Toward a Triadic Approach to Craving in Addictive Disorders: The Metacognitive Hub Model

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Craving is a key factor in substance-related and behavioral addictions, as illustrated by its recent inclusion as a diagnosis criterion in the most recent, fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders*.[1] It globally refers to the subjective motivational state facilitating the emergence of addiction-related obsessive thoughts and compulsive approach behaviors, despite repeated efforts to stop the addictive behavior.[2–5] Reduced craving may produce a cascade of downstream benefits, ultimately preventing relapse,[6,7] but its underlying mechanisms remain uncertain.[8]

We present a conceptual, experimental, and clinical approach to craving that aligns it with the triadic model[9] — a recently developed, prominent model of addictive disorders. Our central assumption is that craving should not be considered as a unitary process but rather as the emerging consequence of the interplay between three subcomponents, namely (1) *cognitive craving* (or "obsessive craving")[10] related to cognitive abilities (e.g., executive functions), (2) *automatic craving*,[10] linked to cue reactivity and implicit processes (e.g., attentional biases), and (3) *physiological craving* (or "relief craving"),[10] corresponding to bodily perceptions and related to withdrawal symptoms. We relate these three components to the systems proposed by the triadic model and suggest that metacognitive abilities may constitute a mechanism bridging the three subcomponents. This approach offers an innovative and integrative conceptual framework to explore the interactions between craving and other key processes involved in addictive disorders.

RECENT CONCEPTUALIZATIONS OF CRAVING

In a comprehensive review, Skinner and Aubin[11] identified 18 craving models: four "conditioning" models (considering craving as an automatic reaction to a stimulus), seven "psychobiological" models (considering craving as mostly related to biological factors), three "motivational" models (considering craving as a consequence of larger decision-making impairments), and four "cognitive" models (considering craving as related to biased information-processing systems). While identifying the major dimensions of craving and highlighting the variety of its related symptoms,[8] these earlier models have all focused on specific factors without offering an integrative theoretical approach to craving. Moreover, these models have mostly considered craving as a phenomenon in seclusion, whereas research has shown that craving actually interacts with a large range of processes assumedly involved in addictive disorders (e.g., implicit cognitions, attentional bias, approach/avoidance tendencies).[12] Skinner and Aubin[11] recommended the development of an integrated model to overcome the limitations of these approaches to craving. To do so, they suggested that the model incorporate the multifaceted nature of the processes involved in craving (e.g., conditioning, psychobiological, motivational, cognitive) and address craving's interactions with other key factors of addictive disorders.

Dovetailing with this proposal, a recent review by Sayette[13] identified four major concerns to be urgently clarified regarding craving:

–*Craving's underlying psychological processes*. Some models focus on impulsion and thoughts (i.e., automatic craving), whereas others focus on behavioral features (compulsion and actions; i.e., behavioral craving).

–*Craving's temporal stability*. Several models consider craving as a dynamic state (relying on the presence of specific stimulations and thus presenting large intra-individual variability), whereas others describe it as a stable individual characteristic.

–*Awareness of craving*. Strong inconsistencies persist across models regarding craving's explicit or implicit nature.

–*Craving's links with desire*. Some models propose a continuum with craving merely considered an intensified

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desire, whereas others propose a qualitative discontinuity between healthy desire and pathological craving.

We argue that these critical questions can be addressed by reconsidering craving in the light of a recent and promising neurocognitive conceptualization of addictive disorders—that is, the triadic model.

THE TRIADIC MODEL OF ADDICTIONS

In their triadic neurocognitive approach to addiction, Noël and colleagues[9] enriched the classical dual-process models by adding a third component focusing on interoception and insular pathways. The most prominent models of addictive disorders have long been dual-process models,[14] positing that decision making related to substance use is determined by the balance between two functionally and neuroanatomically distinct but interacting systems: (1) the *automatic/impulsive system*, an appetitive system underpinned by limbic structures and involved in the impulsive processing of stimuli, triggering automatic responses based on associative learning, and (2) the *controlled/reflective system*, an inhibitory system underpinned by frontal regions, involved in the cognitive processing of stimuli, and relying on memory and executive functions to initiate controlled-deliberate response. According to these models, addictive disorders emerge when these two systems are out of balance, such that the impulsive system becomes sensitized (e.g., by repeated exposure associating substance intake and reward) while the reflective system is compromised (e.g., following the frontal dysfunctions related to the neurotoxic effects of a substance), leading to dysregulated consumption. Although experimentally robust and widely accepted, dual-process models have neglected another key system involved in addictive disorders—namely, interoception.[15]

