Addiction, Cognition, and the Visceral Brain*

Douglas Bernheim
Department of Economics
Stanford University
and
NBER

Antonio Rangel
Department of Economics
Stanford University
and
NBER

March 2002

Abstract

This paper develops a new model of the consumption of addictive substances. The basic premise of the theory is that environmental cues can trigger "viscerally states" that lead the brain to provide an incomplete characterization of the decision problem, and that this can lead the decision-maker to make systematic mistakes. Importantly, cues affect behavior because they affect how the brain characterizes the problem, not because they change the underlying preferences. We show that the model is able to explain the basic stylized facts associated with addiction, that it has good foundations in neuroscience and psychology, and that it generates plausible consumption patterns for different substances. We also use the model to study the welfare properties of six drug policies: laissez-faire, taxation, subsidization of treatment programs, criminalization, regulated dispensation, and "behavioral policies" such as education and "shock-based" marketing campaigns.

1

1 Introduction

Although more than four million chemical compounds have been catalogued to date, only a few hundred generate the consumption patterns and behaviors that we associate with the addictive substances or drugs (Gardner and David [1999]).[1] These "rare" substances are of interest to economists for at least two reasons. First, at a conceptual level, they raise the challenge of understanding why agents knowingly engage in destructive behavior which they often feel powerless to control without costly rehabilitation therapies. Second, at a practical level, there is an open debate concerning the manner in which public policy should deal with this phenomenon. In this paper we study both questions.

Passionate advocates can easily be found at both ends of the policy spectrum. Analysts who believe that people are mostly rational favor a laissez-faire approach. By contrast, analysts who believe that people have basic self-control problems favor strong government intervention, from taxation to criminalization.[2] Clearly, the development of a model that can explain the basic stylized facts associated with addiction is an essential pre-requisite for sound policy analysis.

The consumption of drugs has received substantial attention in neuroscience, psychology, epidemiology, sociology, and economics. This body of research has established the following stylized facts that any theory of addiction needs to explain:[3]

1. Short-term abstention is common even for the most addictive substances, but long-term recidivism rates are high.[4]

2. Consumption and recidivism are associated with cue-conditioned cravings. For example, upon exiting rehab, addicts who return to their old environment are more likely to experience cravings and resume use.[5]

3. There are significant differences across substances. For example, caffeine users tend to consume it on a continuous basis and do not enter detox programs, while cocaine users frequently go through binging cycles, and many visit rehab centers several times during their lives.[6]

---

[1] The compounds listed by these authors include: (1) alcohol, (2) barbiturates, (3) amphetamines, (4) cocaine, (5) caffeine and related methylxanthine stimulants, (6) cannabis, (7) hallucinogenics, (8) nicotine, (9) opioids, (10) dissociative anasthetics, and (11) volatile solvents.

[2] In the economics literature, the first view is represented by the rational addiction model of Becker and Murphy [1988], and the second view is represented, among others, by the application of the quasi-hyperbolic discounting model to addiction (O'Donoghue and Rabin [1999,2000] and Gruber and Koszegi [2001]).

[3] Many of the studies referenced below are discussed in more detail in section 3.

[4] See Goldstein [2001], Hser, Anglin, and Powers [1993], Harris [1993], and O'Brien [1997].

[5] See Goldstein [2001], Goldstein and Kalant [1990], O'Brien [1976,1997], and Robins [1975,1993].

[6] See Goldstein [2001] for a description of the neural pathways through which different drugs

4. There are significant differences across users. In particular, although a sizable fraction of the population experiments with drugs, or uses them recreationally, only a small fraction becomes addicted.[7]

5. Users respond to standard economic incentives such as prices and information about the effects of these substances.[8]

6. Users engage in pre-commitment behaviors such as checking into rehabilitation centers and taking substances like disulfiram, which generates unpleasant side effects if alcohol is consumed subsequently.[9]

7. Exogenous attention shocks can affect drug demand temporarily *without providing new information.* For example, a recovering addict is less likely to use (at least temporarily) if, while experiencing a strong craving, he is reminded of some of the consequences of use.[10]

8. Recovering addicts exhibit a demand for attention management therapies. For example, even addicts who have stayed clean for years attend the meetings of support groups, such as AA, in which no individual therapies or drugs are provided, and in which no new information is revealed.[11]

9. Addicts describe their relationship with the substances as one of powerlessness and conflict. They report making mistakes during strong visceral states, sometimes *even while in the act of consumption.*[12]

In this paper we develop a new model of addiction that is able to account for these nine stylized facts, generates a plausible mapping from the exogenous characteristics

---

affect the brain, and some of the problems and behaviors that they generate.

[7]See Goldstein and Kalant [1990], Gazzaniga [1990, 1994], and Koob and Moal [1997].

[8]See Chaloupka and Warner [2001] and MacCoun and Reuter [2001] for a review of the evidence.

[9]See O'Brien [1997] and Goldstein [2001].

[10]Although this phenomenon is common place, it has not been emphasized explicitly in previous academic studies of addiction. See Massing [2000] for a compelling description of the life of addicts and the role that exogenous attention shocks play in their behavior. The impact of exogenous attention shocks in self-control (for non-drug situations) has been established in a series of experiments by Walter Mischel which are described in section 3.

[11]Goldstein [2001] reports that there is a shared impression among the professional community that 12-step programs such as AA (p. 149) "are effective for many (if not most) alcohol addicts". However, given the nature of these programs, objective performance tests are not available. He also describes that, in the view of these programs, there are recovering alcoholics, but not ex-alcoholics; hence the dictum "once an addict, always an addict". Their treatment philosophy is based on (p.150) "keeping it simple by putting the focus on not drinking, on attending meetings, and on reaching out to other alcoholics."

[12]Goldstein [2001,p.249] describes this familiar phenomenon: He had been "suddenly overwhelmed by an irresistible craving, and he had rushed out of his house to find some heroin. ... it was as though he were driven by some external force he was powerless to resist, *even though he knew while it was happening that it was a disastrous course of action for him*" (italics added).

of substances to consumption patterns, and has good foundations in psychology and neuroscience. The model is an application of a more general theory of decision-making developed in Bernheim and Rangel [2002].

By contrast, as discussed in appendix A and summarized in table 1, none of the existing theories can account for *all* of the facts.[13] Four main classes of theories have been proposed. First are theories based on tolerance and withdrawal (column 1). These theories fail to explain pre-commitment behaviors, the role of attention shocks, or the fact that addicts report making mistakes during hot visceral modes; and cannot provide plausible or complete explanations for the heterogenous experience of users, the demand of management programs such as AA, and some of the differences across substances. Second are theories based on cue-triggered taste shocks (column 2) which can explain the role of cues and why addicts often consume in binging cycles and the effect of exogenous attention shocks (subject to some caveats), but otherwise do not improve the explanatory power of the tolerance/withdrawal theories. Third are theories based on agents who have dynamic inconsistent preferences (either in the form of quasi-hyperbolic discounting or of "temptation costs") and understand that their preferences change with time (columns 3 and 4). These type of theories are unable to explain why agents are able to abstain in the short-run (often suffering painful withdrawal symptoms) but not in the long run, the role of cues, the impact of attention shocks, and agents that report making mistakes *while in the act of consumption*. They also have difficulty explaining the differences across substances and users, and the demand for therapies such as AA. Finally, there are theories based on the idea that agents systematically misforecast their future tastes, and are not aware of this problem (column 5). This class of theories cannot explain why agents can abstain in the short-term but not in the long-term, the role of cues, the demand for pre-commitment, or the presence of mistakes.

The basic idea of our model goes as follows. Behavior results from the operation of two types of brain structures: the neocortex and the visceral brain. Decision-making is depicted as a stylized, three-stage process. In stage 1, which we call *characterization*, the neocortex characterizes the decision problem by identifying a set of potential actions and by generating images about the present and future consequences of each action under consideration. Most importantly, the neocortex may fail to provide an accurate characterization of the problem. In Stage 2, which we call *evaluation*, the visceral brain takes the images generated by the neocortex and assigns an emotional evaluation to each option being considered. Hard-wired mechanisms then carry out the action with the most favorable evaluation. In Stage 3, which we call *experience*, the visceral brain generates a hedonic experience.

---

[13]We recommend that this appendix be read last.

Note that this framework provides a "neuro-foundation" for the standard exponential utility maximization model as long as the following three assumptions are satisfied. First, in the characterization stage, the neocortex considers *all* options and correctly anticipates *all* consequences. Second, in the evaluation stage, the visceral brain accurately anticipates the experiential pleasure/pain associated with the envisioned consequences of each action in all future periods and states of nature. Third, in the evaluation stage, the visceral brain aggregates the anticipated hedonic experiences over time and states of nature in some time and state consistent fashion.

We depart from the standard model by relaxing the assumption of perfect characterization. Shortcuts at the characterization stage are unavoidable since it would be impossible for any finite computational mechanism (including the neocortex) to consider every possible option and forecast every potential consequence. Thus, the neocortex makes decisions based only on a subset of actions and consequences. This does not undermine the standard model of economic choice, provided that the most pertinent alternatives and consequences are always considered (at least as an approximation). The key idea of the theory is that cues can affect the cognitive activation that takes place during the characterization stage. It then follows that cues can have an effect on behavior, and even induce mistakes, *without changing the preferences with which options and consequences are evaluated* in the second stage. We call this phenomenon *characterization failure*.

To concentrate in the problem of addiction, in this paper we provide a reduced form representation of the brain processes described above. More concretely, we assume that the brain can operate in two modes: a *cold* mode, and a *hot* mode. Normally the brain operates in the cold mode. The hot mode is activated when environmental cues trigger a strong visceral state. In the cold mode the neocortex considers every option and generates correct expectations about all consequences. By contrast, in the hot mode the neocortex generates many thoughts concerning the pleasure derived from the "high," but relatively few thoughts about other consequences. In fact, we assume that the characterization bias is so strong that an agent in the hot mode *always* chooses to use the drug. Under these simplifications, decision-making in the cold mode is easily modeled as a standard dynamic programming problem that takes as given the process of visceral activation and the nature of behavior in the hot mode. The resulting model is fairly tractable; it looks like a variation of the rational addiction model in which agents can make mistakes.

One of the advantages of this model is that it gives rise to a well-defined welfare criterion. The reason is simple. The mistakes that agents make in the hot mode are due to a malfunction of the neocortex during the characterization stage, and not to a change in preferences during the evaluation stage. Therefore, the natural welfare

criterion to evaluate public policy is given by the preferences that are coded in the visceral brain. This is not a paternalistic notion of welfare: it merely addresses a basic technological limitation of the brain. In this setting, a policy intervention is welfare improving if it restores the proper operation of the neocortex, or of it induces the brain in the hot mode to make the same choices that it would have made had the neocortex generated all of the relevant information.

We also study the policy implications of characterization failure in an idealized economy without enforcement costs and limited black markets. We derive three main conclusions. First, some commonly used policy instruments are ineffective in addressing the behavioral problems generated by characterization failure: laissez-faire dominates taxation and subsidized rehabilitation; criminalization dominates laissez faire only for some substances and under special circumstances. Second, a policy of regulated dispensation, in which drugs are legalized but must be purchased one period in advance, dominates all of the previous policies. Third, behavioral policies that address directly the brain mechanisms behind characterization failure, such as education and public information, can be welfare improving.

The remainder of the paper is organized as follows. Section 2 describes the model. Section 3 provides the foundations. Section 4 develops a fairly complete characterization of the comparative dynamics. Section 5 shows the consumption patterns that the theory generates for different substances. Section 6 shows how the theory explains the stylized facts. Section 7 discusses the welfare criterion. Section 8 studies the welfare properties of various public policies. Section 9 concludes. Appendix A discusses alternative theories of addiction. Appendix B contains the proofs.

2   Model

Consider a decision maker (DM) who lives for an infinite number of discrete periods. At the beginning of each period he makes one of three choices: use a substance (U), attempt to abstain from use on his own (A), or enter a rehabilitation center for the period (R). As discussed further below, attempted abstention may or may not be successful. By contrast, entering a rehab center guarantees abstention for the current period. In each period, the DM is in one of $S+1$ *addictive states*, labelled $s = 0, 1, ..., S$, which provide a summary statistic of the history of use. They evolve as follows. Usage in state $s \geq 1$ leads to state $\min\{S, s+1\}$ in the next period. No use leads to state $\max\{1, s-1\}$. Note that it is impossible to reach state 0 from any state $s \geq 1$. However, the reverse is not true. In state $s = 0$, use leads to state 1, while no use leads to state 0. The usage state $s = 0$ represents a "virgin state"

in which the DM has had no contact with the drug. Once the drug is used, the DM can never return to the virgin state.

For every period in which the DM's state is s, he receives income y_s, which he divides between ordinary expenditures e, expenditures on the drug qx (where q is the price of the substance, and x is the quantity consumed), and rehabilitation expenditures $r_s d$ (where $r_s \geq 0$ is the price of rehabilitation, and $d = 0, 1$). For simplicity we assume that the drug is only consumed at two levels, 0 or 1, and that the DM cannot borrow or save. Both income and rehabilitation costs can depend on the addictive state s.

The DM's brain can operate in two modes: a cold mode ($\mu = C$), in which the neocortex characterizes the decision problem perfectly, and a hot mode ($\mu = H$), in which there is characterization failure.

The timing of a period is as follows. The DM begins each period in the cold mode. At this time he contemplates all of the alternatives and establishes intentions for the current period (either to use, U, to attempt abstention, A, or to enter rehabilitation, R). At this time the neocortex considers the three courses of action and perfectly forecast all of future consequences. In particular, it anticipates the effects of today's usage on the probability of entering the hot mode which may lead to unwanted usage. He is then exposed to various hedonically neutral environmental cues. Some of these cues trigger the DM into the hot state. This occurrence depends to some extent on random events, and is therefore not completely predictable in advance. Moreover, the likelihood of being triggered varies with the addictive state. Accordingly, we assume that, if the DM enters the period in state s and attempts abstention (A), randomly-occurring environmental cues will trigger the hot state with probability p_s. If this happens, the DM tries to consume the substance regardless of his intention to abstain as of the beginning of the period. With probability $1 - p_s$, the DM remains in the cold mode and sticks to his plan, successfully abstaining. We assume that the brain cannot enter the hot mode from the virgin state: $p_0 = 0$. If the DM chooses U (R), he uses (abstains) regardless of whether he enters the hot state.[14]

The visceral brain evaluates consumption paths by assigning a "payoff" $w_s(e, x)$ to the consumption bundle (e, x) in usage state s. Payoffs are discounted at a constant discount factor δ. By assumption, cues are hedonically neutral, and thus the same function is used to evaluate outcomes in the hot and cold modes. The function w_s is strictly increasing in both of its arguments. The dependence of the payoff function on the addictive state incorporates the effect of tolerance and other

---

[14]Environmental cues may also trigger the hot mode when the individual chooses U or R, but this has no effect on choice. Accordingly, the model implicitly allows for the possibility that the probability of entering the hot mode may vary with the individual's intentions (e.g. if the DM selects A, he may avoid bad influences).

health and socioeconomic costs of drug use.

The individual's budget constraint requires $e + qx + r_s = y_s$. Define $u_s \equiv w_s(y, 0)$; $b_s \equiv w_s(y - q, 1) - u_s$, and $c_s \equiv u_s - w_s(y_s - r_s, 0) \geq 0$. Intuitively, u_s represents the baseline payoff level for state s, b_s represents the marginal instantaneous benefit from use that the individual receives in state s, and c_s represents the cost of rehabilitation in state s. In other words, u_s is the payoff associated with successful abstention (without rehab), $u_s + b_s$ is the payoff for usage (irrespective of the DM's mode), and $u_s - c_s$ is the payoff associated with rehab (again irrespective of the DM's mode). Let $\theta_s = (p_s, b_s, c_s, u_s)$ and $\theta = (\theta_0, ..., \theta_S)$ denote the parameters that describe the consumption problem. These parameters are affected by the characteristics of the substances, the characteristics of the individual users, and the public policy environment.

Under these assumptions the operations of the brain in the cold can be modeled as a simple dynamic programming problem. Given the irreversibility at $s = 0$, the decision rule in the deliberative mode is derived in two steps. First, we solve the problem for states 1 through S. Second, we consider the much simpler problem facing the brain in state 0. For technical reasons we allow the DM to randomize over possible choices in each period. In each period he chooses a vector (π^U, π^A, π^R), where π^i denotes the probability of choosing $i \in \{U, A, R\}$.

Consider the problem for states $s \geq 1$. Since the problem is stationary, we can restrict attention to stationary decision rules. Let Π_s be a probability vector governing choices in state s. $\Pi = (\Pi_0, ..., \Pi_S)$ denotes the complete decision rule. Let $V_s(\Pi, \theta)$ denote the expected discounted payoff, evaluated at the beginning of the period, when the addictive state is s, the parameters are given by θ, and Π is the decision rule. The functions $V_s(\Pi, \theta)$ correspond to the solution of the following system of equations, for $s = 0, ..., S$:

$$V_s(\Pi, \theta) = \pi_s^U V_s^U(\Pi, \theta) + \pi_s^A V_s^A(\Pi, \theta) + \pi_s^R V_s^R(\Pi, \theta); \tag{1}$$

where

$$V_s^U(\Pi, \theta) = u_s + b_s + \delta V_{\min\{S, s+1\}}(\Pi, \theta), \tag{2}$$

$$V_s^A(\Pi, \theta) = (1 - p_s)\left(u_s + \delta V_{\max\{1, s-1\}}(\Pi, \theta)\right) + p_s\left(u_s + b_s + \delta V_{\min\{S, s+1\}}(\Pi, \theta)\right), \tag{3}$$

and

$$V_s^R(\Pi, \theta) = u_s - c_s + \delta V_{\max\{1, s-1\}}(\Pi, \theta). \tag{4}$$

Since this is a standard time-consistent dynamic programming problem with a discrete choice space, there exists a unique optimal value function $V_s^*(\theta)$ and, generically, a unique optimal decision rule $\Pi^*(\theta)$ that maximizes it. Our object is to characterize $\Pi^*(\theta)$ and $V_s^*(\theta) \equiv V_s(\Pi^*(\theta), \theta)$ as a function of the parameter vector θ. Since the payoffs are linear in π we can, without loss of generality, focus on deterministic decisions (randomizations play a role only in the proof of theorem 2). In particular, action i is chosen in state s if and only if it is a solution to

$$\arg \max_{j=U,A,R} V_s^j \left(\Pi^*(\theta), \theta \right). \tag{5}$$

Let $\chi_s^*(\theta)$ denote the set of optimal actions in state s.

