

A Model of Natural Recovery from Addiction

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Abstract

A substantial fraction of individuals with a history of pathological addiction quits without professional assistance. This paper proposes a model of natural recovery, this widespread yet unexplained aspect of addictive behavior. The model is grounded on the neuroscience and on recent theories regarding addiction as a progressive susceptibility to stochastic environmental cues that can trigger mistaken usage. We introduce a "cognitive appraisal" process in individual decision making depending on past addiction experiences as well as on the future expected consequences of addictive consumption. Individual choice is affected in two ways: the reward from use decreases as the decision maker grows older and cognitive incentives emerge reducing the probability of making mistakes. While allowing for cue-triggered mistakes in individual decision making, our model recovers a role for other factors, such as subjective self evaluation, with implications for social policy and the treatment of drug and alcohol dependence.

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Key words: Addiction models, natural recovery, cognitive policy.

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...isn't it remarkable that the behavior of even reasonably intelligent individuals can be as idiosyncratic, seemingly irrational, and sometimes patently counterproductive as it often appears to be?...

Cohen and Blum (2002)

1 Introduction

Addiction is defined as the consequence of repeated use of psychoactive drugs. It is characterized principally by a loss of control over drug seeking behavior with harmful effects on the individual and a high probability of relapse even months or years after cessation of drug taking (Volkow and Fowler, 2000; Kelley, 2004; Weiss, 2005). The main problem is to understand how this phenomenon “moves”, meaning how the various components of its multifactoriality (individual, substance and environment) can trigger the start, sustain recurrence or generate the frustrating relapse.

Economists have developed theories to model addiction, their interest stemming from social costs and externalities generated by the consumption of addictive substances. These theories can be loosely classified as generalizations of the rational addiction model (Becker and Murphy, 1988). Generalizations allow for the presence of random cues that increase the marginal utility of consumption (Laibson, 2001); “projection bias” (Loewenstein *et al.*, 2003); present-biased preferences and sophisticated or naive expectations (Gruber and Koszegi, 2001); “temptation” (Gul and Pesendorfer, 2001) where preferences are defined both over chosen actions and over actions not chosen. Bernheim and Rangel (2004), in an attempt to harmonize economic theory with evidence from psychology, the neuroscience and clinical practice, regard addiction as a progressive susceptibility to stochastic environmental cues that can trigger mistaken usage. This explains, in this model¹, the relationship between behavior and the characteristics of the user, substance and the environment. Neuroscience and clinical practice have indeed shown that addictive substances systematically interfere with the proper operation of a process used by the brain to forecast near term hedonic rewards and lead to strong impulses to consume that may interfere with higher cognitive control. In

¹Bernheim and Rangel analysis is related to previous work by G. Loewenstein (1996, 1999) on the “cold-to-hot empathy gap”.

this case individual consumption choices are sometimes driven by a rational decision making process, sometimes by strong impulses leading to mistakes defined as a divergence between preferences and choices.

These theories explain several patterns of addictive behavior, but there is one aspect left unexplained which is spontaneous remittance also known as natural recovery. Although addiction is defined as a chronic and persistent disease by the scientific community (see e.g. the American Psychological Association's Diagnostic and Statistical Manual of Mental Disorders, known as the DSM-IV), recent longitudinal studies have called into question whether this is an accurate representation (Slutske, 2006). Clinical practice shows that natural recovery characterizes a substantial fraction of individuals with a history of pathological addiction and that this is not an infrequent pattern of behavior in long term addicts, but the reasons for it are still to be understood.

This paper offers two contributions. First, it tries to solve the interesting puzzle of natural recovery, by identifying some of its determinants and its dynamics. Second, it tries to throw up a bridge between behavioral and "mainstream" economic models of addiction as cognitive processes play a relevant role to explain natural recovery even in individuals with an important addiction history.

Starting from the hot/cool interactive system developed by Metcalfe and Mischel (1999) we introduce a "Cognitive Appraisal" function, depending on past addiction history as well as on the expected future consequences of addictive consumption. This affects the decision process in two ways: it erodes the payoff from use as the decision maker grows older and it increases the cognitive control competing with the hedonic impulses to use, thus reducing the probability of entering the impulsive decision making process. Performance analysis of the extended model is carried out.

The remaining of the paper is structured as follows. Section 2 and 3 provide a clinical description of addiction and of natural recovery. Section 4 contains the model of natural recovery starting from recent theories. Section 5 concludes.

2 The neuroscience of addictive behavior

In human beings drugs produce an increase of dopamine concentration at target-cells' receptor levels, as they stimulate the nigrostriatal (controlling motor coordination) and corticolimbic (controlling emotions and cognitive abilities) dopaminergic systems (Wise, 2004). These cerebral systems have evolved not to entertain addictive substances, but to ensure the survival of the individual by controlling basic functions such as mating, searching for food and water, etc. Once these systems are engaged by natural rewards (food or sex for example) or by addictive substances (Kelley, 2004; Nestler, 2005) dopamine release in the nucleus accumbens

and in other cerebral sites increases, causing specific emotional states (for example, euphoria) that are powerful drivers and reinforce that behavior. Addictive substances have an advantage over natural rewards: they produce a higher dopamine concentration by stimulating the (wanting) system more powerfully and for longer periods (Hyman, 2005). The individual is thus induced to repeat such positive experiences (or avoid them when negative), precisely because he associates them to their hedonic responses (Kelley and Berridge, 2002; Bechara, 2005; Kalivas and Volkow, 2005). However, there is another important difference between natural rewards and drugs of abuse. In the case of natural rewards, a habit develops after repeated use which reduces the importance of the experiential act. The quality and quantity of the gained pleasure diminishes. On the contrary, addictive substances activate each time a similar hedonic response (Berridge and Robinson, 2003).

