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A Model of Spontaneous Remission From Addiction

Chiara Mocenni, Department of Information Engineering and Mathematics, University of Siena, Siena, Italy
Giuseppe Montefrancesco, Prevention of Pathological Addictions, ASL Sudest, Siena, Italy
Silvia Tiezzi, Department of Economics and Statistics, University of Siena, Siena, Italy

ABSTRACT

This article develops a formal model of spontaneous recovery from pathological addiction. It regards addiction as a progressive susceptibility to stochastic environmental cues and introduce a cognitive appraisal process in individual decision making depending on past addiction experiences and on their future expected consequences. This process affects consumption choices in two ways. The reward from use decreases with age. At the same time, cognitive incentives emerge that reduce the probability of making mistakes. In addition to modeling the role of cue-triggered mistakes in individual decision making, the analysis highlights the role of other factors such as subjective self-evaluation and cognitive control. The implications for social policy and for the treatment of drug and alcohol dependence are discussed.

KEYWORDS

Addiction, Cognitive Policy, Individual Decision Making, Natural Recovery

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1. INTRODUCTION

Addiction is defined as the consequence of repeated use of psychoactive drugs. It is characterized by a loss of control over drug seeking with harmful effects on the individual and a high probability of relapse even months or years after cessation of drug taking (Volkow & Fowler 2000; Fertig et al. 2004; Koob et al. 2004). The main problem is to understand how the individual, substance and environment-related factors involved can trigger the start, sustain recurrence or generate relapse.

Economists have developed theories to model addiction, their interest stemming from the 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 & 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 & 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[1], thus explaining the relationship between behavior and the characteristics of the user, of the substance and of the environment. Neuroscience and clinical practice have 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. Therefore consumption choices are sometimes driven by a rational decision making process, sometimes by strong impulses leading to mistakes, i.e. divergences between preferences and choices.

These theories explain several patterns of addictive behavior, but one aspect left unexplained is spontaneous remission 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-V), recent studies have called into question whether this is an accurate representation (Slutzke 2006; Breidenbach & Tse 2016). 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. However 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 highlights the role of cognitive processes to explain natural recovery even in individuals with an important addiction history.

Building on the work of Mocenni et al. (2011), we extend Bernheim and Rangel (2004) addiction theory by introducing a "cognitive appraisal" function depending on

future expected losses as well as on 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 Hedonic Forecasting Mechanism 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. We also explore the role of other factors, such as learning and individual heterogeneity. Our model is well suited to explain the following dynamics of quitting behavior: (i) natural recovery occurring as cold turkey quitting without an exogenous shock; (ii) gradual quitting after a period of decreasing consumption; and (iii) quitting occurring after a series of failed attempts. Performance analysis of this extended model is carried out.

Our result poses 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.

The remainder of the paper is structured as follows. Sections 2 and 3 provide a narrative of addiction and of natural recovery. Section 4 contains the formal analysis. Section 5 concludes. Appendix A reports the results of the stability analysis of equilibria. Appendix B develops the optimization method and algorithm.

2. THE NEUROSCIENCE OF ADDICTIVE BEHAVIOR

Drugs stimulate the nigrostriatal and corticolimbic dopaminergic systems (Wise 2004) thus increasing dopamine concentration at target-cells' receptor levels. 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 or searching for food and water. When these systems are engaged by addictive substances (Fertig et al. 2004; Nestler 2003) the dopamine release that occurs in the nucleus accumbens causes specific emotional states (e.g. euphoria) that are powerful drivers of behavior. Addictive substances produce higher dopamine concentrations than natural rewards. Thus, there is an incentive to repeat experiences with those substances (Kelley & Berridge 2002; Bechara 2005; Kalivas & Volkow 2002). Moreover, while in the case of natural rewards the development of habits over time reduces the quality and quantity of pleasure, this does not happen with addictive substances as they activate each time the same hedonic response (Berridge & Robinson 2003).

Chronic substance abuse induces profound alterations of the cerebral mechanisms just mentioned forcing the user to make compulsory choices. By powerfully activating dopamine transmission, drugs reinforce the associated learning process ending up by constraining the individual's behavioral choices (Berke & Hyman 2009). In other words, drugs seem to affect the basic Hedonic Forecasting Mechanism[2] (HFM henceforth), a simple and fast system for learning correlations between current conditions, decisions and short term rewards. 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. 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 & 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 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 and conveniences considered up till then as indispensable. When this happens without professional help natural recovery occurs.

3. NATURAL RECOVERY

Natural recovery is more common than suggested by conventional wisdom and characterizes the whole spectrum of drugs such as alcohol (Cunningham *et al*. 2005; Bischof *et al*. 2003; Weisner *et al*. 2003; Matzger *et al.* 2005; Grella and Stein 2013; Breidenbach & Tse 2016), marijuana (Copersino *et al.* 2006), heroin (Waldorf and Biernacki 1979), binge eating, smoking, sex and gambling (Nathan 2003). Longitudinal studies have shown evidence of natural recovery even in pathological gambling (Slutske 2006) questioning the definition of addiction as given by the DSM-V.

A well-established strand of literature has focused on natural recovery and several reviews of studies exist (Carballo et al 2007; Klingemann et al 2009; Smart 2007; Sobell et al 2000; Watson & Sher 1998; Klingemann et al. 2009), but findings in these reviews have yet to be systematized into a well formulated conceptualization explaining why decisions to change occur (Klingemann et al. 2009).

Clinical and experimental research has studied natural recovery from substance abuse since the mid-1970s (Vaillant 1982) focusing on triggering mechanisms, maintenance factors and on trying to identify common reasons for change in substance use (Prochaska et al. 1992). According to Matzger et al. (2005) factors leading a person to move from problematic to non-problematic alcohol use, for example, can be heterogeneous. 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 used to assess the determinants of sustained remission from problem drinking. The two most frequently endorsed reasons for drinking less were the same in the two groups: a cost-benefit analysis of drinking, and major life changes. Drinking causing health problems was also an important reason for quitting. Medical personnel and family interventions were found to be unrelated to improvements in treated drinkers. Carballo et al. (2007) recognize cognitive appraisal

and self-evaluation as a central aspect of self-change in other addictive behaviors. In Cunningham et al. (2005) cognitive appraisal and life changes are the main reasons for quitting. Cognitive appraisal is described as a process of self-appraisal of the costs and benefits of quitting. In the life changes motivation the patients' lifestyles are linked to successful attempts to quit. 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. Finally, Breidenbach & Tse (2016) also reported natural recovery from alcohol and drug addiction among English speaking Hong Kong residents as an evolutionary process occurring over a long period of time with intervals of cognitive appraisal and quit attempts.

Despite this empirical evidence, there are 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. The motivation to quit is based instead on changes in the addict's perspective as she 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 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 the 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 & Rangel 2004). These new theories, however, do not explicitly model pathways to natural recovery[3].

