

Hava T. SIEGELMANN

Addiction as a dynamical rationality disorder

© Higher Education Press and Springer-Verlag Berlin Heidelberg 2011

Abstract Addiction is frequently modeled as a behavioral disorder resulting from the internal battle between two subsystems: one model describes slow planning versus fast habitual action; another, hot versus cold modes. In another model, one subsystem pushes the individual toward substance abuse, while the other tries to pull him away. These models all describe one side winning over the other at each point of confrontation, represented as a simple binary switch: on or off, win or lose. We propose however, an alternative model, in which opposing systems work in parallel, tipping toward one subsystem or the other, in greater or lesser degree, based on a continuous rationality factor. Our approach results in a dynamical system that qualitatively emulates seeking behavior, cessation, and relapse—enabling the accurate description of a process that can lead to recovery. As an adjunct to the model, we are in the process of creating an associated, interactive website that will enable addicts to journal their thoughts, emotions and actions on a daily basis. The site is not only a potentially rich source of data for our model, but will in its primary function aid addicts to individually identify parameters affecting their decisions and behavior.

Keywords addiction, emotional-cognitive rationality, dynamical systems, dynamical disease, dynamical rehabilitation, relapse, higher power

1 Introduction

Drug and alcohol abuse affects over 24 million individuals in the U.S [1] and its reach is increasing globally. The complexity of addiction is immense, encompassing diverse, but associated spheres: genetic predisposition [2], altering brain structures that change size over the duration of abuse [3], social and economic status [4] and

depression [5], to name only a few.

The complexity of the disease is daunting; the economic toll—vast: from governmental expenditures, rehabilitation programs, personal economic loss, legal costs, etc.; the devastating effect addiction has on the lives of the addicts and their families is equally costly both economically and emotionally. Since 2003, I have researched this complex phenomenon in an effort to use my computer science, mathematic and dynamical systems skills to find ways to assist addicts in their efforts to overcome drug use.

A number of different addiction models exist. At one end of the spectrum, addiction is portrayed as a disorder that becomes monotonically worse, leaving the addict no way out, due to the positive feedback created by addictive substances [6]. At the other end of the spectrum, addiction is viewed as a sort of life phase people grow into and out of: youngsters, who during high school and college, try different drugs, then at a later point in their lives overcome substance abuse with no need of external help [7]; our view lies somewhere in between.

Animal modeling in rats reveals parameters causing rodents to reinstate drug use, including stress, drug cues, and priming [8], as well as genetic factors [9], and living conditions [10]. Valid analogies can be drawn from the rat studies, but obviously, some issues pertaining to higher cognition and rationality are undoubtedly missing. Studies describe addiction as a cyclical condition [11], characterized by periods of abstinence followed by relapse. It is relapse that makes addiction so difficult to treat. Both clinical and anecdotal evidence show clearly that short-term treatment does not help in avoiding relapse. It is this observation that made us resort to dynamical system theory to look for recovery and relapse prediction.

Recent seminal studies [12] followed addicts, providing them with mobile devices and communicating with them a few times a day. Addicts were asked to input any significant, drug-use related events. This sort of approach has the potential to be able to predict tendencies that forewarn of upcoming relapse, which can be extraordinarily beneficial in clinical recovery. Unfortunately, statistical

Received November 1, 2010; accepted December 17, 2010

Hava T. SIEGELMANN (✉)
Department of Computer Science, UMass Amherst, Amherst, MA
01003, USA
E-mail: Hava@cs.umass.edu

correlations described in this particular study were too weak to be of immediate use. We propose that the weak points in the study were related to the fact that the analysis did not consider dynamical trends in the subjects' emotional and behavioral history and ignored connections between parameters within the same categories. We suggest that our dynamical systems approach, with its analog values, active memory, and consideration of internal state, can more fully and accurately describe the global state of the addict.

In this paper we propose an abstract dynamical system approach to drug addiction. We will emulate moments of abstinence and the internal state prior to and during loss of sobriety to grade addiction and to predict relapse. We will show that through the use of non-monotonic dynamics, even hard core addicts (those with genetic predispositions to addiction and those with difficult socio-familial backgrounds) have a chance at rehabilitation, if treatment is applied consistently and interventions are correctly timed.

While our mathematical model can accurately describe efficacious treatments, actual clinical intervention is beyond the scope of the current work. Although it is very desirable to treat actual compulsion and craving, our present model does not yet address these issues and focuses on the behavioral aspects of refraining from drug abuse. 12 step programs, stress relief therapies, such as acupuncture and meditation, religious groups and motivational support programs do carry some successes. However the success of these programs is sporadic, fits some and not others; it is impossible to predict at the outset of treatment which treatments fit which individuals and which individuals will benefit most. All parties involved would like to see a higher ratio of success.

