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Serotonin Receptors Involved in Neuropsychiatric Disorders

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DESCRIPTION

The serotonergic system spans the central nervous system and the gastrointestinal tract. In the central nervous system, serotonin regulates a wide range of functions including mood, cognition, anxiety, learning, memory, reward processing, and sleep. Defects in the serotonergic system can lead to various pathological conditions, especially depression, schizophrenia, mood disorders and autism. In this review, we have reviewed the complexities of serotonergic regulation of physiological and pathological processes. In addition, we provided experimental and clinical evidence for the involvement of serotonin in neuropsychiatric disorders and discussed the molecular mechanisms underlying these diseases and their contribution to new therapeutics. The neurotransmitter serotonin has been implicated in the pathophysiology and treatment of major depression since the recent serendipitous discovery of antidepressants. Despite the widespread use of serotonin-raising drugs, such as selective serotonin reuptake inhibitors and dual serotonin and norepinephrine reuptake inhibitors, the precise neurobiological mechanisms responsible for the therapeutic effects of these drugs is not well understood. Increasing our knowledge of these mechanisms will allow us to identify new therapeutic targets and overcome two major limitations of current therapies.

Mental disorders represent a major economic burden in modern society. However, pharmacological treatment is still suboptimal. A drug used to treat major depressive disorder and anxiety disorders. Serotonin-norepinephrine reuptake inhibitors are pharmacological modifications of the serendipitously discovered first-generation tricyclics that have shown lower potency and slower onset of action. Furthermore, antipsychotics are partially effective for positive symptoms of schizophrenia but are inadequate for treating negative symptoms and cognitive deficits. This article provides an overview of the neurobiological basis of serotonin receptor function and the role of pre and postsynaptic serotonin in the treatment of major depressive disorder, anxiety and psychiatric disorders. Activation of postsynaptic serotonin in corticolimbic regions appears to be beneficial for the therapeutic effects of antidepressants. However, presynaptic serotonin plays an unfavorable role in major depressive disorder, as individuals with high presynaptic serotonin density or function are more prone to mood disorders and suicide, and less responsive to antidepressants. Furthermore, indirect activation of presynaptic serotonin reduces serotoninergic neuron activity and terminal serotonin release, resulting in extracellular serotonin produced by forebrain serotonin transporter blockade. Offset the increase. Chronic antidepressant treatment desensitizes presynaptic serotonin; thereby reducing the effectiveness of serotonin auto receptor mediated negative feedback. Several studies in transgenic mice have also identified the respective roles of presynaptic and postsynaptic serotonin in major depressive disorder and anxiety. Consistent with pharmacological studies, activation of presynaptic and postsynaptic serotonin receptors appears to be required for anxiolytic and antidepressant effects, respectively, although neurodevelopmental roles for serotonin are also likely. Postsynaptic serotonin in the prefrontal cortex also appears to be important for the superior clinical efficacy of clozapine and other second-generation antipsychotics in the treatment of schizophrenia and related psychiatric disorders. It seems. Although clozapine exhibits moderate *in vitro* affinity for serotonin in binding assays, clozapine exhibits functional agonist properties at this receptor type *in vivo*. Stimulation of serotonin in the prefrontal cortex results in distal activation of mesocortical pathways and increased dopamine release in the prefrontal cortex. This effect may be responsible for the clinical effects of clozapine on negative symptoms.

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CONFLICT OF INTEREST

The author's declared that they have no conflict of interest.

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