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Interrelationships among Parental Family History of Substance Misuse, Delay Discounting, and Personal Substance Use

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Abstract

Rationale—Despite consistent evidence of the familiality of substance misuse, the mechanisms by which family history (FH) increases the risk of addiction are not well understood. One behavioral trait that may mediate the risk for substance use and addiction is delay discounting (DD), which characterizes an individual's preferences for smaller immediate rewards compared to larger future rewards.

Objectives—To examine the interrelationships among FH, DD, and diverse aspects of personal substance use, and test DD as a mediator of the relationship between FH and personal substance use.

Methods—The study used crowdsourcing to recruit a community sample of adults (N = 732). Family history was assessed using a brief assessment of perceived parental substance use problems, personal substance use was assessed using the Alcohol Use Disorders Identification Test and a measure of frequency of use, and delay discounting was assessed using a latent index of discounting preferences across six reward magnitudes.

Results—Steeper discounting was significantly associated with personal alcohol, tobacco, and marijuana use, and level of substance experimentation. Steeper DD was also associated with a denser parental FH of alcohol, tobacco, and overall substance misuse. Parental FH density was significantly associated with several aspects of personal substance use, and these relationships were partially mediated by DD.

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Conclusions—The current study suggests that impulsivity, as measured by DD, is one proximal mechanism by which parental FH increases substance use later in life. The causal role of DD in this relationship will need to be established in future longitudinal studies.

Keywords

Delay discounting; impulsivity; family history; alcohol; tobacco; marijuana

INTRODUCTION

It is well established that substance misuse aggregates among family members, with not only robust parental influences but increased risk expanding as far as fifth-degree relatives (Elliotet al. 2012; Tyrfingsson et al. 2010). Family, twin, and adoption studies indicate that the higher risk is partially due to genetic factors. Heritability estimates vary, but, on average, genetic variation appears to account for approximately half of the individual risk to develop a substance use disorder (Goldman et al. 2005). Beyond genetics, environmental processes are also thought to contribute to this relationship (e.g., Barnow et al. 2002). Despite consistent evidence of the familiality of substance misuse, however, the genetic and environmental processes by which family history (FH) confers its influence are not clearly understood. Understanding these mechanisms has considerable potential for ultimately tailoring prevention strategies for individuals with this risk factor.

Delay discounting (DD) may serve as a proximate mechanism by which a positive FH of substance misuse contributes to the development of an addictive disorder (MacKillop 2013). Delay discounting (DD) is a behavioral economic measure of impulsive decision-making, akin to the ability to delay gratification. DD reflects the rate at which an individual devalues a reward based on its temporal delay, with more impulsive individuals discounting delayed rewards at higher rates than less impulsive individuals, and it is an established correlate of addictive behavior (Stein and Madden 2013). Categorical studies comparing DD in high users versus matched controls for several substance classes have revealed significant differences between groups (for a meta-analysis, see MacKillop et al. 2011), typically of medium effect size magnitude. The direction of the relationship between DD and personal substance use is likely to be bidirectional: impulsive decision-making predates the onset of substance use, contributes to the maintenance of addictive disorders, and may also result from extended use of substances (Audrain-McGovern et al. 2009; Mendez et al. 2010; Fernie et al. 2013; Mitchell et al. 2014). Alternatively, it is possible that the link between DD and substance use is mediated by a third, unmeasured variable, such as genetic variation or adverse developmental factors.

One way to study how a trait or condition develops among family members is by characterizing the condition status and its association with family history status, either categorically, as affected/nonaffected, or continuously, in terms of how densely affected the family is. Theoretically, if DD is associated with the presence, or density, of substance misuse in the family, it would suggest that DD may serve as a pathway to addictive behavior. As such, the DD construct could aid in identifying high-risk individuals for preventive purposes. Only a small number of studies have investigated these relationships, with mixed

findings (Crean et al. 2002; Petry et al. 2002; Herting et al. 2010; Acheson et al. 2011). However, early studies were generally limited by relatively small sample sizes, and other potential confounding variables. For example, the majority of these studies focused exclusively on FH of alcoholism and personal drinking behavior, without explicitly including or excluding FH of other addictive substances or other personal substance use. This is problematic given the link between DD and a range of addictive disorders (MacKillop et al. 2011). Moreover most of these studies utilized a dichotomous FH variable (i.e., a positive FH of addiction [FH+] or a negative FH of addiction [FH−]), rather than density of FH, functionally reducing effect size, power, and measurement reliability (MacCallum et al. 2002).