We sometimes assume that the substance in question has the following characteristic:

Definition A substance is *destructively addictive* if $b_k \geq 0$ for all k and, for $s = 0, ..., S-1$, $p_s \leq p_{s+1}$, $u_s \geq u_{s+1}$, $u_s + b_s \geq u_{s+1} + b_{s+1}$, and $c_s \leq c_{s+1}$.

Destructively addictive substances generate an immediate "high". The probability of entering the hot mode and the cost of rehab increase with the addictive state, whereas the immediate utility associated with both abstention and use declines. The marginal benefit of use may increase, decrease, or remain constant.

We conclude this section with a few remarks about the model. First, it reduces to a standard utility maximization problem when $p_s = 0$ for all s. As we will see in the next section, this is a reasonable assumption for substances that do not impact the brain with the same strength that drugs do. Second, in this model rehabilitation does not serve any purpose other than pre-commitment. When the DM is in the cold mode, he can check himself into a rehabilitation facility to prevent consumption even if he subsequently enters the hot mode.[15] Third, we have assumed that the DM can commit to rehabilitation only one period at a time. Since the DM starts each period in the cold mode, this is without loss of generality.

---

[15] In practice, however, rehabilitation programs may also teach self-management skills and desensitize addicts to cues. The practice of self-management skills may be able to alter the thoughts and images that the brain activates during the hot mode. Similarly, desensitization decreases the probability of entering the hot mode at any usage state. Both treatments can be modeled as a reduction in p_s that takes place after rehabilitation and/or therapy. Since the evidence suggests that these treatments are not 100% effective (Goldstein [2001, p.188]), the forces described in this model would still be at work.

3 Foundations

This model of addiction is based on one set of premises concerning decision-making, and another concerning addictive substances. These premises have foundations in psychology, neuroscience, and medicine which we describe in this section. Further details are provided in Bernheim and Rangel [2002].

Decision-Making I

We assume that decision-making results from two types of brain operations. First, the neocortex characterizes the decision problem by identifying a set of options and their consequences. Then, the visceral brain evaluates the pairs of identified options and consequences. Hard-wired mechanisms carry out the action with the "highest" evaluation. These brain operations are modeled here in reduced form.

This premise of the model is based on McLean [1977] theory of the triune brain, which holds that the brain is composed of three types of brain structures: the reptilian brain, the limbic brain, and the neocortex. The reptilian brain plays a central role in the regulation of basic survival behaviors (sustenance, shelter, safety, and sex). The limbic brain plays an essential role in the regulation of other emotions and in learning. The neocortex is the source of higher cognitive skills.[16]

Direct evidence concerning the exact roles of these specific brain structures in decision-making is still scarce.[17] Based upon the available information (some of which is discussed below), we hypothesize that the neocortex plays the leading role in characterizing the options available and their consequences, whereas one of the central roles of the visceral brain is the evaluation of these options. In crude but familiar terms, the visceral brain is the home of preferences, while the neocortex is an add-on cognitive processor that improves the quality of decision-making.

This hypothesis is supported by studies in comparative neuroanatomy. The primitive structures of the visceral brain are shared with countless species, but the size and scope of the human neocortex is unique. In humans, the neocortex accounts for about 33 percent of the encephalic mass, compared with 17 percent of chimpazees, and 3 percent for rats. These differences in brain structure are correlated with behavioral differences. For example, whereas human cocaine users experience cycles of binging and abstention (Gawin [1991]), rats allowed to self-administer cocaine, ignore hunger, reproductive urges, and all other drives, consuming the drug until they die (Pickens and Harris [1968] and Gardner and David [1999]).

---

[16]See any basic neuroscience textbook (e.g. Klein [2000]) for a more detailed description of these three structures.

[17]However, this is an extremely active area of research in neuroscience. See Panksepp [1998] and Rolls [1999] for recent reviews of this literature.

Decision-Making II

The second premise of the model is that, since the neocortex has limited computational abilities, shortcuts during the characterization stage are unavoidable. Plainly, the neocortex can only focus on a subset of the available actions and their consequences. Based on the evidence described below, we assume that a second role of the visceral brain is to assist in the selection of the alternatives and consequences that are considered during the characterization stage. In particular, we hypothesize that the visceral brain learns to associate visceral states with environmental patterns (i.e., cues), and that visceral states affect cognitive activation during the characterization stage.

As described in the introduction, the fact that the brain is capable only of incomplete characterization does not mean that mistakes must be made. Only the most clumsy numerical optimization algorithm would compute the maximum of a function by computing its value for every element in its domain.

Several related pieces of evidence provide support for this part of the model. First, to take a familiar example, emotions such as fear focus attention on the possibility of environmental threat (Janis [1967]) and on a limited number of "flight-or-flight" responses (Panksepp [1988], ch. 11). Second, Damasios' research group has studied the decision-making abilities of patients with damage on the ventromedial sector of the prefrontal cortex.[18] Injuries of this sort lead to abnormal (often muted) emotional responses, but the neocortex seems to be unaffected, since a standard battery of tests reveals no cognitive impairments. Although their "logical" reasoning facilities are intact, these individuals nevertheless exhibit an impaired capacity for sound decision making.[19] Based on these studies, Damasio has formulated the "somatic marker hypothesis," which holds that, in normal individuals, unconscious biases (visceral states in our language) guide behavior by influencing cognition. Damasio suggests that the brain uses somatic markers (i.e., visceral states) to simplify complex decision problems. In particular, pre-conscious visceral

---

[18]See Damasio [1994], Behara et al. [1997], Bechara et. al. [1994], and Bechara et. al. [1996].

[19]Consider a typical experiment comparing the decision-making performance of patients with brain damage versus normals. Agents need to choose cards from 4 decks. Cards from decks A and B generate a payoff of $100, cards from decks C and D generate a payoff of $50 dollars. Cards also generate random losses, which are large in the first two decks and small in the second two. The payoffs are chosen so that it is advantageous to choose from decks C and D. The participants do not know the payoff process or the number of rounds (equal to 100) in which they have to make choices. The experiment is interrupted at several times to question the subjects about their interpretation of what is going on. The main findings are: (1) normals began to choose advantageously before they can report the correct strategy, prefrontal patients continue to choose disadvantageously even after they know the correct response; and (2) normals begin to develop anticipatory skin conductance responses (SCRs) whenever they ponder a choice that turns out to be risky, even before they know the correct strategy, whereas prefrontal patients never develop the anticipatory SCRs, although some eventually realize which choices are risky.

processing focuses attention on a small set of alternatives, discarding the rest. In Damasio's theory, the ventromedial frontal cortices holds dispositional information, accumulated through previous experience, that is used to carry out this evaluation. A third source of evidence is given by the work of Mischel described below.

Decision-Making III

The third premise is that cues trigger strong visceral states which lead to characterization failure. During these states, which are subjectively experienced as a strong emotion such as cravings, cognitive activation is biased towards "hot" options and consequences. When the characterization bias is sufficiently strong, it can lead to mistakes. This premise is modeled by assuming that the DM can act in two modes $\mu = C, H$. In the cold mode there is perfect characterization: individuals consider every option and correctly forecast every consequence. In the hot mode the bias is so strong that the agent always end up using the drug. The cue process is modeled by the probability $p_s > 0$ of entering the hot mode.

Direct experimental evidence comes from the work of Mischel and coauthors on delay of gratification.[20] A subject (often a child) is placed in a room and is offered a choice between two prizes, an inferior and a superior one (one versus two candies). Subjects can obtain the inferior prize at any time by calling the experimenter, but must wait until the experimenter returns (usually around 15 minutes) to obtain the superior prize. Variations of this basic experiment reveal the following patterns. First, the child's ability to wait depends crucially on whether the inferior prize is visible. Merely covering the object significantly enhances the child's ability to wait. In fact, any stimulus that focuses the neocortex on the "tempting" features of the inferior prize increases the likelihood that the child will select it. This behavior is consistent with the hypothesis that seeing the candy triggers strong visceral states (cravings) during which the child's thoughts are restricted to a limited range of activities and outcomes. [21] Second, delay of gratification becomes less difficult with age, and is virtually impossible for children under 4. Since the visceral system is fully operational at birth, but the neocortical structures are not, this finding suggests that successful self-control depends on the neocortex. Third, the deployment of attention is a key determinant of self-control. Children are significantly more likely to wait if they are advised to distract themselves by thinking about something else,[22] or if

---

[20]See Mischel [1974], Mischel and Moore [1973], Mischel, Shoda, and Rodriguez [1992], and Metcalfe and Mischel [1999].

[21]Metcalfe and Mischel [1999] derive similar conclusions.

[22]Specifically, the experimenter suggested that while she was gone the child could think of anything that is fun: "You can also think about singing songs, or playing with toys, or anything that is fun to think of."

they are provided with a distracting toy, such as a slinky (even though children in a control group did not show any interest in the slinky). Most suggestively, telling a child *not* to think about the prize is counterproductive.[23] When the children are asked *not* to think about the prize, they repeatedly check whether or not they are thinking about it, activating in the process thoughts about the prize. This reduces their ability to wait. By contrast, the type of instructions that increase waiting times are those that ask the children to focus on something else.

Other evidence corroborates the view that cues influence behavior at least in part by inducing visceral states that affect cognitive deliberation. Shoppers tend to purchase more food at the grocery store when they are hungry, even though they know that the state of hunger is temporary (see Abratt and Goodey [1990]). It is natural to conjecture that hunger focuses attention on choices and consequences related to food and consumption. Salespeople often attempt to manipulate consumers' choices by using cues to stimulate visceral desires, even when the good in question is durable while the visceral state is not (e.g. selling a sports car by emphasizing its sex-appeal). Likewise, manipulation tactics used in interrogation and legal depositions are often designed to elicit specific responses with long-lasting implications by inducing temporary emotional states (Loewenstein [1996]).

The use of cue-triggered visceral states to influence cognition is an adaptive computational shortcut. This is nicely illustrated by LeDoux's work on the neural mechanisms of fear.[24] Information about the environment reaches the amygdala (a structure of the visceral brain) via two principal routes: a short "direct" route, and a long cortical route . Along the first route, information passes directly from the sensory thalamus to the amygdala without intermediate processing by the neocortex. Along the second route, information is sent from the sensory thalamus to various neocortical structures, where it is processed before proceeding to the amygdala. The short route is more primitive (in an evolutionary sense) and permits the organism to initiate rapid responses in critical survival situations. Though slower, use of the long route permits more deliberate responses. The existence of the short route implies that, in some circumstances, human behavior can result with little (if at all) cognitive deliberation. Consider the following example. While hiking through a park, an individual glimpses a long stick, resembling a snake, lying on the ground. This information first reaches his amygdala through the short route. The amygdala automatically initiates defensive responses, including autonomic changes such as increased blood circulation, endocrine changes such as the release of adrenaline, and neocortical changes such as heightened alertness. Before "fully thinking about it,"

---

[23]This variation of the experiments is closely related to the work of Wegner [1994].

[24]See LeDoux [1992,1993,1998] and also Davis [1992a,1992b].

the hiker stops short or leaps to safety.

Unfortunately, from the perspective of modern humans, this process was fine-tuned over an evolutionary time scale to the world of the hunter-gatherer. As a result, environmental stimuli in the modern world can trigger counterproductive responses. This is one of the central premises of evolutionary psychology (see Barkow, Cosmides, and Tooby [1995]). "Characterization failure" refers to circumstances in which environmental cues induce visceral states in which the neocortex focuses on alternatives that are undesirable from the individual's own perspective (either directly or by restricting attention to a limited range of consequences).

An important part of this premise is the assumption that cues can affect behavior through their impact on the characterization stage, even if they do not affect "preferences". Note that we do not argue that cues do not affect tastes. In fact, later on we will consider what happens when this force is added to the model. The key assumption is that, even if there are taste changes, cues also operate through an independent characterization failure mechanism. Two additional pieces of evidence provide direct support for this. First, in a series of experiments, Berridge has explored the possibility of disassociating "wanting" (incentive salience) from "liking" (experienced utility).[25] He has shown that stimulation of the lateral hypothalamus induces rats to make choices that they do not seem to like. The conclusion that the rats do not like the choices is based on observable behaviors, such as licking of tongues and legs, which in "voluntary choice" experiments correlates well with revealed preference. After the rats are trained, the onset and termination of behavior is perfectly determined by the experimenter's stimulation. These experiments suggest that there are parts of the brain that strongly influence choice without affecting hedonic evaluation.[26] Second, additional evidence comes from Mischel's experiments. Cognitive instructions that reveal no new information neutralize the effect of the exposure cue. A natural interpretation is that the experimenter's instructions to focus on the certain types of thoughts neutralize the ability of the cue to trigger thoughts about the hot features of the prize.

Decision-Making IV

Another premise of the model is that agents have an understanding of the characterization failure mechanism and take actions to manage it. For example, agents may avoid a particular action if it increases the probability of making mistakes due

---

[25] See Berridge [1995,1999], and Robinson and Berridge [1993]. Knutson et. al [2001a,b] provide related evidence using neuro-imaging experiments.

[26] Our theory does not assume that all the steps of the characterization process are accessible to consciousness. In fact, most of them are unlikely to be. Thus, similar mechanisms may be at work in the decision-making of humans and animals which may or may not posses consciousness.

to characterization failure in the future. In the model, self-understanding occurs in the cold mode when the neocortex perfectly forecasts the distribution of future consequences, and based on this chooses to take preventive action.

Once more, work on delay of gratification by Mischel and others provides an important source of evidence. Several studies (summarized in Metcalfe and Mischel [1999]) show that in the course of development children become conscious of self-regulation ("metacognitive awareness"). One example involves a variant of the experiments discussed in the preceding section. When children between the ages of 3 and 8 were asked whether they would prefer to have the prize exposed or covered, those under 4 exhibited no preference and were unable to justify their choice. In contrast, those over 5 preferred to wait with the prize hidden, and offered explanations that suggested some understanding of the principle that exposure to the prize influenced their attention.

Another source of evidence is that adults frequently choose to foreclose future options (pre-commitments). For example, they place alarm clocks across the room (Schelling [1984]), and publicly commit to deadlines. This has been widely interpreted as evidence for time-inconsistent preferences.[27] However, it is also the mark of an individual who is susceptible to cue-triggered characterization failure, and who possesses self-understanding. In particular, individuals foreclose options upon which they tend to focus during hot visceral states. Agents also engage in active cue-management, such as staying away from supermarkets when they are hungry, that has no effect on the budget constraint, but reduces the likelihood of experiencing cravings.

In the case of drugs, agents use agonist, antagonist, and "metabolic" medications to alter their future behavior. Methadone, an agonist, activates the same opioid receptors as heroin, and thus produces a mild high, but has a slow-onset and a long-lasting effect. This reduces the high produced by the consumption of heroin. Naltrexone, an antagonist, blocks the brain receptors activated by opioids, and thus also interferes with their ability to produce a high. Disulfiram, which is used to treat alcoholism, interferes with the liver's ability to metabolize alcohol; as a result, ingestion of alcohol produces a highly unpleasant physical reaction for a period of time. All of these treatments reduce the frequency of relapse. [28]

Addictive Substances I

An important question that any theory of addiction must answer is why drugs generate consumption patterns that are so different from other substances and so

---

[27]See, for example, Laibson [1997], O'Donoghue and Rabin [1999], and Thaler and Shefrin [1981].
[28]See O'Brien [1997] and Goldstein [2001].

pathological. After all, the brain processes described above are at work in all decision problems. A central premise of this theory is that drugs have an unprecedented ability to induce characterization failure. This is modeled as a positive and increasing probability p_s of entering the hot mode in which they always attempt to consume the substance.

The consensus in neuroscience is that the single most important characteristic of these substances is their impact on the pleasure/reward circuitry (PRC) of the visceral brain. Gardner and David [1999, p.102] summarize as follows the consensus view: "The single essential commonality of the addicting drugs is the neurobiological one - their ability to acutely enhance the *PRC* of the brain. The hypothesis that addictive drugs act on these brain mechanisms to produce the intense subjective pleasure/reward that constitutes the "hit" or "rush" or "high" sought by drug users is, at present, the most compelling hypothesis available on the neuro biology of drug abuse."[29] Some drugs, for example cocaine, stimulates the PRC by blocking the reuptake of neurotransmitters like dopamine, which increases their levels in the synapses. Others, such as heroin, stimulate the release of similar neurotransmitters.[30]

This property of drugs is important because the evidence suggests that the PRC plays a key role in decision-making.[31] First, studies with human subjects have shown that electrical stimulation of the PRC produces intense feelings of pleasure and euphoria (Heath [1964]). Second, as summarized by Gardner and David [1999], direct stimulation of the PRC is "one of the most powerful rewards known to biology, rivaled only by the rewards of the most intensely addictive drugs (e.g., cocaine)".

