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The Behavioral Economics of Substance Use Disorders: reinforcement pathologies and their repair

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Abstract

The field of behavioral economics has made important inroads into the understanding of substance use disorders through the concept of reinforcer pathology. Reinforcer pathology refers to the joint effects of (*a*) the persistently high valuation of a reinforcer, broadly defined to include tangible commodities and experiences, and/or (*b*) the excessive preference for the immediate acquisition or consumption of a commodity despite long-term negative outcomes. From this perspective, reinforcer pathology results from the recursive interactions of endogenous person-level variables and exogenous environment-level factors. The current review describes the basic principles of behavioral economics that are central to reinforcer pathology, the processes that engender reinforcer pathology, and the approaches and procedures that can repair reinforcement pathologies. The overall goal of this review is to present a new understanding of substance use disorders as viewed by recent advances in behavioral economics.

Keywords

addiction; behavior economics; reinforcer pathology; elasticity of demand; discounting; treatment

INTRODUCTION

> Effects vary with the conditions which bring them to pass, but laws do not vary. Physiological and pathological states are ruled by the same forces; they differ only because of the special conditions under which the vital laws manifest themselves.
>
> Claude Bernard, *An Introduction to the Study of Experimental Medicine*

DISCLOSURE STATEMENT

W.K.B. is a principal in HealthSim, LLC, which specializes in the research and development of prevention science products.

Claude Bernard, the father of experimental medicine, suggested that pathological and normal processes differ quantitatively, not qualitatively. Indeed, pathological processes often result from normal processes that go awry. For example, cell growth and division are processes necessary for continuation of multicellular organisms. However, if cell growth and division are not regulated, then the pathological process of cancer results. The same can be said for behavioral systems, as normal behavioral development and functioning require regularly seeking and obtaining appropriate reinforcers contingent on adaptive and prosocial behavior. For some individuals, these reinforcement processes can go awry when substances with a high potential for dependence are used, particularly in contexts where those substances are easily available and when alternative opportunities for reinforcement are lacking. The role of inordinate reinforcement in substance dependence has been recognized for some time. However, only recently has reinforcement pathology been explicitly recognized and defined (Bickel et al. 2011a, Carr et al. 2011). Reinforcement pathologies have previously been defined as the joint effects of (*a*) the persistently high valuation of a reinforcer, broadly defined to include tangible commodities and experiences, and/or (*b*) the excessive preference for the immediate acquisition or consumption of a commodity despite long-term negative outcomes (Bickel et al. 2011a, p. 407). Excessive consumption of psychoactive drugs and food as well as excessive gambling exemplify reinforcement pathology and constitute substantial public health problems in the United States. Reinforcement pathology, from this perspective, results from the recursive interactions between endogenous (e.g., physiologically mediated subjective response to drugs, steep devaluation of delayed reward) and exogenous factors (e.g., drug and alternative reinforcement price/availability).

The understanding of reinforcement pathology has been facilitated by behavioral economics, a discipline that hybridizes economics and psychology. As we use it here, behavioral economics refers to the application of economic concepts and approaches to the molar study of individuals' choices and decisions. Extending from basic operant learning approaches, application of behavioral economics to substance use disorders began in the 1990s, with considerable success in relating the quantity of drug-taking behavior to substance price. Some years later, this research domain used controls and began to compare the trade-offs between immediate and delayed outcomes in the decision making of those with substance dependence. Over the intervening period, these approaches have been increasingly applied to an ever-widening range of phenomena. In this article, we explore the concept of reinforcement pathology by reviewing (*a*) the basic principles of behavioral economics that are central to reinforcer pathology, (*b*) the processes that engender reinforcer pathology, and (*c*) the approaches and procedures that can repair it.

PRINCIPLES OF BEHAVIORAL ECONOMICS

Reinforcement pathologies are, in part, the result of patterns of choices that have an additive influence on one's subsequent options and choices. Said another way, the decision-making processes underlying the consumption of a pathological reinforcer change as prolonged use of the commodity increases. Clarifying the processes that undergird these choices allows a deeper understanding of reinforcement pathologies. An exhaustive review of the factors that influence choice is beyond the scope of this review. Instead, we focus on two primary

principles of behavioral economics that permit the objective study and precise characterization of reinforcement pathology as we have defined it: demand and discounting.

Demand

Descriptively, individuals who exhibit reinforcement pathology value their substance of dependence more than other commodities. However, by saying "value more," we are referring not only to the total amount consumed or the subjective hedonic value (e.g., drug liking or enjoyment), but also to the total level of resource allocated toward obtaining the substance and to the extent to which consumption is sensitive (or insensitive) to a change in price. Moreover, outside of controlled laboratory environments, price is defined broadly and could include monetary cost, effort, or time required to obtain a commodity or combination of commodities. This inclusive definition of price allows for the application of behavioral economics across many different types of experimental designs and can also be used to compare the behavior of different species. As a convention, price can be examined as a cost-benefit ratio, referred to as the unit price (see below for greater detail on this topic) where the cost (monetary, effort, time, or some combination) is divided by the unit of the commodity. In behavioral studies, unit price is often modeled with response requirement (e.g., with a fixed-ratio schedule of reinforcement) defined as the cost, divided by reinforcer magnitude (e.g., number of reinforcers delivered upon completion of the response requirement) defined as the benefit (Hursh 1980).

The effects of a changing price, or unit price, on purchasing behavior can be discerned by measuring elasticity of demand. Demand, the amount of a commodity purchased at a given price, is described as inelastic when it is relatively insensitive to a price change and elastic when it is relatively sensitive to a price change. Quantitatively, elasticity of demand is defined as the proportional change in consumption relative to the proportional change in price; a proportional change of consumption is either less than the corresponding proportional change in price (inelastic) or proportionally greater than the corresponding change in consumption (elastic) (Hursh 1980, 1984).

Demand can be further investigated by generating a demand curve, which shows consumption as a function of price. Indeed, the two forms of assessment (quantitative and graphical) share many common features; that is, the slope of a demand curve, when plotted in double logarithmic coordinates, is equivalent to a changing elasticity of demand. Demand analysis can distinguish the subjective value for two different commodities (see Figure 1). By quantifying two dimensions of reinforcer consumption, demand curve analysis shows that the reinforcing efficacy of a commodity is inherently dependent on the unit price at which it is assessed and that initial demand for two products cannot necessarily be used to predict preference at higher prices.

As also shown in Figure 1, two additional variables may be determined from demand curves: P_{max} and O_{max} (Hursh & Winger 1995, Winger et al. 2006). P_{max} is closely related to elasticity and is the price at which response output (e.g., money spent or responses emitted) reaches its maximum value. It is also the price at which demand shifts from inelastic to elastic. The corresponding maximal response output at P_{max} is referred to as O_{max}. The price associated with peak responding (P_{max}) is 300. The corresponding expected

peak response output (O_{max}) is 10,000 responses. As described below, individual differences in demand for substances may provide clinically relevant indicators of addiction severity (MacKillop & Murphy 2007).

Unit price—Demand analysis quantifies the effect of price on consumption. As discussed above, price can be more than cost; it can be a cost-benefit ratio that is also referred to as unit price. Unit price has brought parsimony to otherwise disjointed findings in the literature, serving as a standardized rubric to compare studies that used differing prices and reinforcer magnitudes (see also Bickel et al. 2000). Re-analyses of previously published drug self-administration data, along with other more recent studies, have shown that the two components that contribute to unit price (response requirement and reinforcer magnitude) have functionally equivalent effects on drug self-administration (Bickel et al. 1990, DeGrandpre et al. 1993). For example, studies support the implication of unit price that doubling the response requirements and halving reinforcer magnitude yield comparable decreases in drug consumption. This provides parsimony by functionally integrating two processes previously considered independent into one functional process, thus yielding policy importance. Consider this point: The Food and Drug Administration (FDA) can regulate the amount of nicotine contained in cigarettes (but cannot eliminate nicotine altogether) but has no control over the price manufacturers charge for their product. As a method of decreasing demand and consumption for cigarettes, the advances made through the use of unit price in behavioral economic studies discussed above suggest that decreasing the amount of nicotine in a cigarette by half will function the same as if the price were doubled (DeGrandpre et al. 1993).

Reinforcer interaction—Demand for a commodity is important for the choices drug-dependent individuals will make to obtain the reinforcer that contributed to their pathology; indeed, the individual may at times select the substance over opportunities that may have afforded greater long-term advantages. This suggests the importance of also understanding how that specific commodity interacts with the vast number of other commodities that constitute the context within an individual's life. Behavioral economics provides an easily operationalized and sophisticated framework to quantify the degree and type of interaction between different reinforcers. Behavioral economics specifies that commodities can interact in one of three ways that describes a continuum. At one end of the continuum, commodities can function as substitutes; that is, as price increases for a commodity such as butter and its consumption decreases, consumption increases for another commodity (the substitute) that has remained constant in price (e.g., margarine). The slope of increase in consumption of margarine as a function of the unit price for butter would indicate the degree of substitution (e.g., steeper increase indicates greater substitute). At the other end of the continuum, two reinforcers can interact as complements. For example, as the price of soup increases and its consumption decreases, so might the consumption of soup crackers also decrease despite the absence of a change in their price. Between these two extremes, commodities can be independent; that is, as the price of butter increases and its consumption decreases, the consumption of soup may remain unchanged.

