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Computational Hypothesis for Maturing Out of Addiction and Mindfulness-Based Cognitive Techniques

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Abstract. Use and misuse of addictive substances has been an ongoing phenomenon from early civilizations to the present. Experimental observations endorse the implication of a cognitive component during the addictive course. The present investigation proposes a learning mechanism affecting the cognitive level of a multiscale model of addiction. Simulations account for plausible initiations of natural recoveries through non-traditional techniques, such as meditation. This framework suggests that such plasticity mechanism within the cognitive substrate of an addict may be necessary in order for a maturing out experience to begin and possibly endure over time.

Keywords. Computational Model, Multiscale, Dynamical System, Drug Addiction, Natural Recovery, Cognitive Learning

Introduction

While addiction has been widely regarded as detrimental and even criminal, popular beliefs have converged to consider addiction as a "bio-psycho-social-spiritual disorder" [1]. Through the 1990s, the predominant standpoint to describe the strong desire to use a drug was based on the psychological theory of classical conditioning. Increasing positive association between drug intakes and gratification was postulated as the main aspect inducing the intensification of drug intakes. In 1990, Tiffany expanded on this view by introducing a cognitive process model for addiction, and further characterized the relationship between drug use and craving [2]. His model proposes that a longtime addict will undergo a drug intake not because of the strong association between drug intake and consequent feeling, but as a result of a mindless and automatic process triggered by an external event (e.g., the meeting with a drug supplier). In this model, craving experiences arise when the automatic response to the external event fails (e.g., the drug supplier is not available), and the addict requires cognitive efforts to overcome the situation [3]. Neuroscientific evidence introduced a decade later suggests that drug intoxication correlates, in addition to neural activities in the limbic system, with

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structures in the prefrontal cortex, which further supports Tiffany's model. In 2002, Goldstein and Volkow introduced the "impaired response inhibition and salience attribution" view of addiction to account for the addict's archetypal progression (intoxication, craving, compulsive intakes, withdrawal, and again intoxication) [4]. Two processes compose this framework: one is a progressive transition from deliberate to automatic drug-related behaviors, and the other is an increased predisposition for immediate rewards, even if possibly harmful in a longer term. The prefrontal cortex, pivotal to high-level cognitive functions is "likely to play an important role in the cognitive behavioral and emotional changes that perpetuate drug self-administration" [4].

Craving is a major component leading to relapse, and understanding how to rehabilitate an addict is an important objective in addiction studies. The present investigation presents a multiscale formal model of addiction, which considers recovery and emulates the theories discussed above. This framework evaluates a computational hypothesis on how cognition may operate as a mediator between behavior and neural activity, while accounting for observations known as natural recoveries. The results support the thesis for which a simple computational learning mechanism within the cognitive substrate could significantly impact the success of a recovery process.

1. A Multiscale Model of Addiction

Formal models of addiction encompass epidemiological models [5,6], economic models [7,8], pharmacological models [9-11], abstract models of dopaminergic functions [12-14], and the more recently proposed systemic models [15,16]. In this paper, a systemic model is advanced, as shown in Figure 1, which aims to characterize the comportment of a human being through its tendency of drug-seeking behavior.

This computational framework was defined and validated [17], its dynamics and sensitivity were analyzed [18, 19], and its recovery scale initiated [20].

The model shown in Figure 1 comprises neuropsychological, recovery, cognitive, and behavioral elements. The neuropsychological scale incorporates internal and external processes describing the neural ongoing activity. Internal processes include the level of negative consequences such as poor health or social relations P(t), the level of negative emotional state S(t), the level of dopamine craving D(t), and the saliency of drug-associated cues Q(t). The external processes characterize sudden experiences that, when activated, instantly influence the subject's neural activity. These are: drug-associated cues $A_Q(t)$, that may be triggered by an event such as visiting a "drug buddy"; painful traumas $A_P(t)$, that may cause an addict to stop taking drugs immediately for a period of time; strong stressful episodes $A_S(t)$, that may lead a former addict into immediate drug-use; and drug priming $A_D(t)$ such as social drinking, that may bring the virtual subject into drug-use again. The output of the model G(t), by way of the cognitive scale, depends on both internal and external processes. The process G(t) defines a feedback loop to the neuropsychological scale. The behavioral scale includes the model's output G(t), which is a qualitative evaluation of a virtual subject's tendency for drug-seeking, and arises from the antagonism between inhibitory and compulsory elements. Negative values of G(t) correspond to maladaptive behavior, whereas positive ones accounts for healthy behavior. For the sake of clarity, the processes of inhibition and compulsion are considered constants even though explicit time dependencies of these processes were previously defined [17].