The PRC seems to be play a central role in the process of learning to make good decisions. Although, the evidence on how exactly the PRC accomplishes this

---

[29]Of the addictive substances listed in footnote 1, hallucinogenics (or psychedelics) are the only substances which do not seem to produce intense stimulation of the PRC. Instead, hallucinogenics act on a (Goldstein [2001, p.231]) "subtype of serotonin receptor which is widely distributed in areas of the brain that process sensory inputs." Interestingly, laboratory animals and humans learn to self-administer the same set of substances, with the possible exception of hallucinogenics. (See Gardner and David [1999, p.97 and 98]). Since our understanding of psychedelics is still fairly incomplete, there is some debate about whether they belong to the list of addictive substances (see Goldstein [2001, ch. 14]).

[30]See Klein [2000, ch.5] and Goldstein [2001] for more details about the various mechanisms through which drugs impact the brain.

[31]The existence of this brain structure was established by Olds and Milner [1954]. They showed that rats learn to return to portions of an experimental environment in which they have been receiving direct electrical stimulation to the PRC. When provided with opportunities to self-administer by pressing a lever, they acquire the habit rapidly, giving themselves approximately 5,000-10,000 "hits" during each one hour daily session, and ignoring other stimuli, including food, water, and rats of the opposite sex. In a variation of the experiment, after several weeks of self-administration the rats encounter a new situation: to reach the lever they must cross a region in which they experience painful electric shocks. After a few moments of trial and hesitation, the rats incur the cost (see Gardner and David [1999] for a summary of these experiments).

is still scarce, we hypothesize that it operates through two mechanisms. First, the "utility" output of the PRC is used to update the evaluation function of the visceral brain. For example, if a few times I become sick after eating strawberries, my taste for them will decrease and I might even develop a visceral reaction against them from that moment on.[32] Plainly, this leads to evolving preferences. If the learning process takes into account the state of the world (for example if I get sick only in certain states), this leads to state-contingent preferences. Second, the "utility" output of the PRC is also used to update the process of cognitive activation that takes place during the characterization stage. This is an essential part of the process given the neocortex's computational limitations. Its role is to increase the likelihood that the most "hedonically" relevant information will be activated in future decision problems. Since the state of the world is likely to affect the agent's situation, this learning process leads to a state-contingent probability of cognitive activation so that, for example, I activate thoughts related to fighting only when my brain has detected an "anger state" where such information is likely to be useful.

The brain has acquired these two learning channels because they are adaptive. As long as only adaptive outcomes are able to activate the PRC, the algorithm generates acceptable average outcomes. So what is special about drugs? Based on the available evidence we hypothesize that, as a result of the unprecedented power with which drugs stimulate the PRC, their administration leads to a malfunction of the second learning channel.[33] In particular, during the characterization stage, the brain "learns" to over-activate the option of consuming the drug and images about the "high", at the expense of other options and consequences that are hedonically relevant. This leads to characterization failure: agents sometimes mischaracterize the problem and choose alternatives that do not generate highest possible experiential utility. Robinson and Berridge [1993] reach a similar conclusion.

Several pieces of evidence provide support this hypothesis. First, Gawin [1991] reports that, during binges and cravings, virtually all of the addict's thoughts are concentrated on the substance.[34] Second, the work of Berridge and coauthors discussed above shows that it is possible to stimulates rat's brains to get them to choose things that they do not seem to like. Third, Vorel et. al. [2001][35] have shown that stimulation of the ventral subiculum (a region associated with memory) can rein-

---

[32]This example is taken from Romer [2000].

[33]In order to trigger this mechanism, a substance need to impact the drug with sufficient strength. The technology of administration and size of the dosage plays a key role in this. Consider the case of cocaine. Peruvian peasants have self administered cocaine by chewing coca leaves. This method of administration leads to a slow and steady levels of the substance in the brain. By contrast, snorting cocaine or smoking it (crack), lead to spikes. Studies have shown that the same population becomes "addicted" to snorted cocaine, but not to chewing coca leaves. See Goldstein [2001, ch. 11].

[34]Goldstein and Kalant [1990] make a similar assessment.

[35]See also Vorel and Gardner [2001] and Holden [2001a,b] for a non-technical discussion.

state self-administration in rats that have "kicked a cocaine addiction", while direct stimulation of the medial forebrain bundle (a region of the PRC) does not. These have found that stimulation of the memory centres can trigger something analogous to strong cravings. Also, Ungless et. al. [2001] have shown that similar cellular mechanisms may be at work in memory and addiction.[36] These last two findings suggest a relationship between drug consumption and the process of memory activation.

Additional supporting evidence, although of a more circumstantial nature since it is also consistent with taste-shocks, is given by the well-documented fact that cue-triggered cravings play a central role in addiction. First, studies of drug consumption patterns establish that both short-term abstinence and long-term recidivism are common (Hser, Anglin, and Powers [1993], Harris [1993], and Goldstein [2001]). Second, recidivism rates are especially high when addicts are exposed to cues related to their past drug consumption (O'Brien [1976]). For this reason, drug treatment programs advise recovering addicts to move to new locations, or at least to avoid the places where previous consumption took place. Third, long-term usage is considerably lower among those who experience significant changes of environment. Robins [1975,1993] found that Vietnam veterans who were addicted to heroin and/or opium at the end of the war experienced much lower relapse rates than other young male addicts during the same period. A plausible explanation is that veterans encountered fewer environmental triggers (familiar circumstances associated with drug use) upon returning to the U.S. Finally, a recovering addict is significantly more likely to "fall off the wagon" if he receives a small taste of his drug-of-choice (Goldstein [2001]). This phenomenon, known as "priming," suggests that the taste serves as a powerful cue that activates cravings.

It is important to emphasize that the ability of chemical substances to "over stimulate" the PRC depends on the technology of administration. Consider the case of cocaine. For centuries, peruvian peasants have self administered cocaine by chewing coca leaves without becoming addicted. This method of administration leads to a slow and steady levels of the substance in the brain. By contrast, snorting cocaine or smoking it (crack) lead to spikes. Studies have shown that same peruvian peasants who do not become addicted to chewing coca leaves become addicted to snorted cocaine. Similarly, differences on "drug metabolisim" affect consumption patterns. For example, cocaine produces a shorter lived impact than heroin, which might be related to its shorter administration cycle. See Goldstein [2001, ch. 6 and 11].

---

[36]Helmuth [2001] provides a non-technical discussion of these findings.

The ability of drugs to activate the PRC is an evolutionary fluke. Only a handful of substances generate the consumption patterns that we associate with addiction. For the most part, the evolutionary processes that produced the brain's algorithm for selecting decision-making shortcuts did not take place in an environment where these few substances were readily available. As described above, animals ignore other basic drives (such hunger, thirst, pain, and sex) to self-administer these substances, sometimes to the point of killing themselves. This is clearly not adaptive.

Addictive Substances II

A final premise of the model is that drugs generate tolerance and physical dependence. Tolerance results from the interaction of the addictive substances and the body's homeostatic mechanisms. Every organism constantly relies on these mechanisms to maintain itself in a "normal" state (Koob and Le Moal [1997]). For example, excessive consumption of sugar increases the blood sugar level and triggers the release of insulin, which brings the sugar level back to normal. Since addictive substances destabilize the brain's natural operations, as well as other physical processes, homeostatic mechanisms come into play. For example, when exposed to a drug that mimics the release of natural opioids, thus inducing activation in the PRC, the brain reacts by decreasing the level of naturally occurring opioids. This creates tolerance and physical dependence: as the addiction progresses, the average activation of the PRC decreases (u_s declines with s), and the same dose of the drug no longer generates the same sense of well-being ($u_s + b_s$ also declines). Also, an addict who stops taking the substance experiences the highly unpleasant symptoms of withdrawal, including temporary illness and anhedonia. For many substances, the withdrawal symptoms worsen with the addictive state (b_s increases in s). [37]

In response to the unprecedented impact that drugs have on the PRC, the body also deploys feed-forward mechanisms, which arise from the interplay of learning and homeostasis. Given the highly destabilizing impact of drugs, it is advantageous for the brain to recognize cues that are related to use, and to prepare for their effects. Specifically, the brain reduces the level of activation of the PRC in anticipation of the increased stimulation that will result from the consumption of the drug. This reduces the user's baseline sense of well-being (producing a cue-triggered decrease in u_s), and, depending on the substance, user, and level of addiction, it may even

---

[37]In some cases, sufficiently addicted agents experience withdrawal symptoms that peak after a few dates of abstinence. This effect cannot be captured in a model with a one-dimensional state variable, but could be incorporated by defining a second state variable that measures the number of periods since the drug was last used. The introduction of this complication would generate more realistic consumption and welfare patterns, but it would not alter affect the insights derived here.

make the drug more attractive (a cue-triggered increase of b_s).[38]

One can think of feed-forward mechanisms as a neurological foundation for state-contingent utility. Since our focus in this paper concerns the implications of characterization failure, our basic model and analysis does not incorporate feed-forward responses. However, the two mechanisms are not incompatible. In fact, in section 9 we discuss the positive and normative implications of introducing cue driven taste-shocks into the model.

4   Basic Comparative Dynamics

In this section we characterize the comparative dynamics of the model. Usually one needs to solve for $\Pi^*(\theta)$ and $V^*(\theta)$ simultaneously. In this model, however, it is possible to derive some basic properties of $V^*(\theta)$ without knowledge of $\Pi^*(\theta)$, and then to use these properties to characterize $\Pi^*(\theta)$.

We start with a characterization of the optimal decision rule based on the value function. Suppose that $V^*(\theta)$ is known. Then it follows from (1) to (5) that the optimal decision rule for state $s \geq 1$ has the following form:

$$U \in \chi_s^*(\theta) \ \text{ if and only if } V_{\max\{1,s-1\}}^*(\theta) - V_{\min\{S,s+1\}}^*(\theta) \leq \frac{b_s}{\delta} \qquad (6)$$

$$A \in \chi_s^*(\theta) \ \text{ if and only if } V_{\max\{1,s-1\}}^*(\theta) - V_{\min\{S,s+1\}}^*(\theta) \in \left[\frac{b_s}{\delta}, \frac{b_s + c_s/p_s}{\delta}\right] \qquad (7)$$

$$R \in \chi_s^*(\theta) \ \text{ if and only if } V_{\max\{1,s-1\}}^*(\theta) - V_{\min\{S,s+1\}}^*(\theta) \geq \frac{b_s + c_s/p_s}{\delta} \qquad (8)$$

The optimal choice in state 0 is straightforward. Since $p_0 = 0$, the DM never chooses rehab in this state. If the DM uses the substance in state 0, his payoff is $u_0 + b_0 + \delta V_1^*(\theta)$. If he abstains his payoff is $u_0 + V_0^*(\theta)$. Thus, $U \in \chi_0^*(\theta)$ if and only if

$$V_0^*(\theta) - V_1^*(\theta) \leq \frac{b_0}{\delta}. \qquad (9)$$

Conditions (6) through (9) imply that, once the value function is known, the characterization of the optimal decision rule is straightforward and intuitive. Since usage today increases the usage state by one unit, and non-usage decreases it by

---

[38]See Laibson [2001] for a more detailed discussion of the foundations behind feed-forward mechanisms.
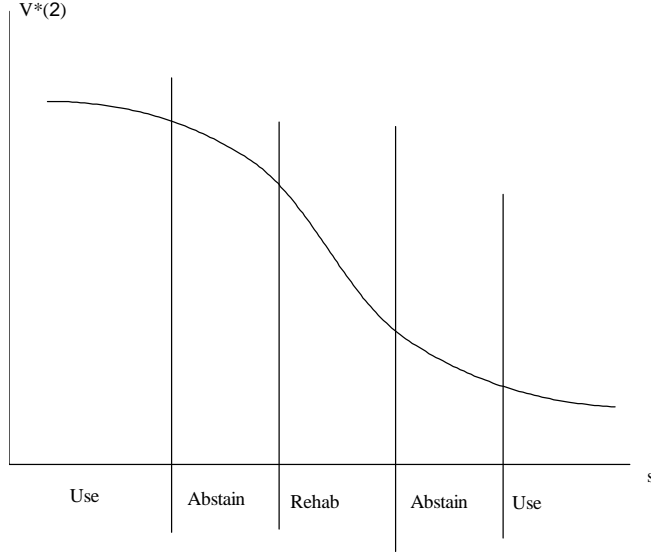
Figure 1: Characterization of the optimal decision rule

one unit, $\delta(V^{*}_{\max\{1,s-1\}}(\theta) - V^{*}_{\min\{S,s+1\}}(\theta))$ represents the future cost of using the substance today. Thus, the DM uses the substance if the benefit of usage, b_s, is large compared to the cost.

Figure 1 illustrates this. Consider a substance for which the value function is decreasing in s. $V^{*}_{\max\{1,s-1\}}(\theta) - V^{*}_{\min\{S,s+1\}}(\theta)$ measures the "steepness" of the value function. Thus, the DM uses the drug in states for which the value function is flat, enters rehabilitation in states for which it is steep, and attempts to abstain for intermediate cases.

We now turn our attention to the properties of the value function.

Theorem 1: *For any destructively addictive substance, $V^{*}_{s}(\theta) \geq V^{*}_{s+1}(\theta)$.*

In other words, for a destructively addictive substance, the DM's optimized well-being declines monotonically with the addictive state.

Theorem 2: *For all s, $V^{*}_{s}(\theta)$ is continuous in θ, weakly increasing in b_k and u_k, and weakly decreasing in p_k and c_k.*

This result is intuitive. When b_k or u_k increase, or when c_k decreases, the same decision rule must yield weakly higher valuations for every state s; hence, $V^{*}_{s}(\theta)$ cannot decline. A subtler argument is needed to establish the monotonicity with respect to p_k.

We make use of the fact that there is a natural ordering of the three actions. In particular, we say that action U involves *higher usage* than A or R, and A involves higher usage than R. We also say that *usage in state s is (weakly) increasing* in a parameter if an increase in the parameter leads to (weakly) higher usage.

Our next result provides a useful tool for characterizing comparative dynamics.

Lemma 1: *Consider a move from θ to θ' such that: (1) $\theta'_k \neq \theta_k$, (2) $\theta'_j = \theta_j$ for $j \neq k$, and (3) $V^*_s(\theta') \geq V^*_s(\theta)$ for all s. Then:*

(i) For $s < k$, usage in state s weakly increases,

(ii) For $s > k$, usage in state s weakly decreases.

Lemma 1 is also intuitive. Consider a change in state k's parameters that makes the state more attractive. Intuitively, the DM should be more inclined to take actions that lead to state k; i.e., he will exhibit a greater tendency to use the substance in lower states, and be less inclined to use it at higher states.[39]

The next result provides a fairly complete characterization of comparative dynamics:

Theorem 3: *Usage in state s is:*

(i) weakly increasing in b_k and u_k, and weakly decreasing in p_k and c_k, for $k > s$,

(ii) weakly decreasing in b_k and u_k, and weakly increasing in p_k and c_k, for $k < s$,

(iii) weakly decreasing in p_k and weakly increasing in c_k, for $k = s$.

This theorem establishes that use in state s is monotonic with respect to most parameters and indicates the direction of the effect. The only two exceptions concern the effects of b_k and u_k on usage in state k, which depending on the parameter values can be positive or negative.[40] Interestingly, while changes in p_k or c_k affect usage in states $s \neq k$ in the same direction, they have the opposite effect in state k.

---

[39]This intuition is somewhat naive because it ignores the effects of the change in θ_k on the value function for states $s \neq k$. The remarkable feature of lemma 1 is that, to characterize certain aspects of comparative dynamics, one need only know the direction of the effect of a parameter change on the value function. This is surprising because optimal choices depend on the *differences* in the value function across states, rather than on the *level* of the value function in any given state (equations (6) through (8)).

[40]Consider first the effect of b_k. Let $S = 4$ and the cost of rehab be arbitrarily high so that the agent always uses or abstains. Consider two vector of parameters θ and θ' with the following features. $b_s = b'_s = 0$ for all s except for b'_3 described below. $u_0 = u'_0 = u_1 = u'_1 \approx -\infty$ and $u_s = u'_s = 0$ for all other s. Then *an* optimal decision rule at θ is given by (U, U, U, A, A), where the kth component denotes the decision state k. Use is optimal in states 0 to 2 to avoid the bad outcomes u_0 and u_1; abstention is optimal in states 3 and 4 because the agent is indifferent between

Theorem 3 underscores the fact that policy changes can have complicated behavioral effects. For example, a policy that reduces usage in the late stages of addiction by decreasing the cost of rehabilitation may also increase use and discourage rehabilitation at earlier stages of addiction. This effect may be particularly strong when subsidized rehabilitation is only offered to the most serious addicts. Indeed, an increase in the cost of rehabilitation for highly addicted states may unambiguously reduce both total use, and use at higher states, by inducing a shift to rehabilitation at an earlier state. This is an argument for early intervention. Similarly, a reduction in p_k reduces unintended usage, but it increases intentional usage among new users.

Theorem 3 provides conditions under which changes in the parameters produce monotonic changes. By contrast, the next result catalogs circumstances for which parameter changes have no effect on the optimal decision or the value function.

