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# A Quantitative Signature of Self-Control Repair: Rate-Dependent Effects of Successful Addiction Treatment

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Warren K. Bickel<sup>1</sup>, Reid D. Landes<sup>2</sup>, Zeb Kurth-Nelson<sup>3</sup>, and A. David Redish<sup>4</sup>

<sup>1</sup>Addiction Recovery Research Center, Virginia Tech Carilion Research Institute; <sup>2</sup>Department of Biostatistics, University of Arkansas for Medical Sciences; <sup>3</sup>Wellcome Trust Centre for Neuroimaging, Institute of Neurology, University College London; and <sup>4</sup>Department of Neuroscience, University of Minnesota

## Abstract

Excessive temporal discounting undergirds addiction, and the quantitative relationships of changes in discounting have yet to be investigated. The quantitative relationship between pre- and posttreatment discount rates was examined using data from five of our studies with diverse interventions across different groups of substance users. Discounting and treatment-outcome drug-use data from 222 drug-dependent individuals were analyzed. The primary measures were discounting of delayed reinforcers and objective measures of drug use. Results revealed that change in discounting was inversely related to baseline rates of discounting, such that participants with low discount rates showed little change in discounting with treatment, whereas participants with high discount rates showed large reductions in discounting. As importantly, those treatments that produced the largest gains in drug abstinence had the largest effects on discount rates. Temporal discounting changes with the specific quantitative signature of rate dependence, and more efficacious treatments remediate high discounting rates.

## Keywords

self-control, addiction, rate dependent, temporal discounting, neurobehavioral, biomarker

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The inability to delay gratification along with the excessive devaluation of delayed reinforcers constitute important aspects of self-control failure evident in substance-dependence disorders (Bickel, Koffarnus, Moody, & Wilson, 2014). This failure of self-control appears persistent, which raises questions about its modifiability by treatment and how treatment interacts across the diversity of that bias evident among individuals with substance dependence (Odum, 2011). At one extreme, the more traitlike this bias, the more likely that treatment will have positive outcomes among those individuals exhibiting a less severe form of this bias. Alternatively, this bias may function as an enduring but changeable state that is alterable by treatment with the degree of change perhaps proportional to the extent of this bias. Importantly, how the relationships of changes in discounting and extent of dysfunction interact with treatments of differing efficacy is unknown.

To investigate these issues, we reanalyzed five of our prior studies in which we examined different treatments

in which patients with different forms of addiction were randomly assigned to study conditions (in four of the five studies) to ascertain whether and to what extent measures of this immediate bias change. All five studies measured temporal discounting at the beginning and the end of the intervention. Also, four of these studies collected biological measures of drug use, thereby permitting a measure of treatment efficacy that allows an assessment of the impact of treatment efficacy on immediacy bias.

The approach we have taken here is generally consistent with novel approaches being developed to investigate psychopathology (for a review, see Bickel, Jarmolowicz, Mueller, Koffarnus, & Gatchalian, 2012; Redish, 2013). Those approaches seek to obtain new

## Corresponding Author:

Warren K. Bickel, Addiction Recovery Research Center, Virginia Tech Carilion Research Institute, 2 Riverside Circle, Roanoke, VA 24016  
E-mail: wkbickel@vt.edu

empirical insight into the psychopathology by discerning crosscutting processes shared across psychopathologies, identifying previously unrecognized subtypes among individuals with a particular disorder, and identifying processes that distinguish individuals who do and do not respond to a given intervention.

To accomplish these aims, we employ methods developed in behavioral economics and neuroeconomics that have successfully identified dysfunctional decisions strongly evident in addiction (Bickel, Jarmolowicz, Mueller, & Gatchalian, 2011). As a measure of immediacy bias, these economic approaches use a specific procedure referred to as temporal discounting (also referred to in various literatures as delay discounting, discounting of delayed reinforcers, intertemporal choice, time preference, or the continuum ranging from impulsivity to self-control). *Temporal discounting* refers to the decreased value of a reinforcer as a function of its temporal distance (Mazur, 1987). Excessive discounting seems strongly related to substance dependence (Bickel et al., 2014): Excessive discounting predicts the start of drug use (Audrain-McGovern et al., 2009), increases with amount of drugs used (Johnson, Bickel, & Baker, 2007; Ohmura, Takahashi, & Kitamura, 2005; Takahashi, Ohmura, Oono, & Radford, 2009; Vuchinich & Simpson, 1998), and distinguishes addicted individuals from those without addiction (for a review, see Bickel et al., 2012; MacKillop et al., 2011).

