Disinhibitory Psychopathology and Delay Discounting in Alcohol Dependence: Personality and Cognitive Correlates

Lyuba Bobova, Peter R. Finn, Martin E. Rickert, and Jesolyn Lucas
Indiana University, Bloomington

Increased discounting of delayed rewards may reflect a decision bias that contributes to excessive use of alcohol and more generally, to an impulsive, disinhibitory predisposition that is characterized by a preference for immediate over long-term rewards. The current study examined the association between delay discounting of rewards and the covariation among several types of disinhibitory problems that are often comorbid with alcohol dependence (AD). Lifetime problems with alcohol, marijuana, other drugs, childhood conduct disorder, and adult antisocial behavior were assessed in a sample of 426 young adults, 257 of whom had a lifetime diagnosis of AD. Higher delay discounting rates were associated with the covariation among all domains of disinhibitory problems and were not uniquely associated with any one domain. Higher delay discounting rates also were associated with lower intelligence, lower working memory capacity, and higher trait impulsivity. The results suggest that increased delay discounting of rewards may reflect aspects of a general vulnerability to externalizing, disinhibitory disorders.

Keywords: delay discounting, alcoholism, comorbidity, impulsivity, antisocial behavior

Individuals with alcohol dependence (AD) tend to discount future rewards relative to immediate rewards at higher rates compared to those without AD (Bjork, Hommer, Grant, & Danube, 2004; Finn, 2002; J. M. Mitchell, Fields, D'Esposito, & Boettiger, 2005; Petry, 2001). This pattern of increased discounting of delayed rewards may reflect decision biases that contribute to excessive use of alcohol and more generally, to an impulsive predisposition that is characterized by a preference for immediate over long-term rewards and reduced cognitive capacity (Finn, 2002; J. M. Mitchell et al., 2005).

However, because increased delayed discounting of rewards is thought to be a key component of impulsivity (Crean, de Wit, & Richards, 2000; S. H. Mitchell, 1999; Reynolds, 2006b) and because AD is highly comorbid with other disinhibitory disorders (Krueger et al., 2002), high rates of delay discounting also should be associated with other forms of disinhibitory psychopathology, such as childhood conduct disorder (CCD). In fact, considerable evidence suggests that this is the case. Abuse of and dependence on other substances, such as opiates, stimulants, and tobacco, also has been associated with increased delay dis-

counting rates (Bornovalova, Daughters, Hernandez, & Richards, 2005; Epstein et al., 2003; Heil, Johnson, Higgins, & Bickel, 2006; Hoffman et al., 2006; Johnson, Bickel, & Baker, 2007; Kirby & Petry, 2004; Kirby, Petry, & Bickel, 1999; Madden, Petry, Badger, & Bickel, 1997; S. H. Mitchell, 1999; Ohmura, Takanashi, & Kitamura, 2005; Reynolds, 2006a; 2006b; Reynolds, Karraker, Horn, & Richards, 2003). Furthermore, several studies show that elevated discounting rates are associated with other forms of disinhibitory psychopathology including antisocial personality disorder (Crean et al., 2000; Dom, De Wilde, Hulstijn, van Brink, & Sabbe, 2006a; 2006b; Petry, 2002).

The primary aims of this study were to investigate the association between delay discounting and disinhibitory disorders that are comorbid with AD and to explore the personality and cognitive correlates of delay discounting. The overarching hypothesis was that increased delay discounting will be associated with different disinhibitory phenotypes, such as AD, CCD, other disinhibitory problems/symptoms, and self-reported impulsivity, and reductions in cognitive ability associated with behavioral disinhibition. The central thesis was that increased delay discounting rates reflect specific processes that contribute to impulsivity, which is a core feature of disinhibitory disorders (Finn, 2002).

The basic study design was a 2 × 2 (AD:CCD) independent-groups model to enable an evaluation of the relative association between delay discounting and both AD and CCD. CCD was chosen as an indicator of antisocial disinhibitory psychopathology because our sample is younger (ages 18 to 30 years old) and although a history of CCD is a prerequisite for adult antisocial personality, CCD is often not assessed in studies of early-onset AD. The first hypothesis was that the combination of AD and a history of CCD

would be associated with higher delay discounting rates than AD without CCD because the combination of AD and CCD represents a more severe form of disinhibitory psychopathology (Finn, Mazas, Justus, & Steinmetz, 2002).

However, recent research suggests that the high level of covariation among externalizing disorders is better conceptualized as a single latent dimension of disinhibitory problems rather than as the co-occurrence of distinct disorders (Krueger et al., 2002; Krueger & Markon, 2006; Krueger, Markon, Patrick, & Iacono, 2005). Although some studies have assessed delay discounting in AD with and without comorbid antisocial personality or other cluster B personality disorders (Dom et al., 2006a; 2006b; Petry, 2002), these studies have failed to directly evaluate the relative influence of delay discounting on specific domains of comorbid externalizing problems, or the covariance among comorbid externalizing problems. This study addressed this issue by using structural equation modeling to examine the association between delay discounting and a single latent disinhibitory disorders factor that reflected the covariation among externalizing problems. Our recruitment strategy was designed to ensure sufficient variation and range in severity of lifetime problems with alcohol, marijuana, other drugs, CCD, and adult antisocial behavior. The second hypothesis was that increased delay discounting rates would be positively associated with the covariation among different types of externalizing-disinhibitory problems, rather than being specific to any one problem domain.

This study also explored the association between discounting of delayed rewards, self-reported traits of impulsivity and harm avoidance, measures of cognitive ability (i.e., executive working memory capacity and intelligence), and increased responsiveness to reward cues, all of which have been associated with both disinhibitory disorders and impulsive behaviors. Several delay discounting studies reported a significant positive association between delay discounting rates and self-reported measures of trait-impulsivity (de Wit, Flory, Acheson, McCloskey, & Manuck, 2007; Kirby & Petry, 2004; Kirby et al., 1999; J. M. Mitchell et al., 2005), although others reported no association (Crean et al., 2000; Dom et al., 2006a; Dom, De Wilde, Hulstijn, & Sabbe, 2007; S. H. Mitchell, 1999). Nonetheless, preference for a smaller immediate reward over a larger delayed reward has been used as a behavioral model of impulsivity and has been observed in impulsive substance using populations (Dom et al., 2006a; Johnson & Bickel, 2002, 2008; Kirby & Petry, 2004; Kirby et al., 1999; Madden, Begotka, Raiff, & Kastern, 2003; Petry, 2002; Reynolds, 2006b). We hypothesized that increased discounting rates would be associated with higher levels of trait impulsivity, but not with low harm avoidance, a personality trait that has been associated with externalizing behaviors via different disinhibitory mechanisms (Finn, 2002; Finn et al., 2002).