Recent studies have addressed some of the preceding methodological limitations and are supportive of the link between FH of several substances and both DD and addictive behavior. For example, Acheson et al. (2011) conducted a thorough classification of FH status across an array of substances and addictive behaviors in a relatively large, late-adolescent, high-risk sample (N=298). Individuals who were FH+ for any of several drug classes exhibited steeper DD than FH− participants. Additionally, Dougherty et al. (2014) found a significant association between categorical FH of any substance use disorder and DD in 386 children, ages 10–12. The presence of this risk factor in individuals, even before the onset of substance use and other risk-taking behaviors associated with adolescence (e.g., reckless driving, unprotected sexual activity, engaging in physical fights, gambling; Romer 2010), provides further support for familial influences on DD. These studies suggest that with adequate power and careful characterization of an individual's family history, steeper DD does appear to be a credible link between FH and personal substance use.

The goal of the current study was to extend our understanding of the relationship between FH of several substances, DD, and personal substance use. Using the validated Amazon Mechanical Turk (MTurk) crowdsourcing platform (e.g., Buhrmester et al. 2011; Casler et al. 2013; Holden et al. 2013), the study examined the interrelationships among parental FH of substance misuse, DD, and personal substance use in a large sample of community adults and tested a mediational relationship between these variables. To address limitations of the existing literature by maximizing power and resolution, the current study employed a large sample, characterized density of parental misuse continuously, and broadened the assessment of parental and personal substance use to include four domains: alcohol, tobacco, other illicit drug use, and a novel index, number of different classes of drugs used. The latter was selected to also permit examination of level of experimentation. These four domains permitted a fuller exploration of DD's association with parental FH across diverse domains of substance use. Given the existing evidence that DD is linked to an array of addictive disorders, the hypotheses were that parental FH, DD, and personal substance use would be significantly positively intercorrelated and that these relationships would be present across substance classes. Additionally, it was hypothesized that DD would statistically mediate the relationship between parental FH of substance misuse and personal substance use.

METHOD

Participants

Participants were recruited via Amazon's Mechanical Turk (MTurk) Web-based data collection platform, an online marketplace where "Requesters" can hire paid "Workers" to complete tasks and surveys. MTurk Workers were pre-filtered such that a Worker could view the study posting only if he/she met the following inclusion criteria: (i) 18 years of age or older; (ii) geographically located in the United States; (iii) must have provided consistently acceptable data on at least 85% of all previously completed MTurk surveys (Requesters have control over accepting and rejecting submitted data). The study posting was titled, "Complete a research study on validating internet-based behavioral economic assessment." It listed a brief description of the assessment battery and study purpose, and stated that workers would be compensated $1 for participation, but only for their first participation. The study posting linked participants to a third-party platform, Inquisit 3.0.6.0 (Millisecond Software), for survey and task completion. Prior to entering the assessment battery environment, all participants completed an electronic consent form approved by the Institutional Review Board of the sponsoring institution. The sample comprised 732 individuals (41% male, 59% female) aged 18–72 years (M = 32.28 years; SD = 11.20) who provided complete data for all assessments; sample characteristics are provided in Table 1.

Measures

Delay Discounting Task—Delay discounting was assessed using an expanded version of the *Monetary-Choice Questionnaire (MCQ)* (Kirby and Maraković 1996; Kirby et al. 1999). The *MCQ* is a validated measure that provides a reliable, quantifiable index for characterizing an individual's delay discounting decision-making preferences. It comprises 27 dichotomous choices between smaller, immediate and larger, delayed monetary rewards (e.g., "Would you rather have $19 today, or $25 in 53 days?"). A "discounting rate," or k value, represents the rate at which an individual devalues a reward based on its delay. A k value is inferred from the individual's choices across pre-configured items. A higher k value indicates a steeper discounting rate and suggests a stronger preference for smaller, immediate rewards. While the standard *MCQ* offers choice preferences across three delayed reward magnitudes, small ($25–$35), medium ($50–$60), and large ($75–$85), the current study also employed a "large" version of the *MCQ* (i.e., the MCQ+) (Amlung and MacKillop 2014). The MCQ+ increases the monetary values by one order of magnitude above the standard *MCQ* values, such that participant choice preferences were also assessed across three additional reward magnitudes, $250–$350, $500–$600, and $750–$850, for a more comprehensive assessment of DD. In addition, the delay discounting task included six control items (e.g., "Would you rather have $55 today or $30 today?") to detect low effort/attention. Data was considered invalid for participants who provided incorrect responses (i.e., selected the smaller monetary amount) for more than two of the control items. The original, high magnitude, and control items were mixed together in the assessment.