Although discounting at the group level robustly distinguishes those who are substance dependent from those who are not (MacKillop et al., 2011), overlap between the groups is observed when the distributions of discounting from individual participants are examined (Bickel, Yi, Kowal, & Gatchalian, 2008). Those substance-dependent individuals who discount in the normal range may not substantively change after intervention, which perhaps suggests that their dependence results from a different dysfunction (Redish, Jensen, & Johnson, 2008). In this case, an orderly relationship may exist between the extent of discounting at baseline (e.g., how close to or far away from the normal range) and the extent of change after an intervention. Such an orderly relationship may reveal a signature of change between preintervention and posttreatment temporal discounting.

Quantitative signatures of change in decision tasks have not previously been reported among drug dependent individuals. However, a signature of change, referred to as rate dependence, has been observed in behavioral pharmacology and may be applicable to temporal discounting. *Rate dependence* generally refers to an inverse relationship between baseline rates of responding and rates of responding after an intervention (Witkin & Katz, 1990). Rate-dependent effects have also been observed in both drug and nondrug interventions (Bickel, Higgins,

Kirby, & Johnson, 1988; Koffarnus, Jarmolowicz, Mueller, & Bickel, 2013) and have been posited as a basis of the therapeutic effects of stimulant medication seen among individuals with hyperactivity (Bowers, Winett, & Frederiksen, 1987). We, therefore, set out to ascertain whether temporal discounting changed in a rate-dependent manner and to examine whether those changes were systematically related to treatment efficacy.

## Method

### *Participants*

Across the five studies, 222 of 514 initial participants contributed usable temporal-discounting data for this analysis. A total of 165 participants dropped out of their respective studies, which is consistent with retention outcomes reported in a variety of studies in addiction (see Dutra et al., 2008, for a meta-analysis); 70 were excluded from analyses according to criteria of Johnson and Bickel (2008) for the identification of nonsystematic discounting performance; data from 44 individuals were lost in a computer crash; and data from 13 were not available for unknown reasons. In the following sections, we describe each study and the number of participants associated with each (see also Table 1 for study descriptions and details on participant results and discounting tasks). All of the studies were approved by an institutional review board, and written informed consent was collected from all participants before any data were collected or treatment was administered.

**Working Memory Trial (WMT).** The WMT had two treatment arms, one receiving a working memory training (WMTraining) and the other a control training (WMTc; Bickel, Yi, Landes, Hill, & Baxter, 2011). These two arms are treated as separate study groups. Twenty-seven patients at a residential treatment facility for stimulant dependence and abuse were randomized to WMTraining ( $n = 14$ ) or WMTc ( $n = 13$ ). Usable discounting data were obtained from all 14 WMTraining participants and from 11 WMTc participants at the start and end of treatment. No objective measure of drug use was reported in this study; however, randomly tested urine samples had to be negative for participants to continue.

**Opioid Dependence Trial 1 (ODT1).** The ODT1 had three treatment arms. All participants received the opioid replacement medication, buprenorphine. In addition, the first arm received standard counseling, the second arm received abstinence-contingent modifications of buprenorphine dosing, and the third arm received abstinence-contingent vouchers for services or goods from local businesses and money (see Chopra et al., 2009, for

**Table 1.** Brief Descriptions of Each Study

Study-group abbreviation ( <i>n</i> )	Description
WMTraining (14) and WMTc (11)	Working Memory Trial. Two treatment arms (training: WMTraining; control: WMTc). WMTc is considered a separate study group because it was a true sham. Abstinence was not monitored. Originally, 27 participants entered treatment, which lasted 25 days on average (Bickel, Yi, Landes, Hill, & Baxter, 2011). All but 2 participants (both WMTc) supplied usable pre- and posttreatment discounting measures.
ODT1 (39)	Discounting tasks: R100, H100, and H1,000 administered at baseline and treatment end. Opioid Dependence Trial 1. Three treatment arms. Abstinence monitored thrice weekly. Originally, 120 participants entered the 12-week treatment (Chopra et al., 2009; Murphy, MacKillop, Vuchinich, & Tucker, 2011). Discounting data were lost on 44 participants in a computer crash, which left 76 who supplied pretreatment discounting measures, of which 57 supplied both pre- and posttreatment measures, of which 39 supplied usable pre- and posttreatment measures. Discounting tasks: H1,000 administered at baseline and treatment end. Completers' mean pretreatment discounting was $-4.35$ ; noncompleters' mean was $1.17$ higher—95% CI for the difference = $[-0.41, 2.76]$ , $t(73) = 1.47$ , $p = .15$ .
ODT2 (83)	Opioid Dependence Trial 2. Two treatment arms. Abstinence monitored thrice weekly. Originally, 152 participants entered the 12-week treatment (Christensen et al., 2013), of which 150 supplied pretreatment discounting measures, of which 108 supplied both pre- and posttreatment measures, of which 83 supplied usable pre- and posttreatment measures. Discounting tasks: H1,000 and H10,000 administered at baseline, midtreatment, and treatment end. Completers' mean pretreatment discounting was $-6.11$ ; noncompleters' mean was $0.04$ lower—95% CI for the difference = $[-0.90, 0.98]$ , $t(141) = 0.08$ , $p = .93$ .
SRS (35)	Smoking Relapse Study. One treatment arm. Abstinence monitored weekly starting in Week 4. Originally, 80 participants entered the 8-week treatment (Murphy et al., 2011; Sheffer et al., 2012), of which 72 supplied pretreatment discounting measures, of which 37 supplied both pre- and posttreatment measures, of which 35 supplied usable pre- and posttreatment measures. Discounting tasks: R100, H1,000 (future and past) administered at baseline. H100 (future and past) administered at baseline and weekly thereafter. Completers' mean pretreatment discounting was $-5.39$ ; noncompleters' mean was $0.56$ higher—95% CI for the difference = $[-0.77, 1.90]$ , $t(70) = 0.84$ , $p = .40$ .
SDT (40)	Stimulant Dependent Trial. Two treatment arms. Abstinence monitored thrice weekly. Total of 135 stimulant-dependent participants in 12-week treatment, of which 132 supplied pretreatment discounting measures, of which 63 supplied both pre- and posttreatment measures, of which 40 supplied usable pre- and posttreatment measures. Discounting tasks: H1,000 administered at baseline and treatment end. Completers' mean pretreatment discounting was $-2.43$ ; noncompleters' mean was $0.84$ lower—95% CI for the difference = $[-0.41, 2.09]$ , $t(122) = 1.33$ , $p = .19$ .