Klingemann et al. (2009) highlighted the following as potential drivers of natural recovery:

- Some individuals confronted with addiction can make informed decisions and develop resolution strategies;
- An individual's capacity to terminate chronic substance misuse is very much a function of the resources she has developed and maintained over the course of her life. These resources consist of personal attributes, physical and socio-environmental structures, cultural dispositions and related life circumstances;
- Consequent-driven reasons (e.g. particular life events) for recovery compared to drifting-out reasons (e.g. role changes, growing older) occur significantly more frequently among those with more severe addiction problems;
- Much self-change research has highlighted the importance of cognitive decision processes (e.g. balancing) as a central characteristic of individual change;

- A core concept of self-change research is to regard change as a process that, in most cases, occurs over time.

In order to include these factors in an addiction theory, we extend Bernheim and Rangel (2004) model by explicitly considering learning, self-appraisal and cognitive control. As shown in the next Section, natural recovery is one possible outcome of the extended model.

Figure 1 outlines a schematic representation of our model. Consumption choices result from the combination of exogenous cues, innate propensity, age, lifestyle and sensitization to addictive goods. The reinforcing mechanism leading the DM to become an addict can be affected by cognitive factors and by the evaluation of the past and future losses due to compulsive consumption of addictive goods.

4. A MODEL OF NATURAL RECOVERY

Bernheim and Rangel's (2004) theory is based on the assumption that substances' consumption is often a cue-triggered mistake. However addicts can develop a growing awareness leading to attempts to control the process.

Drawing from this theory, we consider a decision maker (DM henceforth) who can operate in either cold or hot decision-making mode (Loewenstein 1996 and 1999). In hot mode, she always consumes the substance irrespective of her true preferences. In cold mode all alternatives and consequences are considered, including the likelihood

Figure 1. Scheme of the mathematical model. External and innate factors influencing the DM. Sequence of decisions taken by the DM (central path). Hot and cold modes of decision leading to compulsive and non compulsive behaviors (left and right cycles). Natural recovery (triangle) may be activated by increasing costs (loss function) and cognitive factors producing switches in the DM's behavior (dotted line).

Characteristics of the Hot/Cool Framework

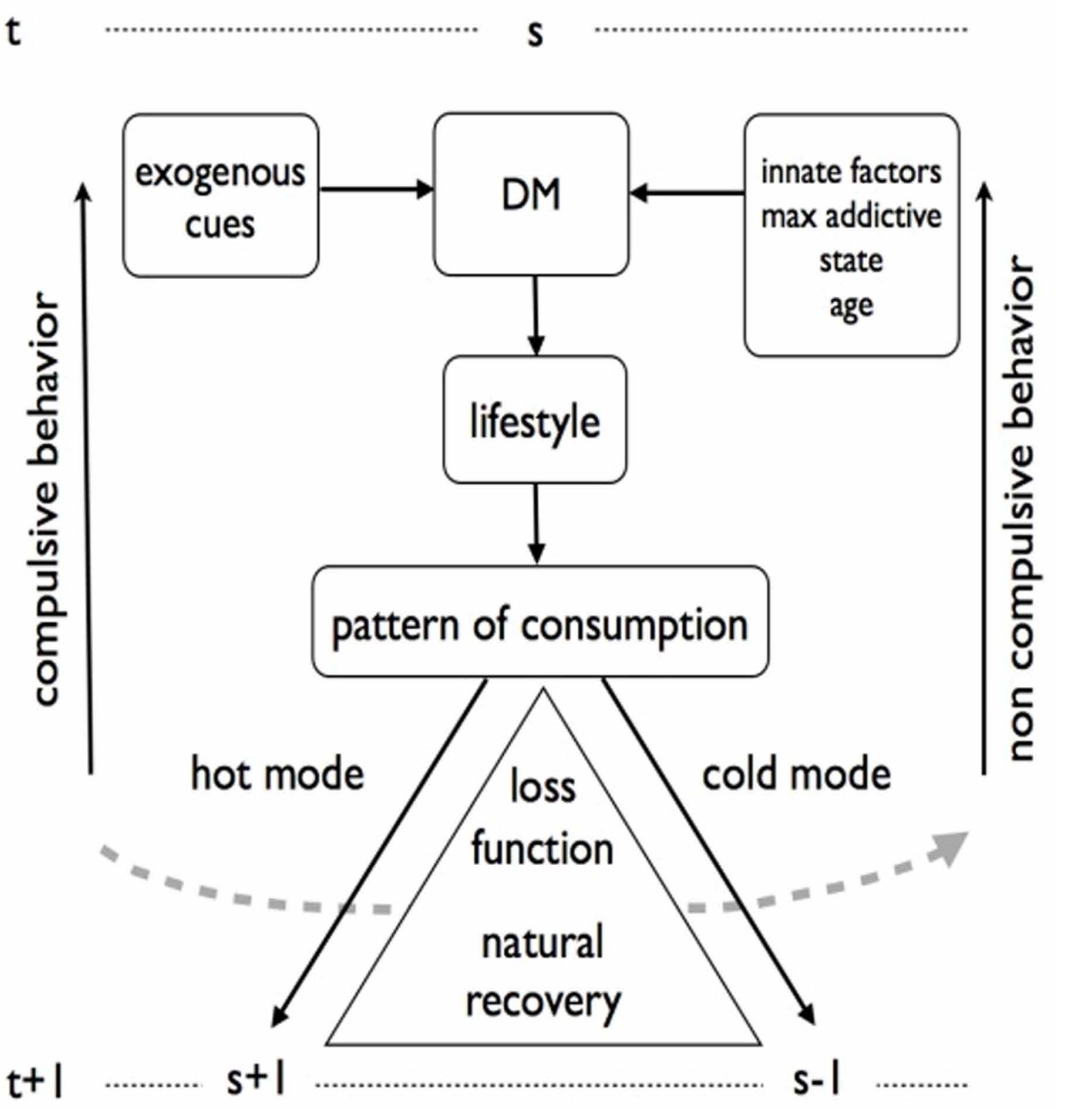
Hot System	Cool System
Emotional	Cognitive
"Go"	"Know"
Simple	Complex
Reflexive	Reflective
Fast	Slow
Develops Early	Develops late
Accentuated by Stress	Attenuated by stress
Stimulus control	Self-control

Source: Metcalfe and Mischel, 1999, p. 4.

of entering the hot mode in the future. Metcalfe & Mischel (1999) adopted such hot/cold framework to explain the dynamics of willpower. They also reviewed previous studies and fields of investigation in psychology in which a hot/cool framework had been adopted (Figure 2).