Family History Assessment—Family history of substance misuse was indexed across three substance classes and separately for a participant's mother and father. Participants were asked to report on perceived substance misuse only in their biological parents. Parental

alcohol misuse was measured using the *Children of Alcoholics Screening Test, Six-item Scale (CAST-6)*, a validated brief screening self-report measure for identifying adult children of alcoholic or drug-abusing parents, in combination with an additional item (i.e., "*Have you ever considered your parent to be an alcoholic?*") (Hodgins et al. 1993). The *CAST-6* consists of six Yes/No items such as, "Have you ever thought that your parent had a drinking problem?" and "Did you ever encourage your parent to quit drinking?" The additional item was included because a previous study found it to function well in identifying children of alcoholics (Hodgins and Shimp 1995). Thus, it was considered to increase the resolution of the assessment with minimal increases in duration. Parental illicit drug misuse was measured using the same items, revised so that each question referenced illicit drug use in place of alcohol consumption. The instructions for these questions stated, "'Drugs' refer to marijuana, prescription pills (when taken other than as described by a doctor), cocaine, amphetamines, opium, heroin, or any other illicit drugs." Parental smoking was assessed using three Yes/No self-report items: (i) "Is your parent currently a smoker?" (ii) Was your parent ever a daily smoker (but has since quit)?" (iii) "Do you think your parent has smoked more than 100 cigarettes in his/her life?" (Shopland et al. 1996). The measure was scored 0/3 with one point given for each "Yes" response (range = 0 [never smoker] - 3 [current smoker]). In contrast to previous studies, parental FH was characterized continuously, rather than dichotomously (FH+ or FH−), in the current sample. Three domain-specific parental FH indices were calculated by combining maternal and paternal misuse within each substance class and converting values to proportions of scale maximum for equivalence across domains. An overall parental FH of substance misuse index was calculated so that each of the three domains contributed equally to the overall density index. Higher index values represent a higher parental FH density for the relevant substance(s).

Personal Substance Use—Personal alcohol use was assessed using the *Alcohol Use Disorders Identification Test* (*AUDIT*), a 10-item measure of alcohol use patterns and related problems over the last 12 months (Saunders et al. 1993). The Cronbach's alpha for the *AUDIT* in the current sample was 0.87. Current scoring standards recommend that total scores of eight or higher are suggestive of hazardous alcohol use (Babor et al. 2001). Personal substance use was characterized using the Alcohol, Smoking, and Substance Involvement Screening Test (ASSIST; World Health Organization 2010) for nine different substances: marijuana, cocaine, methamphetamine, LSD, ecstasy, painkillers (not as prescribed), stimulant medications (not as prescribed), heroin, and opium. Participants were assigned a drug experimentation score reflecting the number of illicit substances they endorsed ever using. Additionally, those who endorsed ever using a particular substance were administered an additional item to characterize their average frequency of use in the last three months. The last three months timeframe is standard to the ASSIST measure and response options included: none, monthly or less, weekly, daily, and multiple times daily. Last three months use frequencies were examined on an individual substance basis. Personal smoking status was classified based on the individual's response to an item assessing frequency of tobacco use during the last three months. Item response options included: none, less than monthly, monthly, weekly, and daily.

