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Delay Discounting Decreases in Those Completing Treatment for Opioid Dependence

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Abstract

Several studies examining both control and substance-dependent populations have found delay discounting to remain stable over time. In this report, we examine whether delay discounting changes in opioid-dependent individuals who complete a 12-week treatment. The 159 subjects who completed discounting assessments at baseline and treatment-end come from two separate clinical trials: 56 from Chopra et al. (2009) and 103 from Christensen et al. (2012). Mean discounting at 12 weeks significantly decreased to less than half (44.8%) of the baseline level (95% CIs (27.5, 73.2)). Analyzing each subject's discounting data individually, over 3 times (95% CIs (1.9, 5.5)) as many subjects statistically decreased their discounting from their own baseline levels than those who exhibited a statistical increase. Though we failed to find any relationship among discounting measures and abstinence outcomes, the results from this large study suggest that treatment for substance dependence promotes decreases in delay discounting.

Keywords

stability; temporal discounting; impulsivity; opiate dependence; abstinence

Recently, theorists from different research orientations have suggested that drug addiction can be understood, along with other constructs, as a preference for immediate outcomes (Hariri et al., 2006; Reynolds, 2006; Wittmann & Paulus, 2008; Johnson et al., 2010), and this has been described as both an example of impulsive behavior (Stevens & Stephens, 2009) and a psychological trait (Odum & Baumann, 2009). Experimental studies have characterized impulsive behavior as a preference for the smaller and immediately available

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Note that Warren K. Bickel, in addition to having academic affiliations, is affiliated with HealthSim, LLC., the health-promotion software development organization that developed the fluency-based, Computer-Assisted Instruction (CAI) technology employed in the computer-based program used in studies that were reanalyzed in the present study. This technology is unique to HealthSim, LLC and was included in the present project because it is an integral part of the web-based program that was evaluated in the study. Dr. Bickel has worked extensively with Virginia Tech Carilion Research Institute to monitor the relationship between these organizations, oversee all aspects of collaborative projects between the organizations, and ensure that no conflict exists between the author's roles in each organization. The analysis plan and results were conducted by a university statistician with no relationship with HealthSim LLC.

alternative over a larger and delayed alternative (Bickel, Amass, Crean, & Badger, 1999), and this has been observed in different species (Green, Myerson, Holt, Slevin, & Estle, 2004; Johnson & Bickel, 2002), populations (Madden, Bickel, & Jacobs, 1999), and contexts (Dixon, Jacobs, & Sanders, 2006). The preference for smaller, sooner outcomes is called delay discounting and is typically measured with a series of (often hypothetical) choices between an immediately available, smaller reinforcer and a delayed, larger reinforcer. Usually, the smaller reinforcer is adjusted so the indifference point, the choice situation where the subject has no preference between alternatives, can be estimated. This is repeated at several delays. These indifference points are then used to calculate a single measure that quantifies the strength of the preference for smaller, sooner reinforcers. This measure has been used profitably to investigate the problems of drug dependence and to make comparisons between drug-dependent and nondependent groups. For example, steeper discounting rates have been found for opioid users (Kirby, Petry, & Bickel, 1999; Madden et al., 1999; Madden, Petry, Badger, & Bickel, 1997), cocaine users (Heil, Johnson, Higgins, & Bickel, 2006), methamphetamine users (Hoffman et al., 2006), alcoholics (Vuchinich & Simpson, 1998), and smokers (Bickel, Odum, & Madden, 1999) compared with controls. In addition to this discriminate ability, there is also some evidence of delay discounting measures being able to predict smoking treatment outcomes. Discounting measures for prenatal smokers were found to predict relapse following childbirth (Yoon et al., 2007), while discounting measures predicted abstinence in adolescents (Krishnan-Sarin et al., 2007) and lower-socioeconomic-status adults (Sheffer et al., in press) during a smoking-cessation program.

