WE ARE ALL PROGRAMMED TO LIKE SUGAR. New research shows some are genetically much more prone to sugar and food addiction than others. I have observed this in my patients, but now it is becoming clear why some have more trouble kicking the sugar habit than others.

As I reviewed in my previous article on food addiction, the science demonstrating that people can be biologically addicted to sugar in the same way we can be addicted to heroin, cocaine or nicotine is clear. Bingeing and addictive behaviors are eerily similar in alcoholics and sugar addicts. In fact, most recovering alcoholics often switch to another easily available drug: sugar.

It seems that we all vary a bit in our capacity for pleasure. Some need a lot more stimulation to feel pleasure driving us to a range of addictive pleasures that stimulate our reward center in the brain – drug and alcohol addictions, compulsive gambling, sex addiction and, of course, sugar, food addiction and compulsive eating. We often see these as moral failures or results of character defects. In fact, it may be that addicts of all stripes are simply unlucky and born with unfortunate genetic variations in our reward and pleasure mechanisms.

The Genetics of Pleasure

In our brain, a little receptor, the dopamine receptor D2 or DRD2 for short, must be activated or switched on for us to feel pleasure. The amino acid dopamine triggers this response. Sugar and other stimulating addictions increase dopamine in the short term. The only problem is it appears that those with sugar addictions, compulsive eating, and obesity have DRD2 systems that need much more stimulation to feel pleasure. Those who have sugar addiction, it seems have fewer D2 dopamine receptors and they need extra stimulation to make them "turn on".[ii]

Functional MRI studies of teenagers, both lean and obese, found that the obese teenagers whose brains didn't light up as much in the dopamine reward centers were more likely to be obese and gain weight later.[iii] They also were more likely to have the DRD2 gene that coded for fewer receptors. Some studies have pointed to drugs or nutrients that can modulate this defective dopamine reward response. In one study, naltrexone, an opioid blocker (blocks the effects of heroin and morphine on the brain) was used in sugar addicts. When they took this drug, which prevented the reward response, they ate less.[iv]

We also know that amphetamines are natural appetite suppressants and reduce cravings. That is why children who take stimulant ADHD drugs (which are actually just fancy amphetamines) that stimulate dopamine receptors have trouble gaining enough weight as they grow.

There are also some promising studies of nutraceuticals[i] that can modulate dopamine receptor function and appetite regulation.[iv] Bruce Ames, PhD found that high levels nutrients can reduce disease in people with 50 different gene variants, nutrients may modulate the function of our genes, improve their function, or affect the activity of enzymes that genes produce.[v] In fact, one third of our entire DNA has one simple job: To code for and produce enzymes controlled by nutrient co-factors. This means that nutrients have a powerful ability to modify the expression of your genes. This is the important field of nutrigenomics.

Overcoming Your Addiction to Sugar

Despite being stuck with the sugar addiction low pleasure gene, you may be able to modify its activity by modulating your brain chemistry and receptor function with the use of specific nutrients that either improve gene expression, or modify the activity, the enzymes, or the receptors, even if they are somewhat impaired.

I have used some of these in my practice, such as glutamine and other amino acids, with success. Regulation of hormones and neurotransmitters that affect appetite and cravings is complex and involves many factors including how quickly food spikes our blood sugar, stress, getting enough sleep, nutritional deficiencies, chemicals such as artificial sweeteners, food sensitivities which drive inflammation, and more.

For those with personal struggles with food addiction, remember it is not a moral failing or lack of willpower. Here are a few suggestions I offer my patients to help them break their food addictions.

1. **Balance your blood sugar:** Research studies say that low blood sugar levels are associated with LOWER overall blood flow to the brain, which means more BAD decisions. To keep your blood sugar stable:
Eat a nutritious breakfast with some protein like eggs, protein shakes, or nut butters. Studies repeatedly show that eating a healthy breakfast helps people maintain weight loss.

Also, have smaller meals throughout the day. Eat every 3-4 hours and have some protein with each snack or meal (lean animal protein, nuts, seeds, beans).

Avoid eating 3 hours before bedtime.