Data Analysis

Because participants were allowed to skip items for all measures except for DD, specific imputation and exclusion criterions were set for each measure. For FH, a participant's data would be excluded from all analyses if more than a third of item responses for any parental FH index were missing (i.e., 2/3 items for parental tobacco use and 5/7 items for parental alcohol and illicit drug use). For participants who did not answer every item but had at least a two-thirds response rate for all parental FH measures (n = 78; 10.7%), mean imputations were generated for missing parental FH index items. Because the *AUDIT* is a screening measure for drinkers, a number of participants skipped item number two, which did not offer a non-drinker response option. For those who skipped item two but endorsed "never" having a drink for item one (n = 88; 12%), skipped responses were imputed as the lowest value response option for item two. Participants were excluded from all analyses if they skipped any *AUDIT* items, other than item number two (which is inapplicable to nondrinkers). Internal consistency was calculated for the *AUDIT* and all parental FH indices prior to imputations. As personal smoking and substance use were measured via single items, participants were excluded from all analyses for skipping the relevant item. One exception is for personal marijuana use, where two participants skipped the marijuana use item but were retained in all analyses, except for analyses involving the marijuana variable. All analyses including marijuana use include n = 730 participants. Additionally, for personal substance use, only marijuana use was considered for analysis, as marijuana was the only substance that a relatively large percentage of the sample (20.6%) endorsed using in the last three months. The DD k values were skewed, as is common, and were log10 transformed to improve normality. To generate a magnitude-independent index of discounting, a principal components analysis (PCA) was used to generate a single latent component, using oblique, direct oblimin rotation. Pearson correlation coefficients were generated to examine the uncorrected patterns of relationships in this sample. The PCA-derived index (PCAk) was significantly negatively correlated with income level (Table 4), which supported the inclusion of income as a covariate in the proposed mediation models. No additional variables were significantly correlated with PCAk after accounting for income. If all three variables in the model (i.e., parental FH, delay discounting, and personal substance use) were significantly intercorrelated, mediation was assessed using Preacher and Hayes' (2004, 2008) recommended bootstrapping method for assessing indirect effects in mediator models. The bootstrapping procedure is recommended over other methods because it does not assume a normal distribution and affords higher power (Preacher and Hayes 2004; Preacher and Hayes 2008). Within each model, the indirect effect of parental FH of substance misuse (X) on personal substance use (Y) through PCAk (M) was calculated as the total effect of X on Y (b[YX]) minus the direct effect of X on Y (b[YX.M]). The Preacher and Hayes (2004, 2008) technique using the recommended 5,000 bootstrap resamples with replacement and 95% bias-corrected confidence intervals (CIs) was then used to test the significance of the indirect effect in each model. Analyses were completed using Preacher and Hayes' (2008) SPSS INDIRECT macro, which generates direct and total effects and then tests the indirect effect of the independent variable (IV) on the dependent variable (DV) through the proposed mediator (see Figure 1). The macro generates bootstrap-derived percentile confidence intervals that evidence a significant indirect effect (i.e., mediation) when the CI does not contain zero.

RESULTS

Preliminary Analyses

For the delay discounting data, PCA*k* accounted for 85.07% of the variance. PCA*k* was used in all subsequent analyses; see Table 2 for intercorrelations among *k* values. Internal reliability, as measured by Cronbach's alpha, was generated to assess the value of adding the additional item to the *CAST-6* questionnaire when characterizing maternal and paternal alcohol and illicit drug misuse. Internal reliability was high and, in all four cases, inclusion of the additional item modestly increased the internal consistency of the respective parental FH index by 1–2%; internal reliability results are presented in Table 3.

Interrelationships among Parental Family History, Delay Discounting, and Personal Substance Use

Interrelationships among study variables are presented in Table 4. As predicted, delay discounting was significantly positively correlated with overall parental FH of substance misuse. Significant relationships were also observed between PCA*k* and substance-specific parental FH densities. The highest magnitude association was between PCA*k* and parental FH of smoking. PCA*k* was also associated with parental FH of drinking; however, no relationship was observed between PCA*k* and parental FH of illicit drug misuse. All personal substance use variables were associated with PCA*k* and were positively intercorrelated and significant.

Overall parental FH of substance misuse was robustly associated with personal smoking frequency, level of drug experimentation, and marijuana use. Similar relationships were observed between substance-specific parental FH densities and their personal substance use counterparts, except for personal alcohol use. Contrary to expectations, overall parental FH of substance misuse did not show an association with *AUDIT* scores in this sample.

Mediation Models

Since parental FH of illicit drug misuse was not significantly correlated with PCA*k,* proposed models including parental FH of illicit drug misuse were not included in the mediation analyses. Similarly, the overall parental FH of substance misuse → PCA*k* → *AUDIT* score model was not tested, as the lack of a significant association between the IV and DV precluded mediation for this relationship in the current sample.

Five models were tested for mediation, all including income as a covariate. Significant direct and total effects were observed in all models tested. Bias-corrected CIs for all models did not include zero, demonstrating the significant contribution of PCA*k* to the effect of X on Y and implicating PCA*k* as a presumptive mediator in each relationship. Specifically, PCA*k* partially mediated the relationships between parental smoking and personal smoking, parental alcohol misuse and personal alcohol use, overall parental substance misuse and personal smoking, overall parental substance misuse and personal drug experimentation, and overall parental substance misuse and personal marijuana use. Results of the mediation analyses are presented in Table 5.