Disinhibitory disorders and increased discounting of delayed rewards also have been associated with low intelligence and reduced working memory capacity (de Wit et al., 2007; Dom et al., 2007; Finn & Hall, 2004; Finn et al., 2002; Hinson, Jameson, & Whitney, 2003; Hoffman et al., 2006; Kirby & Petry, 2004). Lower intelligence and reduced working memory capacity are thought to contribute to impulsive decisions by interfering with an individual's ability to optimize goal-directed behavior while keeping distal, less salient stimuli in mind (de Wit et al., 2007; Finn & Hall, 2004; Hinson et al., 2003). Therefore, we hypothesized that increased discounting of delayed rewards would be associated with lower intelligence and working memory capacity. Finally, studies also suggest that highly salient and temporally proximal stimuli exert greater influence on decision making in antisocial alcoholic individuals than in controls (Finn, 2002; Finn et al., 2002). This study used a reward incentive salience manipulation to test the hypothesis that delay discounting rate would be more strongly associated with AD and/or CCD when the rewards were highly salient.

In summary, this study was designed to add to our knowledge of the association between increased delay discounting and AD by examining the association between delay discounting rates and lifetime disinhibitory problems, disinhibitory personality traits, and cognitive capacity. The overarching hypothesis was that increased delay discounting would be associated with the covariation among AD and related disinhibitory disorders, high trait impulsivity, and reduced cognitive capacity.

Method

Participants

Recruitment. Participants were recruited using advertisements placed in local and student newspapers and around the community. These advertisements were designed using the approach used by Widom (1977) and others (Bauer & Hesselbrock, 1993; Finn et al., 2002) to elicit responses from individuals varying in terms of the level of impulsive, disinhibited traits and levels of alcohol use. The advertisements and flyers asked for "daring, rebellious, defiant individuals"; "carefree, adventurous individuals who have led exciting and impulsive lives"; "impulsive individuals"; "heavy drinkers wanted for psychological research"; persons with a "drinking problem"; persons who "got into a lot of trouble as a child"; persons "interested in psychological research"; "quiet, reflective and introspective persons"; and "social drinkers." This approach has been very effective in attracting responses from individuals who vary in the expected levels of disinhibited traits, AD, CCD, antisocial personality (ASP), and behavioral disinhibition (Finn et al., 2002).

Telephone screening interview. Each respondent was screened via telephone to determine if they met study inclusion criteria. Respondents who met study inclusion criteria were told that they must abstain from using alcohol and other drugs for at least 12 hr before testing. Those who met study inclusion criteria were between ages 18 and 30, read and spoke English, had at least a Grade 6 education level, did not report any severe head injuries, had no history of psychosis, and had consumed alcohol on at least one occasion in their life.

Group inclusion and exclusion criteria. The inclusion criteria for the control group were (a) not meeting *Diagnos-*

Table 1
Group Characteristics and Recent Substance Use

	Control	CCD-alone	AD-alone	ADCCD
n (male/female)	98 (50/48)	58 (31/27)	121 (65/56)	116 (71/45)
Variable: M (SD)				
Age in years[a]	21.3 (2.0)	21.8 (2.9)	21.9 (2.7)	22.5 (3.2)
Years of education[a,b]	14.6 (1.4)	13.3 (2.0)	14.2 (1.6)	12.9 (2.1)
Alcohol frequency[b,c,d]	1.7 (1.3)	2.4 (1.9)	3.9 (1.9)	3.9 (2.3)
Alcohol quantity[b,d]	2.5 (1.9)	3.0 (2.5)	6.9 (4.3)	7.8 (5.3)
Alcohol density[a,b,c,d]	5.3 (4.9)	10.0 (11.8)	16.0 (9.2)	18.0 (16.9)
Marijuana frequency[a,b,c]	0.0 (0.1)	2.3 (2.7)	2.6 (3.0)	3.6 (3.2)
Stimulant frequency[a,b,d]	0.0 (0.0)	0.3 (1.1)	0.3 (1.1)	0.5 (1.1)
Sedative frequency[b]	0.0 (0.2)	0.1 (0.5)	0.4 (1.4)	0.7 (1.6)
Opiate frequency[a,b]	0.0 (0.0)	0.1 (0.9)	0.2 (1.1)	0.6 (1.7)

Note. CCD = childhood conduct disorder; AD = alcohol dependence; alcohol frequency = number of drinking occasions during a typical week; alcohol quantity = average number of drinks on one occasion; alcohol density = greatest number of drinks consumed in one day; marijuana, stimulant, sedative, and opiate frequencies = number of days each drug was used during a typical week.
[a] Significant main effect of CCD, $p < .05$. [b] Significant main effect of AD, $p < .05$. [c] Significant CCD × AD interaction, $p < .05$. [d] Significant main effect of gender, $p < .05$.

tic and Statistical Manual of Mental Disorders (4th ed. [*DSM–IV*], American Psychiatric Association, 1994) criteria for a lifetime history of AD, CCD, ASP, alcohol abuse, or any other substance abuse or dependence; (b) not using marijuana more than 8 times in the last 6 months; (c) not using any other mood altering drug in the last 6 months; (d) not using marijuana more than 15 times in their life; and (e) not using other mood altering drugs recreationally more than 4 times in their life. The inclusion criteria for the AD-alone group were meeting *DSM–IV* criteria for a history of AD but not CCD or ASP. The inclusion criteria for the ADCCD group were meeting *DSM–IV* criteria for a history of both AD and CCD. The inclusion criteria for the CCD-alone group were meeting *DSM–IV* criteria for a history of CCD but not AD. We chose to focus on CCD instead of ASP during recruitment because CCD is a prerequisite to ASP and directly associated with adult antisocial behavior, and because delinquent behaviors after the age of 15 are often associated with alcohol or drug use.