2. Eliminate sugar and artificial sweeteners and your cravings will go away: Go cold turkey. If you are addicted to narcotics or alcohol you can't simply just cut down. You have to stop for you brain to reset. Eliminate refined sugars, sodas, fruit juices, and artificial sweeteners from your diet. These are all drugs that will fuel cravings.

3. Determine if hidden food allergies are triggering your cravings. We often crave the very foods that we have a hidden allergy to. For a simple allergy elimination program, consider trying The UltraSimple Diet, or The UltraSimple Diet Challenge Home Study Coaching Program.

4. Get 7-8 hours of sleep. Research shows that lack of sleep increases cravings.

5. Optimize your nutrient status with craving cutting supplements

- **Optimize your vitamin D level:** According to one study, when Vitamin D levels are low, the hormone that helps turn off your appetite doesn't work and people feel hungry all the time, no matter how much they eat.
- **Optimize omega 3c:** Low levels of omega three fatty acids are involved in normal brain cell function, insulin control and inflammation.
- **Consider taking natural supplements for cravings control:** Glutamine, tyrosine, 5-HTP are amino acids that help reduce cravings. Stress reducing herbs such as Rhodiola can help. Chromium balances blood sugar and can help take the edge off cravings. Glucomannan fiber is very helpful to reduce the spikes in sugar and insulin that drive cravings and hunger.

Now I'd like to hear from you.

Have you ever been addicted to sugar? What was it like?

Do you think the food industry is feeding us products we become addicted to so they can increase profits?

Have you tried overcoming food addiction using any of these steps? How did they work for you?

Please share your thoughts by leaving a comment below.

To your good health,

Mark Hyman, MD

**References**


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**Junkie food: Tastes your brain can't resist**

**POSTED BY ADMIN ON SEP 13, 2010 IN HEALTH | 0 COMMENTS**

Is that cupcake an innocent indulgence? Or your next hit? We're finding that a sweet tooth makes you just as much an addict as snorting cocaine

SETTLED on the sofa watching the usual rubbish on TV, I notice that predictable, uncontrollable, nightly craving. At first I sit there, fighting it. But the longer I fight, the worse it gets. After 20 minutes, I can't concentrate on anything, I feel anxious, and start fidgeting like crazy. Finally, admitting my addiction, I break. I go to the freezer – to my stash of white stuff – and take a hit. Almost instantly, I relax, my brain in a state of bliss as the chemical courses through my veins. Isn't it amazing what a few scoops of ice cream can do?

Before you dismiss my agitation as mere weakness, consider this: to my brain, sugar is akin to cocaine.
There is now compelling evidence that foods high in sugar, fat and salt – as most junk foods are – can alter your brain chemistry in the same way as highly addictive drugs such as cocaine and heroin.

The idea, considered fringe just five years ago, is fast becoming a mainstream view among researchers as new studies in humans confirm initial animal findings, and the biological mechanisms that lead to “junk-food addiction” are being revealed. Some say there is now enough data to warrant government regulation of the fast food industry and public health warnings on products that have harmful levels of sugar and fat. One campaigning lawyer claims there could even be enough evidence to mount a legal fight against the fast food industry for knowingly peddling food that is harmful to our health, echoing the lawsuits against the tobacco industry in the 1980s and 90s.

“We have to educate people about how their brains get hijacked by fat, sugar and salt,” says David Kessler, former commissioner of the US Food and Drug Administration and now a director of the Center for Science in the Public Interest, based in Washington DC. With obesity levels rocketing across the world, it is clear that I am not alone in my love of sweet things, but can it really be as bad as drug addiction?

We have to educate people about how their brains get hijacked by fat, sugar and salt. Arguably, it was the weight-loss industry that first introduced the idea to the public, long before there was any scientific evidence for it. For example, in her book Lick the Sugar Habit, published in 1988, the self-confessed “sugarholic” Nancy Appleton offered a checklist to determine whether you, too, are addicted to sugar. Since then, the notion has become commonplace.

In 2001, intrigued by this nascent cultural phenomenon, neuroscientists Nicole Avena, now at the University of Florida in Gainesville, and Bartley Hoebel at Princeton University, together began exploring whether the idea had a biological basis. They started by looking for signs of addiction in animals that had been eating junk food.