Chronic substance abuse induces profound alterations of the cerebral mechanisms just mentioned which “force”, in a way, the user to make compulsory choices. By powerfully activating dopamine transmission, drugs excessively reinforce the associated learning process, ending up by constraining the individual’s behavioral choices (Berke and Hyman, 2000). In other words, drugs seem to affect the basic forecasting mechanism, a simple and fast system for learning correlations between current conditions, decisions and short term rewards². There is a growing consensus in the neuroscience according to which addiction results from the impact addictive substances have on the HFM. With repeated use of a substance, the cues associated with past consumption cause the HFM to forecast exaggerated pleasure responses, creating a disproportionate impulse to use leading to mistakes in decision making. The pleasure following use, the excessive and rapid hedonic expectation induced by the HFM, the progressive failing of the frontal cortex to counterbalance with rational choices the more alluring offer of drugs, all portray a process that invariably regenerates itself and seems to have no end (Kelley and Berridge, 2002; Berridge, 2004).

Although drug addiction seems to lead to just one possible result, for still unclear reasons often the patient stops participating in the ineluctable dynamics of her/his case and ceases to have this compulsion for the drug. One could say that the multifactoriality sustaining drug addiction sometimes ceases to offer those profits or conveniences considered up till then as indispensable. When this happens without professional help, such phenomenon gives rise to the nice puzzle of natural recovery.

²Bernheim and Rangel (2004) call this process Hedonic Forecasting Mechanism (HFM henceforth).

3 Natural Recovery

Epidemiological studies considering pathways out of alcohol abuse give evidence that the majority of quitting taking place without professional assistance in various countries reveals rates between 66.7% in Germany to 77% in Canada (Bischof *et al.*, 2003). Other studies show that, although there may be differences in the ways in which it occurs, spontaneous remittance characterizes the whole spectrum of drugs such as alcohol (Cunningham *et al.* 2006; Bischof *et al.* 2000; Weisner *et al.* 2003; Matzger *et al.* 2005; Bischof *et al.*, 2003), marijuana (Copersino *et al.*, 2006), heroin (Waldorf and Biernacki, 1979), binge eating, smoking, sex and gambling (Hanninen *et al.*, 1999; Nathan, 2003) and that the rate of natural recovery may be much higher than conventional wisdom would suggest. Recent longitudinal studies have highlighted that a substantial fraction of individuals with a history of pathological gambling have recovered from their problems without professional help (Slutske, 2006) calling into question the definition of addiction as a chronic and persistent disorder as given by the DSM-IV.

Despite this evidence, natural recovery remains an unexplained phenomenon. Yet it is of interest to different major disciplines, such as economics, psychology and sociology. Natural recovery may occur in at least four different ways: (i) cold turkey quitting due to an exogenous shock; (ii) cold turkey quitting happening without an exogenous shock; (iii) gradual quitting occurring after a period of continuous decrease in consumption; (iv) recovery occurring after a series of failed attempts to quit. We are particularly interested in cases (ii), (iii) and (iv) and hypothesize that quitting is the manifestation of a long-term process of "self appraisal" that ultimately brings to quitting consumption of the addictive substance.

Clinical and experimental research has studied natural recovery from substance abuse since the mid-1970s (Vaillant, 1982; Klingemann, 1991) focusing on triggering mechanisms, maintenance factors and on trying to identify common reasons for change in substance use (Prochaska, Di Clemente and Norcross, 1992). There are, however, very few models of decision making describing pathways to natural recovery. The Becker and Murphy (1988) model generates cold turkey quitting through exogenous shocks or stressful events. Suranovic *et al.* (1999) extend Becker and Murphy to generate cold turkey quitting of cigarettes' smoking without relying on exogenous shocks or stressful events. The motivation to quit is based instead on changes in the addict's perspective as he grows older. In addition, this model shows that some individuals may quit addiction by gradually reducing consumption over time. These results are obtained by explicitly taking into account the withdrawal effects (quitting costs) experienced when users try to quit and by explicit recognition that the negative health effects of addiction generally appear late in an individual's life. Both models presuppose a standard inter temporal decision making implying a complete alignment of choices and

time consistent preferences, thereby denying the possibility of mistakes. Insights from psychology and neuroscience have led to new theories of addiction trying to bridge the gap between neuroscience and decision making and depicting addiction as a progressive susceptibility to stochastic environmental cues triggering mistaken usage (Loewenstein, 1999; Bernheim and Rangel, 2004). They do not, however, explicitly model pathways to natural recovery.

We extend the Bernheim and Rangel (2004) model by introducing a process that we call "cognitive appraisal" incorporating future expected losses as well as the past addiction history. Such process leads to a reduction of the reward from use as the decision maker grows older and it increases cognitive incentives competing with the HFM, thus reducing the probability of making mistakes. A similar mechanism, based on the struggle between the impulsive and reflective systems, is proposed by Bechara (2005) in his neurocognitive theory of decision making processes. We also explore the role of other factors, such as learning and individual heterogeneity. This explanation places a high value on policy measures increasing cognitive control such as education, creation of counter cues and policies that help the accumulation of social capital, but it does not rule out the effectiveness of more conventional policy measures, such as regulation or taxation of legal addictive substances which could still play a role.

3.1 Reasons for spontaneous quitting

Matzger *et al.* (2005), in a study of the reasons for drinking less, consider that triggering mechanisms, the interpersonal and environmental influences that cause a person to move from problematic alcohol use to sustained abstinence or non-problematic use, can be varied and multidimensional and often involve a combination of both short and long term pressures. In this study two groups of problem drinking adults, who reported drinking less at the one year follow up, were identified in Northern California. The first group came from a probability sample in the general population; the second was originated through a survey of consecutive admissions to public and private alcohol and drug problems. A logit model was then used to assess the determinants of sustained remission from problem drinking. Results showed that the two most frequently endorsed reasons for cutting down were the same for both groups and were long-term reasons: self-evaluation, i.e. weighing the pros and cons of drinking and not drinking and experiencing a major change in lifestyle. Drinking causing health problems was also an important reason for quitting. Self-evaluation implies that recovery is not necessarily triggered by negative or traumatic events, but alternatively comes about through a period of self-reflection. Interventions by medical personnel and family members were either non-significant predictors or significantly negatively related to sustained improvement for both the general population and treated drinkers.