Note: For administered discounting tasks, "H" and "R" indicate hypothetical and real rewards, respectively. Numbers are the amount of the reward in U.S. dollars. Unless otherwise indicated, all rewards are in the future. Those participants supplying usable discounting data at both pre- and posttreatment are compared with those supplying only baseline discounting data. CI = confidence interval.

full details). Of the 120 individuals who entered treatment, 88 completed treatment. Some discounting data were lost in a computer crash. Thirty-nine participants had usable discounting data from baseline and posttreatment.

**Opioid Dependence Trial 2 (ODT2).** The ODT2 had two treatment arms, both of which were identical to the third treatment arm in ODT1 with the addition that one arm included a Web-based behavior therapy intervention

(for full details, see Christensen et al., 2013; Everly et al., 2011). A total of 170 participants entered treatment with 111 completing treatment. Of the completers, 83 had usable discounting data from both pre- and posttreatment.

**Smoking Relapse Study (SRS).** The SRS consisted of only one treatment and was designed to examine predictors of treatment outcome; participants received group cognitive-behavior treatment, and nicotine replacement

therapy was not provided (American Cancer Society, 2010). Eighty participants started treatment. Of 58 completers, 35 provided usable discounting data at pre- and posttreatment.

**Stimulant Dependent Trial (SDT).** The SDT had two treatment arms. Both arms received attendance-contingent vouchers for local businesses and cash, and one arm also received Web-based therapy intervention.<sup>1</sup> Of the 152 individuals who entered treatment, 73 completed treatment. Forty participants supplied usable discounting data at pre- and posttreatment.

## Procedure

**Discounting tasks.** Two adjusting-amount discounting tasks were used among the five studies: one based on the double-limit algorithm (Johnson & Bickel, 2002) and the other on a decreasing-adjustment algorithm (Du, Green, & Myerson, 2002). These two tasks have been statistically compared and found to produce comparable outcomes (Kowal, Yi, Erisman, & Bickel, 2007). The former was used in ODT1 and ODT2 and the latter in WMT, SRS, and SDT. Researchers in these studies had participants discount a variety of temporal outcomes; these outcomes, along with the times at which discounting tasks were administered, are shown in Table 1.

**Rate estimation.** Participants in each study completed discounting tasks at the beginning of treatment (baseline) and were scheduled to complete discounting tasks at the end of treatment. Not all participants completed treatment. Noncompleters could not be evaluated in the primary analyses for this work because we had no measure of treatment-end discounting. We compared mean pre-treatment discounting among noncompleters and completers: The largest observed difference in baseline discounting came from the ODT1 study, in which completers' mean was 1.17 lower than noncompleters', 95% confidence interval (CI) = [-0.41, 2.76],  $t(73) = 1.47$ ,  $p = .15$  (see Table 1). Discounting tasks in which the participant provided no variability (i.e., always gave the same response) across time frames were excluded from analyses (i.e., were not usable; Johnson & Bickel, 2008).