We suggest that addiction's cyclical nature with the peaks and valleys of non-use and use necessitate a dynamical treatment solution to achieve recovery. We propose that given data, our model can be used to personalize the description of each addict's disorder in mathematical terms, with the potential to suggest highly coordinated, individual therapies.

2 Previous work

Decision-making in severe addiction is dynamically biased toward drug seeking with a strong proclivity toward drug cues and minimized inhibition [13]. Previous generations of computational models focused on describing the process of drug seeking as monotonic with positive feedback. This kind of mechanism "locks" the system and prevents a way out, e.g., Incentive salience via the temporal difference reinforcement learning (TDRL) framework [14], becoming addicted by a change in evaluation based on a drug's effect on dopamine [6], or neg-

ative reinforcement in a psychopharmacology setup [15]. Berridge and Robinson [16], insightfully appreciating the dynamic nature of addiction, proposed the incentive sensitization theory, where they distinguish between liking the drug's effect, i.e. the reward value, and wanting to consume the drug, i.e., the craving. This suggests dynamics whereby hedonic liking effect may decrease as tolerance builds over time, at the same time that the wanting-craving effect may increase; As addiction models became more complex with multiple subcomponents providing increasingly individualized representations of addicts, the subcomponents remained binary and monotonic, making the model acyclic, binary and monotonic-locked modeled behaviors on rigid paths that did not necessarily accurately reflect reality.

More recent addiction models recognize that addiction is not unidirectional or an unbounded sink. Thoughts to reconcile reinforcement learning paradigms with relapse have been raised [17]. Dezfouli and colleagues [18] incorporated the elevation of basal reward threshold after long-term addiction into TDRL models. Their model addresses the unlearning of seeking behavior, but cannot address mathematically its reinstatement (see page 18). In later work [19] Redish et al. suggest addiction may involve ten different vulnerabilities. Their notion of dynamics is based on the existence of two subsystems: one that plans flexibly and the other that is based on habits, and on a binary behavioral controller that switches between these two systems, as was also suggested in [20,21]. While relapse is mentioned, it is left at the level of discussion, but work by Bickel and Yi [22] suggested how to advance this model toward applicability. We note that biological studies do not provide sufficient evidence that the brain's habit and planning areas are independent of each other, but suggest rather, that they may have information loops between them. This brings us to suggest that conflicting subsystems may be combined by a continuous/real (rationality) value parameter, so that instead of one system taking full control of the other, both systems pull with different biases toward a common, compromised solution.

In Ref. [23], Ahmed et al. provide a review of existing pharmacological models, stating that they are unable to describe even the most basic dynamic changes in addiction. Gutkin's model [24] constitutes an interesting dynamical approach to nicotine addiction, which explains processes at the neural level more closely than the previously mentioned abstract models. Gutkin's model is based on the use of varying time scales and the combination of reinforcing and opponent processes in the development of rigid, addictive behavior. This model though, cannot account for the unlearning of seeking behavior and reinstatement after prolonged drug exposure, as discussed in Ref. [18].

A highly intuitive line of work is found in the adaption

of mathematical economic models to represent addiction. The “rational addict” theory [25] explains addictive behavior in economic terms, as taking steps to maximize enjoyment under the addict’s skewed view of reality and placing a high value on the desired substance. Bernheim and Rangel [26] add to previous theories the notion that the addict can make mistakes and may state a sincere intention to quit without following through. Similar to the above-mentioned flexible and habitual subsystems, they argue the existence of two modes of decision-making. The “hot” mode is mechanical and may be compulsive, while the “cold” mode involves active cognitive control. In their view, the cold mode will not allow for drug use unless it includes mistakes. They urge the need of pre-commitment, meaning a willingness to, without exception, make a commitment to sever certain behaviors. They also state the need for self-control for rehabilitation. Our main dynamical equation, which is the basis of our rationality model, parallels the next stage in economic models, where instead of one rational behavior, there is a dynamic rationality that biases between immediate and delayed rewards.

Viewing addiction as a dynamical rationality disorder, leads to the notion of dynamical rehabilitation. Literature describes addicts who grow out of addiction with no formal treatment program [7,27–29], addicts who minimize their use due to economic, social, or religious incentives [30], addicts who require traumatic or extreme existential distress before seeking help, and others who benefit from medications. We suggest that even given the physiologic changes addicts undergo with prolonged drug abuse, and the resultant reduction in their abilities to plan and inhibit, that in light of “dynamic recovery” as set out in our model, rehabilitation is still practicable.