Cunningham *et al.* (2005) give support to both the "cognitive appraisal" and the "life events" motivations for quitting. They noted that individuals who recovered without treatment went through a process of cognitive appraisal (also known as the motivational explanation for quitting) in which they weighed the pros and cons of drinking and not drinking and decided that the pros outweighed the cons. Anticipated costs and benefits of change is thus one means of measuring the respondents' motivational explanation for quitting. The "life events" motivation is instead based on past life events. It is hypothesized that addicts' life events prior to and after their quit attempt are related to successful quitting attempts. Respondents experiencing the greatest reduction in their negative life events pre to post quit attempts were hypothesized to be most likely to have successfully reduced or quit their addiction. Nathan (2003), in his study of natural recovery from pathological gambling, also argues that self-changers have less severe drinking histories and fewer symptoms of dependence.

4 The Model

We start from Bernheim and Rangel (BR henceforth) addiction theory. This is based on the following premises: a) consumption among addicts is frequently a mistake; b) previous experience with an addictive good sensitizes an individual to environmental cues that trigger mistaken usage; c) awareness of sensitivity to cue-triggered mistakes produces attempts to manage the process with some degree of sophistication.

The model involves a decision maker (DM) living for an infinite number of discrete periods who can operate either in a cold or hot mode. Time is discrete, indexed by the nonnegative integers, $t \in \{0, 1, 2, \dots\}$. In each time period, t , the DM makes two decisions in succession. First, he selects a lifestyle a from the set $\{E, A, R\}$ (e.g. going to a bar or staying at home watching TV or reading a book). If lifestyle E , "exposure", is chosen there is a high likelihood that the DM will encounter a large number of substance-related cues. Activity A , "avoidance", entails fewer substance-related cues and may also reduce sensitivity to environmental cues. Activity R , "rehabilitation", implies a commitment to clinical treatment, the cost of which is r_s , and it may further reduce exposure and sensitivity to substance-related cues. Then she allocates resources between a potentially addictive good/substance, $x \in \{0, 1\}$, the price of which is q , and a non addictive good ($e_s \geq 0$). By assumption the DM can not borrow or save. Each period is entered in cold mode and the DM chooses his lifestyle rationally. This choice, along with the addictive state, s , determines the probability p_s^a with which he encounters cues that trigger the hot mode. With some transition probability p_T , consumption of the addictive substance in state s at time t moves the individual to a higher addictive

state, $s + 1$ at time $t + 1$, and abstention moves him to a lower addictive state $s - 1$ at time $t + 1$. There are $S + 1$ addictive states labeled $s = 0, 1, \dots, S$. The system dynamics is described by the evolution of state s_t according to the following equation:

$$s_{t+1} = \begin{cases} \min\{p_T (s_t + 1) + (1 - p_T)s_t, S\} & \text{if } x_t = 1, a_t \in \{E, A\} \\ \max\{1, p_T (s_t - 1) + (1 - p_T)s_t\} & \text{if } x_t = 0, a_t \in \{E, A, R\} \end{cases} \quad (1)$$

Equation (1) implies that consumption in state s leads to state $\min\{S, s_t + 1\}$ in the next period with probability p_T . No use leads to state $\max\{1, s_t - 1\}$ with probability p_T from state $s > 1$ and to state $s = 0$ from state $s = 0$. The volume of substance related environmental cues encountered, $c(a, \omega)$, depends on the lifestyle and on an exogenous state of nature ω drawn randomly from a state space Ω according to some probability measure μ . We assume that the function $c(a, \omega)$ is driven by a normally distributed random process with variance and mean depending on the life style a . Impulses $c(a, \omega)$ place the DM in hot mode when their intensity $M(c, s, a, \omega)$, denoting the DM's sensitivity to the cues, exceeds some exogenously given threshold M^T . Since people become sensitized to cues through repeated use $M(c, s', a, \omega) < M(c, s'', a, \omega)$ for $s'' > s'$ and $M(c, 0, a, \omega) < M^T$. Moreover $M(c, s, R, \omega) \leq M(c, s, A, \omega) \leq M(c, s, E, \omega)$, i.e. the lifestyle affects the DM sensitization to environmental cues. When $M(c, s, a, \omega) > M^T$ the DM enters the HOT mode. Moreover we assume the power function M strictly increasing and twice continuously differentiable in s . We choose a logistic specification for M :

$$M(c(a, \omega_a), s, a, \omega_a) = c(a, \omega_a) + \frac{M_0 e^{\lambda s}}{1 + M_0 (e^{\lambda s} - 1)} \quad (2)$$

where $a \in \{R, E, A\}$ and $M_0 = M(s = 0)$ and λ is the growth rate of the HFM generated impulses. Di Chiara (2002) defines four different phases of addiction, delimited in Figure 1 by vertical dotted lines: controlled drug use, drug abuse, drug addiction, post-addiction stage. In this figure M is measured on the vertical axis and s is measured on the horizontal axis. In the first stage, as a result of curiosity, peer pressure, social factors, personality traits etc. (lifestyles a and environmental cues c) the DM comes into contact with a drug. Sensitization facilitates further experimentation and increases the power of the HFM (M weakly increasing in s). At this stage the subject responds to the drug-related stimuli in a controlled manner. With repeated drug exposure the DM progressively enters the stage of drug abuse. In this stage sensitization becomes very powerful and drug-related stimuli are associated to craving (M strongly increasing in s). The stage of drug addiction is characterized by the preceding stage to which tolerance

and physical dependence are added (the slope of the M function starts decreasing). In the post-addiction stage abstinence as well as sensitization progressively disappear but the HFM-generated impulses remain active (saturating M function).

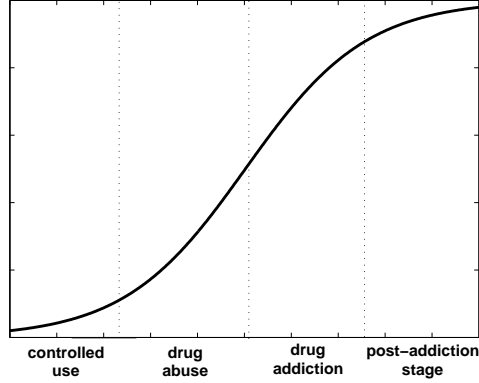


Figure 1: The deterministic portion of the M function.