Follow-up Analyses

Given the large number of studies using the original *MCQ*, the primary analyses were re-run using the average of the three logarithmically transformed *k* values for the original small, medium, and large reward magnitudes. Most of the effect sizes were very similar to those reported using the MCQ+, but the association between personal alcohol use and delay discounting was no longer statistically significant and the indirect effect for personal marijuana use was no longer statistically significant. Specific findings are provided in Supplementary Materials. This suggests that the higher resolution provided by the MCQ+ may substantively affect the observed relationships.

DISCUSSION

The current study examined the intersection of parental FH of substance misuse, delay discounting, and personal substance use using a large crowdsourcing sample. Specifically, the study tested DD as a mediator of the relationship between parental FH of substance misuse and personal substance use for five of the eight proposed models. Overall, the results were generally consistent with the proposed hypotheses: a steeper discounting rate was associated with greater levels of personal substance use and a denser parental FH of substance misuse, parental FH of substance misuse was associated with personal substance use, and this relationship was partially mediated by DD for all models tested. These findings are consistent with previous studies linking DD and personal substance use (e.g., MacKillop et al. 2011) and recent studies examining the relationship between DD and FH of substance misuse (e.g., Acheson et al. 2011; Dougherty et al. 2014).

In contrast to the general pattern of findings, some predicted relationships were not present. Parental history of illicit drug misuse was not associated with DD, which could be due to the covert nature of illicit drug use and/or the low level of reported parental illicit drug use in this sample. Additionally, overall parental FH of substance misuse was not associated with personal alcohol use, which suggests that familial risk for alcohol use may be substance-specific. It is important to consider the relatively low prevalence rates of personal substance use in this sample when interpreting results. Low substance use rates could account for the absence of significant correlations among certain study variables and the relatively small magnitude mediation effects observed. Given that DD has a stronger association with personal substance use in clinical samples, future studies should examine DD as a mediator of the relationship between FH of substance misuse and personal substance use among addicted individuals.

Importantly, in all models, the findings were indicative of partial mediation and a substantial proportion of variance in the relationship between parental FH of substance misuse and personal substance use was unaccounted for. In addition, the observed effect sizes, measured as adjusted R^2 values, were relatively small, ranging from .02 to .09. This evidence of partial mediation supports DD as a *one* proximate mechanism by which FH of substance misuse contributes to personal substance use, but not *the* proximate mechanism. This reveals the complexity and multifaceted nature of FH as an addiction risk factor. A multitude of other interacting genetic and environmental factors (e.g., excessive reward sensitivity, individual differences in drug metabolism and subjective effects, novelty seeking, social modeling,

substance availability, early adversity, low parental monitoring) likely contribute to the unexplained variance in the tested models (Iacono et al. 2008).

It is also worth noting that the mediational relationships evident in the current study findings can arise in a number of ways. For example, parents who misuse substances might inadvertently model impulsivity or provide consistently unreliable rearing environments that can influence a child's beliefs about the likelihood that waiting for a reward will pay off (Kidd et al. 2013). Additionally, non-supportive parenting practices can undermine a child's development of appropriate planning and self-regulatory skills (Brody and Ge 2001), potentially affecting discounting also. Another possibility is that DD functions as an endophenotype, or a geneticallyinfluenced behavioral characteristic that is partially responsible for transmitting risk for developing an addictive disorder (Gottesman and Gould 2003). Growing evidence suggests that DD satisfies several core endophenotype criteria, but the findings to date are by no means definitive (MacKillop 2013). Longitudinal studies of these domains will be necessary in order to further understand DD's role in the development of substance use.

While DD is only one mechanism by which FH of substance misuse is linked to personal substance use, the current and recent previous findings suggest that it is nonetheless an important one. As such, although it is speculative, a logical extension of these findings is that prevention efforts should consider targeting DD in at-risk individuals. For example, a recent study demonstrated that working memory training decreased DD among stimulant addicts (Bickel et al. 2011). Another study demonstrated similar results with episodic future thinking training (Daniel et al. 2013). Although the literature on strategies for reducing delay discounting remains nascent, if these approaches are supported, the promise is very high and would represent a highly novel prevention strategy.

A notable ancillary finding was the value of adding an additional item to the *CAST-6* when characterizing parental alcohol and illicit drug use. Inclusion of the additional item consistently increased the internal consistency of each respective FH index. Although it requires replication, this finding suggests that the *CAST-6* could be augmented to become the *CAST-7*, both expanding its coverage with one additional high-functioning item but maintaining its brevity. This may ultimately improve the assessment of perceived parental substance misuse.