Theorem 4: $\chi_s^*(\theta)$ and $V_s^*(\theta)$ are invariant with respect to:

(i) p_k and c_k when $U \in \chi_k^*(\theta)$

(ii) any increase in p_k and c_k or any decreases in b_k and u_k when $k > s$ and $R \in \chi_n^*(\theta)$ for some $n \in \{s, ..., k-1\}$

(iii) any increase in p_k and c_k or any decreases in b_k and u_k when $k < s$ and $U \in \chi_n^*(\theta)$ for some $n \in \{k+1, ..., s\}$

(iv) local changes in p_k and b_k when $\chi_k^*(\theta) = \{R\}$

(v) local changes in θ_k when $k > s$ and $\chi_n^*(\theta) = \{R\}$ for some $n \in \{s, ..., k-1\}$

(vi) local changes in θ_k when $k < s$ and $\chi_n^*(\theta) = \{U\}$ for some $n \in \{k+1, ..., s\}$

(vii) local changes in c_k when $\chi_k^*(\theta) = \{A\}$

Furthermore, if $U \in \chi_s^*(\theta)$, then $\exists \varepsilon > 0$ such that $R \notin \chi_s^*(\theta')$ for $|\theta - \theta'| < \varepsilon$. Likewise, if $R \in \chi_s^*(\theta)$, then $\exists \varepsilon > 0$ such that $U \notin \chi_s^*(\theta')$ for $|\theta - \theta'| < \varepsilon$.

Parts (iv) to (vii) exploit the discreteness of the choice set, and identify conditions under which a small changes in parameters has no effect on choice. Parts (i) to (iii) are more substantive, and merit further discussion. Part (i) says that, if use is optimal in state k, then no (global or local) change in p_k or c_k can affect behavior in state k. Strikingly, one cannot induce a state k user to enter rehabilitation by

---

states 2, 3, and 4. Now consider a vector θ' where $b_3' = b_3 - \varepsilon$, with $\varepsilon > 0$ but small, and p_3 is large, so that there is a significant probability of use in state 3. In this case the optimal decision rule is (U, U, U, U). Therefore, a reduction in b_3 can increase usage in state 3. Now consider a move from θ to θ'' where b_3'' is quite large, but bounded above. In this case the optimal decision rule is (U, U, U, A), which has the opposite effect.

Now consider the effect of u_k. If the DM uses in states $k + 1$ and $k - 1$, then a small increase in u_k will raise $V_{\max\{1, s-1\}}^*(\theta)$ while leaving $V_{\min\{S, s+1\}}^*(\theta)$ unaffected, which reduces state k use. However, if the DM enters rehabilitation in states $k + 1$ and $k - 1$, the opposite is true.

reducing the state k rehabilitation cost. Anyone who might be attracted by cheaper rehabilitation would already be attempting to abstain.[41] Similarly, one cannot induce a state k user to abstain by reducing the state k probability of entering the hot mode. However, a state k user might switch to abstention or rehabilitation in response to changes in rehabilitation costs and probabilities for *other* states of addiction. Part (ii) says that a (global or local) increase in p_k or c_k, or a (global or local) decrease in u_k or b_k, have no effect on the usage or welfare of an earlier state s when there is an intermediate state $n \in \{s, ..., k-1\}$ for which rehab is optimal. The intuition is as follows. By theorem 3 we know that increases in p_k or c_k, and decreases in u_k or b_k, weakly decrease usage in lower states. This implies that the optimal choice in state n must still be R, and thus the optimal value functions for states s to n remain unchanged. But then, the optimal choice at states s to n also remains unchanged. The logic behind part (iii) is similar.

5   Patterns of Use

In this section we study how changes in the exogenous characteristics of substances (such as the probabilities of entering the hot mode, the short and long term costs of using the substance, and the intensity of the high) affect the consumption patterns of a given group of users. Our focus is necessarily qualitative since the model is too stylized to generate meaningful quantitative predictions. All of the consumption patterns described below are based on robust numerical examples which, given the length of the paper, are omitted.

Case I. Consistent use: Caffeine. To illustrate the simple case of consistent use, imagine an idealized substance for which $(u_s, b_s) = (u_0, b_0)$ for all s, with $b_0 > 0$. Let Π^u denote the behavioral rule that prescribes use in every state. Plainly, $V_s(\Pi^u, \theta) = (u_0 + b_0)/(1 - \delta)$ for all s. But then $V_{\max\{1,s-1\}}(\Pi^u, \theta) - V_{\min\{S,s+1\}}(\Pi^u, \theta) = 0 < b_0/\delta$. This in turn implies that use is optimal in every state (see condition (6)). Intuitively, the DM will never refrain from using a substance that confers positive immediate benefits without sufficiently adverse effects on future well-being.

More generally, consistent use will be optimal provided that b_s is always positive and u_s and $u_s + b_s$ do not decline too rapidly from one state to the next. The aggregate decline from state 0 to state S may, however, be substantial.

Caffeine exhibits the basic properties of this example (see Goldstein [2001,ch.13]). It is a stimulant which produces a "high" in every state ($b_s > 0$). It generates tolerance and withdrawal and, if consumed in large amounts, has long-term adverse

---

[41]However, one can induce a state k user to accept rehabilitation by paying hin to enter rehabilitation, a possibility we have ruled out by assuming that $c_k \geq 0$.

health consequences such as anxiety and sleep disturbances that develop only gradually (u_s and $u_s + b_s$ both decrease gradually with s).

Case II. Non-use: Volatile Solvents. Consider a destructively addictive substance, such as volatile solvents, that produces a relatively mild "high" but inflicts significant and increasing health damages. In this case the value function is steeply decreasing in the addictive state. By condition (9), the DM chooses not to use in state 0 as long as b_0 is sufficiently small. In addition, if b_s is sufficiently small for $s \geq 1$, the DM would never use the substance if, for "exogenous" reasons, he were to find himself at an addictive state $s \geq 1$. In particular, when c_s is sufficiently close to zero for all s, or p_s is sufficiently high, the DM will choose to rehabilitate in all states with a positive probability of entering the hot mode. This rules out both intentional and accidental use.

Case III. Intermittent use and recidivism: Cocaine. Consider a substance, such as cocaine, that quickly generates tolerance. Cocaine is relatively innocuous at low frequency of usage, but begins to take a significant and increasing toll on the health and life of the user with increased use (see Goldstein [2001,ch.11]). In this case, the u and $u+b$ profiles are relatively flat for low states, but decline with s at increasing rates for sufficiently advanced stages of addiction. These parameters generate value functions that are relatively flat in low states, but decline rapidly in high states. The result is an initial region of use followed by a region of abstention (if the rate of decline is more moderate) or rehabilitation (if the value function declines sufficiently rapidly). In this way, the model generates either intermittent use or recidivism.

Intermittent use corresponds to a pattern such as "U, U, U, A, A, A" (where the first U refers to consumption in state 0, the second U refers to consumption in state 1, and so forth). From state 0, the DM uses in three consecutive periods. In the fourth period, he reaches state $s = 3$ and attempts to abstain. As long as abstention is successful, he alternates in subsequent periods between use and abstention. Abstention is not, however, always successful. In some instances, the DM may enter the hot mode and binge in consecutive periods, potentially reaching states 4 and 5. With sufficiently prolonged successful abstention, he emerges from the unintended binge, returning to state 2, at which point he resumes intermittent use. Over time, the DM settles into a stochastic steady state, distributing his time between states 2 to 5.

A slight variation of the pattern just discussed is *recidivism*: "U, U, A, A, R, R." This pattern arises when the rate of decrease of u_s or $u_s + b_s$ accelerates with s. In this case, recreational use occasionally leads to unintentional binges. When the binges go too far (two consecutive periods in this example), the individual elects

rehabilitation. In other words, the DM first attempt to kick the habit by himself (through abstention), but resorts to rehabilitation if successive attempts to abstain meet with little success. Note that, since rehabilitation is never permanently effective, the DM remains caught between the first R and the U that precedes it. This pattern of recidivism is quite common in practice.

Case IV. Resignation: Crack. Now consider a drug with characteristics similar to cocaine, except that beyond some threshold state s^*, the probability of entering the hot mode, p_s increases quite fast and is close to one.[42] This generates a "backward-S-shaped" value function similar to the one depicted in figure 2. Depending on the slope of the value function, and the cost of rehabilitation, it leads to *resignation* consumption patterns of the form "U, U, A, A, A, U, U" or "U, U, A, R, A, U, U". Consider the first pattern. The DM uses regularly until he reaches some state at which he attempts to abstain. If successful, he uses intermittently. However, if the DM is unlucky, the attempt is unsuccessful, and the addictive state rises. The DM continues the attempt to abstain in order to reduce the addictive state. Following a sufficiently long string of bad luck, however, the DM simply gives up and goes back to using the substance. This corresponds to resigning oneself to failure, giving up, or "letting oneself go".

Case V. Sophisticated Junkies. Now consider a destructively addictive substance for which the u profile is relatively flat, the cost of rehabilitation is small, and b_s is large up to some threshold state s^*, beyond which it decreases sharply. This generates a consumption pattern of the form "$U, U, U, R, ...$" The DM chooses use in states 0 to s^*, and rehabilitation in states $s^* + 1$ to S; he resorts to rehabilitation without first attempting to abstain on his own, which leads to cycling between use and rehabilitation. The DM enters rehabilitation in each instance without any desire of staying clean; he knows that he will resume using the substance upon release from rehabilitation, fully expecting to enter rehabilitation once again.

Though this pattern may initially seem peculiar, it actually occurs in practice. According to Massing [2000], serious heroin addicts are known to behave this way when repeated use dilutes the "high". Note the perverse role that characterization failure plays in this pattern. The agent checks into rehab because there is a risk that he might not be able to abstain on his own, decreasing even further his ability to experience the high.

Case VI. Nicotine. Nicotine is highly addictive, and produces strong with-

---

[42]Crack is prepared from cocaine by mixing it with baking soda and water, and then boiling it. This has two important consequences. First, crack can be smoked, which allows the brain to absorb the substance more efficiently, and leads to a quicker and more intense high. Second, crack is significantly cheaper, which leads to a pattern of more frequent administration. We conjecture that these two differences are responsible for the faster rate with which brains exposed to crack learn to enter the hot mode.

drawal symptoms and long-term health damages.[43] In this regard is fairly similar to cocaine. The key difference between the two substances seems to be the speed at which health and socioeconomic damages quick in.[44] Nicotine users remain functional even at high stages of addiction and develop illnesses only in the long-run. As a result, as in the case of caffeine, a fully informed user of nicotine could decide to use constantly.

The fact that millions try to quit tobacco unsuccessfully every year (Harris [1993], Goldstein [2001], Goldstein and Kalant [1990]) suggests that additional mechanisms are essential to understand the consumption of this substance. One possible explanation is that the marginal benefit of smoking decreases with age. For example, smoking is a social activity and agents go out less when they start a family. In this case, agents first-best consumption plan would be to smoke in youth and abstain in middle and old age. Characterization failure plays a role at mid-life when agents try to abstain but find themselves triggered into the hot mode by a multitude of environmental cues. Peer pressure and periods of high stress could have similar long-term consequences since they both increase the value of smoking temporarily, but not in the long-term.

Case VII. Designer Drugs. Given the speed with which our understanding of the brain is increasing, it is likely that new drugs will appear in the near future. In the classic novel "Brave New World", Aldox Huxley [1998] explores the effect on society of a drug called "soma", which makes people feel blissfully happy with their circumstances, however humbly or grand. Huxley's soma can be modeled as a substance that generates an arbitrarily large $u_s + b_s$ at every state of addiction. If such a substance (or device) could be designed, the brain would seek it both in the hot and cold modes, at the expense of any other goal.

Interestingly, regardless of whether or not it triggers the hot mode, soma does not generate behavioral problems since consumption is always optimal. In fact, if one measures welfare based on the experiential utility that results from the visceral brain, soma would be an economic "holy Grail". By contrast, characterization failure creates behavioral problems for substances such as cocaine because it leads to unwanted consumption. The example of soma raises deep questions about what is the right notion of welfare that we do not address in this paper.

---

[43]See Goldstein [2001] for details.

[44]The difference in the costs of cocaine and nicotine use is due, in part, to the fact that nicotine is legal but cocaine is not.

6 Explaining the Stylized Facts

In this section we show the role that characterization failure plays in explaining the nine stylized facts listed in the introduction. In appendix A we show that facts 1 to 7 can also be explained by some of the alternative theories that have been proposed. However, as summarized in table 1, no existing model can explain all the facts, and facts 8 and 9 cannot be explained by any of the alternative theories.

Facts 1 and 2. Short-term abstention and cue-triggered recidivism. Traditional explanations of addiction have emphasized the role of tolerance and withdrawal symptoms.[45] Although the evidence overwhelmingly demonstrates that these forces are at work, this cannot be the main part of the story. It is difficult to understand why agents would frequently abstain successfully for short periods of time, sometimes experiencing extreme pain and discomfort during withdrawal, only to resume use at a later date after the withdrawal symptoms have disappeared. Characterization failure provides a simple explanation.

The visceral brain learns to associate environmental cues with the "high" produced by the drug. As a result, cues trigger strong visceral states that are experienced as cravings. During a cravings episode, the neocortex provides a biased characterization of the decision problem: it generates a sample of options and consequences that is biased towards the "high" produced by the drug. In some cases, this leads to consumption that would not have taken place had the brain characterized the decision problem correctly.

Importantly, addicts only have partial control over their exposure to cues. Although many stay away from obvious cues such as bars or the places where they consumed the substances, they cannot control exposure to all cues. The power of these substances is so strong that a myriad of (unconscious) cues, from a smell to a T.V. commercial, can trigger a visceral memory of the high. This explains the long-rates of long term recidivism.

In this paper we have specified a simple cue process in which the probability of entering the hot mode depends only on the addictive state s. In a more realistic specification, the probability of being triggered in period t would be given by $p(s_t, \mu_{t-1})$; with $p(s_t, H) > p(s_t, C)$. This straightforward extension provides an explanation for the tendency of users to binge.

It is important to emphasize that cue-triggered taste shocks driven by feedforward mechanisms provide a related explanation for these two facts (see table 1). However, as discussed in appendix A, taste shocks cannot explain the other stylized

---

[45]See appendix A for a discussion of economic theories of addiction based on tolerance, and Koob and LeMoal [1997], Goldstein [2001], and Robinson and Berridge [1993] for a discussion of "non-economic models" based on tolerance.

facts.

Fact 3. **Differences across substances.** In the previous section we have shown that, subject to the level of abstraction, characterization failure provides a plausible mapping from the exogenous characteristics of drugs to consumption patterns. It is worth elaborating on the role that characterization failure plays in generating the different patterns.

First, characterization failure plays no role for substances involving consistent use, inasmuch as the DM elects usage in the cold mode for all states (see theorem 4, part i, and the cases of caffeine and soma). Cues may still trigger strong cravings, which may distort the cognitive operations of the brain during the process of choice, but it does not alter the choice itself. This case arises when the cost of usage develops gradually, if at all, so that the "marginal impact" of one more hit is always minor, or when the marginal benefit of usage is sufficiently large.

Second, characterization failure plays an important role for substances in which the first-best consumption dictates an initial period of usage followed by abstention. For example, the optimal path for cocaine involved recreational and sporadic use, and the one for nicotine involved an initial period of usage followed by abstention starting at mid-life. The problem, of course, is that once the brain has been in sufficient contact with the substance, cues can trigger the hot mode and users might find themselves carrying out unwanted use. As a result, temporary taste-shocks such as peer-pressures or stress can induce long-term unwanted conseumption.

Third, characterization failure also plays a role in explaining non-use. Users would abstain from experimental usage of substances for which p_s is large even after a single hit to avoid the risk of becoming addicted. This explanation fits well with casual observation: many people refrain from experimenting with "hard drugs" because they fear that frequent use could undermine future self-control and produce a downward spiral of addiction.

Finally, as explained further below, characterization failure also plays an essential role in explaining rehab.

Fact 4. Differences across users. This theory provides a simple explanation for why only a fraction of the agents who experiment with drugs early in life become addicts. Plainly, the brains of addicts and non-addicts are different: the first group is much more succeptible to characterization failure. An important open question is to what extent is the succeptibility to characterization failure a general trait, and to what extent it is substance specific, so that agents are more succeptible to some drugs than others. Drug-specific sensitivities are plausible since different drugs activate the PRC of the brain through different channels.

It is important to emphasize that these explanations are testable. First, if there

is a general proneness to characterization failure, it should be possible to identify children that are more likely to become addicted later on using procedures similar to the experiments of Mischel described in section 3. Consistent with this prediction, studies have shown that having one addiction decreases the threshold for developing another (Holden [2001]).[46] Second, using brain imaging technologies such as functional magnetic resonance imaging (fMRI) and Positron emission tomography (PET), it should be possible to test if, prior to any exposure to drugs, the brain's of certain groups react more strongly to certain substances, and to see if this trait is correlated with behavioral addiction.[47]

Fact 5. Prices and information matter. In this model, users respond to standard economic incentives such as prices and information because they enter the cold mode with certain frequency and, at those times, they make decisions as in the standard model. Furthermore, in a more complex model of characterization failure developed in Bernheim and Rangel [2002], agents are responsive to prices and information even in the hot mode. Plainly, prices can affect decision making even when agents only consider a subset of options and consequences, as long as they affect the value of the options and consequences that are considered.

Fact 6. Pre-commitment behavior. In this model, agents check into rehab centers when (i) they have reached an addictive state $s \geq 1$ in which they do not want to use, and (ii) p_s is sufficiently large. Agents check into "detox" because they know that there is a probability that they will not be able to do so on their own.

A particularly interesting example of precommitment is the use of agonist, antagonist, and "metabolic" medications described in section 3. Take the case of disulfiram. According to Goldstein [2001,p.151] "if disulfiram is taken by mouth once daily, even a single drink will cause a dreadful aversive reaction. ... an agent who, in the sober state, is motivated to take it regularly will be unlikely to succumb to the craving for a drink, knowing (perhaps from one bad experience) what is bound to happen. ... Experience has shown that alcohol addicts who take disulfiram regularly are able to maintain abstinence reasonably well, but compliance is a major problem. Those with poor motivation either discontinue entirely and relapse, or ... for several days at a time, in order to be able to drink with only minimal discomfort." This suggests that characterization failure can generate unwanted con-

---

[46]Also, longitudinal studies of preschoolers that have participated in Mischel studies have shown that the length of time the 4-year olds were able to delay gratification is correlated significantly with their performance, as adolescents, on standardized tests and parental ratings of competencies such as ability to plan, exert self-control, and focus (Metcalfe and Mischel [1999]).