## Measures

The discounting tasks used in these studies presented the participant with a series of “smaller-now-versus-larger-later” choices between an immediate or delayed reward. These choices are summarized into a single measure of discounting that quantifies the extent to which the participant discounts a delayed reward. We used the method described in Johnson and Bickel (2002) to compute a

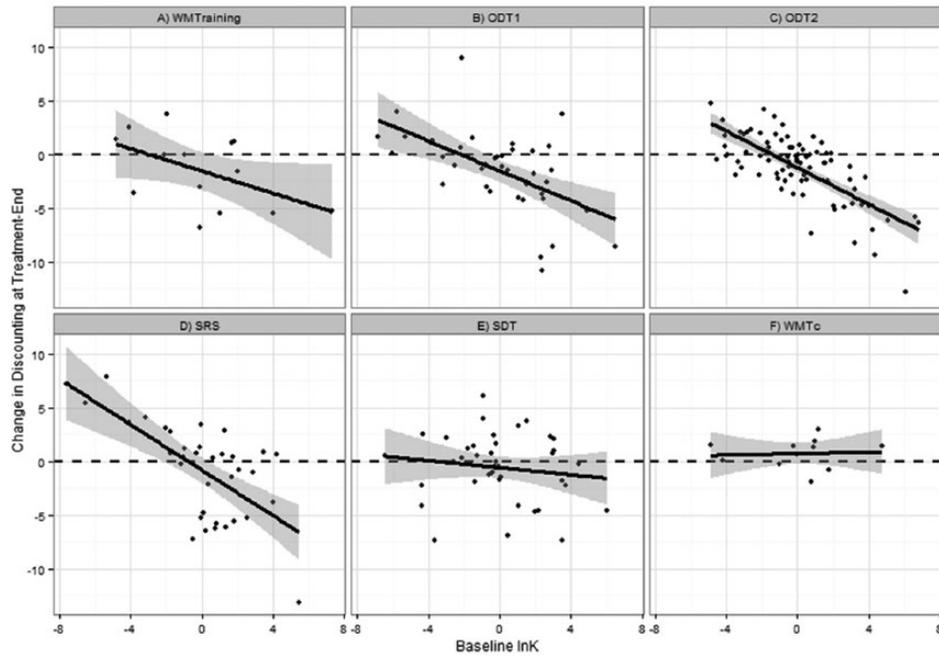
normalized measure of discounting based on Mazur's (1987) hyperbolic function,  $Y = 1/(1 + kD)$ , where  $Y$  is the expected indifference point for a reward delayed by  $D$  days and discounted at a “rate” of  $k$  days<sup>-1</sup>. The natural logarithm of estimated  $k$  values is approximately normal in distribution. For this reason, we used  $\ln(k)$  as our discounting measure for a particular task. We were also interested in how much a person changed his or her discounting between pre- and posttreatment; thus, for same-type discounting tasks, we captured this change with  $d_{\ln(k)} = \ln(k_{post}) - \ln(k_{pre})$ . For studies having more than one type of discounting task, an individual's average  $\ln(k_{pre})$ ,  $\ln(k_{post})$ , and  $d_{\ln(k)}$  were used as his or her data points.

Biochemical measures of drug use were obtained in four of the studies. Specifically, during ODT1, ODT2, and SDT, urine samples were collected under observation at pre- and posttreatment study visits and three times per week throughout the treatment phases. Each urine specimen was tested on-site using a Siemens V-Twin drug-testing diagnostic system with Syva EMIT reagents for methadone, opioids, propoxyphene, cocaine, and benzodiazepines. In addition, Oxycontin was tested using a single-panel Clinical Laboratory Improvement Amendments-waved Oxycontin dipstick (see Chopra et al., 2009). For SRS, exhaled carbon monoxide was measured weekly during the treatment phase of the study (see Sheffer et al., 2012).

## Statistical methods

**Rate dependence and regression to the mean.** When the magnitude and direction of a change measure—here,  $d_{\ln(k)}$ —depend on the initial starting point, or  $\ln(k_{pre})$ , the change is said to be “rate dependent.” Regressing the change,  $d_{\ln(k)}$ , on the initial measure,  $\ln(k_{pre})$ , evaluates rate dependence. The regression slope of  $\ln(k_{pre})$  measures the magnitude and direction of that dependence; when  $\ln(k_{pre})$  is centered about its mean, the regression intercept measures the expected change in  $d_{\ln(k)}$  regardless of  $\ln(k_{pre})$ .

It is possible, though, for there to be evidence of rate dependence, but, on average, there is no difference in the distributions of initial discounting, or  $\ln(k_{pre})$ , and treatment-end discounting, or  $\ln(k_{post})$ ; this is known as “regression to the mean” (RtM). RtM is when more extreme measures converge toward the mean when resampled (Koffarnus & Katz, 2011). Mathematically, this occurs when the mean and variance of  $\ln(k_{pre})$  and  $\ln(k_{post})$  are equal, that is, mean  $d_{\ln(k)}$  is 0, and the expectation of  $\ln(k_{post})$  given  $\ln(k_{pre})$  is  $(1 - \rho)\mu + \rho\ln(k_{pre})$ , where  $\mu$  is the mean of  $\ln(k_{pre})$  and  $\rho$  is the correlation of  $\ln(k_{pre})$  and  $\ln(k_{post})$ . If RtM is true, then the difference  $\ln(k_{post}) - [(1 - \rho)\mu + \rho\ln(k_{pre})]$  should equal 0 on



