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TEMPORAL DISCOUNTING AND CONDUCT DISORDER IN ADOLESCENTS

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Abstract

The current study examined temporal discounting (the decrease in subjective reward value as a function of increasing delay) in youths with conduct disorder (CD) and the extent to which this was modulated by level of psychopathic traits. In the temporal discounting task, participants were asked to choose between immediate rewards of varying values and a larger reward, held at a constant value ($10), whose receipt was delayed by different time intervals across trials (e.g., 7 days, 360 days). The level of immediate reward necessary for selection over the larger, delayed reward is the measure of temporal discounting. Forty-six youths (21 with CD and 25 healthy youths) participated in this study. Compared with healthy youths, youths with CD chose significantly smaller amounts of immediate reward rather than the larger future rewards. This was the case even in youths with CD without comorbid attention-deficit/hyperactivity disorder. However, level of psychopathic traits did not modulate temporal discounting in this sample. These results are discussed in terms of neurobiological models of CD and psychopathic traits.

It has been known for some time that youths with conduct problems show profound impairment in reinforcement-based decision making. This is seen, for example, on paradigms such as passive avoidance learning (Finger et al., 2011), the Iowa Gambling Task (R. J. R. Blair, Colledge, & Mitchell, 2001), reversal learning (Budhani & Blair, 2005), operant extinction (Fisher & Blair, 1998; O'Brien & Frick, 1996), and a form of social discounting task (Sharp et al., 2012). More recently, functional magnetic resonance imaging (fMRI) work has begun to underpin the neural correlates of this impairment. Finger and colleagues (2008), in an initial study using the reversal learning task, demonstrated notable dysfunction in youths with psychopathic traits (i.e., callous-unemotional traits, reduced guilt and empathy, and antisocial behavior) in the response of ventromedial prefrontal cortex (vmPFC) and striatum to reinforcement information. Striatum and vmPFC are critical for aspects of reinforcement processing (K. Blair et al., 2006; O'Doherty, 2004). Since then, a series of studies have reported reduced representation of reward information in youths with conduct problems within striatum and vmPFC (Carré, Hyde, Neumann, Viding, & Hariri, 2013; Crowley et al., 2010; Finger et al., 2008, 2011; Rubia et al., 2009). Importantly, recent *model-based* fMRI work has enabled identification of the computational impairments that

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are present within these regions in youths with CD (White et al., 2013). Specifically, youths with CD show impaired representation of expected value within vmPFC when choosing stimuli and reduced reward prediction error signaling within striatum (prediction errors reflect the difference between expected and received reinforcement; White et al., 2013). There are also structural neuroimaging findings of reduced striatal (Fairchild et al., 2011, 2013) and vmPFC volume (Fahim et al., 2011; Huebner et al., 2008; Hyatt, Haney-Caron, & Stevens, 2012) in CD. The current study examined the relationship among CD, psychopathic traits, and a specific type of reward processing, temporal discounting.

Temporal reward discounting (TD) refers to the decrease of subjective reward value as a function of increasing delay (Critchfield & Kollins, 2001). TD can be measured by asking participants to choose between an immediate reward or a delayed reward of greater value (Mitchell, 1999). By varying both the time delay and the amount of reward, a switch point can be calculated (Mitchell, 1999). The switch point indicates the amount of money that is equally preferable now to a standard amount later (e.g., a 7-day switch point of $9.75 indicates that $9.75 now is equal to $10 in 7 days). Lower switch points have been used to indicate one type of impulsivity, temporal difference impulsivity (TDI). TDI has been seen both in patients with substance abuse disorders (for a review, see MacKillop et al., 2011) and in those with attention-deficit/hyperactivity disorder (ADHD; Barkley, Edwards, Laneri, Fletcher, & Metevia, 2001; Demurie, Roeyers, Baeyens, & Sonuga-Barke, 2012).

Surprisingly, there is less clear evidence of a relationship between TDI and antisocial behavior. There have been no previous studies with youths with CD, and only one study has investigated the relationship between antisocial personality disorder in adults and TDI. Petry (2002) found greater TDI in substance abusers with antisocial personality disorder relative to those without, while both groups showed greater TDI than healthy controls.