A triadic model of addiction has thus emerged, which posits that a third neural system, relying on insular pathways, moderates the dynamics between automatic/impulsive and controlled/reflective systems.[9,16] Under specific circumstances (e.g., homeostatic imbalance, reward deprivation, stress, insufficient sleep), the insular cortex can translate interoceptive signals into what may become subjectively experienced as a feeling of "urge" or "craving." For instance, the insular cortex activity might drive motivation toward a substance by simultaneously exacerbating the automatic/impulsive system's activity and disrupting prefrontal cortex inhibitory functions.[17,18] These interoceptive inputs thus have the ability to "hijack" the cognitive resources necessary for exerting inhibitory control when confronted with rewarding or stressful situations. For instance, people with addictive states may misinterpret body signals (e.g., increased heart rate) related to everyday life situations (e.g., a job interview) as reflecting a physiological urge toward the substance, which may, in turn, reduce inhibitory abilities and activate automatic approach behaviors toward the substance.

The triadic model thus incorporates bodily sensations in addiction models, integrating automatic, controlled, and interoceptive systems. Neuroscience research has recently yielded empirical support for this model. First, the insula has been identified as an integrative interoceptive site connecting autonomic, affective, and cognitive processing.[19,20] For instance, the anterior insula has bidirectional connections to the amygdala, ventral striatum, and orbitofrontal cortex, and the homeostatic imbalance emerging in several psychological states (e.g., stress, sleep deprivation) is related to interoceptive signals received by the insula, which, in turn, influences other neural systems.[17,18,21,22]

Second, a growing body of research indicates that the insula is involved in detecting salient or novel stimuli in the environment, and in initiating the resulting switch between routine brain functioning (based on the default mode network) and activation of the frontal executive network, needed to efficiently process and respond to these incoming stimulations.[19,23–25] The insular system is thus connected with frontal areas and plays a role in the interactions between automatic and controlled processes, with such interactions being strongly involved in addictive disorders.

Third, evidence supporting the role of the insula in reward seeking has come from human brain lesion studies, notably showing that long-term smokers enduring a brain stroke were more likely to abruptly and easily quit smoking, without relapse or persistent craving, when the stroke encompassed insular regions.[26]

Despite the presence of experimental support, the triadic model of addiction needs further elaboration on several levels. Among the dimensions that are still insufficiently conceptualized by the triadic model, craving is prominent. Noël and colleagues[9] considered craving to be exclusively initiated by the interoceptive system, which seems reductive insofar as craving is considered a multidimensional phenomenon. Capitalizing on the triadic perspective, we propose an integrative model of craving that not only encompasses the three subsystems but also sees metacognitive abilities as a hub bridging the interactions among the subsystems.

THE METACOGNITIVE HUB MODEL OF CRAVING

Our main proposal is twofold. First, we suggest that each system of the triadic model corresponds to a specific subcomponent of the craving experience:

–*Cognitive craving*, relying on the *reflective system*, is related to a reduced efficiency of high-level cognitive abilities (i.e., memory and executive functions) and can be approached through executive control[27] or inhibition[16] tasks.

–*Automatic craving*, relying on the *automatic/affective system*, is related to cue reactivity and attentional bias toward addiction-related cues and can be evaluated by measuring approach[28,29] or attentional[30] biases toward addiction-related stimuli. Note that for clarity and to align with the triadic model, we consider automatic craving as encompassing three subcomponents reflecting behavior that is appetitive/reward-driven (i.e., hedonic substance-seeking aiming at enhancing positive affects), aversive/avoidant (i.e., substance seeking focused on the reduction of negative affects), or

compulsive/habit-driven (i.e. substance-seeking related to automatized stimulus-response associations, independent of the actual rewarding/affective value of the substance). These three subcomponents might be differentially involved in automatic craving across distinct clinical populations with addictive disorders (and across the successive stages of these disorders), and the compulsive/habit-driven *versus* appetitive/reward-driven components could even constitute distinct systems. Further studies are needed to clarify this matter.

