Pacini Editore & AU CNS

Letter to the Editor

Heroin Addict Relat Clin Probl 2013; 15(4): 45-48

HEROIN ADDICTION & RELATED CLINICAL PROBLEMS
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A controversial side of addiction: new insight in eating behavior

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TO THE EDITOR: Food addiction is an innovative and controversial theory that aim to explain the increasing prevalence of weight in excess.

Up to a few years ago, the prevalent idea on appetite was that fasting and hunger signals would activate cerebral circuits addressed to food seeking and intake. But current evidence shows that overweight and obese patients overeat despite their hyper-satisfied metabolic needs.

Recently, several Authors have suggested that homeostasis-independent overeating may result from habitual exposure of the cerebral rewarding systems to palatable and hyper-caloric foods, and described remarkable overlaps between drug and food abuse phenomenology [4, 11].

Obesity may in future be partially re-thought as a primary mental condition, involving variable degrees of appetite control disruption and overlapping with substance abuse habits and impulse-control disorders. Several authors unsuccessfully proposed a place for food addiction in the DSM-V, conceiving appropriate psychometric scales for assessment of food attachment such as the Yale Food Addiction Scale [5].

1. Experimental evidences

Typical addiction behavior (craving, withdrawal symptoms, difficulty to suspend administration) can be observed in experimental animals with discontinued access to sugar and/or fat [1].

Dopaminergic neurons in the ventral-tegmental area (VTA) projecting to nucleus accumbens (NAcc)

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physiologically respond to sex, social activities and food-related stimuli. Glucose and insulin peaks, indeed, lead to increase dopamine availability in the NAcc, producing fulfillment and pleasure. PET studies in both overweight and drug-addicted subjects described similar limbic and cortical alterations of dopaminergic transmission: higher substance reinforcing effects, higher reward sensitivity and expectation, higher memory and learning conditioning and higher behavioral disinhibition compared with non-addicted subjects [11].

Long-term stimulation causes dopamine receptor adaptation and a reduced dopaminergic signaling. These events are crucial for developing drug addiction. Recent fRMI studies found the same decreased dopamine responsivity in overweight subjects, in proportion to their BMI [10].

Positive reinforcement driving the overeating lies even on opioid neurotransmission. Endogenous opioid receptors are largely distributed in areas involved in appetite control. Opioid agonists are food intake stimulants, while naloxone and naltrexone cause anorexia [3]. Moreover, a recent study on humans showed that specific mu-opioid receptor gene (OPRM1) polymorphisms are associated with higher body mass index [12].

Starting from these premises, several studies investigated the opioid system as a potential target for pharmacological therapy of overeating. For example, synthetic opioid antagonists such as LY255582 or a combination therapy with bupropion and naltrexone are actually under clinical assessment. Preliminary data suggest that both strategies may be effective on significant weight loss in animals and humans [7].

Other factors influencing appetite control in experimental animals are stress and intense emotional stimuli, increasing ghrelin plasma levels via the release of glucocorticoids. Ghrelin is an orexigenic peptide that increases food intake and fat stocking, and in turn enhances the hypothalamus-hypophysis axis and glucocorticoid levels, in a positive feedback mechanism. Conversely leptin, the main anorectic hormone, reduces physiologic stress response through both CRH and decrease in glucocorticoids [6].

2. Current and future perspectives

Even if the mentioned data suggested a considerable connection between overeating and cerebral rewarding pathways, food and drug abuse cannot be considered overlapping phenomena at all.

According to clinical and experimental experience, full-blown food addiction behavior is not a frequent condition in the general and overweight population. At the same time, considerable proportions of normal-weight and overweight subjects meet the food addiction criteria [5, 13]. A very large number of people, indeed, are affected by sub-threshold forms of food abuse, that not even lead to degrees of overweight and generally considered as innocent habits or auto-concessions [4].

Stress, bad feelings or simply pleasure-seeking are strong drives to overeat for some predisposed individuals. Sometimes this process can lead to full-blown food addiction behavior, but more frequently to mild forms of food attachment, occurring in scientific literature as hedonic eating, emotional eating or specific food craving. These addictive behaviors are not always included in food addiction operational models despite overlapping expressions and, probably, common neurobiological features.

Spectrum models of clinical, behavioral and neurobiological disorders are largely adopted for nosographic and research aims, including the study of mood and substance use disorders [2, 9]. In this perspective, our group proposes a whole eating addiction spectrum model, including all forms of food attachment, from extreme expressions of food addiction to the mild conditions of eating dysregulation, both featured by proportional degrees of psychological, social and medical impairment.

Emerging literature suggests an interpretation of addiction disorders (including food attachment) in terms of learned compulsive habits. DA striatal release specifically mediates the drug/food-related learning and modulates the consummatory response to them in terms of compulsiveness and perseveration.

Short-term effects of drugs include the activation of the nucleus accumbens shell, in the ventral striatum area. The long-term addictive process involves the shifting of prevalent neural activation from ventral to dorsal striatum, an area involved in attributing salience to specific stimuli and conditioned learning. In addition, lack of pre-frontal cortex control causes loss of substance-related behavioral inhibition. In this perspective, both food and drug addiction are behavioral patterns in which the seeking of specific substance effects are overwhelmed by long-term neural modifications fostering compulsive-impulsive substance (food or drug) consume [8].

Future studies should investigate how those neurobiological mechanisms are compromised in the different forms of food over-intake belonging to the

eating addiction spectrum, and which connection between eating addiction disorders, drug addictions and impulsive-compulsive disorders exists.

3. Conclusions

Food addiction is a stimulating but excessively restrictive model for overeating and overweight development, currently excluding mild and diffuse forms of food attachment.

The spectrum model of overeating could contribute to nosographically placing the nuanced eating addiction disorders. At the same time, a spectrum-dimensional approach could help researchers to connect eating disorders, addiction disorders and compulsive disorders in a common theoretical perspective, supporting the finding of their common neurobiological matrix.

Finally, we hope that deeper knowledge in the broad area of the eating addiction spectrum will help clinicians to diagnose and treat contingent psychopathological aspects of overweight, that currently are almost unheard-of.

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Role of the funding source

No grant funding.

Contributors

All the authors contributed to, and have approved, the final manuscript.

Conflict of interest

Authors declared no conflict of interest.

Received and Accepted August 4, 2013