Time is discrete, indexed by the nonnegative integers, $t \in \{0,1,2,...\}$. In each time period t the DM first selects a lifestyle a from the set $\{E, A, R\}$. (e.g. going to a bar or staying at home watching TV). If lifestyle E, "exposure", is chosen there is a high probability that the DM will encounter a large number of substance-related cues. Activity A, "avoidance", entails fewer substance-related cues and may also reduce

Figure 2. Hot-cool framework



sensitivity to them. 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. The DM allocates resources between a potentially addictive good $x \in \{0,1\}$ the price of which is q, and a standard good $(e_s \geq 0)$. By assumption the DM cannot borrow or save. Each period begins in cold mode and the choice of lifestyle, together with the starting addictive state s gives the probability p_s^a of cues triggering 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 $s = 0,1,\ldots,S$. The system dynamics is described by the evolution of state s_t according to the following equation:

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

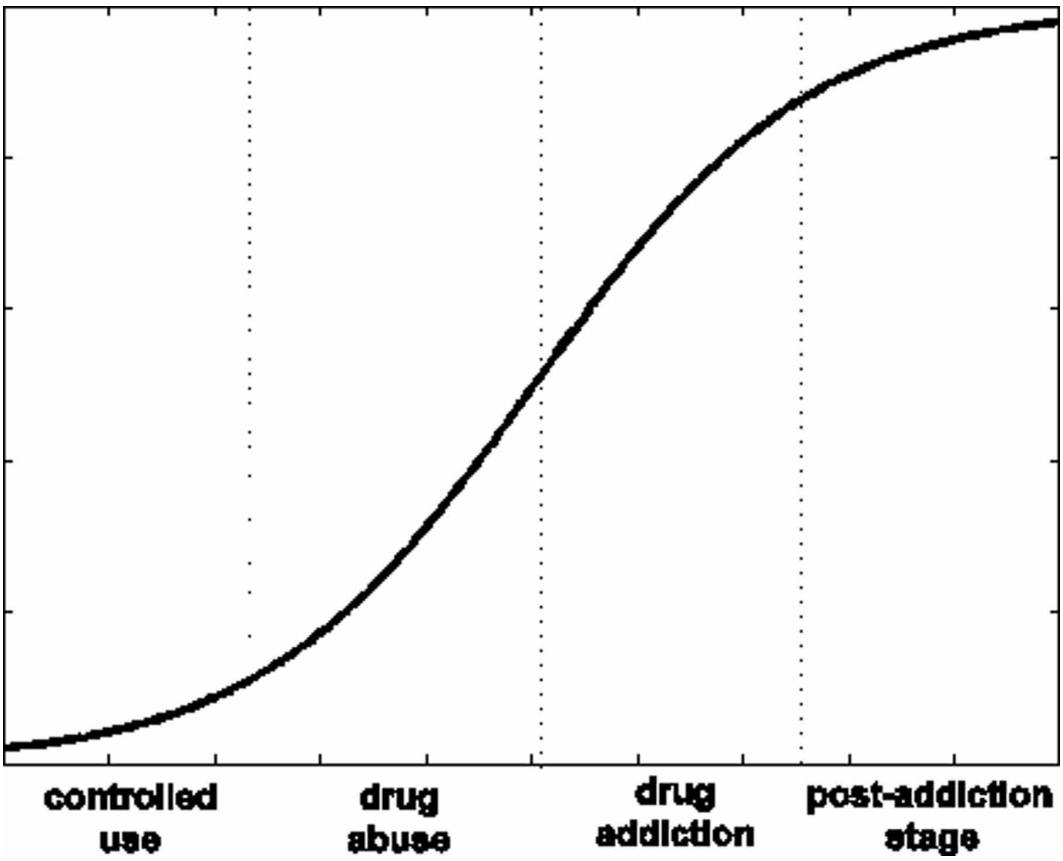
Equation (1) implies that consumption in state s leads to state $min\{S, s_{t+1}\}$ in the next period with probability p_T. No use leads to state $Max\{1, s_{t-1}\}$ with probability p_T from state $s>1$ and to state $s=0$ from state $s=0$. The volume of substance-related environmental cues $c(a,\omega)$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

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

where $a \in \{R, E, A\}$ and $M_0 = M(s=0)$ and λ is the growth rate of the HFM generated impulses.

An example of the power function M is in Figure 3 depicting four different phases of addiction (Di Chiara, 2002) by vertical dotted lines: controlled use, drug abuse,

Figure 3. The deterministic portion of the M function



drug addiction, post-addiction stage. In Figure 3 M is measured on the vertical axis and s is measured on the horizontal axis. In the first stage, the DM starts consuming a drug driven by curiosity, peer pressure, social factors (lifestyles a and environmental cues c.)

Sensitization triggers further experimentation and increases the HFM (M weakly increasing in s). In the first stage subjects react to the drug stimuli in a controlled way. Repeated drug consumption leads to the drug abuse stage in which sensitization is very powerful and craving is generated by drug stimuli (M strongly increasing in s). The drug addiction stage is characterized by tolerance and physical dependence. Finally, post-addiction is characterized by decreasing sensitization and periods of abstinence even though the HFM-generated impulses are still active.

We consider $T\left(s,a\right)=\left\{\omega\in\Omega:M\left(a,s,a,\omega\right)>M^{T}\right\}$. The DM enters the hot mode if and only if $\omega\in T\left(s,a\right)$. Letting $p_{s}^{a}=\mu\left(T\left(s,a\right)\right)$ 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 probability of entering the hot mode at any moment, because the

sensitivity to random environmental cues has increased. So $p_{s+1}^{a} \geq p_{s}^{a}$, $p_{0}^{a} = 0$ and $p_{s}^{E} \geq p_{s}^{A} \geq p_{s}^{R}$ in each time period.

In state s the DM receives an immediate hedonic payoff $w_{s}\left(e_{s}, x, a\right) = u\left(e_{s}\right) + v_{s}\left(x, a\right)$ where utility derived from non addictive goods $u\left(e_{s}\right)$ is assumed to be separable from utility derived from addictive consumption v_{s}. w_{s} is increasing, unbounded, strictly concave and twice differentiable with bounded second derivative in the variable e_{s}. Moreover $v_{s}\left(x, a\right) \equiv u_{s}^{a} + b_{s}^{a}$, 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 $u_{s}^{E} \geq u_{s}^{A} \geq u_{s}^{R}$ and $u_{s}^{E} + u_{s}^{E} > u_{s}^{A} + u_{s}^{A}$. 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 $V_{s}\left(\theta\right)$ and Bellman equation equal to:

$$V_{h}\left(\theta\right) = \max_{(a,x)\in C} u_{h}^{a} + \sigma_{h}^{a,x} b_{h}^{a} + \delta\left[\left(1 - \sigma_{h}^{a,x}\right) V_{h-1}\left(\theta\right) + \sigma_{h}^{a,x} V_{h+1}\left(\theta\right)\right] \tag{3}$$

s.t.

$0 \leq h \leq S$

$h - 1 = Max\left\{1, s-1\right\}$

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

C is the set of decision states $\left\{\left(E,1\right),\left(E,0\right),\left(A,0\right),\left(R,0\right)\right\}$; $\sigma_{s}^{a,x}$ represents the probability of consuming the substance in state x with contingent plan $\left(a,x\right)$ and θ is a vector specifying the model parameters. The stationarity of Equation (3) follows from the assumption that the DM takes her decision at the beginning of each period[4].

We are interested in the choice set $\left(E,0\right)$. 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.

