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judgment in a *predictable* manner as they commit optical illusions (Ariely 2008). No such predictability exists, as Redish et al. admit, with regard to addictions. For instance, most agents are vulnerable to the switch from the loss frame to the gain frame in the Asian disease experiment (Tversky & Kahneman 1981). Also, most agents fall victim to overconfidence and the conjunction fallacy (Baron 2008, Ch. 6). But, with addictions, individuals vary widely in the manner they may or may not become addicted.

The same decision framework seems unable to explain both biases and addictions. Redish et al.'s framework might not be the proper tool to explain addictions. Addictions, at first examination, are maladaptive actions in the sense that they reduce O. In contrast, the errors that arise from heuristics might be minor nuisances that the organism tolerates because the heuristics, on average, are efficient. In this case, the heuristics are tolerable "bad habits" given that such habits, in comparison to their absence, have positive net effect on O. Addictions, in contrast, totally diminish the ability to produce O. If so, we need another framework, aside from deliberation and habits, to tackle addictions. This framework may have to attend to the urge to be creative, to have a meaningful life, and how it may lead to addiction when the urge is frustrated.

## Role of affective associations in the planning and habit systems of decision-making related to addiction

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**Abstract:** The model proposed by Redish et al. considers vulnerabilities within decision systems based on expectancy-value assumptions. Further understanding of processes leading to addiction can be gained by considering other inputs to decision-making, particularly affective associations with behaviors. This consideration suggests additional decision-making vulnerabilities that might explain addictive behaviors.

Redish et al. show that a fuller understanding of the processes and outcomes of substance use and abuse can be gained by probing the underlying decision-making and self-regulatory mechanisms involved in initiation and maintenance of use. Their analysis of decision-making systems and vulnerabilities in those systems stems from expectancy-value model tenets in the decision-making and behavioral economics literatures, and from conditioning principles and theories in the learning literature. Although the framework put forward by Redish et al. draws nicely on these literatures to propose an integrative model of substance use, there are important processes involved in decision-making and self-regulation which are not well included in this framework.

In particular, affective processes are not well represented in the framework presented in the target article. We know that affective processes are implicated in a variety of issues around substance use and abuse. For example, affective states are reported as antecedents of smoking behavior and of relapses after quitting (Gilbert et al. 2000; Shiffman et al. 1996). In the context of alcohol use, negative affect resulting from acts of discrimination is associated with drinking by members of minority groups (Simons et al. 2006; Terrell et al. 2006). Finally, as Redish et al. point out, intake of some substances directly leads

to affective states (e.g., euphoria; Koob & Le Moal 2006). Moreover, research from multiple domains has shown that affective processes are an integral part of "normal" decision-making and both impact and are influenced by the expectancy-value processes discussed in Redish et al.'s analysis. Use of expected-utility rules changes with decision tasks that arouse negative emotion (Darke et al. 2006; Greene et al. 2001). Behavioral choices are influenced by anticipation of experiencing regret, guilt, or other emotions as a result of engaging in a behavior (Richard et al. 1996).

An integrative model of the influence on behavioral choice of cognitively based inputs and affective associations with behaviors has been proposed recently (Kiviniemi et al. 2007). The *behavioral affective associations model* focuses on affective associations with a behavior – feelings and emotions associated with a particular behavioral practice. The model proposes that affective associations with a behavior influence actual behavior; more positive affective associations lead to a greater likelihood of engaging in a behavior. Moreover, according to the model, affective associations mediate influences of cognitive beliefs on behavior. Finally, the model argues that affective associations influence behavioral practices both via mediating cognitive beliefs and through a path that is dissociable from and distinct from the mediation of cognitive beliefs (see Kiviniemi & Bevins 2007 for additional discussion of the model). Affective associations have been documented for alcohol and marijuana use (Simons & Carey 1998) and smoking (Trafimow & Sheeran 1998). Recent data from the Kiviniemi lab shows that affective associations both directly predict use behavior and mediate the influence of expected utility beliefs on use for alcohol, cigarette smoking, and marijuana use.