Consider $T(s, a) = \{\omega \in \Omega : M(c, s, a, \omega) \geq M^T\}$. The DM enters the hot mode if and only if $\omega \in T(s, a)$. Moreover let $p_s^a = \mu(T(s, a))$ denote the probability of entering the hot mode at time t in addictive state s and lifestyle a . An increase in the addictive state s raises the likelihood of entering the hot mode at any moment, because the sensitivity to random environmental cues has increased. So at each time instant $p_{s+1}^a \geq p_s^a$, $p_0^a = 0$ and $p_s^E \geq p_s^A \geq p_s^R$.

In state s the DM receives an immediate hedonic payoff $w_s(e_s, x, a) = u(e_s) + v_s(x, a)$ where utility derived from non addictive goods, $u(e_s)$, is assumed to be separable from utility derived from addictive consumption. w_s is increasing, unbounded, strictly concave and twice differentiable with bounded second derivative in the variable e_s . Moreover $v_s(x, a) \equiv u_s^a + b_s^a$, where u_s^a represents the baseline payoff associated with successful abstention in state s and activity a and b_s^a represents the marginal instantaneous benefit from use the individual receives in state s after taking activity a . By the same assumption, at any instant $u_s^E > u_s^A \geq u_s^R$ and $u_s^E + b_s^E > u_s^A + b_s^A$. Future hedonic payoffs are discounted using an exponential discount factor δ . Choices in cold mode correspond to the solution of a dynamic stochastic programming problem with a value function $V_s(\theta)$ and Bellman equation equal to:

$$V_h(\theta) = \max_{(a,x) \in C} u_h^a + \sigma_h^{a,x} b_h^a + \delta \left[(1 - \sigma_h^{a,x}) V_{h-1}(\theta) + \sigma_h^{a,x} V_{h+1}(\theta) \right], \quad (3)$$

$$\begin{aligned}
& s.t. \\
& 0 \leq h \leq S, \\
& h - 1 = \max \{1, s - 1\}, \\
& h + 1 = \min \{S, s + 1\}
\end{aligned}$$

C is the set of decision states $\{(E, 1), (E, 0), (A, 0), (R, 0)\}$; $\sigma_s^{a,x}$ represents the probability of consuming the substance in state x with contingent plan (a, x) and θ is a vector specifying the model parameters. The stationarity of equation (3) follows from the assumption that the DM takes his decision at the beginning of each period.³

We are interested in the choice set $(E, 0)$. In this case impulses to use are not forcedly controlled through rehabilitation, but abstinence occurs for high enough M^T , the threshold level of the impulses' intensity required to defeat cognitive control. We look for mechanisms that decrease the probability of entering the hot mode and the convenience to use when in cold mode so that the DM is inclined to abstain from consumption for a reasonably long periods of time⁴.

4.1 Accounting for Expected Future Losses and Past Addiction Histories

We assume that consumption of addictive substances has negative effects as the addictive state s increases and introduce a loss function which accounts for past experiences with addictive goods and for the future negative effects of current addiction. Due to increasing awareness of both, the DM may experience a change of perspective as he grows older sufficient to induce quitting even without an exogenous shock or a stressful event occurring to generate this outcome.

Drawing from Suranovic *et al.* (1999) we assume the DM to be Y years old and $T(Y)$ is the number of years remaining representing a non addict's life expectancy at age Y . $T(Y)$ is linear in Y with $T'(Y) < 0$. An addict's life expectancy at age Y can be represented as $T(Y) - \beta H$ with β being a parameter weighting the reduction in life expectancy caused by H . The present value of an addict's expected future utility stream (V) from consumption at age Y can be defined as:

³BR show that this model generates a number of addiction patterns: *Unsuccessful attempts to quit* occur when there is an unanticipated or anticipated and sufficiently slow shift in parameters $\theta_s = (\mathbf{p}_s, \mathbf{u}_s, \mathbf{b}_s)$, from θ' to θ'' ; *Cue-triggered recidivism* associated with high exposure to relatively intense cues, e.g. high realizations of $c(a, \omega)$; *Self-described mistakes* the DM chooses $(E, 0)$ or $(A, 0)$ in cold mode, but then he enters the hot mode; *Self-control through pre-commitment* given by the choice $(R, 0)$ which implies a costly pre-commitment; *Self-control through behavioral and cognitive therapy* through choice $(A, 0)$ which implies costly cue avoidance.

⁴At least two years of sustained abstinence from use according to clinical practice.

$$V_{Y,H}(s) = \int_{t=Y}^{T(Y)+Y-\beta\left(\frac{H+s}{2}\right)} e^{-r(t-Y)} b_s dt \quad (4)$$

where r is the fixed discount rate; $e^{-r(t-Y)}$ is the discount factor at time t and b_s is the individual's expected utility of consuming the addictive good at time t . $\beta\left(\frac{H+s}{2}\right)$ is the average lost life caused by the maximum addictive state reached in the past and by the current addictive state s . For a DM aged Y and maximum addictive state H the present value of the expected future losses at time t is given by⁵:

$$L_{Y,H}(s) = V_{Y,H}(s) - V_{Y,H}(s+1) = \int_{T(Y)+Y-\beta\left(\frac{H+s+1}{2}\right)}^{T(Y)+Y-\beta\left(\frac{H+s}{2}\right)} e^{-r(t-Y)} b_s dt \quad (5)$$

Differentiation of equation (5) with respect to s leads to:

$$L'_{Y,H}(s) = -\frac{\beta}{2} e^{-r\left[T(Y)-\beta\left(\frac{H+s}{2}\right)\right]} b_{s,T(Y)+Y-\beta\left(\frac{H+s}{2}\right)} + \frac{\beta}{2} e^{-r\left[T(Y)-\beta\left(\frac{H+s+1}{2}\right)\right]} b_{s,T(Y)+Y-\beta\left(\frac{H+s+1}{2}\right)} \quad (6)$$

