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Publication Date

2003

IS THE ADDICTION CONCEPT USEFUL FOR DRUG POLICY?

Robert MacCoun *

The development of behavioral economics, with its prospect of integrating insights from economics and psychology, is surely one of the most exciting intellectual developments in the social and behavioral sciences in the past 20 years. And if any domain could benefit from this development, it would seem to be the domain of psychoactive drug use, where choices are so often pathological.

Thus, one can imagine my surprise and dismay when I was asked to prepare an essay on new policy insights that might follow from the leading behavioral economic theories of addiction¹, and I discovered that there weren't any. Or at least, hardly any. In this essay, I present evidence for that assertion, offer some speculative hypotheses about why it is true, and ask whether it is likely to remain true in the future.

1.1. Some evidence

A piece of evidence, I offer the behavioral record -- the behavior of professional drug policy analysts, in the form of two lengthy monographs on drug policy, both of which were published in 2001. Both monographs were prepared by interdisciplinary teams that included both psychologists and economists. I should emphasize that "the psychologist" in both cases was me.

The first is my recent book with Peter Reuter, *Drug War Heresies: Learning from Other Vices, Times, and Places* (MacCoun & Reuter, 2001). The book is a comprehensive analysis of alternative legal policy regimes for controlling marijuana, cocaine, heroin, and other recreational drugs.² It is thoroughly interdisciplinary in scope -- Peter is an economist, I am an experimental social psychologist by training, and our collaborators included the economist Tom Schelling and

the historian Joe Spillane. The book includes chapters on economic theory, psychological theory, moral philosophy, history, cross-national analysis, and so on. But in a 479-page book, with 44 single-spaced pages of bibliographic references, we made almost no use of the theoretical literature on addiction.

The other monograph is *Informing America's Policy on Illegal Drugs: What We Don't Know Keeps Hurting Us* (Manski, Pepper, & Petrie, 2001), the final report of the National Research Council's Committee on Data and Research for Policy on Illegal Drugs. The monograph was produced by 16 members spanning a host of disciplines. This 407-page monograph devotes several pages to neuroscience and behavioral economic concepts of addiction (though not particular models), yet those concepts played almost no detectable role in the subsequent analyses of supply reduction policies, user sanctions, drug prevention, or drug treatment.

One might respond to these observations in by suggesting that behavioral economics simply has a marketing problem—that theorists simply need to more aggressively disseminate and promote their theories. That is almost certainly correct. But I don't believe it is the source of my observations. In neither case did the authors simply overlook these theories in the preparation of the monographs. For example, during the nearly full decade Peter and I spent working on our project, I immersed myself in the neuroscience, economic, psychological, and philosophical literatures on addiction, assembling large collections of papers by the other presenters at this conference. It is riveting stuff, and I learned a great deal in the process. We simply found very little we could use in analyzing the question of the relative benefits and weaknesses of alternative drug-control regimes.

A related response might be that we as policy analysts simply failed to comprehend and appreciate the relevance of these models for drug policy. I am not well -situated to assess this possibility; by definition, one cannot assess whether one suffers from miscomprehension or a failure of imagination. If others respond to this essay by demonstrating that I overlooked profound new implications of these theories for drug policy, I will happily concede and judge this essay to have failed in its arguments but succeeded in its consequences.

After reviewing these policy implications, I will consider a number of alternative explanations for why behavioral economic theories of addiction (henceforth, “BETA”) have produced relatively few policy insights. I conclude that the limited policy implications stem from several features shared by BETA: The overlap in the causal factors that motivate “addictive” and “non -addictive” psychoactive drug use; the overlap between the policy implications of addiction theories and more conventional theories of drug control; and the notion that addiction is a unitary phenomenon with one correct theoretical explanation.

1.2. Some caveats

Before I plunge headlong into my arguments, it is worth briefly clarifying what I am not arguing:

1. I am not decidedly not arguing against behavioral economics as a scientific enterprise.
2. I am not arguing that there is nothing interesting or worthwhile about developing behavioral economic models of drug use or other potentially addictive behavior, although I will argue that the addiction construct is a distraction from the most useful aspects of the behavioral economic analysis of drug use.
3. I am not disputing the existence of drug addiction, or the enormity of its consequences, though I do question whether “addiction” forms a discrete, coherent category. I am not

simply echoing the positions of critics like Stanton Peele (e.g., 1996), Sally Satel (e.g., 2001), or Thomas Szasz (e.g., 1974), each of whom have criticized conventional uses of the addiction concept, though for differing reasons. My arguments in some way overlap with theirs, but I approach the issue from a very different perspective, working backwards from policy analytic considerations rather than working forwards from a set of first principles about human conduct, liberty, or morality.

2. Why the addiction concept may seem more relevant than it is

2.1. What do policy analysts want to know? ³

The left column of Table 1 lists the key levers that are conceptually (if not always politically) available to drug policymakers (see MacCoun, Reuter, & Schelling, 1996):

Table 1. Policy levers and related empirical uncertainties

Policy levers	Key empirical uncertainties
<ul style="list-style-type: none"> • Drug prevention, education, and rhetoric from the bully pulpit • Drug treatment • Criminal sanctions against users • Criminal sanctions against dealers • Interdiction and source country controls • Taxes, advertising controls, and other regulatory mechanisms • Drug testing • Banning employment, welfare, and other benefits 	<ul style="list-style-type: none"> • Cost-effectiveness and cost-benefit ratios of various interventions • Prevalence and incidence of drug use, and statistical distribution of frequency and quantity of consumption • Price elasticity of demand for drugs • Time sensitivity and/or impulsivity of drug users • Dose-responder relationship between consumption and its acute and chronic effects • Relative contribution of psychoactive effects vs. illegality in producing drug-related harms • Possible substitution, complementarity, and “gateway” relationships among