Little is known about the role of addiction treatment on delay discounting, and the evidence so far has been contradictory. Research of those in treatments for substance dependence has found apparent stability in discounting over 1 month in a heterogeneous sample of substance abusers (Aklin, Tull, Kahler, & Lejuez, 2009) and over 2 months in alcoholics (Takahashi, Furukawa, Miyakawa, Maesato, & Higuchi, 2007). Common to both of these studies is that the participants were inpatients. We also note that baseline discounting was obtained 1 month after hospital intake in the Takahashi et al. (2007) study, and whether their discounting 1 month after intake was different from what it may have been at the beginning of treatment is unknown. In contrast, one report using a within-individual design demonstrated a decrease in discounting when participants lowered their carbon monoxide over a week's time in response to a voucher incentive program (Yi et al., 2008). Hence, in the present study, we seek to provide more data by examining whether discounting changes over 12 weeks of addiction treatment and whether discounting behaviors are associated with clinical outcomes. The study is a secondary analysis of data collected in two clinical trials of outpatient treatments for opioid dependence.

Method

The two clinical trials from which the data come have clinical outcomes and detailed descriptions of the trials' methods described in Christensen et al. (2012) and Chopra et al. (2009), and are respectively referred to as Trials A and B. We provide trial information relevant to the current study here, and refer the reader to the original papers for further details.

Participants

Two hundred seventy-two participants (152 from Trial A and 120 from Trial B), aged 18 to 63 years, who met *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; DSM-IV; American Psychiatric Association, 1994) criteria for opioid dependence and the FDA qualification criteria for buprenorphine treatment completed a 12-week treatment study for

opioid dependence. All Trial A participants were recruited at the University of Arkansas for Medical Sciences (UAMS). For Trial B, 75 participants were recruited from the University of Vermont and 45 were recruited from UAMS. For both trials, we required participants to be in good health, report a history of opioid dependence, and have a significant level of current opioid use. We excluded persons with evidence of an active psychiatric disorder or unstable medical illness, those who were pregnant, and those who were incarcerated. For participants living with other participants (e.g., spouses, family members), we randomly chose one from each household to supply data for the study. Participants reporting codependence on alcohol, cocaine, or sedative-hypnotics were not excluded, but those who required detoxification for their own safety were referred to other facilities prior to participation in our study. We recruited participants using radio and newspaper advertisements, flyers, and received referrals from existing participants and local clinics.

To be included in the analyses reported here, the participants had to complete discounting tasks at both baseline and treatment-end 12 weeks later. From Trial A, 103 subjects met this stipulation. Data from only 56 Trial B subjects met the criterion and survived a computer crash that claimed data from up to 44 other participants who completed the 12-week treatment. See Table 1 for demographic and key baseline characteristics.

Procedure

Participants were consented in a 6- to 8-hr session where they were also assessed using several psychological, behavioral, and drug-taking measures. They also met with trained therapists and completed a functional analysis of their drug-taking behaviors and developed a treatment plan to achieve abstinence. All participants signed the appropriate university institutional review board (IRB) approved consent form.

Once inducted, all participants were stabilized on a buprenorphine dose (8–16 mg). Trial A participants were randomly assigned in approximately equal allocation to a voucher contingency (VC) management treatment, enhanced with a computerized version of the community reinforcement approach (CRA), or a voucher contingency management treatment lacking CRA (CM alone). The VC treatment was replicated in Trial B, which also had a buprenorphine medication contingent (MC) treatment, with CRA and standard counseling (SC). Trial B participants were approximately equally randomized among these three treatments. In both trials, the treatment course was 12 weeks in duration, during which time the participants visited the clinic on Monday, Wednesday, and Friday of each week. At each visit, participants supplied a urine specimen that was tested for evidence of methadone, opiates, propoxyphene, cocaine, oxycontin, and, once a week, for benzodiazepines. The treatment protocols were reviewed and approved by the appropriate university IRB.

Treatments

All participants in Trials A and B were stabilized on a buprenorphine dose (6, 12, or 18 mg) on which they were maintained during the 12-week treatment regimens (see Chopra et al., 2009, and Christensen et al., 2012, for complete details). Additionally, three of the four treatments had a contingency management component. Contingency management is the systematic reinforcement of desired behaviors and the withholding of reinforcement or punishment of undesired behaviors (Higgins & Petry, 1999).

