, Germans Gard, & Gard, 2011; Ursu et al., 2011). Furthermore, impairment on these value representation tasks has been associated with greater severity of negative symptoms in SZ (Strauss et al., 2014). Such findings have led some to propose that value representation may be the most fundamental deficit underlying negative symptoms, preventing intact reward responses from being used to guide decision-making processes needed to initiate approach motivation behaviors (e.g. social, recreational, role) (Barch & Dowd, 2010; Gold, Waltz, Prentice, Morris, & Heerey, 2008). Whether such deficits also occur in CHR and predict greater severity of negative symptoms has yet to be examined.

One commonly employed measure of value representation is the DD task (Kirby & Marakovic, 1996). DD assesses an individual's preference for hypothetical smaller, temporally proximal rewards over larger, temporally remote rewards. Based on these preferences, a DD function (*k*-value) is generated to estimate the rate at which an individual discounts reward size based on the delay of reward receipt. Smaller *k*-values reflect a slower discounting rate, or a preference for larger delayed rewards (LDRs), whereas larger *k*-values reflect a steeper discounting rate, or a preference for smaller immediate rewards (SIRs). Outside of schizophrenia (SZ), the steeper discounting rate has been associated with lower intelligence (Shamosh & Gray, 2008), reduced working memory capacity (Shamosh et al., 2008), obesity (Amlung, Petker, Jackson, Balodis, & MacKillop, 2016), and greater impulsivity and addictive behavior (MacKillop et al., 2011).

Several studies have examined DD performance in SZ. Most have found that individuals with SZ have steeper discounting rates than healthy controls (Ahn et al., 2011; Avsar et al., 2013; Brown, Hart, Snapper, Roffman, & Perlis, 2018; Heerey et al., 2007; Weller et al., 2014; Yu et al., 2017); however, findings have not been consistent across all studies (Horan, Johnson, & Green, 2017; Kirschner et al., 2016; MacKillop & Tidey, 2011; Wang et al., 2018; Wing, Moss, Rabin, & George, 2012). Neuroimaging studies indicate that steeper discounting is associated with aberrant activation in brain regions associated with executive function (inferior frontal gyri, dorsal anterior cingulate cortex, posterior parietal cortex) and reward processing (ventral striatum and mid-brain) (Avsar et al., 2013). The steeper discounting rate has been associated with greater severity of cognitive impairment (Ahn et al., 2011; Brown et al., 2018; Heerey et al., 2007; Yu et al., 2017) and negative symptoms (Horan et al., 2017) in some studies; however, other studies have found null or inconsistent results (Heerey et al., 2007; Horan et al., 2017; Weller et al., 2014). Similar findings have emerged in first-degree relatives (Ho, Barry, & Koeppel, 2018) and individuals with high negative schizotypy traits (Cai et al., 2018) [however, see Wang et al. (2018) and Yu et al. (2017)]. Collectively, these findings suggest that impairments in value representation exist both in individuals with SZ, as well as first-degree relatives and those with schizotypal traits who are putatively at risk for psychosis. Given these prior findings and that value representation has been proposed to be a core facet of negative symptoms in adults with SZ (Gold et al., 2008), DD may offer insight into the mechanisms underlying negative symptoms in the prodromal phase.

Two studies were conducted to evaluate value representation impairments in adults with SZ (Study 1) and CHR youth (Study 2) using the DD paradigm. These studies had multiple aims. First, to replicate past findings of altered DD in SZ. Based on abundant evidence from previous studies, we hypothesized that individuals with SZ would discount rewards more steeply than controls. Second, to determine whether youth at CHR exhibits altered value representation. We predicted that CHR participants would also discount rewards more steeply than controls. Third, to determine whether DD is related to negative symptoms and cognition across phases of illness. We expected that the steeper discounting rate would be associated with more severe negative symptoms and greater cognitive impairment in both youth at CHR and adults with SZ. Additionally, we predicted that steeper discounting would be associated with higher risk for conversion in CHR participants using a cross-sectional psychosis risk calculator (Zhang et al., 2018).

Method

Study 1

Participants

Forty Healthy controls (CN) and 36 outpatients meeting DSM-5 criteria for schizophrenia or schizoaffective disorder (SZ) were included in the study. SZ participants were recruited from local outpatient mental health clinics or online/printed advertisements, and evaluated during periods of clinical stability (defined as no change in medication type of dose within the past 4 weeks). Diagnosis was established via a best-estimate approach based on psychiatric history and the Structured Clinical Interview for DSM-5 [SCID-5: (First, Williams, Karg, & Spitzer, 2015)]. All SZ patients had experienced multiple episodes and were generally in the chronic phase of illness.