Opportunity cost—According to behavioral economics, reinforcers can also interact in another way: "Opportunity cost" refers to situations where options are mutually exclusive; that is, selection of one renders the other choices as unavailable. Economics stipulates that the opportunity cost of a choice is equal to the best alternative not taken (Bickel et al. 1993). The concept of opportunity cost for drug reinforcement has been frequently demonstrated in both nonhuman animal and human studies; that is, alternative reinforcement decreases the consumption of abused substances by increasing the cost of substance use (e.g., Bickel et al. 1993, Higgins et al. 1994, Nader & Woolverton 1991). Said another way, providing the alternative reinforcer in explicit competition with substance use increases the opportunity cost of substance use (i.e., what could be gained from abstinence); therefore, substance use decreases. As described below, individuals with few alternatives to substance use are less likely to change their use successfully (Murphy et al. 2005, Vuchinich & Tucker 1996). Moreover, treatment approaches that attempt to increase levels of substance-free reinforcement---whether by enhancing social support or goal-directed/prosocial behaviors---are generally more effective than treatments that focus only on reducing substance use (Murphy et al. 2012).

Discounting and Related Processes

Discounting processes are another behavioral economic concept that has proven extremely relevant to the study of such reinforcer pathologies as addiction. The general discounting framework determines how much value a reinforcer loses as a function of a manipulated variable. Three primary variables that have been manipulated and are germane to this discussion are delay, probability, and social distance. Delay (or temporal) discounting examines the extent to which the value of a reinforcer decreases as a function of its temporal distance (Mazur 1987, Rachlin et al. 1991). Probability discounting determines the degree to which uncertainty of a reinforcer decreases the value of that reward (Rachlin et al. 1991). Social discounting determines the degree to which a person's valuation of a reinforcer delivered to another person decreases as a function of the social distance between those two individuals (Jones & Rachlin 2006). Other parameters that have been manipulated in the context of discounting include the magnitude and type of the reward (e.g., money, drugs, health, or sex) (e.g., Baker et al. 2003, Johnson & Bruner 2012, Lawyer et al. 2010), whether the rewards are gains or losses (Thaler 1981), combinations of delays and probabilities (Yi et al. 2006), whether the rewards are received in the future or the past (Yi et al. 2006), the influence of delay on social decisions (Bickel et al. 2012b), and cross-commodity (e.g., money now versus drug later) interactions (e.g., Bickel et al. 2011b, Yoon et al. 2009).

Delay discounting—Of all the discounting processes, delay discounting was the first identified and has been the most extensively studied. Experimental research into delay discounting has its origins in Herrnstein's matching law, which specified that allocation of responses in a choice situation is proportional to the relative rates of reinforcement among the options (Chung & Herrnstein 1967; Herrnstein 1961, 1970). This, in turn, suggested that subjective reinforcing value decreases, owing to delay, by dividing reinforcer magnitude by delay (Baum & Rachlin 1969).

Experimentally, delay discounting is commonly studied through assessments of one's preferences between smaller immediate rewards and larger delayed rewards. In humans, money is the reward most typically used in this work owing to its generality as a reinforcer, relative ability to maintain value into the future, and amenability to precise quantification. Many studies have used hypothetical rewards, to which direct behavioral and neural comparisons of real and hypothetical rewards have shown comparable effects (Baker et al. 2003; Bickel et al. 2009; Johnson & Bickel 2002; Johnson et al. 2007; Lagorio & Madden 2005; Madden et al. 2003, 2004). Delay discounting is an intuitive concept, in that when offered a choice between receiving $100 now, versus receiving $100 after a 1-month delay, most individuals would prefer the immediate $100. However, by systematically adjusting choices, as in psychophysical experiments, this intuitive concept becomes a powerful tool for assessing individual differences and understanding fundamental processes in decision making. So, to continue with the example, when faced with the choice between receiving $75 now versus receiving $100 after a 1-month delay, some people may still prefer the immediate option, but others would prefer to wait for the larger delayed option. Likewise, if the immediate option is $50, an even larger proportion of individuals may prefer to wait for the delayed option. By assessing such choices in individuals across a variety of delays, delay-discounting procedures can specifically quantify the devaluation of rewards across delays, which in turn allows an assessment of a graphical representation of this behavior in the form of the discounting function (see Figure 2) and an index of discounting rate. In addition to strict assessment of delay discounting by tracking preferences between smaller-sooner and larger-later rewards, some studies falling within the domain of intertemporal choice have assessed the discounting of different commodities across time, for example, choices between drug reward now versus money reward later (e.g., Bickel et al. 2011b, Yoon et al. 2009).

Building from the framework provided by the matching law, Mazur (1987) determined the relationship between reinforcing value and delay approximated a hyperbola and proposed that the model represented by Equation 1 best described this relationship:

$$V = \frac{1}{1 + kD}. \quad (1)$$

In this equation, V is the present value of the delayed reward; that is, the magnitude of immediate reward subjectively equivalent to a delayed larger reward. V is expressed here as a proportion of the later outcome. D is the delay from the choice until the occurrence of the outcome, and k is a free parameter that serves as an index for discounting rate. Figure 2 shows a hypothetical example of delay-discounting data, showing reward value plotted against delay until the reward, with the best-fitting function defined by Equation 1. Although some newer multiparameter mathematical models provide an improved fit and in turn provide further insights about the nature of the temporal (e.g., Green et al. 1994, Rachlin 2006, Yi et al. 2009), the hyperbolic model expressed in Equation 1 typically provides a good fit across studies that have used varied techniques, such as the use of real and hypothetical rewards. Furthermore, the single parameter k is more interpretable than the parameters from multiparameter models, which are inherently interdependent on one

another. Finally, the hyperbolic model is able to account for an empirically demonstrated feature of delay discounting---the reversal of preferences from larger-later to smaller-sooner rewards as the time until receipt of the smaller-sooner reward draws closer (Frederick et al. 2002, Green et al. 1994) (see Figure 3).

With exponential discounting, if a larger-later reward is preferred when both it and a smaller-sooner reward are distant in the future (as at Time A), then that larger-later reward is preferred even when the smaller-sooner reward is imminently available (as at Time B). Time does not result in a change in preference as the reward draws closer. In contrast, Figure 1 shows hyperbolic discounting and the resulting possibility of a preference reversal. At Time A when both a smaller-sooner and a larger-later reward are far into the future, the discounted-value curve of the larger-later reward is above that of the smaller-sooner reward. Thus, a person would indicate a preference for the objectively larger reward at this point. However, as predicted by the crossing of discounting curves allowed by the hyperbolic functions, at Time B when the smaller-sooner reward is imminently available, people reverse their previous preferences for the larger reward and instead select the smaller-sooner reward.

The choice dynamic implied by the hyperbolic discounting model is reflected in characteristic patterns of decisions made by many substance abusers. Substance abusers often have a fairly consistent stated preference for the larger delayed rewards associated with abstinence that often goes unrealized. Problem drinkers may wake up every morning with a desire to abstain to attain future positive health, education, or vocational outcomes, which they, at 8:00 AM with no immediate access to alcohol in the home, value more than the smaller more proximal rewards associated with drinking. However, when the availability of the smaller-sooner reward is imminent---for example, at the end of the workday when a coworker mentions that there will be a happy hour right after work---the value of drinking, now immediately available, may surpass the delayed value of abstinence resulting in a preference reversal and the decision to drink. This phenomenon may also explain the loss of control that often occurs within substance use episodes. Substance users often violate initial self-imposed limits when faced with the immediate opportunity to consume more of the substance. These self-control failures and instances of behavior that are seemingly at odds with “true preferences” or values are a key feature of relapse and addiction.

Closely related to delay discounting, delay of gratification refers to the observation that waiting for a consequence reduces reinforcement. This framework was pioneered in a series of studies in children by Mischel and colleagues (1972). Participants were asked to wait an unspecified period of time for the experimenter to return to the room before consuming a preferred food reward. In the meantime, the child could signal the experimenter to return to the room at any time, but the participant would receive a less-preferred food reward. Although delay-discounting and intertemporal choice tasks are similar, delay of gratification tasks differ in that receiving the preferred delayed reward requires not only a single choice, but rather continual resistance from opting for the sooner less-preferred reward. In long-term follow-up research, those preschool children who were able to wait for the preferred delayed reward scored higher on college entrance exams, thus suggesting the robustness of such tasks for indexing important future life consequences (Mischel et al. 1989). As reviewed in a

subsequent section, a similar assessment of preference between sooner less-valuable and delayed more-valuable rewards in delay-discounting tasks is also associated with important real-life consequences such as propensity to addiction.

Excessive probability discounting—Probability discounting, or the devaluation of rewards due to their uncertainty, is assessed by tasks very similar to the delay-discounting task described above, except that the smaller and larger rewards differ by certainty rather than delay (smaller certain reward versus a larger uncertain reward, e.g., a choice between a 100% chance of receiving $50 or a 50% chance of receiving $100). In probability discounting procedures, subjects who gamble have shown less discounting due to uncertainty relative to control nongambling participants (Holt et al. 2003, Madden & Bickel 2009). That is, gamblers value uncertain rewards more highly than others. However, the association may be less robust in substance abuse disorders: Some studies have failed to find differences (Andrade & Petry 2012, Reynolds et al. 2003, Takahashi et al. 2009), but others have found that substance users showed greater probability discounting than nonusers (Reynolds et al. 2004; Yi et al. 2007, 2012). Thus, although gamblers may overvalue uncertain rewards, substance abusers may undervalue uncertain rewards. Many of the delayed rewards that may compete with substance use are also uncertain (e.g., future health, educational outcomes), relative to the consistent immediate rewards associated with substance use (e.g., euphoria, sedation, analgesia). Steep delay and probability discounting may reflect a maladaptive decision-making bias, and individuals who devalue delayed or uncertain outcomes may prefer drug use relative to many alternative behaviors (Rachlin et al. 1991). More research is needed to explore the complex relationship among substance use, gambling, and probability discounting.