4.1. Expected Losses and Past Addiction Histories

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

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

where r is the discount rate; $e^{-r\left(t-Y\right)}=\delta$ 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 by[5]:

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

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

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

where *L'* is the time derivative of L. (6) is weakly positive because:

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

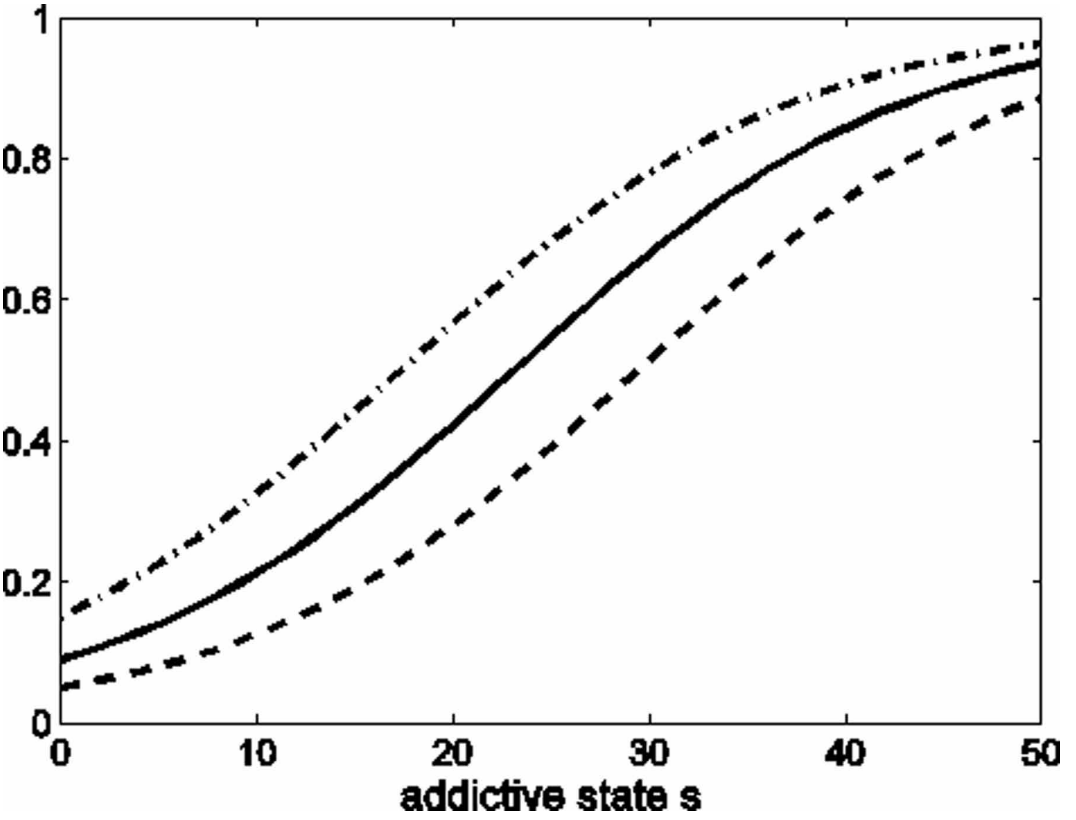
and:

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

As a consequence of (5) and (6), 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 $L_{Y,H}\left(s\right)$ rises:

$$\begin{aligned}&\frac{\partial L_Y}{\partial Y}=\left(T'\left(Y\right)+1\right)b_{s,T\left(Y\right)+Y-\beta s}e^{-rT\left(Y\right)-\beta s}\\&-\left(T'\left(Y\right)+1\right)b_{s,T\left(Y\right)+Y-\beta\left(s+1\right)}e^{-rT\left(Y\right)-\beta\left(s+1\right)}\\&+\int_{T\left(Y\right)+Y+\beta\left(s+1\right)}^{T\left(Y\right)+Y+\beta s}re^{-r\left(t-Y\right)}b_s dt\geq 0\end{aligned} \tag{7}$$

Figure 4. *M* and *I* functions corresponding to different assumptions on I_0. Dashed line: $I_0 < M_0$ (for $\gamma L_{Y,H} < M_0 - I_0$); solid line: $I_0 = M_0$ (for $\gamma L_{Y,H} = M_0 - I_0$); dashdot line: $I_0 > M_0$ (for $\gamma L_{Y,H} > M_0 - I_0$).



In addition, future losses also increase with age, as the discount factor increases as one gests closer to the end of life. On the other hand, end-of-life utilities will be weighted less at younger ages because they are far too distant.

Future expected losses affect consumption behavior in the following ways: (i) by increasing M^T and by decreasing the probability of entering the hot mode; (ii) by affecting the Bellman Equation (3) through the decreased probability of use σ; (iii) by eroding the marginal benefit in V_S as $b_s^a - L_{Y,H}$. The effect of past experiences is accounted for introducing the variable $H = Max\{s_i\}$, $i = 0,1,\ldots,t-1$ indicating the DM's maximum addictive state reached up to the current period t.

4.2. Increasing Cognitive Appraisal

We consider a variation of the M function and an initial condition denoting the *a priori* level of cognitive control. The population of DMs is split between *non addicts* $I_0 \geq M_0$ and *potential addicts* $I_0 < M_0$ (Orphanides and Zervos 1995). *Non addicts* may never become addicted if their competing cognitive incentives are high enough

Table 1. Variables, functions and model parameters

Parameter Variable Function	Description	Meaning	Notes
M_0	Initial value of function M representing the DM's sensitivity to exogenous cues	Parameter accounting for the tendency of individuals to become addicts	Parameter (DM specific)
M^T	Threshold parameter. The DM enters the hot mode when the function M exceeds M^T	It indicates the critical value beyond which the DM enters compulsive behavior	Parameter (a priori)
λ	Argument of the exponential function defining the growth of function M	Positive constant indicating the growth rate of the HFM generated impulses	Parameter (a priori)
γ	Weighting parameter of function g in I_0	It indicates the presence of learning processes related to the past history of consumption	Parameter (a priori)
I_0	Initial condition	A priori level of cognitive control	Parameter (DM specific)
Y	DM's age		Parameter (DM specific)
H	Maximum addictive state reached by the DM		Model variable
s_t	State variable describing the degree of addiction at time t	Intensity of addiction at time t	Model variable
E, A, R	life styles chosen by the DM	It defines the environment in which the DM operates	Model variable
g	Part of function I_0	Additional cognitive control arising from the present value of future expected losses	Model function
I	Cognitive Appraisal Function	It models the fundamental mechanism driving natural recovery	Model function
I_0	Initial condition of the function I including the effects of the loss function L	It determines the possibility for the DM of entering the hot mode	Model function
p_t	Transition probability from state s to state s+1 or s-1		
c	Function describing substance related environmental cues	Environmental factors triggering the DM to enter the hot mode	Model function
M	Power function accounting for the intensity of exogenous cues	The shape of this function accounts for the potential addictive behavior of the DM	Model function
V	DM's expected utility	Present value of expected future utility stream from consumption	Model function
L	Loss function depending on addictive state s, age Y and the maximum addictive state H	Expected future losses at time t	Model function
W	Payoff function	It incorporates the effects of the addictive good's past consumption	Model function

