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Perspective

Conscious and Unconscious Mechanisms in the Emotional Brain Underlying Difficulties to Stop Overeating, Drinking and/or Using Drugs: Challenges and New Treatment Perspectives for Obesity and Addiction

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Abstract

Recent data from neuropsychology, neuroimaging, and experimental research suggest that obesity and addiction share common neurocognitive endophenotypes. The objective of this article was to review the main common neurobiological basis of obesity and addiction, investigating how evidence-based translational neuroscience would explain conscious and unconscious mechanisms in emotional brain underlying difficulties to stop overeating, drinking and/or using drugs. Based on this, we aimed to delineate new possible strategies for the treatment of these very similar conditions. In general, studies have shown that obesity and addiction are associated with brain dopaminergic (DA) circuitry dysfunction and deficits in executive functioning/decision making. Both implicit and explicit memory mechanisms have been implicated in addiction and obesity, suggesting a prominent role of unconscious (implicit) mechanisms in the perpetuation of both chronic pathological conditions. Results on traditional pharmacotherapy methods have been limited, so here we argue that an integrative approach combining psychotherapy and neurorehabilitation could be promising for the treatment of these complex conditions.

INTRODUCTION

Recent data from clinical psychiatry, neuropsychology, neuroimaging, and experimental research suggest that obesity and addiction share common symptoms and similar neurocognitive endophenotypes [1-4]. Apparently, the majority of the population has already accepted the idea that certain types of food can be addictive, but at the same time they do not believe that typical medical treatments such as surgery or medication could be effective, considering a long-term perspective [5]. Scientific evidences have shown that diet, regular exercises and psychotherapy are considered the most important types of treatment for obesity, maybe because they emphasize the persons' 'responsibility' for their behaviors [5]. At the same time, clinical trials have been conducted with obese and addicted patients, but results are still limited. Considering that addiction and obesity involve unconscious or more specifically an implicit learned pattern of repetitive (both impulsive and compulsive) behavior, here we argue that the available traditional treatments have neglected these important components. In sum, there is a lack of integrative studies considering both implicit (conscious) and explicit (unconscious) aspects of addictive diseases, which makes difficult for the clinician to comprehend and to make effective decisions along the course of the treatment. The objective of this article is to review the common neurobiological

basis of obesity and addiction, including how evidence-based translational neuroscience would explain conscious and unconscious mechanisms in the "emotional brain" underlying difficulties to stop overeating, drinking and/or using drugs, in order to delineate new possible strategies for the treatment of these very similar conditions, helping to guide future research.

PREVALENCE, COURSE, AND PROGNOSIS

Both addiction and obesity are considered high prevalent clinical conditions, involve high costs to society and represent increasing public health concerns [6]. They are basically characterized by relevant overlapping symptoms such as loss of behavioral control and a compulsive pattern of use [2,4]. The clinical similarities of both disorders are described in the Table 1 [3].

Considering these commonalities, for the first time the psychiatric nosology of the DSM-V has included binge eating as a psychiatric eating disorder [6]. Interestingly, rates of success for addicted and obese patients are quite similar. Quantitatively, results show that about 40-70% of alcoholic, 50-80% of opioid, 50-60% of cocaine, 20-40% of tobacco and only 15% of obese patients have success in treatment [7,8] suggesting that addiction and obesity are both chronic disorders with similar rates of success (Table 2). As with the treatment of addiction and other chronic conditions [8], treatment for obese people depends prominently on behavioral change. In general, a poor prognosis for those conditions may be associated with low socioeconomic status, comorbid psychiatric conditions, lack of family support, end executive dysfunction [8,9]. For obese patients, the lower percentage of success may be attributed by the fact that an over-eating patient could not exclude food from his life because it is necessary for survival, while an addicted patient is stimulated to strategically exclude entirely the substance from his life.

Especially here, among individuals genetically predisposed to obesity and addiction, environmental influences such as emotional stress and parenting practices can be relevant factors for their expression in a given subject [8].