This is weakly positive because

$$e^{-r\left[T(Y)-\beta\left(\frac{H+s}{2}\right)\right]} < e^{-r\left[T(Y)-\beta\left(\frac{H+s+1}{2}\right)\right]}$$

and

$$b_{s,T(Y)+Y-\beta\left(\frac{H+s}{2}\right)} \leq b_{s,T(Y)+Y-\beta\left(\frac{H+s+1}{2}\right)}.$$

Future losses increase with the addictive state as higher addictive states cut off the expected benefits of the final moments of life. As the DM gets older, the loss function $L_{Y,H}(s)$ rises:

$$\frac{\delta L_Y}{\delta Y} = (T'(Y) + 1) b_{s,T(Y)+Y-\beta s} e^{-rT(Y)-\beta s} - (T'(Y) + 1) b_{s,T(Y)+Y-\beta(s+1)} e^{-r[T(Y)-\beta(s+1)]} \quad (7)$$

$$+ \int_{T(Y)+Y-\beta(s+1)}^{T(Y)+Y-\beta s} r e^{-r(t-Y)} b_s dt \geq 0$$

Future losses also rise with age, because the discount factor used to weight end-of-life utility rises as aging draws one closer to the terminal date. Due to discounting, end-of-life utilities are given more weight as one gets closer to the

⁵In writing equation (5) we do not account for transition probabilities affecting the evolution of addictive state s , because the DM evaluates future losses independently from the speed of transition between addictive states.

terminal date, because they are closer to the present. On the other hand, at a younger age, end-of-life utilities are given much less weight because they are far away in the future. Future expected losses may affect behavior in three distinct ways: (i) they increase the threshold M^T thus reducing the probability of entering the hot mode; (ii) They affect the Bellman equation (3) through the decreased probability of use σ ; (iii) They erode the marginal instantaneous benefit from use as they enter the cold mode of operating, in the value function V_s (see equation (3)), as $b_s^a - L_{Y,H}$. The effect of past experiences is instead accounted for introducing the variable $H = \text{Max}\{s_i\}$, $i = 0, 1, \dots, t - 1$ which is the DM's maximum addictive state reached up to the current time period t .

4.2 Increasing Cognitive Appraisal

Natural recovery is conceived as the long-term outcome of a competition between HFM-generated impulses and a “cognitive appraisal” process (I) representing cognitive incentives competing with the HFM’s generated impulses to use. This is modeled as a variation of the deterministic part of the M function with initial condition $I_0 = I(s = 0)$ representing the a priori level of cognitive control. To simplify the analysis, the population of DMs is divided in two: *non addicts* and *potential addicts* (Orphanides and Zervos, 1995). For *non addicts* $I_0 \geq M_0$ and for *potential addicts* $I_0 < M_0$. A *non addict* DM may never become an addict, because its level of competing cognitive incentives is high enough to decrease the probability of entering the hot mode. On the other hand $I_0 < M_0$ represents the case of a DM who has not yet gained experience with the addictive good and is thus less aware of its potential consequences. We focus on this class of DMs. The I function for *potential addicts* is related to the loss function $L_{Y,H}(s)$ as follows:

$$I(s, Y) = \frac{\bar{I}_0 e^{\lambda s}}{1 + \bar{I}_0 (e^{\lambda s} - 1)}, \quad (8)$$

where λ is the same as in equation (2). The initial condition is now defined as

$$\bar{I}_0 = I_0 + \gamma g(L_{Y,H}), \quad (9)$$

where the function g is increasing in $L_{Y,H}$ and is defined as the additional cognitive control arising from the present value of future expected losses $L_{Y,H}$. I satisfies the following properties: $I(s', Y) < I(s'', Y)$ for $s' < s''$; $I(s, Y') < I(s, Y'')$ for $Y' < Y''$ and it is also assumed to be strictly increasing in $L_{Y,H}(s)$ and twice continuously differentiable in the variable s . In equation (9) γ indicates the presence of learning processes related to past history of consumption, age and awareness of future expected losses. We assume $0 \leq \gamma \leq 1$, where $\gamma = 1$ implies perfect learning and $\gamma = 0$ absence of learning. Given I_0 , the presence of learning may drive cognitive

incentives to override the HFM impulses to use for sufficiently high Y and H . Since different individuals have different learning capacities and histories I_0 and γ account for DMs heterogeneity. In Figure 2 we plot the I function against the addictive state s corresponding to different values of the initial condition \bar{I}_0 .

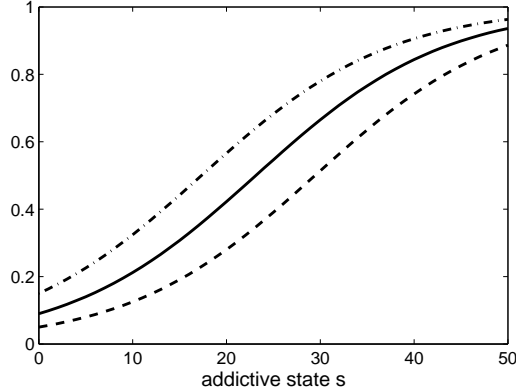


Figure 2: M and I functions corresponding to different assumptions on \bar{I}_0 . Dashed line: $\bar{I}_0 < M_0$ (for $\gamma L_{Y,H} < M_0 - I_0$), solid line: $\bar{I}_0 = M_0$ (for $\gamma L_{Y,H} = M_0 - I_0$), dashdot line: $\bar{I}_0 > M_0$ (for $\gamma L_{Y,H} > M_0 - I_0$).

For a given γ , the I function shifts upwards as time t and the addictive state s increase, so that different values of I may be associated with the same s reached at different time periods. Such process may continue until the I function overrides the HFM and the probability of entering the hot mode declines to zero. An analogous process arises when the a priori level of cognitive control I_0 increases. Spontaneous remission can thus be a result of the extended model. In Appendix A we study the conditions under which $I(s, Y) > M(s, a, \omega_a)$ and show that the equilibrium solution $s^{eq} = 1$ is globally asymptotically stable for the dynamic system described by equation (1).

