

# Influencing Your Appetite Pt. II

Emotional Eating



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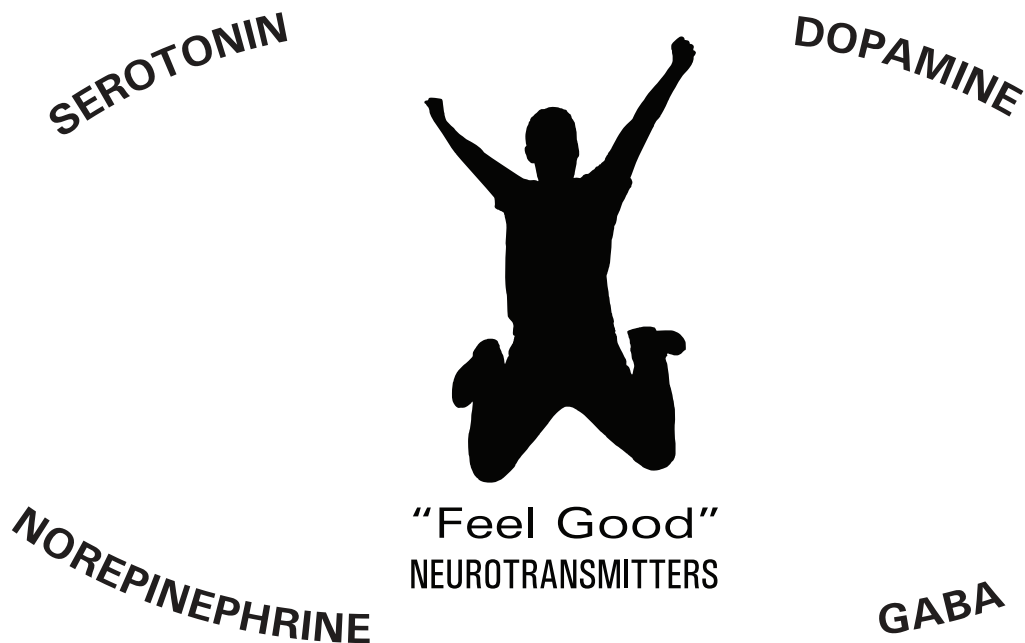
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## What Causes Hunger?

Hunger plays an important biological role in energy balance. Regulation of appetite is mediated by the gut-brain axis, which receives signals of satiation from the adipose tissue, gut response to nutrient sensing, and gut response to stretching (being full). During ideal circumstances, these complex systems can communicate with one another to appropriately regulate energy intake.

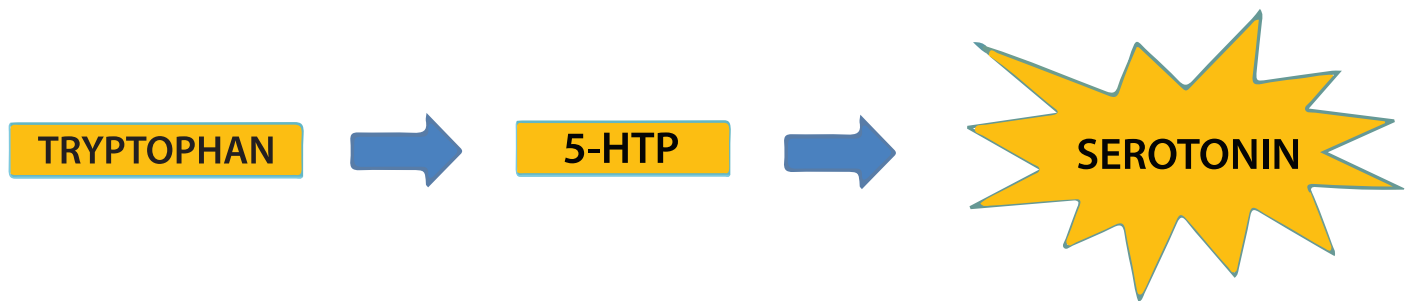
We now are learning that food composition, particularly when high in sugar, can trigger an altered response of these gut hormones, which might amplify the reward pathway and promote continued feeding beyond the point of satiation. This phenomenon is sometimes referred to as **emotional eating**. This distortion in energy-regulation may lead to obesity as well as make it incredibly challenging to make the dietary changes necessary to properly treat obesity and its peripheral conditions.

Our body's reward system is controlled by neurotransmitters, chemical messengers your brain cells use to communicate with one another influencing your behavior. Neurotransmitters regulate a wide variety of processes such as fear, pleasure, joy, anger, mood, memory, cognition, attention, concentration, alertness, energy, sleep, cravings, and appetite. The four major neurotransmitters are **Serotonin, Dopamine, GABA and Norepinephrine**.



## Serotonin

Serotonin is an important neurotransmitter released by the brain. Depression is often considered to be a result of insufficient serotonin. The symptoms of depression include depressed mood, poor quality of sleep, and increased energy consumption (decreased satiety as well as eating to 'feel good') - all of which are functions that are regulated by serotonin.



