RESEARCH ARTICLE

Effects of Ziprasidone in the Brain among Adolescent Versus Adult Schizophrenic Patients

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Abstract

The study explores the neurochemical and structural mechanisms underlying schizophrenia, focusing on the dysregulation of D2 and 5-HT2A receptors within mesolimbic and cortical pathways, their contributions to the disorder's symptoms, and how the antipsychotic medication ziprasidone affects adolescents compared to adults. This paper analyzes how neurotransmitter imbalances influence cognitive behavior and emotional functioning, while evaluating treatment efficacy across different developmental stages. Synthesizing established scientific literature, clinical findings, and medical resources from the National Library of Medicine (NIH), the study examines core neurobiological processes and treatment outcomes. Findings indicate that hyperactive dopamine and serotonin signaling is strongly associated with positive symptoms, such as hallucinations and paranoia, whereas structural brain changes are linked to cognitive and negative symptoms. Ziprasidone helps regulate these imbalances by acting as a receptor antagonist. However, side effects are more pronounced in adolescents, while adults generally exhibit better tolerance. Ultimately, this paper emphasizes the importance of age-specific treatment strategies and highlights the need for continued advancements in psychopharmacology and comprehensive patient care.

Keywords

Neuroscience, Neurobiology, Receptors, Ziprasidone, Adolescents

Introduction

Schizophrenia is a severe mental illness that affects approximately 0.5% of the total population in the United States.[1] Patients diagnosed with schizophrenia frequently exhibit reduced volumes of gray matter within the left hemisphere of the frontal and temporal lobes.[2] Gray matter contains the majority of the brain's neuronal cell bodies, which are responsible for processing and relaying signals throughout the central nervous system. The loss of these neurons causes disruptions to the brain's information-processing capabilities, leading to symptoms such as hallucinations, executive dysfunction, deficits in attention and concentration, slowed cognitive processing speed, catatonic or unusual motor behavior, and disorganized thought and speech.[3] Although there is no known cure, researchers have developed second-generation antipsychotics, such as ziprasidone (commonly known as Geodon), to help manage the disorder. While ziprasidone is considered an effective therapeutic option, its physiological and psychological effects in adolescents differ significantly from those observed in adults.

A recent study by the National Institutes of Health evaluated the effects of ziprasidone in adolescents with schizophrenia and found that the drug failed to demonstrate significant separation from the placebo, indicating minimal efficacy in this age group. In contrast, ziprasidone demonstrated clear efficacy in adult populations, suggesting that the medication requires further modification and continued research to effectively treat younger patients. Mechanistically, ziprasidone binds to 5-HT and D2 receptors, inhibiting the hyperactive serotonin and dopamine signaling that typically drives positive symptoms such as hallucinations. However, because the adolescent brain is still maturing, dynamic shifts in neurodevelopment and baseline neurotransmitter activity can cause fluctuations in the drug's effectiveness when these receptor pathways are blocked.

It is absolutely critical that the effects of ziprasidone are thoroughly studied before they are provided to juveniles ages 13–17, as their brain, most specifically the prefrontal cortex, is still underdeveloped and does not function the same as an adult brain. Since the frontal lobe is responsible for major parts of an adolescent's development, the uncertainty of its side effects as well as its effectiveness due to lack of proper research could potentially be detrimental for the patients that are being treated with it. Although schizophrenia within juveniles is scarce and doesn't make up a large part of the population, they are affected by this disorder just as much as adults, if not even more.

This difference in developmental stages between the two groups, combined with the limited amount of research currently available in this field, can lead to a range of challenges regarding the performance and reliability of the drug during the periods in which it is prescribed. Variations in brain development, metabolism, and overall physiological responses may influence how the medication functions in adolescents compared to adults, potentially affecting both its efficacy and safety.

The objective of this paper is to examine a broader scope of existing research on the effectiveness of ziprasidone, with a particular focus on its impact in younger patients. Additionally, this paper aims to provide a comprehensive literature review of the available data on the side effects associated with schizophrenia treatments, highlighting any differences observed between age groups and identifying gaps that warrant further investigation.

Methods

The study uses a literature review to examine the neurochemical and functional effects of ziprasidone in the prefrontal cortex in adolescents compared to adult patients diagnosed with schizophrenia using qualitative methods of data collection. The principal purpose of this paper is to determine developmental differences in drug response that could potentially influence the efficacy of the treatment. The paper consists of 14 systematically collected, relevant clinical studies and peer-reviewed articles from academically reputable databases including PubMed, Google Scholar, and the National Library of Medicine. The keywords used to find articles were: "ziprasidone," "ziprasidone side-effects," "ziprasidone adolescents," "schizophrenia symptoms," "schizophrenia symptoms adolescents," and "ziprasidone functions."