We have run behavioral simulations solving the stochastic dynamic programming model⁶ (Appendix B). The specific numerical assumptions about the characteristics of the substance and the user are taken from Bernheim and Rangel (2005). We consider $S = 50$; $y_s=800$ \$; each time period t : 1 week; simulation length: 1000 periods (≈ 20 years); cost of addictive substance: 200 \$; cost of rehabilitation: 250 \$; decisions set: $(E, 1), (E, 0), (A, 0), (R, 0)$. Figure 3 shows the probability of each choice as a function of the addictive state s .

In Table 1 and 2 show some statistics of simulations performed by varying income y and initial cognitive control I_0 with respect to a baseline value (y^*, I_0^*)

⁶Numerical simulations and dynamic programming are run on MATLAB 7.0.4.

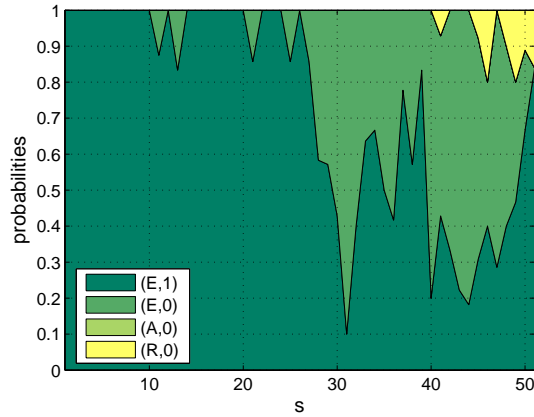


Figure 3: Frequency of decisions for each additive state s .

of the same parameters. Means (1st column), Standard Deviations (2nd column), Absolute Maxima (3rd column) and time periods at which natural recovery occurs (5th column) are shown. STD of Max (4th column) is the standard deviation of the absolute maximum corresponding to each run. All statistics are evaluated on 10 runs of the evolution of the addictive state s .

Table 1: Summary statistics on income y .

	Mean of $s(t)$	STD of $s(t)$	H	STD of H	Nat. Rec. (t)
$y = 700$	7.88	7.69	30	1.94	808
$y = 800^*$	7.42	7.39	28	1.88	737
$y = 900$	6.97	7.29	27.6	1.64	702

Table 2: Summary statistics on initial cognitive control I_0 .

	Mean of $s(t)$	STD of $s(t)$	H	STD of H	Nat. Rec. (t)
$I_0 = 0.05$	8.3	8.97	33.1	2.18	733
$I_0 = 0.07^*$	7.42	7.39	28	1.88	737
$I_0 = 0.09$	6.8	6.39	24.8	1.61	741

Figure 4 shows decisions in cold mode and actual decisions (Hedonic Forecasting Mechanism active) (see also Figure 5).

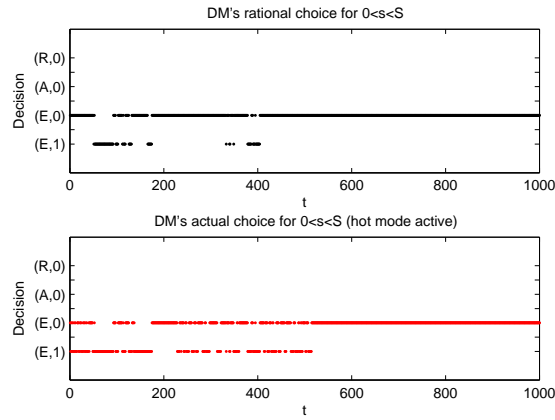


Figure 4: Choices over time: cold mode (top) and hot mode (bottom) decision making.

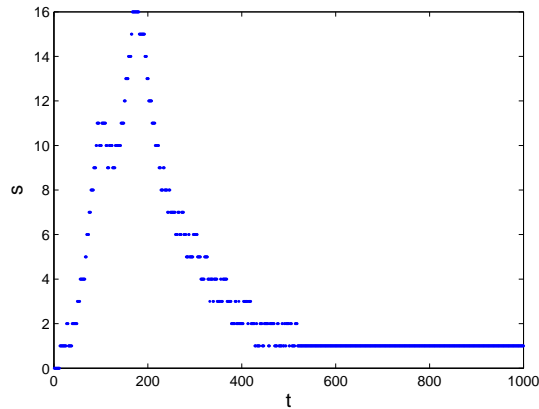


Figure 5: Evolution of the addictive state s as a function of time.

5 Conclusions

If consumers are sometimes rational and sometimes driven by cue-triggered mistakes, public policy approaches such as taxation of legal addictive substances or strict regulation may only raise the cost of consumption.

However, if spontaneous remittance occurs through increased awareness of future expected costs and learning from past experiences, these traditional public policy approaches can still play a role. Policy strategies could be differentiated according to the age profile of addicts. In young consumers cognitive therapies, education and information campaign can have a positive impact not only to discourage initial experimentation, but also on I_0 and γ and can help them activate

cognitive control mechanisms. Trosclair *et al.* (2002) stress that more educated individuals are far more likely to quit smoking successfully, for instance, as education helps activating the competing cognitive incentives necessary to override the HFM. The oldest consumers, on the other hand, may be more responsive to monetary incentives or regulation because their cognitive control is more developed and is more likely to prevail over impulses to use.

The model presented in this paper provides a reasonable explanation of how even long term addicts may find their way out of substance abuse without the utilization of professional help. Drawing from clinical and experimental research we introduce a cognitive appraisal function depending on past addiction histories as well as on future expected consequences of addictive consumption. This affects the DM in two ways: it erodes the payoff from use as the decision maker grows older and it increases the cognitive control competing with the hedonic impulses to use, thus reducing the probability of entering the hot mode. Identifying factors leading to natural recovery and its long-run dynamics may help designing policy measures aimed at reducing consumption of addictive goods. Other explanations could also be reasonable and having data on the demographic characteristics of those who recover spontaneously would help assessing this and alternative explanations.