As noted, there is clear evidence that clinical and forensic samples of youths and adolescents with psychopathic traits show impairment on reinforcement-based decision-making tasks (R. J. R. Blair et al., 2001; Budhani & Blair, 2005; Finger et al., 2008, 2011; Fisher & Blair, 1998; O'Brien & Frick, 1996). However, previous investigations of temporal discounting and psychopathic traits have not clearly indicated greater TDI in individuals with elevated psychopathic traits relative to controls. Thus, studies have investigated the impact of psychopathic traits on the severity of TDI in heroin addicts (Vassileva et al., 2007), smokers (Melanko, Leraas, Collins, Fields, & Reynolds, 2009; Vassileva et al., 2007), and healthy participants (Miller & Lynam, 2003; Morgan, Gray, & Snowden, 2011). Level of psychopathic traits was related to level of TDI in the two studies of healthy participants (Miller & Lynam, 2003; Morgan et al., 2011), but not in the studies with smokers or heroin addicts (Melanko et al., 2009; Vassileva et al., 2007).

The lack of a consistent relationship between TDI and antisocial behavior is surprising because appropriate representation of future reward magnitude appears to rely on the responsiveness of striatum (nucleus accumbens) and vmPFC (for a review, see Peters & Buchel, 2011). Youths with CD show reduced representation of reward information within striatum and vmPFC (Crowley et al., 2010; Finger et al., 2008, 2011; White et al., 2013). The suggestion is that the stronger the neural representation of the future reward, the more

likely an individual is to choose to delay receiving a future reward. In line with this view, individuals showing greater TDI show weaker striatal responsiveness to future reward (e.g., Ballard & Knutson, 2009). Moreover, patients with lesions of vmPFC show increased TDI (Sellitto, Ciaramelli, & di Pellegrino, 2010). This would thus predict increased TDI in youths with CD. However, to our knowledge, this prediction has not been formally tested.

The goal of the current study was to examine TD in youths with CD. Given that previous data indicate reduced neural representation of reward information within striatum and vmPFC in youths with CD (Crowley et al., 2010; Finger et al., 2008, 2011; White et al., 2013), we predicted that youths with CD would show increased TDI. Given hypotheses relating psychopathy to dysfunction within striatum and vmPFC (Anderson & Kiehl, 2012; R. J. R. Blair, 2007), we additionally predicted that level of psychopathic traits might relate to extent of TDI in the youths with CD.

METHODS

PARTICIPANTS

Forty-six youths participated: 21 youths with CD and 25 healthy comparison youths (Table 1). Youths were recruited from the community through newspaper ads, fliers, and referrals from area mental health practitioners. A statement of informed assent and consent was obtained from participating children and parents. This study was approved by the National Institutes of Health Institutional Review Board.

All youths and parents completed Kiddie Schedule for Affective Disorders and Schizophrenia (K-SADS; Kaufman et al., 1997) assessments with an experienced clinician, trained and supervised by expert child psychiatrists, with good interrater reliability (kappa > 0.75 for all diagnoses). The K-SADS assesses for substance abuse and substance dependence but, due to exclusion criteria, no children in either group met criteria for these diagnoses. IQ was assessed with the two sub-test form of the Wechsler Abbreviated Scale of Intelligence (WASI; Wechsler, 1999). Exclusion criteria were pervasive developmental disorder, Tourette's syndrome, lifetime history of psychosis, depression, bipolar disorder, generalized, social, or separation anxiety disorder, posttraumatic stress disorder, neurological disorder, history of head trauma, and IQ < 80. Youths in the conduct disorder group met *DSM-IV* criteria as assessed by the K-SADS. Comparison subjects did not meet criteria for any K-SADS diagnosis. The groups did not differ on age [$t(44) = 1.747$, $p = .09$], but the CD group did have a significantly lower IQ [$t(44) = 4.221$, $p < .001$], a greater proportion of minority youths [$\chi^2 = 7.98$, $p = .01$], and a greater proportion of males [$\chi^2 = 5.32$, $p = .03$] relative to the healthy controls (see Table 1). Parents also completed the Antisocial Process Screening Device (APSD; Frick & Hare, 2001), a measure of psychopathic traits.