3 Dynamical system model

Given addiction’s complexity both in terms of multiple underlying causes and in actual behaviors, we propose that the intricate balance underlying sobriety cannot reasonably be described by a binary parameter like “prone to abuse” versus “not prone to abuse”. We agree that in the addict’s inner debate, to use or not use drugs, one voice will ultimately predominate at any given moment. However, we view the internal process as a complex patchwork of gray areas that represent more this and less that, rather than the more simplified-this or that of binary functions. Realizing that the internal state, in terms of a dynamical system, will contain many levels that in combination will lead at the end to one observed behavior or another, we feel a continuous value is necessary to accurately describe the individual state. We further hypothesize that this dynamical view is directly relevant to issues of recovery.

The theory of dynamical systems consists of equations that govern the temporal evolution of the values of an interacting set of variables and parameters [31]. This fundamental mathematical field is used to explain such widespread phenomena as jetlag [32], social and economic interaction [33], and cancer growth [34]. It has also given rise to subfields such as chaos theory and complex systems theory [35]. The term *Dynamical Diseases* [36] refers to qualitative changes in the regular dynamics of physiologic systems, e.g., as occurs in heart arrhythmia and in schizophrenia. We have broadened this concept to include the “dynamical treatments hypothesis” [37], which adds the idea that a given system can be dynamically mutated by some parameters, yet restored by other means.

Dynamical systems theory has yet to be directly applied to addiction. It carries a great potential to better model and analyze an individual’s withdrawals, relapses and to intervene before relapse occurs.

3.1 Emotional-cognitive rationality factor

We suggest a high level model that separates internal processes such as craving, emotional stress and inhibition, from the actual decision-making and behavior leading directly to substance abuse and addiction.

Our suggested master equation is reminiscent of previous models in recognizing two opposing subsystems (e.g., hot versus cold, habit versus planning). Unlike previous models, the two subsystems output scalars, and thus no longer have to be combined via temporal selection of a binary switch, but rather, can be mediated by a continuous parameter that allows both to affect outcome simultaneously. We call this mediating parameter, which is the heart of our model, “the emotional-cognitive rationality factor”, a generalization of the rational model, which reflects the complexity and internal dynamics of addiction.

The rationality factor $R(t)$ is a temporal function, here having the hyperbolic tangent functional form and residing in the interval $[0,1]$. Its value is affected by its previous value as well as by current filtered inputs $f(t)$ (such as drug cue or dopamine flashes) from the external world and other internal systems. $R(t)$ is also affected by the recovery power $H(t)$, to be characterized in Sect. 4. The parameters α , β , and γ balance the influence of these components on R :

$$R(t) = \frac{1}{2} \tanh(\alpha R(t-1) + \beta f(t) + \gamma H(t)) + \frac{1}{2}. \quad (1)$$

This equation is abstract; like previous economic models, it requires a live database, something we are in the process of collecting. The value of $R(t)$ can be thought of as representing an ongoing, changing activity of neuronal patterns, which act on a faster time scale (reminiscent of

Ref. [24]) relative to the other parameters in the master equation described in the next subsection.

This paper is not meant to specify the exact affecting parameters in f and H , since it would be premature prior to data collection, yet Dr. Dino Levy (then a graduate student at the Weizmann Institute) has already suggested two ways to model them: One focuses on neurotransmitters only, translating all internal and external events to levels of Dopamine, Glutamate, and Gaba as well as stress related hormones in different brain areas and with respect to neurotransmitter production or extra-cellular levels. Dino Levy also proposed another level of modeling, where psychological variables focusing on stress, external events, trials and tribulations in life, drug-related cues and environment can also be used; yet significantly, boredom and fatigue and other parameters should be included there as well. We also believe, based on the successes of 12 step type programs, that the inclusion of a variable representing the concept of a “higher power”—encompassing spirituality, motivation, social support, religion, etc. would significantly enhance the model, which is why H is included in Eq. (1). Another physiologic modeling approach might follow the same higher Eqs. (1) and (2) and focus on genetic factors such as D2 receptor physiology, volume of prefrontal cortex and changes in the limbic and reward systems.

3.2 Master equation

Our model is based on four main variables: The “**global abstaining**” variable (G) indicates the strength in resisting substance abuse. The higher G 's value, the less likely an agent is to consume drugs.

The “**inhibition**” variable (I), affected by a combination of genetic and social factors, is related to an individual's self discipline and ability to wait for satisfaction.

