Serotonin and candida are unusually linked. The happiness hormone, Serotonin, is not something you expect to see connected to Candida albicans. It is something we talk about more in relationship to brain dysfunction and mood disorders. Serotonin and candida is something we don’t hear much about.

If we consider that 80-90% of the production of the body’s serotonin happens in the gastrointestinal tract, the distance between the two narrows significantly and a possible relationship between the two makes more sense.

Why the GI Tract produces so much serotonin is not completely known yet (motility and appetite are two effects), but a functional link between serotonin and candida has been established, and it’s not about serotonin making candida happy.
Scientists from Austria have shown that serotonin has anti-fungal activity against candida, helping to control its ability to change into its problematic fungal form and the activity of enzymes produced by it. Depending on the concentration of serotonin present, at lower concentrations, it can first interfere with the activity of candida’s enzymes, and at higher concentrations, it can significantly limit its fungal growth.

It has been shown that the bacteria of the intestinal tract play a role in the production of serotonin and since fungal candida effectively regulates and alters bacterial ratios in the gut after antibiotic use, just as serotonin affects it, it can affect serotonin. In short, gut bacteria regulate serotonin levels and candida regulates gut bacteria. Serotonin and candida are linked.

Also

Another factor is that fungal candida increases inflammation throughout the body and this has been shown to play a role in neurological conditions such as depression and anxiety.
Research from King’s College in London shows that inflammation is often linked to diabetes and depression. Researcher, Dr. Khalida Ismail states, “Inflammation may be driving a number of different long-term conditions. That’s quite a new way of thinking of the mind and the body”.

Fungal candida can also play a role in creating diabetes through its pro-inflammatory effects and the induction of the body’s TH-17 immune response. The creation of blood sugar imbalances is associated with depression and depressive behaviors.

These effects can once again be linked to an increase in brain inflammation associated with hypoglycemia and diabetes. Researchers at the University of Washington found that depression was “significantly associated with...hypoglycemia.”

Fungal candida is implicated in conditions such as MS, CFS, ME, arthritis, psoriasis, and other autoimmune diseases by researchers in Germany and Switzerland. The primary factor in the relationship between candida and depression, as well as a long list of other conditions, is inflammation.

**Antibiotics**

Many of these effects can be traced back to antibiotic use and it’s not the abuse of antibiotics, it’s the simple use of antibiotics. In killing 100 trillion bacteria in the body within 5 to 7 days, antibiotics cause a massive flooding of the body’s tissues with bacterial cell components that humans are highly allergic to. This can prime the body and the brain for a lifetime of inflammation and disease. Fungal candida results from antibiotic use and drives a lot of inflammation in the body linking it to over 125 different conditions.

Of course, not all is lost and the body can be brought back into balance through the application of sound principles and an understanding of the body’s physiology and systems, microbiome, and fungal mechanics. Get started today on Dr. McCombs Candida Plan to re-experience a life of vitality! When all else fails, remember to laugh, as laughter stimulates the release of serotonin, which in turn inhibits candida.

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INTRODUCTION
Several years ago antimicrobial activity was described for psychotropic drugs of the phenothiazine and thioxanthene groups (Brown, 1975). Since then, several non-antibacterial substances have been examined and it has been reported that selective serotonin re-uptake inhibitors (SSRIs) influence the in vitro viability of bacteria (Cederlund & Mardh, 1993; Munoz-Bellido et al., 1996, 2000) and may reverse chloroquine resistance in Plasmodium falciparum (Coutaux et al., 1994). These drugs have significant antimicrobial activity, mainly against Gram-positive bacteria, yet they are inactive against most enteric Gram-negative bacteria (Munoz-Bellido et al., 2000).

Recently, we found that sertraline, a typical SSRI has in vivo and in vitro antifungal activity (Lass-Flörl et al., 2001a, b). Since fungicidal effects were observed at high concentrations, immunomodulatory effects or several modifications of fungal virulence by SSRIs were more likely to explain the in vivo outcome in our patients. In humans, SSRIs modify the behaviour of 5-hydroxytryptamin (5-HT) and act primarily on the 5HT transporter protein (SERT) (Schloss & Williams, 1998). A block in the re-uptake process of 5 HT causes an increase in 5 HT during therapy with SSRIs (Dammock et al., 2000). This fact and the clinical phenomenon found in our patients (Lass-Flörl et al., 2001a) led us to examine the potential antifungal role of 5 HT. We determined the direct influence of 5 HT on the viability of clinical isolates of Candida spp. and studied whether delayed regrowth as a post-antifungal effect follows short exposure to 5 HT. 5 HT showed antifungal activity towards all isolates of Candida spp. The isolates yielded comparable MIC and MFC values of 5 HT in the range 0.01–1.34 mM and 1.83–14.68 mM, respectively. A lag in regrowth was dependent on the concentration tested. Treatment for 3 h at concentrations of 5 HT below and equivalent to the MFC resulted in a delayed regrowth of 8–12 h for isolates of Candida spp. In conclusion, these in vitro studies clearly demonstrate antifungal effects of 5 HT. Identifying the mode of action could be of great help in developing and researching new antifungal drugs.

METHODS
Strains. The in vitro tests were performed on clinical isolates of Candida albicans (n = 10), Candida glabrata (n = 9), Candida tropicalis (n = 10) and Candida parapsilosis (ATCC 22019). Isolates were maintained as suspensions in sterile water at room temperature and subcultures were grown on Sabouraud glucose agar (Merck) incubated at 35 °C for 2 days.

Drug. According to the manufacturer’s instructions, 5 HT (M, 212) Sigma was dissolved and further diluted in sterile water (Fresenius) final concentrations were 0.92 μM–0.22 mM.

Broth microdilution test. Isolates were tested using the microbroth dilution method according to the National Committee for Clinical Laboratory Standards (1997) guidelines. A fungal inoculum size of 6 × 10^6–6 × 10^7 CFU ml^-1 was used. A total of 100 μl of each of the drug dilutions was added with 100 μl of the fungal suspensions, and the mixture was incubated at 35 °C and evaluated after 24 h for growth. The minimal inhibitory concentration (MIC) end point criterion was the lowest drug concentration showing no visible growth after 24 h of incubation. To obtain the minimal fungicidal concentration (MFC), 100 μl volumes were taken from each well and spread on Sabouraud Glucose Agar (Merck). The number of CFU was counted after