Test session exclusion criteria. At the beginning of each testing session, participants were asked about their alcohol and drug use in the past 24 hr and were given a breath alcohol test using an AlcoSensor IV (Intoximeters Inc., St. Louis, MO). Participants were rescheduled if they reported consuming alcohol or drugs in the past 12 hr, if their breath alcohol level was greater than 0.0%, if they reported feeling hung-over, or if they appeared impaired, high, overly sleepy, or ill.

Sample characteristics. The sample consisted of 426 young adults: 129 (69 men, 60 women) with a history of AD with no history of CCD, 62 (32 men, 30 women) with a history of CCD and no history of AD, 128 (75 men, 53 women) with a history of both AD and CCD, and 107 (54 men, 53 women) controls. The sample was 77% White, 13% African American, 6% Asian, and 4% Hispanic. The control group was 60% White, 15% African American, 3% Hispanic or Latino, 21% Asian American or Asian, and 1% other ethnicity; the CCD-alone group was 81% White, 18% African American, and 2% Hispanic or Latino; the AD-alone group was 91% White, 5%

African American, 2% Hispanic or Latino, 2% Asian American or Asian, 1% other ethnicity; the ADCCD group was 84% White, 9% African American, 5% Hispanic or Latino, and 3% Asian American or Asian. Table 1 displays the group means on demographic and recent substance use measures.

Forty-three percent of the total sample ($n = 184$) met *DSM–IV* criteria for lifetime history of marijuana abuse or dependence, including 24 participants in the CCD-alone group, 66 in the AD-alone group, and 94 in the ADCCD group. Within marijuana dependent or abusing individuals, 61% ($n = 112$) were currently experiencing problems and 39% ($n = 72$) had past problems with marijuana use. Independent of marijuana use, 29% of the total sample ($n = 124$), including 25 CCD-alone, 37 AD-alone, and 62 ADCCD participants, met *DSM–IV* criteria for lifetime history of abuse or dependence of one or more illicit drugs. Of these individuals, 93% ($n = 115$) reported past problems associated with illicit drug use, and 25% ($n = 31$) were currently using illicit drugs. Although the drug abusing or dependent individuals most commonly abused marijuana (90%), 52 (25%) also reported abusing cocaine, 72 (35%) reported abusing other stimulants, 56 (27%) reported abusing sedatives, 20 (10%) reported abusing opiates, and 34 (17%) reported abusing other drugs, such as nitrous oxide or ecstasy.

Assessment Procedures and Materials

Diagnostic interview. Participants were administered the Semi-Structured Assessment for the Genetics of Alcoholism (SSAGA–II; Bucholz et al., 1994) to ascertain lifetime diagnoses and symptoms of AD, CCD, adult antisocial personality, and marijuana and other drug abuse or dependence. Indicators of lifetime problems with childhood conduct, adult antisocial behavior, alcohol use, marijuana use, and other drug use were determined by summing positive responses to relevant SSAGA–II questions in the respective diagnostic sections. Table 2 displays the group means for lifetime problems in each of these problem domains.

Table 2
Lifetime Problems Related to Disinhibitory Psychopathology

	Control		CCD		AD		ADCCD	
	n = 98		n = 58		n = 121		n = 116	
	M	SD	M	SD	M	SD	M	SD
Alcohol problems[a,b]	4.2	4.5	15.2	12.2	36.4	15.5	47.6	14.7
Conduct problems[a,b,c]	4.8	3.6	16.9	5.1	12.1	5.4	20.4	4.8
Adult antisocial problems[a,b,c,d]	3.9	3.6	15.6	6.8	11.2	6.3	21.6	7.1
Marijuana problems[a,b,c]	0.5	2.7	10.5	10.5	10.8	11.0	17.4	11.9
Drug problems[a,b]	0.0	0.3	14.3	22.4	13.2	25.4	27.8	34.8

Note. Lifetime alcohol, conduct, antisocial, marijuana, and drug problems were summed positive responses to subsets of Semi-Structured Assessment for the Genetics of Alcoholism–II questions. CCD = childhood conduct disorder; AD = alcohol dependence.
[a] Significant main effect of CCD, $p < .05$. [b] Significant main effect of AD, $p < .05$. [c] Significant CCD × AD interaction, $p < .05$. [d] Significant main effect of gender, $p < .05$.

Recent alcohol and other substance use. Alcohol use was quantified as the average frequency of drinking (days per week), the average quantity consumed per day, and the density of consumption (largest number of drinks consumed on a single occasion) over the past 6 months. Drug use was quantified as the average frequency of use (days per week) for marijuana, stimulants, sedatives and opiates over the past 3 months.

Personality assessment. Impulsivity was assessed with the impulsivity subscale of the Eysenck Impulsivity–Venturesomeness questionnaire (Eysenck & Eysenck, 1978); this self-report measure has been shown to be associated with elevated delay-discounting rates observed in heroin abusers (Kirby et al., 1999; Kirby & Petry, 2004). Impulsivity data were missing for one AD subject and one AD-CCD subject. Harm avoidance was measured with the harm avoidance subscale of the Multidimensional Personality Questionnaire (MPQ; Tellegen, 1982). The MPQ harm avoidance subscale assesses the tendency to avoid dangerous or risky activities and behavioral inhibition (Waller, Lilienfeld, Tellegen, & Lykken, 1991). Harm avoidance is inversely related to impulsive, thrill-seeking, and risk-taking personality traits (Kadden, Donovan, Litt, & Cooney, 1996). Although low harm avoidance has been associated with antisocial traits and substance abuse risk (Finn, 2002; Masse & Tremblay, 1997), it is thought to reflect different processes (i.e., decreased inhibition in the face of threat) than those reflected in impulsivity (Finn, 2002).