The current findings must be considered in the context of the study's strengths and weaknesses. Strengths include a robust DD variable that captured discounting preferences across six reward magnitudes, functionally reducing method variance specific to a particular reward size; FH and personal substance use assessments that characterized substance-specific relationships across three substance classes; and a relatively large, well-powered sample, permitting the detection of even small magnitude relationships (e.g., a significant positive relationship between DD and marijuana use has only been demonstrated in one previous study; Moreno et al. 2012). However, several limitations were present also. First, the FH assessment was based on a brief, participant self-report of perceived substance use problems among their biological parents and may have been susceptible to retrospective informant reporter biases. For example, one participant might consider his/her parent to have

an alcohol problem because the parent consumed three drinks per day, whereas another participant might have considered this level of consumption to be normative and thus not problematic. Furthermore, participants might also be unaware of parental substance use, particularly if their parents recovered from early substance use disorders prior to adulthood. In addition, the FH assessment did not use the full diagnostic criteria employed in more extensive measures, and the current study did not gather informant report/outside confirmation of parental substance use problems. However, it is also notable that the *CAST-6* has been specifically subjected to validation in relation to much more comprehensive assessments of family history and has fared well (e.g., Hodgins and Shimp 1995; Hodgins et al. 1993). Second, the personal substance use measures were relatively brief self-report measures, without the resolution of extended interviews, such as the Timeline Followback (Sobell and Sobell 1992) or the Addiction Severity Index (McLellan et al. 1980). Finally, it is important to note that in a cross-sectional study, mediational relationships are necessarily associations among variables and true causality cannot be inferred.

The crowdsourcing methodology is both strength and a limitation. This methodology afforded an efficient means for obtaining a large and relatively diverse sample. MTurk is superior to other crowdsourcing platforms because it allows recruitment from a large, existing pool of reliably rated workers and has built-in tracking capabilities to flag duplicate and invalid responders. Furthermore, evidence suggests that data collected via MTurk is as reliable as those collected in a traditional, in-person laboratory setting (Buhrmester et al. 2011). For example, Holden et al. (2013) demonstrated strong test-retest reliability for a personality measure administered via MTurk, and Casler et al. (2013) found consistent performance on a behavioral paradigm across three sampling methods (MTurk, social media, and in-person data collection). Two recent MTurk discounting studies, Jarmolowicz et al. (2012) and Johnson et al. (2015), provide additional support for the validity of MTurk data and of DD data gathered via this platform. However, crowdsourcing may affect data validity in unpredictable ways, including where and under what conditions workers are completing measures, the influence of community forums, and the greater probability of prior knowledge of tasks. Furthermore, MTurk participants necessarily reflect individuals with access to computers and adequate computer literacy. Despite these considerations, the current study generated data that were largely consistent with data obtained via traditional laboratory methods, supporting the use of crowdsourcing to examine these constructs in future studies. The MCQ+ was similarly a strength and a potential limitation, both leveraging a higher resolution assessment and employing a version that is less compatible with studies exclusively using the original *MCQ*. However, the data was highly orderly internally and it appeared to be somewhat more sensitive than the original *MCQ*, providing some initial support for high-resolution measurement strategies that span diverse reward magnitudes.

In sum, the current study provided further support for DD as one mechanism linking parental FH of substance misuse and personal substance use. These relationships were most clearly present for parental tobacco use and personal tobacco use; parental alcohol use and personal alcohol use; overall parental family history of substance misuse and amount of lifetime drug experimentation; and overall parental family history of substance misuse and

