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Comorbid Depression, Antisocial Personality, and Substance Dependence: Relationship with Delay Discounting

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Abstract

Background—Within the field of addiction, as many as four-fifths of individuals in treatment for substance use disorder have co-existing lifetime psychopathology and as high as two-thirds have current psychopathology. Among substance-dependent individuals, excessive delay discounting is pervasive. Despite evidence of excessive discounting across substance use disorders, few studies have investigated the impact of co-occurring psychopathologies and SUD on delay discounting.

Methods—We compared delay discounting in currently abstaining substance users with a) SUD (n=166), b) SUD and managed major depressive disorder (MDD; n=44), c) SUD and antisocial personality disorder (APD; n=35), d) SUD and managed MDD and APD (n=22) and e) no SUD or co-occurring psychopathology (n=60).

Results—All groups with SUD discounted future delayed rewards significantly more than healthy controls ($p < 0.001$ in each case, $d = 0.686, 0.835, 1.098$ and 1.650 , respective to groups a-d above). Individuals with both APD and SUD and individuals with MDD, APD, and SUD discounted future rewards significantly more than substance users without comorbid psychopathology ($p = 0.029, d = 0.412$ and $p < 0.001, d = 0.964$, respectively).

Conclusions—Overall, individuals with multiple psychopathologies in addition to substance use have exacerbated deficits in discounting of the future, above and beyond that observed in substance use alone. Increased discounting in combined substance and psychopathology profiles suggest a greater chance of treatment failure and therefore may necessitate individualized treatment using adjunctive interventions to achieve better treatment outcomes.

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Contributors

WKB conceived the study idea and worked on manuscript, CTF oversaw statistical analyses and worked on manuscript, and LM oversaw project progress and worked on manuscript.

Conflict of Interest

No conflict declared.

Keywords

delay discounting; psychopathology; depression; antisocial personality disorder; substance use

1. INTRODUCTION

Excessive delay discounting, or the disproportionate devaluation of delayed rewards, is pervasive across substance use disorders (SUD; Coffey et al., 2003; García-Rodríguez et al., 2013; Heil et al., 2006; MacKillop et al., 2011; MacKillop and Kahler, 2009; Petry, 2001a; Vuchinich and Simpson, 1998). In SUD, increased delay discounting is tied to worsened treatment outcomes (Dallery and Raiff, 2007; Krishnan-Sarin et al., 2007; Passetti et al., 2008; Sheffer et al., 2012, 2014; Washio et al., 2011; Yoon et al., 2007). Moreover, substance users are more likely to have mental health problems than the non-dependent population (Farrell et al., 2001). Given that 1) SUD are associated with excessive rates of discounting, 2) excessive discounting is associated with worsened treatment outcomes, and 3) substance users have increased rates of mental health problems, the impact of comorbid psychopathology on discounting in substance using populations may be important to understand treatment outcomes and may suggest methods to improve treatment efficacy.

The extant literature is conflicting with respect to the impact of comorbidities on delay discounting. One report on attention deficit hyperactive disorder in combination with cocaine dependence did not observe increases in discounting of delayed rewards above the non-combined profile (Crunelle et al., 2013). Another study found that individuals with problem gambling and substance use disorders discounted significantly more than problem gamblers without substance use disorders (Andrade and Petry, 2012; Petry, 2001b). Yet another report found that individuals that smoked cigarettes, used alcohol, and gambled discounted significantly more than nonsmokers with alcohol and gambling problems (Andrade et al., 2013). The heterogeneity of findings and paucity of systematic comparisons across comorbid group profiles highlights the importance of additional clarification within this area. Specifically, here we assess the impact of co-occurring depression and/or antisocial personality disorder in substance users.

Clinically depressed individuals exhibit increased delay discounting (Pulcu et al., 2013; Takahashi et al., 2011). Takahashi et al. (2011) reported increased delay discounting in combined psychopharmacologically medicated (e.g., managed) depressed and bipolar individuals compared to nonaffected controls. So too, Pulcu et al. (2013) reported increased discounting of large rewards in individuals with current MDD symptomology (approximately half of which were currently taking anti-depressants) compared to non-medicated remitted MDD and nonaffected controls. Together, the extant literature on delay discounting and depression indicates that individuals with current depression symptomology show atypical patterns of delay discounting compared to healthy controls such that they discount large rewards more steeply than individuals with past or never depression. However, the impact of combined substance use and depression has not been examined.