Community Reinforcement Approach

The community reinforcement approach (CRA) is a treatment component where environmental contingencies are used to encourage behavior that is incompatible with the problem behavior and also to eliminate the positive benefits from participating in the problem behavior (Miller, Meyers, & Hiller-Sturmhofel, 1999). CRA was delivered by

computer as described in Bickel, Marsch, Buchhalter, and Badger (2008). The computer delivery includes a fluency-building component where participants are required to develop a predetermined level of accuracy and speed in responding for each topic. These questions were initially presented in the same order as presented in the instruction materials, and then presented several times in random order. The read and response timing were progressively shortened as the participant successively completed each presentation in the random order section of the fluency-building component, or increased if the answers were incorrect. Once three correct responses were made on three consecutive presentations of the same question, that question was no longer presented. This component was included in the VC treatment, which appeared in both trials, and in Trial B's MC treatment.

Voucher Contingency Management

Participants were reinforced for supplying urine specimens negative for drugs of abuse with points (each worth \$0.25) that they could subsequently redeem for either gift cards at a variety of local businesses or for cash, when participants had unspent study credit over \$100. The first negative specimen was reinforced with 10 points (\$2.50); each consecutive negative urine specimen increased the point value of a negative urine specimen by 5 points. Three consecutive negative specimens received a \$10 bonus, while a positive specimen reset the subsequent point level to the starting level of 10 points. Earned points were never taken away. Participants needed to submit a negative specimen to receive their vouchers. The maximum possible amount that could be earned was \$997.50. See Chopra et al. (2009) and Bickel et al. (2010) for further details. This component was delivered as a stand-alone treatment in Trial A (i.e., CM alone), and was combined with the CRA component to form the VC treatment, which was used in Trials A and B.

Medication Contingency Management

A different contingency management component was buprenorphine medication contingencies, which consisted of two parts. The first involved participants receiving twice their daily maintenance dose on Mondays and Wednesdays, and three times the daily dose on Fridays, if they provided opioid- and cocaine-free urine samples. If a urine sample was positive for one of the target drugs (methadone, opiates, propoxyphene, cocaine, oxycontin, and, once a week, for benzodiazepines), the participant was required to report for dosing under a 5-days-per-week schedule. This continued until they had provided three consecutive drug-free samples and attended daily for at least 5 consecutive weekdays. At this point, they could return to the 3-day-a-week dosing schedule. The second part of the medication contingency included the use of a dose alteration procedure, wherein the patients' daily buprenorphine dose depended on both clinic attendance and recent abstinence. A cocaine- or opioid-positive urine sample resulted in the participant receiving only half their daily maintenance dose when they attended the clinic each weekday. If the participant submitted a drug-positive sample on a Friday, they received double their daily maintenance dose for the weekend rather than a triple dose. Reduced dosing continued until the first opioid- and cocaine- free urine sample was submitted (whereas release from the daily dosing schedule required three consecutive drug-free urines). Thus, after a drug-positive urine, participants were under the daily dosing requirement for a longer time than the reduced dosing aspect of the contingency. This component, combined with CRA, made up the MC treatment in Trial B.

Standard Counseling Treatment

Participants in this group did not receive voucher points on submission of opioid- and/or cocaine-free urine samples, nor did they receive dose reductions and daily dosing on submission of opioid-and/or cocaine-positive urine samples. However, they continued to receive thrice-weekly dosing with buprenorphine/naloxone and standard methadone-style

counseling sessions once a week throughout the period of the treatment. Study therapists were informed about the urinalysis results and discussed them with the participants. This “standard counseling” (SC) treatment was the only one of the four described that did not have a contingency management component.