Other forms of discounting—Social discounting tasks determine the extent to which a participant's personal value of a reward declines as a function of one's social distance to the reward's recipient (Jones & Rachlin 2006). Social discounting tasks have participants make hypothetical choices between receiving a certain amount of money versus having a person at a certain social distance receive an amount of money (e.g., $5 given to you or $75 given the fifth-closest person to you in your life). Individuals typically value a reward given to a very close individual (e.g., a family member, romantic partner, or close friend) more than one given to a distant individual (e.g., a friendly acquaintance). As with delay and uncertainty sensitivity, individuals vary in their sensitivity in the extent to which a certain social distance reduces value to oneself. Social discounting shows remarkable similarities to delay and probability discounting in its mathematical form (Bradstreet et al. 2012; Kowal et al. 2007; Rachlin 2006; Yi et al. 2011a,b).

As with delay and probability discounting, initial research suggests social discounting may be associated with substance use and abuse. Bradstreet et al. (2012) showed that, among pregnant women who were smokers, those who relapsed to smoking after quitting showed a higher degree of social discounting (i.e., showed a greater decrease in personal reward value as a result of social distance to the reward recipient) than those who quit smoking and did not relapse. Yi et al. (2012) found higher rates of social discounting (as well as delay and probability discounting) in methamphetamine users compared with control participants. In

another study, Bickel et al. (2012b) compared individual delay discounting (standard delay discounting) and social temporal discounting (individuals made intertemporal monetary choices for a group of which they are a member) in hazardous-to-harmful drinkers, smokers, and controls. Compared with controls, hazardous-to-harmful drinkers and smokers discounted more on individual discounting, but only smokers discounted delayed rewards more rapidly under the social condition.

Interactions of Demand and Discounting: Experimental Evidence

The comorbidity of excessive discounting and demand (for the commodity to which they are addicted) in individuals with substance use disorder raises the question of whether a relationship exists between these two concepts. An individual who discounts the future may be willing to expend an inordinate amount of resources to obtain a drug dose even at high prices and may consume large quantities when drug prices are low (Murphy & MacKillop 2006). Several studies have explicitly tested whether a directional relationship between demand and discounting exists, but the results from this incipient line of inquiry have been largely inconclusive. For example, in one study in rats, subjects that had higher discounting rates (i.e., those that preferred smaller-sooner over later-delayed food reinforcement) showed lower elasticity for nicotine self-injections (that is, greater continued consumption of nicotine despite increases in the work required to receive the injection) (Diergaarde et al. 2012). However, delay discounting was unrelated to demand elasticity for alcohol self-administration. Another study found that rats with higher delay discounting for a food reward also exhibited greater demand for cocaine, but not for food (Koffarnus & Woods 2013). In humans, a study in smokers found that delay discounting for money was affected by nicotine deprivation, but demand for cigarettes was not affected (Field et al. 2006). Among heavy drinkers, a greater severity of alcohol use disorder was associated with both greater delay discounting of money rewards and greater alcohol demand (MacKillop et al. 2010a). Moreover, higher alcohol demand was associated with greater discounting. However, compared with smokers without schizophrenia, those with schizophrenia have greater demand for cigarettes but not greater discounting (MacKillop & Tidey 2011). The number of studies relating demand and discounting is currently limited, and definitive results will require additional study. Given the importance of these constructs, the relationships between demand and discounting may have a great impact on the understanding of addiction.

PROCESSES ENGENDERING REINFORCER PATHOLOGY

The preceding sections detail key features of substance use disorders from a behavioral economic perspective in phenomenological and operational terms. In this section, we discuss the putative processes that give rise to the overvaluation and overconsumption of psychoactive substances. We also discuss the empirical evidence supporting this perspective. Importantly, in addition to emphasizing the processes of demand and discounting, a behavioral economic perspective is also highly consilient with mechanisms that have been identified from different psychological and neurobiological perspectives. Where sharp differences are present, they result from the framing and theoretical conceptualization of the same empirical phenomena. Most contemporary perspectives

emphasize endogenous (person-level) factors in the development and maintenance of addictive behavior, such as alterations to biological circuits by chronic drug exposure, maladaptive cognitive templates, or intense experiential cravings. What is unique about a behavioral economic perspective is that it attempts to synthesize the concurrent interplay of processes that are operative within the individual and those in the environment. In the following, we use a behavioral economic perspective to discuss both person-level factors and environmental factors in addictive behavior. In addition, we use the same lens to understand neurobiological and genetic perspectives on the etiology of addictive disorders.

Person-Level Factors: Elevated Demand and Delay Discounting

In terms of intraindividual processes, high levels of drug demand and delay discounting (or immediate preference in other forms of intertemporal choice) are theorized to be recursive etiological markers in the development of substance use disorders. Drug demand and delay discounting putatively capture important variability of an individual's valuation of drugs as reinforcers and preference for smaller-sooner rewards relative to larger-delayed rewards, respectively. As recursive markers, they are theorized to reflect both current preferences and, in turn, predict subsequent preferences. This theoretically applies across the chronology of use, from predicting the acquisition and escalation of substance use to forecasting substance use maintenance or success in treatment. The empirical evidence supporting each domain is discussed in turn.

Persistently elevated drug demand as an etiological process—In studies of drug demand, consistent evidence supports a significant association between indices of demand and substance dependence. For example, indices of alcohol demand have been significantly associated with severity of alcohol misuse in studies using young adult samples (Murphy & MacKillop 2006; Murphy et al. 2009, 2013; Wahlstrom et al. 2012) and community samples (Acker et al. 2012, Gray & MacKillop 2013, MacKillop et al. 2010a). Moreover, higher levels of alcohol demand predict poor response to brief alcohol interventions in young adults (Bernstein et al. 2013, MacKillop & Murphy 2007). Specifically, high alcohol demand was associated with a differentially lower intervention response.

Recent studies have also examined indices of alcohol demand in mechanistic relationships with established person-level variables. For example, high alcohol demand moderates the relationship between impulsive personality traits and alcohol misuse, such that higher O_{max} was associated with a stronger relationship between urgency (i.e., acting out under conditions of negative affect) and alcohol-related problems (Smith et al. 2010). Similarly robust relationships have been observed for tobacco demand and nicotine dependence. In a wide diversity of populations, higher levels of tobacco demand are consistently associated with greater nicotine dependence (Chase et al. 2013, Few et al. 2012, Liao et al. 2013, MacKillop & Tidey 2011, MacKillop et al. 2008, Murphy et al. 2011). Also consistent with a role for drug demand in substance dependence, compared with smokers receiving a placebo, those who were administered the efficacious smoking-cessation medication varenicline showed significant increases in demand elasticity for cigarettes (McClure et al. 2013).

In addition, a substantial body of work connects demand for food to obesity, which, in our framework, is also a reinforcement pathology much like substance use disorders. Early studies showed elevated food reinforcement among obese individuals (Jacobs & Wagner 1984, Johnson 1974), and these findings have been largely supported in more recent work using validated assessments of demand. For example, food incentive value has been positively associated with body mass index and obesity in both children and adults (Epstein et al. 2007, Saelens & Epstein 1996, Temple et al. 2008). Laboratory studies also support the link between food demand and eating behavior. Food demand is positively associated with greater food consumption in the laboratory (Epstein et al. 2004, 2008, 2011; Rollins et al. 2010; Saelens & Epstein 1996; Temple et al. 2008). Indeed, food demand is a better predictor of consumption than are food hedonic subjective ratings (Epstein et al. 2004, Temple et al. 2008). Finally, in a recent weight-loss intervention trial in overweight children, food demand predicted treatment success and moderated the effects of an enriched environment (i.e., availability of alternative nonfood reinforcers) (Best et al. 2012). Specifically, an enriched environment was not associated with a positive outcome for children exhibiting high food demand.

A significant limitation to the existing literature on demand as an etiological process is that the vast majority of studies are cross sectional. The prospective clinical studies to date (Bernstein et al. 2013, Best et al. 2012, MacKillop & Murphy 2007) suggest that high demand is a maintaining factor that reduces the benefits of interventions, but additional research is needed to establish elevated demand as an etiological process relevant to substance dependence and to identify predictors of elevated demand. Two recent longitudinal preclinical studies revealed escalating demand for cocaine and opiates over the course of increased experience (Christensen et al. 2008, Wade-Galuska et al. 2011), which is supportive of the etiological hypothesis but is by no means definitive. As such, a recursive causal role remains primarily a hypothesis, and the need for longitudinal studies, especially in human populations, is very high.

Delay discounting as an etiological process—Returning to impulsive intertemporal choice, the evidence supporting a causal etiological role is relatively robust. As with demand, a consistent body of evidence significantly associates steep delay discounting with substance use disorders. Numerous categorical studies have contrasted addiction-related groups with control groups and found significantly greater discounting for substances such as alcohol (Bjork et al. 2004, Claus et al. 2011, Mitchell et al. 2005, Petry 2001, Vuchinich & Simpson 1998, Yankelevitz et al. 2012), cocaine (Coffey et al. 2003, Heil et al. 2006), tobacco (Baker et al. 2003, Bickel et al. 1999b, Heyman & Gibb 2006, Johnson et al. 2007, Mitchell 1999, Reynolds 2006b), opioids (Kirby & Petry 2004, Kirby et al. 1999, Madden et al. 1997), and methamphetamine (Hoffman et al. 2006, 2008; Monterosso et al. 2007). One study examining marijuana-dependent individuals and controls did not find a significant difference, but it showed a trend toward greater discounting in dependent individuals; this effect, however, had a smaller effect size than those found in many of the previous studies with other drugs (Johnson et al. 2010). A recent meta-analysis of these categorical studies found evidence of a medium effect-size difference across studies (Cohen's $d = 0.58$) (MacKillop et al. 2011), which was moderated by clinical severity. Specifically,

significantly greater discounting (d = 0.61) was found in those studies using participants that met clinical criteria compared with those using nonclinical samples (d = 0.45). Moreover, smoking rates are robustly related to delay discounting (Alessi & Petry 2003; Cheng et al. 2012; Epstein et al. 2003; Johnson et al. 2007; MacKillop et al. 2010a; Ohmura et al. 2005; Reynolds 2004, 2006a; Sweitzer et al. 2008). In addition, increased discounting is a negative prognostic factor in treatment for nicotine dependence (MacKillop & Kahler 2009, Sheffer et al. 2012), stimulant dependence (Washio et al. 2011), and marijuana dependence (Stanger et al. 2012). In a series of longitudinal studies, a naturalistic index of discounting based on relative discretionary expenditures toward alcohol relative to savings predicts the natural resolution of alcohol misuse among middle-aged adults (Tucker et al. 2002, 2006, 2008). In addition to substance use, studies have also suggested that greater delay discounting is associated with important clinical problems associated with substance use, including HIV risk via injection equipment sharing among heroin abusers (Odum et al. 2000) and condom use among cocaine abusers (Johnson & Bruner 2012).

