Addicted to Food? The New Research Suggests It's Possible

Science is now proving what we've long suspected: we're hard-wired to want the foods that are worst for us

Newsweek

Like most recovering addicts, Kay Sheppard has a testimony. Hers is this: the Florida mother of two spent years making trips to the store to buy cookies and chips for her family, eating almost every bag and box in the car on the way home. Then came the year she bought her dad chocolates for Christmas. She stuffed the candy in her dresser drawer and later finished off the entire thing—repeating this cycle five times with five boxes of candy. One day, she spied herself eating in the mirror and was horrified. "That was the first time in my 30-some years that I ever thought what I was doing was abnormal, because I had done the same thing year after year after year," she says.

She had an epiphany. She began to think of herself not as a dieter but as an addict in recovery. In this way of thinking, her drugs of choice were sweets, carbohydrates, and any sugary, fatty, salty substance that never grew from the ground or walked upon it. Like an alcoholic giving up drinking one day at a time, she began a daily abstinence from highly processed, high-carbohydrate foods. She stopped thinking about her weight. Her approach was so successful that she founded a program, wrote a couple of books, and now helps others. "If you focus on weight, you will lose," she says, but "if you focus on recovery, you lose weight."

To Sheppard, some people become obese because food has a druglike power over them. "I mean if you had control—if you weren't out of control—wouldn't you go on a diet and lose some weight?" says Sheppard, now a master's-level mental-health counselor. She adopted her philosophy long before much science backed her up.

But the theory that the brain responds to high-fat, high-calorie foods similarly to how it responds to drugs is now gaining scientific muscle, led by renowned names in the field of addiction. Over the past few years, it has become one of the hottest topics in obesity research, receiving nearly $6 million of funding in 2011 alone from the National Institute on Drug Abuse. "There is a huge amount of research now going on in this area," says the institute's head, Nora Volkow. In general, especially in studies of rodents, the brain appears to uniquely draw us to high-calorie, low-nutrient foods of the kind filling the shelves at every Kwik Chek, 7-Eleven, and corner deli.

Categorizing calorie- and fat-dense foods as addictive would mean, as it did with Sheppard, that "dieting" could become "recovery" and steps might be taken to restrict the way certain foods are marketed and sold. It could open the door to cigarette-style taxes and warning labels. "Do you try to change the nature of our food supply so there's just not so much exposure to this stuff? What kind of food should be marketed to kids?" says Kelly Brownell of Yale University, where he and others have developed a Food Addiction Scale to try to measure whether a person's eating is driven by addiction-like cravings or just appetite.

The idea still has skeptics, but if borne out, it stands to open the door for public-policy changes that could have as profound an effect on the food industry as nicotine research had on tobacco. Of course, not everyone who is overweight may be suffering from an addiction. But figuring out who could be—and more broadly, pinpointing the extent to which a druglike pull of junk food could be contributing to the obesity epidemic—stands to change our understanding of the most serious health crisis in the country.

While scientists have long theorized about an addiction-like quality to certain foods, only recently has the idea been the subject of intense scientific research, scrutinizing what studies call "highly palatable" foods

such as soda, ice cream, french fries, and even pepperoni pizza. It's a definition that has to do with how much the foods appeal to our appetite—and how hard they are to resist.

Mark Gold, chairman of psychiatry at the University of Florida, a longtime tobacco researcher, turned to obesity after noting that addiction recovery commonly leads to hunger and weight gain. To keep off drugs, "we noticed it was important for a recovering addict to stay full," he says. His studies have pointed to the possibility that eating may satisfy the same brain cravings that drive a person to addictive tobacco, alcohol, and drug use. In the *Journal of Addictive Diseases*, he has reported that, generally speaking, the higher a person's body mass index, the lower the consumption of alcohol. Similarly, in a study published in June in the *Journal of the American Medical Association*, researchers from the University of Pittsburgh found that alcohol-use disorders may increase significantly two years after undergoing gastric bypass surgery.

By far the most convincing case for food addiction comes from studies in animals, where researchers are investigating biochemical changes that occur after animals are fed highly palatable foods. When animals eat a high-fat diet, the brain produces a suite of chemicals in response. In experiments at the Rockefeller University, neurobiologist Sarah Leibowitz has injected rats with those chemicals and found that it led animals to eat more fat. "The more you eat, the more you want," she says.

That may help explain why it's difficult to keep from eating badly once it becomes habit. Animals in one recent experiment published in *Nature Neuroscience* that had access to sugary and fatty "cafeteria" foods (among them, cheesecake and bacon) for one hour a day began to binge on those foods, waiting for them even when other options were set out all day long. Another group of animals offered almost unfettered access to the sweeter, fattier food became obese. The scientists, from Scripps Research Institute in Florida, found that animals continued to prefer the cafeteria diet even when threatened with electrical shock. And the late Bart Hoebel from Princeton University also found druglike responses to sugar when his laboratory investigated the effect in rats: when sugar was offered in high doses and then taken away, animals experienced classic withdrawal symptoms like anxiety, tremors, and chattering teeth.

The animal data even suggest that the possible changes in the brain from a diet of sweet and fatty foods may be passed on to the next generation. Leibowitz, from Rockefeller, reported recently that pregnant rats on a high-fat diet gave birth to offspring with neurological alterations in the hypothalamus, part of the brain heavily associated with appetite. In 2010 in the journal Endocrinology, scientists from the University of Pennsylvania School of Medicine found that pregnant mice that ate a heavily junk-food diet gave birth to pups not only with changes to the reward mechanisms of the brain, but with a preference for sugar and fat.