–*Physiological craving*, relying on the *interoceptive system*, is related to the interpretation of body signals triggered by homeostatic imbalances associated with specific psychological states (e.g., stress, sleep deprivation) or by the perception of substance-related cues (which can modulate insular activity).[31,32] This type of craving can be evaluated using interoception tasks or psychophysiological measures.[33]

Second, we suggest that metacognitive abilities play a key role in the distinction between implicit (i.e., unconscious and related to automatic approach behaviors) and explicit (i.e., accessible to awareness, with an intensity depending on the individual's metacognitive abilities) craving.[34] Both human[35,36] and animal[34] neuroimaging research supports this type of implicit-explicit distinction regarding the control of drug-seeking impulses under laboratory conditions. This research notably showed that patients with alcohol use disorders and with poor metacognitive abilities exhibited a reduced control of socially appropriate emotional, cognitive, and behavioral responses, as well as suboptimal craving appraisal and management. Given these findings, we hypothesize that metacognitive abilities distinctively determine, for each craving component of the triadic model, whether the individual is able to identify and apprehend the presence and intensity of that component.

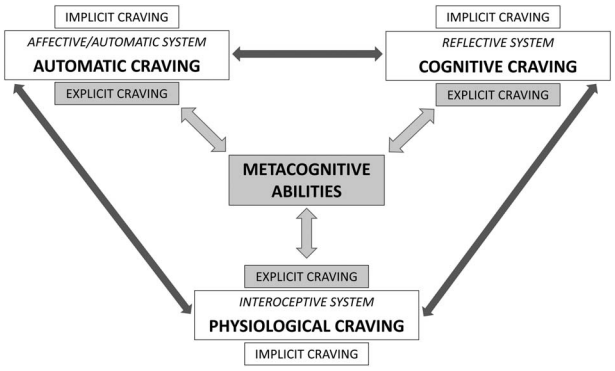

Figure 1. Metacognitive hub model of craving. The original triadic model encompasses three systems (affective/automatic, reflective, and interoceptive), represented by the large white rectangles, which are in constant interaction (as represented by the dark gray arrows). Each system generates one type of craving (automatic, cognitive, and physiological cravings, respectively), each type being characterized by implicit (small white rectangles) and explicit (small gray rectangles) subcomponents. The explicit-craving subcomponent is strongly influenced by metacognitive abilities (as indexed by the light gray arrows), playing the role of a hub bridging the systems.

Metacognition, defined as the understanding of one's own cognitive functioning,[37] is used to monitor and control one's thoughts and behaviors.[38] Accordingly, we suggest adding a metacognitive level to the triadic model (see Figure 1). This metacognitive component would act as a hub for (1) interpreting and integrating signals from each subsystem, (2) managing the interactions between craving subtypes, and (3) allowing the explicit craving experience to emerge. Depending on their metacognitive abilities, patients with addictive disorders will differentially access the cognitive, emotional, and interoceptive subcomponents of craving, leading to the subjective craving experience, with various possible patterns (e.g., preserved emotional but impaired interoceptive metacognition, leading to an affect-focused explicit craving experience, ignoring bodily sensations). Of note, the relations between each system and metacognitive abilities have to be understood as bidirectional: the activation of each system can modulate metacognitive abilities (e.g., overactivation of the automatic system can lead to reduced metacognitive awareness), which can, in turn, modify the functioning of each system either directly (e.g., via the modification of automatic/affective system activation through metacognitive modulation when confronted with substance-related cues) or indirectly (e.g., via the modification of automatic/affective system activation through the metacognitive influence on reflective [increase of attentional/cognitive resources] or interoceptive [reinterpretation of body signals] systems).