Conflicting results have been reported in studies of delay discounting among those with APD. In one study, delay discounting was not observed to differ between SUD with and

without APD (Sargeant et al., 2012). However, an earlier study reported that the SUD group discounted delayed rewards more than healthy controls and the SUD with APD group discounted more than the SUD group (Petry, 2002). The heterogeneity in these studies suggests the value of obtaining additional data to clarify the prior findings. The contrary results regarding SUD and APD could indicate one of two competing hypotheses. Either, combined SUD and APD may result in an additive effect on discounting or combined SUD and APD may result in a ceiling effect such that discounting does not increase above that observed in substance users. Given the current studies findings, we will evaluate these competing hypotheses to bring clarity to the extant literature.

The extant literature on delay discounting in combined SUD and psychopathology profiles is largely heterogeneous. Here, we present data that addresses and systematically replicates previous findings to clarify and provide a unique comparison of comorbid MDD, APD, and SUD. Given that excessive discounting has been demonstrated to be associated with poorer treatment outcomes, any additive effect of discounting may suggest that supplemental treatment to improve future valuation may improve treatment outcomes. Of course, a competing hypothesis is that SUD results in such a degree of excessive discounting that additional psychopathology cannot engender any greater discounting in which case the implications for treatment would be nil.

2. METHODS

In the current analysis, all participants provided written consent that was approved by either the IRB at University of Arkansas or Virginia Tech. Participants were community members from either the greater Little Rock metropolitan area in Arkansas or the greater Roanoke Valley region of Virginia. They were recruited via community outreach including flyers, postings on social media outlets including Facebook and Craigslist, and word of mouth. To participate, all participants had to 1) be at least 18 years of age, and 2) not have ADHD, epilepsy, mania, psychosis, or traumatic brain injury. Further, control participants had to be free from any form of drug dependence, recent drug use, or mental health disorder as screened with the Mini-International Neuropsychiatric Interview (MINI; Sheehan et al., 1998). Substance using participants had to 1) meet Diagnostic and Statistical Manual (DSM; American Psychiatric Association, 1994) criteria for cocaine or methamphetamine dependence and/or alcohol dependence. The current study was collected using DSM-IV criteria for substance dependence. The more recent DSM-V criteria merge what was previously substance abuse and substance dependence into substance use disorder with mild, moderate, and severe classifiers based on number of symptoms endorsed (American Psychiatric Association, 2003). Individuals that previously met diagnostic criteria for substance dependence would have met current DSM-V criteria for substance use disorder. In addition, the current study limited substances to stimulants and alcohol because these data were collected as part of a larger project examining the rehabilitation of executive function deficits with neurocognitive training. All substance-using participants were assessed by trained research associates using the MINI for MDD and APD. Participants that met current diagnostic criteria for MDD were required to provide a note from a health care professional indicating that the MDD was currently managed either through psychotherapy or psychopharmacology. Participants were not excluded for meeting current diagnostic criteria

for APD unless they reported an inability to comply with laboratory rules and regulations. Participants that were substance users were required to be in treatment and/or to have not used in the past three weeks. We selectively sampled individuals that were not currently using substances or currently had unmanaged MDD to avoid an overestimation of dysfunction that may result from acute substance-induced or psychopathologic states; however, this sampling procedure does not capture the full populations of substance using and depressed individuals. Although different forms of substance use disorders may be differentially affected by comorbid psychopathology, in the current study, consistent with other research in this area (Petry, 2001b) we have grouped these two substance use disorders together to maintain adequate power to assess the relationship of delay discounting in comorbid substance users.

After consent was obtained, participants completed demographic information and breathalyzer, urinalysis to confirm current abstinence from alcohol and drugs including cocaine, opiates, marijuana, amphetamines and benzodiazepines. Following confirmation of all eligibility criteria, the delay discounting task (described below in section 2.2) was administered. Participants were compensated \$20-\$40 for completion of consent and assessments based on amount of time spent in the laboratory with a rate of compensation of \$10 per hour spent in the research center.