COMMON NEUROBIOLOGICAL BASIS OF ADDICTION AND OBESITY: CONSCIOUS AND UNCONSCIOUS MECHANISMS

Studies have shown that a pre-existent executive dysfunction combined with higher impulsivity may represent predisposing factors guided by a combined construction of genetic influences and early environmental factors (e.g., emotional stress, parenting life-style), which may negatively impact the normal development of the prefrontal cortex, leading to poor decision-making, which heightens the risk for acquiring addictive disorders such as obesity and addiction [1,2,4,10-14].

We have evaluated 101 adults, including 69 cocaine-dependent subjects and 32 controls, aiming at investigate if cocaine dependent individuals with Attention-Defcit/ Hiperactivity Disorders (CDI + ADHD) would have a distinct pattern of executive functioning when compared with CDI without ADHD (CDI), and our results confirm that a pre-existing dysexecutive symptomatology associated with ADHD have a significant negative impact on executive dysfunction in Addicted individuals, suggesting that they may present an underlying cognitive endophenotype that would substantially increase the risk for acquiring addictive disorders [1].

Table 1: Similarities among clinical phenotypes in addiction and obesity.

Addiction	Obesity
Tolerance: increasing amounts of substance use (i.e., tobacco, alcohol, or drugs) to reach intoxication	Tolerance: increasing amounts of food to maintain satiety
Withdrawal symptoms upon drug discontinuation	Distress and dysphoria during dieting
Larger amounts of drug taken than were intended	Larger amounts of food eaten than were intended
Persistent desire and unsuccessful attempts to cut drug use	Persistent desire for food and unsuccessful attempts to curtail the amount of food eaten
Great deal of time spent on getting the drug, using the substance, or recovering from it	Great deal of time is spent eating
Important social, occupational, or recreational activities are given up or reduced because of substance abuse	Activities are given up from fear of rejection because of obesity
Substance use is continued despite knowledge of having a persistent or recurrent physical or psychological problem caused or exacerbated by the drug	Overeating is maintained despite knowledge of adverse physical and psychological consequences caused by excessive food consumption

Source: adapted from Volkow & O´Brien, 2007.

Table 2: Percentage of success rates in patients with similar addictive disorders.

Chronic Medical Condition	*Percentages of success rate**
Alcohol	50 (40-70)%
Opioids	60 (50-80)%
Cocaine	55 (50-60)%
Nicotine	30 (20-40)%
Food (obesity)	15 (0-49)%

Source: adapted from O´Brien & Mc Lellan, 1996 and Ayyad & Andersen, 2000. **Note:** follow-up 6 months. Data are mean (range).

The ADHD is commonly characterized by inattention, hyperactivity, and impulsivity symptoms (APA, 2000) [15] and its cognitive endophenotype includes deficits in response inhibition and impulsivity [2,16], which in turns are considered possible vulnerability markers for substance dependence and obesity [1,2]. Recent studies suggest that obese and addicted patients suffer from significant alterations in dopaminergic pathways that regulate neuronal systems associated with reward sensation, motivation, conditioning, self-control, and stress reactivity [4]. Obesity and addiction has been associated with reduced availability of Dopamine 2 receptors (D2R) in striatum [4,17-19]. More recently, other experiments have cleared how evidence-based translational neuroscience would explain conscious and unconscious mechanisms in the "emotional brain" underlying difficulties to stop overeating, drinking and/or using drugs. Neuroscientific experiments (in both humans and rodents) confirm D2R alterations [20] and also indicate other common brain abnormalities in the prefrontal cortex and hippocampus, involved in mechanisms of memory formation [19]. For more details of the commonalities among the neurobiological basis of obesity and addiction, please see Johnstone et al., 2013 and Volkow et al., 2013 [1,6].