**Fig. 1.** For each study, individuals' change in discounting (ln scale) was plotted against baseline discounting (ln scale, centered about the sample mean). Lower values of both baseline discounting and change in discounting indicate more self-control and becoming more self-controlled. Solid lines are linear regressions with standard errors; horizontal lines are at 0, the null hypothesized regression. WMTraining = Working Memory Trial training; ODT1 = Opioid Dependence Trial 1; ODT2 = Opioid Dependence Trial 2; SRS = Smoking Relapse Study; SDT = Stimulant Dependent Trial; WMTc = Working Memory Trial control.

average. Because of the mathematical relationship between correlation ( $\rho$ ) and simple linear regression and the relationship among  $\ln(k_{pre})$ ,  $\ln(k_{post})$ , and  $d_{\ln(k)}$ , RtM is estimated with the aforementioned regression intercept.

To examine rate dependence within each of the six study groups, we regressed  $d_{\ln(k)}$  on  $\ln(k_{pre})$  centered about its mean (see Fig. 1 and Table 2 for results). Residuals of  $d_{\ln(k)}$  had homogenous variance and no substantial violations of normal errors. In addition to original regression coefficients, we report standardized regression coefficients to allow easier comparison of effect sizes among the studies. CIs for the intercept from these regressions also provide a goodness-of-fit test for RtM; CIs containing 0 imply that RtM is a plausible explanation of any rate-dependent effect. We also used a bootstrap method (using 1,000 bootstrapped samples per study group) to obtain a nonparametric verification of RtM's goodness of fit and RtM's effect size to compare among studies (Efron & Tibshirani, 1993; see Table 2).

**Biochemical measures of abstinence.** For each individual within a study, we computed the percent of documented abstinence out of the total number of originally scheduled visits: 36 visits for ODT1, ODT2, and SDT and

4 visits for SRS. We plotted within-study means of these percentages by mean change in discounting.

We used an alpha of .05 for all tests and present 95% CIs.

## Results

### Rate-dependent effects

On average, participants in the active-treatment study groups (excluding WMTc) had improved discount rates at treatment end relative to pretreatment levels (see Table 2, intercepts), and patients with higher discount rates tended to improve (decrease) more than their counterparts with closer-to-normal discounting (see Table 2, slopes), although not statistically improved in all cases. Examination of relationships between baseline discounting and changes in discounting at treatment end revealed a common pattern in three of the six study groups (ODT1, ODT2, and WMTraining) but not in the other three groups (SRS, SDT, and WMTc): that substantially more participants than expected (i.e., half) decreased their discount rates (improved) from baseline to treatment end. Furthermore, improvements in discounting were more robust relative to the other studies (see Table 2, standardized estimates).

**Table 2.** Regression Coefficients of Change in Discounting at Treatment End on Baseline Discounting and Tests of Goodness of Fit From Regression to the Mean

Study	<i>n</i>	Regression of discounting change on baseline discounting					Regression-to-the-mean goodness of fit <sup>a</sup>			
		Intercept		Slope		Standardized estimate ( <i>b<sub>st</sub></i> )	Estimate		Standardized estimate (RtM <sub>st</sub> )	
		Estimate	95% CI	Estimate	95% CI		Estimate	95% CI		
WMTraining	14	-1.53	[-3.30, 0.24]	-0.52	[-1.07, 0.03]	-0.51	-1.53	[-3.34, 0.13]	-0.52	
WMTc	11	0.75	[-0.24, 1.74]	0.03	[-0.35, 0.42]	0.07	0.75	[-0.08, 1.47]	0.53	
ODT1	39	<b>-1.52</b>	<b>[-2.55, -0.50]</b>	<b>-0.69</b>	<b>[-1.04, -0.35]</b>	<b>-0.56</b>	<b>-1.52</b>	<b>[-2.72, -0.38]</b>	<b>-0.49</b>	
ODT2	83	<b>-1.23</b>	<b>[-1.69, -0.76]</b>	<b>-0.85</b>	<b>[-1.03, -0.67]</b>	<b>-0.72</b>	<b>-1.23</b>	<b>[-1.94, -0.58]</b>	<b>-0.58</b>	
SRS	35	-0.78	[-1.96, 0.39]	<b>-1.06</b>	<b>[-1.47, -0.64]</b>	<b>-0.67</b>	-0.78	[-2.32, 0.61]	-0.23	
SDT	40	-0.56	[-1.56, 0.44]	-0.16	[-0.54, 0.21]	-0.14	-0.56	[-1.56, 0.31]	-0.18	

Note: Confidence intervals (CIs) not containing 0 indicate statistical significance ( $\alpha = .05$  level and are in bold font). Baseline discounting was centered about the sample mean. WMTraining = Working Memory Trial training; WMTc = Working Memory Trial control; ODT1 = Opioid Dependence Trial 1; ODT2 = Opioid Dependence Trial 2; SRS = Smoking Relapse Study; SDT = Stimulant Dependent Trial.