Healthy control participants (CN) were recruited through printed and online advertisements. CN participants did not meet criteria for any current major psychiatric illnesses (i.e. mood, anxiety, obsessive compulsive disorders, etc.) or SZ-spectrum personality disorders as determined by the SCID-5 (First et al., 2015) and SCID-5-PD (First, Williams, Benjamin, & Spitzer, 2015). CN also had no family history of psychosis and did not meet lifetime criteria for psychotic disorders. Exclusion criteria for all participants included substance dependence in the last 6 months and lifetime history of neurological disorders associated with cognitive impairment (e.g. Traumatic Brain Injury, Epilepsy).

Of the 36 SZ and 40 CN, 3 SZ and 0 CN met criteria for inconsistent responding on the DD task. As it is common to exclude inconsistent responders (Kirby, 2000), these 3 SZ were not included in analyses. The final group included 33 SZ and 40 CN. These final groups did not significantly differ in age, parental

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Table 1. Study 1 demographic and clinical characteristics

	SZ ($n = 33$)	CN ($n = 40$)	Test statistic	p value
Age	39.4 (13.0)	39.0 (10.9)	$F = 0.02$	0.89
Parental education	14.4 (2.9)	13.5 (2.8)	$F = 1.68$	0.20
Participant education	13.3 (1.9)	15.7 (2.6)	$F = 19.36$	<0.01
% Female	72.7	77.5	$\chi^2 = 0.22$	0.79
Race (%)			$\chi^2 = 4.9$	0.43
Caucasian	63.6	50		
African American	24.2	20		
Asian American	0	5		
Hispanic/ Latino	6.1	12.5		
Biracial	6.1	7.5		
Other	0	5		
MCCB t-score	43 (14.6)	51.8 (9.9)	$F = 9.08$	<0.01
Symptom ratings				
BNSS total.	14.4 (13.4)			
LOF total	24.8 (6.9)			
LOF work	4.2 (3.2)			
LOF social	6.0 (2.5)			
PANSS total	63.7 (12.6)			
PANSS positive	16.8 (5.4)			
PANSS general	33.4 (8.3)			
PANSS disorganized	6.6 (2.6)			

SZ, schizophrenia group; CN, control group; MCCB, MATRICS Consensus Cognitive Battery; BNSS, Brief Negative Symptom Scale; LOF, Level of Function Scale; PANSS, Positive and Negative Syndrome Scale.
Note. A total of 19 SZ were prescribed second-generation antipsychotics and 1 was on both first- and second-generation antipsychotics. Thirteen SZ participants were stably unmedicated and not prescribed an antipsychotic at testing.

education, sex, or ethnicity; however, SZ had lower personal education than CN (see Table 1).

Procedure

All participants provided written informed consent and received monetary compensation for their participation. Study procedures were approved by the University of Georgia Institutional Review Board. Participants completed a series of clinical interviews conducted by a clinical psychologist (GPS) or examiners trained to reliability standards (>0.80) using gold standard training videos developed by the PI (GPS). In cases of the latter, examiners consulted with the PI for consensus. To establish diagnosis, all participants were rated on SCID-5 criteria (First et al., 2015), and SCID-5-PD (First et al., 2015) SZ-spectrum criteria for CN only. SZ participants were also rated on the Brief Negative Symptom Scale [BNSS: (Kirkpatrick et al., 2010)], Positive and Negative Syndrome Scale [PANSS: (Kay, Fiszbein, & Opler, 1987)], and Level of Function Scale [LOF: (Hawk, Carpenter, & Strauss, 1975)]. Participants then completed the MATRICS Consensus Cognitive Battery [MCCB: (Nuechterlein et al., 2008)] and the DD task (Kirby, Petry, & Bickel, 1999). The 5-factor model of the PANSS was used to examine symptom associations (Wallwork, Fortgang, Hashimoto, Weinberger, & Dickinson, 2012).