Cognitive measures. Working memory capacity was assessed with the Operation-Word Span test (Conway & Engle, 1994). This test taps into complex working memory functions including the competition for attentional resources (i.e., divided attention) and the maintenance, or decay, of mental representations over brief distraction periods. The test involves solving simple mathematical operations while trying to remember words. The participants were presented with a stack of index cards, each card containing a short mathematical equation followed by a word (e.g., 6/3 + 2 = 4 DOG). They were instructed to read the math operation aloud, respond "yes" or "no" to indicate whether the answer was correct, and then say the word (e.g., "dog") aloud. After a series of operation-word pairs, the partici-

pants were prompted to recall the words in that series in the order they were given. Series varied in length from 3 to 6 operation-word pairs and each series length was repeated three times during the task. The dependent measure on this test was calculated by adding the number of correctly recalled words across all test series. Test data was missing for one ADCCD participant on this measure.

General intelligence was measured using Shipley Institute of Living Scale estimates of IQ (Zachary, 1986). The Shipley is a self-administered measure of intelligence that assesses both verbal intelligence and logical reasoning and is strongly correlated with the WAIS Full Scale IQ (Zachary, 1986). Research shows that Shipley IQ estimates are associated with antisocial traits and alcohol abuse (Finn & Hall, 2004). Data for one control participant and one ADCCD participant were missing on this measure. Table 3 displays the group means for personality and cognitive measures, and delay discounting rates.

Delay Discounting Task

The delay discounting task consisted of "ascending" and "descending" choice trials on which participants were asked to indicate whether they preferred a specific amount of money "now" (i.e., the immediate reward) or $50 "later" (i.e., 1 week, 2 weeks, 1 month, 3 months, 6 months, 1 year). On ascending trials, the immediate reward value began with $0.05, followed by $1.25 and was then increased from $2.50 to $50 in 20 steps of $2.50. On descending trials, the immediate reward was reduced from $50 to $2.50 in 20 steps of $2.50 and ended with values of $1.25 and $0.05. Thus, participants made 22 choices on both ascending and descending trials for each of the six payment delays. The immediate reward values were printed on a deck of index cards that were flipped by the participants after each trial. At each delay, the 44 ascending and descending trials were administered in order regardless of participants' behavior. The delay blocks were always administered shortest to longest. During each delay block, the experimenter placed a laminated card displaying the delay on the table and instructed the participants that the $50 reward would

Table 3
Group Means and Standard Deviations on Personality, Cognitive, and Delay Discounting (k) Measures

	Control		CCD		AD		ADCCD	
	n = 98		*n* = 58		*n* = 121		*n* = 116	
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Impulsivity[a,b]	7.0	4.2	9.7	4.5	11.0	4.2	12.7	3.8
Harm avoidance[b,c]	16.3	6.5	15.0	6.6	14.4	5.6	13.3	6.2
Intelligence[a,b]	108.9	8.7	105.9	9.4	106.0	7.3	102.5	12.2
Word span[a,b]	45.8	10.8	42.1	12.1	41.1	10.8	38.6	12.9
Delay discounting rate (*k*)	0.0084	0.015	0.0143	0.024	0.0115	0.018	0.0195	0.031

Note. CCD = childhood conduct disorder; AD = alcohol dependence; impulsivity = Impulsivity scale from the Eysenck Impulsivity–Venturesomeness questionnaire; harm avoidance = Multidimensional Personality Questionnaire Harm Avoidance scale; intelligence = Shipley Institute of Living Scale Estimates of IQ; word span = Operation–Word span test; rate *k* = the estimated delay discounting parameter.
[a] Significant main effect of CCD, $p < .05$.   [b] Significant main effect of AD, $p < .05$.   [c] Significant CCD × AD interaction, $p < .05$.

now be available after the time displayed on the card. The experimenter recorded all 264 responses made by the participants during the task.

Reward incentive salience. The salience of any particular stimulus is determined by attributes, such as size, color, and brightness, and by context-dependent factors including validity and uncertainty or both (Estes, 1994). Differences in incentive salience are also derived from the stimulus' rewarding or motivational properties and from outcomes associated with that stimulus through past experiences. For example, monetary rewards may vary in their degree of incentive salience (Robinson & Berridge, 2003), such that more tangible, highly probable, and larger amounts of money carry more incentive salience than less tangible, lower probable, smaller amounts of money. Small amounts of money also may vary across individuals in their motivational properties based on subjective utility of those amounts. In the present study, participants were randomly assigned to one of two reward salience conditions (high or low).

The high salience condition included the presence of real money (i.e., highly tangible) and a 100% probability (highly probable) of receiving any money after the task. The low salience condition included a promissory note for money (i.e., less tangible) and a 17% probability (less probable) of receiving any money after the task. The purpose of manipulating reward salience was to assess group differences in response to salience of monetary rewards, not to systematically test the effects of varying the two parameters of tangibility and probability of reward. In the high salience condition, $50 in USD ($10 and $20 denominations) was counted in full view of the participant and then placed on the table in front of the participant. The participant was told that she or he would receive an amount of money either immediately or at a delayed date, determined by drawing a numbered bead (1 to 264) from a plastic container after the task was completed. The numbered beads reflect all possible trial choices. If the number corresponded to a choice in which the immediate amount was preferred, she or he would immediately be paid the amount indicated in cash. If the number corresponded to a choice in which the delayed amount was preferred, the participant would receive a payment voucher that could be redeemed at the specified delay

date. In the low salience condition, a $50 voucher was placed on the table in front of the participant. The participant was told that she or he had one in six chance of obtaining the money associated with her or his choice, which would be determined by drawing a bead with the number "6" printed on it at the end of the task. Those who drew a "6" received money in the same manner as in the high salience condition by drawing a numbered bead (1 to 264) that corresponded to a specific choice trial. In the high reward salience condition, there were 27 control men and 27 control women, 16 CCD-alone men and 14 CCD-alone women, 35 AD-alone men and 30 AD-alone women, and 38 ADCCD men and 26 ADCCD women. In the low reward condition, there were 27 control men and 26 control women, 16 CCD-alone men and 16 CCD-alone women, 34 AD-alone men and 30 AD-alone women, and 37 ADCCD men and 27 ADCCD women.