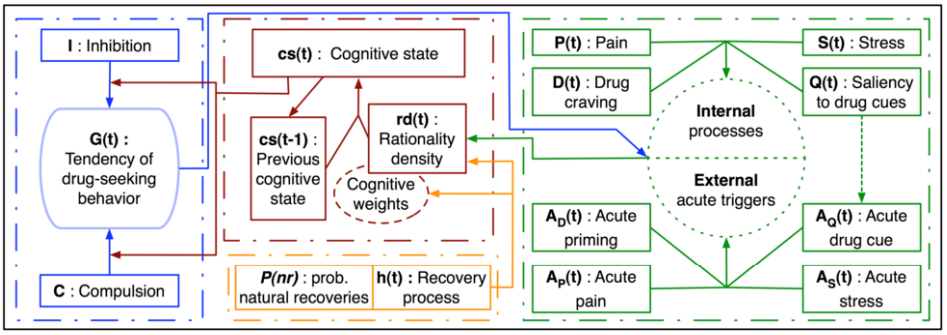

Figure 1. Diagram of the computational model, where the output of the model G(t) represents the tendency for drug-seeking behavior. The levels of observations represented include the neuropsychological scale in green, the recovery scale in orange, the cognitive scale in red, and the behavioral scale in blue.

The cognitive scale complies with the definition in [21]: "A system is cognitive if and only if sensory inputs serve to trigger actions in a specific way, so as to satisfy a viability constraint." This scale is established as a mediator between low and high level of behavioral control. In a perceptron-like architecture, the neuropsychological processes are weighted and integrated to define the degree of rationality rd(t), which drives the virtual subject's cognitive state cs(t) toward a more inhibited or a more compulsive behavior. The recovery scale is a computational hypothesis which relies on the definition of natural recovery, for which the addict ceases using the drug "without the help of treatment intervention" [22], suggesting a recovery process solely impacting the cognitive scale (no pharmacological nor behavioral interventions). The recovery process is designated as a sudden cognitive change which, when induced, makes achievable a drug abstinence period that may or may not endure. The recovery scale affects the virtual subject's cognitive state by direct intervention on its rationality estimation, and by modulating the weights of internal and external processes. In the next section are presented the formal descriptions of the cognitive and recovery scales. Computational definitions of the neuropsychological and behavioral scales are reported in [17].

2. Methods

This section presents the details of the cognitive and recovery scales, as well as the data used to emulate instances of the recovery process.

The rationality density rd(t) is defined as a weighted sum of the neuropsychological and recovery processes:

$$rd(t) = \sum \omega_i(t) \cdot \Phi_i(t) \quad , \quad \Phi_i = \{S, P, D, A_S, A_P, A_D, A_Q, h\} \tag{1}$$

The weights ω_i for the processes P(t) and $A_P(t)$ are in $\mathbb{R}^+$, whereas the other weights are in $\mathbb{R}^-$. This rationality estimate drives the cognitive state cs(t) of the virtual subject:

$$cs(t) = \frac{1}{2}\tanh\left(\alpha \cdot cs(t-1) + \beta \cdot rd(t) + \gamma\right) + \frac{1}{2} \tag{2}$$

Values of α, β, and γ are constants and their sensitivity is discussed in [19].

The recovery process h(t) is equal to a constant value h_0 while triggered or active. This process is active for a certain time Θ(t), which increases for consecutive instances of h(t):

$$h(t) = \begin{cases} h_0 & if\{p > P(nr) \mid t_h \in [1,\Theta(t)]\} \\ 0 & else \end{cases} \tag{3}$$

$$\Theta(t) = \begin{cases} \Theta(t) + \delta_i & if\{h(t) = triggered\} \\ \Theta(t-1) & if\{h(t) = active\} \\ \max\left(0,\Theta(t) - \delta_d\right) & if\{h(t) = 0\} \end{cases} \tag{4}$$

The probability P(nr) defines the possibility of a natural recovery and is discussed in the next subsection. The constants δ_i and δ_d are in $\mathbb{N}$, and t_h is a time step counter reset to 1 at every recovery instance.