2.1 Measures

A computer-based delay discounting task was used to assess participants' impulsive compared to self-controlled behavioral strategies. Participants were presented with hypothetical scenarios in which the extent of their discounting of a delayed "reward" (i.e., \$1000) relative to an immediate "reward" was determined at seven delays (i.e., 1 day, 1 week, 1 month, 6 months, 1 year, 5 years, and 25 years) presented in chronological order. The smaller sooner amount titrates until an indifference point where the subjective value of the immediate reward is approximately equal to the value of the delayed reward (Du et al., 2002). The indifference points are then fit to the following equation:

$$V=A/(1+kD) \quad \text{Equation 1 (Eq. 1)}$$

where V is the subjective value of the objective monetary amount A, to be delivered after some delay, D (Mazur, 1987). The parameter k describes the hyperbolic function and is used as an index of the extent to which participants discount the value of future rewards. Taken together, higher k values indicate a tendency to devalue future rewards at a higher rate and this, in turn, suggests greater impulsivity.

A demographic questionnaire was administered that included age, race, gender, marital status, educational attainment, and employment status. Assessment of relevant medical history included history of head injury with loss of consciousness lasting more than 10 minutes; history of stroke, seizure or other neurological disorder; and history of treatment for alcohol or substance abuse disorders.

The Mini-International neuropsychiatric Interview (MINI; Sheehan et al., 1998) is a short structured diagnostic interview for DSM-IV psychiatric disorders. It was designed to meet

the needs of being a short but accurate structured psychiatric interview. The MINI provides symptoms and diagnostic criteria for individuals on a range of disorders (e.g., major depressive disorder, post traumatic stress disorder, bipolar disorder, panic disorder, antisocial personality disorder). The modules analyzed here include Major Depressive Episode current, APD lifetime, alcohol dependence, and primary substance dependence.

The timeframe of questions asked on the MINI are meant to assess for current symptomology. For example, the Major Depressive Episode module asks about symptoms of depression over the past two weeks, the APD lifetime module asks about behaviors before the age of 15 years old and since the age of 15 years old to assess for pervasive personality constructs. Furthermore, the alcohol and substance dependence modules ask about substance-using habits over the past twelve months. While assessing for current symptomology, participants were excluded if they met diagnostic criteria for MDD and symptoms were not managed through psychotherapy and/or psychopharmacology. Thus in this study, participants could meet diagnostic criteria for MDD as long as the MDD was managed.

2.2 Data Analytic Plan

Descriptive statistics were computed for all demographic variables including: gender, age in years, race (white, African American, other), ethnicity (Hispanic or non-Hispanic), employment status (full-time, part-time, unemployed, retired), years of education, and monthly income to characterize the substance-dependent and control individuals. Data were analyzed with IBM SPSS Statistics Software (Version 22.0.0.0) unless otherwise noted.

The primary research goal was to explore the interplay between substance and various psychopathology profiles and delay discounting. Normality of the residuals was visually assessed for delay discounting to determine if ordinary linear regression assumptions were met. Area under the curve (AUC) was calculated for each of the substance use and psychopathology groups using the indifference points to create a trapezoid and determine the amount of area under the curve between 0 and 1 where 0 is very steep discounting and 1 is no discounting. Transformations of the k values were considered to assess normality in the residuals. In previous work, k values have been widely observed to restore normality via natural logarithm transformation (e.g., Heil et al., 2006; Kirby and Petry, 2004; Kirby et al., 1999). To address the possibility that other study variables might affect the association between delay discounting and the substance and psychopathology profiles, an exhaustive model selection routine was implemented to determine which covariates should be modeled alongside delay discounting. In order to select demographic covariates for the model, a Morgan-Tater model search was implemented which considered each of the 256 models (i.e., 2^8 for the eight demographic variables) corresponding to the possible permutations of the candidate variables in R Version 3.0.2 (R Development Core Team, 2013) using the `bestglm` package (McLeod and Xu, 2010) to choose between eight candidate covariates (i.e., age in years, monthly income, employment status, marital status, ethnicity, race, years of education, and gender) for the delay discounting measure. The Bayesian Information Criterion (BIC; Schwarz, 1978) was used as a metric to decide which of the available predictors best described observed delay discounting. BIC weighs the likelihood of a

candidate model for a given set of data and includes a penalty term for complexity, so that a model with fewer parameters would be chosen over a more complex version if the predictive ability of both models were similar. Upon obtaining the optimal (i.e., lowest) BIC model for delay discounting, the chosen variables for the model that provided the best fit were used as covariates in remaining analyses.