<sup>a</sup>H<sub>0</sub>:  $E(d_{RM}) = 0$ , where  $d_{RM} = \ln(k_{post}) - [(1-p)\mu + p\ln(k_{pre})]$ .

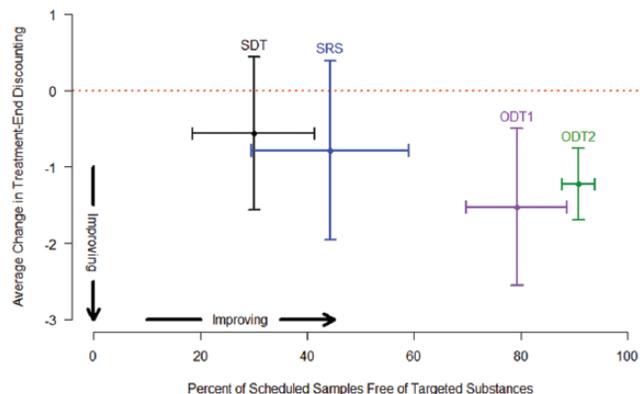
Starting with the study on WMT, we found that 9 out of 14 (64%) WMTraining participants showed improvement in discounting rate from baseline to treatment end. Those participants who discounted most at baseline showed the greatest change, which implied a rate-dependent effect ( $b_{st} = -0.51$ ,  $p = .0633$ ). Although RtM cannot be ruled out, it was only marginally plausible as a parsimonious explanation of how extreme discounters change over treatment, thus implying that some other process may have been driving the change. (We note that the standardized RtM effect, RtM<sub>st</sub>, was  $-0.52$ , and a 90% CI for the RtM effect did not include 0.) In contrast, only 3 out of 11 (27%) of the WMTc participants decreased their discounting from baseline levels to the end-of-treatment test, and the relationship between baseline discounting and change in discounting was near 0 ( $b_{st} = 0.07$ ,  $p = .8437$ ; see Fig. 1 and Table 2).

Among the clinical studies, 26 out of 39 (67%) of ODT1 participants decreased their treatment-end discounting from baseline levels. Plotting treatment-end change as a function of baseline discounting revealed a rate-dependent effect, such that those participants who discounted most at baseline showed larger improvements (decreases) than did those who initially discounted less ( $b_{st} = -0.56$ ,  $p = .0002$ ). Likewise, 55 out of 83 (66%) of ODT2 participants decreased their treatment-end discounting from baseline, and ODT2 participants who discounted most at baseline showed the greatest improvement ( $b_{st} = -0.72$ ,  $p < .0001$ ). For both ODT1 and ODT2, the distributions of treatment-end discounting differed from baseline distributions; thus, RtM failed to explain the observed rate dependence—ODT1: RtM<sub>st</sub> =  $-0.49$ ; ODT2: RtM<sub>st</sub> =  $-0.58$  (see Table 2 and Fig. 1).

In contrast, only 16 out of 35 (46%) of SRS participants discounted less at treatment end than at baseline. As with the previous three active-treatment groups, SRS participants showed a rate-dependent effect ( $b_{st} = -0.67$ ,  $p < .0001$ ). Although rate dependence was clearly evident, the changes individuals experienced may have been RtM, with a standardized effect size less than half the magnitude of that for WMTraining, ODT1, and ODT2 (RtM<sub>st</sub> =  $-0.23$ ). Similarly, only 21 out of 40 (53%) of SDT participants exhibited a decrease in treatment-end discounting compared with baseline levels. The relationship between change and baseline discounting was not significantly different from 0 ( $b_{st} = -0.14$ ,  $p = .3794$ ), nor was there any compelling evidence to suggest anything beyond RtM (RtM<sub>st</sub> =  $-0.18$ ; see Table 2 and Fig. 1).

### **Relationship of changes in discounting to treatment outcomes**

The four random-assignment clinical studies documented abstinence from the substance of abuse targeted for cessation. We measured treatment efficacy within each study group by averaging the participants' percentages of their total number of scheduled visits that were negative (i.e., drug-free) samples. Plotting the change in discounting (adjusted for baseline discounting) by percent of drug-free samples, we found that study groups with the largest decreases in discounting rates also documented the most abstinence (see Fig. 2). Among the four study groups, ODT1 participants exhibited the largest average decrease in treatment-end discounting (intercept =  $-1.52$ ,  $p = .0048$ ), and 79% of the scheduled urines were negative for paneled drugs. Similarly, ODT2 participants showed