When a recovery process arises, it influences rd(t) through two mechanisms. On the one hand, h(t) has a direct effect on rd(t) as defined in Equation 1. On the other hand, the cognitive weights ω_i of the processes P(t), S(t), and D(t) provisionally change their values according to the relationship:

$$\omega_i(t) = \begin{cases} \kappa_i + \Delta_{\kappa i} & if\{h(t) = h_0\} \\ \kappa_i & else \end{cases} \tag{5}$$

where $\Delta_{\kappa i}$ is in $\mathbb{R}^-$ for S(t) and D(t), and in $\mathbb{R}^+$ for P(t).

At the last active time step of a recovery process h(t), the temporary effect on ω_i of processes P(t), S(t), and D(t) can become permanent with arbitrary probability θ_i:

$$\kappa_i(t) = \begin{cases} \kappa_i + \Delta_{\kappa i} & if\{t = \Theta(t)\} \quad and\{p > \theta_i\} \\ \kappa_i & else \end{cases} \tag{6}$$

2.1. Probability of a recovery process P(nr)

In 1962, Charles Winick popularized the phenomenon of maturing out of narcotics addiction, revealing cases where regular heroin and synthetic opiate abusers ceased using the substance without any psychological or pharmacological treatment [23]. In 1980, Maddux and Desmond discussed the possible overestimation of Winik's statistics, and proposed further data to increase the accuracy of the study [24]. Maddux and Desmond confirmed that the trends of age distribution for withdrawal initiations were consistent in both studies, and argued the possible overestimation due to the disregard of cessation onset rates in the base addict population.

In the present investigation, data reported in [23] and [24] are combined to quantify the likelihood of a narcotic addict to undergo a "maturing out" experience. Winick based his investigation on the number of addicts reported to the Federal Bureau of Narcotics in 1955 that were not reported again during a five-year period [23]. As reported in Table 1, the probability for an addict to experience a maturing out

experience is inferred (fourth column in Table 1). This probability is scaled using the subsequent results by Maddux and Desmond, which report the annual rates of abstinence onset in the base population [24]. For simplification purposes, the age category "All ages" in [24] is considered to describe the age range from 0 to 19 years old, and the category "40-49" to additionally include ages exceeding 49 years old. The scaled cumulative distribution function for a maturing out event to arise can be approximated in terms of the age in years T of a virtual subject as:

$$P(nr) = \frac{0.02359}{1 + e^{-0.154 \cdot T + 5.037}} \tag{7}$$

Table 1. Data about the US narcotics users population in 1955 and the related former addicts population at the end of 1959 are used to calculate the cumulative distribution function (CDF) describing the occurrences of maturing out from narcotics addiction, which is subsequently scaled in accordance to new observations about the onset age of abstinence in the base population. AP = Number of active addicts in total addict population; FS = Number of former addicts in sample; AOA = Annual onsets of abstinence per 1000. Columns AP, FS, and AOA are reproduced from [23,24] with permission of the United Nations Office On Drugs and Crime (UNODC).

Age	AP	FS	CDF P(FS|AP)	AOA	CDF P(FS|AP) * AOA
< 20	1743 (3.8%)	13 (0.2%)	0.75%	23	0.17‰
20-30	24343 (53.6%)	2820 (39.0%)	12.33%	21	2.59‰
30-40	14058 (31.0%)	2857 (39.5%)	32.65%	22	7.18‰
> 40	5247 (11.6%)	1544 (21.3%)	62.08%	38	23.59‰

3. Results: plausible scenarios of drug-seeking and maturing out

Two scenarios are presented of a virtual subject denoted as B.T, who had a healthy mental and physical development and became an addict in her early adulthood. In the first set of simulations, the weights ω_i of the processes P(t), S(t), and D(t) can only change their values accordingly to Equation 5, whereas in the second set of simulations also Equation 6 applies. The graphs reported in this section represent the mean of 100 simulations of 600 time steps (~25 days) each and their corresponding standard errors of the mean (SEM) for B.T. at the age of 35.