Complementing these findings is a large preclinical literature examining the role of discounting in the etiology and maintenance of addiction. This work has revealed the fundamental neurobiological circuits that subserve delay-discounting decision making (for a review, see Cardinal 2006). Furthermore, these studies demonstrate the independence of the neurobiological processes that underlie delay discounting from those underlying response-disinhibition measures of “impulsivity” (Winstanley et al. 2006). More importantly, these studies have found that individual differences in discounting are predictive of initiation and progression of drug self-administration in rodent models (Anker et al. 2009; Marusich & Bardo 2009; Perry et al. 2005, 2007). This finding is important because it establishes that high levels of discounting, prior to any drug exposure, set the stage for the development of addictive behavior. In addition, chronic exposure to addictive drugs significantly increases delay-discounting rates (Dallery & Locey 2005, Koffarnus & Woods 2013, Mendez et al. 2010, Roesch et al. 2007, Simon et al. 2007).

Complementing these findings are a number of longitudinal studies showing that greater delay discounting predicts the development of addictive behavior. In a study of early adolescents over a 5-year period, discounting significantly predicted increases in smoking over time (Audrain-McGovern et al. 2009a). Furthermore, discounting remained generally stable as a characteristic, and alternative path modeling did not support the opposite relationship, of greater smoking behavior leading to greater discounting. In a twin study, greater delay discounting predicted the development of substance use and other externalizing behaviors from ages 12 to 14 (Anokhin et al. 2011). Most recently, a 4-year longitudinal study examining determinants of alcohol misuse showed that working memory predicted the development of alcohol misuse, and this relationship was partially mediated by discounting (Khurana et al. 2013). Although more longitudinal studies are needed, these provide converging evidence that greater delay discounting plays a causal role in the development of addictive behavior. Together, the preclinical findings and longitudinal findings support the view of steep delay discounting as a recursive etiological factor in the development of addictive behavior, serving as both a predisposing risk process and a process further exacerbated by excessive substance use.

Factors that Dynamically Affect Demand and Discounting

The processes that give rise to elevated demand and discounting are typically not the focus of direct experimental examination. However, a small number of studies imposed experimental manipulations on these variables to understand how in vivo changes in both variables may take place. For example, environmental drug cues significantly increased demand for alcohol and tobacco (Acker & Mackillop 2013; MacKillop et al. 2010b, 2012b), suggesting that drug-related associative conditioning leads to dynamic increases in demand. Similarly, acute withdrawal from nicotine significantly increased tobacco demand (MacKillop et al. 2012b) and also made increased discounting even more precipitous among smokers and opiate-dependent individuals (Field et al. 2006, Giordano et al. 2002, Mitchell 2004, Yi & Landes 2012). In a preclinical study, a parallel relationship was reported in which morphine withdrawal increased demand for a substitute opiate (Wade-Galuska et al. 2011). These studies contextualize demand and discounting with two well-established processes in substance dependence: reactivity to drug-related environmental cues and withdrawal following the development of physiological dependence. As such, they suggest that associative conditioning and acute withdrawal have direct effects on these domains and, in turn, on choice behavior. Accordingly, these factors acutely affect substance use decision making via behavioral economic indices. Furthermore, these individual experiences are predicted to summate over time, partially contributing to the high levels of demand and discounting that are stably observed in affected individuals. This relatively incipient line of research holds promise for characterizing microlevel events that give rise to persistent dysregulation of decision making.

Environmental Factors: Availability of Alternative Reinforcers (Opportunity Cost)

The preceding domains are theorized to represent only one major class of influences, the motivational factors within the individual. However, because of its foundations in economics and behavioral psychology, a behavioral economics perspective gives equal weight to the larger environmental context within which addictive behavior takes place and, more specifically, the alternative reinforcers to which a person has access. As noted above, a behavioral economics perspective is a molar account of addiction, focusing on aggregates of behavior, not instances, and both the endogenous conditions of the organism and exogenous characteristics of their environment. Put simply, a person’s internal motivational state and the alternative reinforcers available in the environmental context are theorized to jointly determine the decision to use or not to use a drug. From this perspective, the yin and yang of addiction are high levels of endogenous factors (high demand and impulsive discounting) and low levels of exogenous factors (alternative reinforcers). Furthermore, a recursive etiological process is proposed to be operative for alternative reinforcers over the course of the development of a substance use disorder. For example, an individual may begin drinking recreationally and as part of a diverse repertoire of positively and negatively reinforcing behaviors. But, as use escalates and negative consequences mount (e.g., dissolution of a relationship, loss of a job, legal difficulties), the availability of these alternative reinforcers diminish, which commensurately increases the reinforcing value of alcohol (Rachlin 1997). This recursive etiological feedforward loop illustrates the classic vicious cycle of addiction (e.g., the “primrose path” model of addiction formulated by Herrnstein & Prelec 1992).

In terms of empirical research, considerable experimental and descriptive evidence supports the importance of alternative reinforcers (Cosgrove & Carroll 2003, Cosgrove et al. 2002). In preclinical animal models, the highest rates of substance use are present in contexts with the least availability of substance-free reinforcements, such a palatable food, exercise, enriched housing, and social access (Alexander et al. 1978, Carroll et al. 2009, Cosgrove & Carroll 2003, Cosgrove et al. 2002, Hadaway et al. 1979). Similar results have been found in human laboratory and clinical studies (e.g., Bickel et al. 1995, Vuchinich & Tucker 1988). Recent functional magnetic resonance imaging (fMRI) studies also indicate that chronic substance abusers report diminished neural activation to nondrug rewards (Lubman et al. 2009). Using measures that characterize the proportionate reinforcement received from a variety of sources, substance misusers report less reinforcement from nondrug activities compared with control participants (Correia et al. 1998, 2003; Van Etten et al. 1998). In continuous relationships, alcohol-free reinforcement is significantly negatively associated with alcohol consumption and alcohol-related problems (Murphy et al. 2005). In one longitudinal study, the presence of alternative reinforcers negatively predicted the development of smoking over a four-year period (participants ages 18–22) (Audrain-McGovern et al. 2011). Furthermore, an indirect relationship was evident, such that higher levels of baseline depression predicted reduced alternative reinforcement over time and, as a result, increases in smoking. Another longitudinal study with heroin-dependent adults found that subjective valence ratings of pleasant (drug-free) pictures negatively predicted future heroin use after controlling for baseline craving and heroin use (Lubman et al. 2009).

Clinical studies also point to the importance of alternative reinforcers (Higgins et al. 2004). For example, among young adult smokers, those who increased substitute reinforcers across treatment had approximately double the rate of treatment success (Audrain-McGovern et al. 2009b). Similarly, proportionate alcohol-free reinforcement has been associated with poorer response to a brief alcohol intervention in young adults (Murphy et al. 2005) and young adult drinkers who were asked to increase engagement in exercise and creative activities spontaneously reduced their drinking compared with control participants (Correia et al. 2005). Successful treatment is associated with the development of more sources of alternative reinforcement (Rogers et al. 2008). More broadly, several of the behavioral economic treatments operate by enhancing alternative reinforcers (discussed below) (Murphy et al. 2012).

Neurobiological Substrates

Dramatic advances have taken place in our understanding of the neurobiology of addictive behavior, including the role of genetic factors in liability for addictive disorders. Although a comprehensive review is beyond the scope of the current article, this section briefly summarizes the literature through the lens of behavioral economics. In its distilled form, the neurobiology of addictive behavior is theorized to reflect the result of functional and structural changes to the brain resulting from long-lasting neuroadaptations that are induced by chronic exposure. Three key neural regions and circuits are disrupted by this process, including the prefrontal cortex, which is responsible for diverse forms of executive function such as decision making; subcortical motivational circuits, which include structures such as ventral striatum and nucleus accumbens; and what are referred to as “antireward” circuits,

which include the stress systems that subserve the acute and protracted aversive states that follow from intoxication into acute and protracted withdrawal (for reviews, see Kalivas & Volkow 2005, Koob & Le Moal 2008, Volkow et al. 2012). Together, these changes confer distinct vulnerabilities for continued addictive behavior, including altered decision making, powerful motivational drives, reduced sensitivity to substance-free rewards, and a semichronic aversive state (Kalivas & Volkow 2005, Koob & Le Moal 2008, Lubman et al. 2009, Volkow et al. 2012).