**Fig. 2.** For each study having clinical verification of abstinence, mean change in discounting, adjusted for baseline discounting, plotted against the percent of scheduled visits that were documented abstinences within the particular study. Vertical error bars are 95% confidence intervals for the mean change in discounting; horizontal error bars are 95% confidence intervals for the true percent of documented abstinences. SDT = Stimulant Dependent Trial; SRS = Smoking Relapse Study; ODT1 = Opioid Dependence Trial 1; ODT2 = Opioid Dependence Trial 2.

decreased discounting (intercept =  $-1.23$ ,  $p < .0001$ ) and 91% drug-negative samples. In contrast, the decrease in discounting experienced by SRS participants was attenuated and failed to significantly differ from no baseline discounting (intercept =  $-0.78$ ,  $p = .1839$ ). Furthermore, only 44% of scheduled breath samples had carbon monoxide less than or equal to 8 ppm. SDT participants showed the least decrease in baseline discounting levels (intercept =  $-0.56$ ,  $p = .2636$ ), and only 30% of scheduled urine samples were drug negative (see Table 2 and Fig. 2).

## Discussion

In this study, we report for the first time that temporal-discount rates changed in a rate-dependent fashion after highly efficacious treatments and working memory training. Less efficacious treatments and the control condition for working memory training did not produce rate-dependent effects. These findings extend the understanding of how temporal discounting as a measure of self-control can change as a function of interventions. Koffarnus et al. (2013) recently reviewed 30 studies in which researchers attempted to change temporal discounting. An overwhelming number of these studies were conducted in nonpatient populations (e.g., college students) and used transitory interventions (e.g., examining acute doses of alcohol or framing the choices differently), and none focused on the relationship between baseline rate of discounting and the rate observed after the intervention.

Moreover, only 4 of the 30 research reports reviewed by Koffarnus et al. (2013) were therapeutic interventions.

Two of those 4 were not reanalyzed here. One study reported a decrease in monetary and cigarette discounting from pre- to postintervention, and relative to a control group, among participants who completed a 5-day contingency management procedure reinforcing reductions in carbon monoxide among cigarette smokers (Yi et al., 2008). The other study reported a decrease in temporal discounting among alcohol- and cocaine-dependent individuals after participants' completion of an extensive monetary-management training program (Black & Rosen, 2011). Thus, the finding from the present report that temporal discounting showed a systematic relationship between preintervention and posttreatment across participants and the relationship between that effect and the efficacy of the treatment constitute a novel finding in this research domain. Whether rate dependence relates to the transitory interventions remains to be determined. In the following paragraphs, we address five aspects of our findings.

First, our results suggest a provocative hypothesis that effects of an intervention on temporal discounting may be a marker of its efficacy. The only treatments that changed discounting were highly efficacious. Working memory training was not examined as a clinical treatment, but it also changed discounting in a rate-dependent fashion. Therefore, if that change is a marker of efficacy, then working memory training should be an efficacious treatment. Studies of working memory have shown that it decreases alcohol consumption (Houben, Wiers, & Jansen, 2011), helps sustain weight loss among obese adolescents (Verbeke, Braet, Goossens, & van der Oord, 2013), and improves symptoms in children with attention-deficit/hyperactivity disorder (Beck, Hanson, Puffenberger, Benninger, & Benninger, 2010). These studies did not measure discounting, but these disorders have been shown to be associated with excessive discounting (Bickel et al., 2012). Moreover, the money-management intervention referred to earlier that changed temporal discounting has been shown to be efficacious as a treatment (Rosen, Rounsaville, Ablondi, Black, & Rosenheck, 2010). Future studies on other putative treatments may be able to use the effects of those treatments on temporal discounting, in those disorders associated with excessive discounting, as a marker of efficacy.

Second, the rate-dependent effects observed here may also be interpreted from the contemporary neuroeconomic perspectives in addiction. Specifically, literature on neuroeconomics has suggested that addiction results from an imbalance between two neurobehavioral decision systems (Bechara, 2005; Bickel et al., 2007); that is, the impulsive decision system, embodied in the limbic and paralimbic brain regions and associated with an immediacy bias, is relatively stronger than the executive decision system, embodied in aspects of the prefrontal

cortices and associated with the valuation of delayed outcomes. These two systems also have been shown to contribute to discounting, and the excessive discounting exhibited by individuals with addiction is consistent with stronger control by the impulsive decision system. The rate-dependent effects we observed may suggest that the efficacious treatment appears to render these two decision systems into something approximating regulatory balance. Of course, this inference will await neuroimaging studies to confirm this speculation.