**Hooked on sugar**

Sugar is a key ingredient in most junk food, so they offered rats sugar syrup, similar to the sugar concentration in a typical soda beverage, for about 12 hours each day, alongside regular rat feed and water. After just a month on this diet, the rats developed behavior and brain changes that Avena and Hoebel claimed were chemically identical to morphine-addicted rats. They binged on the syrup and showed anxious behavior when it was removed – a sign of withdrawal. There were also changes in the neurotransmitters in the nucleus accumbens, a region associated with reward.

Crucially, the researchers noticed that the rats' brains released the neurotransmitter dopamine each time they binged on the sugar solution, even after having eaten it for weeks (Neuroscience & Biobehavioral Reviews, vol 32, p 20). That's not normal.

Dopamine drives the pursuit of pleasure – whether it is food, drugs or sex. It is a brain chemical vital for learning, memory, decision-making and sculpting the reward circuitry. You would expect it to be released when they eat a new food, says Avena, but not with one they are habituated to. “That's one of the hallmarks of drug addiction,” she says. This was the first hard evidence of a biological basis for sugar addiction, and sparked a slew of animal studies.

Those results were among the most exciting news in obesity research in the last 20 years, says Mark Gold, an international authority on addiction research and chairman of the psychiatry department at the University of Florida College of Medicine.

Since Avena and Hoebel's landmark study, scores of other animal studies have confirmed the findings. But it is recent human studies that have finally tipped the balance of evidence in favor of labeling a love of junk food as a proper addiction.

**Addicted brains**

Addiction is commonly described as a dulling of the “reward circuits” triggered by the overuse of some drug. This is exactly what happens in the brains of obese individuals, says Gene-Jack Wang, chairman of the medical department at the US Department of Energy's Brookhaven National Laboratory in Upton, New York. In another landmark study published in 2001, he discovered a dopamine deficiency in the striatum of the brains of obese individuals that was virtually identical to those of drug addicts (The Lancet, vol 357, p 354).

In subsequent studies, Wang showed that even when (not obese) individuals are shown their favorite foods, an area of their brain called the orbital frontal cortex – involved in decision-making – experiences a surge of dopamine. The same area is activated when cocaine addicts are shown a bag of white powder. It was a shocking discovery that showed you don't have to be obese for your brain to exhibit addictive behavior. “I can tell they want it,” says Wang.

Another critical leap in identifying junk food as addictive was made by Eric Stice, a neuroscientist at the Oregon Research Institute in Eugene. Stice has been trying to predict a person's propensity to junk food addiction. He has been watching how people's brains respond when they are fed a brief burst of creamy chocolate milkshake. He then compares the brain activity of lean and obese individuals, to see if it differs.

In an as-yet-unpublished study he found that when fed milkshakes, lean adolescents with obese parents experienced a greater surge of dopamine – indicating a greater sense of satisfaction – than those who had lean parents. Stice suspects that this is where the problem begins. “There are people born for whom eating is just more orgasmic,” he says. It is this innate enjoyment of food that primes certain people to overeat.

There are people born for whom eating is just more orgasmic. Ironically, as they overeat, their reward circuitry dulls, which makes the food less satisfying and
Stice has also shown that people with certain variants of the DRD2 and DRD4 genes are endowed with less active dopamine circuits, and as a result have a dulled dopamine response when eating appetizing foods. Paradoxically, this places them at greater risk of obesity than a person without those gene variants because it means they have to eat more to get a sufficiently rewarding level of dopamine release (Science; vol 322, p 449; NeuroImage, vol 50, p 1619).

Together, these studies suggest there are two routes to food addiction corresponding to overactive or underactive dopamine systems, respectively: one if you find food more rewarding than the average person, and another if it isn’t rewarding enough.

Of course, fast food is more than just a sugar rush, it is often a rich cocktail of sugars, fats and salt. Neuroscientist Paul Kenny at The Scripps Research Institute in Jupiter, Florida, is probing the impact of a junk food diet on rat behavior and brain chemistry. One of his recent studies showed that these foods trigger the same changes in the brain as those caused by drug addiction in humans. In animals, as in humans, repeated cocaine or heroin use dulls the brain’s reward system. This leads to heavier use because the memory of a more pleasurable effect spurs the user to take more to get the same feeling, essentially chasing the high.