These neuroadaptive changes are highly compatible with a behavioral economic perspective. In the context of drug demand, exaggerated drive for a drug is an obvious substrate for persistently high incentive value. However, attenuated executive functioning would also be expected to increase demand by reducing cognitive capacity to fully consider the associated costs, as would a protracted aversive state, which would putatively drive up the value of the drug on the basis of its negatively reinforcing capacity to alleviate that state. Reduced sensitivity to drug-free rewards would similarly increase the demand for substances. With regard to impulsive discounting, the prefrontal and subcortical circuits are largely those that have been implicated with discounting decision making in fMRI research (Bickel et al. 2009, McClure et al. 2004), suggesting that commensurate impairment in discounting may be a behavioral manifestation of these neuroadaptive changes. With regard to the antireward system, the role of stress in discounting has been only modestly studied, and results are mixed (Diller et al. 2011, Fields et al. 2009, Torregrossa et al. 2012, White et al. 2008). However, purely from conjecture, given the laboratory evidence on withdrawal effects discussed above, the chronic augmentation of the antireward system may be a further plausible mechanism for increasing discounting. Finally, from the perspective of alternative reinforcers, diminished executive functioning should reduce the probability a person would investigate, plan for, and engage in new activities. Considered in totality, neuroscience and behavioral economics can largely be seen as highly compatible, and the disconnect between them is simply a side effect of their occupying different scientific levels of analysis.

Similarly, in behavioral genetics, major advances have established the substantial role of genetic factors in the liability for addictive disorders (for reviews, see Goldman et al. 2005, Kendler et al. 2012, MacKillop et al. 2010c). However, substantial evidence suggests that there is no single “addiction gene” or small number of critical variants. Rather, the current evidence suggests that liability is conferred via many genetic loci, each exerting a small effect. Where consistent evidence is available, it pertains to genes responsible for a drug’s pharmacokinetics (metabolic processing) (e.g., Luczak et al. 2006) and pharmacodynamics (CNS actions) (e.g., Bierut 2010, MacKillop et al. 2010c).

Where behavioral genetics and behavioral economics may be reciprocally informative is in using behavioral-economic characteristics as intermediate phenotypes or endophenotypes (Gottesman & Gould 2003). These terms refer either to characteristics that are at least mechanistically informative in terms of genetic relationships (intermediate phenotypes) or to characteristics that meet a number of criteria demonstrating independence and links to both genetic variables and clinical phenotypes (endophenotypes). Both classes of variables are currently receiving a great deal of interest because, relative to clinical phenotypes, they offer more stable, focal, reliable phenotypes that are, putatively, more closely related to genetic

variation. As such, associations with specific variants are expected to be larger in magnitude, more reliable, and more interpretable from a mechanistic standpoint. Thus, accumulating evidence suggests that greater delay discounting may be a useful endophenotype (MacKillop 2013). Specifically, evidence supports the notion that variation in discounting is heritable (Anokhin et al. 2011, Huskinson et al. 2012), reliable (Beck & Triplett 2009, Ohmura et al. 2006, Takahashi et al. 2007), associated with the level of familial addictive behavior (Acheson et al. 2011), and linked to specific polymorphisms (Boettiger et al. 2007, Eisenberg et al. 2007, Paloyelis et al. 2010), albeit with some inconsistency in the latter findings. Furthermore, indices of demand or other behavioral economic indices may also be useful for clarifying genetic influences on addictive behavior.

THE REPAIR OF REINFORCER PATHOLOGIES

Constraint of Unhealthy Choice

In the preceding sections, we have outlined the behavioral economic processes associated with reinforcement pathology. We have also discussed how they may function in the development and maintenance of substance use disorders. In the following sections, we examine the evidence and the implications of behavioral economics for the repair of reinforcement pathologies.

Direct environmental constraints: reducing the availability and increasing the price of unhealthy choice commodities—As noted above, behavioral economics provides a molar account of behavior in that it relates aggregates of behavior (e.g., patterns of substance use and substance-free activities over time) to aggregates of reinforcement or price contingencies as measured over some interval (Tucker et al. 2002, Vuchinich 1995). This perspective does not attempt to predict the outcome of a decision to use (or not to use) drugs on a particular day or moment, which would be the goal of a molecular account of behavior (Vuchinich & Heather 2003). As predicted by the law of demand and described above, consumption of most commodities, including psychoactive substances, is inversely related to price or response requirement (Bickel et al. 2012a, DeGrandpre & Bickel 1996, Higgins et al. 2004, Hursh & Roma 2013, MacKillop et al. 2012a, Murphy & MacKillop 2006). As also discussed above, price is defined broadly and may include the monetary cost of purchasing a drug as well as time or effort costs associated with obtaining a drug (e.g., operant response requirement or, in a natural environment, the time/effort required to purchase a drug). Although difficult to quantify, the potential legal and health costs as well as the negative impact of substance use on access to other rewards are also “costs” that influence an individual’s substance use decisions. Increases in any of these costs can decrease substance use, and conversely, decreases in these costs can increase substance use (Hursh & Roma 2013). These observations are consistent with the broader econometric studies comparing the relationships between differing tax levels and licit drug consumption (Chaloupka et al. 2012, Wagenaar et al. 2010).

At a behavioral level, the impact of both price and lost opportunity cost is illustrated in Figure 4, which plots hypothetical drink purchases as a function of price and next-day responsibility (Skidmore & Murphy 2011). The sample included 207 college-student binge drinkers who were instructed to make drink purchases across a range of prices and preceding

three responsibility conditions for the next day: no class, morning class without a test, and morning class with a test. Consumption was reduced markedly as a function of both price and the availability of a competing next-day alternative reinforcer that would presumably be negatively impacted by heavy drinking (see also Gentile et al. 2012). The results of this study are consistent with behavioral economics theory and suggest that substance abuse prevention and intervention approaches are most likely to be successful when they attempt to increase direct constraints on substance use while simultaneously increasing the availability of particular classes of alternative reinforcers that are incompatible with substance use.

Moreover, a variety of effective public-health-level prevention approaches are consistent with behavioral economics research (Hursh & Roma 2013). These include enforcing age limits on cigarette and alcohol purchases, maintaining or increasing taxes and/or legal sanctions (related to alcohol/tobacco or illicit substance use, respectively), reducing the density of outlets that sell regulated substances (alcohol, tobacco, and, in some areas, marijuana), carefully regulating the prescribing practices related to commonly abused prescription medications (opioids, benzodiazepines, stimulants), and enhancing the availability of prosocial activities that may increase the opportunity costs of substance abuse (e.g., access to education, employment, recreational activities) (DeFulio & Silverman 2011, Gentile et al. 2012, Weitzman & Kawachi 2000). Interventions that attempt to increase constraints on drug choices are discussed next.

Contingency management as constraint—Research on the relationship between price and consumption has been successfully translated into a number of effective intervention strategies: Contingency management (CM) may be the most direct exemplar. CM approaches attempt to change the user's environment such that (*a*) substance use and abstinence are readily detected, (*b*) abstinence is readily reinforced, (*c*) substance use results in a loss of reinforcement, and (*d*) the density of reinforcement derived from substance-free sources is increased to compete with the reinforcing effects of substance use (Higgins et al. 1991). Thus, one of the active mechanisms of CM is the increased costs (loss of reinforcement) associated with the detection of substance use. CM protocols typically strive to achieve continuous abstinence through the use of escalating schedules of reinforcement. For example, a substance user on an escalating CM schedule may receive a voucher worth $5 for day one of verified abstinence, but that amount would increase with each consecutive day of abstinence such that after seven days a single episode of substance use may "cost" the user $50 in addition to the actual cost of the substance. Thus, the opportunity cost of substance use, as defined above, is increased over time. Importantly, because substance abusers generally undervalue delayed outcomes (i.e., show steep delay discounting), effective CM approaches require that drug abstinence be objectively verified frequently so that the "cost" of substance use (lost voucher) is incurred soon after use.

CM procedures have been effectively applied to reduce the use of a number of substances (Hartzler et al. 2012, Stitzer et al. 2007), including cigarettes (Alessi et al. 2004, Yi et al. 2008), alcohol (Alessi & Petry 2013, Koffarnus et al. 2011, Petry et al. 2000), cocaine (Higgins et al. 1993, Petry et al. 2013), marijuana (Carroll et al. 2012), and opiates (Bickel & Marsch 1999, Bickel et al. 1999a, DeFulio & Silverman 2011, Silverman et al. 1996). CM

treatment is also associated with increases in quality of life in substance abusers (Petry et al. 2007), and cost-effectiveness studies suggest that these interventions result in a net economic gain owing to their relative efficacy and the expense associated with treatment failures in substance-abusing populations (e.g., inpatient treatment, incarceration, increased health care utilization) (Olmstead et al. 2012, Sindelar et al. 2007). CM interventions have also been modified and are efficacious when (*a*) relatively low-value vouchers are delivered probabilistically, thereby reducing the total voucher costs incurred by treatment facilities (Olmstead & Petry 2009), and (*b*) when access to paid employment, rather than vouchers for goods or services, is used to reinforce abstinence (DeFulio & Silverman 2011). Both of these adaptations have the potential to increase the dissemination of CM.

Other Behavioral Approaches to Reduce Consumption by Increasing Constraints on Drug Choice

Several other substance abuse intervention approaches may work in part by increasing constraints on substance use, either by reducing the availability of a substance or increasing the real or perceived cost of alcohol or other substance use. The community reinforcement approach (CRA) focuses on assisting individuals to develop alternative reinforcement that is mutually incompatible with substance use. CRA has been used to treat clients who abuse alcohol (Smith et al. 1998), marijuana (Diamond et al. 2002), cocaine (Carroll et al. 1994), opiates (Bickel et al. 1997), and cigarettes (Roozen et al. 2006). Behavioral couples therapy (BCT) also attempts to increase the cost of substance use though the use of daily sobriety contracts, through ingestion of disulfiram (when indicated), and by encouraging significant others to withhold reinforcement if the substance-abusing partner drinks or uses other substances. A recent meta-analysis in the treatment of alcohol and other substance use disorders found that BCT was superior to a variety of control treatments, including cognitive behavioral therapy that did not include a significant other (Powers et al. 2008).