Third, the fact that a portion of substance-dependent participants did not exhibit excessive discounting suggests that self-control failure is only one of several possible processes that can contribute to drug dependence (Bickel, Jarmolowicz, Mueller, Gatchalian, & McClure, 2012; Koffarnus & Katz, 2011; Redish et al., 2008). This view suggests that addiction results from multiple endophenotypes (MacKillop, 2013) and that these transdisease processes will be exhibited across different addicted populations, with subsets of patients exhibiting different profiles of dysfunctional processes (Redish et al., 2008). Moreover, this view suggests that when the specific processes are identified, treatment can be organized to target the specific dysfunction exhibited by a given patient. For example, the effects of treatment on normalizing impulsivity in the participants with greatest discounting suggests that these interventions may be useful with other impulsive patient groups, such as those with attention-deficit/hyperactivity disorder (Barkley, 1997), gambling problems (Petry, 2001), and obesity issues (Weller, Cook, Avsar, & Cox, 2008). In addition, it is important to note that the heterogeneity of the extent of discounting suggests that replication of this rate-dependent effect will depend on the extent and range of temporal discounting exhibited by the target population. Specifically, recruitment of a sample with a restricted baseline discount rate should result in limited or no changes in discounting.

Fourth, there are at least two substantive weaknesses associated with this report. One potential weakness is that the majority of studies reported here used hypothetical monetary rewards in temporal-discounting procedures, and, perhaps, hypothetical outcomes are not reflective of temporal discounting with real money. In that regard, a substantial body of literature has demonstrated that real and hypothetical outcomes produce comparable behavioral choices (Johnson & Bickel, 2002; Lawyer, Schoepflin, Green, & Jenks, 2011; Madden, Begotka, Raiff, & Kastern, 2003; Madden et al., 2004) and brain activations (Bickel, Pitcock, Yi, & Angtuaco, 2009) and that hypothetical rewards are predictive of real monetary behavior (Bickel et al., 2010); therefore, this concern should be minimal here. Another weakness concerns a problem that often occurs in random-assignment studies in clinical populations generally and among those with addiction in particular, namely, that participant attrition limited the data for analyses. In addition, some

participants exhibited a pattern of behavior that suggests they did not understand or attend to the task, thus making their data difficult to interpret. These limitations are in some ways offset by the fact that our observations were obtained in a diverse and, overall, large number of individuals with different addiction disorders.

Fifth, and perhaps most important, these data set the occasion for two lines of additional research that would have importance for improving the treatment for substance dependence and abuse and understanding why individuals with excessive discounting have worse outcomes is treatments of moderate efficacy. The first line of research would be to explore the possibility of empirically defining those temporal-discount rates that are modifiable. If so, then temporal discounting could be used as a means to cost-effectively personalize treatments. Consider that several moderately efficacious treatments have reported that baseline discounting rates are predictive of therapeutic outcomes (e.g., MacKillop & Kahler, 2009; Sheffer et al., 2012; Stanger et al., 2012; Yoon et al., 2007); that is, participants who discount the most have the worst treatment outcomes. Perhaps, instead of providing everyone with a costly, highly efficacious treatment, only those with changeable discount rates who might not respond to a moderately efficacious treatment could receive a supplemental intervention (e.g., working memory training) that would improve their discount rates and, if discount rates are causally related to therapeutic outcome, improve their treatment response.

The second research line could test whether treatment attenuates the stronger control by the immediate environment that we speculate is evident among individuals who excessively discount delayed events. If individuals who discount excessively, compared with those who discount future rewards less, exhibit stronger subjective response to drug-related cues or to internal stimuli resulting from drug withdrawal, then this may be a reason why those who excessively discount do poorly in moderately efficacious treatments, as we noted earlier. However, if highly efficacious treatments change discounting and produce the greatest change in discounting among individuals who discount the most, as we have shown here, then those individuals, after the intervention, should be less under the control of the immediate environment relative to baseline. This "stimulus-bound" hypothesis of excessive discounting could be tested using either highly efficacious treatment in clinical trials or working memory training in more experimental settings. Indeed, if this hypothesis is valid, then a gradient of change should be obtained where the most stimulus-bound individuals show the greatest proportional change in that dimension. Given that excessive discounting may function as a transdisease process (Bickel et al., 2012), such a demonstration could be relevant to a variety of disorders.

## Author Contributions

WKB conceptualized the analyses and wrote the majority of the paper. RDL analyzed data, wrote portions of the manuscript, and provided comments/edits to drafts. ZKN provided suggestions for analyses, ideas for discussion, and contributed comments/edits to drafts. ADR provided suggestions for analyses, ideas for discussion, and contributed comments/edits to drafts.

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R. D. Landes is now at the Radiation Effects Research Foundation, Hiroshima, Japan.

## Declaration of Conflicting Interests

W. K. Bickel is a principal owner of HealthSim, LLC, which specializes in the research and development of prevention and therapeutic-educational software, and several studies employed a product for which he has a proprietary interest. However, no financial support resulted from this involvement for this study. The other authors have no financial interests to disclose.

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## Note

1. This trial did not demonstrate efficacy, and we have not reported these unpublished data in this article (but they can be obtained from W. K. Bickel).

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