Measures

Delay discounting—The delay discounting assessment used in both trials is described in detail in Johnson and Bickel (2002). The task was administered with a computer program. For each of the following hypothetical delays (in order of presentation)—1 day, 7 days, 14 days, 1 month, 6 months, 1 year, 5 years, or 25 years—a series of trials was presented. A trial was the choice between a smaller sooner (SS) and larger later (LL) reward, which remained at \$1,000 throughout the task. The purpose for each series of trials was to determine an indifference point—a proportion of the LL reward that is deemed equivalent to the LL amount when available immediately. A double-limit algorithm was used to narrow the series of choices to the indifference point. The first choice trial for each delay was between the LL amount and SS amount that was 50% of the LL (e.g., \$500). The computer program randomly chose subsequent SS choices within a range of values. The range would shrink to an indifference point when choices were consistent and would reset when choices were not. For example, if a participant chose an immediate \$125 over a delayed \$1,000 on one trial, and the subsequent trial offered an immediate \$85, choosing either the SS or LL would be consistent with the first choice and the range would shrink. If, however, the participant was offered \$195 as the SS, and chose the delayed \$1,000, this would be inconsistent with the previous \$125 choice and the range would reset.

The choices made between smaller, immediate outcomes and larger, delayed rewards are indicative of impulsive or self-controlled behavior (Madden & Johnson, 2009). Specifically, a tendency to choose the SS amounts are associated with impulsivity, while preferences for larger delayed rewards are associated with self-control (Madden & Johnson, 2009). The degree to which a participant’s preferences lie on this continuum is often quantified using Mazur’s (1987) hyperbolic function:

$$Y=1/(1+kD) \quad (1)$$

where Y is the true indifference point (a proportion) at delay D for a person having k as his or her discounting parameter.

Abstinence outcomes—In both trials, participants provided urine samples three times each week (Monday, Wednesday, and Friday) for 12 weeks. On the same day as collected, samples were tested for the panel of substances listed previously. We recorded the participant as abstinent only when the provided sample was negative for the paneled substances; that is, missed samples were treated as “positive.” Primary abstinence measures were longest continuous abstinence and total abstinence, both counts with a maximum of 36.

Statistical Methods

Using a reparameterized version of Mazur’s hyperbolic function (Equation 1), we estimated the discounting parameter normalized with the natural logarithm, $\ln(k)$:

$$Y=1/(1+\exp(\ln(k))D) \quad (2)$$

where Y , k , and D were as before. Squaring the correlation of observed indifference points with predicted indifference points provided a pseudo- R^2 for assessing model goodness-of-fit. Since the amount of variability in the estimates of $\ln(k)$ differed among the individuals, we

accounted for this heterogeneity of variance problem by constructing weights for the estimated $\ln(k)$ values, as recommended by Landes, Pitcock, Yi, and Bickel (2010). We also computed area under the curve (AUC) for each discounting task (Myerson, Green, & Warusawitharana, 2001). The weighted $\ln(k)$ values and AUCs were analyzed in an analysis of variance (ANOVA) context, accounting for repeated measures. Further, to learn whether an individual statistically changed his or her discounting from one time point to another, we estimated the difference in $\ln(k)$ values between two time points using the “direct estimation method” recommended in Landes et al. (2010, p. 178, Equation 4). That is, to each pair of discounting tasks differing only in their time of administration, we fitted the indifference points with

$$Y = 1 / (1 + \exp[w \ln(k_1/k_0) + \ln(k_0)]D) \quad (3)$$

where k_0 and k_1 are, respectively, Mazur’s discounting parameters from the baseline and treatment-end time points, and w is a covariate indicating whether the indifference points come from baseline ($w = 0$) or from treatment-end ($w = 1$). We note that $\ln(k_1/k_0)$ is the difference in the log-transformed discounting parameters, k_0 and k_1 , and the statistical inference on this parameter tests whether the difference is zero. Based on the results of these tests, particular to the individual, we also provide percentages of those statistically changing their discounting from baseline levels and Pearson correlations between the discounting measures from baseline and treatment-end. To examine possible relationships among discounting measures and abstinence outcomes, we computed Pearson correlations. Since a substantial portion of the subjects (39.6%) documented complete abstinence over the 12-week period, we also partitioned the subjects into those who were and were not completely abstinent, and then used ANOVA (accounting for abstinence group, study, and their interaction) to evaluate whether these two abstinence groups differed in their discounting measures. All analyses were conducted in SAS version 9.2, with mixed models being fitted in the MIXED procedure. “Significant” refers to results with $p < .05$.