After choosing variables to be modeled alongside delay discounting, the bivariate relationship of delay discounting was compared alongside the selected covariate model to the substance use and psychopathology groups. Since group means analysis revealed statistically significant relationships at the $p < 0.05$ level, simple comparisons of the five substance and psychopathology groups were computed following Bonferroni adjustment to maintain a familywise Type I error rate of 0.05. Both raw and Bonferroni adjusted p -values are presented. Note that the p -values do not incorporate uncertainty associated with the model selection approach. Effect sizes of simple comparisons are reported using a variation of Cohen's d where the contrast estimates from the individual substance use and psychopathology groups were divided by the root mean squared error (RMSE). Conventions for interpreting these effect sizes are 0.20, 0.50, and 0.80 are small, medium, and large, respectively (Cohen, 1992).

2.3 Descriptive statistics

The full sample ($n = 327$) was comprised of the following groups: CON group ($n = 60$), SUD group ($n = 166$), MDD/SUD group ($n = 44$), APD/SUD group ($n = 35$), and MDD/APD/SUD group ($n = 22$). See Table 1 for overview of demographic characteristics.

2.4 Delay Discounting Analyses

The k values from the delay discounting task were natural logarithm transformed. The aggregate fit of indifference points to Eq. 1 was adequate (median $R^2 = 0.912$, and did not differ across groups). The top 10 models selected using the BIC are provided in Table 2. The BIC revealed that the model with years of education alone was the most parsimonious (See Tables 1 and 2). Using the selected model, the omnibus group effect of delay discounting $\ln(k)$ was statistically significant ($F(4, 321) = 13.444$, $p < 0.001$, Partial $\eta^2 = 0.143$). The omnibus group effect using the delay discounting AUC also indicated a statistically significant omnibus group effect ($F(4, 321) = 11.602$, $p < 0.001$, Partial $\eta^2 = 0.127$; See Table 3, Figure 1, and Figure 2).

Simple comparisons were conducted on the delay discounting task to elucidate the unique effects of group membership. Given the comparable omnibus models of the $\ln(k)$ parameter and AUC, the following simple comparisons were conducted on $\ln(k)$. To be conservative in protecting against Type I error while also considering the risk of Type II error, results are discussed both with and without a Bonferroni adjustment for multiple comparisons. Type I error rates of $\alpha = 0.05$ were used to declare statistical significance for unadjusted p -values. To control familywise Type I error rate at five percent, the $\alpha = 0.0056$ (based on nine comparisons) level was used. For the delay discounting task, statistically significant comparisons were found between CON and SUD, MDD/SUD, APD/SUD, and MDD/APD/SUD ($p < 0.001$ for all), SUD and APD/SUD ($p = 0.029$), SUD and

MDD/APD/SUD ($p < 0.001$), MDD/SUD and MDD/APD/SUD ($p = 0.002$), and APD/SUD and MDD/APD/SUD ($p = 0.043$; See Table 4 and Figure 1).

3. RESULTS

The goal of this study was to look at the effects of delay discounting on currently abstaining substance users with managed MDD and/or APD. We found increased delay discounting in combined substance use and psychopathology profiles. The CON group discounted significantly less than all other profiles (i.e., SUD, MDD/SUD, APD/SUD, MDD/APD/SUD) indicating that the CON group was less impulsive and displayed more self-control than all substance using groups. Adding to prior work on discounting in substance using populations, we found that when substance use disorders are combined with mental health problems the rate of discounting increases. Individuals with multiple psychopathologies (i.e., both MDD and APD) and SUD discounted more than individuals with SUD alone. Notably, the MDD/SUD group did not discount more than SUD alone and the APD/SUD group did not discount significantly more than the SUD group when held to the more conservative standards of the Bonferroni correction (See Table 4).

4. DISCUSSION

The current systematic evaluation of combined psychopathology and substance use offers clarity to the heterogeneous findings from the extant literature. The current finding that substance use in combination with managed MDD or APD is not associated with significantly greater discounting suggests a ceiling effect wherein the combination of MDD, or APD does not engender significantly greater dysregulation of the executive and impulsive decision systems. However, the combined profile of MDD, APD, and SUD was associated with significantly greater discounting than substance use alone suggesting an additive effect wherein the previously observed ceiling was no longer applicable. The observation of further exacerbation of discounting in the combined MDD, APD, and SUD group could be due to either more compromised executive functioning or more impulsive functioning. Analysis with fMRI may clarify the specific origin of the dysregulation resulting in the observed additive effect of MDD, APD, and SUD on delay discounting.