STUDY MEASURES

Antisocial Process Screening Device (Frick & Hare, 2001)—The APSD is a 20-item parent-reported rating of callous-unemotional traits and conduct and impulsivity problems, designed to detect psychopathic traits in youths. A three-factor structure has been

characterized and comprises the following dimensions: callous-unemotional (CU), narcissism (NAR), and impulsivity (IMP). The parent-report version of the APSD has been found to have adequate overall internal consistency (α = .85–.89) and subscale internal consistency (CU, α = .72–.76; NAR, α = .79–.82; IMP, α = .65–.75; Munoz & Frick, 2007).

Delayed Discounting Task (Bjork, Momenan, & Hommer, 2009)—Temporal discounting was assessed using the computer-based delayed discounting task (Figure 1). Participants were required to choose between immediate rewards of varying values ($0, $0.25, and from $0.50 to $10.50 in varying $0.50 increments) and a larger reward, held at a constant value ($10), whose receipt was delayed by different time intervals across trials (0, 7, 30, 90, 180, 360 days). Participants were made aware that they would not receive the money they won in the task. The immediate reward appeared on the left side of the screen in 50% of trials and on the right side of the screen for the other 50% of trials (the delayed reward was on the other side of the screen). The side of the screen that the immediate reward appeared on changed randomly throughout the task. All possible combinations of time delays and reward amounts were presented randomly for a total of 138 trials. Each trial ended when participants made a selection. For each of the six delay intervals, a switch point was calculated, defined as the dollar amount above which the participant opted for the immediate reward over the standard amount at a delay (the $10 at 0 delay versus $10 now combination was not considered in calculations). This can be considered the "worth" of the standard $10 amount at that particular delay interval. A lower switch point is indicative of greater TDI.

RESULTS

PRELIMINARY ANALYSES

Mean duration of the task across all participants was 560.91 seconds (*SD* = 196.86), and duration did not significantly differ between healthy and CD youths [*t* = .018, *p* = .986].

MAIN ANALYSIS

A 6 (delay interval: 0, 7, 30, 90, 180, 360 days) × 2 (group: CD youths, healthy comparison youths) analysis of covariance (ANCOVA) was conducted on switch point scores while including IQ and level of psychopathic traits as covariates (see Figure 2). A main effect of delay interval was observed [$F(5, 38) = 2.780$, $p = .019$], where participants required increasing amounts of money to accept increasing delays in gratification. Critically, a significant main effect was observed for group, where CD youths showed significantly greater TDI relative to healthy youths [$F(5, 38) = 7.589$, $p = .009$] and a significant interval-by-group interaction was observed [$F(5, 38) = 2.347$, $p = .042$]. Significantly lower switch points were observed for CD youths at every delay interval [*t*s from 3.144–4.363, $p = .003$ – < .001] except for the 0 days interval [$t(37) = .881$, $p = .383$]. Against hypotheses, no significant effect of psychopathic traits [$F(5, 38) = 1.419$, $p = .240$] or interval-by-psychopathic traits interaction [$F(5, 38) = .789$, $p = .558$] was observed. No significant effect was observed for IQ [$F(5, 38) = .243$, $p = .624$]. No significant interactions were observed for interval-by-IQ [$F(5, 38) = .458$, $p = .807$].

This analysis was repeated three times, using each of the APSD's subscales, callous-unemotional, impulsive/antisocial, and narcissistic, as the covariate in place of the APSD total score. For all three subscales, a main effect of delay interval was observed [*F*s = 3.56–4.25, *p*s = .047–.003], where participants required increasing amounts of money to accept increasing delays in gratification. A significant main effect was observed in each analysis for group, where CD youths showed significantly greater TDI relative to healthy youths [*F*s = 10.02–12.62, *p*s = .003–.001]. Significant delay interval-by-group interactions were observed for narcissism and callous-unemotional [*F*s = 3.01 & 3.07, *p*s = .012 & .011, respectively]. Significantly lower switch points were observed for CD youths at every delay interval. The delay interval-by-group interaction for impulsive/antisocial was observed at a very weak trend [$F = 1.95$, $p = .172$]. No significant effects were observed for any of the APSD subscales [*F*s = .57–.98, *p*s = .456–.202], nor were any interval-by-psychopathic traits [*F*s = .32–.60, *p*s = .640–.701] interactions. No significant main effects of IQ [*F*s = .35–.40, *p*s = .561–.532] and no significant interaction effects for interval-by-IQ [*F*s = .69–.78, *p*s = .630–.564] were observed.