[47]Using these type of techniques, Volkow (1997) and Volkow et. al. (1997) have shown that the brain's of long-term methamphetamine users are different: they have a lower number of dopamine receptors. This research does not show, however, whether the difference is due to drug exposure or to pre-existing brain differences (or sensitivities).

sumption directly or indirectly by failing to take the pre-commitment medication. A simple extension of the model in which agents do not start every period in the cold mode can explain why some alcoholics stop taking disulfiram. Consistent with this model, an important focus in pharmacotherapy is the development of drugs that need to be taken less frequency since this has been shown to reduce the frequency of relapse (O'Brien [1997]).

Fact 7. Exogenous attention shocks. Consider an agent in a highly addicted stage who is experiencing strong cravings and keeps having thoughts about what it would feel like to get high. The agent is about to consume "on impulse." Before he actually consumes, someone reminds him of other consequences of usage (say the damage he has caused to his family, or the likelihood of dying from an overdose). The agent reacts to the information by choosing not to use (at least temporarily).

Fact 8. Demand for attention management therapies. Recovering addicts who have stayed clean for years choose to attend the meetings of support groups, such as AA, in which no individual therapies or drugs are provided, and in which no new information is revealed. Characterization failure provides a natural explanation for this behavior. Agents attend the meetings because they help them to improve their self-control, and thus to stay clean. During the meetings, agents share their experiences with relapse. By exposing recovering addicts to the experiences of others who are still struggling with the addiction, the cognitive links between usage and the experience of the high are strengthened. This improves the probability that the right thoughts are generated during the hot mode, which improves self-control.

Similarly, characterization failure provides a plausible explanation for the observation that individuals deliberately spend large amounts of resources trying to develop their self-control powers (e.g. techniques for staying calm in the presence of environmental stimuli that ordinarily induce hot visceral states). In fact, society has developed a wide range of tools, from cultural and religious norms to modern behavioral therapies, that teach methods of self-control. Our theory explains, for example, why an individual would learn to defer important decisions until he is calm. It rationalizes therapies that teach individuals to deal with emotional situations by calling to mind specific thoughts and images. It also accounts for therapies that reduce the probability of hot visceral states through deliberate relaxation and desensitization.

Fact 9. Addicts report making mistakes. Addicts describe their relationship with the substance as one of powerlessness. When they "cool down," they frequently describe the usage that took place during the hot mode as a *mistake*. In some instances, they report making a mistake even while in the act of consumption.[48]

---

[48]The model is too stylized to provide a satisfactory explanation of how an agent can simulta-

Characterization failure provides an intuitive explanation for this phenomenon. To make sound decisions, the brain needs to consider the right set of options and consequences. During hot modes, the neocortex focuses excessively on drug usage and on the experience of the high, which leads to a decision that does not generate the maximum possible experiential utility.

An individual without characterization failure can certainly regret a decision in the following sense: he may recognize that he is currently worse off due to some past choice. However, he cannot regard the past choice as a mistake when viewed from the point in time at which the decision was made. In contrast, an individual with characterization failure can certainly view past choices in this way. Once the hot visceral mode subsides, the neocortex resumes normal operation, and the full consequences of actions immediately become apparent.

7 Welfare

The key assumption of this theory is that environmental cues can trigger visceral states that lead to characterization failure. In effect, the hot mode causes the DM's optimization algorithm to break down, without changing his preferences. When the agent characterizes the problem incorrectly, he can make choices that do not generate the greatest possible experiential utility. As a result, it is appropriate to evaluate the DM's well-being using the "preferences" that are coded in the visceral brain. The resulting welfare function is given by

$$\sum_{t=0}^{\infty} \delta^t w_{s_t}(e_t, x_t),$$

where $w_{s_t}(e_t, x_t)$ denotes the experiential utility generated by the visceral brain in period t and addictive state s_t. Note that the brain's operation mode ($\mu = H, C$) has no effect on the evaluation function.

With characterization failure, a policy is welfare improving if it restores the proper operation of the neocortex, or if it gets the agent to make the same choice that he would have made in the cold mode. However, characterization failure, by itself, is not sufficient to justify intervention. A policy can improve welfare only

neously mischaracterize the problem and be aware that he is making a mistake. To explain this observation it would be necessary to add a model of self-awareness. In such a model, an agent that has been taken over by the hot mode might experience thoughts recognizing that he is making a mistake, without being able to generate all of the thoughts that are needed to avoid making the mistake. The awareness could be based on experience: the agent would recognize that the current situation looks like previous occasions in which he was "taken over" by emotions and made a decision which later, after cooling down, was identified as a mistake. Of course, this explanation assumes that the act of self-awareness has a limited ability to restore all of the neo-cortical operations that take place in the deliberative mode.

when, as in the case of substances such as cocaine and nicotine, characterization failure introduces a gap between the actions taken in hot and cold modes. As the example of caffeine shows, this is not always the case.

8   Public Policy

In this section we study the public policy implications of characterization failure. We emphasize at the outset that we do not perform a full cost-benefit analysis of drug policy, which is beyond the scope of this paper. Instead, we study the impact of the different policies on the welfare of users who exhibit characterization failure. The goal is to identify policies that are able to improve the welfare of users and thus are good candidates for further analysis.

To do this we make the following simplifying assumptions.[49] First, we ignore consumption externalities.[50] We do this not to contaminate the analysis. It is well known that externalities provide a rationale for government intervention. The question here is whether characterization failure, *by itself*, also does. Second, we ignore the existence of black markets to circumvent policies, such as taxation or regulated dispensation, that legalize usage but regulates it heavily. Third, we do not model enforcement costs.

Throughout this section we assume that the addictive substance is produced using a constant-returns-to-scale technology, so that supply is infinitely elastic at a price equal to some marginal cost q.

8.1   Taxation

Consider first the introduction of a per-unit tax τ on the substance. Let $V_s^{*\tau}(\theta)$ denote the value function for an agent in state s when the tax rate is τ and all revenues are distributed as described above. The value function under laissez faire is given by $V_s^*(\theta)$. Theorem 5 below shows that, irrespective of the individual's addictive state, taxation is dominated by laissez faire.

The standard measure of welfare is deadweight loss. It is based on a thought experiment in which the revenues raised by the tax are returned to the individuals in a

---

[49]See McCoun and Reuter [2001] and Miron and Zwiebel [1995] for a comprehensive discussion of drug policy that takes into account some important factors, such as enforcement costs and externalities, that we ignore in this paper.

[50]There are three distinct classes of externalities to consider. First, the consumption of addictive substances may inflict costs on others directly (such as accidents due to drunk driving or the spread of infectious diseases). Second, for some substances, consumption is a social activity; i.e., preferences might depend on how many other people are consuming. Third, consumption by one individual may generate cues that trigger hot modes in other individuals. When one considers the first and third types of externalities, policies that discourage use appear more attractive from a welfare perspective. The opposite holds for the second type of externalities.

lump-sum fashion. In this model, however, one must be careful about the manner in which the lump-sum transfers are distributed across addictive states. This is important because we do not permit the DM to borrow and lend, thereby redistributing income over time, and consequently over addictive states. A policy that gives the same transfers to all agents, regardless of their addictive state, would contaminate the experiment by introducing cross-state transfers that, by assumption, are not available to the DM. To isolate the effect of taxation on usage, and to eliminate these spurious welfare gains or losses, one must modify the notion of deadweight loss by requiring that any revenue raised from an individual in a given period and state of addiction is redistributed back to that individual in lump-sum fashion in the same period and state of addiction. In effect, one visualizes a large population wherein DMs are grouped by addictive state in each period, and all resources affected by taxes and transfers remain within these groups. While this construction is artificial, it is necessitated by the artificiality of the no savings assumption.

Theorem 5: *Suppose that the agent has additively separable preferences of the form: $w_s(e, x) = \phi_s(x) + v_s(e)$, where v_s is an increasing differentiable function. Then laissez faire dominates a per-unit tax τ: for every state s, $V_s^{*\tau}(\theta) \leq V_s^*(\theta)$, with strict inequality if $\Pi_s^{*\tau}(\theta) \neq \Pi_s^*(\theta)$.*[51]

The intuition is simple. To be welfare improving, a policy must correct the mistakes made in the hot mode without distorting excessively behavior in the cold mode. The tax has exactly the opposite effect: it has no effect during the hot mode, and distorts behavior during the cold mode, when there is no behavioral problem to be corrected. Also, given the discrete nature of the choice space, a small enough tax may have no effect on behavior, and thus on welfare. Laissez faire *strictly* dominates taxation only if the tax is large enough to distort behavior in at least one state. A similar qualification applies for the other results in this section.

A related class of policies that have been proposed are "harm reduction" or "Dutch policies" such as needle exchanges. The goal of these policies is to eliminate some of the negative consequences associated with drug usage. This can be interpreted as a negative tax or subsidy. As long as the subsidy is small enough theorem 5 still holds.

This result depends on the absence of externalities such as driving under the influence. In the rational addiction model taxation can be welfare improving only

---

[51]The rationale for the restriction on preferences (which is used in step 1 of the proof) is the following. Consider an environment in which agents in state s choose to abstain either with or without the tax. The lump-sum transfer to this group is $T_s = p_s \tau$. For these agents, the the tax is equivalent to the introduction of a lottery that pays $p_s \tau$ in the cold mode, and $-(1 - p_s)\tau$ in the hot mode. The restriction on preferences guarantees that the introduction of such a lottery is not welfare improving. The result can be extended to any preferences satisfying this property.

in the presence of externalities. The same is true here. Characterization failure, by itself, does not provide a rationale for corrective taxation.

Theorem 5 is driven by the extreme form of characterization failure that we have modeled in this paper. In particular, behavior in the hot mode is price inelastic. In Bernheim and Rangel [2002] we provide a more general model of characterization failure in which this need not be the case. Suppose, for example, that in the hot mode the neocortex exhibits a bias towards short-term consequences. Then a large enough tax can induce the agent to abstain even though he is not characterizing the problem properly. Observe, however, that in this more general model the tax still imposes costly distortions to users in the cold mode. As a result, the net welfare effect of the tax would depend on the parameters of the problem.

8.2   Treatment: Subsidized Rehabilitation

Now consider a policy that focuses on treatment by subsidizing the cost of rehabilitation. Let σ_s be the per-treatment subsidy for an addict in state s, and let $\sigma = (\sigma_1, ..., \sigma_T)$. Note that we allow the rate of subsidization to vary with the addictive state. Let $V_t^{*\sigma}(\theta)$ denote the value function associated with this policy. For the same reasons discussed above, the appropriate policy experiment provides financing for the subsidies through lump-sum taxes that avoid redistributions across periods and states of addiction.

The following result shows that the welfare consequences of this policy are similar to those of taxation. The intuition is also similar. Subsidization of treatment distorts behavior in the cold mode, where there is no behavioral problem, but has no effect on the hot mode, where mistakes are made.

Theorem 6:   *Laissez faire weakly dominates any pattern of subsidies to rehabilitation:* $V_s^{*\sigma}(\theta) \leq V_s^{*}(\theta)$ *for all s, with strict inequality if $\Pi_s^{*\sigma}(\theta) \neq \Pi_s^{*}(\theta)$.*

8.3   Criminalization

There are two types of criminalization policies: (1) demand-side criminalization, which outlaws usage; and (2) supply-side criminalization, which proscribes production and distribution. Here we focus on the second type. The analysis of demand-side criminalization, where usage of illegal substances is punished with fines or jail, is similar to the case of taxation.[52]

Sellers react to criminalization by moving to the black market. This has an impact on demand through the following two effects: a *price effect*, since criminal-

---

[52]Like taxation, demand-side criminalization increases the marginal costs of usage. However, unlike taxation, it generates no revenue, and probably involves greater enforcement costs. Thus, in this simple model, demand-side criminalization is Pareto dominated by taxation.

ization increases the cost of bringing drugs to the market; and a *scarcity effect*, since the policy interferes with the process of matching buyers and sellers. Let Δq denote the price increase, and γ_s denote the probability that a DM who wants to buy the substance in state s is able to complete the purchase. Let $V_t^{*C}(\theta)$ denote the value function associated with supply-side criminalization.

It is instructive to consider the scarcity and price effects separately. Consider first a policy that only generates a price increase. This policy is equivalent to a per-unit-tax policy ($\tau = \Delta q$) in which the revenue raised by the tax is destroyed. It follows that criminalization is dominated by taxation. Then, by theorem 5, it is also dominated by laissez-faire.

Now consider an idealized policy that only generates scarcity effects.[53] Regardless of the DM's mode, this policy reduces the probability that an agent who wants to use the substance can do so.[54] This is always costly in the cold mode, but can be beneficial in the hot mode if it stops unwanted consumption from taking place. The net welfare effect depends on the parameters.

To see why, consider first a substance for which usage is optimal in every addictive state. A replication of the arguments used in the proof of theorem 4, part (i), shows that decreases in γ_s have no effect on the optimal decision rule: the DM still wants to use in every period. The scarcity effect then necessarily makes the agent worse off: he always prefers to consume in the cold mode but sometimes is not able to find a seller. Now consider a substance for which laissez faire generates the pattern "$A, A, A, A, R, R, R, R,$" before and after the policy change. The policy is clearly welfare improving since it reduces usage only when the brain enters the hot mode in states 1 through 3. Finally, consider a substance that generates the intermittent use pattern "$U, U, U, A, A, A, R, R, R,$" before and after the introduction of the policy. In this case the policy decreases well-being in states 1 through 3, but can increase welfare in more highly addicted states. The net welfare effect depends on the exact parameters of the value function.[55]

---

[53]This policy changes somewhat the value function described in (1)-(4), but the results in section 4 extend to this case.

[54]See Goldstein and Kalant [1990] for evidence establishing that drug usage decreases as these substance become less available.

[55]Agents could respond to the scarcity effect by stocking up drugs. One would expect that extending the model in this direction would improve the attractiveness of criminalization. Here is the intuition. Agents in the cold mode would stock drugs only when future use is optimal. Agents in the hot mode could also try to stock drugs, but since they enter the cold at the beggining of every period, they can get rid of unwanted stocks.

8.4 Legalization with Regulated Dispensation

Criminalization is a poor policy instrument because it does not discriminate between consumption in the hot and cold modes. By contrast, an ideal policy would eliminate consumption only when (i) the DM is in the hot mode, and (ii) he would have chosen to abstain in the cold mode. Any such policy would achieve the first-best outcome, mimicking the case of a consumer who never exhibits characterization failure ($p_s = 0$ for all s).

The preceding observations lead us to consider a policy of legalization with regulated dispensation. Imagine, in particular, that the government licenses vendors and requires them to take all orders for controlled substances one period in advance. A consumer can place an order at any point during any period t. However, the product is not delivered until period $t+1$. At the beginning of period $t+1$, prior to taking delivery on an order placed in period t, the consumer is permitted to cancel the order. We assume that supply is only available through these regulated vendors.

Equipped with this transaction technology, consumers in our model can achieve the first best outcome. Solving the dynamic programming problem with $p_s = 0$ for all s yields a deterministic consumption path. The consumer can mimic this outcome as follows. At the beginning of each period t (when the consumer is in the cold mode), he (i) forms an intention to use in the current period if and only if this is his first-best choice, (ii) places an order for delivery at time $t + 1$ if and only if consumption in $t + 1$ is part of the first-best solution, and (iii) cancels any orders placed in $t - 1$ (possibly while in the hot mode) if consumption in t is not part of the first-best solution. In this way, the consumer himself selectively creates optimal scarcity: the substance is available to him only when he would choose to use it while in the cold mode. To put it somewhat differently, this policy allows consumers to make optimal pre-commitments.[56]

Individual heterogeneity presents a difficult challenge for drug policy: some usage is unwanted and driven by characterization failure, other usage is rational. Since the government cannot distinguish between both types of users, it must impose a common policy to all of them. An attractive feature of regulated dispensation is

---

[56]Naturally, the problem considered here is highly stylized. However, even in more realistic circumstances, it is possible to imagine policies of regulated dispensation that permit consumers to achieve better outcomes by selectively contriving scarcity. For example, the preceding argument appears to depend on the assumption that the consumer enters each period in the cold mode. In fact, one can dispense with this assumption. Imagine, that hot modes can potentially last for several periods. Consider a policy of regulated dispensation that permits consumers to (1) place advance orders for any future period, (2) prior to delivery, cancel orders placed in any previous period, and (3) voluntarily establish restrictions on the orders that they are permitted to place in any future period. Consumers are not permitted to remove a voluntary restriction once they establish it. Provided that the consumer occasionally enters the cold deliberative mode, he can use this transaction technology to contrive optimal scarcity and achieve the first-best outcome.

that it is able to curtail unwanted usage without imposing a cost in "rational users". By contrast, all the previous policies hurt "rational users."

It is important to emphasize that legalization with regulated dispensation works well in this context because the mistakes made in the hot mode always involve overconsumption. This policy would not work in a setting where hot modes could produce either overconsumption or underconsumption.[57]

8.5 "Behavioral" Policies

Given the brain processes responsible for characterization failure, a natural class of policies are those that reduce the number of cues that trigger cravings, or minimize the cognitive bias during the hot mode. By contrast, the policies discussed above attempt to curtail unwanted consumption by modifying the budget constraint. In terms of the model, the two policies considered here generate a reduction in the probability of entering the hot mode. For some substances, as theorem 3 shows, this may lead to higher intended and experimental use.

