Neuro-endocrinology

52 BRIEFINGS

Obesity prevention: Moving beyond the food addiction debate

SUMMARY

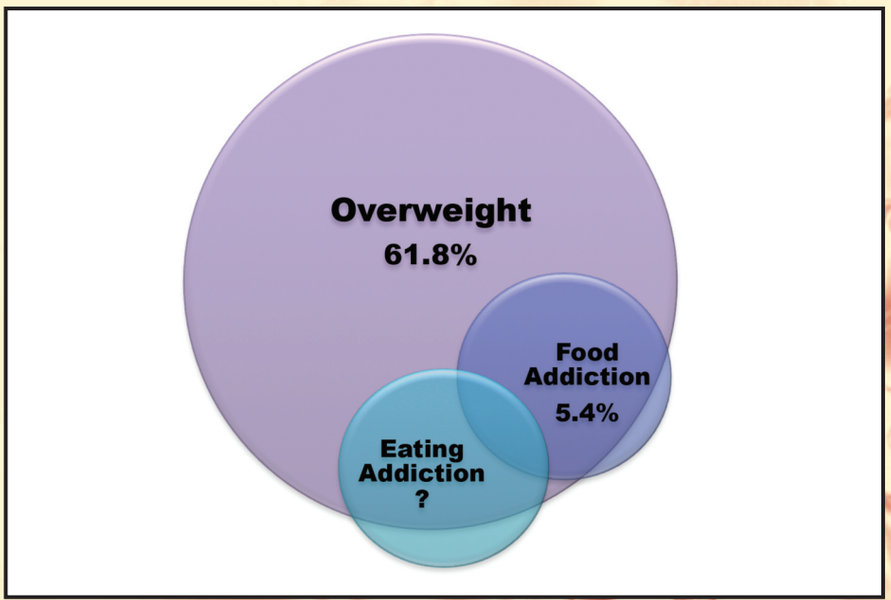
In light of the dire therapeutic situation for obesity, we are witnessing an unfolding discussion on the relevance of addiction in this condition. If substances in food lead to addiction, pressure would mount to pursue structural prevention: social, economic and policy strategies to curtail obesity rates. On the other hand, if eating addiction is best conceptualized as a behavioral addiction, the affected individual is seemingly at fault, reducing this pressure. I propose to move beyond this divisive discussion and argue that structural prevention does not depend on proof that obesity ensues from addiction.

Making choices

Food choice is under the control of at least two interconnected brain systems. The homeostatic systems of the hypothalamus and caudal brainstem, under the influence of leptin, insulin, ghrelin and other signals from the periphery, ensure that our overall intake of calories and nutrients balances energy expenditure to maintain a stable body weight. The hedonic, or reward, system of limbic brain areas drives the motivation to preferentially consume more palatable and energy-dense foods. The reward system has thus been suggested to underlie overeating, hence contributing to the obesity epidemic. Palatable foods activate opioid and cannabinoid pathways in the brain's reward system, so it is natural to speculate whether certain foods could act like drugs of abuse, even to ask whether such foods could have addictive properties.

Diagnosing addition

The Yale Food Addiction Scale, a diagnostic tool for food addiction, diagnoses approximately 5-10% of the general population with this condition, with higher rates in obese than in normal weight subjects. The concept of food addiction has also been promoted by researchers, who have argued that rats can become addicted to sugar. In humans, imaging studies have revealed an overlap of brain regions of the reward system involved in both substance induced disorders and overeating.



Population based prevalence rates of 'Food Addiction' (Yale Food Addiction Scale), 'Eating Addiction' (criteria not yet defined), and overweight (BMI ≥ 25 kg/m²). A large fraction of the population is overweight, but only a small fraction of these are diagnosed with food addiction. The degrees of overlap of the proposed disorder 'Eating Addiction' with overweight and 'Food Addiction', remain as yet undetermined.

Mental disorders are diagnosed using the Diagnostic and Statistical Manual of Mental Disorders (DSM). The latest edition, DSM-5, now refers to the category *Substance-Related and Addictive Disorders*, and includes for the first time Non-Substance Related Disorders (behavioral addictions). This novel extension of the category, previously merely termed Substance Related Disorders (DSM-4), has fuelled the discussion as to whether addiction-like overeating can best be conceptualized as a substance use disorder or as a behavioral addiction to eating.

Paths to preventing obesity

"Food addiction" suggests parallels to nicotine addiction – in terms of prevention policies and legal issues. The consensus is that success in reducing smoking was largely based on efforts to limit advertising, increase the price of, and restrict the access to cigarettes. This success was based on the proof that smoking can result in nicotine dependence. As such, food addiction in the context of a (currently not substantiated) substance use disorder recapitulates this theme with the option for exploitation in political terms. By contrast, eating addiction as a non-substance related (addictive) disorder seemingly lets the food industry off the hook; human behavior is to blame, not specific food products.

Will using the term food addiction indeed help us to promote structural prevention efforts and to engage the food industry in this process? As long as unequivocal evidence in humans does not exist, the notion that specific food products give rise to addiction will be challenged; in fact the debate may prove counterproductive, because the food industry has an excuse to remain on the sidelines. On the other hand, agreed criteria for eating addiction still need to be found.

Clearly, both substance-related and non-substance-related disorders can be prevented via structural efforts. For example, age restrictions on gambling are effective in preventing the development of a behavioural addiction to gambling in minors. However, irrespective of whether scientific evidence will justify use of the term food and/or eating addiction, most obese individuals have neither a food nor an eating addiction. Obesity frequently develops slowly over many years; only a slight energy surplus is required to in the longer term develop overweight. Genetic, neuroendocrine, physiological and environmental research has taught us that obesity is a complex disorder with many risk factors, each of which have small individual effects and interact in a complex manner. The notion of addiction as a major cause of obesity potentially entails endless and fruitless debates, when it is clearly not relevant to the great majority of cases of overweight and obesity.

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Accordingly, it would seem short sighted to rely on scientific evidence for food/eating addiction in an effort to convince politicians and the food industry that something must be done to curtail obesity. The danger is that prevention efforts based on a single, minor cause of obesity risk undermining a holistic strategy that aims to reduce weight gain. We need strategies for preventing obesity that do not overly depend on any particular etiology of obesity. Lessons can be learned from previous successful public health programs such as the large-scale immunization programs which have all but wiped out specific contagious diseases, and programs aimed at preventing traffic accident casualties.
The food industry must contribute to obesity prevention irrespective of whether, and to what extent, addiction is involved. The car industry did likewise: while the driver is often responsible for the accident, the safety features in modern cars have reduced harm and collateral damage.

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