Delay discounting data from a total of 33 participants (9 controls: 4 men & 5 women; 4 CCD-alone: 1 man & 3 women; 8 AD-alone: 4 men & 4 women; 12 ADCCD: 4 men & 8 women) were not included in the analyses. Exclusion criteria were consistent with the guidelines proposed by Johnson and Bickel (2008) for identifying and eliminating unsystematic discounting data. The authors suggested eliminating individuals who (a) did not discount by at least 10% from the first to the last delay (31 cases excluded here), and (b) exhibited increases in the magnitude of switch points (starting at the second delay) by a magnitude greater than 20% of the larger reward (2 cases excluded here). All subsequent analyses were conducted using the remaining 393 cases.

Estimation of discounting rate. We assumed that the general form of delay discounting of future rewards can be adequately modeled using a single-parameter hyperbolic function (Mazur, 1987) $V_p = V/(1 + k \times dt)$, where V_p was the present (discounted) value, the constant V was the amount of the delayed reward (i.e., $50), dt was the length of time the reward is delayed expressed in days (i.e., 0, 7, 14, 28, 91, 182, and 364), and k was the discounting rate. All k estimates carry the reciprocal of days (1/days) as units in the modeling. At each delay, the V_p represented an arithmetic average of the ascending and descending run values at which the participant switched from selecting the

present to the delayed reward or vice versa. Using nonlinear regression, we estimated k separately for each participant by fitting the single-parameter hyperbolic model to the observed data with the discounted value V_p set to \$50 at zero delay. Resulting k values of zero reflected no discounting, whereas k values greater than 0 indicated that the delayed reward is discounted relative to the immediate reward. The dependent measure used in all analyses was the $\log_{10}$ transformed k value.

Data Analyses

Prior to the analyses, missing data on the cognitive and impulsivity measures were imputed using a single imputation procedure with a multiple regression equation using all available data as predictors. The relative influences of AD and CCD on delay discounting rates were investigated using a $2 \times 2 \times 2 \times 2$ (AD by CCD by gender by reward salience condition) analysis of variance (ANOVA). Planned comparisons were used to test the hypotheses that the ADCCD group would have significantly higher delay discounting rates than both the AD-alone and CCD-alone groups. Structural equation modeling (SEM) using Amos 6.0 (Arbuckle, 1995) was used to test the hypothesis that delay discounting rates would be positively associated with the covariance among these disorders, rather than being specific to any one disinhibitory disorder. SEM examined the association between a latent disinhibitory disorders (DD) factor and the rate of delay discounting ($\log_{10}(k)$). The DD factor was indicated by lifetime problems with alcohol, marijuana and other drug use, CCD, and adult antisocial behavior. Lifetime problem measures were Blom transformed to minimize skew prior to SEM analyses and the resulting distributions of alcohol and CCD plus adult antisocial symptoms are displayed in Figures 1a and 1b. Blom transformation is considered an optimal approach to handling psychiatric symptom counts in multivariate modeling analyses (Krueger et al., 2002; van den Oord et al., 2000). Modification indexes were used to assess whether the individual indicators of the DD factor were uniquely associated with the delay discounting rate, beyond their covariance with other DD indicators in the model. A modification index greater than 4.0 for a path from a DD indicator to the $\log_{10}(k)$ variable indicates that overall model fit will improve by adding that path to the model. Table 4 displays the intercorrelations among all variables used in the SEM analyses.

A second analysis of gender differences in the association between the DD factor and discounting rate $\log_{10}(k)$ were examined using a multiple-group SEM analysis. This analysis compared the fit of a model in which the regression coefficients between the DD factor and delay discounting rate were allowed to vary freely across gender (Model A) to the fit of a model in which the coefficients were constrained to be equal for men and women (Model B).

A third SEM assessed whether recent substance use accounted for the association between the DD factor and delay discounting. This SEM included an alcohol use latent factor indicated by drinking quantity, frequency, and density, and a drug use latent factor indicated by the frequencies of marijuana, opiate, sedative, and stimulant use. All substance use indicators were Blom transformed.

The final SEM was used to explore the association between exogenous observed personality and cognitive variables (impulsivity, working memory capacity, and intelligence) and discounting rate $\log_{10}(k)$.

The goodness-of-fit for all models was assessed with chi-square goodness-of-fit, the Bentler–Bonett normed fit index (NFI; Bentler & Bonett, 1980), and the root mean square error of approximation (RMSEA; Browne & Cudek, 1993). A close fit of the model is reflected in a nonsignificant NFI (higher than .94), RMSEA of .08 or less, and a nonsignificant chi-square statistic (Bentler & Bonett, 1980; Browne & Cudek, 1993).

Results

AD, CCD, and Gender on Delay Discounting

The ANOVA revealed a main effect of AD, $F(1, 377) = 6.16$, $p < .05$, a main effect of CCD, $F(1, 377) = 14.13$, $p < .001$, and a main effect of gender, $F(1, 377) = 6.31$, $p < .05$ on $\log_{10}(k)$. The AD $\times$ CCD interaction was not significant. Group means are displayed in Figure 2. Planned comparisons revealed that the ADCCD group had higher delay discounting rates than both the AD-alone group, $F(1, 154) = 4.82$, $p < .05$, and the CCD-alone group, $F(1, 172) = 4.58$, $p < .05$. The main effect of gender indicated that men discounted delayed rewards at significantly higher rates than did women. This effect is displayed in Figure 3.

Delay Discounting and Disinhibitory Disorders

Figure 4 illustrates the structural model of the association between the DD factor and the rate of delay discounting $\log_{10}(k)$. This model fit the data very well, $\chi^2(5, N = 393) = 2.04$, $p = .84$, NFI = .99, RMSEA = 0.0001. The path between the latent DD dimension and $\log_{10}(k)$ was significant, standardized $\beta = .323$, $p < .0001$. The strong model fit suggested that none of the paths from the individual DD indicator variables would significantly predict $\log_{10}(k)$ beyond their covariance with other DD indicator variables. In fact, modification indices indicated that model fit would not be improved by adding any of the individual indicator variables of the DD dimension.