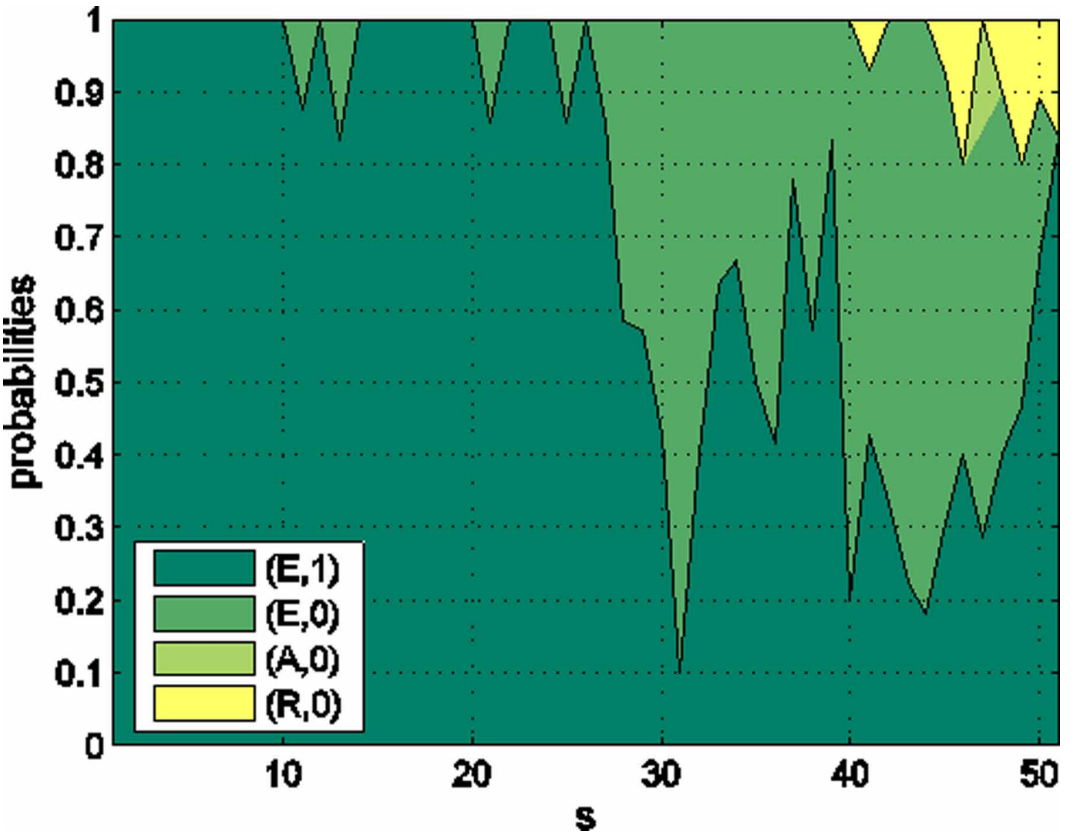
to avoid the hot mode. When $I_0 < M_0$ the DM is not yet acquainted with the substance and its potential consequences. In this case the I function for *potential addicts* is related to the loss function $L_{Y,H}(s)$ in the following way:

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

where λ is the same as in Equation (2). The initial condition is defined as:

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

Figure 5. Frequency of decisions for each addictive state s



where I_0 and g account for DMs heterogeneity, and the increasing in $L_{Y,H}$ function g 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 $Y' < Y''$. Moreover, it is strictly increasing in $L_{Y,H}(s)$ and twice continuously differentiable in s. γ indicates the presence of learning processes related to the past history of consumption, age and awareness of future expected losses. We assume $0 \leq \gamma \leq 1$ with γ =1 implying perfect learning and $\gamma = 0$ implying absence

Table 2. Summary statistics on income y

y	Mean s(t)	STD s(t)	H	STD H	Nat. Rec.
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 3. Summary statistics on initial level of cognitive control I_0

I_0	Mean s(t)	STD s(t)	H	STD H	Nat. Rec.
I_0=0.05	7.88	7.69	30	1.94	808
I_0=0.07*	7.42	7.39	28	1.88	737
I_0=0.09	6.97	7.29	27.6	1.64	702

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. In Figure 4 we plot the I function against the addictive state s corresponding to different values of the initial condition $\bar{I}_0$.

As time t and addictive state s increase, the I function moves up for any γ so that different values of I are associated with the same s. When the I function overrides the HFM, the probability of entering the hot mode is driven to zero. Similar

Figure 6. Choices over time: cold mode (top) and hot mode (bottom) decision making