3.1. Direct Influence of the Recovery Process h(t)

According to Equation (7), a 35 year old virtual subject can be exposed to at most 5 recovery processes within the year. Figure 2 shows the graphs corresponding to the processes defining B.T.'s profile. The recovery process h(t) has a direct effect solely on her cognitive scale. In other words, the weights ω_i of the processes P(t), S(t), and D(t), used to estimate the cognitive state, can only temporarily change their values, during an active process h(t), but are not subject to any permanent alteration. There are 4 recovery processes that occur during these simulations, at t={120, 200, 210, 420}, which correspond to an immediate and strong change in the model's output G(t). For a limited time, B.T. expresses a healthy behavior because of the new value of the weights ω_i. During this period, the neuropsychological processes of B.T. adapt their computational definitions, since they rely on the model's output G(t), but this sudden change doesn't last for a sufficient time for B.T. to acquire a permanent healthy

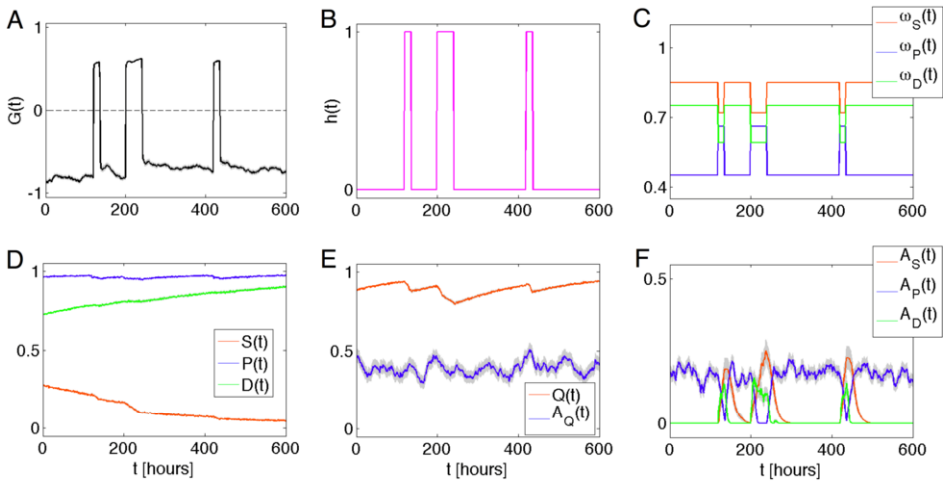

Figure 2. Mean and SEM for 100 simulations of B.T.'s profile at 35 years old, 600 time steps long (25 days) under the direct effect of h(t) at t={120, 200, 210, 420}. (A) The drug-seeking behavior G(t) is mostly negative (maladaptive behavior) and the effect of the recovery process h(t) is strong enough to temporarily change it into positive (healthy behavior). (B) The recovery process h(t) is activated 4 times. (C) The cognitive weights ω_i change value when h(t) is active. (D) The internal processes S(t), P(t), and D(t) change behavior when h(t) is active. (E) The internal process Q(t) and the its related external process AQ(t) are influenced by h(t). (F) The external processes $A_S(t)$, $A_P(t)$, and $A_D(t)$. The processes $A_S(t)$ and $A_D(t)$ occur only when G(t)>0, that in this scenario also corresponds to an active h(t).

behavior, and her maladaptive behavior regains predominance when the active effect of h(t) ceases.

3.2. Direct and Potentially Long-Term Influences of the Recovery Process h(t)

In the previous scenario, the direct effect of the recovery process by itself is not durable enough for the whole system to acquire the necessary dynamic allowing B.T. to start a potentially long-lasting period of abstinence. The non-monotonic property of this model [18] computationally grants B.T. with a possible lifelong rehabilitation, but the values of the constants defining the model that are necessary to achieve such a condition will correspond to a situation beyond biological plausibility (e.g., an event h(t) lasting several months). In the simulations presented in Figure 3, the weights ω_i of the processes P(t), S(t), and D(t), can permanently change their values once the active effect of h(t) ceases. After completing the 3rd recovery process, B.T. expresses a fragile healthy behavior (positive G(t) values), which is further consolidated by the 4th recovery process. This simulation exemplifies a plausible trajectory of an addict that starts an abstinence period within a period of about one month, as a result of 4 long-lasting recovery events, as for example could correspond to instances of non-traditional healing techniques to help overcoming addiction. Instances of these techniques were discussed in the late 1970s (e.g. meditation, faith healing, holistic medicine, etc.) [25], and more recently were at the center of two issues of the journal serving the Association for Medical Education and Research in Substance Abuse (e.g. "attentional control", Mindfulness-Based Cognitive Therapy, etc.) [26,27].