Gender-related differences. The multiple-groups analyses indicated no significant gender-related differences in the path between the latent DD factor and $\log_{10}(k)$, $\Delta\chi^2(1, N = 393) = 0.011$, $p = .918$. The path between the DD factor and $\log_{10}(k)$ was statistically significant for both men, $\beta = .350$, $p < .0001$ and women, $\beta = .257$, $p < .001$. The multiple-groups analyses indicated that the strength of the paths did not differ between men and women.

Recent substance use. The SEM that assessed the influence of recent alcohol and drug use on the association between the DD factor and delay discounting rate fit the data adequately, $\chi^2(53, N = 393) = 171.77$, $p < .0001$,
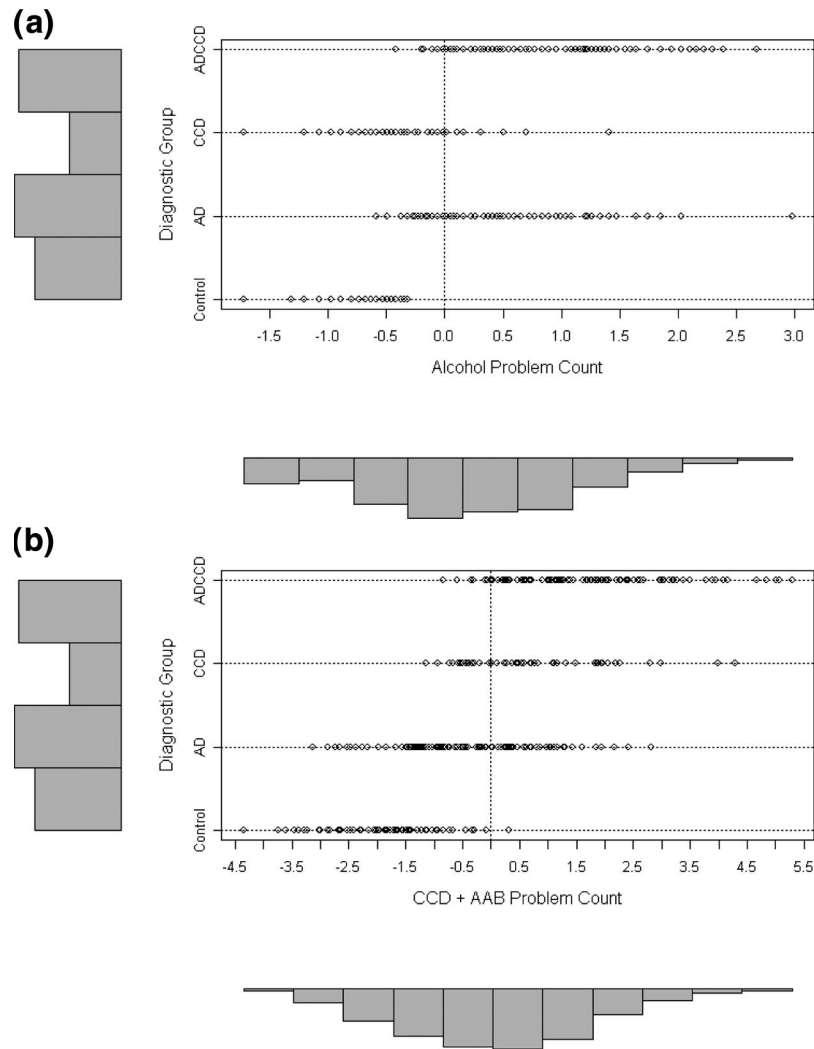
Figure 1. (a) and (b): Blom-transformed lifetime problem counts by diagnostic group for alcohol problems in the top panel (1a) and an aggregate measure of childhood conduct disorder (CCD) plus adult antisocial behavior (AAB) problems in the bottom panel (1b). The bars on the left represent number of participants in each diagnostic group, whereas the bars below are frequencies of problem counts in the entire sample.

NFI = .94, RMSEA = 0.076. The latent DD factor remained significantly associated with $\log_{10}(k)$, $\beta = .338, p < .0001$. Neither the alcohol use nor the drug use latent variables were associated with $\log_{10}(k)$, $\beta s = -.01$ and $-.01$, respectively, $ps > .05$. This analysis suggests that recent substance use did not account for the association between the DD factor and $\log_{10}(k)$.

Personality, Cognitive Factors, and Delay Discounting

The SEM that assessed the association between four exogenous predictor variables (impulsivity, harm avoidance, working memory capacity, and intelligence) and delay discounting rates revealed that impulsivity, working memory capacity, and intelligence, but not harm avoidance were

significantly associated with $\log_{10}(k)$, $\beta s = .168, -.148$, and $-.186$, respectively, $ps < .01$. Increased delay discounting was associated with higher levels of impulsivity, lower working memory capacity, and lower intelligence. Figure 5 illustrates this model.

Discussion

The pattern of results observed in this study suggest that increased delay discounting is associated with a general disinhibitory disposition, reflected in increased externalizing problems, higher levels of trait impulsivity, and lower scores on measures of cognitive capacity. Delay discounting rates were associated with the covariation among lifetime problems with alcohol, marijuana, other drugs, childhood conduct disorder, and adult antisocial behavior, but were not