Tryptophan, an essential amino acid, is a precursor to serotonin. High carbohydrate meals increase the availability of tryptophan in the brain, leading to a flood of serotonin. This is the source of the tryptophan myth. Every Thanksgiving you hear this myth spread by coworkers and family members alike. The truth is that while tryptophan is part of the equation, it is actually the carbohydrates that cause this feeling by removing molecules that would otherwise limit serotonin production.

"The mechanism of the rise in brain serotonin [from carbohydrates] involves the secretion of insulin, and the decrease that the hormone produces in plasma levels of certain amino acids that compete with tryptophan, serotonin's precursor, for transport across the blood-brain barrier. The rise in serotonin can thus be produced by any carbohydrate that elicits insulin secretion, independent of its sweetness." (Wurtman, 1988) In other words, it is not the turkey that is making you sleepy, it's the potatoes and pie!

## Diet's Impact

The neurological impact of our diet is not limited to Thanksgiving. Feeling drowsy after any carbohydrate-rich meal is the effect of serotonin being released. Serotonin can improve one's mood and provide feelings of satiation but it also relaxes the nervous system resulting in cognitive decline and sleepiness. Once these effects fade, you are left craving more carbohydrates.



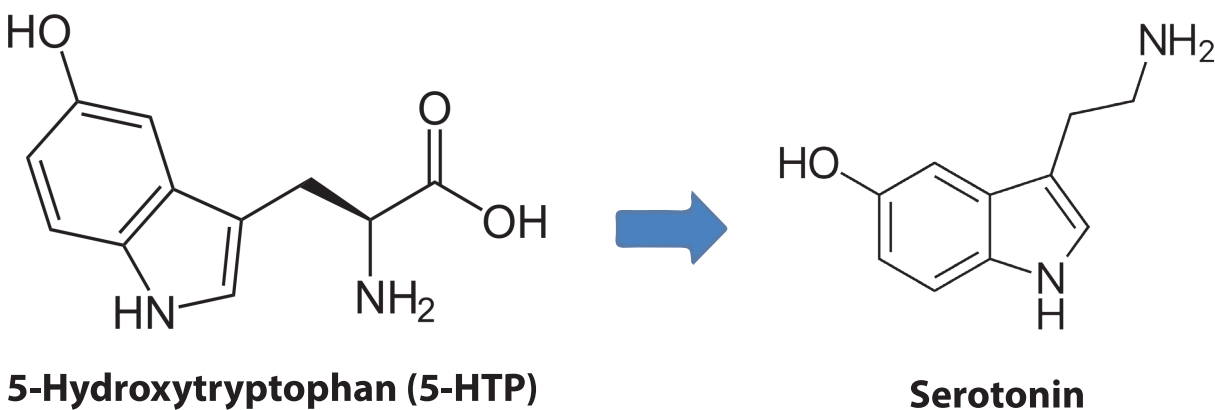
"Carbohydrate consumption--acting via insulin secretion and the 'plasma tryptophan ratio'--increases serotonin release; protein intake lacks this effect" (Wurtman, 1995). This relationship between consumption and neurological signaling leads people to overeat carbohydrates and snack foods for emotional reasons, in response to stress or depression.

This relationship makes it incredibly difficult to give up unhealthy foods and frequently results in obesity. "Patients describe positive subjective responses to the dietary carbohydrates which are unrelated to hunger. These responses may be mediated by the rise in brain serotonin." (Wurtman, 1988)



## 5-HTP

Despite weight gain and changes in cognition, the mood enhancing benefits of serotonin can cause cravings that tempt you to return to unhealthy habits. We may be able to reduce these cravings using supplements that have a positive effect on serotonin, such as 5-HTP.



5-HTP (5-Hydroxytryptophan) is a direct precursor to serotonin. As an intermediate in the tryptophan-serotonin conversion, 5-HTP is not bound by the same limits as tryptophan and thus can be more freely converted to serotonin. One study found that “5-HTP is well absorbed from an oral dose, with about 70 percent ending up in the bloodstream”. The research found that the 5-HTP was then able to cross the blood-brain barrier to increase serotonin. (Birdsall, 1998). This is important because since serotonin cannot cross the blood-brain barrier, it must be produced in the brain.

Furthermore, it was discovered that 5-HTP’s conversion to serotonin is not limited by the enzyme tryptophan hydroxylase, which is the rate-limiting step in traditional serotonin production and is often suppressed by poor diet and insulin resistance, as well as other factors, resulting in insufficient serotonin production.

Using 5-HTP to increase serotonin may help provide satiation for carbohydrate cravings, and make those sweet snacks seem less tempting. The increased serotonin will also help to boost mood, eliminating the need for 'emotional eating'.