Results

Discounting Decreases Over Time in Treated Subjects

The median (1st quartile, 3rd quartile) pseudo- R^2 , after fitting Equation 2 to the discounting assessments from Trials A and B participants, was 0.898 (0.758, 0.950). The subjects from both trials were analyzed together, with a repeated measures ANOVA accounting for study, treatment (nested within study), time period, and all interactions. Mean $\ln(k)$ at treatment-end was lowered by 0.8090 (95% CI (0.3321, 1.2853), $p = .0016$), from a baseline level of -2.9102 . In terms of k , the treatment-end k was 44.5% of the baseline k (95% CI (27.7, 71.7); Figure 1). The analysis of AUC values returned a similar inference with the AUC at treatment-end being 47.1% more (indicative of *less* discounting) than baseline levels (95% CI (24.7, 69.5), $p < .0001$). Though not of primary interest, analyses of both $\ln(k)$ and AUC revealed that those in Trial A discounted less than those in Trial B ($p_{\ln(k)} = .0010$ and $p_{\text{AUC}} = .0009$). None of the other effects in the ANOVAs were found to be significant (all $ps > .60$).

We also analyzed the data from each trial independently in repeated measures ANOVAs that accounted for treatment, time period, and their interaction. For both trials, treatment-end discounting was significantly lower than baseline discounting (Trial A: $p_{\ln(k)} = .0028$, $p_{\text{AUC}} = .0020$; Trial B: $p_{\ln(k)} = .0134$, $p_{\text{AUC}} = .0101$). Additionally, there was no evidence of a difference among the treatments (from ANOVAs for 2 trials $\times$ 2 discounting measures; all $ps > .72$), or of an interaction between treatment and time period (all $ps > .76$). Table 2 provides summary statistics of the discounting measures for both trials.

More Treated Subjects Decrease Discounting Over Time Than Increase

Though a population may experience a mean shift over time, individuals can deviate from the pattern by either not shifting at all or shifting in the other direction. Table 3 describes how individuals changed (or did not change) their discounting. There was no evidence that the two trials differed in the proportions of those significantly decreasing, not significantly changing, and significantly increasing their baseline discounting, $\chi^2_{[2]}=0.483, p = .786$. Considering only those that statistically changed their discounting (82 participants across both trials), the proportion of those showing a significant decrease was 3.1 times (95% CI (1.8, 5.4)) that of those showing a significant increase.

Correlations of the same measure taken over time are often used to evaluate stability of the measure at the individual level, with higher magnitude positive correlations being indicative. We correlated the estimated discounting parameters between the two time periods and found a low-magnitude positive correlation, Pearson $r = .337$ for $\ln(k)$, and $r = .396$ for AUC (both $ps < .001, N = 159$).

Relationships of Discounting With Abstinence Outcomes

Pearson correlations of abstinence measures with discounting measures are in Table 4; no relationship was evidenced (all unadjusted $ps > .10$). We split the 159 participants into those who documented complete abstinence for the 12 weeks ($n = 63$) and those who did not ($n = 96$), and tested for mean differences in the discounting measures between these two groups, while accounting for study and their interaction. Though those who were completely abstinent had a mean baseline $\ln(k)$ that was 0.01035 lower and decreased their treatment-end $\ln(k)$ by 0.5597 more than those who were not, these differences were not significant ($t_{[155]} = 0.03, p = .9729$ and $t_{[155]} = 1.24, p = .2165$, respectively); AUC results were similar. We also examined whether the distributions of individually based statistical change differed among those who documented complete abstinence (36 negative urine samples) for the 12 weeks and those who did not; the percentages were nearly equal, differing by no more than 2 percentage points ($\chi^2_{[2]}=0.028, p = .9861$; see Table 5). Finally, those in Trial A tended to achieve longer abstinences than those in Trial B: 6.3 (95% CI (2.6, 10.0)) and 4.0 (95% CI (1.8, 6.3)) more visits in longest continuous abstinence and total abstinence, respectively.

