The Synapse: Intercollegiate science magazine

Volume 17 | Issue 1 Article 11

2018

The Truth on Seratonin: For A Sound Mind and Healthy Mood

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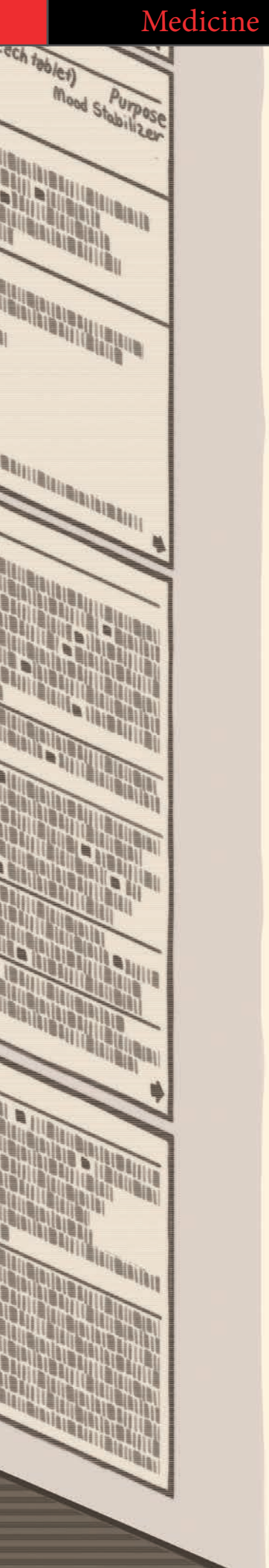
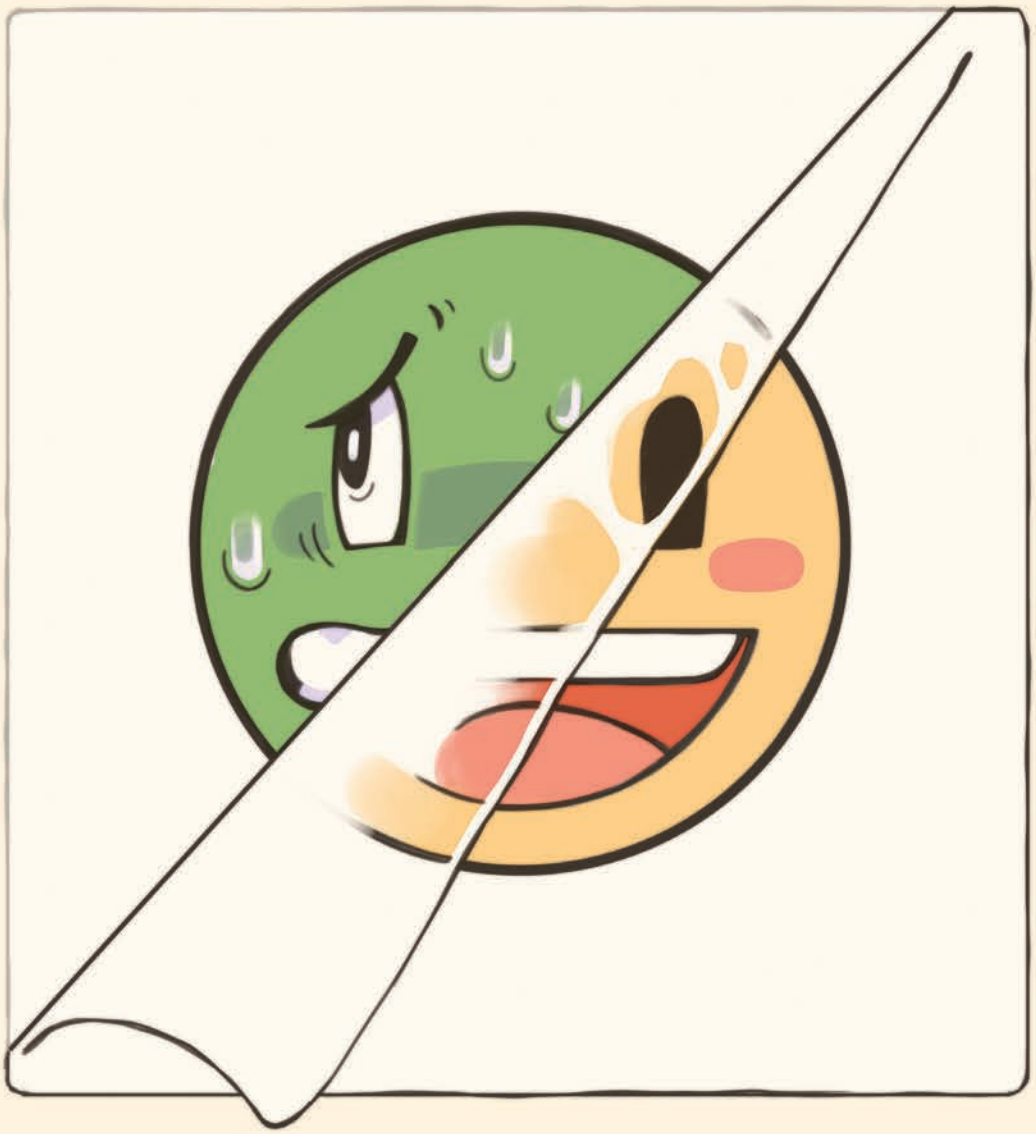


THE TRUTH ON SEROTONIN*

100 mg 70 Capsules Dietary Supplement

For a Sound Mind and Healthy Mood™

*MISCONCEPTIONS AND THE REALITY ABOUT THE NEUROTRANSMITTER AND ITS FUNCTION



- Doctor-approved, attuned to ensure and protect the body's natural balance of neurotransmitters.
- Scientifically proven to improve mood, reduce stress, and promote a sound mind and body.