The “**craving**” variable (C), which mathematically serves as the balancing factor of I , is related to concepts of liking, craving, wanting and rewards. I may be associated with frontal cortex development and volume, as well as social rules. There are genetic and social risk factors that increase the probability of becoming an addict; only about 17% of people who consume drugs become addicted [38]. If C is high, then the individual demonstrates a more compulsive nature and will have a stronger predisposition to addictive behavior. Conversely, the higher the I value is, the higher is inhibitory control, which helps prevent addiction. Both I and C are also dependent on the emotional-cognitive rationality variable R , in addition to being weighted by it for the purpose of calculating G . The dependence is not described in this paper.

The “**emotional-cognitive rationality**” (R) variable, weighs the relative balance between craving and inhibition. Connections between emotions and decision

making are well documented [39]. Under severe stress, rationality is low and craving takes over, yet with self acceptance I is higher and thus inhibition has a higher role in the final behavior. In our model, drug seeking is explicitly controlled by the mediating parameter, and has proven to be affected by both internal (e.g., emotional stress during withdrawal) and external processes (e.g., drug-related cues); social support and economic incentives may also increase R .

For normalization purposes we can bind I and C like R in the $[0,1]$ domain. The behavior parameter G is bounded in $[-1,1]$ where we think of positive numbers as no-use and negative numbers as higher likelihood of use. The relations between these parameters is as follows: Low levels of R cause craving to dominate, which may decrease the global abstaining value G . High levels of R will cause inhibition to dominate, exerting an inhibitory control over behavior that might otherwise have negative, long-term consequences. When I and C are similar to each other (within the 0.5 range of each other) the behavior oscillates around the 0 point and is highly dependent on R . If $R = 0$, then our model is reminiscent of previous theories describing addiction in terms of rationality, craving or wanting. The cyclical behavior in our model increases due to the rapid changes in the values of R relative to the C and I parameters.

One possible function with these characteristics is

$$G(I, C, R) = \tanh\left(\frac{R}{1 - RC} \frac{I}{C} - 1\right). \quad (2)$$

Figure 1 demonstrates the dynamics of the function G . For demonstration purposes, we take R to be the time dependant, periodic sinus function:

$$R = \frac{1 + \sin t}{2}.$$

There are physiologic justifications to our proposal of multiple systems working in parallel with a soft bias between them. Extensive data exists suggesting that cognitive action planning and inhibition is associated with “loops” between cortical (frontal and medial temporal) areas and the dorsomedial striatum, whereas habitual and compulsive behavior is more associated with “loops” between frontal cortical areas and the dorsolateral striatum. The pathophysiology of addiction is heavily mediated by alterations in neurotransmitter systems and in these loops. It can be expected that one of the main underlying problems of addiction is the altered function of these pathways, which can result in a shift from a balanced compulsive-inhibition system to a biased situation.