Discussion

This study examined delay discounting data taken longitudinally from participants in two separate clinical trials of treatments for opioid dependence. The main finding from this study is that, on average, participants' delay discounting decreased over time—a result found in the combined, more powerful analysis, and verified in trial-specific, less powerful analyses. Decreases were independent of study and treatment received. Finally, there were 3 times more individuals whose treatment-end discounting had statistically decreased from their own baseline than those who statistically increased. This latter finding was substantiated by the positive, low-magnitude correlations of discounting parameters taken across time. We will discuss four points related to these findings.

First, the opioid treatments used in the two trials considered in this study were designed to move behaviors of substance-dependent persons more in line with those having no substance dependencies. Besides refraining from drug use more often than opioid dependents, those not dependent on substances also tend to discount less than their dependent counterparts (Madden et al., 1997). Further, those who are not undergoing some sort of manipulation tend to exhibit stable discounting (Appendix; Audrain-McGovern et al., 2009; Beck & Triplett, 2009; Kirby, 2009; Ohmura, Takahashi, Kitamura, & Wehr, 2006). With respect to

abstinence, the subjects in this study documented, on average, high percentages of 36 scheduled urine collections that were negative for the paneled drugs: 91% and 80% for Trials A and B, respectively. Additionally, 46% of Trial A and 29% of Trial B participants were completely abstinent for the entire 12 weeks. As for discounting, these subjects tended to decrease from baseline discounting at both the population and individual levels. However, we found no evidence of a relationship between discounting measures (either baseline discounting or discounting changes) and overall abstinence outcomes, thus leaving the clinical relevance of discounting ambiguous.

Second, rather than casting discounting into a “state or trait” mold, we suggest that discounting is stable under stable conditions. For discounting to change, conditions must change, and the individual must adapt to the new state, which may take time. Recently, Bickel, Yi, Landes, Hill, and Baxter (2011) recruited stimulant-dependent individuals from a substance-abuse treatment facility and randomized them to receive executive function training or a sham training program. Bickel and colleagues (2011) found discounting to decrease over time in those undergoing the actual treatment, but no such decrease in the sham-trained subjects. Coupled with the findings of Bickel et al. (2011), the notion that discounting can change in response to an intervention is strengthened.

Third, though comparison between Trials A and B was not a primary objective, we did find Trial B participants to generally discount more and document fewer negative urines than those in Trial A. These differences between the two trials could be related to the recruited populations, trial staff, execution of the trials, some combination of these, or something else entirely. Our analyses of these potential relationships failed to find any statistical relationship; however, reduced variability in abstinence outcomes (due in part to the ceiling on possible negative urines) may have reduced statistical power for detecting any existing relationship. In the expected direction, we note that those who documented complete abstinence experienced, on average, a larger decrease in their treatment-end discounting than those who did not; and this mean difference (0.56) was not much smaller than the overall drop in treatment-end discounting of 0.80.

Fourth, to speak of within-individual stability across a population, it is reasonable to describe the proportions of those who statistically change one way or the other as well as those for whom the evidence is inconclusive. A method for detecting within-individual change of discounting (Landes et al., 2010) allowed us to provide these descriptions in this study. We learned that among the approximate half who statistically changed their discounting from baseline to treatment-end, a large majority (75%) of these decreased their discounting. Others have described within-individual (i.e., “differential” or “relative”) stability with correlations (e.g., Beck & Triplett, 2009; Kirby, 2009; Takahashi et al., 2007; Ohmura et al., 2006). This is certainly valid yet somewhat troubling, in that, if the sample size is large enough, even low-magnitude correlations become statistically significant, much like the correlation we observed between baseline and treatment-end discounting, $r = .337$ for $\ln(k)$. Hence, in addition to correlations, we recommend including proportions of those who statistically decrease, increase, or fail to change their discounting in future within-individual discounting studies.