Importantly, the findings of the current study are in line with a broad literature of previous work reporting that substance users discount more than healthy controls (Bickel et al., 1999; Businelle et al., 2010; García-Rodríguez et al., 2013; Heil et al., 2006; MacKillop et al., 2011; Vuchinich and Simpson, 1998). Also consistent with previous reports that depression is associated with increased discounting (Pulcu et al., 2013; Takahashi et al., 2011), the MDD/SUD group discounted significantly more than the CON group; however, managed MDD did not add significantly to the rate of discounting above and beyond substance use alone. The finding that the MDD/SUD group did not discount significantly more than the SUD group shows that managed depression may not result in significant increases in impulsivity above and beyond that of substance use. This implication is also supported by the small effect detected when comparing discounting in the SUD and MDD/SUD group ($d = 0.149$).

Discounting in combined APD and SUD was significant at the $\alpha < 0.05$ but not at the Bonferroni corrected $\alpha < 0.0056$ level. This finding is consistent with Petry's (2002) findings that individuals with both APD and SUD discount more than SUD alone. However, the current findings are in contrast to the reports of Dom et al. (2006) and Sargeant et al. (2012) which find that comorbid APD and substance use was not associated with significantly greater discounting than substance users alone. A potential explanation for the conflicting findings is that the current study and the consistent findings of Petry (2002) were community samples; conversely, Dom et al. (2006) and Sargeant et al. (2012) were both samples from inpatient substance use treatment centers possibly indicating that participants from inpatient residential treatment centers represent a unique subset of the population where additive effects on discounting in substance use and antisocial personality disorder were not apparent. The finding that the APD/SUD group increased discounting compared to the SUD group did not withstand the Bonferroni multiple correction threshold possibly indicating that SUD with a comorbidity of APD is not so interfering as to result in a distinctly greater discounting profile. This conclusion is also supported by the comparatively smaller (in the small-to-medium range) effect size of combined APD/SUD compared to SUD alone ($d = 0.411$), potentially providing an explanation for the heterogeneity of previous results. Interestingly, a significant difference was found between SUD and MDD/APD/SUD ($d = 0.962$), indicating that the combined comorbidities of both a managed Axis 1 and Axis 2 disorder in conjunction with substance dependence is associated with greater discounting. Furthermore, the combined MDD/APD/SUD group compared to the SUD alone group had a greater effect size (in the large range) than MDD/SUD or APD/SUD, suggesting an allostatic overload wherein discounting becomes excessive. Although not all of these profiles are significantly different, we observed what may be considered an orderly dose-related relationship (see Figure 1) as we systematically increase from healthy controls (CON), to substance users (SUD), to single comorbidity substance and psychopathology profiles (MDD/SUD and APD/SUD), to the dual psychopathology in addition to substance use profile (MDD/APD/SUD).

Strengths of this study include sampling individuals at a point of theoretically low symptomology within the time course of their illnesses. By assessing delay discounting during times of managed substance use and depression, instead of at clinically acute periods, we intend to capture that excessive discounting that is not likely to be attributable to psychiatric exacerbation. Consequently, the decision making of these individuals may continue to be compromised despite being in non-acute states. Moreover, differences in delay discounting in the present study versus those in the extant literature may reflect discounting that is underlying the pathologies versus dysfunction as a result of psychiatric exacerbation or current substance consumption.

Weaknesses of this study include the heterogeneity of the treatment participants received to manage their comorbid depression. For example, one could hypothesize that individuals receiving psychotherapeutic interventions as opposed to psychopharmacological might evidence greater shifts in behavioral self-regulation as measured by delay discounting. As such, future research should investigate the differences between those with psychotherapeutically managed, psychopharmacologically managed, and unmanaged

psychopathology so as to characterize what is chronic compared to acute dysfunction. Also, consideration of other psychopathologies that are associated with increased impulsivity, such as attention deficit hyperactivity disorder, will aid in understanding if all comorbid profiles lead to discounting exacerbations (Bickel et al., 2007), or if these observations are unique. Finally, the current analysis assessed alcohol dependence and stimulant dependence (i.e., either cocaine or methamphetamine) together to maintain adequate power to test for differences across comorbid psychopathologies. In future studies, different substance use disorders should be assessed for unique effects of alcohol dependence compared to stimulant dependence on delay discounting in comorbid profiles. Further, future studies may consider looking at the combined effects of other substance use disorders such as opioids to ascertain if they have differential effects.

