The Facts of Antidepressant Target Serotonin and Its Role in Reducing Depression

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Letter to the Editor

Modern day people are subjected to great stress irrespective of the work they do and many reasons add to this. Stress was an important factor in the past as well, but the intensity is growing more in the recent days and is taking the form of depression. Psychiatric drugs play an important role in treating stress, depression and many other mental ailments. Lot of research is being done in this field, leading to the designing of new drugs with better effect. Hence, our objective in this article is to bring focus on the recent findings of the transport protein structures and their significance in discovering new drugs.

Keywords

Serotonin; Antidepressants; Depressants; Drugs

Introduction

Neurotransmitters are the key molecules in regulating the proper functioning of the central nervous system. Serotonin is one such important neurotransmitter that regulates the psychological and physiological functions as well. Serotonin is a potent neurotransmitter required for proper transmission of signals within the neurons. It has been considered as the key factor in mood imbalances and also depression. Major proportion of this neurotransmitter is synthesized more in the recent days and is taking the form of depression. Psychiatric drugs play an important role in treating stress, depression and many other mental ailments. Lot of research is being done in this field, leading to the designing of new drugs with better effect. Hence, our objective in this article is to bring focus on the recent findings of the transport protein structures and their significance in discovering new drugs.

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Introduction

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Chahrour M et al, 2008 [1] revealed that, mutations in the gene encoding the transcriptional repressor methyl-CpG binding protein 2 (MeCP2) have caused the neurodevelopmental disorder Rett syndrome. They’ve also reported the tiss of function as well as increased dosage of MECP2 gene causing a host of neuropsychiatric disorders. To explore this, molecular mechanism (s) underlying these disorders, the researchers examined gene expression patterns in the hypothalamus of mice that either lack or over express MeCP2. In both models, MeCP2 dysfunction induced changes in the expression levels of thousands of genes, but to their surprise, the majority of genes (~85%) appeared to be activated by MeCP2. This led to delve more into a research of this kind.

Beuten J et al, 2005 [2] in their study reported that, Brain-Derived Neurotrophic Factor (BDNF) influences dopamine and serotonin neurotransmission in the brain, both of which are involved in the reward system of addiction. They’ve also said, the significant association of BDNF variants with ND (Nicotine Dependence) implies that this gene plays a role in the etiology of ND in EAs and that its involvement is gender specific. BDNF may warrant further investigation in ND.

Bowel activities, Aiding formation of blood clots, Nausea, bone density are the major biological events being regulated by the levels of serotonin in human body. These neurotransmitters are reabsorbed back into the nerve cells of the brain and this process is called as reuptake. The antidepressants are a group of reuptake inhibitors which termininate the reabsorption of serotonin and thereby helps retaining back the neurotransmitter within the nerve connections called the synapses, which is a key step in keeping a check on depression.

Irving Kirsch [3] in his study said that, Antidepressants are supposed to work by fixing a chemical imbalance, specifically, a lack of serotonin in the brain. Indeed, their supposed effectiveness is the primary evidence for the chemical imbalance theory. When on that, he narrated, the serotonin theory is as close as any theory in the history of science to having been proved wrong. Instead of curing depression, popular antidepressants may induce a biological vulnerability making people more likely to become depressed in the future.

Well, these few lines definitely create some interest to put our pen and paper in this area of research. Am I right?

Past research showed that, retaining back neurotransmitters within the synapses makes the communication between the neurons stronger which further prevents depression. There are many selective serotonin reuptake inhibitors which are being used as the potent antidepressants.

Therese Brochard [4] has this very important piece of information. In an article published by her, she gave us an important data. She took an interview with Peter J Kramer MD, Brown University. These are her excerpts.

“It’s true that depression is not a serotonin deficiency”, Peter J. Kramer, MD, told me in an interview, “but it’s also true that serotonergic drugs allow for recovery from depression, and sometimes they seem to reverse it directly”. Dr. Kramer is clinical professor of psychiatry and human behavior at Brown University and the bestselling author of six books, including Listening to Prozac.

During that time, she also spoke with Ron Pies, MD, professor
of psychiatry at SUNY Upstate Medical University and author of Psychiatry on the Edge.

These are her excerpts.

“There is little question that the role of serotonin in depression was over-emphasized and over-marketed in the 1990s”, Dr. Pies explained to me, “though most psychopharmacologists understood that the neurobiology of depression was much more complicated. Indeed, the term ‘SSRI’ is itself a misnomer, since some of these agents also affect other brain chemicals (e.g., sertraline has mild effects on dopamine). None of this, however, should be used in service of the equally mythological claim that ‘antidepressants don’t work’ or are no better than a sugar pill’. This is demonstrably false, at least with respect to moderate-to-severe depression”.

Conclusion

Allosteric sites of the transport proteins, where antidepressant drug molecules bind to, have brought into focus, the additional drug target sites of these proteins. This is the significant finding in this research which added a possibility in designing new anti-depressants.

The mental strength from yoga quickened the healing process of my fracture. I started feeling confident and told myself ‘I am normal and fit’. I slowly began leading life happily after meeting my psychiatrist and after she advised me to go for an early walk and perform yoga. Now, after all the stress and pain, thanks to my doctor, I am in a position lead my life the beautiful way it once was. Yoga, Meditation, and a walk in the morning and evening helped me in maintaining my concentration and always kept my optimism high. Physical exercise kept me in good shape and finally brought me back into leading normal life.

My personal experience inspires me in leaving the comfort bed, and makes me realize the importance of physical activity in daily life. I hope to be happy, healthy and peaceful always.

References