SYMPTOM SEVERITY AND BEHAVIORAL RESPONSE IN CD YOUTHS

In order to investigate the impact of psychopathic traits on TDI in youths with CD, four repeated-measures ANCOVAs examining the impact of delay interval (0, 7, 30, 90, 180, 360 days) using APSD total and subscale scores (Narcissism, Impulsive/Antisocial, & Callous-Unemotional traits) as covariates for the 21 youths meeting CD criteria. Significant main effects were observed for delay interval [*F*s = 3.491–5.988, *p*s < .01]; however, no main effect of APSD total/subscale scores [*F*s = .176, .009, .710, .182, *p*s > .675] or delay interval-by-APSD total/subscale scores interaction were observed [*F*s = .181–.777, *p*s > .569].

POTENTIAL CONFOUNDS

Given the significant and substantial differences in IQ, gender, and race between the groups, the main analysis was repeated with an IQ- [$t(30) = 1.330$, $p = .19$], gender- [$\chi^2 = 3.24$, $p = .15$], and race-matched sample [$\chi^2 = 2.67$, $p = .22$] of 16 healthy and 16 CD youths. Replicating the results of the main analysis, a main effect was observed for delay interval [$F(5, 25) = 12.706$, $p = < .001$], where participants required increasing amounts of money to accept increasing delays in gratification [*t*s from 2.02–5.76, $p = .05 – < .001$]. Importantly, a significant main effect was again observed for group, where CD youths showed significantly greater TDI relative to healthy youths [$F(5, 25) = 7.163$, $p = .012$]. Again, no effect of psychopathic traits was observed [$F(5, 25) = 1.490$, $p = .232$], nor was a significant interval-by-psychopathic traits interaction observed [$F(5, 25) = 1.207$, $p = .281$]. However, an interval-by-group interaction approached significance [$F(5, 25) = 3.834$, $p = .060$], where significantly lower switch points were observed for CD youths at every delay interval [*t*s from 2.617–3.349, $p = .038–.002$] except the 0 days interval [$t(30) = 1.103$, $p = .279$].

Because five of the CD youths also met criteria for ADHD, the main analysis was repeated excluding these participants. Again, similar to the results of the main analysis, a main effect was observed for delay interval [$F(5, 33) = 3.253$, $p = .008$], where participants required increasing amounts of money to accept increasing delays in gratification [*t*s from 3.303–

6.043, $p = .002 – \leq .001$], although no difference was observed in the switch points between a delay of 90 and 180 days [$t(42) = 1.083$, $p = .285$]. Also, a significant main effect was observed for group, where CD youths showed significantly greater TDI relative to healthy youths [$F(5, 33) = 6.4221$, $p = .016$]. Again, no effect of psychopathic traits [$F(5, 33) = 1.053$, $p = .311$] or IQ [$F(5, 33) = .539$, $p = .467$] was observed, nor were significant interval-by-psychopathic traits [$F(5, 33) = .467$, $p = .800$] or interval-by-IQ [$F(5, 33) = .793$, $p = .556$] interactions observed. When ADHD youths were removed, the interval-by-group interaction fell to weak trend levels [$F(5, 33) = 1.791$, $p = .117$].

Because of the potential concern that youths with CD might have shown response biases or task boredom that exaggerated over time, we examined the first 50% of trials versus the second 50% of trials in both healthy and CD youths (excluding the 0 days delay interval trials). We conducted a 2 (diagnosis: CD, healthy) × 2 (half: first half, second half) analysis of variance (ANOVA). Consistent with the analyses we have described, this ANOVA revealed a significant main effect of diagnosis [$F = 15.09$, $p < .001$]; youths with CD were significantly more likely to choose immediate rewards than comparison youths. There was also a main effect of half; all youths were more inclined to delay reward in the second half of the task relative to the first [$F = 5.976$, $p = .019$]. There was no significant diagnosis-by-half interaction [$F = 1.238$, $p = .273$]. Importantly, these data show that from the first to the second half of trials, the tendency of youths with CD to choose immediate rewards diminished.