IMPLICATIONS

Theoretical and Translational Implications

Our new model—which disentangles craving into three distinct subcomponents (thanks to the triadic perspective) and also features the metacognitive abilities as a core interface between subsystems activation and explicit craving experience—clarifies the above-mentioned current debates in the following ways:[13]

–*Craving's underlying psychological processes*. Our model offers a clear theoretical framework to explore each craving subcomponent—namely, cognitive (e.g., inhibition, monitoring, planning), automatic (e.g., attentional biases, affective response), and interoceptive (e.g., body sensations, physiological activation)—as well as their interactions and their reinterpretation by metacognitive abilities, leading to the explicit craving experience reported by individuals with addictive disorders. This perspective will allow us to move beyond the classical dissociation between obsessive and compulsive craving subcomponents, by considering that this obsession-compulsion balance should be revisited by a triadic craving system, overseen by metacognitive abilities.

–*Craving's temporal stability*. Our model identifies the crucial variables explaining why craving experience evolves over time, in the short and mid term, and thus offers perspectives to determine the role of each subcomponent in intra-individual and inter-individual craving variations, as repeatedly observed in clinical settings. Given the limited

experimental data available regarding the differential lability of craving subcomponents, future research should attempt to identify the key factors modulating the stability of craving subcomponents during the detoxification process (e.g., mood-dependent variations of affective reaction to addiction-related cues) or during abstinence (e.g., improvement of cognitive functions, interoceptive accuracy, or metacognitive abilities).

–*Awareness of craving*. While earlier models did not provide a precise mechanism explaining the frequent discrepancy between implicit and explicit craving, we propose that metacognitive abilities—which are crucial for perceiving implicit signals coming from the three craving subcomponents—are the key factor in explaining such a dissociation and in integrating them into a conscious representation, leading to explicit craving. Strong craving activations related to each subsystem can, for example, end up in a reduced reported explicit craving among patients with low metacognitive abilities, leading to serious inconsistencies between implicit and explicit craving measures.

–*Craving's links with desire*. While desire corresponds to a specific overactivation of the automatic system, craving emerges from a combination of automatic, cognitive, and interoceptive subcomponents. Craving is thus a multidimensional phenomenon based not only on affective desire resulting from the limbic system but also on its modulation by frontal and insular networks. In other words, we suggest that *desire* corresponds to the automatic subcomponent of craving and that it can be modulated or controlled by the two other craving forms (cognitive and physiological) and also by metacognitive abilities—leading, in the end, to the integrated craving experience. For example, a patient with an addictive disorder who is experiencing low automatic craving (and thus healthy desire initiated by affective activation) may still exhibit a pathological craving experience initiated by overactivated interoceptive or cognitive craving subcomponents.

Clinical Implications

While the present model requires further experimental support, it might nevertheless serve as the foundation for some innovative clinical research agendas regarding craving management. Indeed, interventions currently available in most clinical settings tend to focus on each subcomponent separately,[39] thus showing low to moderate efficiency and limited generalization to everyday life. Our model emphasizes the articulation and interdependence of the three subcomponents, and therefore the significance of developing programs that will (1) offer a valid evaluation of patients' abilities on each subcomponent as well as of their metacognitive abilities and; (2) highlight, on the basis of this evaluation, the need for integrated therapeutic programs that aim at targeting the impaired subcomponents. In this way, we suggest that an initial evaluation is needed for each system, as proposed above, as well as a metacognitive awareness task,[40] in order to have a precise evaluation of each component's integrity.

A clinical assessment of craving should be conducted to explore the explicit craving experience—for example, by using a visual analogic craving scale, an obsessive-compulsive scale,[41] and a metacognitive assessment.[42] This evaluation—all-inclusive, short, and implementable in clinical settings—may help in drawing a precise picture of the craving extent and lead to personalized, evidence-based therapeutic programs potentially including (1) inhibition training[43] for the *reflective system*, (2) attentional-bias modification[44] for the *automatic/affective system*, (3) interoceptive training through biofeedback[45] for the *interoceptive system*, and (4) metacognitive therapy (in line with what has been proposed for depression and anxiety)[46,47] for metacognitive abilities.