Thus, there are a variety of reasons to argue that affective associations play a central role in both decision-making about and ongoing self-regulation of substance use. What implications might an affective association analysis have for Redish et al.'s framework for studying addiction? First, consider two components of Kiviniemi et al.'s (2007) behavioral affective associations model: (a) affective associations mediate the influence of expected utility beliefs on behavior, and (b) affective associations influence behavior both in conjunction with (through the mediational pathway) and independent of cognitively based expected utility beliefs. The mediational path suggests that affective associations may serve a self-regulatory role by functioning as an indicator of the expected utility of a behavioral choice or, more broadly, by indicating the overall positivity or negativity of one's cognitively based beliefs (e.g., attitudes, social norms). This would allow decision making to proceed in a faster and more efficient manner than directly accessing cognitive beliefs. Such an analysis is consistent with the work of Damasio and colleagues on the somatic marker hypothesis (e.g., Damasio 1994). The tenet that affective associations can exist and can influence behavior independent of one's cognitive beliefs suggests that the content of one's affective associations with a behavior could conflict with one's cognitive beliefs (e.g., one might perceive a number of negative consequences from alcohol use but still have overall positive affective associations with alcohol and its use).

In the context of substance abuse, this suggests the possibility for an additional vulnerability in the decision-making system. To the extent that affective associations are created relatively independently of one's cognitive beliefs about the behavior (as might be the case for euphoria resulting from use or from associating the drug and its use with other positively valued things), the independent affective associations–behavior pathway might push behavior in different directions than the cognitive beliefs path. Such a conflict between decision-making inputs then raises the important question of which input will "win" and influence behavior. Because cognitively based processes often require some effort by the individual, whereas affective processes are more automatic, it may be the case that affective associations will be more likely to guide behavior. This may be especially

likely in the context of substance use where impaired cognitive functioning may be a consequence of use (e.g., Hoffman et al. 2006; Kim et al. 2005).

Supporting this point about vulnerability and affective associations are the published examples of unconditioned stimulus revaluation using Pavlovian conditioning with alcohol (Molina et al. 1996; Revusky et al. 1980; Samson et al. 2004). For instance, in a retrospective revaluation design Molina et al. (1996) found that an aversion to a tactile stimulus conditioned with ethanol was abolished if ethanol was later paired with sucrose. More specifically, rat pups first had an aversion conditioned to the tactile stimulus by pairing it with intragastrically administered ethanol. If rat pups then had the ethanol paired with a sucrose solution via intra-oral cannula, the robust tactile aversion was no longer expressed. The previously acquired tactile aversion was *not* lost if ethanol and sucrose were presented in an unpaired fashion (i.e., no temporal contiguity). Molina et al. concluded that the representation of the ethanol unconditioned stimulus (US) was changed by the appetitive conditioning history with sucrose. As such, expression of the earlier conditioned association (memory) was also changed. A similar possibility has been discussed for nicotine (Bevins, in press; Bevins & Palmatier 2004). Applied to the early discussion, here is an example of a choice behavior (avoid aversive stimulus) that was modified not by direct and contrary learning history in that situation. Rather, choice was presumably altered by changing the positive affective qualities of ethanol. Perhaps effortful cognition was involved in this revaluation. However, such an assumption is not necessary to explain the change in choice behavior and, in fact, seems a priori.

In summary, Redish et al. in this target article outline an integrative model of substance use from a decision-making and self-regulation perspective. This model provides much to think about, as well as indicates interesting and likely important paths for future research. However, we suggest that going beyond considering vulnerabilities within an expectancy-value decision system to consider how other inputs to decision-making might inform our understanding of substance use and abuse, can strengthen the framework proposed by Redish et al. In particular, considering the role of affective associations with behavior suggests that an additional decision-making vulnerability influencing substance use might be conflict between affectively based and cognitively based decision systems. Such conflict can explain why behaviors, including substance use and abuse, may depart from expected-utility model predictions.