DISCUSSION

The current study examined whether youths with CD showed TDI and whether level of TDI related to level of psychopathic traits. There were two main findings. First, as expected, youths with CD showed greater levels of TDI relative to healthy control youths. Second, and against predictions, psychopathic traits showed no relationship to TDI.

In line with data showing reduced representation of reward information within striatum and vmPFC in youths with conduct problems (Carré et al., 2013; Crowley et al., 2010; Finger et al., 2008, 2011; Rubia et al., 2009; White et al., 2013), the current study revealed increased TDI in youths with CD. Striatum and vmPFC appear critical for the appropriate representation of future reward magnitude, and it is the strength of this representation that determines whether the individual waits for the higher future reward or prefers the lower immediate reward (for a review, see Peters & Buchel, 2011). Given fMRI and neuropsychological findings (Ballard & Knutson, 2009; Sellitto et al., 2010), dysfunction in these systems in CD should result in the observed TDI.

There have been suggestions that psychopathic traits are related to dysfunction within striatum and vmPFC (Anderson & Kiehl, 2012; R. J. R. Blair, 2007). Given this possibility, we predicted that we would see a relationship between level of TDI and level of psychopathic traits. However, this prediction was not supported. This might represent a Type II error. Yet it is worth noting that the three studies examining youths with CD (and oppositional defiant disorder) also found no relationship between level of psychopathic traits in the youths with CD and extent of pathophysiology within striatum and vmPFC (Finger et

al., 2008, 2011; White et al., 2013). A fourth study of healthy young adults (Carré et al., 2013) found no relationship between striatal activity to reward and level of callous-unemotional traits (reduced guilt and empathy, the emotional core of psychopathy). However, Carré et al. did report that increased levels of antisocial and impulsive behavior were associated with reduced striatal activity. In short, difficulties in the representation of reward may underpin decision-making difficulties in antisocial individuals irrespective of whether they also present with psychopathic traits. In line with this, it is worth noting that both of the studies on substance abusing populations found that TDI was related to substance abuse but was not exacerbated by level of psychopathic traits (Melanko et al., 2009; Vassileva et al., 2007).

Psychopathic traits have been associated with both emotional and cognitive deficits (R. J. Blair, 2010). Recent evidence has bolstered support for a primary emotion account of psychopathic traits (Sylvers, Brennan, & Lilienfeld, 2011; White et al., 2012), and there is relatively consistent data showing that the core emotional component of psychopathic traits, callous-unemotional traits (reduced guilt and empathy), relates to reduced processing of distress cues. Specifically, level of CU traits predicts amygdala response to fearful expressions in both clinical (Marsh et al., 2008; White et al., 2012) and subclinical samples (Jones, Laurens, Herba, Barker, & Viding, 2009; Sebastian et al., 2012; Viding et al., 2012). The current study and several others have failed to link psychopathic traits, including callous-unemotional traits, to the *severity* of decision-making deficits associated with CD (Fairchild et al., 2009; White et al., 2013). Furthermore, disruption in decision making has been found consistently in substance abusing populations (Crowley et al., 2010; Schutter, van Bokhoven, Vanderschuren, Lochman, & Matthys, 2011) even when directly examining psychopathic traits (Melanko et al., 2009; Vassileva et al., 2007). This is not to say that these deficits are not present in psychopathy. They clearly are (K. S. Blair, Morton, Leonard, & Blair, 2006; Epstein, Poythress, & Brandon, 2006; Newman & Kosson, 1986; Newman, Widom, & Nathan, 1985), and youths with CD and psychopathic traits show profound impairment in the representation of reinforcement information within caudate and vmPFC (Finger et al., 2008, 2011; White et al., 2013). Instead, we would suggest that these deficits might also be present in other populations engaged in externalizing behaviors, such as CD with low callous-unemotional traits and substance abuse.