Of course, extrapolating from rats to humans is difficult because people have more variable genes and lifestyles than rats in a cage. This complicates research design and interpretation. Nonetheless, in some experiments the brain-imaging scans of an obese person resemble the brain scans of an addict. One of the most notable studies in this vein came in the early 2000s, when Nora Volkow and her team at Brookhaven National Laboratory placed 10 obese volunteers into a PET scanner to study the reward mechanisms in their brains. (Volkow later became head of the National Institute on Drug Abuse.) They found that the brains of these obese people looked different from those of people of normal weight; specifically, the obese people lacked certain receptors to dopamine, one of the brain chemicals associated with reward and drug abuse.

Dopamine receptors can reveal a great deal about the dynamics of pleasure, reward motivation, and addiction. Two widespread channels for dopamine signaling in the brain occur through receptors known as D1 and D2. In general, stimulating D1 receptors motivates a specific behavior; stimulating D2 inhibits it. If you lack D2 receptors, as Volkow found in her subjects, you will not be able to inhibit the strong urges that are then sent into the areas of the brain involved in action, she says, leading to overeating. She says the

deficit in D2 could also encourage overeating by making people less sensitive to the pleasure of eating itself, needing greater quantities of food to feel the reward.

But the biology that separates an almond from an Almond Joy goes far beyond dopamine. One of the key hormones involved in appetite, called ghrelin, is also involved with the reward system in the brain that drives us to eat, says Jeffrey Zigman of the University of Texas Southwestern Medical Center at Dallas, who studies appetite mechanisms that often overlap with the circuitry of addiction. He and others theorize that some people overeat because they produce more ghrelin. In fact, so much wiring in the brain appears to drive us toward high-calorie eating that some scientists suspect food is perhaps the reason drug addiction exists in the first place. The neurons in the brain that make a milkshake feel good "likely are the same neurons as when you're talking about drug reward," Zigman says.

That programming is probably there to make sure our ancestors took advantage of times when they could load up on calories. But along came hyper-refined substances like cocaine and alcohol that could hijack the system and take it to a new intensity. And maybe, if the research is correct, that's possible with other highly processed substances like high-fructose corn syrup and 1,000-calorie cheeseburgers.

While compelling, the addictive nature of food is by no means a universally accepted theory. It follows a credible narrative, says Paul Fletcher, a neuroscientist and psychosis expert at the University of Cambridge. But he says the literature is fuzzier when viewed as a whole. "There are huge inconsistencies across the data," he says. He and others are not ready to dismiss the possibility of food addiction, but they say the evidence is not strong enough to embrace it.

What they don't dispute is that the brain has some kind of programming that has drawn us to foods that are sweet and fatty as a survival mechanism over the course of human evolution. Debate arises when this system is likened directly to drug abuse. "I am sure whenever I present my work not to use the word[s] addiction or craving," Zigman says. Why? The substance-abuse community has a strict definition for what constitutes addiction, he says. It encompasses behaviors that go beyond simple enjoyment and involve other dimensions, like tolerance and life disruptions because of the drug and using it long after the joy is gone. While the pleasure of a potato chip may be widely felt, only a small fraction of people (say, those with disorders like binge eating) may be truly addicted. To the skeptics, it is still unclear how much of a role addiction might play in the obesity epidemic and who might be affected.

But for those pushing this research forward, the fact that most people would not have an addiction to food does not diminish the implications of the research. Psychiatrist Mark Gold makes the comparison with alcohol. Most people can have a happy-hour drink or two and stop, but those who can't suffer serious consequences. "What's the difference between a person that has the ability to do that and the 15 percent of the people in the United States that will be expected to lose control?" he says. "There will be genetic differences. There will be prenatal dietary differences and prenatal exposure differences. There will be early-childhood differences. There may be other health issues like pain or depression." Even though most people do not become addicted to alcohol, it is still highly controlled because some people do.

Even this preliminary research is beginning to change the way some scientists are thinking about the approach to weight loss. Historically, that treatment has focused on a model of appetite suppression, says Gold. But in October, at a meeting of the Society for Neuroscience, he described his work using two substance-abuse medications—baclofen (which may reduce cravings) and naltrexone (which blocks opiates)—to try to prevent binge eating in animals. After injection of the drugs, rats that had been raised to binge on fat and sugar were less likely to overeat, and the combination of the two medications worked better than either alone.

Beyond medication, relegating some kinds of food to pariah status—much like cigarettes have been—might also reduce their visibility and therefore reduce the prompts to eat. David Kessler, the former head of the Food and Drug Administration known for leading the regulation of cigarettes, calls this mechanism "cue-induced wanting," or a sudden need triggered by the sight of something. And he thinks we need to learn from the anti-tobacco model with highly processed, high-calorie foods. "What's changed in the last four decades?" he says. "We changed our environment. We increased the number of cues. We made it socially acceptable to eat any time."

Many advocates of the food-addiction hypothesis also hope to eventually limit access to certain foods, especially for children, whose immature brains are vulnerable to forming the foundations for dependency and addiction. "If parents start to believe that these foods are having a negative effect on the brains of their children," says Brownell, of Yale, "they might very well want to keep them away from their kids. They may not want schools to be selling them. To me, there are the big implications."