	<p>drugs</p> <ul style="list-style-type: none"> • Unintended effects of use - reduction strategies on drug harms, and of harm - reduction strategies on drug use • Distribution of effects of drug use across bearers – user, family, friends, neighbors, community, taxpayers
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Analysis of these policy levers follows two approaches, direct program evaluation (common for prevention and treatment, rare for enforcement) or theoretical analysis. The right column of Table 1 lists explanatory constructs relied on most heavily in recent theoretical analyses of American drug policy (e.g., Behrens et al., 2000; Caulkin et al., 2000; Kleiman, 1992, 1998; MacCoun & Reuter, 2001; Manski, Pepper, & Petrie, 2001). It is clear that BETA make contact with these explanatory factors in myriad ways. But in the remainder of this section, I will attempt to illustrate how behavioral economic theories of addiction largely generate policy implications that are redundant with existing strategies. And the novel implications they do offer follow from general principles of self-control rather than an narrow and extreme end state called “addiction.”

2.2. BETA’s implications for demand reduction

2.2.1. Prevention Some authors have argued that BETA have implications for drug prevention. For example, Herrnstein and Prelec (1992, p. 357) argue that their model “suggests that society should at least provide people with more information, on the ground that they are less likely to go down the path if they know where it is headed.” Heyman (1996, p. 573) argues that “... the ideas presented here indicate that treatments should attempt to bring drug consumption under the control of overall rather than local value functions.... Thus, methods that increase the salience of distant behavioral consequences should move individuals towards more

rational use of drugs. This points suggest that persuasion is a potentially powerful weapon in altering people's behavior.”

These recommendations fall short on two grounds. First, they restate the obvious; public information campaigns on the risks of long-term drug use have been a staple of American drug policy for over 30 years. Second, they ignore the evidence that such information campaigns have been remarkably ineffective at discouraging drug use (and risky sex) and are generally recognized as insufficient by prevention researchers. (See Chapter 7 of Manski et al., 2001 for a detailed review and meta-analysis.) In fairness, past anti-drug information campaigns might have been more effective if they had been more credible and less moralistic. In contrast, the prevalence of cigarette smoking fell by half in a generation following the release of a series of highly factual, morally neutral Surgeon General reports. But even there, it is discouraging that tobacco initiation rates among youth have remained remarkably stable.

2.2.2. Treatment. A more likely mechanism by which BETA might contribute to drug policy would be a new and better method of drug treatment. Behavioral economics research has already made significant contributions to the design of drug treatments. For example, the NRC report (Manski et al., 2001, p. 248) highlighted the behavioral economic work of Stephen Higgins and his colleagues as among the most promising developments in cocaine treatment research. This approach applies community reinforcement techniques and a “token economy” system of vouchers for retail goods to help cocaine users remain abstinent (see Higgins et al., 1995; Bickel, DeGrandpre, & Higgins, 1995). These studies are invaluable. It is highly plausible, but not very helpful, to be told that drug problems might be reduced by eliminating joblessness and poverty. It is nearly incredible, and extremely helpful, to learn that heavy cocaine users will provide three clean urinesamples for a \$10 gift certificate.

But while this treatment method is decidedly “behavioral economic,” it does not depend in any direct way on a behavioral economic account of *addiction*. The same logic would follow from a behavioral economic analysis of self-control difficulties – or indeed from a more traditional applied behavioral analysis (the contemporary term for behavior modification).

For the sake of argument, imagine that insights into effective drug treatment eventually emerge from behavioral economic analyses that require a notion of addiction *per se*, rather than a broader analysis of self-control. A radical improvement in drug treatment effectiveness would dramatically alter the drug policy landscape, although I argue later that it would not eliminate our drug problems. But if the improvements were only incremental in magnitude, they would be unlikely to have a noticeable impact at the policy level. It is difficult to detect any major impact of past treatment research on policy decisions (see Reuter, 2001). And there is sufficient uncertainty about the true efficacy and effectiveness of treatment that any improvement may fall well within existing error bounds (see Manski et al., 2001; Horowitz, MacCoun, & Manski, 2002) – and short of the more extravagant claims. Finally, the drug policy budget is an imaginary construction – the funds aren’t fungible in the sense that dollars could simply be shifted from enforcement to treatment (Murphy, 1994), although there could be a reallocation of funds within the treatment portion of the budget.

2.3. BETA’s implications for supply reduction

Behavioral economic theorists have also drawn various implications of their theories for supply reduction policy.

2.3.1. Availability. Several BETA theorists have suggested the importance of minimizing opportunities to obtain drugs; e.g., “... differences in prevalence rates will depend importantly on exposure to drugs. . . . it seems likely that increasing the availability of addictive drugs would

substantially increase the frequency of addiction” (Heyman, 1996, p. 573). This is surely correct, but, like the advice on prevention, redundant. Over half of our annual national drug control expenditures go to supply reduction efforts; roughly a third for interdiction and source country controls. It is difficult to imagine a more aggressive supply reduction effort than the one we’ve experienced, and yet student surveys show that drugs remain readily available at schools, and cocaine and heroin prices have fallen to about a third of their 1981 levels after controlling for inflation (see MacCoun & Reuter, 2001, Ch. 2).