It is worth briefly considering ADHD in this context. A series of studies have reported TDI in patients with ADHD (Barkley et al., 2001; Demurie et al., 2012; Scheres, Tontsch, Thoeny, & Kaczkurkin, 2010). ADHD is typically highly comorbid with CD (Connor, Ford, Albert, & Doerfler, 2007; Waschbusch, 2002). Other studies have reported reduced striatal activity to reward anticipation in individuals with ADHD (Plichta et al., 2009; Scheres, Milham, Knutson, & Castellanos, 2007; Strohle et al., 2008). This may underpin their TDI (see Demurie et al., 2012). This is interesting because while ADHD may not be related to impairment in reinforcement processing within vmPFC, a shared impairment in striatal responsiveness has been seen both in youths with CD and in youths with ADHD (see Finger et al., 2008). In other words, it can be speculated that some of the comorbidity may represent a shared impairment in striatal reward signaling and consequent “impulsive” decision making as a function of this deficit. Suggestive of this possibility, a recent study found that

TDI was related to hyperactivity/impulsivity, but not to inattentive ADHD symptoms (Scheres et al., 2010). Indeed, it is possible that this impairment may underpin the comorbidity seen between CD and ADHD and other externalizing disorders such as substance abuse.

It is worth briefly considering whether the results might be due to response biases, particularly with respect to boredom as the task progressed. Although this is possible, it appears unlikely for two main reasons: First, the location of the immediate and the delayed rewards differed across trials (i.e., sometimes the delayed choice was on the left-hand side and sometimes on the right-hand side of the screen). This reduces the possibility of a response bias to a particular spatial location. Second, our analysis examining the first versus the last 50% of trials revealed that the tendency of youths with CD to choose immediate rewards actually diminished. In other words, there was an increase in the preponderance of a specific response over time in the youths with CD. However, *the increase was for the more appropriate, delayed response* (which they still showed less than the comparison youths).

Two caveats should be considered with respect to the current data. First, the healthy control youths had significantly higher IQ scores than the CD youths. However, mitigating this caveat, WASI scores were included as a covariate in the main analysis and were not significant. Further mitigating this caveat, our subsequent analysis in an IQ-matched sample yielded results very similar to those of the main analysis. Second, an ADHD comparison group was not included in the current study. Although our results could be attributed to comorbid ADHD, the TDI was present even in youths with CD without comorbid ADHD. It would be interesting to determine whether the severity of TDI is comparable in both disorders. On this note, it would be extremely interesting to determine, using fMRI, the extent of overlap in pathophysiology related to reward processing in these two disorders.

Three limitations to the current study deserve consideration. First, no data on socioeconomic status were collected. Therefore, from the current study is not possible to assess the potential impact of this variable. Second, the modest sample size of the current study may have limited our ability to find a significant effect of psychopathic traits. Finally, the current study did not have a dimensional assessment of CD severity.

It would be interesting to note if the extent of TDI in CD is related to symptom severity. In conclusion, the current study revealed increased temporal discounting impulsivity in youths with CD and showed that this was not affected by severity of psychopathic traits. Overlap in impairment in reward processing may underpin the high comorbidity seen in the externalizing disorders of CD, ADHD, and substance abuse. However, future fMRI work will be important in determining whether this overlap reflects dysfunction in the neural systems involved in reward processing (ventromedial prefrontal cortex, striatum, amygdala, posterior cingulate cortex; K. Blair et al., 2006; O'Doherty, 2004) or more selective disorder-specific dysfunction within particular neural components of this circuitry.

