



## **IOM Workshop Asks “Can Food Be Addictive?”**

July 28, 2014

The Food Forum at the National Academies’ Institute of Medicine (IOM) held a workshop on July 9 and 10 on [Relationships between the Brain, Digestive System, and Eating Behavior](#). The Food Forum is chaired by Francis Busta, University of Minnesota, St. Paul. Eric Decker, University of Massachusetts, Amherst, chaired the workshop planning committee. Early presentations explored the physiological interactions between the brain and the digestive system, and later sessions assessed the science and methodologies behind the “food addiction” model.

### **Interaction between the Brain and the Digestive System**

As part of a session on interactions between the brain and digestive system, presenters Timothy Moran, Johns Hopkins University; Robert Margolskee, Monell Chemical Senses Center; and Robert Ritter, Washington State University, focused their remarks on the physiological mechanisms the gut uses to tell the brain about food that has been ingested.

Laurette Dubé, McGill University, took a different approach and discussed the contextual and environmental influences on food intake. She described four levels of context that impact eating behavior. First, our individual predispositions to food “reward” cues impact how our brains respond to the stimulus of food. Some people have greater tendencies toward “external eating,” or eating in response to cues (the sight, smell, or even discussion of food) regardless of hunger. Second, the fetal environment can have an impact on how people eat; low birth weight is correlated with being overweight later in life. Third, the home and parental environment also plays a role. Growing up skipping breakfast, eating in front of the television, and having food used for reward and punishment can all impact how we eat. Finally, the broader food environment clearly impacts eating behavior. Dubé noted a correlation between soft drink sales and low income neighborhoods, and described the impacts of living in areas with greater density of fast food restaurants. Dubé recommended researchers consider all four of these contexts as part of what she termed a “brain-to-society” model of food behavior.

### **The Science behind Methodologies Used to Characterize Food as Addictive**

A second session focused on the neuroimaging and questionnaire methodologies behind some of the research into whether food can have addictive properties. Dana Small, Yale University, characterized unhealthy food behavior as the result of a vulnerable brain interaction with an unhealthy food environment. She noted that some people demonstrate greater brain responses when presented with food cues (sights, aromas, and tastes), and these people tend to be predisposed to snacking independent from hunger, a behavior she called “hedonic eating.” Small cited research demonstrating that these

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heightened responses to food predict weight gain—even for subjects trying to lose weight. Small noted that our brain’s response to food cues is dependent on dopamine and described her research stimulating this response based on caloric content alone (without the presence of external cues like taste).

Hisham Ziauddeen, University of Cambridge, expressed skepticism that neuroimaging evidence is truly suggestive of food “addiction.” He noted that while we know food and drugs act on overlapping rewards systems in the brain, much of our knowledge of how these processes work come from animal studies and some researchers have had difficulty replicating this work. He also pointed to imprecision in what the concept of food addiction refers to: are certain foods addictive? Or do some people overeat in ways that resemble consumption of addictive substances?

Ashley Gearhardt, University of Michigan, discussed her research into food addiction, as measured by the Yale Food Addiction Scale, which she developed. She postulated that food addiction could be a potential explanation for some of the more intractable problems of the obesity crisis—the rising obesity rate, the high proportion of people who regain weight after losing it, and the rise of binge eating disorder. She observed that the increased potency of fats, sugars, and salt in ultra-processed food is unprecedented and might give these types of foods addictive-like properties. The Yale Food Addiction Scale (YFAS) was developed as a questionnaire to begin to operationalize the food addiction model. It is based on the criteria for addiction in the American Psychiatric Association’s *Diagnostic and Statistical Manual of Mental Disorders* (DSM) IV. Gearhardt’s research showed that those who scored high on the YFAS tended to have higher current and lifetime BMIs, were at greater risk for negative health outcomes, were more impulsive eaters, and had higher emotional dysregulation. Gearhardt readily acknowledged that more research remains to be done, but suggested that food addiction as a diagnosis could potentially reduce the stigma associated with obesity.

Charles O’Brien, University of Pennsylvania, spoke from his experience as chair of the working group that focused on substance-related disorders for the recently updated DSM-V. He explained that the criteria underlying substance use disorder are mostly unchanged from DSM-IV and include tolerance, withdrawal, and loss of control (using more of the substance than intended, craving, unsuccessful attempts to cut down, spending excessive time acquiring the substance, continued use despite negative effects, etc.). A note was added to indicate that tolerance and withdrawal could result from appropriate use of substances under medical supervision. Food addiction was considered for inclusion in DSM-V, but was ultimately left out. Considerations for adding a diagnosis include clinical need, sufficient distinction from other disorders, potential harm adding the disorder might bring, potential treatments, and whether the diagnosis meets the criteria for a mental disorder.

### **Is the Addiction Model Appropriate for Food?**

Another session was structured like a debate, with one speaker presenting arguments that the addiction model is appropriate for food and the other arguing that it was not. Nicole Avena, Columbia University, gave the “pro” perspective. She argued that while obesity has many contributing factors, food addiction should be considered among them. Avena presented evidence demonstrating that rats can exhibit bingeing and tolerance behaviors with sugar water, can show signs of anxiety and distress when the sugar source is removed (withdrawal), and can “crave” the sugar (subjecting themselves to shock in order to acquire sweet foods). She also discussed a study showing that rats presented with a “cafeteria diet” of many different types of junk foods no longer respond to plain rat food. She suggested that food addiction could be considered a “functional” addiction, analogous to nicotine addiction, rather than similar to cocaine or heroin addiction.



Peter Rogers, University of Bristol, argued that the addiction model should not be applied to food. He began by making the distinction between a dependence, where the substance is needed to function within normal limits (e.g. caffeine), and an addiction, where control over use of the drug is lost. Rogers also referenced the “cafeteria diet” rat study, and suggested an alternative explanation: that once the rats gained weight on their buffet of junk food, they lost interest in normal rat food as their bodies returned to equilibrium. He suggested that cravings, which are hard to study because they are a mental process, might be the result of trying to resist our brains’ food cues. He concluded by observing that even if the food addiction model described the eating behavior of some people, most obese people would not qualify as “addicted,” raising the question of whether the model has utility for public policy.