Kenny wondered whether rats that eat junk food would have a similar response to the cocaine-addicted rats he had already studied. He used three groups of rats. The first was a control group that only had access to standard rat feed. The second group could eat junk food – bacon, sausage, icing and chocolate – for only 1 hour each day with regular rat feed and water available for the rest of the time. The third group had an all-you-can-eat, around-the-clock buffet that included junk food and rat feed. After 40 days, Kenny stopped access to the junk food in both experimental groups. The rats with unlimited access to junk food essentially went on a hunger strike. “It was as if they had become averse to the healthy food,” says Kenny. It took two weeks before the animals began eating as much as those in the control group.

Unlimited access to a powerfully addictive drug like cocaine has a big impact on the brain, says Kenny, so you might expect any addictive effect from food to be much less pronounced. But that is not the case. “Changes happened rapidly and we really saw very, very, striking effects. That’s what surprised me.”

The obese, unlimited junk food rats had dulled reward systems and were compulsive eaters. They would even tolerate electric shocks to their feet designed to deter them from eating junk food when the rat feed was still available shock-free. Cocaine-addicted rats behave the same way towards their drug.

When Kenny examined the brains of the obese rats with the unlimited junk food diet, they too had a dopamine deficiency in their striatum, similar to the obese individuals in Wang’s study in humans. In the rats’ brains, Kenny noticed there was a marked drop in a particular dopamine receptor, called D2. But it wasn’t clear whether this drop affected a rat’s propensity to become addicted to junk food.

To test the relevance of D2 receptors, he artificially reduced their number in the brains of a group of rats and then offered them only junk food for two weeks. The effect was dramatic. Compared to the control group, the modified rats showed a dulled response almost immediately. Unlike normal rats, they gorged on junk food even when eating it was penalized with an electric shock. Crucially, rats with reduced D2 receptors fed only regular rat food did not show the same change in their reward circuitry (Nature Neuroscience; vol 13, p 635). It seems there is an interaction between reduced D2 receptors and consumption of junk food that leads to addiction, says Kenny.

Taken together with Stice and Wang’s results, this suggests that people who from birth have a low number of D2 receptors could also be prone to junk-food addiction. Kenny cautions that more studies in humans are needed before the conclusion can be generalized beyond rats.

Gold says there is plenty of evidence that food and drug addiction are so similar that treatments proven safe and effective for other addictions — such as alcohol, nicotine, cocaine and heroin — should be tested for food addiction too. “The real test of the ‘hedonic eating’ or food addiction hypothesis is if it can yield new and effective treatments,” he says.

What some people claim is now beyond doubt is that junk foods rich in salt, sugar and fat switch on biological mechanisms that are just as powerful, and hard to fight, as drugs of abuse. Given that we regulate drugs because of the harms they can cause, is it time to begin tougher regulation of fast food too?

Junk foods switch on biological mechanisms that are just as hard to fight as recreational drugs. John Banzhaf, a lawyer who teaches public interest law at George Washington University Law School in Washington DC, has been following the research for the last decade. In the 1960s, he won a court ruling that forced radio and TV stations across the US to provide free airtime for anti-smoking messages and played a major role in crafting lawsuits against the tobacco industry. Now he is turning his attention to the fast food industry and its role in fueling the obesity epidemic.

Banzhaf believes there is now enough research for the US Office of the Surgeon General to issue a report on food addiction, as it did for nicotine addiction in 1988. “The Health Consequences of Smoking: Nicotine Addiction”, a report weighing in at over 600 pages, concluded that cigarettes were addictive, nicotine was the cause, and that the chemical and behavioral processes that define heroine and cocaine addiction were the same for tobacco. “At that point people began to accept it,” Banzhaf says. But he acknowledges this is going to be a tricky fight. “Fast food isn’t a [single] chemical so you
It is clinically significant that a deficit of serotonin is central to the development of depression, and that 5-HTP helps alleviate depression and reduces aggressiveness directly from the general circulation (by being transported across the blood-brain barrier).