2.3.2. Prices. Changes in prices have little impact for addict’s drug use rates under a traditional “enslavement” view of addiction. (It might, however, influence the number of crimes some addicts commit to finance their habits.) Under that model, addicts were considered to be extremely insensitive to prices. Until Becker formulated his rational addiction theory (e.g., Becker et al., 1992), drug experts largely ignored users’ “price elasticity of demand” (the percent change in drug use for a 1 percent change in price). But recent studies (reviewed in Caulkins and Reuter, 1996) suggest considerable price sensitivity, with elasticities for cocaine ranging from 0.7 to -2.0. In other words, addicts reduce their consumption when prices rise. The emphasis on drug prices is surely one of the most important contributions of the economic approach to drug policy.

Unfortunately, in a prohibition regime, there isn’t much we can do with this knowledge. Prohibition itself keeps prices artificially high, but beyond that, our supply reduction efforts are spectacularly ineffective at influencing prices at the margin. A legal regime would provide considerably more leverage, through taxation, price controls, and other regulatory possibilities (MacCoun, Reuter, & Schelling, 1996). Thus, BETA probably has greater potential policy impact in the tobacco and alcohol domains than in the domain of illicit drugs.

2.3.3. Smart deterrence and coerced abstinence. Kleiman (2000, 2001b) has offered a persuasive behavioral economic analysis of ways we might enforce prohibition more effectively. He argues that hyperbolic discounting implies the need to shift our emphasis from severe but uncertain and delayed sanctions to a regime in which sanctions are modest but swift and probable. His “coerced abstinence” model of aggressive drug-testing of probationers offers a radically different way of deploying law enforcement resources for drug control. But nothing in Kleiman’s analysis requires the notion of “addiction.” Coerced abstinence makes sense if heavy users make impulsive choices; it would make little sense – indeed, it would be inhumane – if they were incapable of choice.

2.4. BETA’s normative (welfare) implications

There is a third category of potential policy implications that are normative rather than empirical.

2.4.1. Is this state justified in prohibiting drugs? Do BETA tell us whether government intrusion into private choices is justified? A tradition dating back to John Stuart Mill considers such intrusion justified if it harms others (see MacCoun & Reuter, 2001, Ch. 4). There is overwhelming evidence associating drug use with such externalities, but we still know very little about the relative contribution of three causal mechanisms to this association: psychopharmacological effects of drug use, overlap in the dispositional propensities to use drugs and commit crimes (see below), and criminogenic consequences of prohibition and its enforcement (MacCoun, Kilmer, & Reuter, in press). Unfortunately, BETA have remained largely silent about this question by focusing on drug consumption but not its consequences.

2.4.2. Is drug addiction an involuntary state? A second normative question is whether drug addiction is involuntary, such that addicts aren’t capable of making rational choices. In

theory, penalties and sanctions are unjustified factors are incapable of controlling their actions. In theory, paternalistic government behaviors are justified if factors are not capable of protecting their own welfare.

Becker's "rational addiction" model is provocative precisely because it suggests that addicts freely choose their situation with full recognition of its eventual consequences. Analyses by O'Donoghue and Rabin (e.g., 1999) and Gruber and Koszegi (2001) persuasively challenge this extreme characterization. But it is not entirely clear what's at stake in this debate for the normative choice among policy regimes. BETA model addiction as a choice process, but a constrained and distorted choice process. From a moral philosophical perspective, the models offer not black or white but shades of gray.

Politically, it might not matter, for the public is not teetering on a moral knife edge where evidence might tip us one way or the other. Americans of a conservative stripe insist on strict norms of individual responsibility; American liberals endorse paternalism for far more trivial consumer choices than heroin consumption (Skitka & Tetlock, 1993). Yet Americans of both stripes largely reject the notion that drug dependence is completely involuntary; if the addict doesn't choose today's injection, she certainly chose her first injection (see Mannetti & Piero, 1991; Weiner et al., 1988). Hamilton (1981) argues that Americans judge others not by scientific causation but by the question "could the actor have done otherwise." It is not clear that Becker and his BETA competitors actually differ in their answer to that question.

2.5. BETA's policy implications: Summary

To date, most of the proposed policy implications of BETA are either redundant with current policies, or have less policy import than meets the eye. Significantly, almost all the policy implications discussed here were also suggested by Bickel and DeGrandpre (1996, pp. 46

47) in their analysis of behavioral economic principles of reinforcement. This point is noteworthy because that analysis made only a passing reference to the notion of “dependence” and no reference to the word “addiction.” This suggests, to me at least, that most of the important implications of behavioral economic analysis don’t actually require the concept of addiction.

3. Possible explanations for the limited usefulness of the addiction concept

I am confident in my thesis that BETA have offered few new policy insights, at least so far -- a rather depressing conclusion. I am less confident that I know why. Here I offer six speculative explanations, one which seems unpersuasive and five that seem more plausible.

3.1. Theory where no theory is needed?

One possibility is that this is just an example of the classic division between “basic and applied research.” On this account, it is foolish to ask for policy relevance from basic science. This proposition might be correct at the extremes, but it is certainly not defensible as a general proposition. There is usually good reason to accept Kurt Lewin’s (1951) classic dictum that “there’s nothing so useful as a good theory.” And it is clear that at least some major theorists in this area do in fact desire to inform drug policy. ⁴

In a classic essay, Milton Friedman (1953) defended an “as if” meta-theory of economics, drawing an analogy to a billiard expert who behaves “as if” solving a complex set of differential equations without actually doing so. One possibility is that a formal model of addiction might yield useful predictions of this sort, even though it is not a valid model of the actual addiction process. It would serve as a valid “black box” model of the functional relationship between causal antecedents and consequences, while remaining mute as to the underlying mediational processes.