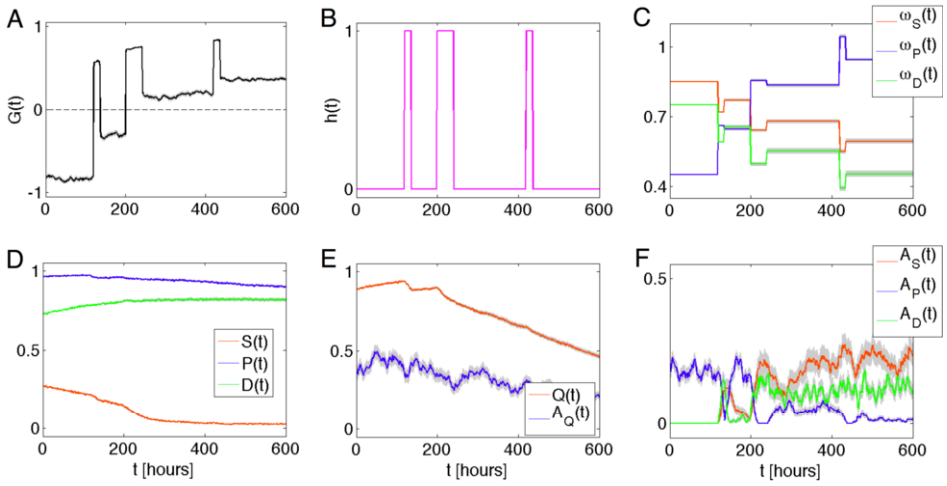

Figure 3. Mean and SEM for 100 simulations of B.T.'s profile at 35 years old, 600 time steps long (25 days) under the effect of h(t) at t={120, 200, 210, 420}. (A) The drug-seeking behavior G(t) starts as negative (maladaptive behavior) and the effect of the recovery process h(t) allows its transition into positive values (healthy behavior). (B) The recovery process h(t) is activated 4 times. (C) The cognitive weights ω_i change value when h(t) is active and are subject to a permanent change when an h(t) event becomes inoperative. (D) The internal processes S(t), P(t), and D(t) change behavior when G(t) transition to positive values. (E) The internal process Q(t) and the its related external process AQ(t) are influenced by h(t) and the positive value of G(t). (F) The external processes $A_S(t)$, $A_P(t)$, and $A_D(t)$.

4. Analysis: a cognitive learning mechanism to enable maturing out

The parameter γ in Equation 2 significantly influences the output of the model and its value can be calculated to mathematically ensure that the virtual subject has no cognitive preference toward a particular behavior [19]. Comparisons of simulations defined by an arbitrary γ with simulations using the unbiased γ, defined in [19], are presented in Figure 4. The value of γ is the same for simulations presented in Figures 2, 3, and the ones in Figure 4 labeled as "*B.T.'s original γ*". All other parameters are equivalent for all simulations. In Figures 4A and D there are no recovery processes h(t); in Figures 4B and E only direct influences of h(t) are considered; and in Figures 4C and F both Equations 5 and 6 apply.

The baseline simulations presented in Figures 4A and D, which don't include any recovery occurrences, show the cognitive predilection of B.T. toward a healthier behavior. B.T.'s original portrayal expresses a less accentuated likelihood of maladaptive behavior than its correspondent cognitively unbiased description. The computed behaviors accounting solely the direct influence of the recovery process h(t), compared in Figures 4B and E, describe a situation in which B.T. successfully abstains from drugs use for a limited time, but the correspondent cognitively unbiased profile constantly preserves a maladaptive behavior. The behavioral and neuropsychological characteristics of the subject make this abstinence difficult to preserve for B.T.'s original profile, and establish a challenging environment for her unbiased profile to reach a healthy behavior. The model's outcomes presented in Figures 4C and F compare the original and unbiased profiles of B.T. when both the direct and the potentially long-term influences of the recovery process h(t) are active. Both cases tend