Policies that attempt to reduce an addict's exposure to cues include regulating the public consumption of drugs, regulating or even prohibiting drug advertisement, and regulating the places where they can be sold - for example, by prohibiting their public display. In each one of these cases, public intervention is justified since a consumer or seller is imposing an externality in potential users by creating cues that can trigger the hot mode.

Similarly, consider a public advertising campaign that repeatedly presents viscerally charged images about the consequences of consuming drugs (blackened lungs, shrunk brains, gruesome car wrecks). If the campaign influences the pattern of cognitive activation during the hot mode, it can reduce unwanted consumption. For example, repeated exposure to visceral images regarding the effects of nicotine might produce a cognitive link such that, whenever a cue triggers the hot mode, it also activates thoughts about the gruesome consequences of drugs usage.

Since agents can purchase this type of "therapy" as a private good, public provision is justified only if (1) there are increasing returns in the provision of these goods, (2) agents have an imperfect understanding of the value of these therapies, or (3) an agent in the hot mode imposes an externality in others.

Like regulated dispensation, an attractive feature of these types of policies is that they are able to decrease unwanted usage without distorting rational usage.

---

[57]In a related analysis, Loewenstein, O'Donoghue, and Rabin [2000] emphasize the role of "mandatory waiting periods" in a model where agents systematically overconsume.

9   Final Remarks

Even a casual reading of the literature reveals that one of the most difficult problems is how to define addiction. For example, is this phenomenon confined to drugs, or can agents also be addicted to television, love, and french fries? This theory suggests the following answer to this question. An agent is addicted to a substance or activity whenever he finds himself engaging in *repeated* and *unwanted* consumption. Both features are an essential part of the definition. Repeated but wanted use, as in the case of caffeine, does not represent an addiction. Similarly, a substance which triggers the hot mode only sporadically does not lead to addiction since the unwanted consumption rarely takes place. A prototypical example of an addiction is given by a the binging cycles that are common among cocaine and nicotine users. These agents want to quit after an initial period of use but find themselves unable to do so. At that point, they are addicted to the substance. Any activity that generates repeated and unwanted consumption leads behavioral problems similar to those generated by the addictive substances, and is likely to be driven by similar mechanisms. Examples include pathological gambling, overeating, and, at the end of the spectrum, compulsive shopping and compulsive shoplifting (kleptomania). Interestingly, compulsive shoppers experience a hot mode analogous to cravings, are affected by cues such as stress or advertisements, and exhibit the same type of "binging cycles".[58]

In order to isolate the role that characterization failure plays in explaining the stylized facts, and to study its public policy implications, we have assumed that cues do not trigger taste shocks. However, the evidence suggests that cue-triggered taste-shocks are also likely to be at work. Here we discuss the positive and normative implications of adding them into the model. Cue-triggered taste shocks are easily incorporated by assuming that the basic pay-off function is given by $w_s(e, x, \mu)$. This makes the baseline payoff and the marginal benefit of use dependent on cues (i.e., $u_s(\mu)$ and $b_s(\mu)$). In this more general model, a cue that triggers cravings affects decision-making through its impact on cognitive activation and through its impact on the function used by the visceral brain to evaluate choices.

The positive implications of adding taste-shocks are straightforward. If cravings increase the marginal benefit of usage $(b_s(H) > b_s(C))$, they provide an additional mechanism for why agents use when cues trigger the hot mode. If cravings are hedonically unpleasant $(u_s(H) < u_s(C))$, they provide an additional reason for

---

[58]See Holden (2001) for a review of the commonalities between the addictive substances and these behavioral problems that have been uncovered by recent research. For example, as drug addicts, compulsive gamblers and kleptomaniac's respond to drugs such as naltrexone which block the brain's ability to experience the high, and compulsive gamblers and bulimics experience sudden relapse even after many years of abstinence.

cutting early use to avoid the possibility of increasing the addictive state, and thus the probability of entering the hot mode. Finally, as $b_s(H)$ increases, the demand for rehab decreases. Plainly, agents demand rehab only when it is optimal to abstain but there is a significant probability of entering the hot mode. If $b_s(H)$ is large enough, consumption in the hot mode is optimal, and thus agents do not want to stop themselves from using when cravings arise.

The policy implications of adding taste-shocks are more complicated. Theorems 5 and 6 hold: laissez-faire still dominates a per-unit tax and subsidized rehabilitation. To see why, consider a model in which there are only taste shocks, but no characterization failure ($p_s = 0$ for all s). Since there are no externalities, taxes and subsidies necessarily generate a deadweight-loss.

The analysis of criminalization is complicated. In section 8 we showed that criminalization can be welfare improving only when it generates scarcity effects that curtail unwanted demand in the hot mode. Now consider again the extreme case in which there are only taste shocks. In this case the scarcity effect must decrease welfare since it only curtails "wanted" demand. Thus, the introduction of taste shocks decreases the attractiveness of the criminalization policy. In fact, if the taste effects are strong enough, criminalization is always dominated by laissez-faire.

The introduction of taste shocks also reduces the attractiveness of the regulated dispensation policy. This policy is ideal to control the problems generated by characterization failure because it provides a pre-commitment technology to stop unwanted consumption. But then, since the introduction of taste shocks decreases the demand for pre-commitment, it also reduces the welfare value of this policy.

Finally, consider two behavioral policies. The first one decreases exposure to cues. The second reduces the cognitive bias that takes place during the hot mode. If cravings are hedonically aversive, they introduce an additional rationale for the first behavioral policy. To the extent that taste shocks reduce the amount of unwanted demand, they also weaken the case for the second behavioral policy.

An important question for future research is to quantify the relative importance that taste shocks, characterization failure, and other mechanisms such as projection biases (see the appendix), play in the consumption of the different substances and populations. This is essential since, as described above, in some cases these mechanisms have opposite policy implications. The enormous advances that the brain sciences have made in the last few decades, including the development of neuro-imaging technologies, makes this a feasible and exciting project. In tackling the problems of addiction and the optimal design of drug policy, there are important synergies between neuroscience, psychology, and economics.

Appendix A: Other Theories of Addiction

In this paper we show that a simple model of addiction based on characterization failure can explain the nine stylized facts listed in the introduction. Here we review the main competing theories of addiction and show that none of them can explain all of the facts. The discussion is summarized in table 1.

A. Rational addiction

Consider first the rational addiction model of Becker and Murphy [1988], which is based on tolerance and withdrawal.[59] Agents are fully informed and maximize standard preferences. The key feature of the model is the presence of "addictive capital": present usage increases the future marginal benefit of usage, and decreases the future baseline level of well-being.

This standard model of decision-making explains stylized fact number 5. However, it is easy to see that a rationally addicted agent would never consume in "random" binging cycles, be affected by cues, engage in pre-commitment, be affected by attention shocks that provide no new information, or make mistakes.

The model also has difficulties explaining the remaining three facts. To explain why recovering addicts keep attending AA meetings, the model needs to assume that addicts, but not others, derive *direct utility* out of them. It is not clear why this should be the case. By contrast, our theory provides an explanation that is not based on "addict specific tastes". Next, to explain why only a small fraction of people who repeatedly use in young age eventually become long-term addicts, the model needs to resort to changes in preferences: many people like consuming the substance in youth, but non-addicts loose their taste for it at some point in mid-life. (A similar comment applies to all of the other theories discussed here, except for projection bias). By contrast, the explanation for this fact provided in this paper does not rely on changes in preferences. Finally, by selecting an appropriate preference specification, the model can also explain some, but not all of the differences across substances. For example, the model can explain why some drugs are consumed more frequently than others, or in smaller dosages, but not why some of them involve "random" binging cycles and the use of detox programs. By contrast, the explanation for the differences across substances developed here is based on how the characterization failure mechanism interacts with exogenous properties of the substances.

B. Rational addiction with taste shocks

---

[59]See also Orphanides and Zervos [1995].

Laibson [2001][60] has shown that the explanatory power of the rational addiction model increases significantly with the introduction of cue triggered "taste-shocks", or cravings. Their introduction is justified since the body reacts to the consumption of addictive substances by releasing *feed-forward mechanisms* (see section 3) which affect the body in a way that looks like a cue triggered taste shock.

This small change provides an explanation for binging patterns, the role of cues, and some of the differences across substances. It can also explain the demand reducing effect of attention shocks that reveal no new information, but only if, in contrast to the explanation provided in this paper, one assumes that they affect tastes. As far as we know, there is no foundational evidence establishing that this is the case.

The introduction of cue-triggered taste shocks cannot overcome the other shortcomings. In particular, it cannot explain why agents choose to pre-commit not to use the substance by checking into a rehab center. If cues increase the taste for drugs, agents would want to consume the substance whenever they experience cravings, and thus would never check into a detox program.

Loewenstein [1996,1999] convincingly argues, using empirical and experimental evidence, that visceral states such as cravings and hunger have a large impact in decision-making and that they induce agents to make decisions that are against their self-interest.[61] Based on this, he sketches a model in which cues change the preferences that agents maximize, but not their "true" utility function. No foundation is provided for this departure from the standard model. By contrast, here we develop a full model based on characterization failure, not on taste shocks.

C. Temptation preferences models of addiction

Gul and Psendorfer [2001a,2001b] and Laibson [2001] have argued that an additional mechanism, which we refer here as "temptation taste-shocks", plays an important role in the consumption of addictive substances.[62] They propose a model in which agents' preferences depend on the budget constraint in a very specific way. If at time t the agent has to choose whether or not to consume the drug at time t, and the drug is in the budget constraint, he receives a "temptation taste-shock" that (1) increases the marginal utility of consuming the drug, and (2) generates a utility cost. However, the temptation taste shock does not appear when the agent

---

[60]See also Hung [2000].

[61]This theory is greatly indebted to the ideas developed by Loewenstein in these papers. See also Loewenstein and Lerner [2001] for an excellent review of the empirical evidence on the effect of emotions on decision-making.

[62]Gul and Psendorfer [2001a] show that these types of preferences can be derived from an axiomatization that, at first sight, looks like a minor departure from the standard axioms of expected utility.

makes choices one or more periods in advance (for example, when choosing in period t whether or not to consume the drug in period $t + k$, with $k \geq 1$). The authors interpret the temptation shock as cravings.

Consider adding temptation to the rational addiction model with taste-shocks. (Such a model has not been written, but it is the most general version of this class of theories). This modification can explain some, but not all pre-commitment. To see why, note that in this theory agents pre-commit to avoid cravings, a costly taste-shock. However, they would not choose to pre-commit if they knew that they were going to experience cravings anyway. This is problematic since it runs counter to the experience of addicts: the reason that they give for checking into rehabs is that they know that they will experience cravings (in or out of the center) and they want to make sure that they will not consume when this happens.

Another short-coming of temptation costs is that they make preferences dependent on the budget constraint. This is tricky because, if one is free to specify the form of this dependency, any behavior can be explained. In principle, evidence from disciplines such as neuroscience could guide the choice of the functional form. However, such foundational evidence has not been provided.[63]

D. Present-biased preferences models of addiction

O'Donoghue and Rabin [1999,2000] and Gruber and Koszegi [2001] have proposed a variation of the rational addiction model in which exponential discounting is substituted for quasi-hyperbolic discontinuing. Agents have dynamically inconsistent preferences that change through time and exhibit a present bias: in any period t, the discount rate between periods t and $t + 1$ is higher than the discount between periods $t + k$ and $t + k + 1$. There are two versions of this model, which differ on the degree to which agents are aware of their self-control problem. In the sophisticated version, agents fully anticipate the consequences of the present bias. In the naive version, agents consistently mischaracterize their future actions. The naive version of the model generates behavior similar to the rational addiction model. Thus, here we focus our discussion on the case of sophisticated agents.

The introduction of quasi-hyperbolic discontinuting can explain pre-commitment. Consider parameters for which agents ideal choice is to use the drug now, but not in the future. In the absence of pre-commitment, this is not feasible since their future selves have the same type of present bias, and thus always choose to use. Pre-commitment is valuable because it allows them to control their future selves.

---

[63]Laibson [2001] provides foundational evidence for why exposure to the drug, or cues related to the drug, can trigger feed-forward mechanisms that look like regular taste-shocks. However, no foundational evidence is provided for temptation costs.

It is important to emphasize that in this model agents are short-sighted, but they do not make mistakes: they always choose the option that maximizes their objectives. Thus, they would never report making a mistake, or experience regret, while in the act of consumption.

Gruber and Koszegi [2001] also study the implications of this model of addiction for optimal taxation. Their analysis is complicated by the fact that, since there are many "selves" with different preferences, there is no straightforward notion of welfare. They choose a welfare function equal to the preferences that agents would have if there were no present bias. Under this notion of welfare the model generates overconsumption of drugs. Using data from cigarette consumption, they argue that quasi-hyperbolic discounting provides a rationale for a sizable per-unit sales tax, which stands in sharp contract with theorem 5 in this paper. Besides the fact that the underlying model cannot account for all the stylized facts, there is an additional concern with this result. The model predicts overconsumption of all addictive substances, from caffeine to cocaine, and that all of them should be taxed.[64] In fact, it predicts that agents overconsume any good that generates pleasure in the present (from movies to shoes). This runs counter to intuition. By contrast, the model of addiction developed here predicts that some substances are overconsumed, but not others.

E. Projection bias models of addiction

Loewenstein [1996,1999], Loewenstein, O'Donoghue, and Rabin [2001] have argued that projection bias plays an important role in the consumption of addictive substances. Projection bias says that agents over-estimate the extent to which their future preferences will look like their current ones. Importantly, in these models agents are unaware of their projection bias. As a result, they mistakenly predict the future consequences of their actions. For example, an unaddicted agent might not understand that using the drug today will increase his future taste for the substance, and thus it will lead to more use in the future future.

The introduction of projection bias into the rational addiction model improves its explanatory power in two dimensions. First, it provides a more plausible explanation for individual heterogeneity. During the youthful period of experimentation, some agents learn to forecast their future preferences, while others do not. As long as their addictive capital is not too high, the first group is able to successfully stop consuming the substance. Second, as long as exogenous attention shocks (such as a reminder of the effects of consuming the substance) reduce the projection bias, it can explain stylized fact number 7.

---

[64]Note that the case for taxation that they make is based on overconsumption, not on the presence of externalities.

Projection bias cannot explain the other stylized facts. Pre-commitment does not arise since the brain does not anticipate a change in preferences between itself and its future "selves". There is no role for cue-driven recidivism, since present bias is always at work and does not depend on cues. Finally, agents do not report making mistakes since they always choose the path of consumption that maximizes their perceived well-being at the time. For this same reason, agents do not choose to attend informational meetings, such as AA, which reduce their projection bias.

In this paper we show that projection biases are not necessary to explain the basic stylized facts. However, since a significant amount of evidence suggests that agents are not good at forecasting their future states, it is natural to consider what would be the effect of adding projection biases to our model. From a positive point of view, an important effect would be to increase the amount of early usage that takes place, and thus the eventual size of the addicted population. The reason is simple. In section 6 we show that one of the reasons agents abstain early on is to avoid the possibility of moving to higher addictive states where there is an increased danger of entering the hot mode. Agents who do not fully understand the power of characterization failure would engage in less preventive abstention. From a policy perspective, projection biases introduces a role for "behavioral policies", such as public information campaigns and education, that reduce the size of the bias.

F. Hypothetical "mega-theory"

Now consider a hypothetical model that includes all of the features discussed above: agents exhibit taste-shocks, quasi-hyperbolic discounting, temptation-shocks, and projection bias. Such a theory would be able to explain all of the facts except the last two: why agents actively seek to receive cognitive instructions, such as those provided in AA meetings, and why agents report making mistakes. This is not a minor shortcoming since characterization failure, a mechanism that is able to explain them and has good neuro-foundations, has important positive and norma-tive implications. Furthermore, since preferences change with time, this theory does not lead to an uncontroversial welfare criterion that can be used to evaluate public policy.