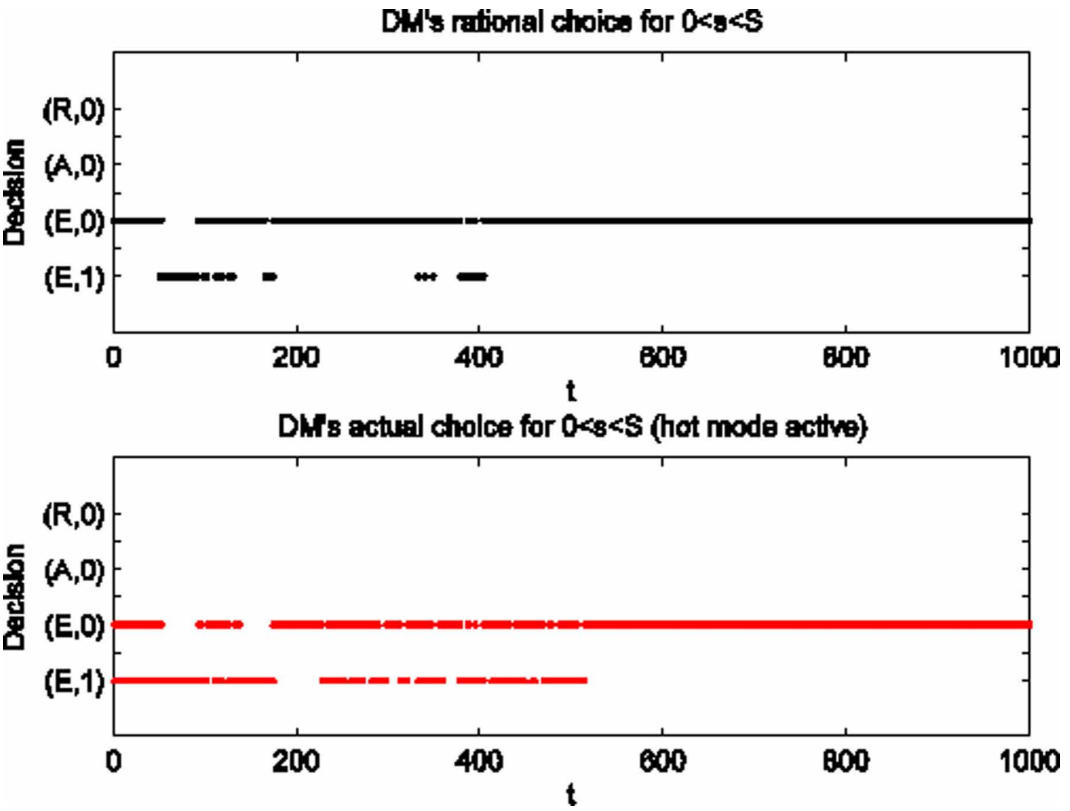
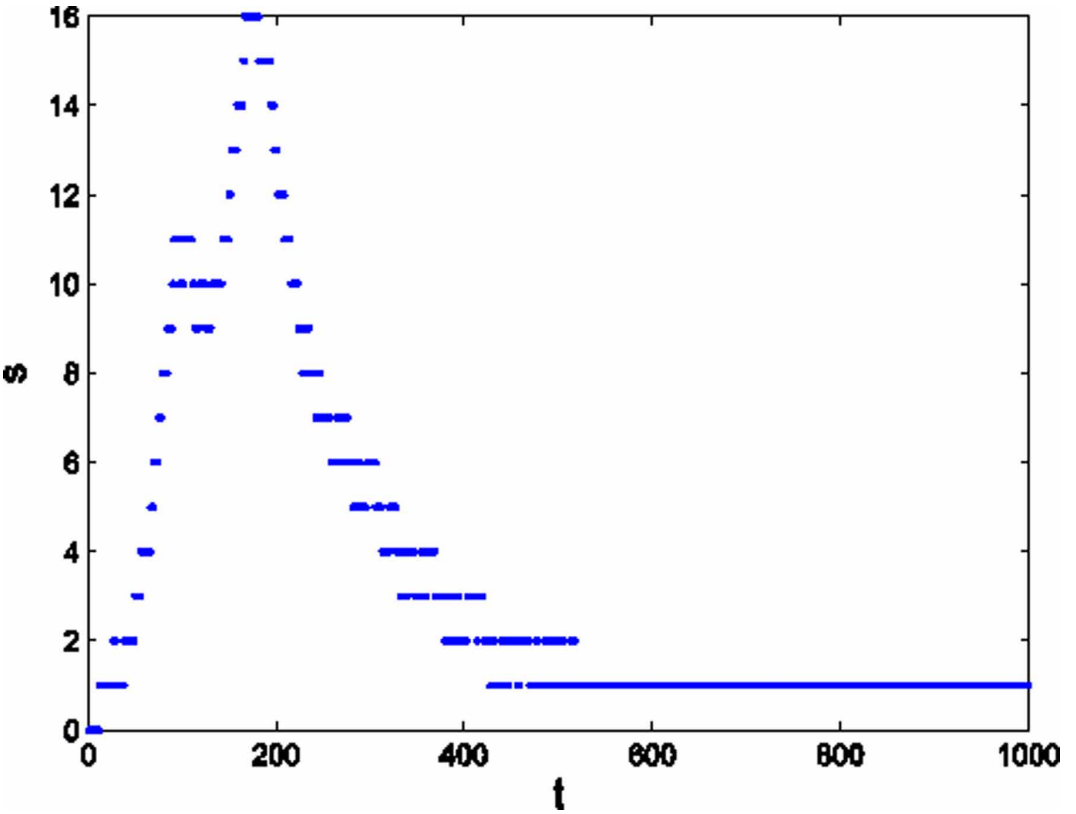


Figure 7. Evolution of the addictive state S as a function of time



dynamics occur when I_0 increases. Natural recovery can thus be the long-term outcome of a competition between HFM-generated impulses and cognitive incentives *I*.

Appendix A details the conditions under which $I\left(s,Y\right) > M\left(s,a,\omega_a\right)$ and shows that the equilibrium solution $s^{eq} = 1$ is globally asymptotically stable for the dynamic system described in (1). In particular, we prove the following propositions in Appendix A.

Proposition 1:

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

(2) Higher values of γ decrease the probability p_s^a and thus the probabilities $\sigma_s^{E,0}$ and $\sigma_s^{A,0}$.

Proposition 2:

The equilibrium solution $s = s^{eq}$ is globally asymptotically stable.

4.3. Simulations and Discussion

This section shows numerical simulations of the extended model and discusses the theoretical results. The numerical solutions are obtained solving the stochastic dynamic programming problem implied by the model and described in Appendix B. Table 1 contains a description of the main variables and the model's parameters.

The numerical assumptions on the characteristics of the substance and the user are taken from Bernheim and Rangel (2005). We consider $S = 50$; y_s =800\$; 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 5 shows the probability of each choice as a function of the addictive state s.

Tables 2 and 3 show some summary statistics of the simulations performed by varying income *y* and initial cognitive control I_0 with respect to their baseline value (y^*, I_0^*). Means (1st column), Standard Deviations (STD, 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. Choices over time are shown in Figure 6. Figure 7 shows the evolution of the addictive state *s* leading to natural recovery.

Additional results concern the relationship between the model solutions and the parameters. In particular, we prove the following propositions in Appendix A.

Proposition 3:

Assume fixed all the parameters in φ except for I_0:

(1) 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.

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

Proposition 4:

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

Both the numerical simulations and the theoretical results show that natural recovery is a possible outcome of an extended model accounting for learning, cognitive control and self-appraisal. Identifying factors leading to natural recovery and its long-run dynamics may help designing policy measures aimed at reducing consumption of addictive goods. If consumers are sometimes rational and sometimes driven by cue-triggered mistakes, measures such as taxation of legal addictive substances or strict regulation may only raise the cost of consumption. However, if spontaneous remission occurs through increased awareness of future expected costs and learning from past experiences, standard public policy approaches can still play a role. The

implication of our model is that more attention should be paid to education and information policy measures relative to health and pharmacological ones. Policy strategies differentiated by the age profile of the patients could be useful. In young consumers cognitive therapies, education and information campaigns can have a positive impact not only to discourage initial experimentation, but also on I_0 and γ, and can help individuals activate cognitive control mechanisms. Trosclair et al. (2002) stress that more educated individuals are far more likely to successfully quit smoking, for example, as education helps them activating the competing cognitive incentives necessary to override the HFM. Oldest consumers, on the other hand, may be more responsive to regulation because their cognitive system is more developed and more likely to prevail over impulses to use.

5. CONCLUSION

We propose a decision making model explaining how even long term addicts may find their way out of substance abuse without the utilization of professional help. Even though natural recovery characterizes a substantial fraction of individuals with a history of pathological addiction, research is still scarce. Spontaneous remission becomes a possibility when additional decision making factors, neglected by the previous literature, are taken into account. 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 decision maker 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.

Future research could focus on empirical tests and calibration of the model, if appropriate longitudinal data are available. The estimated parameters incorporate information on individual traits that may be crucial for natural recovery. Moreover, parameters estimation would allow classification of population groups based on their addictive behavior. This information could then be used to design appropriate addiction control measures.

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ENDNOTES

1 Bernheim and Rangel (2004) analysis is related to previous work by Loewenstein (1996 and 1999) on the "cold-to-hot empathy gap".