CONCLUSIONS AND RESEARCH PERSPECTIVES

We have proposed a theoretically grounded model of craving that includes three components (automatic, cognitive, and physiological) orbiting around a metacognitive hub. This proposal might initiate research avenues allowing a better understanding of the craving experience, of the dissociation between explicit and implicit craving, of the interactions between craving subcomponents, and of the role of these subcomponents in the etiology and maintenance of addictive disorders. More specifically, the present theoretical framework proposes the three following avenues for research, with each testing a key assumption of our model.

The Role Played by Metacognitive and Interoceptive Abilities in Substance Use Disorders

Although the primary assumption of our model is that interoceptive and metacognitive systems are actively involved in substance use disorders, the currently available evidence regarding the role of these abilities is much more limited than for automatic and reflective systems. A first experimental test of our model would thus be to determine whether interoceptive and metacognitive abilities also constitute central factors in addictive disorders—which is a prerequisite for switching from the classical dual-process view (focusing on well-documented reflective and automatic systems) to the triadic model (adding the interoceptive system) and then to our metacognitive hub model (further adding metacognitive abilities). The related prediction is that if our model is valid, interoceptive and metacognitive deficits should be demonstrably present in substance use disorders. Moreover, we can hypothesize that (1) the intensity of these deficits should be proportional to the seriousness of the substance use disorder, (2) the extent of these deficits should be correlated with the risk of relapse, and (3) effective interventions to address these deficits should be associated with improved clinical outcomes (e.g., reduced relapse rates after detoxification).

The Dissociation Between Explicit and Implicit Craving, and the Role of the Metacognitive Hub

A second central assumption of our model is that implicit and explicit craving can be separated out by exploring the objective automatic, reflective, and physiological activations (indexing implicit craving, as measured through experimental/neuroscience tools) and the subjective craving experience that results from these activations (indexing explicit craving, as measured through self-report measures). A second experimental test of our model would thus be to compare implicit and explicit craving measures, as well as their similarities and differences, among patients with substance use disorders. Moreover, we have proposed that metacognitive abilities are the key factor in explaining the disconnection between implicit and explicit craving, as these abilities are crucial in perceiving and integrating the implicit signals coming from the three craving subcomponents (i.e., implicit craving), eventually resulting in the explicit craving experience. This proposal can also be experimentally tested by exploring how metacognitive abilities can modulate the difference between explicit and implicit craving. The related prediction is that if our model is valid, experimental and self-report measures should be able to distinguish explicit versus implicit craving. In addition, the differences between these two types of craving should vary according to the metacognitive abilities of each patient.

The Interactions Between the Craving Subcomponents Related to Each System

A third central assumption of our model is that the automatic, cognitive, and physiological subcomponents of craving are distinct but interconnected. In other words, we postulate that these three types of craving can independently vary in intensity across patients with substance use disorders (as well as within each patient, according to internal state or external stimulations), leading to a wide range of craving experiences (e.g., low physiological craving combined with intense automatic and moderate cognitive ones). A third experimental test of our model would thus be to propose a simultaneous measure of the three craving types (in their implicit and explicit dimensions) to determine whether they can indeed be considered as separate and whether all their possible combinations can be found among patients with substance use disorders. Moreover, our model postulates that each subcomponent can influence the other two—which can, again, be tested by experimentally manipulating one craving type (e.g., increasing automatic craving through cue exposure) and checking whether such manipulation does modify the intensity of the others (e.g., can increase cognitive and physiological craving). The related prediction is that if our model is valid, the three craving types should be influencing each other but should be distinguishable.

Finally, a direct experimental implication of our model is that, rather than considering craving as a unitary concept (as it is still currently done in most empirical work), future studies should attempt to evaluate the three craving subcomponents separately or should at least clearly state which craving subcomponent their experimental design addresses (and also justify the specificity of the tools chosen to measure it). Nevertheless, this model obviously needs empirical support, especially regarding the interactions among the three proposed subsystems, as well as their temporal evolution. Recent computational tools from network analysis may help to identify how distinct subsystems bridge together.[48,49] One also needs to consider that the triadic model, which is at the center of the extended model presented here, was developed within the field of tobacco dependence. This extension to substance-use and behavioral addictions should thus be considered, in effect, an effort to formulate a transdiagnostic approach to craving in addictive disorders.

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