Appendix A: Stability of the Equilibrium Solution

PROPOSITION 1. (i) Higher values of I_0 decrease the probability p_s^a and thus the probabilities $\sigma_s^{E,0}$ and $\sigma_s^{A,0}$; (ii) higher values of γ decrease the probability p_s^a and thus the probabilities $\sigma_s^{E,0}$ and $\sigma_s^{A,0}$.

PROOF. (i) Let I_0' and I_0'' be two distinct initial conditions of the I function, such that $I_0' < I_0''$. From equation (8) it follows that $I(s, Y, I_0') < I(s, Y, I_0'')$ $\forall s = 0, 1, \dots, S$ and $T(s, a, I_0'') \subset T(s, a, I_0')$. It follows that $\mu(T(s, a, I_0')) > \mu(T(s, a, I_0''))$. (ii) Analogously, $I(s, Y, \gamma') < I(s, Y, \gamma'')$ for $\gamma' < \gamma''$ and $\mu(T(s, a, \gamma')) > \mu(T(s, a, \gamma''))$.

We next show that the equilibrium solution $s^{eq} = 1$ is globally asymptotically stable for the dynamic system described by (1). We start by noting that equation (1) is a hybrid dynamic systems as it evolves according to different dynamics depending on the specific point in the state-input space under consideration. In general, for example, Piecewise Affine Systems allow to consider fundamental hybrid features such as linear-threshold events and mode switching. In our case the regime shifts depend on the DM's choices at each time period and the resulting dynamic systems are the following:

$$s_{t+1} = \begin{cases} s_t + 1, & \text{if } x_t = 1, \\ s_t - 1, & \text{if } x_t = 0, \\ s_t, & \text{if } (x_t = 1 \wedge s_t = S) \vee (x_t = 0 \wedge s_t = 1). \end{cases}$$

In the first two regimes, no equilibrium solutions exist and the dynamics is always increasing or decreasing; in the third dynamics there are two equilibria: $s^{eq1} = 1$ and $s^{eq2} = S$. Asymptotic stability of one of them corresponds to either addictive behavior leading to chronic addiction ($s = S$) or to natural recovery ($s = 1$). Any oscillating dynamics is due to shifts or to transient behavior. In order to study the stability properties of the dynamic system we focus only on the third regime and on the two single-point sets $M = \{s^{eq1}\}$ and $N = \{s^{eq2}\}$.

PROPOSITION 2. *The equilibrium solution $s = s^{eq1}$ is globally asymptotically stable.*

PROOF. Let $L(s_t) = V_{max} - V_t(s_t)$ be a function defined in the open set $G = \{0, 1, 2, \dots, S - 1\}$ of the values reached by the state variable s . L is a Liapunov function on G ; in fact it is continuous on G and $\dot{L}(s_t) = L(s_{t+1}) - L(s_t) \leq 0$ for all $s_t \in G$. Figure 6 plots the function L on the set G of the state variable s . Different colors correspond to simple moving averages (SMA) of L with increasing orders: L (green); $SMA(L, 3)$ (blue); $SMA(L, 5)$ (red); $SMA(L, 7)$ (black).

Furthermore, M is the largest invariant set in G and G is a bounded open positively invariant set. Then, the theorem on asymptotic stability of the set M

(La Salle, 1997) shows that the equilibrium $s = s^{eq1}$ is asymptotically stable on G . This completes the proof.

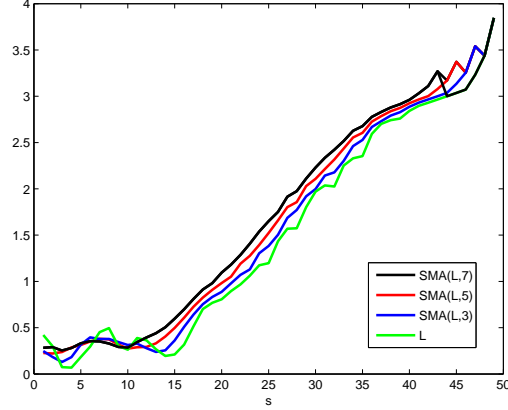


Figure 6: The function L on the set G of the state variable s .

Since the loss function decreases the instantaneous marginal benefit from use we expect this self evaluation process to lead⁷ the DM to choose (E,0) when in cold mode and for a number of time periods sufficient to generate natural recovery. Taking into account clinical evidence reported in Section 3, spontaneous remittance may be a result of the model depicted in this Section.

Now let ϕ be the parameters' vector, $\phi = (\delta, r_s, q, y, I_0, M_0, \gamma)$ such that natural recovery may occur.

PROPOSITION 3. *Assume fixed all the parameters in ϕ except for I_0 :*

(i) *on average an increase in I_0 lengthens the time interval between the initial use and the maximum addictive state H and shortens the interval between H and natural recovery.*

(ii) *On average an increase in I_0 lowers the maximum addictive state H .*

PROOF. (i) Given $\bar{I}_0 = I_0 + \gamma L_{Y,H}$, an increase in \bar{I}_0 is determined by a change in the a priori level of cognitive control I_0 . For a given stochastic process ω and lifestyle a , this causes p_s^a to decrease (see Proposition 1) at each t thus reducing consumption in hot mode and reducing the velocity with which s increases.

(ii) Let I'_0 and I''_0 be two distinct initial conditions of the I function, such that $I'_0 < I''_0$. The maximum levels of s $H'(I'_0)$ and $H''(I''_0)$ are reached at two different time instants t' and t'' . From (i) it follows that $t' \leq t''$. Since by definition $L(H, Y)$

⁷Stated differently, there exist a subset of the relevant parameters satisfying the conditions leading to natural recovery.

is increasing in time, $H''(I_0') \leq H''(I_0)$.

PROPOSITION 4. *Assume fixed all the parameters in ϕ except for γ . An increase in γ shortens the interval between the initial use and the maximum addictive state H and anticipates natural recovery.*

PROOF. A decrease in γ shifts the I function downwards. From Proposition 1 this implies an increase in p_s^a which causes a delay in the effects of the loss function.