4 Higher (recovery) power

The inherent ability of a perturbed dynamical system

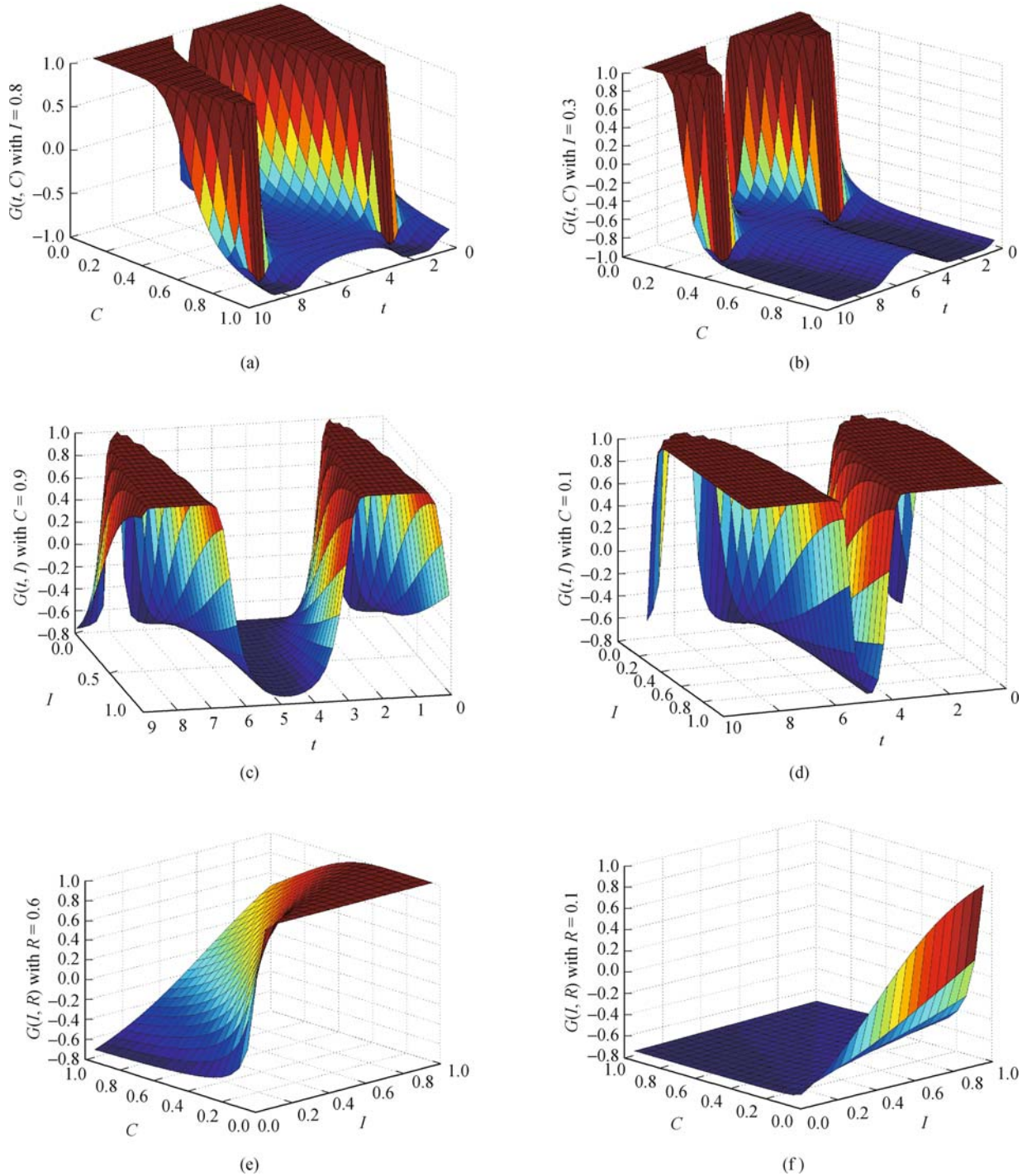


Fig. 1 (a) and (b) depict G where the value of I is fixed to high (0.8) and low (0.3), respectively. We see in the first case that the function is mainly in the positive domain while in (b), G follows C and tends to be negative except for very high R and low C . (c) and (d) depict G for a fixed value of C , being 0.9 and 0.1 respectively. We can see that the function G is low most of the time during high C , except for very high values of R and I , and is high most of the time for low C . (e) and (f) draw G relative to R . (e) shows $R = 0.6$, which balances the function between I and C , (f) biases G to low number since it contains low value of the rationality parameter, $R = 0.1$

to recover is intrinsic to our model. After detoxification, in the confines of a rehab facility, particularly post-withdrawal, the individual is protected against drug cues. But outside the facility, such an ideal environment is impossible to maintain. For recovery, an elevation of endurance and resistance to relapse is needed. In real world situations, 12 step groups have successfully used

the concept of a “higher power” to increase resistance to drug cues, elevate endurance and resistance to relapse. Mathematically, this higher recovery power represented in our model, by “ H ”, is a temporal function as well. To provide a solution and raise the global abstaining variable $G(t)$ sufficiently, $H(t)$ should have the following dynamic memory property: that it can respond to im-

pulse by increasing its value; each time it is boosted, it remains elevated for a longer time than before, and if it was elevated for some time (due to well timed impulses), it will remain at higher values longer. Conversely, if no impulse has arrived for some time, the memory of prior impulses starts fading. The H function is somewhat like an electrical circuit with memory that counts impulses, but whose effect fades with time. H is a parameter of R and it may keep R in decent rationality by causing rationality to elevate as a response to H 's value remaining high and thus it also elevates G , see Fig. 2. Hence, when the rationality is at risk of descending, H must be elevated at once.