Or one might ignore causal antecedents altogether. James Q. Wilson (1983) argued that "...one can intelligently make policies designed to reduce crime without first understanding the causes of crime..." It is hard to know how seriously to take this quote, since only two years later Wilson published (with Richard Herrnstein, 1985) a lengthy tome on the causes of crime. At any rate, BETA researchers clearly aspire to develop valid models of causal processes as well as input-output associations, as they surely should.

3.2. The wrong level of analysis?

Another possibility is that BETA are framed at the wrong level of analysis to be relevant for policy analysis. Interestingly, George Ainslie has referred to his BETA as "picoeconomics," as distinguished from microeconomics and macroeconomics. It is often the case that collective social phenomena are more than the sum of individual actions. Indeed, the public health movement has made important conceptual advances by adopting "population thinking" as an alternative to an individual-based clinical perspective. But I would not try to defend the position that good policy analyses can do without a model of the individual actor, and that is surely not what Ainslie has in mind either.

Still, it is conceivably the case that the notion of "addictiveness" might be useful for individuals governing their own conduct (individual policy), without being useful for the governing of aggregate conduct (public policy). A personal theory about addiction might itself be an important self-control device (Ainslie, 2001; Bateson, 1971). Ainslie (2001, p. 109) suggests that

"people cultivate the belief that street drugs are always irresistible once tried, rather than just making an overrule against trying them. This cultivation is apt to take the following form: An authority teaches that irresistibility is a fact; you encounter evidence

to the contrary, for instance in statistics on ex-users who used only casually; you discount or somehow don't incorporate the contrary evidence, not because it seems to be of poor quality, but out of a feeling that it's seditious."

Later, he notes that when Ockham, Galileo, and Darwin "pointed out that the 'facts' on which people based moral norms weren't found in nature, they encountered violent objections on the grounds that these discoveries would undermine morality" (p. 112).

3.3. Overlap with other theories

Another reason why BETA might fail to produce novel insights is that it overlaps in broad ways with more popular conceptualizations of drug use, even when they differ radically in their details.

One source of overlap is lay common sense or folk psychology. One can describe heavy drug users as "giving in to temptation," that they are "self-indulgent," "impulsive," "short-sighted," and "selfish," without any knowledge of the subtleties of BETA.

But there is also considerable overlap with contemporary criminological theory. In their highly influential "general theory of crime," Gottfredson and Hirschi (1990, p. 41) argue that:

"Crime and drug use are connected because they share a feature that satisfies the tendencies of criminality. Both provide immediate, easy, and certain short-term pleasure. ... Evidence to support our contention is found in the correlation between the use of cheap drugs, such as alcohol and tobacco, and crime... [and] by the connection between crime and drugs that do not affect mood or behaviors sufficient to cause crime (such as tobacco)."

Whether Gottfredson and Hirschi's central construct of "low self control" is isomorphic with the BETA notion of hyperbolic discounting is still unclear. Vuchinich and Simpson (1998) found

only weak and inconsistent correlations between personality measures of impulsivity and hyperbolic discounting behavior among light and heavy drinkers. The personality measures were better discriminators of light vs. heavy drinking than were discounting scores, at least in that experimental setting and sample.

3.4. A problematic construct?

Analytic use of the addiction concept may be hindered by its lack of adequate construct validity, in the psychometric sense of a unitary concept that can be adequately delineated and distinguished from other concepts. One can dispute the usefulness of the addiction construct without disputing the ontological reality of addiction or making sidereference to a metaphysical “ghost in the machine.” The question is whether the construct would be more useful if it were disaggregated into distinct features.

The DSM-IV permits a diagnosis of substance dependence when any three of the following are observed in a 12-month period: Tolerance, withdrawal, using more than intended, desire to quit and/or difficulty quitting; considerable time spent obtaining, using, or recovering from the drug; interference with other activities; and/or persistent use despite problems caused by use. The DSM-IV definition of dependence has fairly high interrater reliability (Heyman, 2001), and the interitem correlations are reasonably high (Feingold & Rounsaville, 1995). But a construct can have high reliability without having high constructor predictive validity (e.g., astrological signs).

The DSM items may hold together empirically, but it is not clear that they do so conceptually in a way that makes the best analytic use of the data. At least as currently used (with the “any three” criterion), these items don’t form a meaningful Guttman scale, as they would if the components had a logical, cumulative order (e.g., None, A only, A+B, A+B+C,

etc.). One can interpret the debate between Ole -Jørgen Skog and Nick Heather in this volume as a debate about what a defensible Guttman scale of addiction might look like.

The DSM -IV dependence checklist items don't look anything like interchangeable, substitutable indicators of a latent construct, in the psychometric "domain sampling" sense. They aren't like items on a personality scale or intelligence test which can be thought of as tapping identical construct variance plus idiosyncratic item error. Instead, each component on the checklist is conceptually distinct. Moreover, the criteria confound the condition of addiction with its antecedents, its consequences, and its context, thereby begging the very questions that theory (and policy analysis) need to answer. Finally, epidemiological studies (e.g., Anthony et al., 1994) demonstrate considerable heterogeneity in the qualifying criteria displayed across individuals receiving the same "dependence" diagnosis, and even greater heterogeneity across addictive substances (alcohol vs. tobacco vs. opiates vs. cocaine vs. cannabis).

3.5. An overdetermined phenomenon?

Discussions about the relative merit of addiction theories of ten seem to accept two implicit assumptions: that addiction is a single, unitary phenomenon, and that it is caused by a single process.

Addiction theorists too often rely on "sufficiency" arguments in favor of their theories (MacCoun, 1996). Some stylized facts about addiction are reviewed, and it is then demonstrated that the theory in question can produce such patterns. Even if correct, such arguments show that the theory is sufficient to produce "addictive" behavior; they do not establish that the hypothesized mechanisms actually produce the actual addictive behavior we observe in the world. In essence, behavioral economic theorists have tended to start with the model (rational

choice theory) rather than actual behavior; the goal has been to teach the model new tricks – how to act addictively – in the fewest steps possible.