Delay discounting task

All participants completed a computerized version of the DD task for hypothetical monetary rewards (Kirby et al., 1999). Over the course of 27 trials, subjects were asked to choose between a SIR and a LDR by responding via left and right button presses on a keyboard. SIRs included rewards of three sizes: small ($11–34), medium ($20–54), and large ($31–80). LDRs also included rewards of three sizes: small ($25–35), medium ($50–60), and large ($75–85). Based on methods from Kirby (2000), each choice was assigned a k-value, which represents the hyperbolic discount parameter at indifference between choosing LDR and SIR values, and is calculated using the equation by Mazur, Mazur, and Nevin (1987):

$$k = \left(\left(\frac{\text{LDR}}{\text{SIR}}\right) - 1\right)/\text{Delay}$$

Subjects' k-values were estimated independently for each reward size by calculating the geometric mean of the largest k-value at which they chose the LDR and the smallest k-value at which they chose the SIR. For example, if an individual chose the LDR on a question with a k-value of 0.1 (a choice consistent with k smaller than 0.1), and then chose the SIR at the next smallest k-value of 0.041, the participant's k-value would be determined by taking the geometric mean of the two k-values (0.1 and 0.041). Therefore, a person selecting the aforementioned choices would have an estimated k-value of 0.064. Monitoring response consistency is important for accurately estimating k-values. Inconsistent responding refers to switching between LDR and SIR choices multiple times. k-Values and consistency values were calculated for each individual within each reward size (small, medium, and large) based on procedures by Kirby (2000). Three participants in the SZ group demonstrated inconsistent response patterns (⩽89% consistent, determined a priori) and were excluded from analysis.

Data analysis

The discounting rate was analyzed using a 2 Group (SZ, CN) x 3 Reward Magnitude (small, medium, and large k values) repeated measures ANOVA. Bivariate correlations were used to determine associations between discounting rate and symptoms, functional outcome, and cognition.

Study 2

Participants

Forty-five healthy controls (CN) and 37 CHR youth were included in the study. CHR participants were recruited from two psychosis risk evaluation programs directed by the PI (GPS), which received referrals from local clinicians (e.g. Psychiatrists, Psychologists, Social Workers, School Psychiatrists) to perform diagnostic assessment and monitoring evaluations for youth displaying psychotic experiences. Additional recruitment methods included online and

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print advertisements, in-person presentations to community mental health centers, and calls or in-person meetings with members of the local school system (e.g. superintendent, principals). All CHR youth met criteria for a prodromal syndrome on the structured interview for prodromal syndromes [SIPS; (Miller et al., 2003)]: (1) attenuated positive symptoms (i.e. SIPS score of at least 3–5 on at least one positive symptom item, with worsening symptoms over the past year) ($n = 34$); (2) genetic risk and deterioration syndrome (i.e. 1st degree relative with a psychotic disorder and decline in global functioning over the past year) ($n = 2$); and (3) brief intermittent psychosis syndrome (i.e. SIPS score of 6 on at least one positive symptom item, with symptoms present at least several minutes a day at a frequency of at least once per month) ($n = 1$). None of the CHR participants met lifetime criteria for a DSM-5 psychotic disorder as determined via SCID-5 interview (First et al., 2015).

CN participants were recruited from the local community using posted flyers, newspapers advertisements, and electronic advertisements. CN participants had no current major psychiatric disorder diagnoses and no SZ-spectrum personality disorders as established by the SCID-5 (First et al., 2015) and SCID-5-PD (First et al., 2015), no family history of psychosis, and were not taking psychotropic medications. All participants were free from lifetime neurological disease. Groups did not significantly differ on age, ethnicity, sex, personal education, or parental education (see Table 2).

Table 2. Study 2 demographic and clinical characteristics

	CHR ($n = 37$)	CN ($n = 45$)	Test statistic	p value
Age	19.9 (1.9)	20.1 (1.6)	$F = 0.20$	0.66
Parental education	15.0 (2.7)	15.3 (2.2)	$F = 0.27$	0.60
Participant education	13.4 (1.7)	14.1 (1.5)	$F = 3.45$	0.07
% Female	75.7	77.8	$\chi^2 = 0.05$	1.0
Race (%)			$\chi^2 = 3.51$	0.48
Caucasian	73	71.1		
African American	0	4.4		
Asian American	13.5	17.8		
Hispanic/ Latino	10.8	6.7		
Biracial	2.7	0		
MCCB t-score	54.1 (8.7)	58.6 (6.3)	$F = 2.12$	0.16
Symptom ratings				
SIPS positive	10.4 (4.4)			
SIPS negative	6.4 (5.0)			
SIPS disorganized	4.0 (2.4)			
SIPS general	7.5 (4.9)			
BNSS total	12.9 (11.3)			
GFS: S	7.9 (1.4)			
GFS: R	8.0 (1.4)			

CHR, clinical high-risk group; CN, control group; MCCB, MATRICS Consensus Cognitive Battery; SIPS, Structured Interview for Prodromal Syndromes; BNSS, Brief Negative Symptom Scale; GFS: S, Global Functioning Scale: Social; GFS: R, Global Functioning Scale: Role.
Note. Twenty-six CHR participants were not on any psychiatric medication, one CHR participant was prescribed an antipsychotic, seven were prescribed an antidepressant, four were prescribed an anxiolytic, and two were prescribed mood stabilizers.

