

Food Addiction: Cause or Consequence of Obesity

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Editorial

Poor nutrition and lack of exercise are the primary risk factors that are studied in relationship to weight gain and obesity. Addictive behaviors, such as food craving and overeating may, however, represent the underlying reason for poor food choices and could be an important determinant of obesity. There is conflicting evidence about whether food addiction is a viable behavior that leads to obesity or whether obesity promotes food addiction among individuals who were not addicted to food prior to weight gain [1]. While many investigators concur that food addiction is associated with development of obesity in certain individuals [2], not all obese individuals gain weight because they are addicted to food.

Food addiction has behavioral and neurological qualities that resemble substance abuse and dependence. Increased craving for food or food-related substances leads to a heightened state of pleasure, energy, or excitement [3]. Obese individuals behave differently than normal weighted individuals in relationship to food stimuli and reward [4-7].

Classic pathways and locations in the brain that are well studied in relationship to drug addiction have been linked to food addiction. The reward pathway, most often associated with dopamine, can be initiated by food stimuli. Extracellular dopamine in the dorsal striatum is significantly correlated with increase in self-reports of hunger and desire for food [4]. The somato sensory cortex, location of oral, lip, and tongue sensation, is activated in relationship to pleasure from the taste of food, and may lead to overeating as a result. Taste sensation primarily reinforces food consumption [8], with obese subjects more sensitive to the rewarding properties of food related to palatability [5].

Food craving is an intense desire to eat particular foods that is much greater than normal feelings of hunger [9,10]. Specific foods such as sugar and fat have distinct tastes and provide rewarding sensations. Individuals who crave foods have an intense desire for a particular food, and only eating the food that is craved will satisfy their urges [9]. Animal models have shown that the craving for intense sweetness can exceed the craving for cocaine among rats given the choice of cocaine or water sweetened with saccharin [11], and human studies of opiate addicts show the addicts have stronger preference for intake of sweets compared to controls [12]. Carbohydrate craving could therefore represent an important underlying determinant of obesity due to the strong evidence linking sugar-sweetened beverages and weight gain [13].

Eating beyond satisfying hunger and nutritional needs has multiple determinants, including pursuit of reward and feelings of pleasure. The opioid receptor system regulates intake of palatable food and is linked to pleasurable feelings of 'fullness'. If the opioid receptor system, designed to promote overeating as a protective mechanism against future starvation, becomes dis-regulated then the potential for weight gain and obesity is greatly increased.

While the evidence is clear for obesity influencing food addiction, there is less evidence that food addiction promotes the development

of obesity among previously thin individuals. Associations between heritable candidate genes for addiction and obesity may provide plausibility for food addiction promoting development of obesity. Polymorphic variants of addiction genes such as the Taq1A allele from the dopamine D2 receptor gene, and the functional A118G polymorphism of the mu opioid receptor gene, have been linked to food addiction and obesity [14-16], and combinations of the two alleles linked to binge eating [15]. Evidence from prospective studies is generally lacking, although a study conducted in the screening arm of the Prostate, Lung, Colon, and Ovarian Prevention cohort found an association between DRD2 and obesity [17].

Evidence from prospective studies is needed to establish the link between food addiction and development of obesity. Animal models, genetic studies in targeted populations, and brain scanning studies provide strong biologic evidence that requires verification in population-based samples. Many investigators agree that food addiction is associated with development of obesity in certain individuals [2]. Obesity has multiple determinants and but not all obese individuals gain weight because they are addicted to food. Other individuals that are not addicted to food can develop obesity, for instance, through modest energy imbalances over a sustained period of time [18]. Studies of food addiction and obesity in a population-based sample will undoubtedly include individuals that are not addicted to food, and, therefore a mixture of studies with positive and negative associations between food addiction and obesity are to be expected.

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