But there are good reasons to believe that real-world addiction is *overdetermined*, with a complex set of interrelated distal and proximal causal antecedents. A very partial list would include factors discussed in detail in this volume:

- classical conditioning of cues
- operant conditioning (especially schedules of reinforcement)
- tolerance, withdrawal, opponent processes, and other neurochemical adaptations
- impulsivity due to hyperbolic temporal discounting

But many researchers would list additional mechanisms falling outside the theoretical framework of either neuroscience or BETA, such as:

- biased cognitive expectancies (e.g., Stacy, Widaman, & Marlatt, 1990; Tversky & Kahneman, 1974), including “optimism bias” (the tendency to believe that generic population risks don’t apply to oneself”; e.g., Weinstein & Klein, 1995)
- sensation seeking (Zuckerman, 1994)
- “social scripts” (automatized behavioral schemata; see Wegner & Bargh, 1998)
- maladaptive self-regulatory strategies for dealing with conflicting goals (Baumeister, 1997; Baumeister et al., 1994; Carver & Scheier, 1998; Tice et al., 2001; Wegner et al., 1989)
- attentional control (e.g., Steele & Josephs, 1990)
- self-handicapping and other self-presentation strategies (e.g., Higgins & Harris, 1988; Islieb, Vuchinich, & Tucker, 1988)

With such a lengthy list, it seems strange that many experts still consider addiction to be

“paradoxical.” For example, Elster and Skog (1999, p. 1) argue that “On a theoretical level,

addiction raises the paradox of *voluntary self-destructive behavior*. The challenge is to explain

why people engage in behaviors that they know will harm them.” (Elster & Skog, 1999, p. 1).

This notion of a paradox follows naturally from a rational choice perspective, or from a less sophisticated “folk psychological” theory in which actors are conceived as making coherent, conscious choices on the basis of a stable set of beliefs and desires. But it is less clear why addictive behaviors should be viewed as “paradoxical” from the perspective of contemporary scientific psychology or neuroscience. There is ample evidence that self-defeating behaviors are commonplace among otherwise well-functioning, non-clinical populations (Baumeister, Heatherton, & Tice, 1994). Baumeister (1997) notes that none of these mechanisms require any explicit self-destructive motives. They are overdetermined by a variety of fairly normal processes, especially cold, warm, or hot cognitive biases of information processing; and/or perverse side-effects of self-regulatory strategies for pursuing conflicting goals.

Self-regulatory models in cognitive, social, personality, and developmental psychology do imply a purposive actor, but they are not built on rational or quasi-rational choice principles. This makes them less rigorous deductively, but the models do make clear, testable predictions that can and have been tested using experimental methods (see Carver & Scheier, 1998; Muraven & Baumeister, 2000; Tice et al., 2001; Wegner & Bargh, 1998).

3.6. Undue Emphasis on the Extremes

3.6.1. The distribution of drug consumption across users. The proposition that “addiction” is overdetermined has testable implications. “Single mechanism” theories may propose qualitative discontinuities—thresholds beyond which a user passes from “non-addiction” to “addiction.” But such analyses are *ceteris paribus*. Presumably, the multiple mechanisms of “addictiveness” are highly correlated, but they are not isomorphic, so a discontinuity in one mechanism might well be obscured by the operation of other mechanisms. A series of superimposed step functions might collectively form a smoothly continuous function.

If so, one would not expect to observe stark discontinuities between “addicted” and other heavy users. In principle, this should be testable using psychometric techniques for empirically distinguishing discrete typologies from continuous, dimensional traits (Meehl, 1995).

Some indirect evidence on this point comes from the National Household Survey on Drug Abuse (NHSDA) for 2000. ⁵ Figure 1 shows the number of days of drug use per year among Americans age 12 and older who used in the past year, separately for marijuana, cocaine, and alcohol. (Unfortunately, cigarette data are not available for this variable.) For marijuana and alcohol, but not cocaine, the distributions are bimodal. The largest mode is at “1 to 11 days per year” (very casual use), but the second mode is at “100 to 299 days per year,” not “300 or more” as one might expect given the ease with which we use the label “addict.”