2 From Bernheim and Rangel 2004.

3 Bernheim & Rangel (2004) actually mention the possibility of their model to generate natural recovery.

4 Bernheim and Rangel (2004) 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 $\theta_s = \left(p_s, u_s, b_s\right)$ from θ' to θ''. *Cue-triggered recidivism* is associated with high exposure to relatively intense cues, e.g. high realizations of c(a, ω). *Self-described mistakes* in which 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) implying a costly pre-commitment. *Self-control through behavioral and cognitive therapy* through choice (A, 0) implying costly cue avoidance.

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

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

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

APPENDIX A: STABILITY OF THE EQUILIBRIUM SOLUTION

Proposition 1:

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

(2) Higher values of γ decrease the probability p_s^a and thus the probabilities $\sigma_s^{E,0}$ and $\sigma_s^{A,0}$.

Proof:

(1) Let $I_0^{'}$ and $I_0^{''}$ be two distinct initial conditions of the I function, such that $I_0^{'} < I_0^{''}$. From Equation (A8) $I(s,Y,I_0^{'}) < I(s,Y,I_0^{''}) \quad \forall s = 0,1,...,S$ and $T(s,a,I_0^{''}) \subset T(s,a,I_0^{'})$. It follows that $\mu(T(s,a,I_0^{'})) > \mu(T(s,a,I_0^{''}))$.

(2) 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). (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, 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 is:

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

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: $s^{eq_1} = 1$ and $s^{eq_2} = S$. Asymptotic stability of one of them corresponds to either addictive behavior leading to chronic addiction $\left(s = S\right)$ or to natural recovery $\left(s = 1\right)$. 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 $M = \{s^{eq_1}\}$ and $N = \{s^{eq_2}\}$.

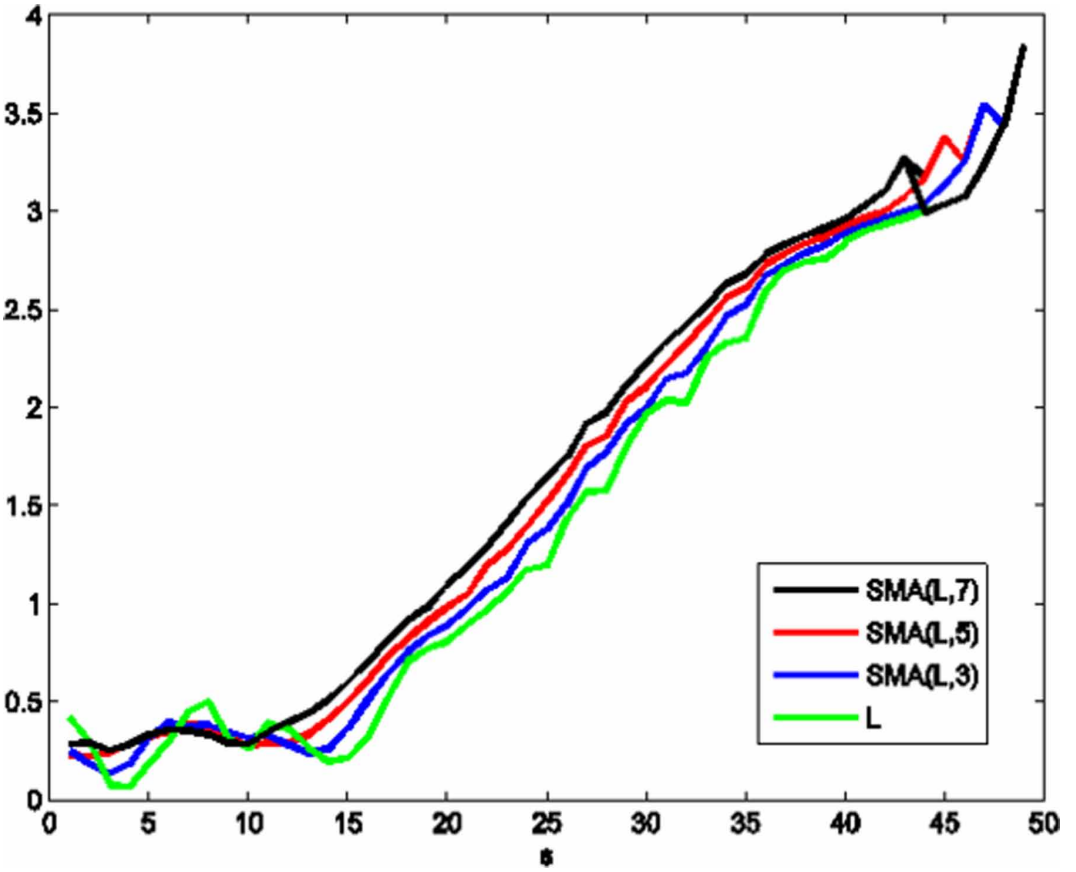
Proposition 2:

The equilibrium solution $s = s^{eq_1}$ is globally asymptotically stable.

Proof:

Let $L(s_t) = V_{max} - V_t(s_t)$ be a function defined in the open set $G = \{0,1,2,...,S-1\}$ of the values reached by the state variable s. L is a continuous on G Liapunov function with $\dot{L}(s_t) = L(s_{t+1}) - L(s_t) \leq 0$ for all $s_t \in G$. Figure 8 plots the function

Figure 8. The function L on the set G of the state variable s



L on the set G of the state variable s. Different colors correspond to simple moving averages (SMA) of L: L (green); $SMA(L,3)$ (blue); $SMA(L,5)$ (red); $SMA(L,7)$ (black).

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 $s = s^{eq_1}$ is asymptotically stable on G. This completes the proof.
Since the loss function decreases the instantaneous marginal benefit from use we expect the DM to choose (E,0) when in cold mode and for a number of time periods sufficient to generate natural recovery[6].
Now let φ be the parameters' vector, $\varphi = (\delta, r_s, q, y, I_0, M_0, \gamma)$ such that natural recovery may occur.

Proposition 3:
Assume fixed all the parameters in φ except for I_0:

(1) 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.
(2) On average an increase in I_0 lowers the maximum addictive state H.

Proof:

(1) 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 at which s increases.
(2) 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 $L(H,Y)$ 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 shorthens 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.