ARTICLE by Rajitha Narreddy
ART by Roger Ort

I remember reading a New York Times article a while back, entitled 'Prozac Nation is now the United States of Xanax'. It was about the explosion of anxiety disorders in not only the general population, but also in college students. The number of Google searches about anxiety has doubled in the last fifty years while the number of searches about depression has remained constant. According to the National Institute of Mental Health, 38% of girls and 26% of boys between the ages of 13 and 17 have an anxiety disorder. On college campuses, anxiety is the most common mental health concern.

Selective Serotonin Reuptake Inhibitors (SSRIs) are often the first line of defense against anxiety disorders, after therapy. This has lead to the general perception that the cause of anxiety disorders is a lack of serotonin in the brain. However, most recent studies have shown that this is not the case. And because SSRIs are so widely used, this misperception is problematic.

Serotonin is a neurotransmitter, which is one of the ways in which neurons communicate with each other. Neurons release neurotransmitters into synapses, which are the gaps in between neurons. The neuron that releases the neurotransmitter into the synapse is called the presynaptic neuron and the neuron that the neurotransmitter is "talking" to is called the postsynaptic neuron. Serotonin is commonly thought of as one of the "happy chemicals," but its effects are very complicated.

SSRIs increase the concentration of serotonin in synapses. Generally, after a presynaptic neuron releases a neurotransmitter, there needs to be a mechanism in place to "clean up" the synapse. Otherwise, synapses would be clogged up with neurotransmitters. One of the ways that neurons clear the synaptic cleft is by reabsorbing serotonin into the presynaptic neuron through a process is called reuptake. SSRIs increase concentration of serotonin in synapses by preventing the presynaptic neuron from reabsorbing the serotonin in the synapse. They block the serotonin reuptake transporter, so there will be more serotonin in synapses.

While SSRIs were originally intended to help with depression, they are now used to help with a host of conditions including anxiety disorders. Oftentimes, the general public takes this to mean that anxiety stems from a decreased concentration of serotonin in the brain. This, however, is an oversimplification which many recent studies have shown to be false. The latest research suggests that in the short term, increased concentrations of serotonin actually increase anxiety even though long term increases in levels of serotonin in the brain do help decrease anxiety.

> While SSRIs are effective in treating many anxiety disorders, we don't really have a clear understanding of how they work.

One explanation of why this may be the case can be explained by looking at how SSRIs affect people who take them. SSRIs generally take a few weeks to begin working. The lack of immediate effects indicates that there is not a direct correlation between higher levels of serotonin and decreased anxiety. Furthermore, the initial side effects of SSRIs look a lot like an an increase in anxiety levels! According to the UK's National Health Service (NHS), side effects of SSRIs include "feeling agitated, shaky or anxious; insomnia or drowsiness; suicidal thoughts; and a desire to self harm," and these side effects typically decrease over time.

A recent study (Marcinkiewcz et al., Nature, 2016) looked at the neurocircuitry of serotonin in mice and also found results that align with this idea that serotonin initially increases anxiety level before mitigating anxiety. The researchers found that both SSRIs and paw-shocks to mice increase symptoms of anxiety with an increase in serotonin levels. Serotonin is produced in the dorsal raphe which projects into the Bed Nucleus of the Stria Terminalis (BNST). In rats, the increased serotonin interacts with and inhibits a different neural circuit in the BNST that silences anxiety decreasing outputs. The BNST projects to the ventral tegmental area (VTA) and lateral hypothalamus. These areas are associated with mood and relieving anxiety. When the BNST is inhibited, these areas are also inhibited. All of this together means that increases in serotonin increase anxiety. While we don't have concrete evidence that this pathway exists in humans, there are surprising similarities between rodent brains and human brains. Through further study, we may be able to better understand how this process works in humans.

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Researchers have also found that people with anxiety disorders, specifically panic disorder, have a decreased density of 5-HT1A receptors, which inhibit serotonin release. Because of this, we understand that people with anxiety disorders likely have an increased level of serotonin in their brains. There is also an inverse relationship between the density of 5-HT1A receptors and amygdala activation. The amygdala is involved with the processing of emotions. This suggests that people with anxiety disorders not only have an increased concentration of serotonin, but that one of the causes of this increased concentration is correlated with an increase in emotional perception.

So, why do SSRIs work to decrease anxiety in the long term? The short answer is that we really don't know. One theory is that increases in serotonin lead to an increase in neurogenesis, or the making of new neurons. Until relatively recently, it's been thought that after childhood, it is impossible for human brains to make new neurons. However, more recently, scientists have found that this is not true. While adult humans have a very limited capacity for neurogenesis, it still does happen. There is some evidence that suggests that SSRIs and increases in serotonin lead to an increase in neurogenesis in the hippocampus. The hippocampus is involved in short term memory regulation and emotion processing, so it is possible that the increase in neurons in the brain can allow for improved emotional processing, decreasing anxiety symptoms.

While SSRIs are effective in treating many anxiety disorders, we don't really have a clear understanding of how they work. What we do know is that their effects are very complicated and that there are many common misperceptions of how serotonin interacts with anxiety.

The next step to fully understand the relationship between serotonin levels and anxiety is to test drugs to see if they can change the way that this circuit works. One possible option is to find a drug that targets proteins in the BNST. That way, we could directly target the part of the brain that the serotonin acts on to increase anxiety in the short term. By taking this drug with the SSRI for the first few weeks, we could combat the initial side effects. This is especially important since one of the big problems with SSRIs is noncompliance. One study determined that almost 50% who have been prescribed SSRIs stopped taking them within 60 days. By decreasing side effects, we can help decrease noncompliance rates and help the large number of people with mental illness feel better. ●