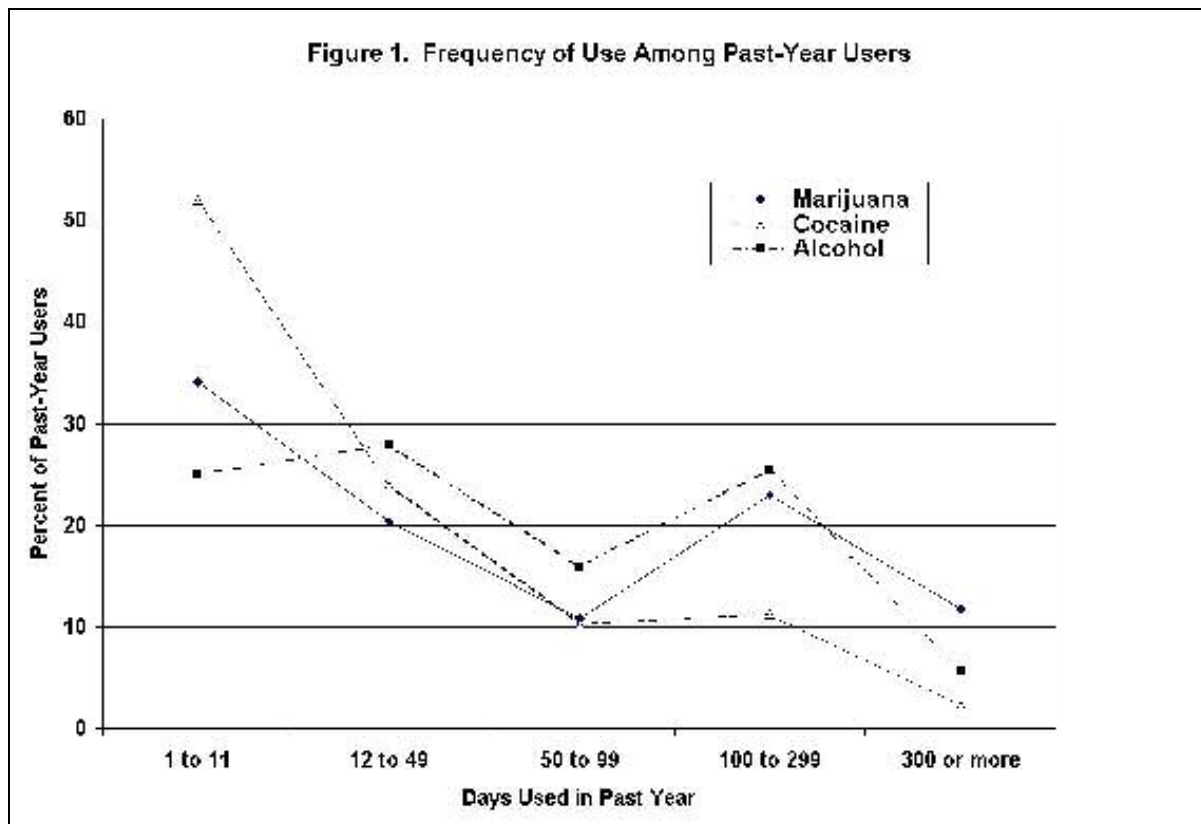


Figure 2. Days of Use in Past Month

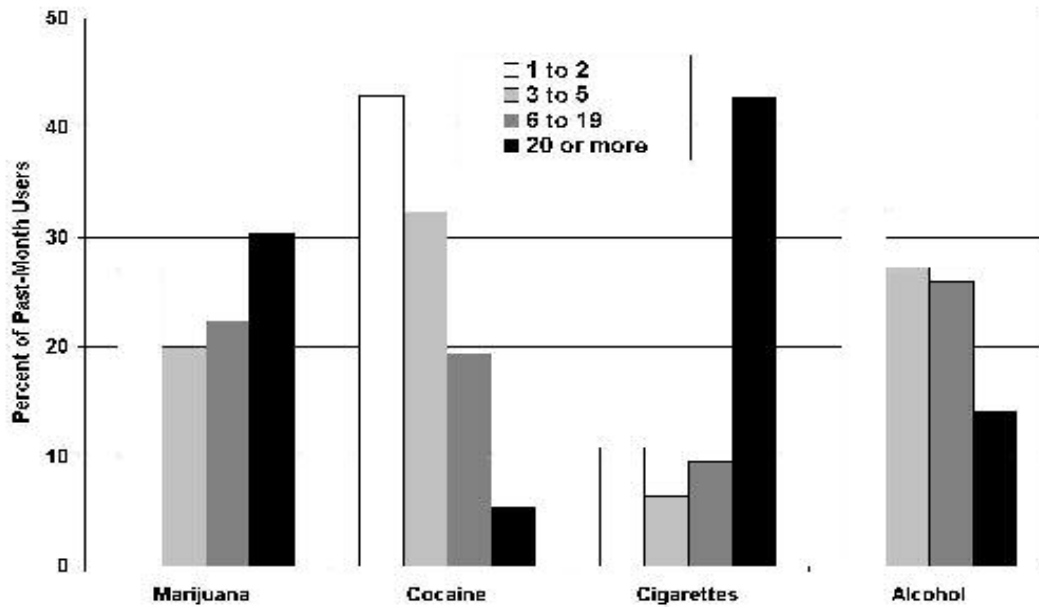
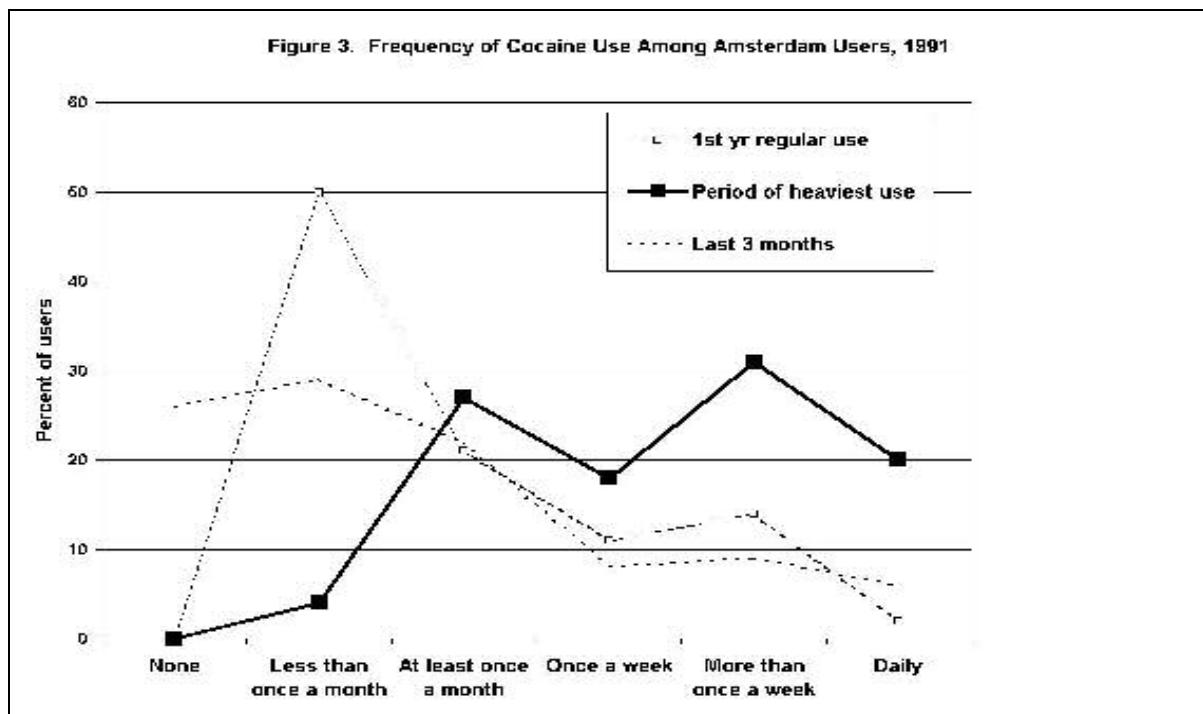