Procedure

Participants provided written informed consent and received monetary compensation for their participation. Study procedures were approved by the Binghamton University and University of Georgia Institutional Review Boards. Participants completed a structured clinical interview to rate the SCID-5 (First et al., 2015) and SCID-5-PD (First et al., 2015), SIPS (Miller et al., 2003), BNSS (Strauss & Chapman, 2018), Global Functioning Scale: Social [GFS:S (Cornblatt et al., 2007)], and Global Functioning Scale: Role [GFS:R (Cornblatt et al., 2007)]. Interviews were conducted by a clinical psychologist (GPS) or examiners trained to reliability standards (>0.80) using gold standard training videos developed by the PI. In cases of the latter, examiners consulted with the PI for consensus. A cross-sectional conversion risk prediction score was calculated for CHR participants using an algorithm by Zhang et al. (2018) that uses functional decline, positive, negative, and general symptom scale items from the SIPS. After the interview, participants completed the MCCB (Nuechterlein et al., 2008) and the DD task.

Delay discounting task

Task procedures were identical to study 1. All participants demonstrated consistent responding and were included in the analysis.

Data analysis

The analytic plan was identical to study 1.

Results

Study 1

The within-subjects effect of value level, $F\ (2, 71) = 18.61$, $p < 0.001$, $\eta_p^2 = 0.21$, and between-subjects effect of group, $F\ (1, 71) = 6.17$, $p = 0.02$, $\eta_p^2 = 0.08$, were significant. However, the 2 group (SZ, CN) X 3 value level (mall, medium, and large) interaction was nonsignificant, $F\ (2, 142) = 0.40$, $p = 0.67$, $\eta_p^2 = 0.01$. Pairwise contrasts used to follow-up the significant main effect of the value level indicated that discounting was steeper for large than medium, large than small, and medium than small values ($p < 0.001$ for all). One way ANOVA confirmed that SZ had steeper discounting than CN for large $F\ (1, 71) = 5.51$, $p = 0.02$, $\eta_p^2 = 0.07$, medium $F\ (1, 71) = 6.96$, $p = 0.01$, $\eta_p^2 = 0.10$, and small reward values $F\ (1, 71) = 4.51$, $p = 0.04$, $\eta_p^2 = 0.06$. These findings suggest that value representation is impaired in SZ and characterized by a preference for smaller, immediate rewards (see Fig. 1a).

In SZ, steeper discounting (measured via the average DD geomean score) was associated with poorer working memory ($r = -0.56$, $p < 0.001$), poorer global cognition ($r = -0.61$, $p < 0.001$), number of cigarettes smoked per day ($r = 0.49$, $p = 0.03$), and greater severity of BNSS asociality ($r = 0.39$, $p = 0.03$). Partialing out the effects of depression did not attenuate this significant correlation with asociality. The discounting rate was not