It is useful to emphasize the contribution that the different components would make to the explanatory value of such a theory: taste-shocks would explain the binging cycles and the role of cues, quasi-hyperbolic discounting would explain the pre-commitment, projection bias the heterogeneity across individuals and the effect of exogenous attention shocks.

	rational addiction	rational addiction with taste schocks	rational addiction with temptation costs	rational addiciton with present biased preferences	rational addiction with proejction bias	characterization failure
Binging cycles: ST-abstention, LT-recidivism	N	Y	N	N	N	Y
Central Role for cue-conditioned cravings	N	Y	N	N	N	Y
Differences across substances (caffeine vs. cocaine)	~	~	~	~	~	Y
Differences across users	~	~	~	~	Y	Y
Responiveness to prices and information	Y	Y	Y	Y	Y	Y
Pre-commitment behaviors (detox, disulfiram)	N	N	~	Y	N	Y
Effect of exogenous attention shocks	N	~	N	N	Y	Y
Demand for attention management therapies	~	~	~	~	~	Y
Mistakes: unwanted consumption	N	N	N	N	N	Y

Figure 2: Summary of the literature

Appendix B: Proofs

Proof of Lemma 1: To simplify the notation in the proof, let $V_s(i) = V_s^i(\Pi^*(\theta), \theta)$ and $V_s'(i) = V_s^i(\Pi^*(\theta'), \theta')$ for $i = U, A, R$, $V_s = V_s^*(\theta)$, and $V_s' = V_s^*(\theta')$.
(Step 1) We claim that for all $s < k$, $V_s' - V_s \leq V_{s+1}' - V_{s+1}$. Consider first the case $s = 1$. (The case $s = 0$ is almost identical and thus is omitted).[65]

$$
\begin{aligned}
V_1' - V_1 &= \max\{V_1'(U), V_1'(R), V_1'(A)\} - \max\{V_1(U), V_1(R), V_1(A)\} \\
&\leq \max\{V_1'(U) - V_1(U), V_1'(R) - V_1(R), V_1'(A) - V_1(A)\} \\
&= \delta \max\{V_1' - V_1, V_2' - V_2, (1 - p_1)(V_1' - V_1) + p_1(V_2' - V_2)\}.
\end{aligned}
$$

Consider the last expression. Given the linearity of the third term, there are two possible cases. If the first term is the maximand then, since $\delta \in (0, 1)$, $V_1' - V_1 = 0 \leq V_2' - V_2$. The last inequality follows from the statement of the lemma. If the second term is the maximand, the claim trivially holds.
Now consider the following induction step. We show that for all $s < k$,

$$
V_{s-1}' - V_{s-1} \leq V_s' - V_s \Rightarrow V_s' - V_s \leq V_{s+1}' - V_{s+1}.
$$

Again, we get that

$$
V_s' - V_s \leq \delta \max\{V_{s-1}' - V_{s-1}, V_{s+1}' - V_{s+1}, (1 - p_s)(V_{s-1}' - V_{s-1}) + p_s(V_{s+1}' - V_{s+1})\}. \tag{10}
$$

As before, there are two possible cases. If the first term is the maximand we get that

$$
V_s' - V_s \leq \delta(V_{s-1}' - V_{s-1}) \leq \delta(V_s' - V_s);
$$

where the last inequality follows from the induction hypothesis. This implies that $V_s' - V_s = 0$ and the rest of the argument proceeds as before. If the second term is the maximand, the claim trivially holds. (Note that this establishes the claim only for $s < k$; (10) does not hold for $s = k$ since $\theta_k' \neq \theta_k$).
(Step 2) We show that for all $s > k$, $V_s' - V_s \leq V_{s-1}' - V_{s-1}$. The argument is symmetric to the one in step 1 and thus is omitted.
(Step 3) Step 1 implies that, for all $0 \leq s < k$,

$$
V_s' - V_s \leq V_{s+1}' - V_{s+1} \text{ and } V_{\max\{1, s-1\}}' - V_{\max\{1, s-1\}} \leq V_s' - V_s.
$$

---

[65]The argument makes use of the fact that, for any 6 real numbers a to f,

$$
\max\{a, b, c\} - \max\{d, e, f\} \leq \max\{a - d, b - e, c - f\}.
$$

This implies that

$$V'_{\max\{1,s-1\}} - V_{\max\{1,s-1\}} \leq V'_{s+1} - V_{s+1},$$

and thus

$$V'_{\max\{1,s-1\}} - V'_{s+1} \leq V_{\max\{1,s-1\}} - V_{s+1}.$$

Since the parameters for state s have not changed, (6) to (8) imply that in states 1 to $k-1$ usage is weakly higher with θ' than with θ. An almost identical argument shows that usage also weakly increases in state 0.

(Step 4) Step 2 implies, using an argument symmetric to step 3, that in states $k+1$ to S usage is weakly lower with θ' than with θ. Q.E.D.

The following lemma is used in the proofs below:

Lemma 2: For any vector of parameters θ and decision rule $\Pi \neq \Pi^*(\theta)$, there exist a finite number of decision rules $\Pi^1,...,\Pi^m$ such that: (1) $\Pi^1 = \Pi$, (2) $\Pi^m = \Pi^*(\theta)$, (3) for all t, $\Pi^t_s \neq \Pi^{t+1}_s$ for exactly one state s (call it j^t), and (4) $V_k(\Pi^{t+1}) \geq V_k(\Pi^t)$ for all t and k, with strict inequality for $k = j^t$.

Proof: Consider the following algorithm. For any decision rule Π^t, which may be probabilistic, compute $V_0(\Pi^t)$, ..., $V_T(\Pi^t)$ using equations (1) to (4). Let $j^t = \min\{k = 0, ..., S | \Pi^t_k$ does not satisfy the optimality condition in (5)$\}$. If this problem does not have a solution, the algorithm stops and $\Pi^t = \Pi^*(\theta)$. If it has a solution, define a new value function Π^{t+1} as follows: $\Pi^{t+1}_s = \Pi^t_s$ for all $s \neq j^t$; and

$$\Pi^{t+1}_{j^t} = \arg\max_{i=U,A,R}\{V^i_{j^t}(\Pi^t)\}$$

(in case of indifference, choose the action that involves higher usage as defined in section 4). Set $\Pi^0 = \Pi$.

(Step 1) We show that $V_k(\Pi^{t+1}) \geq V_k(\Pi^t)$ for all t and $k > j^t$. Given that Π^{t+1} and Π^t only differ on the action taken in state j^t, the value functions for any state $k > j^t$ can be written as:

$$V_k(\Pi^t) = \alpha(\Pi^t_{j^t+1}, ..., \Pi^t_S, \delta, k)V_{j^t}(\Pi^t) + \beta(\theta_{j^t+1}, ..., \theta_S, \Pi^t_{j^t+1}, ..., \Pi^t_S, \delta, k)$$

and

$$V_k(\Pi^{t+1}) = \alpha(\Pi^t_{j^t+1}, ..., \Pi^t_S, \delta, k)V_{j^t}(\Pi^{t+1}) + \beta(\theta_{j^t+1}, ..., \theta_S, \Pi^t_{j^t+1}, ..., \Pi^t_S, \delta, k),$$

where α and β depend on δ, $\Pi^t_{j^t+1}$ to Π^t_S, θ_{j^t+1} to θ_S, and k. Then, step 3 (which for expositional reasons is proven below) implies that $V_k(\Pi^{t+1}) \geq V_k(\Pi^t)$.

(**Step 2**) We show that $V_k(\Pi^{t+1}) \geq V_k(\Pi^t)$ for all t and $k < j^t$. The argument is similar to the one in step 1.

(**Step 3**) We show that $V_{j^t}(\Pi^{t+1}) > V_{j^t}(\Pi^{t+1})$ for all t. By construction of Π^{t+1}, $V_{j^t}^{\Pi^{t+1}(j^t)}(\Pi^t) > V_{j^t}(\Pi^t)$; where $V_{j^t}^{\Pi^{t+1}(j^t)}(\Pi^t)$ denotes the payoff at state j^t of choosing $\Pi^{t+1}(j^t)$ now, but sticking to Π^t in the future, including choosing $\Pi^t(j^t)$ if the state j^t is reached again.

Define a sequence of non-stationary decision rules $\widetilde{\Pi}^n$, for $n = 0, 1, ...,$ as follows. Let h denote the history of actions and states. Define a counter function $\eta(h)$ that keeps track of the number of times the DM has visited state j^t in the past. The n-th decision rule is given by $\widetilde{\Pi}_s^n(h) \equiv \Pi_s^t$ for all $s \neq j^t$; and

$$\widetilde{\Pi}_{j^t}^n(h) \equiv \begin{cases} \Pi^{t+1}(j^t) & \text{if } \eta(h) \leq n \\ \Pi^t(j^t) & \text{otherwise} \end{cases}.$$

For all n, let $V_{j^t}(\widetilde{\Pi}^0)$ denote the value function when state j^t is reached for the first time (i.e., when $\eta(h) = 0$). Note that $V_{j^t}(\widetilde{\Pi}^0) = V_{j^t}^{\Pi^{t+1}(j^t)}(\Pi^t)$ and $\widetilde{\Pi}^\infty = \Pi^{t+1}$.

We need to consider three possible cases.

(**Case 1**) $\Pi^{t+1}(j^t) = U$. In this case $V_{j^t}(\widetilde{\Pi}^0)$ can be written as

$$\begin{aligned} V_{j^t}(\widetilde{\Pi}^0) &= u_{j^t} + b_{j^t} + \delta V_{j^t+1}(\Pi_t) \\ &= u_{j^t} + b_{j^t} + \delta(\alpha V_{j^t}(\Pi^t) + \beta); \end{aligned}$$

where α and β are identical to the constants defined in step 1. Similarly,

$$\begin{aligned} V_{j^t}(\widetilde{\Pi}^1) &= u_{j^t} + b_{j^t} + \delta(\alpha V_{j^t}(\widetilde{\Pi}^0) + \beta) \\ &= (1 + \delta\alpha)(u_{j^t} + b_{j^t}) + (\delta + \delta^2\alpha)\beta + (\delta^2\alpha^2)V_{j^t}(\Pi^t). \end{aligned}$$

An iteration of this arguments shows that

$$\begin{aligned} V_{j^t}(\widetilde{\Pi}^n) &= (1 + \delta\alpha + ... + \delta^n\alpha^n)(u_{j^t} + b_{j^t}) + \\ &\quad (\delta + \delta^2\alpha + ... + \delta^{n+1}\alpha^n)\beta + (\delta^{n+1}\alpha^{n+1})V_{j^t}(\Pi^t). \end{aligned}$$

This implies that

$$V_{j^t}(\widetilde{\Pi}^{n+1}) - V_{j^t}(\widetilde{\Pi}^n) = \delta^n\alpha^n(V_{j^t}(\widetilde{\Pi}^0) - V_{j^t}(\Pi^t)) > 0. \tag{11}$$

If $\alpha = 0$, then $V_{j^t}(\Pi^{t+1}) > V_{j^t}(\Pi^t)$ since

$$V_{j^t}(\Pi^{t+1}) = V_{j^t}(\widetilde{\Pi}^0) = V_{j^t}^{\Pi^{t+1}(j^t)}(\Pi^t) > V_{j^t}(\Pi^t).$$

If $\alpha > 0$, then (11) implies that $V_{j^t}(\widetilde{\Pi}^n) < V_{j^t}(\widetilde{\Pi}^{n+1})$ for all n. A standard continuity argument then shows that $V_{j^t}(\Pi^{t+1}) > V_{j^t}(\Pi^t)$ since $V_{j^t}(\Pi^t) = V_{j^t}(\widetilde{\Pi}^0)$ and $V_{j^t}(\widetilde{\Pi}^n)$ converges to $V_{j^t}(\widetilde{\Pi}^\infty)$, which is equal to $V_{j^t}(\Pi^{t+1})$.

(Cases 2 and 3) $\Pi^{t+1}(j^t) = A$ or R. The argument is similar and thus is omitted.

(Step 4) To conclude the proof it suffices to notice two facts. First, the algorithm defined in step 1 must stop at a deterministic decision rule satisfying the optimality conditions in (5). Second, the algorithm stops in a finite number of steps since there is always a deterministic optimal decision rule, and there are at most $3(T+1) - 1$ such decision rules. Q.E.D.

Proof of Theorem 1:
As before, to simplify the notation in the proof let $V_k = V_k^*(\theta)$.
(Step 1) We show that $V_0 \geq V_1$. Since U is an option at $s = 0$, we have that $V_0 \geq u_0 + b_0 + \delta V_1$. Furthermore, since the substance is destructively addictive, we must have that $V_1 \leq \frac{u_1 + b_1}{1 - \delta} \leq \frac{u_0 + b_0}{1 - \delta}$. These two inequalities imply that $V_0 \geq (1 - \delta)V_1 + \delta V_1 = V_1$.
(Step 2) Now we show that $V_{s-1} \geq V_s \Rightarrow V_s \geq V_{s+1}$. There are four possible cases.
(i) $\Pi_s^* = U$. In this case, $V_s = u_s + b_s + \delta V_{s+1}$. Furthermore, since states lower than s are never reached (once the DM is in state s), and the substance is destructively addictive, we have that $V_{s+1} \leq \frac{u_s + b_s}{1 - \delta}$. The argument then proceeds as in step 1.
(ii) $\Pi_{s+1}^* = R$. In this case we have that

$$V_{s+1} = u_{s+1} - c_{s+1} + \delta V_s \leq u_s + b_s + \delta V_s.$$

Also, $V_s \geq u_s + b_s + \delta V_{s+1}$ since U is an option. This implies that $V_s \geq V_{s+1} - \delta V_s + \delta V_{s+1}$ and thus $V_s \geq V_{s+1}$.
(iii) $\Pi_{s+1}^* = U$. In this case $V_{s+1} \leq \frac{u_{s+1} + b_{s+1}}{1 - \delta} \leq \frac{u_s + b_s}{1 - \delta}$. Also, at state s we have that $V_s \geq u_s + b_s + \delta V_{s+1}$. The argument then proceeds as in case (i).
(iv) $\Pi_{s+1}^* = A$ and $\Pi_s^* \neq U$. From condition (7) and $b_{s+1} \geq 0$, we have that $V_s \geq V_{s+2}$ (otherwise A would not be optimal at $s+1$). Suppose, towards a contradiction, that $V_{s+1} > V_s$. Then $V_{s+1} > V_{s+2}$. Then we have that

$$
\begin{aligned}
V_{s+1} &= p_{s+1}(u_{s+1} + b_{s+1} + \delta V_{s+2}) + (1 - p_{s+1})(u_{s+1} + \delta V_s) \\
&\leq p_{s+1}(u_{s+1} + b_{s+1} + \delta V_{s+1}) + (1 - p_{s+1})(u_{s+1} + \delta V_{s-1}) \\
&\leq p_{s+1}(u_s + b_s + \delta V_{s+1}) + (1 - p_{s+1})(u_s + \delta V_{s-1}) \\
&\leq p_s(u_s + b_s + \delta V_{s+1}) + (1 - p_s)(u_s + \delta V_{s-1}) \quad \text{(see below)} \\
&\leq V_s;
\end{aligned}
$$

which is a contradiction. If $\Pi_s^* = A$, the third inequality follows because $p_s \leq p_{s+1}$ and, for abstention to be optimal, it must be the case that $u_s + \delta V_{s-1} \geq u_s + b_s + \delta V_{s+1}$. If $\Pi_s^* = R$, the third inequality follows since $p_s \leq p_{s+1}$ and it must be the case that

$$u_s + \delta V_{s-1} \geq u_s - c_{s-1} + \delta V_{s-1} \geq u_s + b_s + \delta V_{s+1}.$$

The last inequality follows from the definition of V_s. Q.E.D.

Proof of Theorem 2:

(Step 1) The proof of continuity is standard, and thus omitted.

(Step 2) Consider an increase in b_k or in u_k, or a decrease in c_k. Let θ' denote the new vector of parameters. Clearly, we must have that $V_s(\Pi^*(\theta), \theta') \geq V_s^*(\theta)$ for all s. If $\Pi^*(\theta') = \Pi^*(\theta)$ we are done. If not, the result follows from lemma 2.

(Step 3) Let θ' denote the vector of parameters obtained by decreasing p_k by Δ. Consider a (probabilistic) decision rule $\widetilde{\Pi}$ defined as follows: (i) $\widetilde{\Pi}_s = \Pi_s^*$ for all $s \neq k$, (2) $\widetilde{\pi}_k^R = \pi_k^{*R}(\theta)$, (3) $\widetilde{\pi}_k^A = \frac{1-p_k}{1-p_k+\Delta}\pi_k^{*A}(\theta)$, and (4) $\widetilde{\pi}_k^U = \pi_k^{*U} + \frac{\Delta}{1-p_k+\Delta}\pi_k^{*A}(\theta)$. It is straightforward to verify that $V_s(\widetilde{\Pi}, \theta') = V_s^*(\theta)$ for all s. If $\widetilde{\Pi} = \Pi^*(\theta)$ we are done. If not, the result follows from lemma 2. Q.E.D.

Proof of Theorem 3:

(Step 1) The proof of (i) and (ii) follows from a straightforward application of lemma 1 and theorem 2. For example, consider an increase in b_k. By theorem 2 the value function weakly increases in every state. By lemma 1, usage weakly increases in states 0 to $k-1$ and weakly decreases in states $k+1$ to S.

(Step 2) Consider first a θ' that is equal to θ except for p_k and/or c_k. We claim that $U \in \chi_k^*(\theta) \Rightarrow \Pi^*(\theta) = \Pi^*(\theta')$. Since $U \in \chi_k^*(\theta)$ implies that $V_s^*(\Pi^*(\theta), \theta) = V_s^*(\Pi^*(\theta), \theta')$, the claim then follows (6) to (9).

(Step 3) Now suppose that $U \notin \chi_k^*(\theta)$ and that θ' is equal to θ except for p_k. By step 2, $U \notin \chi_k^*(\theta')$. Thus, $\chi_k^*(\theta') = \{A\}, \{A, R\}$, or $\{R\}$. We claim that there exists $\overline{p}_k \in [0, 1]$ such that (1) $\chi_k^*(\theta') = A$ if $p_k' < \overline{p}_k$, (2) $\chi_k^*(\theta') = R$ if $p_k' > \overline{p}_k$, and (3) $\chi_k^*(\theta') = \{A, R\}$ if $p_k' = \overline{p}_k$. To show this note that $R \in \chi_k^*(\theta')$ and $p_k'' > p_k'$ implies that $\chi_k^*(\theta'') = R$. This follows from (8) and the fact that $V_s^*(\Pi^*(\theta'), \theta') = V_s^*(\Pi^*(\theta), \theta'')$ for all s. The claim then follows since this establishes the existence of a lower threshold $\overline{p}_k$ for R.

(Step 4) Finally suppose that $U \notin \chi_k^*(\theta)$ and that θ' is equal to θ except for c_k. An argument analogous to the one in step 3 shows that there exists $\overline{c}_k \geq 0$ such that (1) $\chi_k^*(\theta') = R$ if $c_k' < \overline{c}_k$, (2) $\chi_k^*(\theta') = A$ if $c_k' > \overline{c}_k$, and (3) $\chi_k^*(\theta') = \{A, R\}$ if $c_k' = \overline{c}_k$.