Figure 2 focuses more narrowly on past -month users, thereby screening out most of the very casual users. The data for cigarettes match the profile of “an addicted drug”, with the modal user using 20 or more days out of the month. But for marijuana, cocaine, and alcohol, even among past -month users few use 20 or more days a month.

Figure 3. Frequency of Cocaine Use Among Amsterdam Users, 1991



Unfortunately the NHSDA sampling and self-report procedures are thought to underrepresent the heavy cocaine use. Figure 3 shows use frequencies in a sample arguably less susceptible to such biases – a snowball sample of recent cocaine users in Amsterdam (Cohen & Sas, 1995). Despite a very different sampling strategy and a far more tolerant culture, self-reports of use during the last 3 months, and during the users' first year of use, look quite similar to the pattern in the NHSDA data. Even for the "period of heaviest use," only 20 percent reported daily use. Compulsive use, in which lives are dominated by drug consumption, is an extremely important part of the policy picture, but it is clearly not the whole picture by any means.

3.6.2. Loss of information due to the choice of study populations. By treating addiction as a category rather than a continuum, BETA researchers frequently rely on clinical populations that fail to represent the full range of patterns of consumption of a given drug. According to Heyman (2001, p. 91), "most addicts recover, but this is only apparent if the addicts are selected independently of their treatment history." Two BETA studies present evidence suggesting that hyperbolic discounting might vary gradually rather than discontinuously between clinical "addict" populations and other users. Vuchinich and Simpson (1998) found that while heavy drinkers and/or problem drinkers showed stronger temporal discounting than light social drinkers, the 75th percentile for discounting among light drinkers fell near the median for heavy drinkers. Bickel, Odum, and Madden (1999) found a multimodal distribution of delay discounting parameter values among current cigarette smokers, overlapping considerably at the low end with values for ex-smokers and never-smokers.

3.6.3. Would we eliminate the drug problem if we eliminated addiction? One can reasonably defend a focus on clinically defined addicts as follows: Not all drug use is harmful; a

society that values individual liberty ought to concentrate its attention on those users who are harming themselves and/or others. I have much sympathy for this viewpoint. But matters are not so simple; the risks of drug use vary continuously across users with no apparent “step function.”

For the sake of argument, let's say we actually cured addiction -- i.e., any user who crossed a certain behavioral threshold could be recalibrated -- restored to a state of non-addiction, perhaps even one permitting "controlled use" of the drug in the future. Clearly, this would significantly shift American policy away from a primary emphasis on law enforcement to a greater emphasis on treatment; indeed, it may be the only way such a shift could occur given the great political advantages of being “tough on drugs.” And the “drug problem” would be reduced dramatically. But not completely. How much of a problem would remain?

The answer depends on some empirical questions that have received some attention in alcohol epidemiology (Edward et al., 1994), but have been largely neglected in the illicit drug domain (MacCoun, 1998): What does the consumption distribution look like? What are the dose-response functions that link consumption to various health and safety harms?

We know that the distribution of users by consumption levels is positively skewed. Presumably, addicts are mostly located in the long right tail. We can reduce the harm of drug consumption by either targeting the heaviest users (the right tail) or, as some alcohol experts recommend, by trying to target the great majority of users near the middle of the distribution (see Rose, 1992; Skog, 1993). Presumably, the greater the share of total consumption due to heavy users, the greater the efficacy of targeting them. So if addiction were cured, would the right tail be eliminated, or just “thinned out”?

A cure for addiction might reduce, but will surely not eliminate the acute harms of intoxicated driving, parenting, work behavior, and so on. A few facts about alcohol are sobering. It is estimated that teenager drinkers – few of whom are likely to be “addicted” (at least not yet) – account for 11.4 percent of all alcohol consumed in the U.S. (CASA, 2002). In the 2000 Drug Abuse Warning Network study, 12–17 year olds account for 17.7 percent of all emergency room mentions of alcohol (SAMHSA, 2001). Among drivers in fatal accidents, the age 21–24 group consistently has the highest proportion with blood alcohol levels exceeding 0.10 (NHTSA, 2000).

Indeed, the literature on “compensatory behavioral responses” to risk reduction (reviewed by MacCoun, 1998) suggests that a cure for addiction might actually encourage much intoxication that would not otherwise take place. Existing users might have less reason to fear a binge; non-users would have less reason to fear initiation. Whether these incentives would be large enough to offset the sizeable reductions due to the elimination of addiction is not clear.