Better characterization of co-occurring psychopathologies is essential to the recent National Institute of Mental Health initiative to identify Research Domain Criteria. The Research Domain Criteria highlight the importance of identifying transdiagnostic processes (Bickel et al., 2012) to inform assessment, diagnosis, and treatment of psychopathology. Indeed, the extant literature indicates that delay discounting in temporally stable, biologically based, and heritable. As such, delay discounting is identified as a behavioral marker (Bickel et al., 2014) and more recently as a candidate endophenotype of addiction and other temporally relevant disorders (Bickel, 2015; Gottesman and Gould, 2003). Importantly, the purpose of endophenotypes is to base diagnoses on phenotypic categorizations that are more stable and heritable than the current diagnostic classification system (MacKillop, 2013). As such, future work to identify substance and psychopathology profiles that are characteristic of excessive discounting may prove important not only to inform current treatment needs but also to lay the groundwork for future diagnostic criteria informed both by phenotypic and genetic etiology. The case may be that discounting functions as an endophenotype in other forms of psychopathology than substance use. If so, then identifying the alleles associated with the endophenotypic expression of excessive discounting in these psychopathologies may suggest alternative and potentially more efficacious psychotherapeutic interventions.

In summary, psychopathology, especially MDD and APD, is more prevalent in SUD than in the population at large. Given the increased prevalence, awareness of how comorbidities affect treatment responses is critical. Given the current study's finding of increased discounting of future rewards in SUD with both psychopathologies, personalized adjunctive treatments to target excessive discounting may improve treatment response.

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Highlights

- Many with substance use disorder also have comorbid psychopathology
- Discounting is exacerbated in comorbid psychopathology and substance use
- Excessive discounting may be related to increased treatment failure

Figure 1.

Delay discounting $\ln(k)$ by group. Bar graph of the mean rate of discounting on the delay discounting task by substance and psychopathology group. Error bars indicate ± 1 standard error. Black arrow indicates direction of more optimal performance. The covariate (i.e., years of education) appearing in the model is evaluated at 12.495 years.

Figure 2.

Area under the curve (AUC) by group. Bar graph of the median area under the delay discounting curve by substance and psychopathology group. Error bars indicate ± 1 standard error. The covariate (i.e., years of education) appearing in the model is evaluated at 12.495 years.

Table 1

Demographic and discounting information by group.

Variable		CON (<i>n</i> = 60)	SUD (<i>n</i> = 166)	MDD/SUD (<i>n</i> = 44)	APD/SUD (<i>n</i> = 35)	MDD/APD/SUD (<i>n</i> = 22)
Age (years)		35.15 (13.48)	43.05 (10.35)	45.61 (9.71)	40.57 (9.91)	43.00 (11.04)
Education (years)	Mean (SD)	12.95 (1.69)	12.61 (1.82)	12.16 (1.24)	12.01 (1.52)	11.84 (1.67)
Discounting (lnk)		−6.59 (2.64)	−4.76 (2.44)	−4.27 (2.36)	−3.57 (2.87)	−2.12 (3.22)
Income (\$ monthly)	<i>a</i>	836 (0-1412)	200 (0-710)	200 (0-697)	200 (0-750)	0 (0-200)
Employment Status						
Full-time		11 (18.3)	10 (6.0)	2 (4.5)	3 (8.6)	-
Part-time		14 (23.3)	22 (13.3)	3 (6.8)	3 (8.6)	1 (4.5)
Unemployed		29 (48.3)	133 (80.1)	38 (86.4)	29 (82.9)	21 (95.5)
Retired		4 (6.7)	1 (0.6)	-	-	-
Missing		2 (3.3)	-	1 (2.3)	-	-
Gender (% male)		37 (61.7)	126 (75.9)	36 (81.8)	27 (77.1)	19 (86.4)
Marital Status						
Single/Never Married		31 (51.7)	63 (38.0)	16 (36.4)	16 (45.7)	7 (31.8)
Married	Frequency (%)	14 (23.3)	17 (10.2)	1 (2.3)	1 (2.9)	3 (13.6)
Divorced		7 (11.7)	63 (38.0)	20 (45.5)	14 (40.0)	8 (36.4)
Separated		-	17 (10.2)	6 (13.6)	4 (11.4)	4 (18.2)
Widowed		-	5 (3.0)	1 (2.3)	-	-
Missing		8 (13.3)	1 (0.6)	-	-	-
Race						
White		46 (76.7)	98 (59.0)	22 (50.0)	23 (65.7)	13 (59.1)
African-American		14 (23.3)	62 (37.3)	20 (45.5)	10 (28.6)	9 (40.9)
Native American		-	2 (1.2)	-	-	-
Pacific Islander		-	1 (0.6)	-	-	-
Other		-	3 (1.8)	2 (4.5)	2 (5.7)	-