In sum, for both addiction and obesity, now we have information about altered mechanisms in hippocampus which are associated with problems in acquisition of explicit (conscious) new information about facts (semantic memory) and events (episodic memory), as well as reduced dopamine signaling in the striatum, which is a core brain region implicated in reward, motivation, conditioning, and habit formation [19,20], which are associated with involuntary (unconscious) components of memory habituation (implicit memory, see Figure 1).

At a behavioral level, after continued repetition of voluntary drug or food-taking, there are neuroplastic neuronal changes in the brain, especially in dopaminergic pathways in the PFC, striatum, insula and amygdala, that makes the subject loses the voluntary ability to control its use, in a continuum process, when progressively the "drug misuser" or "binge-eater" becomes "addicted" and compulsive [1,8]. In this period, the voluntary aspect of a controlled behavior is overwhelmed by an implicit progressively and unconsciously acquired knowledge that reveals as a more powerful force to maintain the continued repetitive maladaptive behavior, such as over eating and drug using. This is the basis to explain the recurrence of harmful and self-destructive behaviors such as over-eating and substance abuse, considering conscious and unconscious aspects.

INTEGRATING THE SCIENCE OF ADDICTION AND OBESITY: IT NECESSARY TO USE COMMON STRATEGIES TO DEAL WITH SIMILAR DISORDERS?

Some years ago, we did not know relevant insights about the neural mechanisms underlying this change from drug-taking and food consumption to addiction and obesity, respectively, but now we have made significant advances for the comprehension of this process. At that time, we were particularly looking for pharmacological treatments to reverse this process [8]. However, considering recent advances in translational neuroscience, it would be discouraged to suppose that medication, surgeries and/or vaccines would be effective to "solve" the problem of addiction. From this point of view, they may be effective for "suture of bleeding" in an acute condition (for example, during a strong craving, a health crisis related to overeating or excessive drug intoxication), but not for sustained behavioral changes, restoring and brain health in a long term perspective. Particularly the idea here is that these strategies (i.e., medication, vaccines,

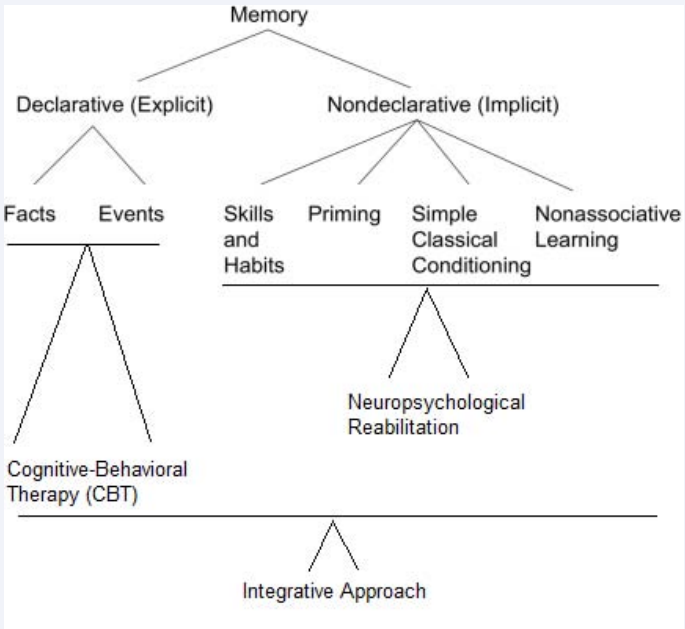

Figure 1 Implicit and explicit memory systems involved in addiction and obesity: evidences for a necessary new and integrative approach to treat these similar psychiatric conditions.
Source: adapted from Squire, 2004.

etc) are based on the same mechanism which is responsible for maintaining addictive behaviors: decision making based on limited and immediate gains, in this case by clinicians and industry. If addiction and obesity are complex behaviors based on explicit (conscious) and implicit (unconscious) mechanisms, highly influenced by neurobiological, environmental and evolutive characteristics, it is reasonable to suggest that just to stop using one type of substance or food would not help these patients to deal with their maladaptive pattern of behavior, which corresponds to a neurocognitive endophenotype that reflects an altered reward mechanism as a whole [4].