The major limitation of this study is that we had no control group; that is to say, we had no sample of opioid-dependent treatment seekers who were observed over time without receiving any treatment for their dependency. The two clinical trials from which the discounting data came were designed to test experimental opioid cessation treatments against established controls. However, since the original control treatments are known to have at least some efficacy, these are not appropriate controls for the objectives of this study. Indeed, usual care standards for the treatment of opioid-dependent participants make

it difficult to consider how to construct a control group that would not receive that standard. Of the studies we found that specifically examine discounting over time, none used the same procedure to assess discounting that was used in the two trials reported here. (We also note that there was no common discounting procedure among them.) We were, however, given access to longitudinal discounting data from Yoon et al. (2007) that (a) were obtained with the same discounting assessment used in our two trials, (b) were collected from a substance-dependent group who received no specialized treatment for their dependency, and (c) revealed stable discounting over an 18-week time period (see Appendix). The data from Yoon and colleagues (2007) rule out any concerns about the test–retest reliability of the discounting assessment used. More importantly, their data suggest that in the absence of treatment for substance dependence, discounting remains stable.

One may also question whether the results seen for discounting of hypothetical outcomes would be the same as for discounting of real outcomes. Though we had no discounting of real outcomes in this study to verify, others have found estimated discounting parameters from hypothetical and real outcomes to be highly and positively correlated and not different in brain regions activated (e.g., Bickel, Pitcock, Yi, & Angtuaco, 2009; Johnson & Bickel, 2002; Madden, Begotka, Raiff, & Kastern, 2003). Additionally, our discounting model (Equation 1) assumed utility was constant over the hypothetical delays. We hence considered the possibility that utility is nonlinear (specifically assuming Equation 4 in the supplementary material of Pine et al., 2009) and reanalyzed our data. The findings fully supported those reported here.

Lastly, our main finding of decreased discounting differs from reports by Takahashi et al. (2007), studying alcoholics, and Aklin et al. (2009), studying a heterogeneous sample of substance users. The former concluded discounting was stable over a 2-month period after finding no statistical evidence of a difference between baseline and study-end discounting measures. The latter also failed to find hypothesized discounting decreases over a month's time. Common to both studies, subjects were in a residential treatment facility, where abstinence was required to stay in treatment. The difference in results between their studies and ours may be due to treatment types (inpatient vs. outpatient), statistical power ($N = 35$ in their studies and $N = 159$ in ours), timing of the discounting assessments (not exceeding 2 months in their studies vs. 3 months in ours), some other factors, or a combination of these.

Ultimately, our study shows that discounting tended to decrease for the majority of individuals who completed a range of opioid-dependence treatments while achieving high percentages of abstinence during treatment. This suggests that discounting is a mutable quality reflecting the incremental changes akin to improving physical fitness, learning a new skill, or, in our case, becoming drug free. This may even map the developmental growth of a person. We suggest that discounting is a primal reflection of the evolving nature of a person, where even stable characteristics can change to meet the demands of the new environment.

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Appendix. Stability of Discounting in Untreated Substance-Dependent Subjects

Regarding the specific discounting task used to assess discounting in this study (for a detailed description, see Johnson & Bickel, 2002), no other published work has rigorously considered whether discounting is stable or changes in a control population over time. (Others have reported stable discounting before, but with different discounting tasks.) Yoon et al. (2007) used the same task we used in their longitudinal study of discounting in pregnant women who had spontaneously quit smoking cigarettes. The participants discounted hypothetical future gains of \$1,000, as in both of our clinical trials. Yoon and colleagues (2007) graciously provided access to their discounting data. They had a control arm of 25 pregnant women who spontaneously quit smoking and who received no incentive for smoking cessation except for participation in the study. These women received “usual care for smoking cessation provided through their obstetric clinics, which typically involved provider inquiry regarding smoking status and a discussion of the advantages of quitting during pregnancy (pg. 1017, Higgins et al., 2004).” Estimated discounting parameters (k , equivalent to $\ln(k)$) were collected 3 times before childbirth, at approximately 10, 14, and 28 weeks antepartum, and 5 times afterwards. We only report on discounting before childbirth, as we could not evaluate the psychological issues arising from childbirth, like postpartum depression, which may have affected subsequent discounting behaviors.