Much depends on the parameters of the relevant dose-response curves linking drug use to its various consequences. Such curves are usually S-shaped. When they are very steep, even moderate consumption levels are risky. Presumably, some “acute” risks are primarily sensitive to dosage per incident (e.g., driving accidents, overdoses, unsafe sex, and what Goldstein [1985] calls “psycho-pharmacological violence”) whereas other risks are triggered more by chronic use overtime (e.g., deteriorating health, bad parenting, and Goldstein’s [1985] “economic compulsive violence”).

Interestingly, the recent Swiss heroin maintenance trials suggest that the dose-response functions can vary dramatically with legal context (Reuter & MacCoun, 2002). Registered addicts who were eligible to receive heroin from government clinics massively increased their

daily doses, yet they significantly increased their legitimate work participation and significantly reduced their income -generating criminal behaviors.

Elsewhere my colleagues and I have decried the American tendency to almost single-mindedly equate drug policy with “prevalence reduction” – a reduction in the number of Americans whose use of drug. Arguably, a more sensible overarching goal is “total harm reduction” – reducing the total social harm caused by drug use. But $total\ harm = average\ harm\ per\ user \times number\ of\ users \times average\ amount\ used$, and the emphasis on prevalence reduction (something we’re not very good at) leads to the neglect of two other strategies – quantity reduction and harm reduction (MacCoun, 1998; MacCoun & Reuter, 2001).

It is surely better to categorize users into “addicts” vs .non-addicted users, rather than mindlessly (and moralistically) lumping heavy users together with extreme casual light users (see Caulkins, 1997). But we should beware of reifying an extreme corner of a continuous, multidimensional space constituted by the dimensions of frequency of use, quantity consumed per use, and harmfulness of conduct while intoxicated. Doing so begs the questions I noted above – the need to know the shape of the consumption distribution and the relevant dose-response functions linking use to harms.

4. With Friends Like These...

It is regrettable that this chapter has such a critical tone. My purpose in raising these arguments is not to discourage behavioral economic work on drug use – far from it. But a candid assessment suggests that, at least so far, BETA’s insights into drug policy fall into two categories. They are either largely redundant with the conventional wisdom as expressed by existing policy strategies (viz., drug prevention and supply reduction), or they are quite innovative but seem not to require any conception of “addiction” as a distinct state or category of

experience (viz., treatment and self-control strategies). The first category is no fault of the theorists, but the second suggests that the addiction concept just isn't that useful. In my view, the value of the behavioral economic comes from its analysis of self control (a broad category), not from its analysis of addiction (a very narrow one). In short, from BEAT (the behavioral economic analysis of temptations) rather than BETA (behavioral economic theories of addiction).

Are there policy implications I (and the BETA community) have overlooked? Probably. I can see at least four areas for future development:

- Structuring of the very "local" (in time and space) economy to help facilitate better self control (an idea floated in various ways by several authors of this volume; see Wertenbroch, 1998; Read, Loewenstein, & Kalyanaraman, 1999 for marketing examples).
- The development of more psychologically realistic law enforcement deterrence tactics (Kleiman, 2000, 2001b; MacCoun & Reuter, 2001, Ch. 5).
- The incorporation of behavioral economic principles into analyses of the dynamics of drug epidemics and the strategic timing of interventions (Behrens, Caulkins, Tragler, & Feichtinger, 2000).
- A behavioral economic analysis of the triage problem in the design of heroin and other opiate maintenance schemes – deciding who should be eligible, and when (Reuter & MacCoun, 2002).

If I can name four, then hopefully readers can come up with many more. I see no reason why an assessment of the policy payoffs of a behavioral economic analysis won't be considerably more up beat a decade from now.

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6. Endnotes

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¹I take the members of this set to include the recent work of such theorists as Becker, Grossman, and Murphy; Prelec, Herrnstein, and Heyman; Ainslie; Rachlin; Elster; and O'Donoghue and Rabin. Much of this work has been summarized in various chapters in the recent volumes *Addictions: Entries and Exits* (edited by Elster, 1999), *Getting Hooked: Rationality and Addiction* (edited by Elster and Skog, 1999), *Breakdown of Will* (Ainslie, 2001), and *The Science of Self-Control* (Rachlin, 2000). Note that Ole-Jorgen Skog (paper for this conference) questions whether Becker's model is in fact a model of "addiction."

²The book is the major product of a grant from the Alfred Sloan Foundation to the RAND Corporation's Drug Policy Research Center.

³This section draws heavily on arguments developed in much greater detail in MacCoun and Reuter (2001) and Manski, Pepper, and Petrie (2001). But many of these arguments were independently developed and presented by Mark Kleiman at a conference on "The Uses and Misuses of Science in Public Discourse," Boston University, April 1, 2000 (see Kleiman, 2001a).

⁴"To design treatments and policies that will make people quit their addictions or never become addicted in the first place, it is useful to have an understanding of the causes of addiction and relapse" (Elster & Skog, 1999, p. 1). "If economists want to contribute to the policy debate over

how to deal with addictions, we need to develop a systematic approach to analyzing self-control problems and other errors rather than assume them away. We hope our analysis will prove useful in this regard.” (O’Donoghue & Rabin (2001, p. 37 of preprint version). Becker and his colleagues (1992, p. 362) consider “highly tentative inferences concerning the effects of legalization...” and Herrnstein and Prelec (1992) devote three pages to a section on “policy implications” of their theory of addiction.

⁵http://www.samhsa.gov/oas/nhsda/2k_detailed_tabs/Vol_1_Part_4/sect6v1.htm#6.2b