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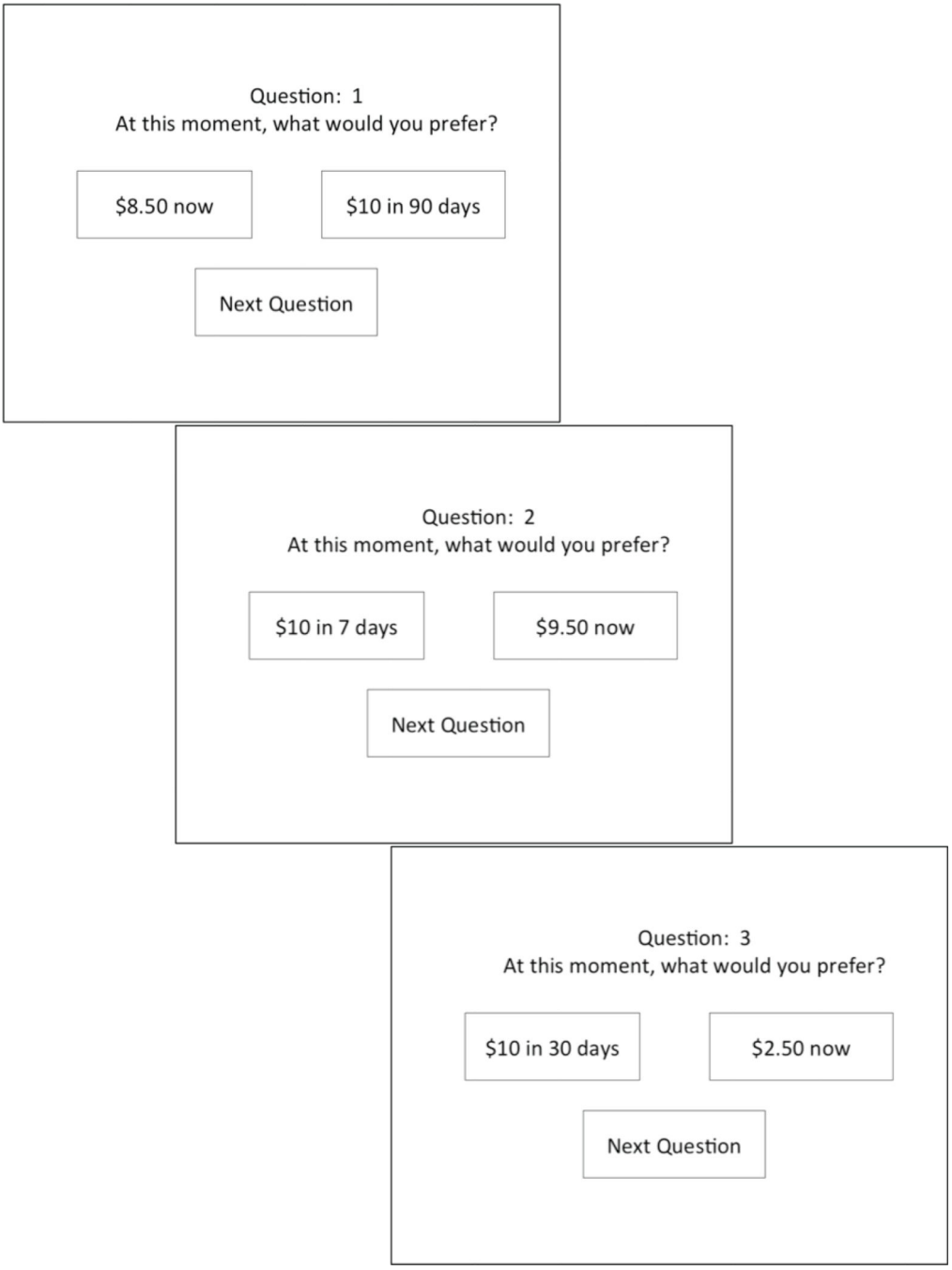

FIGURE 1.

Delayed discounting task.

Participants were required to choose between a smaller, but immediate reward and a larger, standard reward at various delay intervals.

