Study identifies neural activity linked to food addiction

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Persons with an addictive-like eating behavior appear to have greater neural activity in certain regions of the brain similar to substance dependence, including elevated activation in reward circuitry in response to food cues, according to a report posted online today that will appear in the August print issue of *Archives of General Psychiatry*.

"One-third of American adults are now obese and obesity-related disease is the second leading cause of preventable death. Unfortunately, most obesity treatments do not result in lasting weight loss because most patients regain their lost weight within five years," the authors write. "Based on numerous parallels in neural functioning associated with substance dependence and obesity, theorists have proposed that addictive processes may be involved in the etiology of obesity. Food and drug use both result in dopamine release in mesolimbic regions [of the brain] and the degree of release correlates with subjective reward from both food and drug use." The researchers add that to their knowledge, no studies have examined the neural correlates of addictive-like eating behavior.

Ashley N. Gearhardt, M.S., M.Phil., of Yale University, New Haven, Conn., and colleagues examined the relation between food addiction (FA) symptoms, as assessed by the Yale Food Addiction Scale (YFAS), with neural activation (measured via functional magnetic resonance imaging) in response to cues signaling impending delivery of a highly palatable food (chocolate milkshake) vs. a tasteless control solution; and consumption of a chocolate milkshake vs. a tasteless solution. The study included 48 healthy young women ranging from lean to obese recruited

for a healthy weight maintenance trial.

The researchers found that food addiction scores correlated with greater activation in areas of the brain including the anterior cingulate cortex (ACC), medial orbitofrontal cortex (OFC), and amygdala in response to anticipated receipt of food. Participants with high vs. low FA demonstrated greater activation in the dorsolateral prefrontal cortex and caudate during anticipated palatable food intake and reduced activation in the lateral OFC during palatable food consumption.

"As predicted, elevated FA scores were associated with greater activation of regions that play a role in encoding the motivational value of stimuli in response to food cues. The ACC and medial OFC have both been implicated in motivation to feed and to consume drugs among individuals with substance dependence," the authors write. "In sum, these findings support the theory that compulsive food consumption may be driven in part by an enhanced anticipation of the rewarding properties of food. Similarly, addicted individuals are more likely to be physiologically, psychologically, and behaviorally reactive to substance-related cues."

"To our knowledge, this is the first study to link indicators of addictive eating behavior with a specific pattern of neural activation. The current study also provides evidence that objectively measured biological differences are related to variations in YFAS scores, thus providing further support for the validity of the scale. Further, if certain foods are addictive, this may partially explain the difficulty people experience in achieving sustainable weight loss. If food cues take on enhanced motivational properties in a manner analogous to drug cues, efforts to change the current food environment may be critical to successful weight loss and prevention efforts. Ubiquitous food advertising and the availability of inexpensive palatable foods may make it extremely difficult to adhere to healthier food choices because the omnipresent

food cues trigger the reward system. Finally, if palatable food consumption is accompanied by disinhibition [loss of inhibition], the current emphasis on personal responsibility as the anecdote to increasing obesity rates may have minimal effectiveness," the researchers conclude.

More information: *Arch Gen Psychiatry*. Published online April 4, 2011. doi:10.1001/archgenpsychiatry.2011.32