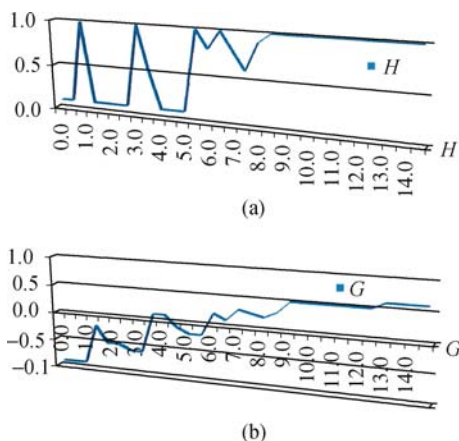


Fig. 2 (a) Higher power value (H) increases with impulses; (b) the global ability to cease drug-seeking behavior is expected to increase due to the change in H

As previously mentioned maturing out, pre-commitment and motivation to recover, family and social support, economic help, rehabilitation programs, 12 step meetings, psychological support, religion, stress relief, as well as pharmacological substances such as methadone or nicotine replacements, have all been proposed as boosting recovery power [40,41]. As these attributes increase in frequency, their effects tend to stay longer. In terms of achieving recovery, the purpose is to achieve as consistent reinforcement as possible. Therefore, increasing support frequency is key to recovery, and when the affect of support is about to diminish it is crucial to provide further support and keep the higher power working since it is easier to keep it up than to rebuild it. As long as an attribute has this quality of staying longer as its frequency increases, it has the potential to significantly aid recovery. This concept of increasing reinforcement frequency to increase reinforcement effect is associated with memory function and learning. Our work in memory and intelligence has numerous parallels and connections to our addiction model and has significantly informed and shaped our work in modeling addiction.

The function H can be elevated by reducing guilt induced stress (e.g., accepting oneself), undermining the

anticipation of substance use as the only thing that can relieve internal pain (e.g., lowering associations with substance cues and positive outcome, re-introducing other rewards and building social support), using positive incentives to increase “recovery power” and reduce vulnerability to environmental cues and real life events. Techniques of recalling a better life without drugs as well as its negative counterpart, recalling problems that stemmed from drug use, may assist in increasing inhibition and reducing anticipation. Finally, reducing substance use cues (e.g., by moving to a new area, staying away from friends associated with drug use) is recommended since acquired endurance can never be perfect.

5 Website

As previously mentioned, increasing the frequency of recovery supports, increases their effect and increases the likelihood of continued recovery. In an effort to build a tool that would assist addicts with daily reinforcement of their recovery, we designed an interactive, “journaling” website. Addicts take part in a brief, daily report (not more than a five minute effort) and always have access to an online journaling feature that provides them with a place to vent feelings and record thoughts. For simple logistic and economic reasons, most addicts seeking it have only intermittent help in their recovery efforts. Organizations like 12 step anonymous groups and sponsorship within these groups have perhaps, come closest to providing frequent, consistent support to the addict. But even with the excellent work these groups do, there is a limit to how much attention any individual addict can get over the long periods of time that make up recovery. Our site is being developed as an automated, continuous contact resource for addicts-not to replace current recovery resources, but to augment them. Enhancements are now being made to our site to incorporate natural language understanding via commonsense databases that would allow intelligent, syntactical analysis of journal entries, intelligent analysis and classification of questionnaire data, even gaining insight from a respondent’s choice not to answer.

As addicts interact with the website, the engine behind the database will become familiar with their lives, work, friends, emotional state, and will gain insight into what preceded craving or relapse and what strengthened the individual. The immediate benefit is feedback to the user: Individuals would receive weekly reports comparing the preceding week in terms of behavior, social marks, work, and emotional state with weeks prior. While users are not required to provide information and can skip questions as they see fit, the more they complete, the more they get out of the process. The longer term benefit of the website is the opportunity to use

the data to create increasingly accurate, more predictive models. Potential uses incorporating data generated through the website, include everything from immediate feedback and advice prior to tactical behavior changes, such as relapse or identifying points when assistance is most needed, to contacting rehab clinics for help-as-needed so the individual has continuous support.

6 Conclusions

This introductory paper describes a mathematical model that stands on the digital shoulders of those models that have preceded it. Our primary contributions, the use of real, analog values versus simple binary, non-monotonic functions, the ability of the model to realistically model non-monotonic relapse and cessation, and importantly the inclusion of H , the higher power function, to name a few, demonstrates the great utility of applying dynamical system modeling to addiction. We call for future work that will provide analytical forms for the H and R functions, to further enhance individualized mathematical modeling for those suffering from addiction and to develop broad-based, personalized treatment plans. The model acts as a bridge between the fields of mathematical modeling, neuroscience, psychology, sociology and information technology - bringing a new and badly needed tool to the front lines of the battle against addiction.

Acknowledgements We are grateful to the Office of Naval Research, which supported this work in part through grant #109-0138R. Kun Tu joined in the development of Eq. (2) and Fig. (1); Eric Goldstein edited the paper thoroughly; Dino Levy, Nora Volkow and Jerry Meyer helped by introducing previous works of addiction, pushing the model to be as realistic as possible.