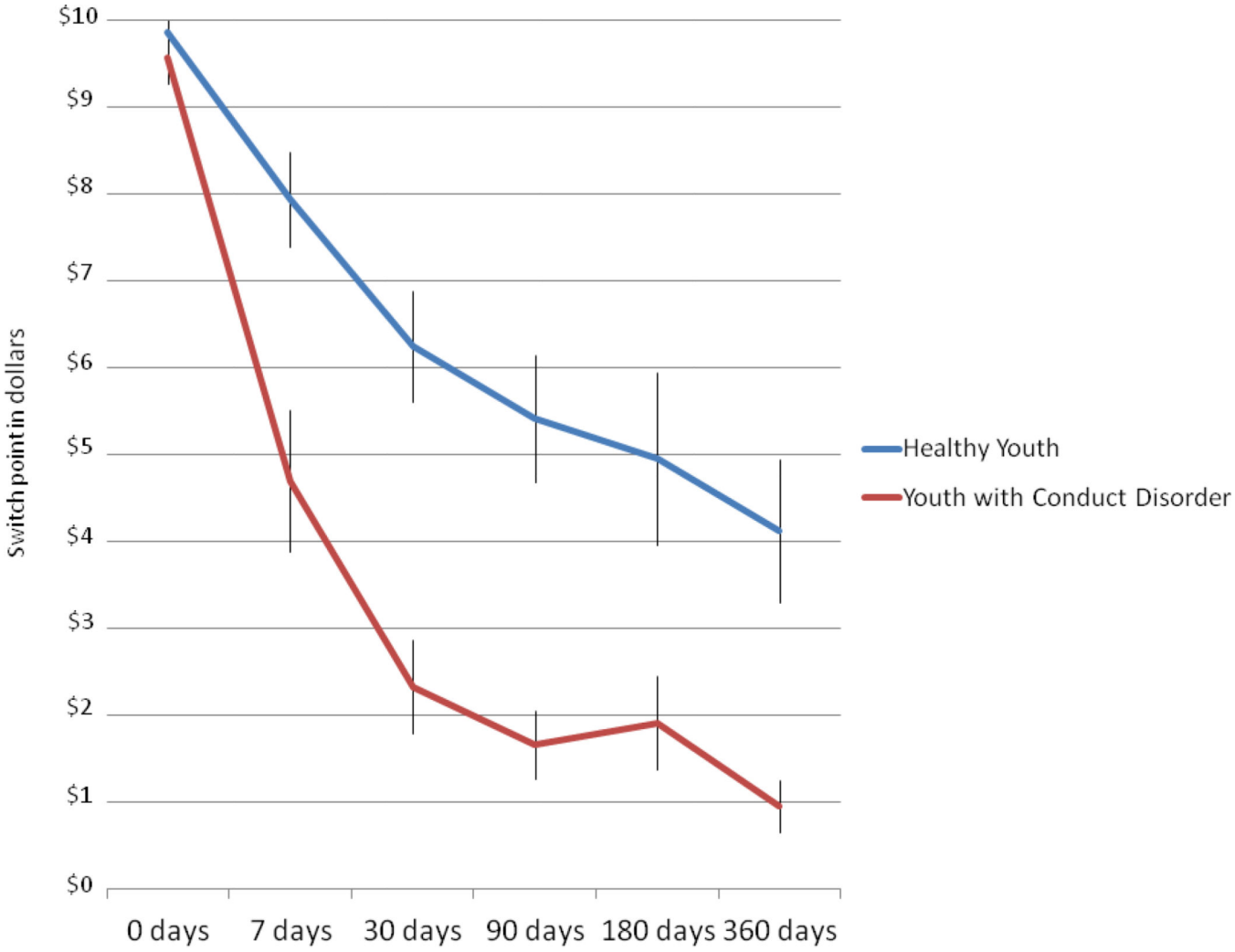

FIGURE 2.

Switch points for the Delayed Discounting Task from 0- to 360-day delay intervals for 21 youths with conduct disorder and 25 healthy youths. Switch points reflect the amount of money (in $) that will be chosen now rather than waiting for the time delay for $10. Error bars indicate standard error of the mean.

TABLE 1

Characteristics of Youths with CD and Healthy Youths

	Youths with CD		Healthy Youths	
	***N* = 21**		***N* = 25**	
Characteristic	**Mean**	**(*SD*)**	**Mean**	**(*SD*)**
Age (years)	15.67	(2.04)	14.53	(2.33)
IQ[a]	90.91**	(8.33)	105.80**	(14.24)
Antisocial Process Screening Device Total and Subscale Scores				
Total score	25.61**	(5.18)	4.92**	(4.35)
CU	7.37**	(2.36)	2.68**	(3.39)
Narcissistic	7.16**	(3.47)	2.16**	(3.04)
Impulsive	6.47**	(2.48)	2.60**	(2.27)
	N	%	*N*	%
Gender	18 male*	78.3	12 male*	48.0
ADHD	5	23.81	0	0

Note.

SD = standard deviation.

[a]Assessed with the Wechsler Abbreviated Scale of Intelligence (two-subtest form).

*significantly different at $p < .05$.

**significantly different at $p < .001$.