We analyzed the estimated $\ln(k)$ values from Yoon et al.’s (2007) study with a one-factor ANOVA, accounting for the within-individual factor, time, and having an unstructured (i.e., general) covariance among observations within an individual. We failed to find an overall difference among the time periods ($F_{[2,23]} = 0.41, p = .6700$). More importantly, we found each pair of time periods to be statistically *equivalent* within a (4/5, 5/4) region (all unadjusted $ps < .003$). That is to say, the smallest of the three means was statistically greater than 80% of the largest mean, and the largest mean was statistically less than 125% of the smallest mean. The Pearson correlations of $\ln(k)$ values between the first and second, first and third, and second and third time periods were 0.87, 0.75, and 0.58, respectively (all unadjusted $ps < .002$).

Figure 1.
Estimated discounting curves at baseline (solid) and treatment-end (dotted).

Table 1

Summary Statistics of Baseline Characteristics for the Participants From Trials A and B

Measure	Trial A (N = 103)	Trial B (N = 56)
Caucasian (%)	93	98
Male (%)	50	57
Never married (%)	46	48
Completed high school (%)	85	86
Employed full time (%)	30	48
Received prior opioid treatment (%)	46	70
Age (mean [SD])	34.9 (9.9)	32.7 (10.5)
Monthly income USD (mean [SD])	1265 (1725)	2073 (3073)
Years of cocaine use (mean [SD])	1.4 (2.7)	3.5 (5.8)
Years of opioid use (mean [SD])	7.9 (7.3)	6.9 (8.0)
Previous month's spending on opioids USD (mean [SD])	567 (710)	2253 (3227)
Preferred Route (%)		
Intranasal	9	38
Intravenous	12	39
Oral	80	23
Alcohol dependence (%)	14	9
Cocaine dependence (%)	5	14
Sedative dependence (%)	11	14
Cannabis dependence (%)	23	38

Table 2
Summary Statistics of $\ln(k)$ and AUC for Each Trial-Time Period Combination

Trial	Time period	$\ln(k)$		AUC	
		Mean	SD	Mean	SD
A	Baseline	-3.419	1.677	0.329	0.289
	Treatment-End	-4.378	2.131	0.445	0.353
B	Baseline	-2.470	1.774	0.209	0.243
	Treatment-End	-3.221	2.937	0.345	0.358

Table 3

Distributions of Individually-Based Statistical Change in \$1000 Discounting From Baseline to Treatment-End

Individual's change	Trial A (<i>n</i> = 103)	Trial B (<i>n</i> = 56)	Combined (<i>N</i> = 159)
Significant decrease (%)	41	36	39.0
No significant change (%)	47	50	48.4
Significant increase (%)	12	14	12.6

Table 4

Pearson Correlations Between Discounting Measures and Abstinence Measures

Abstinence measure	ln(k)			AUC		
	Baseline	Treatment-end	Baseline–Treatment-end	Baseline	Treatment-end	Baseline–Treatment-end
Longest continuous abstinence	–.086	–.131	.069	.089	.081	–.012
Total abstinence	–.083	–.121	.062	.057	.071	–.027

Note. Abstinence measures based on counts of urine samples negative for assayed drugs; *N* = 153.

Table 5

Distributions of Individual-Based Statistical Change Compared Between Completely Abstinent Subjects (36 Negative Urines) and Those Who Were Not (35 Negative Urines)

Group	Individual's change in treatment-end discounting			Total (%)
	Significant decrease (%)	No significant change (%)	Significant increase (%)	
36 Negative urines (<i>n</i> = 63)	40	47	13	100
35 Negative urines (<i>n</i> = 96)	39	49	12	100