Propositions 3 and 4 imply that the process leading to advanced addiction stages can be slowed down by increasing I_0 or γ . This is important, because time, among other factors, activates the self-evaluation process. Thus slowing down the addiction process as represented in figure 1 may increase the likelihood of the onset of the self-evaluation process.

Appendix B: The Stochastic Dynamic Programming Problem

Numerical simulations are obtained by assigning values to the model parameters and maximizing the value function (3).

The parameters of the M and I functions are: $\lambda = 0.1$, $M_0 = 0.09$, $I_0 = 0.07$ and $\gamma = 1$. $c(a, \omega)$ is specified by $c(a, \omega_a) = k_1 + k_2\omega_a$, where ω_a is a normally distributed random process with variance $\sigma^2 = 1$ and mean depending on the life style a . The parameters k_1 and k_2 depend on the lifestyle a .

Taking a quadratic approximation in all the arguments except e_s the instantaneous payoff function w_s is⁸:

$$w_s(e_s, x, a) = b_s^a + w(s) + u(e_s) = b_s^a + u_s^a, \quad (10)$$

where

$$\begin{aligned} b_s^a &= \alpha_x^a x + \frac{\alpha_{xx}^a}{2} x^2 + \frac{\alpha_{xs}^a}{2} xs, \\ w(s) &= \alpha_s^a s + \frac{\alpha_{ss}^a}{2} s^2 + \frac{\alpha_{xs}^a}{2} xs, \\ u(e_s) &= \alpha_e \log(e_s) + \alpha_{ee} \log(e_s) + \alpha_{xe} xe + \alpha_{se} se, \\ u_s^a &= w(s) + u(e_s). \end{aligned}$$

$w(x)$ and $u(e_s)$ are increasing and concave in x and e , $w(s)$ is decreasing in s and the interaction terms α_{xe} and α_{se} are zero by the separability assumption. Monotonicity and concavity of $w(x)$ and $u(e_s)$ follow from standard arguments,

⁸To simplify the notation we omit, henceforth, the time index from variables and equations.

whereas the properties of $w(s)$ incorporate the effect of past usage on current well being, i.e. tolerance, deterioration of health, depression, illness, etc. (see figure 7 in which the payoff functions are plotted against the addictive state s).

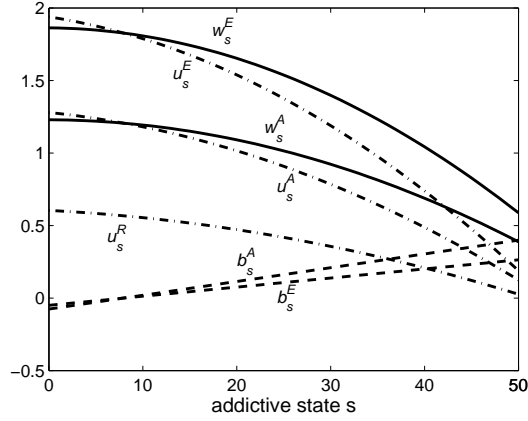


Figure 7: Payoff functions.

The payoff function $w_{s,t}$ is specified by equation (10), where $\alpha_x = 10$, $\alpha_{xx} = -0.5$, $\alpha_s = -1.0$, $\alpha_{xs} = 0.9$, $\alpha_{ss} = -0.1$, $\alpha_e = 30$, $\alpha_{ee} = -1$, $e_s = y_s$.

The solution of the dynamic stochastic programming problem

$$V_h = \max_{(a,x) \in C} u_h^a + \sigma_h^{a,x} b_h^a + \delta \left[(1 - \sigma_h^{a,x}) V_{h-1} + \sigma_h^{a,x} V_{h+1} \right], \quad (11)$$

s.t.

$$0 \leq h \leq S,$$

$$h - 1 = \max \{1, s - 1\},$$

$$h + 1 = \min \{S, s + 1\}$$

is obtained by solving the problem recursively as follows:

Step 1: initialization. By equation (11), for $s = S$ the function V is

$$V_S = \max_{(a,x) \in C} u_S^a + \sigma_S^{a,x} b_S^a + \delta \left[(1 - \sigma_S^{a,x}) V_{S-1} + \sigma_S^{a,x} V_S \right]. \quad (12)$$

Equation 12 implicitly defines V_S as a function of V_{S-1} by

$$V_S = h_S^*(V_{S-1}). \quad (13)$$

We search V_{S-1} within an interval $\left[\underline{V}_{S-1}, \overline{V}_{S-1} \right]$.

Step 2: backward induction For each $k = S - 1, \dots, 2$, by equation (11) we find

$$V_k = g_k^*(V_{k-1}, V_{k+1}) = g_k^*(V_{k-1}, h_{k+1}^*(V_k)), \quad (14)$$

where the function h_{k+1}^* is defined implicitly by the previous steps. Hence, equation (14) implicitly defines a sequence of functions h_k^* such that

$$V_k = h_k^*(V_{k-1}). \quad (15)$$

Step 3: evaluation of terminal condition We find

$$V_1 : g_1^*(V_1, V_2) = g_1^*(V_1, h_2^*(V_1)) \quad (16)$$

by solving equation

$$V_1 = \max_{(a,x) \in C} u_1^a + \sigma_1^{a,x} b_1^a + \delta \left[(1 - \sigma_1^{a,x}) V_1 + \sigma_1^{a,x} h_2^*(V_1) \right].$$

Equation (16) is nonlinear in the variable V_1 and can be solved numerically. Let \hat{V}_1 be the optimal solution. We also evaluate V_0 by solving equation

$$V_0 = \max_{(a,x) \in C} u_0^a + \sigma_0^{a,x} b_0^a + \delta \left[(1 - \sigma_0^{a,x}) V_0 + \sigma_0^{a,x} \hat{V}_1 \right]$$

Step 4: computation of optimal values the optimal sequence $\hat{V}_0, \hat{V}_1, \hat{V}_2, \dots, \hat{V}_S$ is backward recovered by applying the functions h_k^* defined by equation (15).

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