Table 4
Intercorrelations Among All Indicator Measures Used in SEMs

	Lifetime problem counts				Substance-use indicator measures							Personality		Cognitive		
	a	b	c	d	e	f	g	h	i	j	k	l	m	n	o	p
log(*k*)	.24*	.19*	.20*	.31*	.31*	.09	.05	.15*	.15*	.04	.15*	.12*	.25*	-.04	-.29*	-.27*
a.alc	1	.65*	.58*	.66*	.67*	.49*	.46*	.47*	.41*	.23*	.29*	.29*	.49*	-.18*	-.26*	-.24*
b.mar		1	.68*	.56*	.58*	.30*	.21*	.25*	.55*	.30*	.30*	.30*	.35*	-.18*	-.15*	-.21*
c.drg			1	.55*	.59*	.19*	.11*	.15*	.44*	.34*	.43*	.42*	.34*	-.15*	-.26*	-.27*
d.ccd				1	.87*	.27*	.20*	.26*	.39*	.23*	.23*	.25*	.49*	-.23*	-.29*	-.24*
e.aab					1	.30*	.26*	.30*	.41*	.22*	.27*	.27*	.50*	-.19*	-.30*	-.25*
f.aq						1	.64*	.77*	.28*	.15*	.07	.09	.27*	-.14*	-.14*	-.19*
g.af							1	.63*	.30*	.17*	.09	.12*	.31*	-.20*	-.01	-.12*
h.ad								1	.31*	.15*	.14*	.10	.32*	-.16*	-.15*	-.18*
i.mj									1	.37*	.32*	.30*	.27*	-.20*	-.13*	-.21*
j.sti										1	.38*	.37*	.20*	-.11*	-.02	-.11*
k.sed											1	.52*	.23*	-.09	-.12*	-.16*
l.op												1	.21*	-.12*	-.08	-.20*
m.imp													1	-.28*	-.21*	-.20*
n.ha														1	-.20*	-.03
o.iq															1	.50*
p.opws																1

Note. SEM = structural equation modeling; log(*k*) is the log transformed discounting rate *k*; alc, mar, drg, ccd, aab = lifetime problems with alcohol, marijuana, and drug use, childhood conduct disorder, and adult antisocial behavior, respectively; aq, af, ad = quantity, frequency, and density of alcohol consumption, respectively; mj, sti, sed, op = frequency of marijuana, stimulant, sedative, and opiate use; imp = Eysenck Impulsivity scale; ha = Multidimensional Personality Questionnaire Harm Avoidance scale; iq = Shipley intelligence estimate; opws = total Operation-Word span score.
* Correlation is significant at the .05 alpha level.

uniquely associated with any one domain of disinhibitory problems. As hypothesized, higher scores on the disinhibitory disorders factor were associated with higher delay discounting rates. Recent substance use did not account for the association between the disinhibitory disorders factor and delay discounting rates. Additional analyses showed that higher delay discounting rates were significantly associated with higher trait impulsivity, but not with lower trait harm avoidance. Lower general intelligence and working memory capacity also were associated with higher delay

discounting rates, suggesting that lower cognitive capacity may contribute to increased delay discounting of rewards. Finally, men had higher overall delay discounting rates compared to women; however, the association between the disinhibitory disorders factor and delay discounting did not differ by gender. Contrary to our expectations, but consistent with other studies (e.g., Johnson et al., 2007), there was no effect of the manipulation of reward incentive salience.

The SEM fit the data well and highlighted the importance of accounting for the covariation among individual disinhibitory
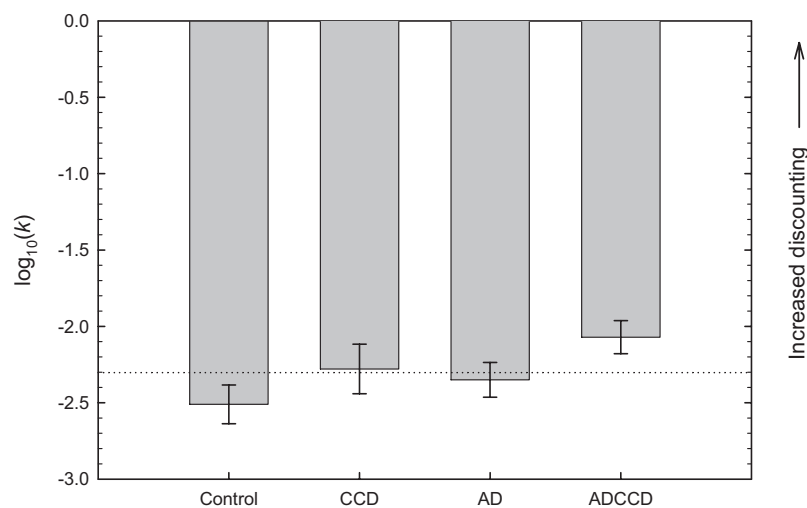

Figure 2. Log$_{10}$ transformed discounting rate *k* by diagnostic group. The error bars represent a 95% confidence interval for the group mean, and the dashed line represents the grand mean.
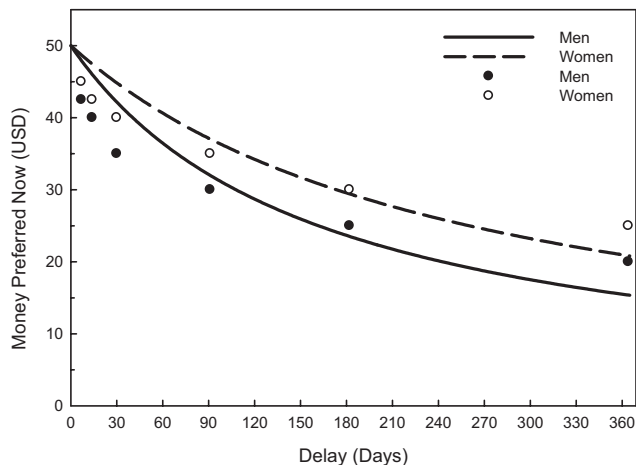
Figure 3. Median points of indifference and predicted discounting curves for men and women.

Figure 5. Structural equation modeling (SEM) of the association among measures of harm avoidance, impulsivity, working memory capacity, and general intelligence (Shipley IQ scores), and delay discounting rate, $\log_{10}(k)$. ** $p < .01$. *** $p < .001$.