Brief motivational interventions (BMIs) have emerged as an effective strategy for reducing substance misuse among a variety of populations (Miller & Rollnick 2012). Although BMIs were not originally developed from behavioral economics theories, they attempt to increase an individual's motivation to change in part by fostering an individual's awareness of the costs and consequences of substance use. This is often accomplished via a discussion of the pros and cons of substance use (i.e., decisional balance exercise) as well as personalized feedback that highlights tangible costs (e.g., money spent on alcohol and other substances, calories and weight gain related to drinking, estimated life years lost from cigarette smoking). All these strategies encourage clients to consider the total costs of substance use, relative to the overall benefits, and are designed to motivate self-regulation and more informed decision making (Miller & Rollnick 2012). Another feature of BMIs that is consistent with, although not explicitly derived from, behavioral economics theory is that the outcomes of temporally extended patterns of choices related to substance use are aggregated into meaningful units such as substance use occasions per week, substance-related problems experienced over time, or money spent on substances over the course of a year.

From a behavioral economics perspective, removing drugs and associated paraphernalia (e.g., pipes, needles, ashtrays, etc.) from the home not only removes powerful triggers, which can increase craving and demand for substances (MacKillop et al. 2012b, MacKillop et al. 2010b), but also increases the effort costs associated with obtaining new drugs. Various forms of substance-use monitoring (e.g., by clinicians, parents, or significant others) via the increasing availability of low-cost point-of-contact drug-testing kits can also increase the costs of substance use. Parental monitoring is a particularly effective element of substance abuse treatment for adolescents (Komro et al. 2001).

Finally, although a full review is beyond the current scope of this article, a number of medications are FDA approved for the treatment of substance use disorders (e.g., O'Brien 1997), including pharmacotherapies for treating nicotine dependence, opiate use disorders, and alcohol use disorders. These treatment modalities are highly consistent with a behavioral economics perspective. The most common pharmacotherapy strategy is direct or indirect alleviation of withdrawal (e.g., nicotine patch/gum/lozenge/spray, methadone, buprenorphine), which can be thought of as a strategy for reducing demand for a particular drug. These medications may also be characterized as behavioral economics substitutes for the drug being abused. Alternatively, disulfiram for AUDs also putatively reduces demand, but it does so by directly imposing a response cost via the sickness that results when consumed with alcohol. Naltrexone pharmacotherapy for AUDs is somewhat distinct in that it putatively attenuates craving, blunts alcohol's positively reinforcing stimulant effects, and potentiates its unpleasant sedative effects (for a review, see Ray et al. 2010). As such, in behavioral economics terms, it reduces demand via multifarious positive and negative reinforcement mechanisms.

Although relatively little research has focused on this area, there is clear conceptual overlap between known pharmacotherapy mechanisms and behavioral economics mechanisms as well as a small amount of empirical data. One recent study illustrates the promise of these intersecting lines of research. In a sample of Asian Americans, Bujarski et al. (2012) investigated the effects of naltrexone on alcohol demand, both in general and following intravenous alcohol administration to raise participants' blood alcohol level to 0.06 g/dl. In addition, on the basis of evidence that possession of the G allele of a single nucleotide polymorphism in the *OPRM1* gene (rs1799971) is associated with greater sensitivity to alcohol's subjective effects (Ray & Hutchison 2004, 2007) and naltrexone's clinical effects (Anton et al. 2008, Kim et al. 2009, Oslin et al. 2003), participants were genotyped for this locus. In Bujarski et al. (2012), compared with a placebo, naltrexone significantly reduced intensity of demand, O_{max}, and breakpoint, thus supporting the hypothesis that one of its mechanisms is the attenuation of the incentive value of alcohol across the demand curve. In addition, possession of the high-alcohol-sensitivity G allele was also associated with greater intensity of demand, suggesting that behavioral economics indices may be promising intermediate phenotypes. Although this study had a relatively small sample size and was exploratory in nature, it nonetheless illustrates the potential of integrating behavioral economics into addiction pharmacotherapy and pharmacogenetic research.

Reduced Constraint of Healthy Choice

Restructuring environments to enhance access to healthy choices—As noted above, experimental studies have shown that high rates of substance use most likely occur in contexts devoid of substance-free sources of reinforcement (Alexander et al. 1978, Carroll et al. 2009, Hadaway et al. 1979), that substance abuse is associated with diminished dopamine response to naturally occurring substance-free rewards (Koob 2006, Volkow et al. 2003), and that substance use will generally decrease if access to alternative reinforcers is increased (Higgins et al. 2004). These basic research findings have led to a variety of interventions that seek to increase the availability of healthy alternatives to substance use. These approaches vary with respect to the intensity of the intervention; that is, chronic substance abusers may require more step-by-step assistance in accessing drug-free rewards, whereas the enhancement of substance-free alternatives via brief interventions are viable in less-severe populations.

Community reinforcement, contingency management, and behavioral couples therapy: approaches enhancing access to substance-free reinforcement contingent on abstinence—Many of the aforementioned intervention approaches that attempt to increase constraints on substance use also seek to increase the availability of drug-free sources of reinforcement. This is a central element of CRA and CM approaches, which include counseling designed to improve employment prospects, marital and family relationships, and nondrinking social and recreational interactions. Vouchers earned from abstinence can be exchanged for a variety of items that are designed to increase substance-free reinforcement, including materials related to hobbies/recreational activities, sporting equipment, tickets to cultural/entertainment events, and educational supplies or tuition. A meta-analytic review concluded that CRA is more effective than usual care and that the addition of incentives for abstinence leads to improved outcomes (Roozen et al. 2003). The Therapeutic Workplace, developed by Silverman and colleagues (Silverman et al. 2001, 2002) is another example of a CM treatment approach that directly targets engagement in drug-free activities. In one series of studies, substance-abusing pregnant and postpartum women were able to earn a livable wage by working and/or acquiring job skills related to data entry. Access to work was made contingent on providing drug-free urine samples collected three times per week. Participants who provided the drug-free urine samples and completed a 3-h work shift received a payment voucher. A 3-year outcome study demonstrated the effectiveness of the Therapeutic Workplace with pregnant and postpartum women (Silverman et al. 2002). Subsequent research suggests that long-term Therapeutic Workplace effects may dissipate following termination of the program (DeFulio & Silverman 2011). This research suggests the importance of transitioning chronic substance abusers to stable employment settings that require regular drug tests and have consequences that are proportional to use severity (e.g., not a catastrophic consequence such as termination from employment for a single positive drug test) (e.g., Kleiman 2009).

A study with opioid-dependent patients demonstrated that CM can also be effective when vouchers are used to directly reinforce engagement in substance-free activities that are consistent with treatment goals (Iguchi et al. 1997). Participants were randomly assigned to one of three treatment conditions: urinalysis-based reinforcement (UA), reinforcement that

was conditional on treatment-plan success (TP), and standard care. Participants in the UA and TP conditions could earn vouchers, described as treatment-assistance coupons, redeemable for expenses linked to a treatment plan (e.g., clothing for job interviews). Participants in the UA condition earned vouchers for opioid-negative urine specimens. Vouchers were awarded to TP participants for meeting objectively defined, and clearly verifiable, treatment-plan tasks. For example, vouchers could be earned for completing job applications or training. In each case, treatment-plan tasks were individually tailored for each client, and vouchers were used to shape behavior toward long-range treatment goals. Even though only UA participants were directly reinforced for abstaining, TP participants were more than twice as likely to produce negative urine specimens, highlighting the importance of reinforcing drug-free alternatives. These results support the notion that clients who are brought into contact with drug-free reinforcers occurring naturally in their environment are likely to maintain behavioral changes after removal of the CM contingencies (Higgins et al. 2004).

Similarly, brief behavioral-activation approaches, designed to reduce depression by increasing engagement in social and goal-directed activities, are effective as a supplemental or stand-alone intervention for substance use (Daughters et al. 2010, Reynolds et al. 2010). Finally, BCT explicitly seeks to involve the couple in rewarding drug-free activities and to increase the rate of reinforcement derived from behaviors aimed at improving the couple's day-to-day interactions (Fals-Stewart et al. 2004, 2005; Powers et al. 2008).

Direct behavioral economics enhancement of a brief motivational intervention for alcohol misuse in young adults—As noted above, BMIs target nontreatment-seeking individuals and employ motivational interviewing to enhance motivation for change. These interventions are more efficacious than no- or minimal-treatment control conditions, and their effect sizes are generally small to moderate (Lundahl et al. 2010, Miller & Rollnick 2012). However, individuals who have few alternatives to drinking or elevated demand or impulsivity may not respond to standard BMIs (Ewing et al. 2009; MacKillop & Murphy 2007; Murphy et al. 2005, 2012; Stein et al. 2011). Importantly, BMIs have recently been modified to take into account behavioral economics principles via the addition of substance-free activity sessions (SFASs) (Murphy et al. 2012). This represents a novel approach of directly importing behavioral economics concepts into treatments that come from entirely distinct perspectives.