References

1. Substance Abuse and Mental Health Services Administration. Results from the 2007 National Survey on Drug Use and Health: National Findings. NSDUH Series H-34, DHHS Publication No. SMA 08-4343, 2008
2. Kreek M J, Nielsen D A, Butelman E R, LaForge K S. Genetic influences on impulsivity, risk taking, stress responsivity and vulnerability to drug abuse and addiction. *Nature Neuroscience*, 2005, 8(11): 1450-1457
3. Volkow N D, Fowler J S, Wang G J. The addicted human brain viewed in the light of imaging studies: brain circuits and treatment strategies. *Neuropharmacology*, 2004, 47(Suppl 1): 3-13
4. Jones A M. Health, addiction, social interaction and the decision to quit smoking. *Journal of Health Economics*, 1994, 13(1): 93-110
5. Kleber H D, Weissman M M, Rounsaville B J, Wilber C H, Prusoff B A, Riordan C E. Imipramine as treatment for depression in addicts. *Archives of General Psychiatry*, 1983, 40(6): 649-653
6. Redish A D. Addiction as a computational process gone awry. *Science*, 2004, 306(5703): 1944-1947
7. Heyman G M. *Addiction: a Disorder of Choice*. Cambridge: Harvard University Press, 2009
8. Soria G, Barbano M F, Maldonado R, Valverde O. A reliable method to study cue-, priming-, and stress-induced reinstatement of cocaine self-administration in mice. *Psychopharmacology*, 2008, 199(4): 593-603
9. Johnson P M, Kenny P J. Dopamine D2 receptors in addiction-like reward dysfunction and compulsive eating in obese rats. *Nature Neuroscience*, 2010, 13(5): 635-641
10. Alexander B K. "The Myth of Drug-Induced Addiction", a paper delivered to the Canadian Senate, 2001
11. Dockner E J, Feichtinger G. Cyclical consumption patterns and rational addiction. *American Economic Review*, 1993, 83(1): 256-263
12. Epstein D H, Willner-Reid J, Vahabzadeh M, Mezghanni M, Lin J L, Preston K L. Real-time electronic diary reports of cue exposure and mood in the hours before cocaine and heroin craving and use. *Archives of General Psychiatry*, 2009, 66(1): 88-94
13. Goldstein R Z, Volkow N D. Drug addiction and its underlying neurobiological basis: neuroimaging evidence for the involvement of the frontal cortex. *American Journal of Psychiatry*, 2002, 159(10): 1642-1652
14. McClure S M, Daw N D, Montague P R. A computational substrate for incentive salience. *Trends in Neurosciences*, 2003, 26(8): 423-428
15. Ahmed S H, Koob G F. Transition to drug addiction: a negative reinforcement model based on an allostatic decrease in reward function. *Psychopharmacology*, 2005, 180(3): 473-490
16. Berridge K C, Robinson T E. Parsing reward. *Trends in Neurosciences*, 2003, 26(9): 507-513
17. Redish A D, Jensen S, Johnson A, Kurth-Nelson Z. Reconciling reinforcement learning models with behavioral extinction and renewal: implications for addiction, relapse, and problem gambling. *Psychological Review*, 2007, 114(3): 784-805
18. Dezfouli A, Piray P, Keramati M M, Ekhtiari H, Lucas C, Mokri A. A neurocomputational model for cocaine addiction. *Neural Computation*, 2009, 21(10): 2869-2893
19. Redish A D, Jensen S, Johnson A. A unified framework for addiction: vulnerabilities in the decision process. *Behavioral and Brain Sciences*, 2008, 31(4): 415-487
20. Daw N D, Niv Y, Dayan P. Uncertainty-based competition between prefrontal and dorsolateral striatal systems for behavioral control. *Nature Neuroscience*, 2005, 8(12): 1704-1711
21. Redish A D, Johnson A. A computational model of craving and obsession. *Annals of the New York Academy of Sciences*, 2007, 1104(1): 324-339
22. Bickel W K, Yi R. Addiction science as a hedgehog and as a fox. *Behavioral and Brain Sciences*, 2008, 31(4): 441-442
23. Ahmed S H, Graupner M, Gutkin B. Computational approaches to the neurobiology of drug addiction. *Pharmacopsychiatry*, 2009, 42(Suppl 1): S144-S152
24. Gutkin B S, Dehaene S, Changeux J P. A neurocompu-