personal marijuana use. Notably, DD was a partial mediator, indicating other variables play a role in this pathway, and a more comprehensive perspective on the mechanisms of this mechanistic relationship could not be examined. These remain priorities for future research in this area.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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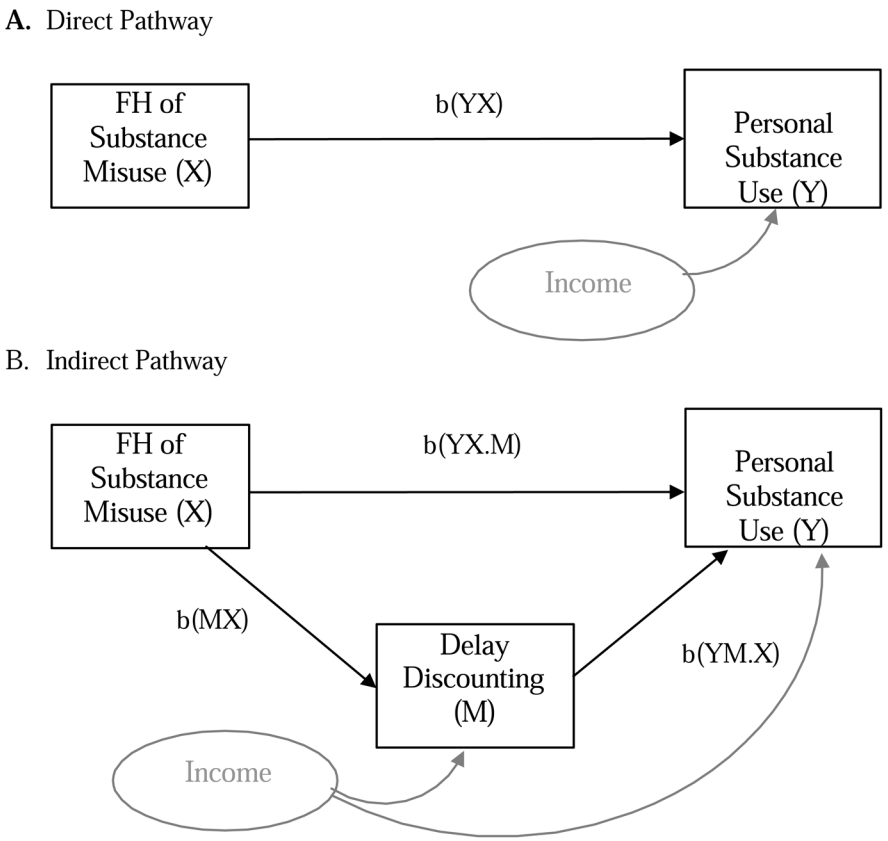

Figure 1.

Delay discounting as a mediator of the relationship between family history (FH) of substance misuse and personal substance use. X refers to the independent variable, Y refers to the dependent variable, XY refers to the direct relationship between the two, M refers to the mediator variable, YX.M refers to the direct effect adjusting for the mediator, and YM.X refers to the indirect (mediating) effect. Income was controlled in all analyses.

Table 1

Participant characteristics

Variable	%/*M* (*SD*)
Demographics	
Gender[a] (% female)	58.49
Age[b] (*M* [*SD*])	32.82 (11.20)
Pretax Income (Median)	$30,000–$44,999
Education (*M* [*SD*])	15.35 (2.76)
Race[c] (%)	
White/Caucasian	79.64
Black/African American	9.70
Asian/Pacific Islander	3.74
Am. Indian/Alaskan Native	1.11
Mixed Race	5.82
Hispanic/Latino Ethnicity[d] (%)	6.87
Personal Substance Use	
Smoking frequency (last 3 months; %)	
None	72.25
Monthly or less	4.51
Weekly	1.91
Daily	2.60
Multiple times daily	18.72
AUDIT	3.94 (4.55)
Marijuana use[a] (last 3 months; %)	
None	79.32
Monthly or less	9.73
Weekly	4.93
Daily	2.47
Multiple times daily	3.56
Drug Experimentation (*M* [*SD*])	1.41 (1.73)
Family History of Substance Misuse	
Parental FH of Smoking (*M* [*SD*])	30.93 (30.86)
Parental FH of Alcohol Misuse (*M* [*SD*])	11.89 (20.66)
Parental FH of Illicit Drug Misuse (*M* [*SD*])	4.36 (14.46)
Overall Parental FH of Substance Misuse (*M* [*SD*])	15.73 (16.45)

Note. M = mean; *SD* = standard deviation; *AUDIT* = *Alcohol Use Disorders Identification Test*; Drug Experimentation = lifetime number of illicit substances ever used; FH = family history. FH mean values represent the percent of total items endorsed for that density index.

[a] *n* = 730,

[b] *n* = 727,

[c] *n* = 722,

$d_n = 728$.

Table 2

Delay discounting task descriptive statistics, and intercorrelations between PCAk and individual magnitude MCQk values. Note that the associations between the individual magnitudes and PCAk reflect component loadings.