SFASs, which target substance-free activities and temporal discounting, were initially developed for heavy-drinking college students and targets increasing engagement in academic and other constructive campus/community activities that are associated with delayed rewards and generally negatively associated with drinking (Murphy et al. 2005). A single 50-min SFAS follows a standard alcohol-focused BMI session and encourages students to identify their college and career goals. Personalized feedback and information is then provided (*a*) to enhance motivation to pursue the stated goals by highlighting the value of the associated delayed reward (e.g., a discussion of the students reasons for pursuing a particular major or career, feedback on the future salary implications of obtaining a college degree and the income implications of college grade-point average) and (*b*) to provide a specific, personally tailored roadmap on how to achieve future goals (e.g., information on

specific educational and experiential requirements for gaining entry into a given profession, including grade-point-average requirements, and specific campus and local internships and organizations that provide relevant experiences). SFASs include personalized feedback on current patterns of time allocation across a variety of activity categories that may be relevant to important goals and values (e.g., time spent studying, drinking, exercising, volunteering). Thus, the overarching goal of SFASs is to increase the salience of delayed outcomes and the extent to which the behavior leading to those rewards or punishers is viewed as part of a coherent molar pattern (rather than an isolated choice) to reduce impulsive response patterns (Hofmeyr et al. 2011, Siegel & Rachlin 1995).

A randomized controlled trial indicated that a two-session (alcohol BMI + SFAS) intervention resulted in larger reductions in alcohol problems ($d_b = 0.52$, $p = 0.01$) (see Figure 5) relative to a two-session (alcohol BMI + relaxation) active control condition in a sample of 82 binge-drinking college students. Moderation analyses indicated that BMI + SFAS was also associated with significantly greater reductions in heavy drinking among those participants reporting at baseline low levels of substance-free reinforcement or high levels of depression. The focus on increasing goal-directed substance-free activities may have been beneficial to these participants. Importantly, the SFASs increased relevant mechanisms such as future time orientation, self-regulation, and participation in evening studying/academic activities (Murphy et al. 2012).

Improved Impulse Control and Executive Function

As discussed above, substance use disorders are associated with decreased executive functioning and decreased impulse control. Therefore, clinical researchers have been interested in training procedures to improve executive function or impulse control as a way of treating the underlying decision-making patterns associated with impulse-control disorders including substance abuse. Various procedures have been developed to change an individual's discount rate (for a review, see Koffarnus et al. 2013). However, most of these manipulations alter discount rate only temporarily, often only in the presence of some impermanent stimulus. Manipulations that influence human intertemporal choice behavior in a more lasting way include fading procedures, behavioral contracts, and pharmacological interventions.

Fading procedures—Fading procedures can increase an individual's choice of larger, delayed rewards, thereby decreasing discount rate. First observed in laboratory animals (Mazur & Logue 1978), these procedures often start with a choice between two reinforcers of different sizes that are both available after the same delay. After choice of the larger reinforcer is established, delay to the smaller reinforcer is gradually decreased with an adjustment schedule that maintains choice of the larger, more delayed reinforcer. Over time, subjects learn to choose delayed reinforcers under conditions where they previously chose only the smaller-sooner option. This approach has been adapted for use in human populations, primarily with children and adults with developmental disabilities or traumatic brain injury (for a review, see Odum 2011). To date, there are no published reports assessing whether fading procedures can effectively increase self-control in substance-dependent

individuals or populations vulnerable for developing substance dependence, although this may be a promising approach given the low self-control demonstrated in these groups.

Contracts including deposit contracts—In the context of substance abuse treatment, a contract typically refers to an agreement made with a counselor, friends, family, and/or oneself to avoid substance use by arranging an undesirable consequence should substance use occur. For example, the substance user may supply a relatively large up-front sum of money to be held by a third party, which is forfeited should substance use occur. Such a deposit contract is effective at controlling behaviors involving self-control including substance use and diet and exercise (Bowers et al. 1987; Dallery et al. 2008; Elliott & Tighe 1968; John et al. 2011; Lando 1977; Paxton 1980, 1981, 1983; Singh & Leung 1988; Volpp et al. 2008; Winett 1973). As discussed above, the average substance-dependent individual has an elevated discount rate, reducing the likelihood of self-controlled choices. Owing to the hyperbolic form of discounting curves, however, people with an elevated discount rate are still likely to make self-controlled choices when faced with two options that are both significantly delayed. A contract works as a precommitment strategy (Rachlin & Green 1972) by moving the decision point for engaging in substance use from the time that drugs are present and available for ingestion to a point in time days or weeks prior to ingestion. As a result, people avoid the preference reversal that often leads substance abusers to choose the immediate reward of substance use over the delayed rewards associated with abstinence. By agreeing ahead of time to forfeit a large sum of money or to experience another negative consequence after substance use, the substance user changes the consequences when drugs are available for use. Instead of the typical choice between engaging in substance use and possibly experiencing delayed negative consequences, the substance user is faced with the choice between engaging in substance use and definitely experiencing relatively immediate negative financial or other agreed-upon consequences.

Pharmacological approaches to impulse control—The drugs most commonly researched and used clinically to improve self-control are the stimulants amphetamine and methylphenidate. These drugs are primarily used to treat attention deficit hyperactivity disorder (ADHD), which is characterized by elevated discount rate (Schweitzer & Sulzer-Azaroff 1995; Solanto et al. 2001; Sonuga-Barke et al. 1992, 1996). In healthy controls, acute amphetamine decreases discount rate (de Wit et al. 2002). However, likely owing to their high abuse liability, these drugs are not typically used to treat self-control in substance abuse (Jasinski et al. 2008, Kollins et al. 2001). As an alternative, the nonstimulant drug atomoxetine is FDA approved for the treatment of ADHD and increases self-control in animal models (Robinson et al. 2008, Sun et al. 2012). Although such efficacy has not been evaluated in humans, should atomoxetine's effect extend to this population, it has some potential as a self-control treatment because it has very low abuse liability in people with and without substance dependence (Heil et al. 2002, Jasinski et al. 2008).

Executive Function Training

Some evidence suggests that self-control and intertemporal choice behavior are associated with an individual's executive function capacity (Bobova et al. 2009, Shamosh et al. 2008). As a result, some researchers have become interested in manipulating discount rate with

interventions that affect executive functioning. Behavioral executive function training regimens have shown utility in improving the executive functioning and associated behaviors of groups with deficits in this area, such as individuals with schizophrenia (Krabbendam & Aleman 2003, Twamley et al. 2003).

Two experiments have assessed the effects of working-memory training in substance users. In one study, in-treatment stimulant-dependent adults were divided into two groups, and a computerized working-memory training regimen with known efficacy was compared with a similar control training regimen wherein participants were supplied with correct answers in the tasks to prevent them from exercising their working memory (Bickel et al. 2011c). In this study, discount rate was substantially reduced in the active group. Another study examined working-memory training in two groups of problem drinkers (Houben et al. 2011). Active working-memory training requiring recall of sequences of visual stimuli that progressively increased in length was compared with a control condition wherein static three-stimuli sequences were recalled. Compared with the control group, participants in the active condition showed increased working-memory capacity, and as a result of this intervention, they reported less alcohol use immediately after training and at a 1-month follow-up. Together, these experiments suggest that working-memory training may be an effective way of treating substance use, perhaps by modifying discount rate. However, this hypothesis remains speculative without further study.

CONCLUSIONS

The problems of substance dependence have largely remained the same. However, the scientific tools, both technological and theoretical, we use to investigate and understand this disorder change and evolve over time (Bickel et al. 2013). These new tools allow us to see dependence disorders in a different light and allow new questions to be addressed. Behavioral economics, in our view, should be properly considered a new theoretical tool, as it has provided new conceptual categories, independent variables, and dependent measures as well as an ever-growing array of data that suggests its insights are robust and fruitful. These tools were initially adapted from behavioral theory and microeconomics and applied in both animal and human laboratories. Over time, the behavioral economics of substance use disorders has been applied to more molecular analyses involving genetics as well as neuroimaging and the important molar issue of therapeutic applications to addiction. As a result, a new conceptual model of what may be an important feature underlying the phenomena of substance use disorders has developed. This model is what we refer to as reinforcer pathologies, that is, the persistent high valuation of a substance of dependence and the excessive discounting of delayed rewards. Much of the data reviewed above support the role of these processes. Substantial evidence suggests that these two factors are related to substance dependence, are predictive of treatment outcomes, and can rightfully be the target of clinical intervention.

One clear future direction is to better understand the interaction of these two components of reinforcer pathologies at any level of analysis. Presumably, a group either of adolescents at risk for substance dependence in a preventive model or of currently substance-dependent individuals entering treatment could be divided into a two-by-two matrix crossing

individuals ranking high and low on measures of drug demand and temporal discounting (see Table 1). We predict, but at this time do not have data to test our hypothesis, that the lower demand and lower discounting group and higher demand and higher discounting groups would have the least and greatest likelihood of moving into substance dependence and show the greatest and least response to treatment, respectively. Such evidence would directly stimulate the development of specific treatment components that could be used to address these challenges and perhaps recognize a functional but nuanced difference among individuals with substance dependence. Such evidence and recognition may permit individuals to be matched to treatment on the basis of the functional phenotype they exhibit. For example, in a study described above, heavy-drinking young adults with low levels of substance-free reinforcement did better in a treatment that specifically addressed this deficit (Murphy et al. 2012). The development of treatments that selectively attenuate excessive valuation and discounting are still in the early stages, but progress has been made particularly in improving excessive temporal discounting.

Of equal importance is the development of behavioral economics procedures for use in treatments for substance dependence. As reviewed above, a variety of efforts have been and are being developed to integrate behavioral economics concepts into existing comprehensive treatments. These examples show that this conceptual model provides different theoretically driven options for treatment development. In particular, some of these approaches, such as emphasizing behavioral economics variables during brief motivational interviewing and the role of implicit or explicit incentives to increase the effort or the opportunity cost of continued substance use, hold promise of being developed at scale.