(Step 5) To conclude the proof note that steps 2 and 3 imply that usage weakly decreases with p_k, and steps 2 and 4 imply that usage weakly increases with c_k.

Proof of Theorem 4:

(i) Follows directly from step 2, in the proof of Theorem 3.

(ii) Is a direct application of lemma 1 and theorem 2. Consider, for example, an increase in p_k and let θ' denote the new parameter. By theorem 2 the value function weakly decreases in every state. By lemma 1, usage weakly decreases in states $s < k$. This implies that the $\Pi_n^*(\theta') = R$ and thus the value functions for all states $s \leq n$ also remain unchanged. Finally, conditions (6) to (9) imply that the optimal decision rule for state s also remains unchanged.

(iii) Analogous to case (ii).

(iv) Let θ' denote the new parameter. The continuity of the value function, and conditions (6) to (9), imply that the optimal decision rule in state k is unaffected by local changes in p_k and b_k. Given that $\chi_k^*(\theta) = \{R\}$, this implies that $V_s^*(\Pi^*(\theta), \theta') = V_s^*(\Pi^*(\theta), \theta)$ for all s. As a result, the value function and optimal decision rule also remain unchanged.

(v)-(vii) Analogous to case (iv).

To establish the final step note that the continuity of the decision rule, and conditions (6) to (9), imply that the optimal decision rule in state s is unaffected by local changes in any of the parameters. Unlike in the previous cases, these changes may affect the value functions.

Proof of Theorem 5:

(Step 1) Let $\theta^\tau = (u_s^\tau, b_s^\tau, c_s^\tau)_{s=0}^S$ denote the parameters for the taxation regime (including the lump-sum transfers), $\Pi^*(\theta^\tau)$ and $V_s^*(\theta^\tau)$ denote the optimal decision rule and value function in the tax regime, and T_s denote lump-sum transfer that agents receive in state s. We claim that, for all s, $V_s^{*\tau}(\theta) \leq V_s(\Pi^*(\theta^\tau), \theta)$. To show this it suffices to show that, in each state s, the immediate payoff generated by action $\Pi_s^*(\theta^\tau)$ is less or equal in environment θ^τ than in environment θ. There are three possible cases:

(case 1) $\Pi_s^*(\theta^\tau) = U$. In this case $T_s = \tau$ and we have that

$$u_s^\tau + b_s^\tau = w_s(y_s + T_s - (q + \tau), 1) = w_s(y_s - q, 1) = u_s + b_s.$$

(case 2) $\Pi_s^*(\theta^\tau) = R$. In this case $T_s = 0$ and we have that

$$u_s^\tau - c_s^\tau = w_s(y_s + T_s - r_s, 0) = w_s(y_s - r_s, 1) = u_s - c_s.$$

(case 3) $\Pi_s^*(\theta^\tau) = A$. In this case $T_s = p_s \tau$ and we have that

$$
\begin{aligned}
p_s(u_s^\tau + b_s^\tau) + (1 - p_s)u_s^\tau &= p_s(\phi_s(1) + v_s(y_s + T_s - (q + \tau))) + (1 - p_s)(\phi_s(0) + v_s(y_s + T_s)) \\
&= p_s\phi_s(1) + (1 - p_s)\phi_s(0) + p_s v_s(y_s - q - (1 - p_s)\tau) + (1 - p_s)v_s(y_s + p_s\tau) \\
&\leq p_s\phi_s(1) + (1 - p_s)\phi_s(0) + p_s v_s(y_s - q) + (1 - p_s)v_s(y_s) \\
&= p_s(u_s + b_s) + (1 - p_s)u_s.
\end{aligned}
$$

(The inequality follows from the following argument. Let $\xi_s(\tau) \equiv p_s v_s(y_s - q - (1 - p_s)\tau) + (1 - p_s)v_s(y_s + p_s\tau)$. Then $\xi_s'(\tau) = p_s(1 - p_s)(v_s'(y_s + p_s\tau) - v_s'(y_s - q - (1 - p_s)\tau)) < 0$ for all $\tau \geq -q$.

(Step 2) To conclude the proof we claim that $V_s^{*\tau}(\theta) \leq V_s^*(\theta)$, with strict inequality if $\Pi_s^{*\tau}(\theta) \neq \Pi_s^*(\theta)$. This follows directly from step 1 and lemma 2.

Proof of Theorem 6:

(Step 1) Let $\theta^\sigma = (u_s^\sigma, b_s^\sigma, c_s^\sigma)_{s=0}^S$ denote the parameters for the subsidy regime (including the lump-sum taxes), $\Pi^*(\theta^\sigma)$ and $V_s^*(\theta^\sigma)$ denote the optimal decision rule and value function in the subsidy regime, and T_s denote lump-sum tax that agents pay in state s. We claim that, for all s, $V_s^{*\sigma}(\theta) \leq V_s(\Pi^*(\theta^\sigma), \theta)$. To show this it suffices to show that, in each state s, the immediate payoff generated by action $\Pi_s^*(\theta^\sigma)$ is less or equal in environment θ^σ than in environment θ. There are two possible cases:

(case 1) $\Pi_s^*(\theta^\sigma) = U$ or A. In this case $T_s = 0$ and it trivially holds that

$$u_s^\sigma + b_s^\sigma = u_s + b_s \text{ and } p_s(u_s^\sigma + b_s^\sigma) + (1 - p_s)u_s^\sigma = p_s(u_s + b_s) + (1 - p_s)u_s.$$

(case 2) $\Pi_s^*(\theta^\sigma) = R$. In this case $T_s = \sigma_s$ and we have that

$$u_s^\sigma - c_s^\sigma = w_s(y_s - T_s - (r_s - \sigma_s), 0) = w_s(y_s - r_s, 1) = u_s - c_s.$$

(Step 2) $V_s^{*\sigma}(\theta) \leq V_s^\sigma(\theta)$, with strict inequality if $\Pi_s^{*\sigma}(\theta) \neq \Pi_s^*(\theta)$. This follows directly from step 1 and lemma 2.

References

[1] Abratt, R. and S. Goodey (1990) "Unplanned Buying and In-Store Stimuli in Supermarkets," Managerial and Decision Economics," 111-21.

[2] Barkow, Jerome, Leda Cosmides, and John Tooby (1995) *The Adapted Mind : Evolutionary Psychology and the Generation of Culture.* Oxford: Oxford University Press.

[3] Bechara, A., A. Damasio, H. Damasio, and S. Anderson (1994) "Insensitivity to Future Consequences Following Damage to Human Prefrontal Cortex," Cognition, 7-15.

[4] Bechara, A., D. Tranel, A. Damasio, and H. Damasio (1996) "Failure to Respond Autonomically to Anticipated Future Outcomes Following Damage to the Prefrontal Cortex," Cerebral Cortex, 215-25.

[5] Bechara, A., D. Tranel, A. Damasio, and H. Damasio (1997) "Deciding Advantegeously Before Knowing the Advantageous Strategy," Science, 1293-95.

[6] Becker, Gary and Kevin Murphy (1988) "A Theory of Rational Addiction," Journal of Political Economy, 96(4), 675-700.

[7] Bernheim, Douglas and Antonio Rangel (2002) "Neurofoundations of Decision-Making: Cognition, Emotions, and Characterization Failure", Stanford manuscript.

[8] Berridge, K. (1996) "Food Reward: Brain Substrates of Wanting and Liking," Neuroscience and Biobehavioral Reviews, 20(1):1-25

[9] Berridge, K. (1999) "Pleasure, Pain, Desire, and Dread: Hidden Core Processes of Emotion," in *Well-Being: The Foundations of Hedonic Pscyhology*, D. Kaheman, E. Diener, and N. Schwarz (eds.), 525-57

[10] Chaloupka, F. and K. Warner (2001) "The Economics of Smoking", in J. Newhouse and D. Cutler (eds.), *Handbook of Health Economics*, North-Holland.

[11] Damasio, Antonio (1994) *Descartes Error: Emotion, Reason, and the Human Brain.* New York: Putnam.

[12] Davis, M. (1992a) "The Role of the Amygdala in Fear and Anxiety," Annual Review of Neuroscience, 353-75.

[13] Davis, M. (1992b) "The Role of the Amygdala in Conditioned Fear," In J.P. Aggleton (ed.), *The Amygdala: Neurobiological Aspects of Emotion, Memory, and Mental Disfunction,* New York: Wiley-Liss.

[14] Gardner, Eliot and James David (1999) "The Neurobiology of Chemical Addiction," in *Getting Hooked: Rationality and Addiction,* edited by Jon Elster and Ole-Jorgen Skog. Cambridge: Cambridge University Press.

[15] Gawin, F.H. (1991) "Cocaine addiction: Psychology and neurophysiology," Science, 1580-6.

[16] Gazzaniga, M. (1990). Transcript of interview published in the *National Review.*

[17] Gazzaniga, M. (1994). Transcript of interview published in the *National Review.*

[18] Goldstein, A. (2001) *Addiction: From Biology to Drug Policy.* Second Edition. New York: Oxford University Press.

[19] Goldstein, A. and H. Kalant (1990) "Drug Policy: Striking the Right Balance," Science 249: 1513-21.

[20] Gratton. A. and R. Wise (1994) "Drug- and Behavior-associated Changes in Dopamine-related Electrochemical Signals During Intravenous Cocaine Self-Administration in Rats," The Journal of Neuroscience, 4130-46.

[21] Gruber, Jonathan, and Botond Koszegi (2001) "Is Addiction "Rational"? Theory and Evidence," Quarterly Journal of Economics, 116(4): 1261-1303 .

[22] Gul, Faruk and Wolfgang Psendorfer (2001a), "Temptation and Self-Control", Econometrica 69(6): 1403-35.

[23] Gul, Faruk and Wolfgang Psendorfer (2001b) "A Theory of Addiction," Princeton mimeo.

[24] Harris, J.E. (1993) *Deadly Choices: Coping with Health Risks in Everyday Life.* New York: Basic Books.

[25] Heath, R. (1964) "Pleasure response of human beings to direct stimulation of the brain: Physiologic and psychodynamic considerations," in R.G. Health (ed.) *The Role of Pleasure in Behavior,* New York: Hoeber, pp. 219-43.

[26] Helmuth, L. (2001) "Beyond the Pleasure Principle," Science, 294:983-84.

[27] Holden, C. (2001a) "'Behavioral' addictions: Do they exist?" Science, 294:980-82.

[28] Holden, C. (2001b) "Zapping Memory Centers Triggers Drug Craving," Science, 292:1039

[29] Hser, Y.M., D. Anglin, and K. Powers (1993) "A 24-year follow-up study of California narcotics addicts," Archives of General Psychiatry, 50:577-84.

[30] Hung, Angela (2000) "A Behavioral Theory of Addiction," Caltech, manuscript.

[31] Huxley, Aldus (1998) *Brave New World.* Harper Perennial. Reprint Edition.

[32] Janis, I. (1967) "Effects of fear arousal on attitudinal change," Advances in Experimental Psychology, 167-224.

[33] Klein, S. (2000) *Biological Psychology.* Upper Saddle River, New Jersey: Prentice Hall International.

[34] Knutson, B., et. al. (2001a) "Dissociation of reward anticipation and outcome with event-related fMRI", Brain Imaging, 12(17):3683-86.

[35] Knutson, B. et. al. (2001b) "Anticipation of Increasing Monetary Reward Selectively Recruits Nucleus Acumbens," The Journal of Neuroscience, 21, RC159.

[36] Koob, G. and M. LeMoal (1997) "Drug Abuse: Hedonic Homeostatic Dysregulation", Science 278, October, 52-8.

[37] Laibson, David (1997) "Golden Eggs and Hyperbolic Discounting," Quarterly Journal of Economics, (112):443-77.

[38] Laibson, David (2001) "A Cue-Theory of Consumption," Quarterly Journal of Economics, 116(1): 81-120.

[39] LeDoux, Joseph (1992) "Emotion and the Amygdala." In J.P. Aggleton (ed.) *The Amygdala: Neurobiological Aspects of Emotion, Memory and Mental Dysfunction,* pp. 339-51. New York: Wiley-Liss.

[40] LeDoux, Joseph (1993) "Emotional Networks in the Brain," In Michael Davis and Jeannette Haviland (eds.), *Handbook of Emotions,* New York: The Guildford Press.

[41] LeDoux, Joseph (1998) *The Emotional Brain : The Mysterious Underpinnings of Emotional Life.* Touchstone books.

[42] Loewenstein, George (1996) "Out of Control: Visceral Influences on Behavior," Organizational Behavior and Human Decision Processes, 65(3) 272-92.

[43] Loewenstein, George (1999) "A Visceral Account of Addiction," in Jon Elster and Ole-Jorgen Skog (eds.) *Rationality and Addiction*. Cambridge: Cambridge University Press.

[44] Loewenstein, George, and Jennifer S. Lerner (2001) "The Role of Affect in Decision Making," in R.J. Davidson, H.H. Goldsmith, and K.R. Scherer (eds.) *Handbook of Affective Science*. Oxford: Oxford University Press.

[45] Loewenstein, George, Ted O'Donoghue, and Matthew Rabin (2000) "Projection Bias in Predicting Future Utility," mimeo.

[46] Massing, Michael. *The Fix*. Los Angeles: University of California Press, 2000.

[47] MacCoun, R. and P. Reuter (2001) *Drug War Heresies: Learning from Other Vices, Times, and Places.* Cambridge University Press.

[48] MacLean, P.D. (1977) "The triune brain in conflict," Psychotherapy and Psychosomatics, 28, 207-220.

[49] Metcalfe, Janet, and Walter Mischel (1999) "A Hot/Cool-System Analysis of Delay of Gratification: Dynamics of Willpower," Psychological Review, 106(1):3-19.

[50] Miron, J. and J. Zwiebel (1995) "The Economic Case Against Drug Prohibition," Journal of Economic Perspectives, 9(4):175-92.

[51] Mischel, W. (1974) "Processes in delay of gratification," In *Advances in Experimental Social Psychology, volume 7,* D. Berkowitz editor, 249-272.

[52] Mischel, W. and B. Moore (1973) "Cognitive Appraisals and Transformations in the Delay of Gratification," Journal of Personality and Social Psychology, 28:172-9.

[53] Michel, W., Y. Shoda, and M. Rodriguez (1992) "Delay of Gratification in Children," in *Choice over Time,* G. Loewenstein and Jon Elster editors, New York: Rusell Sage

[54] O'Brien, C. (1976) "Experimental analysis of conditioning factors in human narcotic addiction," Pharmacological Review, 25:533-43.

[55] O'Brien, C. (1997) "A Range of Research-Based Pharmacotherapies for Addiction," Science, 278:66-70.

[56] O'Donoghue, Ted, and Mathew Rabin (1999), "Doing It Now or Later," American Economic Review, (89):103-24.

[57] O'Donoghue, Ted, and Mathew Rabin (2000), "Addiction and Present-Biased Preferences," manuscript, Berkeley.

[58] Panksepp, J. (1998) *Affective Neuroscience: The Foundations of Human and Animal Emotions.* New York: Oxford University Press.

[59] Pickens, R. and W.C. Harris (1968) "Self-administration of d-amphetamine by rats," Pscyhopharmacologia, 158-63.

[60] Olds, J. and P. Milner (1954) "Positive reinforcement produced by electrical stimulation of septal area and other regions of rat brain," Journal of Comparative and Physiological Psychology, 419-27.

[61] Orphanides, Athanasios, and David Zervos (1995) "Rational Addiction with Learning and Regret," Journal of Political Economy, 103: 739-58.

[62] Robins, L. (1994) "Vietnam Veterans' Rapid Recovery from Heroin Addiction: a Fluke or Normal Expectation," Addiction, 1041-54.

[63] Robins, L., D. Davis, and D. Goodwin (1974) "Drug Use by U.S. Army Enlisted Men in Vietnam: a Follow-up on their Return Home," American Journal of Epidemiology, 235-49.

[64] Robinson. T. and K. Berridge (1993) "The Neural Basis of Drug Craving: An Incentive-Sensitization Theory of Addiction," Brain Research Reviews, 18(3):247-91.

[65] Rolls, E. (1999) *The Brain and Emotion.* New York: Oxford University Press.

[66] Romer, Paul (2000) "Thinking and Feeling," AmAmerican Economic Review Papers and Procedings, 90(2): 439-43

[67] Schelling, T (1984) "Self-Command in practice, policy, and in a theory of rational choice," American Economic Review, 1-11.

[68] Thaler, R. and H. Shefrin (1981) "An Economic Theory of Self-Control," Journal of Political Economy, 89(2):392-406.

[69] Ungless, M., J. Whistler, R. Malenka, and A. Bonci (2001) "Single cocaine exposure in vivo induces long-term potentiation in dopamine neurons," Nature, 411(31):583-87.

[70] Volkow, N.D. (1997) "The role of the dopamine system in addiction," Hospital Practice (Special reports), April: 22-26.

[71] Volkow, N.D., Wang, G.-J., Fowler, J.S. (1997) "Imaging studies of cocaine in the human brain and studies of the cocaine addict. In: Imaging Brain Structure and Function, " in Annals of the New York Academy of Sciences, Lester D.S., Felfer C.C., Lewis E.N. (eds), 820:41-53.

[72] Vorel, S. and E. Garner (2001) "Drug Addiction and the Hyppocampus," Science, 294(9):1235a

[73] Vorel, S., et. al. (2001) "Relapse to Cocaine-Seeking After Hippcampal Theta Burst Stimulation," Science, 292:1175-78.

[74] Wegner, Daniel (1994) *White Bears and other unwanted thoughts: Suppression, obsession, and the psychology of mental control.* New York: Guildford Press.

[75] Wise, R.A. (1996) "Addictive Drugs and Brain Stimulation Reward," Annual Review of Neuroscience, 19:319-40.