	M (SD)	1.	2.	3.	4.	5.	6.	7.
1. PCA*k*	−0.03 (.99)		.88***	.93***	.94***	.94***	.92***	.92***
2. DD: $30	−1.53 (.75)			.86***	.80***	.76***	.71***	.74***
3. DD: $55	−1.70 (.77)				.88***	.84***	.79***	.80***
4. DD: $80	−1.84 (.79)					.87***	.84***	.83***
5. DD: $300	−2.03 (.80)						.86***	.84***
6. DD: $550	−2.20 (.82)							.89***
7. DD: $800	−2.37 (.79)							

Note. M = mean; *SD* = standard deviation; PCA*k* = delay discounting factor score from principal components analysis; DD = delay discounting; monetary amounts listed in parenthesis reflect the average reward amount within the specified DD task version/magnitude.

*** $p < 0.001$.

Table 3

Internal reliability (α) of parental family history (FH) of substance misuse indices

	Maternal	Paternal	Combined
FH of Smoking	0.75	0.78	**0.77**
FH of Alcohol Use	0.92	0.93	**0.90**
FH of Illicit Drug Use	0.96	0.94	**0.92**
Overall FH	0.89	0.88	**0.90**

Note. FH = family history; α = Cronbach's alpha. Internal reliability estimates for the FH indices used in the mediation analyses are formatted in bold font.

Table 4

Zero order relationships among delay discounting, personal substance use, and family history (FH) of substance misuse

	2.	3.	4.[a]	5.	6.	7.	8.	9.	10.
1. PCA*k*	.20***	.08*	.11**	.12**	.14***	.08*	.04	.14***	-.19***
2. Smoking		.23***	.31***	.37***	.26***	.09*	.10**	.23***	-.06
3. Alcohol Use			.21***	.25***	-.00	.08*	.04	.05	.07*
4. Marijuana Use a				.46***	.08*	.07	.14***	.12**	-.13***
5. Drug Experimentation					.23***	.19***	.19***	.28***	-.08*
6. FH of Smoking						.34***	.20***	.82***	-.10**
7. FH of Alcohol Misuse							.41***	.75***	-.01
8. FH of Illicit Drug Misuse								.59***	-.11**
9. Overall FH of Substance Misuse									-.10**
10. Pretax Income									

Note. FH = family history; PCA*k* = delay discounting factor score from principal components analysis.

[a] $n = 730$.

*** $p < 0.001$;

** $p < 0.01$;

* $p < 0.05$.

Table 5

Mediation of the effect of parental FH of substance misuse on personal substance use through DD

Mediation Relationship X→M→Y	Direct and Total Effects		Adjusted R^2	Indirect Effect	Bootstrapping	
					Lower BC 95%CI	Upper BC 95% CI
Model 1:	b(YX)	1.31***				
FH of Smoking → PCA*k* → Smoking	b(MX)	.40***	.09***	.10	.04	.18
	b(YM.X)	.26***				
	b(YX.M)	1.21***				
Model 2:	b(YX)	1.82*				
FH of Alcohol → PCA*k* → *AUDIT*	b(MX)	.38*	.02**	.15	.02	.43
	b(YM.X)	.40*				
	b(YX.M)	1.67*				
Model 3:	b(YX)	2.15***				
Overall FH of Substance Misuse → PCA*k* → Smoking	b(MX)	.71***	.08***	.19	.08	.35
	b(YM.X)	.26***				
	b(YX.M)	1.96***				
Model 4:	b(YX)	2.87***				
Overall FH of Substance Misuse → PCA*k* → Drug Experimentation	b(MX)	.71***	.08***	.10	.02	.22
	b(YM.X)	.14*				
	b(YX.M)	2.78***				
Model 5:	b(YX)	.63**				
Overall FH of Substance Misuse → PCA*k* → Marijuana Use[a]	b(MX)	.72***	.03***	.05	.01	.13
	b(YM.X)	.08*				
	b(YX.M)	.57**				

Notes. Number of bootstrapped resamples = 5,000; FH = family history; DD = delay discounting; *AUDIT = Alcohol Use Disorders Identification Test*; Drug Experimentation = number of illicit substances ever used for those participants who endorsed ever using any illicit drug; PCA*k* = delay discounting factor score from principal components analysis; DV = dependent variable; BC = bias corrected; Y = dependent variable; M = mediator; b(YX) = direct effect of X on Y; b(MX) = direct effect of X on M; b(YM.X) = direct effect of M on Y, controlling for X; b(YX.M) = direct effect of X on Y, controlling for M. The indirect effect of X on Y through M is calculated by subtracting the direct effect of X on Y, controlling for M (i.e., b[YX.M]) from the total effect of X on Y (i.e., b[YX]).

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[a] $n = 730$.

*** $p < 0.001$;

** $p < 0.01$;

* $p < 0.05$.