In sum, we present behavioral economics as a theoretical perspective that allows researchers to see substance dependence in a new light---a view of reinforcer pathologies that has pragmatic value in both understanding the disorder and approaching treatment. Furthermore, behavioral economics provides a suite of methods and tools to examine addictive behavior in systematic, precise, and novel ways. Despite considerable progress, the opportunities ahead in exploring the space made available by this approach abound and continue to have great potential in improving our understanding of addictive behavior.

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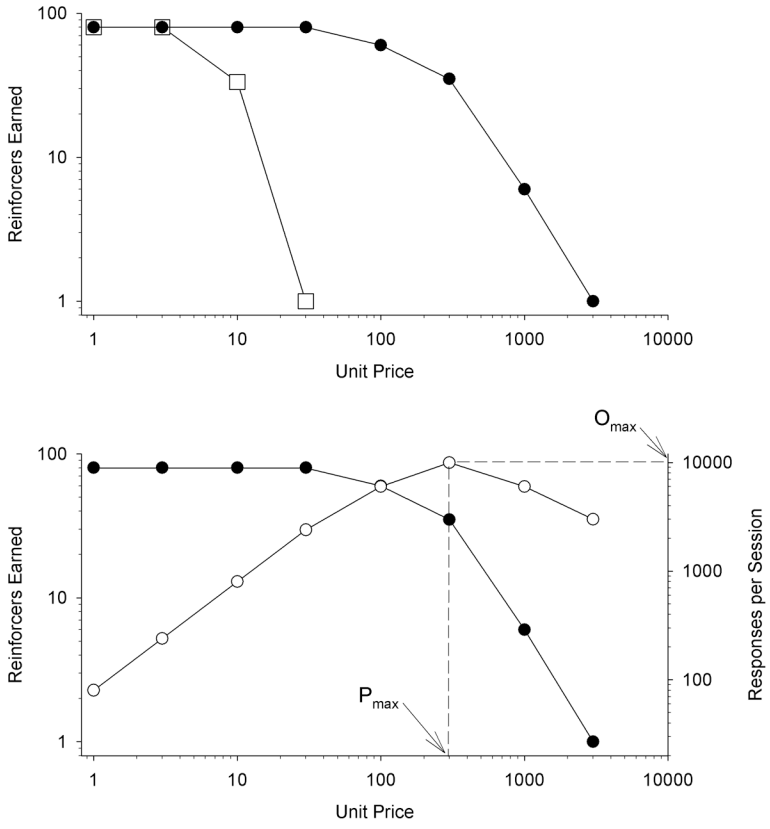

Figure 1.

Panel a shows demand curves for two hypothetical reinforcers. Although both reinforcers show the same intensity of demand (level of consumption when the commodity is available at very low prices), the reinforcer represented by open red squares shows higher elasticity (sensitivity to price) compared with the reinforcer represented by closed blue circles. *Panel b* shows demand curve (*closed blue circles; left* y-*axis*) and corresponding response output curve (*open red circles; right* y-*axis*). Also shown are the price at which maximal responding occurs (P_{max}) and the corresponding maximal response output (O_{max}).

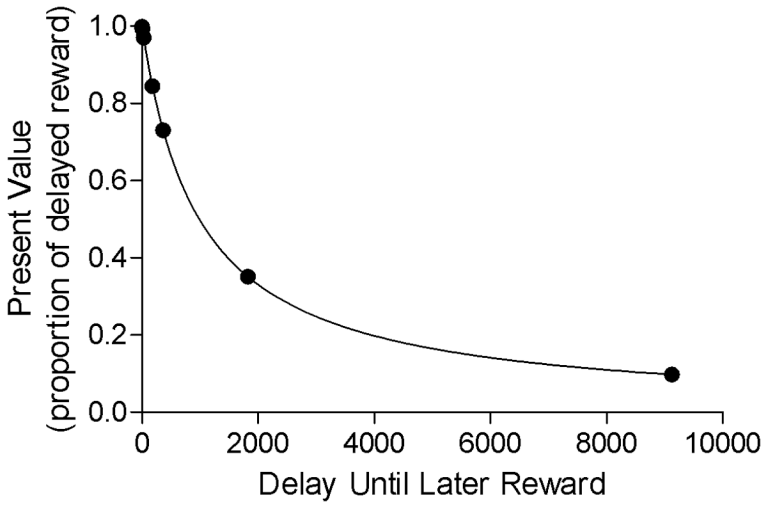

Figure 2.
Hypothetical delay-discounting function. Reward value decreases in a hyperbolic fashion as delay until receipt of the reward increases.

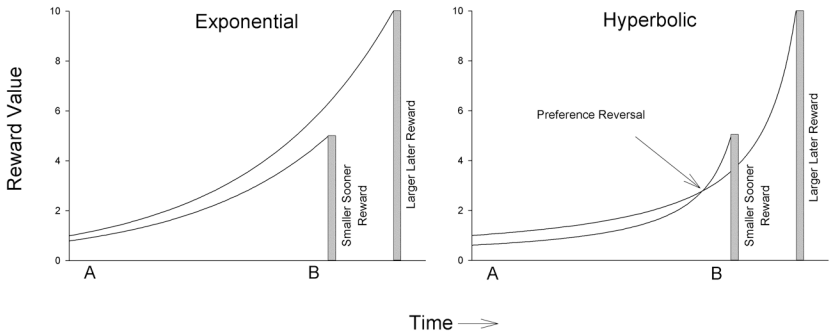

Figure 3.

Panel a shows that assuming exponential discounting, relative reward (preference) between a smaller-sooner (*blue*) and larger-later (*red*) reward remains constant through time (e.g., at both Time A and Time B). *Panel b* shows that assuming hyperbolic discounting, preference reverses from a larger-later (*red*) reward (at Time A) to a smaller-sooner (*blue*) reward (Time B).

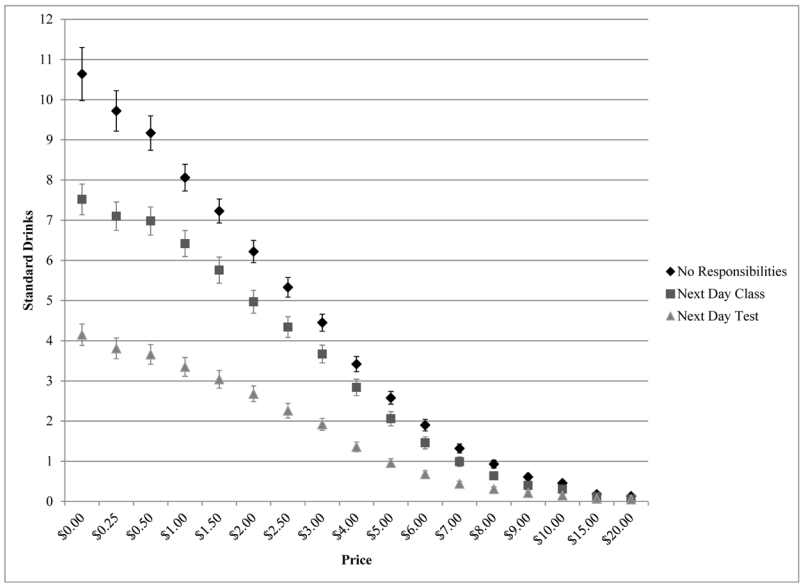

Figure 4.

Mean (±1 standard error of the mean) hypothetical consumption values across 17 different prices for alcohol purchases associated with three next-day responsibility conditions: (*diamonds*) no next-day responsibilities, (*squares*) next-day class, and (*triangles*) next-day test. From Skidmore JR, Murphy JG. 2011. The effect of drink price and next-day responsibilities on college student drinking: a behavioral economic analysis. *Psychol. Addict. Behav.* 25:57–68. Reprinted with permission from the American Psychological Association.

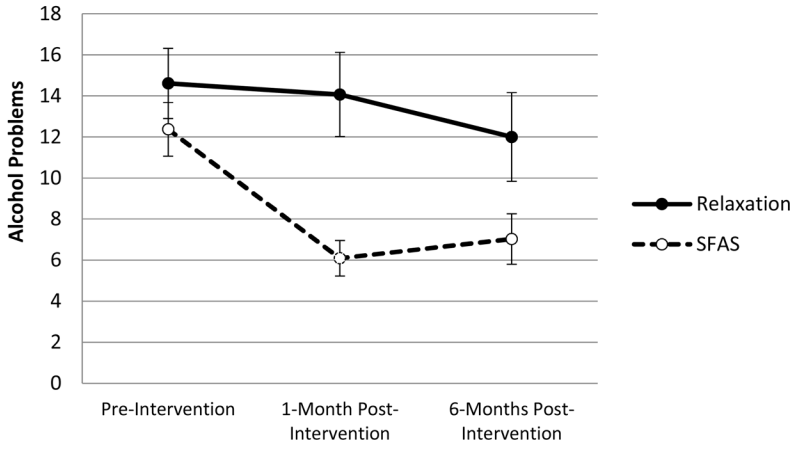

Figure 5.

Changes in past-month number of alcohol problems from baseline to follow-up by intervention condition. Error bars reflect ±1 standard error of the mean. Abbreviation: SFAS, substance-free activity session. From Murphy JG, Dennhardt AA, Skidmore JR, Borsari B, Barnett NP, et al. 2012. A randomized controlled trial of a behavioral economic supplement to brief motivational interventions for college drinking. *J. Consult. Clin. Psychol.* 80:876–86. Reprinted with permission from the American Psychological Association.

Table 1

Potential phenotypes of the two processes that define reinforcer pathology

	Low demand	**High demand**
Limited discounting	No pathology	No pathology
Excessive discounting	No pathology	Reinforcer pathology