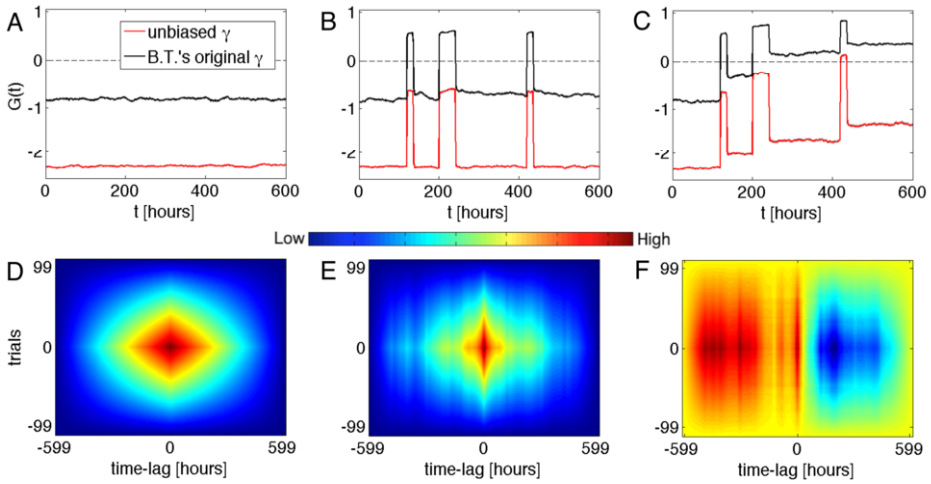

Figure 4. Comparison of simulations using the original and the cognitively unbiased values of γ. On the upper part are represented means and SEMs of the model's output G(t) for 100 simulations of 600 time steps (25 days) each. On the bottom part are plotted the cross-correlations matrices comparing B.T.'s original profiles with its unbiased profile. In (A, D) the recovery process h(t) is not active; in (B, E) h(t) is active but can not induce a permanent change of the cognitive weights; and in (C, F) h(t) is active and can potentially initiate a long-lasting period of abstinence by permanently modifying the cognitive weights values.

towards a healthier behavior, which is reached and maintained by the original profile but is barely touched by the unbiased profile.

The 2-D cross-correlations provide an immediate look at the similarity between B.T.'s original profile and its correspondent cognitively unbiased *alter ego*, considering each simulation rather than the mean of several simulations. Without any recovery processes, the cross-correlation matrix has a smooth circular pattern (Figure 4D), which becomes distorted when only the local effect of the process h(t) is active (Figure 4E), and substantially changes the motif for simulations operating the cognitive learning described above (Figure 4F).

These results suggest that an addict may have the cognitive means to start an abstinence period which, depending on his or her neural substrate and natural surroundings, could persist over time.

5. Concluding Remarks

The fields of psychology and neuroscience provide us with a growing amount of evidence supporting the fundamental role of cognitive components in the course of an addict's life. A pivotal investigation demonstrates that cocaine craving is induced by neural correlates within the frontal cortex, rather than by the dopaminergic circuitry [28], and a recent review paper proposes the hypothesis for which "drug addiction involves a failure of the different subcomponents of the executive systems controlling key cognitive modules that process reward, pain, stress, emotion, habits, and decision-making" [29]. Heeding this belief, and supported by observations of natural recoveries, the framework presented aims to describe a simplistic computational scheme necessary to counteract such cognitive deficiency. Even though the neural correlates of an addict's

limbic system have been modified compared to the brain of a healthy individual, it seems biologically plausible to consider neural changes in the prefrontal cortex to account, at least partially, for a balancing mechanism reconditioning the brain's functions towards a healthy state.

The present investigation proposes formal arguments to support the hypothesis of a cognitive learning mechanism, capable of influencing decision-making processes associated with drug abuse. The emulated abstinence onsets from drug abuse presented above are an initial attempt toward the localization of such a balancing mechanism. To advance this exploration, it could be interesting to emulate similar rehabilitation properties within a more elaborated biologically inspired cognitive architecture, as for example Leabra [30], Clarion [31], and GMU-BICA [32]. To further enhance psychological plausibility of the model presented in this paper, components studied in pathological gambling (stressors, cognitive distortions, ruminations, and distractions) [33] could be incorporated and explored.

The framework presented in this paper supports the view that mindfulness-based cognitive techniques could act as a catalyst for maturing out of addiction.

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