^aMedian (interquartile range)

Table 2

The top ten BIC models for delay discounting. One indicates that the variable was included and zero indicates that the variable was excluded in each model. The lowest relative criterion score indicates goodness of fit of the model.

BIC Model	Age	Years of Education	Income	Gender	Marital Status	Employment Status	Ethnicity	Race	Criterion
1	0	1	0	0	0	0	0	0	710.04
2	0	1	1	0	0	0	0	0	713.95
3	0	1	0	1	0	0	0	0	715.02
4	1	1	0	0	0	0	0	0	715.41
5	0	1	0	0	0	0	1	0	715.71
6	0	1	1	1	0	0	0	1	718.96
7	0	1	0	0	0	0	0	0	719.06
8	1	1	1	0	0	0	0	0	719.26
9	0	1	1	0	0	0	1	0	719.65
10	0	1	0	1	0	0	1	0	720.53

Table 3

Overall GLM model including selected covariate, years of education, using cell reference coding where each coefficient estimate in the table quantifies the difference in discounting between MDD/APD/SUD and respective cell.

	ln(k)					AUC				
	B	SE	t	Sig.	95% Confidence Interval	B	SE	t	Sig.	95% Confidence Interval
Intercept	0.81	1.13	0.71	0.48	-1.42 - 3.04	-0.10	0.10	-1.06	0.29	-0.29 - 0.09
Education (Yrs)	-0.25	0.08	-2.95	0.00	-0.41 - -0.08	0.02	0.01	2.20	0.03	0.00 - 0.03
CON	-4.19	0.64	-6.55	< 0.001	-5.45 - -2.93	0.26	0.05	4.89	< 0.001	0.16 - 0.37
SUD	-2.45	0.58	-4.22	< 0.001	-3.59 - -1.31	0.08	0.05	1.66	0.10	-0.02 - 0.18
MDD/SUD	-2.07	0.66	-3.12	0.00	-3.38 - -0.76	0.05	0.06	0.86	0.39	-0.06 - 0.16
APD/SUD	-1.40	0.69	-2.03	0.04	-2.76 - -0.04	0.03	0.06	0.45	0.65	-0.09 - 0.14
MDD/APD/SUD	0	.	.	.	.	0	.	.	.	.

Planned univariate simple comparisons for delay discounting compared using GLM. Bold values indicate significant at the $\alpha < 0.05$ level. Bonferroni correction for multiple comparisons was used to protect against Type II errors with a significance threshold or $\alpha < 0.0056$ and is indicated by an asterisks.

Table 4

Simple Comparisons	Delay Discounting	Contrast Estimate	95% Confidence Interval	Effect Size (<i>d</i>)	Standard Error
CON v. SUD	< 0.001*	-1.744	-2.500 to -0.989	0.686	0.384
CON v. MDD/SUD	< 0.001*	-2.123	-3.124 to -1.122	0.835	0.509
CON v. APD/SUD	< 0.001*	-2.790	-3.865 to -1.716	1.098	0.546
CON v. MDD/APD/SUD	< 0.001*	-4.193	-5.453 to -2.934	1.650	0.640
SUD v. MDD/SUD	0.382	-0.379	-1.230 to 0.472	0.149	0.433
SUD v. APD/SUD	0.029	-1.046	-1.981 to -0.111	0.412	0.475
SUD v. MDD/APD/SUD	< 0.001*	-2.449	-3.590 to -1.307	0.964	0.580
MDD/SUD v. MDD/APD/SUD	0.002*	-2.070	-3.376 to -0.763	0.815	0.664
APD/SUD v. MDD/APD/SUD	0.043	-1.403	-2.764 to -0.043	0.552	0.692