Levels in foods such as bananas and is important for a variety of brain functions. Serotonin, however, cannot cross the blood-brain barrier, although it is produced in the body from the dietary amino acid tryptophan in only two steps. Serotonin can also be made from 5-hydroxy L-tryptophan (5-HTP), which is simply the modified amino acid obtained from plant sources.

The chemical serotonin is important for a variety of brain functions. Its deficit is now believed to be central to the development of depression, agitation, sleep disorders, obesity and addiction. Serotonin can be produced in the body from the dietary amino acid tryptophan in only two steps. Serotonin can also be made from 5-hydroxy L-tryptophan (5-HTP), which is simply the modified amino acid obtained from plant sources.

The pharmaceutical control of brain serotonin levels is the mechanism of action of two commonly prescribed classes of drugs used in the treatment of depression and anxiety, a well known example being Prozac. A relative deficiency of serotonin is also believed to be associated with the brain's perception of starvation and hunger. For these reasons, 5-HTP has been used as an antidepressant, as an appetite suppressant in obese persons and as an inducer of sleep.

5-HTP: A dietary precursor to an important chemical in the brain

Notwithstanding the fact that electrical impulses travel down an individual nerve fiber, nerves communicate with each other chemically and not electrically. Nerves are classified according to their ability to either secrete or respond to various chemical compounds called neurotransmitters. (These molecules are secreted into the gap between two different nerve cells, called either the synapse or the synaptic cleft. A nerve impulse is fired when enough receptor molecules on the “post-synaptic” nerve bind specifically to one or another class of neurotransmitter that was secreted by the “pre-synaptic” nerve.) Different regions of the brain – and different neurotransmitters – mediate disparate processes, ranging from highly aware mental cognition to unconscious manifestations, such as hunger and sleep.

Neurotransmitters are derived from precursor chemicals found in common food substances. For example, acetylcholine is produced by nerves from choline, a chemical that can be obtained commercially from soybean lecithin. Acetylcholine is secreted by the motor nerves into the neuromuscular junction thereby, stimulating muscular contraction. The neurotransmitters GABA and norepinephrine are derived from proteins that contain the amino acids glutamate and tyrosine, respectively. Adrenaline is chemically related to norepinephrine and is used medically to restart hearts that have been arrested following a heart attack. The neurotransmitter, serotonin, is present in high levels in foods such as bananas and is important for a variety of brain functions. Serotonin, however, cannot cross the blood-brain barrier, although 5-HTP – a nutritional supplement – can enter the brain directly from the general circulation (by being transported across the blood-brain barrier).

5-HTP helps alleviate depression and reduces aggressiveness

It is clinically significant that a deficit of serotonin is central to the development of depression.
agitation, sleep disorders, obesity and addiction. For this reason, the pharmaceutical control of brain serotonin levels is the mechanism of action of two commonly prescribed classes of drugs used in the treatment of depression. Prozac is an example of a selective serotonin reuptake inhibitor (SSRI), which prevents the "presynaptic" nerve from reabsorbing serotonin that it has previously secreted. By inhibiting this normal process, Prozac causes an increase in brain serotonin levels and a non-narcotic anti-depressant effect. Another class of antidepressant drugs, the monoamine oxidase (MAO) inhibitors, cause an increase in serotonin levels by preventing its degradation. Conversely, the experimental depletion of serotonin in animals – by eliminating tryptophan from the diet – causes an increase in aggressiveness. [Note later section entitled “5-HTP or Prozac or Both?”]

5-HTP as an appetite suppressant

Decreased brain serotonin levels are also associated with obesity due to overeating. Drugs like fenfluramine (Fen Phen), which increases serotonin production, are used as an appetite suppressant successfully in the treatment of common obesity. A relative deficiency of serotonin is believed to be associated with the brain's perception of starvation and hunger. Tryptophan is one of the rarest of the essential amino acids – one that the body cannot produce – but one that is needed for the production of vital proteins. Consequently, the dietary depletion of tryptophan, a serotonin precursor, is an ideal homeostatic mechanism in the brain for regulating the desire for food intake.

Hunger sensation in the brain is believed to occur in the region called the hypothalamus. Opposite to the effect of food deprivation, the specific intake of carbohydrates and various sugars cause an increase in brain serotonin levels. This explains why some people are willing to eat an excess of "junk food" that entirely lacks any protein.