From this point of view, treatments for addictive problems should focus on changing patterns of behavior and habits to have real and stable long term results. For example, the available strategies to treat patients with obesity rarely induce long-term behavioral changes because they do not address the neuropsychological functions [6]. A plausible explanation to this is that treatments purely based on immediate strategies such as medication and bariatric surgery are associated with a tendency for most physicians to perceive obesity as being an acute condition, similar to what happens with addiction [8]. The main idea here is to consider them as similar brain disorders involving specific neurocognitive endophenotypes that may represent a higher vulnerability to different kinds of addictive behaviors, such as to take drugs, to drink alcohol, to eat too much, to gamble, to work more than the necessary, to being connected to a social network for all the day, to play videogame all night long, or to have excessive sex, among others. In theory, every behavior that may elicit pleasurable sensations with an over activation of the brain reward system and dopamine (DP) may be addictive. So here we propose an opened and necessary debate about how to improve our available treatments focused on the "underlying disorder" that leads the affected subjects to a tendency to relapse to an active and self-destructive behavior.

For example, CBT helps patients to deal with conscious "advantages" and "disadvantages" of using drugs, but addicted patients tend to persist drug use (or excessive food consumption) despite knowing all adverse consequences (APA, 2000). This important component of drug persisting behavior is associated with implicit mechanisms of substance use, such as impulsivity, poor emotional control, altered stress reactivity, and craving. In this perspective, it is crucial to develop new strategies that would presumably help patients not only to deal with conscious aspects of their addiction (related to substances or to high-caloric food), but also (and mainly) help them to construct new patterns of implicit (unconscious) behavior different from that associated with substance use. For this purpose, an integrative approach combining cognitive behavioral therapy (CBT) with neuroreabilitation strategies could be promising to tackle addictive disorders (Figure 1).

We have recently reviewed several studies on neurorehabilitation for executive functioning and results suggest that executive impairments may be remediated and/or compensated at a certain degree by neuropsychological rehabilitation, but the impact of rehabilitation remains poorly understood [21]. Despite the great advances in the understanding of addiction and obesity as similar psychiatric disorders, sharing several common neural mechanisms, now we must face the major challenge in this area based on how to help patients to improve EF and decision-making, aiming at reduce risk-behaviors and substantially improve their quality of life. There is an urgent need for the development of new and/or combined techniques for a broader impact on executive functioning and on the quality of life of these patients. For example, we have recently developed a new model of treatment for addiction integrating Motivational Interviewing (MI) with chess game, first because motivation plays a crucial role in EF [22] and second because to play chess is thought to be associated with both PFC activation [23] and executive functioning enhancement [24]. Considering the major limitation of neuroreabilitation studies for executive functions that have persistently failed to translate cognitive benefits into real life improvements (ecological factors), here we used Motivational Interviewing to promote the generalization of the explicit and implicit acquired learning (during the game) in the context of real-life problems and perspectives, through stimulation of future planning and decision-making. We called this new intervention as "Motivational Chess" (MC), and details can be found elsewhere [25]. Our results demonstrate that MC was associated with a more significant improvement in working memory during the first month of abstinence in addicts, indicating that specific and integrated tailored interventions focusing on complex executive processes may accelerate the progress of cognitive therapy along this initial period of abstinence [25,26].

CONCLUSION

Despite a large body of evidence indicating strong similarities between addiction and obesity, few studies have developed new strategies based on this accumulated knowledge. New studies integrating neuropsychological rehabilitation and CBT are necessary, to address not only explicit memories (conscious) but also implicit contents (unconscious) underlying the maintenance of drug-seeking and over-eating behavior.

DECLARATION OF INTEREST

The author reports no conflict of interest. The author alone is responsible for the content and writing of the paper.

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