APPENDIX B: THE STOCHASTIC DYNAMIC PROGRAMMING PROBLEM

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

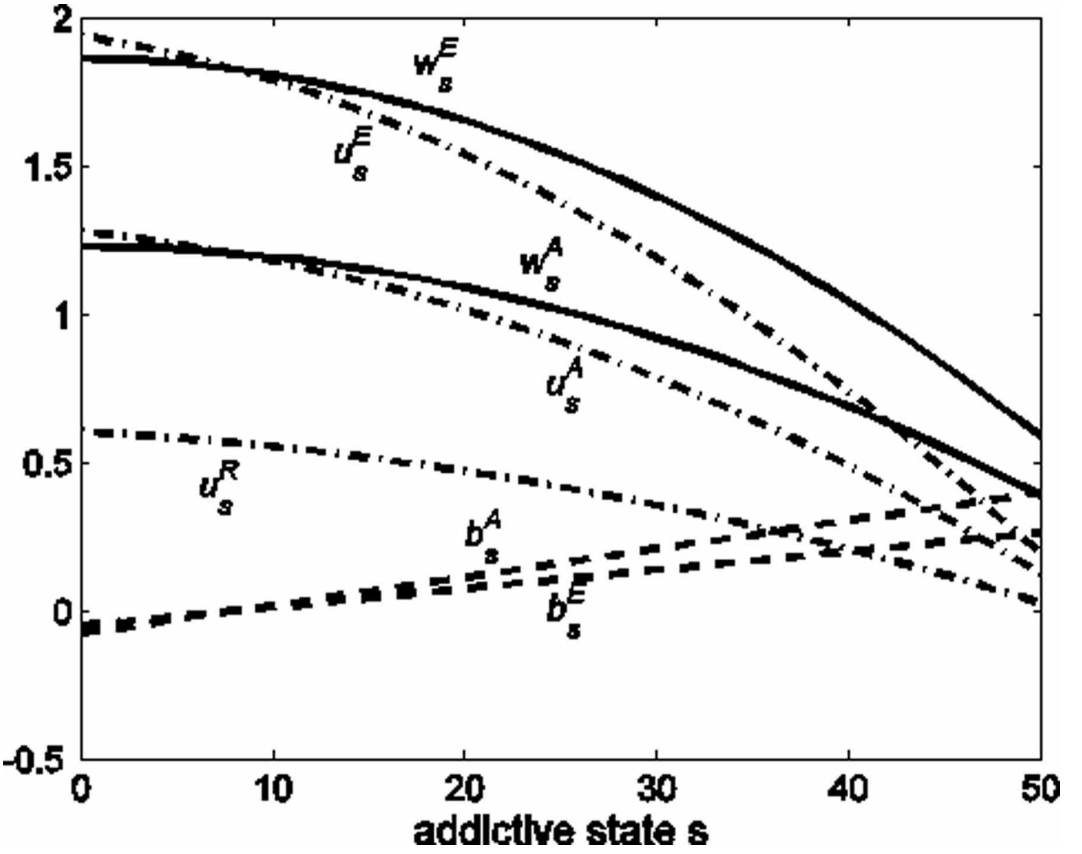
The parameters of the M and I functions are: $\lambda = 0.1$, $M_0 = 0.09$, $I_0 = 0.07$ and $\gamma = 1$. $c(a,\omega)$ is specified by $c(a,\omega_a) = k_1 + k_2\omega_a$, where ω_a is a normally distributed random process with variance $\sigma^2 = 1$ and mean depending on lifestyle a. The parameters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

Taking a quadratic approximation in all the arguments except e_s the instantaneous payoff function w_s is[7]:

$$w_s\left(e_s, x, a\right) = b_s^a + w\left(s\right) + u\left(e_s\right) = b_s^a + u_s^a \tag{B1}$$

with:

Figure 9. Payoff functions



$$b_s^a = \alpha_x^a x + \frac{\alpha_{xx}^a}{2} x^2 + \frac{\alpha_{xs}^a}{2} xs$$

$$w(s) = \alpha_s^a s + \frac{\alpha_{ss}^a}{2} s^2 + \frac{\alpha_{xs}^a}{2} xs$$

$$u(e_s) = \alpha_e \log(e_s) + \alpha_{ee} \log(e_s) + \alpha_{xe} xe + \alpha_{se} se$$

$$u_s^a = w(s) + u(e_s)$$

$w(x)$ and $u(e_s)$ are increasing and concave in x (potentially addictive good) and e (non addictive good); $w(s)$ 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, whereas the properties of $w(s)$ incorporate the effect of past use on current well being, i.e. tolerance, deterioration of health, illness (Figure 9).

The payoff function $w_{s,t}$ is specified by Equation (B1), 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:

$$V_h = \max_{(a,x)\in C} u_h^a + \sigma_h^{a,x} b_h^a + \delta\left[\left(1-\sigma_h^{a,x}\right)V_{h-1} + \sigma_h^{a,x} V_{h+1}\right] \tag{B2}$$

s.t.

$0 \le h \le S$

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

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

is obtained recursively. By Equation (B2), for $s = S$ the function V is:

$$V_S = \max_{(a,x)\in C} u_S^a + \sigma_S^{a,x} b_S^a + \delta\left[\left(1-\sigma_S^{a,x}\right)V_{S-1} + \sigma_S^{a,x} V_S\right] \tag{B3}$$

(B3) implicitly defines V_S as a function of V_{S-1} by:

$$V_S = h_S^*(V_{S-1}) \tag{B4}$$

We search V_{S-1} within an interval $\left[\underline{V_{S-1}}, \overline{V_{S-1}}\right]$. For each $k = S-1,\ldots,2$, by Equation (B2) we find:

$$V_k = g_k^*(V_{k-1}, V_{k+1}) = g_k^*(V_{k-1}, h_{k+1}^*(V_k)) \tag{B5}$$

where the function h_{k+1}^* is defined implicitly by the previous steps. Hence, Equation (B5) implicitly defines a sequence of functions h_k^* such that:

$$V_k = h_k^*(V_{k-1}) \tag{B6}$$

We find:

$$V_1 : g_1^*(V_1, V_2) = g_1^*(V_1, h_2^*(V_1)) \tag{B7}$$

by solving:

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

Equation (B7) is nonlinear in V_1 and can be solved numerically. Let $\widehat{V}_1$ be the optimal solution. V_0 is evaluated solving:

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

the optimal sequence $\widehat{V}_0$, $\widehat{V}_1$, $\widehat{V}_2$, ..., $\widehat{V}_S$ is backward recovered by applying the functions h_k^* defined in (B6).

Silvia Tiezzi is Associate Professor of Economic Policy in the Department of Economics and Statistics at the University of Siena. Her Research Fields are: Consumer Economics, Environmental Economics, Health Economics, Applied Micro Econometrics, Design and Analysis of Laboratory Experiments. She holds a PhD from the University of Siena and an M.Phil. from the University of Cambridge (UK). She has been a Marie Curie Fellow at Carnegie Mellon University, Pittsburgh (PA).

Chiara Mocenni is Associate Professor of Systems and Control at the Department of Information Engineering and Mathematics (DIISM), University of Siena, where she teaches Complex Dynamic Systems and Game Theory and serves as Head of the Teaching Committee of the Engineering Management MSc. CM received the Laurea degree in mathematics in 1992 and the Ph.D. degree in physical chemistry in 1998 with a thesis on modeling biological complex systems. She joined the Systems and Control Group of DIISM in 1998 as Research Associate. From 2000 to 2012, she served on the Board of Directors of the Center for the Study of Complex Systems of the University of Siena. Research interests are: mathematical modeling and identification of nonlinear dynamic systems, nonlinear time series analysis, modeling of biological systems, evolutionary games on networks.