- tational hypothesis for nicotine addiction. Proceedings of the National Academy of Sciences of the United States of America, 2006, 103(4): 1106–1111
25. Becker G S, Murphy K M. A theory of rational addiction. *Journal of Political Economy*, 1988, 96(4): 675–699
 26. Bernheim B D, Rangel A. Addiction and cue-triggered decision processes. *American Economic Review*, 2004, 94(5): 1558–1590
 27. Winick C. Maturing out of narcotic addiction. *Bulletin on Narcotics*, 1962, 14(1): 1–7
 28. Maddux J F, Desmond D P. New light on the maturing out hypothesis in opioid dependence. *Bulletin on Narcotics*, 1980, 32(1): 15–25
 29. Waldorf D. Natural recovery from opiate addiction: some socio-psychological processes of untreated recovery. *Journal of Drug Issues*, 1983, 13(2): 237–280
 30. Tucker J A. Different pathways to knowledge about different pathways to recovery: a comment on the people awakening study. *Addiction*, 2008, 103(2): 216–217
 31. Alligood K T, Sauer T, Yorke J A. *Chaos, An Introduction to Dynamical Systems*. New York: Springer-Verlag, 1997
 32. Leise T, Siegelmann H. Dynamics of a multistage circadian system. *Journal of Biological Rhythms*, 2006, 21(4): 314–323
 33. Rapatski B L, Suppe F, Yorke J A. HIV epidemics driven by late disease stage transmission. *Journal of Acquired Immune Deficiency Syndromes*, 2005, 38(3): 241–253
 34. Olsen M M, Siegelmann-Danieli N, Siegelmann H T. Dynamic computational model suggests that cellular citizenship is fundamental for selective tumor apoptosis. *PLoS ONE*, 2010, 5(5): e10637
 35. Glass L, Mackey M C. *From Clocks to Chaos: The Rhythms of Life*. Princeton: Princeton University Press, 1988
 36. Belair J, Glass L, An Der Heiden U, Milton J. Dynamical disease: identification, temporal aspects and treatment strategies of human illness. *Chaos*, 1995, 5(1): 1–7
 37. Siegelmann H T. Complex systems science and brain dynamics. *Frontiers in Computational Neuroscience*, 2010, 4(7): 1–2
 38. Anthony J C, Warner L A, Kessler R C. Comparative epidemiology of dependence on tobacco, alcohol, controlled substances, and inhalants: basic findings from the national comorbidity survey. *Experimental and Clinical Psychopharmacology*, 1994, 2(3): 244–268
 39. Koenigs M, Young L, Adolphs R, Tranel D, Cushman F, Hauser M, Damasio A. Damage to the prefrontal cortex increases utilitarian moral judgements. *Nature*, 2007, 446(7138): 908–911
 40. Monterosso J, Ainslie G. The behavioral economics of will

in recovery from addiction. *Drug and Alcohol Dependence*, 2007, 90(Suppl 1): S100–S111

41. Niv Y, Joel D, Dayan P. A normative perspective on motivation. *Trends in Cognitive Sciences*, 2006, 10(8): 375–381



Hava T. SIEGELMANN is an associate professor of Computer Science and Neuroscience at the University of Massachusetts at Amherst, and the director of the BINDS (Biologically Inspired Neural and Dynamical Systems) laboratory. Dr. Siegelmann's research focuses on mathematical

modeling of biological systems with particular interest in memory, epigenetics, cellular development and disease evolution. Her research into biologically inspired memory and artificial intelligence has led to machine systems, which are more autonomous: capable of learning, tracking, clustering, associating, and inferring and are more robust and capable of operating in real-world environments. She introduced the highly utile Support Vector Clustering algorithm with Vladimir Vapnik and colleagues. Siegelmann's seminal Turing machine equivalence of recurrent neural networks theorem and the super-Turing theory, which greatly impacted current thinking on computation, have found new utility in her work on machine memory reconsolidation and intelligent cellular function. Her work is often interdisciplinary, and combines methods from the fields of Complexity Science, Networks Theory, Dynamical Systems, Artificial Intelligence and Machine Learning. *Dynamical Health* is Siegelmann's recent thesis stating that an unbalanced dynamic is the cause of most systemic disorders, that returning the system to balance is extremely beneficial to healing and further, that it is too limiting to focus only on primary causes, when any treatment that returns balance is sufficient for healing. Modeling these systems mathematically provides a means of exploring many possible solutions, which can then be translated to actual treatment. Her recent system biology studies include genetic networks, circadian system, memory reconsolidation, miRNA, cancer, and now addiction. She remains active in supporting young researchers and encouraging minorities and women to enter and advance in the sciences.