disorders when predicting delay discounting performance. The model indicated that none of the individual disinhibitory problem domains was associated with delay discounting rates beyond its covariance with other problem domains. This result suggests that higher rates of delay discounting reflect a process that is common to all disinhibitory disorders and is not unique to any one disinhibitory disorder. Furthermore, although recent drug use was weakly associated with delay discounting in the univariate correlational analyses, it was not significantly associated with delay discounting when included in the multivariate analyses along with the disinhibitory disorders factor. These analyses suggest that higher delay discounting rates are associated with a process that is common to the covariance among disinhibitory disorders and not simply due to recent substance use. The group-based ANOVAs supported this interpretation. Both AD and CCD were significantly associated with elevated rates of delay discounting, and the ADCCD group had the highest delay discounting rates. The ADCCD group
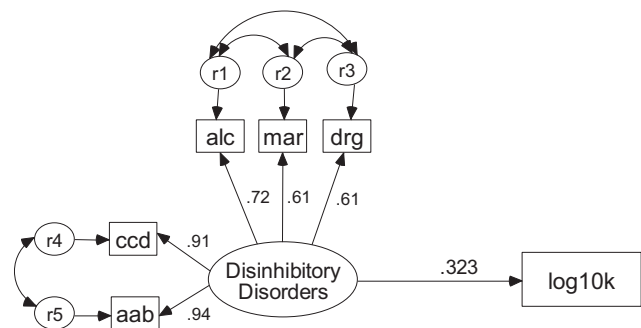

Figure 4. Structural equation modeling (SEM) of the association between disinhibitory disorders (DD) and $\log_{10}$ transformed discounting rate *k*. Alc = lifetime problem with alcohol; mar = lifetime problem with marijuana; drg = lifetime problem with drug use; ccd = lifetime childhood conduct problems; aab = lifetime adult antisocial behavior problems.
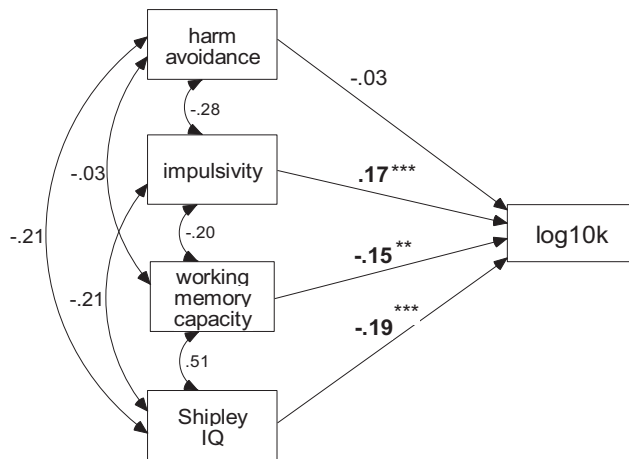
reflects the upper range of the disinhibitory disorders dimension, characterized by more severe symptoms and greater behavioral disinhibition compared to both the AD-alone and CCD-alone groups (Finn et al., 2002; cf. Table 2). Other research indicates that this disinhibitory-externalizing dimension reflects a vulnerability that is mostly genetic in origin (Kendler, Prescott, Myers, & Neale, 2003; Krueger et al., 2002) and distinct from a vulnerability to internalizing problems (Kendler et al., 2003). Thus, increased delay discounting may reflect aspects of a general vulnerability to externalizing, disinhibitory disorders.

Delay discounting also was positively associated with self-reported trait impulsivity. This finding is consistent with previous studies (Kirby et al., 1999; Kirby & Petry, 2004) and was expected because increased discounting of delayed relative to immediate rewards is thought to be a core feature of impulsivity (Monterosso & Ainslie, 1999). Although some studies report no significant association between delay discounting and trait impulsivity (Crean et al., 2000; Dom et al., 2006a, 2007; S. H. Mitchell, 1999), this may have resulted from either insufficient variation in trait-impulsivity or limited sample sizes. The current study adds to the literature by showing that delay discounting is associated with trait impulsivity, but not with low harm avoidance. Low harm avoidance, although associated with behavioral disinhibition (Finn, 2002; Kadden et al., 1996), is thought to reflect a different disinhibitory mechanism, for example, decreased sensitivity to aversive outcomes or events (Finn, 2002; Finn et al., 2002; Waller et al., 1993).

Delay discounting also was associated with reduced cognitive ability on measures of general intelligence and working memory capacity. Only a handful of studies have assessed the cognitive correlates of delay discounting and the results are inconsistent. Although Crean and colleagues (2000) found no association between intelligence and delay discounting, Kirby and Petry (2004) reported that increased delay discounting was associated with lower intelligence. There is additional evidence

that compromising working memory capacity may result in increased delay discounting. For example, Hinson and colleagues (2003) observed higher rates of discounting in a working memory load condition compared to a no-load condition. Evidence from our study suggests a modest association between increased delay discounting and lower intelligence and working memory capacity. The large sample size and substantial variation in disinhibitory problems in this study likely enhanced the possibility of detecting associations between delay discounting, cognitive capacity, and trait impulsivity. However, more research is needed to clarify the potential mechanisms underlying the association among delay discounting, intelligence, working memory capacity, and impulsive, disinhibited behavior.

Finally, the results did not support our hypothesis that increased reward salience would be associated with higher discounting rates in those with AD and or CCD. The results revealed that the reward incentive salience manipulation had no effect on delay discounting rates. This result is similar to other studies that found that the actual reinforcement of choices is not associated with delay discounting rates (Johnson & Bickel, 2002; Johnson et al., 2007; Madden et al., 2003; Reynolds, 2006b).

This study had a few limitations that need to be considered. First, this study used a nonrandom sample of substance abusers, who volunteered to participate after seeing various advertisements. The sample cannot be considered to be representative of all individuals with substance use disorders, or externalizing disorders in general. It is biased by self selection in response to specific advertisements. It also is possible that participants responding to a specific advertisement may present themselves in a manner that is consistent with the content of the advertisement to gain entry into the study. Although the screening, diagnostic interview, and substance use assessments serve as a check against gross misrepresentation, it remains possible that some participants biased their responses to meet their expectations of study inclusion criteria. Finally, a number of participants failed to discount delayed rewards or insisted on choosing immediate rewards only. These individuals may have misunderstood the task, may have been inattentive, or may have been influenced by processes other than delaying the consequence (Johnson & Bickel, 2008).

In summary, increased delay discounting of future rewards likely reflects a vulnerability common to several disinhibitory disorders. Delay discounting was directly associated with the covariance among lifetime problems with alcohol, marijuana, other drugs, childhood conduct, and adult antisocial behavior, and was not uniquely associated with any one problem domain. There was additional evidence that increased delay discounting reflects other processes related to poor self-regulation, such as lower intelligence and working memory capacity as well as higher levels of trait impulsivity.

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