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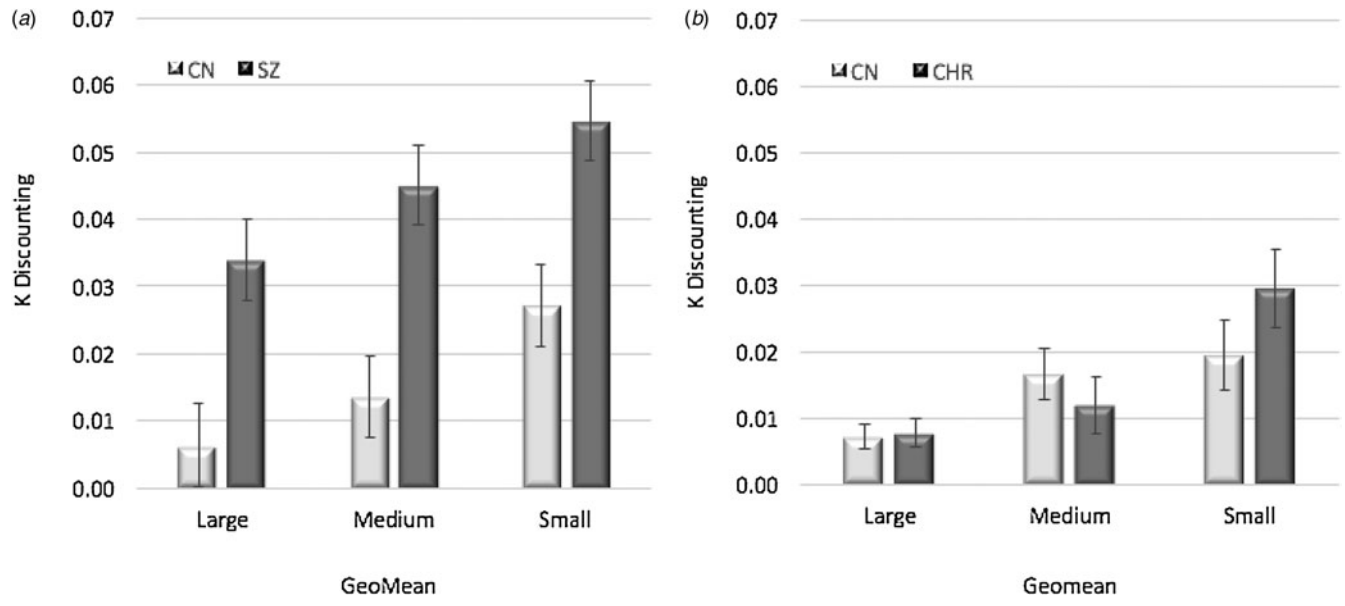

Fig. 1. Delay discounting in SZ and clinical high-risk for psychosis participants.
Note. SZ, schizophrenia group; CN, control group; CHR, clinical high-risk group. A, discounting rate by reward size and group for SZ and CN. B, discounting rate by reward size and group for CHR and CN. Results indicated that SZ had steeper discounting than CN for all 3 value levels (Fig. 1*a*); however, CHR and CN did not differ for any value level (Fig. 1*b*).

significantly associated with the other BNSS domains or total score, LOF scores, or other PANSS factors.

Study 2

The within-subjects effect of the reward value was significant, F (2, 80) = 19.78, $p < 0.001$, $\eta_p^2 = 0.20$. However, the between-subjects effects of group, F (1, 80) = 0.04, $p = 0.84$, $\eta_p^2 = 0.001$, and the group X reward value interaction, F (2, 80) = 1.48, $p = 0.23$, $\eta_p^2 = 0.02$, were nonsignificant. Pairwise contrasts used to follow-up the significant main effect of the value level indicated that discounting was steeper for large than medium, large than small, and medium than small values ($p < 0.001$ for all). The significant differences among value levels indicate that the DD task functioned as expected, but DD performance is unimpaired in CHR participants (see Fig. 1b).

In CHR, steeper discounting was associated with greater severity of BNSS anhedonia ($r = 0.34$, $p = 0.04$). Partialing out the effects of depression did not attenuate this significant correlation. The discounting rate was not associated with GFS: S or GFS: R scores, MCCB working memory or MCCB total score, SIPS positive, negative, disorganized, or general scores, or predicted conversion risk. The rate of smoking was too low for correlations with smoking behavior to be conducted in CHR.

Discussion

The primary goal of the current study was to determine whether similar mechanisms underlie negative symptoms across phases of psychotic illness. In particular, we focused on a reward processing deficit thought to be core to negative symptoms of SZ, value representation (Gold et al., 2008), and conducted two studies using the DD paradigm to determine whether value representation was: (1) impaired in adults with SZ (study 1) and CHR youth (study 2) and (2) associated with greater negative symptom severity and more severe cognitive impairment across phases of illness.

The majority of prior studies using DD in SZ have found that patients have steeper discounting rates than CN, consistent with a deficit in value representation (Ahn et al., 2011; Avsar et al., 2013; Brown et al., 2018; Heerey et al., 2007; Weller et al., 2014; Yu et al., 2017). The current study replicated the significant between group effect observed in most prior studies. Correlations between the discounting rate and clinical outcomes have been inconsistent in past SZ studies. Specifically, steeper discounting has been associated with negative symptoms and working memory impairment in several studies (Ahn et al., 2011; Brown et al., 2018; Heerey et al., 2007; Horan et al., 2017; Yu et al., 2017); however, findings have been nonsignificant or in the opposite direction in other studies (Heerey et al., 2007; Horan et al., 2017; Weller et al., 2014). In the current study, steeper discounting was associated with greater working memory impairment and more severe negative symptoms, replicating findings of several prior studies (Ahn et al., 2011; Brown et al., 2018; Heerey et al., 2007; Yu et al., 2017). Inconsistencies in group effects and negative symptom correlations among past DD studies may reflect methodological differences, such as use of first *v.* second generation negative symptom scales, whether *K*-values were normalized or not, and differences in equations used to calculate K, or DD task version.