5-HTP – which increases serotonin levels – is an appetite suppressant at low doses (50 to 200 milligrams) if taken one-half hour before meals. At high doses, a common side effect of 5-HTP is nausea. During clinical trials in obese subjects, the intake of 5-HTP caused a voluntary decrease in caloric intake of both carbohydrates and fats, but not of protein. A significant loss of weight occurred, due to a voluntary decrease in caloric intake and not because of a restrictive diet. 5-HTP should also help in the adherence to a diet that is both low in calories and fat, but that is high in protein. 5-HTP should always be taken with adequate amounts of protein in the diet – not with a starvation weight loss regimen. [Note the later section entitled "5-HTP or Prozac or Both?" for a short discussion on the cardiac side effects of fenfluramine (Fen Phen) and the brain damage associated with the use of dexfenfluramine (Redux) another drug commonly prescribed to suppress appetite.]

5-HTP, addiction and pain

Serotonin levels are also increased by the intake of addictive substances, such as alcohol, tobacco, certain narcotics and caffeine. Individuals attempting to kick these habits develop a chemical withdrawal syndrome when serotonin levels plummet. These results are observed in both experimental animals and in people. The above findings indicate that overeating is, in part, chemical dependency related – by low serotonin levels – to other chemical addictions and to depression.

Pain sensitivity increases, as well, when brain serotonin levels are low. This has been presented by some researchers as one contributing factor in pre-menstrual syndrome (PMS). Agitation, pain irritability and depression are characteristic aspects of PMS, perhaps each related to a sex hormone-induced decrease in "serotonergic" activity. Conversely, pain sensitivity is markedly impaired during alcohol intoxicification, which temporarily increases serotonin levels in the brain.

5-HTP gently induces sleep

Perhaps the most immediate effect of 5-HTP is its ability to induce sleep when taken on an empty stomach about one hour before going to bed. A 100 mg dose is effective in a large adult male. Both 5-HTP and serotonin (5-HT) are precursors to another neurotransmitter – melatonin – that also induces sleep. 5-HTP, like melatonin, can now be obtained in health food stores. Millions of people have safely taken melatonin for sleep and for eliminating jet lag. Melatonin is produced in the pineal gland deep within the brain, especially at night. Melatonin production is indirectly suppressed by light going into the eye and its levels are directly augmented by the availability of precursors, such as 5-HTP.

5-HTP, stress reduction and aging

The thalamus, is the region in the brain responsible for emotions and for controlling hypothalamic – hormone related – activity. Chemical releasing factors from the hypothalamus direct the adjacent pituitary gland to produce a variety of hormones in all mammals, including man. Pituitary hormones then direct 'endocrine 'glands in the periphery – such as the adrenal glands and the gonads – to produce secondary hormones. Different emotions, situations and behaviors – especially stress – are frequently associated with the bodies production of different hormones.

Over a century ago, the great physiologist Hans Selye observed that stress – in both humans and in experimental animals – resulted in an increased level of the (adrenal) hormone cortisol. Cortisol is responsible for the “fight, flight or fright” response; its elevation produces states ranging from marked wakefulness to panic. Both high cortisol levels and experimental stress reduce levels of brain serotonin. Long-term exposure to cortisol actually damages certain serotonin producing nerves in the brains of animals.

A number of factors are involved in depression in elderly individuals, exposed to a lifetime of various stresses, including an age-related decrease in brain serotonin levels. Elderly persons frequently have difficulty falling to sleep at right as well. Prozac, which raises serotonin levels, is sometimes prescribed for elderly depression for those reasons.

Interestingly, while moderate food restriction in rodents is known to increased lifespan, it also increases levels of the neurotransmitter melatonin – a metabolite of serotonin (and therefore, of 5-
"Stopping Addiction to Sugar: Willpower or Genetics?" We are all programmed to like sugar. New research shows that some of us are genetically much more prone to sugar and food addiction than others. "Mastering Leptin". Byron J. Richards, Board Certified Clinical Nutritionist, is the first to explain the meaning of over 15,000 studies on the hormone leptin and its link to solving obesity.