## Research

There have been numerous studies that have sought to verify that stimulating serotonin production using 5-HTP can be an effective method to accelerate satiety.

Manipulation of the serotonin pathway was shown to effect feeding behavior in mice, with 5-HTP resulting in “reduction in meal size and a curtailment of eating rate” (Blundell, 1986). “5-HTP brought about a dose related inhibition of food intake” (Blundell, 1979).

“[S]timulating a number of different serotonergic mechanisms can reduce food intake in rats” (Simansky, 1990). “Peripherally administered [5-HTP] also decreases food intake in rats in a behaviorally specific manner (Simanski, 1996).”

During a study performed at the Kobe University in Japan, Leptin, a hormone that regulates satiety, was found to be increased in mice treated with 5-HTP which was directly related to a decrease in feeding. “Serum leptin levels in fasted mice treated with 5-HTP were similar to those control mice after milk intake” (Yamada, 2006). This indicates that 5-HTP can produce a level of satiety similar to that acquired by eating.

## References

1. Wurtman, RJ., Wurtman, JJ. (1995). Brain serotonin, carbohydrate-craving, obesity and depression. *Obesity Research*. Retrieved from: <https://www.ncbi.nlm.nih.gov/pubmed/8697046>
2. Wurtman, RJ., Wurtman, JJ. (1988) Do carbohydrates affect food intake via neurotransmitter activity? *Appetite*. Retrieved from: <https://www.ncbi.nlm.nih.gov/pubmed/2903717>
3. Fischer, K., Colombani, PC., Langhans, W., Wenk, C. (2002). Carbohydrate to protein ratio in food and cognitive performance in the morning. *Physiology & behavior*. Retrieved from: <https://www.ncbi.nlm.nih.gov/pubmed/11897269>
4. Spring, B., Maller, O., Wurtman, J., Digman, L., Cozolino, L. (1982). Effects of protein and carbohydrate meals on mood and performance. *Journal of psychiatric research*. Retrieved from: <https://www.ncbi.nlm.nih.gov/pubmed/6764932>
5. Birdsall, T. (1998). 5-Hydroxytryptophan: a clinically-effective serotonin precursor. *Alternative medicine review : a journal of clinical therapeutic* 3, 271-80. Retrieved from: [https://www.researchgate.net/profile/Timothy\\_Birdsall/publication/13558789\\_5-Hydroxytryptophan\\_A\\_Clinically-Effective\\_Serotonin\\_Precursor/links/09e415086bd3b3d5ab000000/5-Hydroxytryptophan-A-Clinically-Effective-Serotonin-Precursor.pdf](https://www.researchgate.net/profile/Timothy_Birdsall/publication/13558789_5-Hydroxytryptophan_A_Clinically-Effective_Serotonin_Precursor/links/09e415086bd3b3d5ab000000/5-Hydroxytryptophan-A-Clinically-Effective-Serotonin-Precursor.pdf)
6. Blundell, J. (1986). Serotonin manipulations and the structure of feeding behaviour. *Appetite, Volume 7, 39-56*. Retrieved from: <https://www.sciencedirect.com/science/article/pii/S0195666386800514>
7. Blundell, J. & Latham, CJ. (1979). Serotonergic influences on food intake. *Pharmacol Biochem Behav.*, 11(4), 431-7. Retrieved from: <https://www.ncbi.nlm.nih.gov/pubmed?Db=pubmed&Cmd=ShowDetailView&TermToSearch=316547>
8. Simansky, KJ. & Vaidya, AH. (1990). Behavioral mechanisms for the anorectic action of the serotonin uptake inhibitor... *Brain Res Bull.*, 25(6), 953-60. Retrieved from: <https://www.ncbi.nlm.nih.gov/pubmed?Db=pubmed&Cmd=ShowDetailView&TermToSearch=2149668>
9. Simansky, KJ. (1996). Serotonergic control of the organization of feeding and satiety. *Behav Brain Res.*, 73(1-2), 37-42. Retrieved from: <https://www.ncbi.nlm.nih.gov/pubmed?Db=pubmed&Cmd=ShowDetailView&TermToSearch=8788474>
10. Yamada, J., Sugimoto, Y., Ujikawa, M. (2006). Involvement of leptin in hypophagia induced by the serotonin precursor 5-hydroxytryptophan (5-HTP) in mice. *Biol Pharm Bull.*, 29(3), 557-9. Retrieved from: <https://www.ncbi.nlm.nih.gov/pubmed?Db=pubmed&Cmd=ShowDetailView&TermToSearch=16508167>
11. Donovan, M. & Tecott, L. (2013). Serotonin and the regulation of mammalian energy balance. *Frontiers Neuroscience*, 7, 36. Retrieved from: <https://www.frontiersin.org/articles/10.3389/fnins.2013.00036/full>
12. Guyton, A.C. & Hall, J.E. (2000). *Medical Physiology*. Philadelphia, PA: Saunders.

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