Contrary to hypotheses, CHR did not differ from CN on the discounting rate. This may suggest that value representation is intact in the CHR population. Alternatively, it is possible that sampling bias led to an over-representation of CHR subjects with clinical and neural profiles that were less likely to show a deficit. Since only ~20% of CHR convert to a full psychotic disorder, it may be that value representation deficits are only pronounced in those who eventually transition. Longitudinal studies are needed to test this possibility. However, as hypothesized, greater severity of negative symptoms was associated with a steeper discounting rate, similar to SZ. Partialing out the effects of

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depression did not attenuate this significant correlation, suggesting that the increased rates of depression observed in the CHR population (Addington et al., 2017; Kline et al., 2018) do not account for this negative symptom effect. These findings suggest that value representation may be a key mechanism of negative symptoms transphasically.

Some have proposed that basic cognitive impairments, particularly in the domain of working memory, may play a role in DD deficits (Collins, Brown, Gold, Waltz, & Frank, 2014; Gold et al., 2008; Heerey et al., 2011). This appeared to be true in our SZ sample, but not our CHR sample. Our CHR sample, like many others (Brewer et al., 2006), did not display cognitive impairments of the same magnitude as SZ. Thus, it is possible that the intact DD performance observed in the CHR sample is a byproduct of relatively preserved working memory performance. As suggested by Collins et al. (2014), it may be that reward processing and working memory deficits are intricately interrelated and critical to the etiology of negative symptoms. Future studies are needed to examine this possibility using tasks that manipulate both reward and working memory demands concurrently in both longitudinal CHR and SZ samples.

Certain limitations should be considered. First, although this CHR data is part of a larger longitudinal study, only baseline cross-sectional data were available for this initial report. We were therefore unable to determine whether participants in our sample who will eventually transition to a psychotic disorder display value representation deficits, whereas non-converters do not. Future reports will address this question. Second, although only one CHR subject was prescribed an antipsychotic, we cannot rule out a potential role of antipsychotics in driving DD deficits occur in SZ but not CHR. Third, differences have been observed between DD tasks that manipulate hypothetical *v.* actual monetary rewards (Horan et al., 2017). We did not examine this manipulation in the current study, and it is possible that the clinical groups might behave differently in the presence of actual compared to hypothetical rewards that were implemented here. Finally, we conceptualized the DD task as a measure of value representation because it requires participants to generate mental representations related to both time and value, combine them, and use those representations to guide decision-making. However, some have conceptualized this task as a measure of impulsivity (Bickel, Odum, & Madden, 1999). We believe our conceptualization of the task as a measure of value representation accurately reflects the construct, as supported by the inclusion of this measure in the 'Reward Valuation' domain of the NIMH RDoC matrix. But, other tasks may measure value representation more purely by eliminating the temporal discounting element. Other tasks measuring the value representation construct, such as reversal learning (Culbreth, Gold, Cools, & Barch, 2016) might yield different results and should be evaluated in future studies

Identifying mechanisms underlying negative symptoms in CHR youth is an urgent need for our field. Negative symptoms are highly prevalent during this phase, one of the key factors that bring CHR youth into contact with the medical system, and a strong predictor of conversion (Demjaha, Valmaggia, Stahl, Byrne, & McGuire, 2010; Johnstone, Ebmeier, Miller, Owens, & Lawrie, 2005; Lencz, Smith, Auther, Correll, & Cornblatt, 2004; Piskulic et al., 2012; Schlosser et al., 2012; Valmaggia et al., 2013; Yung & McGorry, 1996). The current study is one of a series of studies by our group [see also Strauss, Ruiz, Visser, Crespo, and Dickinson (2018)] attempting to determine whether reward processing mechanisms related to negative symptoms in SZ are also impaired in CHR and associated with negative symptoms. Value representation may be a key mechanism contributing to negative symptoms transphasically, leading SZ and CHR to be less able to use mental representations of reward value to influence decision-making processes that are needed to initiate approach behaviors (e.g. goal-directed, social, or recreational activities).

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