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## Negative affects are parts of the addiction syndrome

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**Abstract:** Decision-making is a complex activity for which emotions and affects are essential. Maladaptive choices depend on negative affects. Vulnerabilities to drug or non-drug objects depend on previous psychopathological comorbidities. Premorbid individual characteristics allow us to understand why some individuals – and not others – enter into the addiction cycle. Moreover, plasticity of reward neurocircuitry is, at least in part, responsible for these vulnerabilities leading to compulsive drug use.

The field of addiction research is becoming more and more complex, and many hypotheses have been proposed to account for the transition from recreational use to impulsive consumption and to the last stage of this chronic, relapsing disease: compulsive use and addiction. Redish et al. have reviewed some of these theories and have proposed a classification under eight categories in such a way that some researchers will be surprised to find themselves listed under this or that category. The theories cited each stress different aspects, functional or neuropsychological, and different phase of the process, or consider either physiological mechanisms or structural neurobiology. In the target article, Redish et al. propose one more theory, which is more specific and cognitively oriented: the process of decision (decision-making) is hypothesized to be a “unified framework for addiction” and to be operational to provide a classification of potential vulnerabilities. From a Herculean analysis of the literature, but from this restricted point of view, they have identified ten potential different constitutive vulnerabilities.

Scientific analysts alert us about the breaking down and fragmentation of knowledge, a crisis due in part to the reductionism inherent in modern scientific progress. What is needed is to turn toward a more difficult task: to try and propose holistic theories and to conform to the principle of parsimony. Entities should not be multiplied without necessities, according to the principle of Occam’s razor. Moreover, most authors now agree about the reality of a common clinical syndrome for all the drug and non-drug addictions (Goodman 1990; 2008) and, underlying it, a common set of neuronal systems, whose dysregulations is supposed to be responsible for the set of symptoms (see Koob & Le Moal 1997; 2001; 2006). The question is to know at which stage the process is examined. It seems that Redish et al. are considering the stage of addiction when maladaptive choices are made in spite of their deleterious consequences, whereas vulnerability is generally studied as an intrinsic factor operating at the beginning of the process, accounting for the huge individual differences in the propensity to move toward impulsive drug-taking or gambling (Anthony et al. 1994; Piazza & Le Moal 1996; Piazza et al. 1989; Substance Abuse and Mental Health Services Administration 2003).

At one moment of the process, there is a passage from impulsive control disorder to compulsive disorder – from a stage where increasing tension and arousal occur before the impulsive act, with pleasure, gratification, or relief during the act, followed by regret or guilt, to a stage of recurrent and persistent thoughts (obsessions) that cause marked anxiety and stress followed by repetitive behaviors (compulsions) that are aimed at preventing or reducing distress. The first stage is most closely associated with positive reinforcement (pleasure, gratification); the compulsive stage is most closely associated with negative reinforcement and relief of anxiety and/or stress (Koob & Le Moal 1997). Addiction involves persistent plasticity in the activity of neuronal circuits mediating a decreased function of the brain reward system and a recruitment of anti-reward systems, now well identified, driving aversive states (Koob & Le Moal 2005; 2008). For the purpose of this commentary, the withdrawal/negative affect stage can be defined as the presence of motivational signs of withdrawal in humans, that is, chronic instability, emotional pain, malaise, dysphoria, and loss of motivation for natural rewards. As dependence and withdrawal develop, brain anti-reward systems are recruited (Koob & Le Moal 2008). Another critical problem is chronic relapse in which addicts return to compulsive drug-taking after acute withdrawal; relapse corresponds to the preoccupation/anticipation stage of the addiction cycle just outlined. A unified framework for addiction cannot avoid the fact, well documented from clinical observations, that affects and emotions are important, if not central, neuropsychological dimensions in this human condition. Needless to say, these dimensions interact with the process of decision-making.

All the neurobiological theories of addiction (we refer to the last stage of the process) agree (see Koob & Le Moal 2006)