There were no physical tools, experiments, or materials involved during the research process. The 14 sources were selected based on the impact of ziprasidone on neurotransmission activity, specifically dopamine and serotonin receptors, and age-related variations in brain development, primarily prefrontal cortex function. All papers cited range from the year 1999 to 2026. This paper analyzes data from studies comparatively to identify patterns in treatment response between adolescents and adults. The primary idea resides in developmental neurobiology, recognizing that the adolescent brain undergoes ongoing maturation, which may result in variations in neurotransmitter regulation and receptor sensitivity. The factors listed above were considered when interpreting differences in the efficacy of ziprasidone and its side effects.

Results and Discussion

Primary Neurochemical Effects of Schizophrenia

Schizophrenia is a severe mental illness that affects how an individual thinks, feels, and responds to the environment around them. There are multiple different stages of schizophrenia as well as different types of symptoms. Individuals who have schizophrenia suffer from a loss of both white and gray matter, which play critical roles in the functionality of the brain.

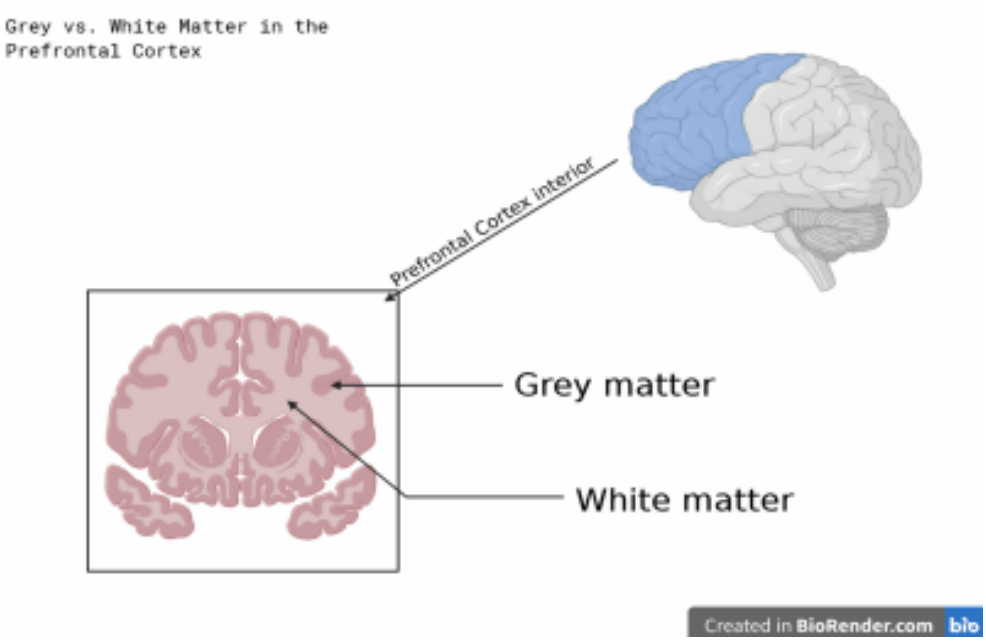

Figure 1. Grey Matter vs. White Matter Diagram. Gray matter resides within the brain's central nervous system, mainly found on the outer layer of the brain. It contains a high concentration of neurons, which are in charge of relaying information to and from the brain. Gray matter, in particular, is in charge of processing information, reacting to stimuli, and regulating emotions.8 White matter, on the other hand, resides within the brain and enables cognitive processing, learning, as well as motor functionality in various parts of the central nervous system. Source. Made with BioRender.

A decrease in gray matter within the following anatomical areas, as presented in Figure 1, in schizophrenic patients results in dissonance within the brain's ability to process information, leading to symptoms such as hallucinations, poor executive functions, attention and concentration deficit, slow mental processing speed, unusual physical behavior, and disordered thoughts and speech. A reduction in white matter would cause a decline in an individual's manual dexterity and overall physical functionality. This could result in muscle spasms and attention deficits, all leading to symptoms of schizophrenia.

Methods of Diagnosis

Most patients are diagnosed after a psychotic break, occurring at different periods for men and women. The symptoms of schizophrenia vary depending on the person; however, they can be broken into three main parts: positive symptoms, negative symptoms, and cognitive symptoms. Positive symptoms are phenomena that do not usually occur within mentally stable individuals; these include delusions, hallucinations, unusual physical behavior, and disordered thoughts and speech. Negative symptoms consist of qualities that are lost within those affected by schizophrenia; these symptoms cause a patient to lose motivation, emotional expression, speech abilities, and sociability. Cognitive symptoms include neurological limits that affect daily life functions for the affected individuals; these include poor executive functions, attention and concentration deficits, slow mental processing speed, as well as difficulty recognizing emotions in conversations and picking up on social cues.

The Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition—a manual used for psychological and mental illness diagnoses—used to contain five different forms of schizophrenia: paranoid type, disorganized type, catatonic type, undifferentiated type, and residual type. Paranoid type schizophrenia is when the patient is experiencing delusions as well as auditory hallucinations once or on multiple different occasions. Symptoms for disorganized type schizophrenia consist of disorganized speech and behavior, along with a severe reduction in emotional expression and inappropriate emotions

toward the situation. Catatonic type involves the repetition of often bizarre movements, words, and phrases, extreme negativity, and little to no emotions or speech being conveyed. Undifferentiated type is defined by significant motor abnormalities, such as catatonic stupor, the maintenance of unusual body positions, or episodes of agitated, non-goal-directed motion. It's essentially a mix of paranoid, disorganized, and catatonic; however, it does not fit any one completely. Residual type schizophrenia is diagnosed in those who have a history of psychosis and negative symptoms.

In the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, however, the different subsections of schizophrenia are not used nor appointed in the diagnosis process, making schizophrenia easier to identify as well as allowing equal methods of treatment for all individuals affected.

Chemical Effects of Schizophrenia

Schizophrenia is mostly the result of an increase in dysfunction within the mesolimbic pathway, as the pathway becomes disrupted when schizophrenia develops.[1] The mesolimbic pathways are responsible for synthesizing and carrying dopamine throughout the brain, and they become hyperactive when an individual begins to develop schizophrenia. Additionally, the D2 receptors and 5-HT2A receptors within the brain are also affected by the disorder. The D2 receptors are mostly located in the striatum and are in charge of regulating dopamine—the chemical that regulates motivation and memory—synthesis, activity, release, and absorption. The 5-HT2A receptors reside mainly in the cerebral cortex and are in charge of releasing glutamate, which strengthens connections between neurons, and serotonin, which regulates an individual's overall mood.[11]

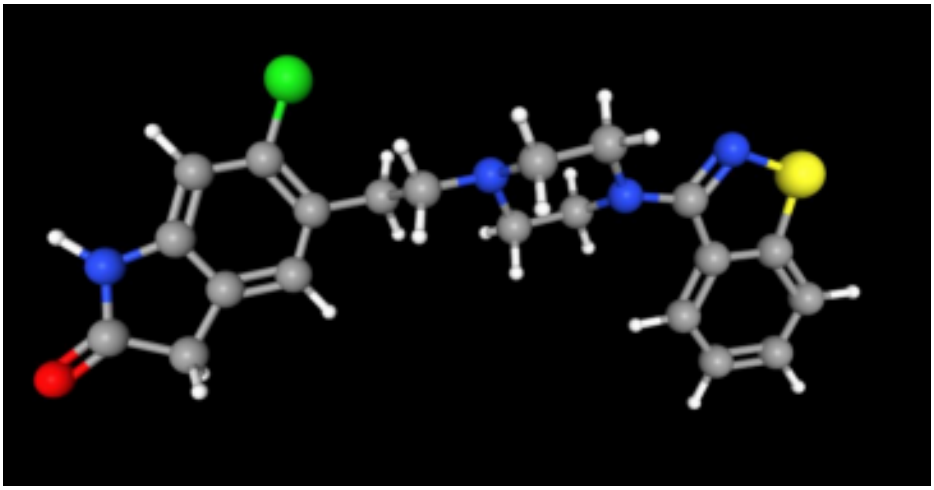

Figure 2. Chemical Anatomy of Ziprasidone. Chemically, ziprasidone is a serotonergic antagonist as well as a dopaminergic antagonist, meaning it contains properties that restrict the production of serotonin and dopamine, respectively. Ziprasidone binds to the D2 receptors inside the mesolimbic pathways, latching onto the site so that it inhibits the protein. Ziprasidone also binds to the 5-HT2A receptors, which decreases the output of serotonin.[21] Source: National Center for Biotechnology Information

When a patient develops schizophrenia, their D2 and 5-HT2A receptors become overly active, releasing more dopamine and serotonin than necessary.[12] When the brain excessively increases its generation of dopamine, it causes loss of impulse control, insomnia, and paranoia due to the excessive cognitive load. An excessive increase in serotonin can cause agitation, anxiety, and muscle spasms. These symptoms are due to overstimulation of 5-HT2A receptors within the central nervous system. These shifts within the brain's functionality can be treated with ziprasidone. Ziprasidone is a second-generation antipsychotic that is used to treat schizophrenia as well as other disorders. It treats the disorders by balancing dopamine and

serotonin levels within the brain.

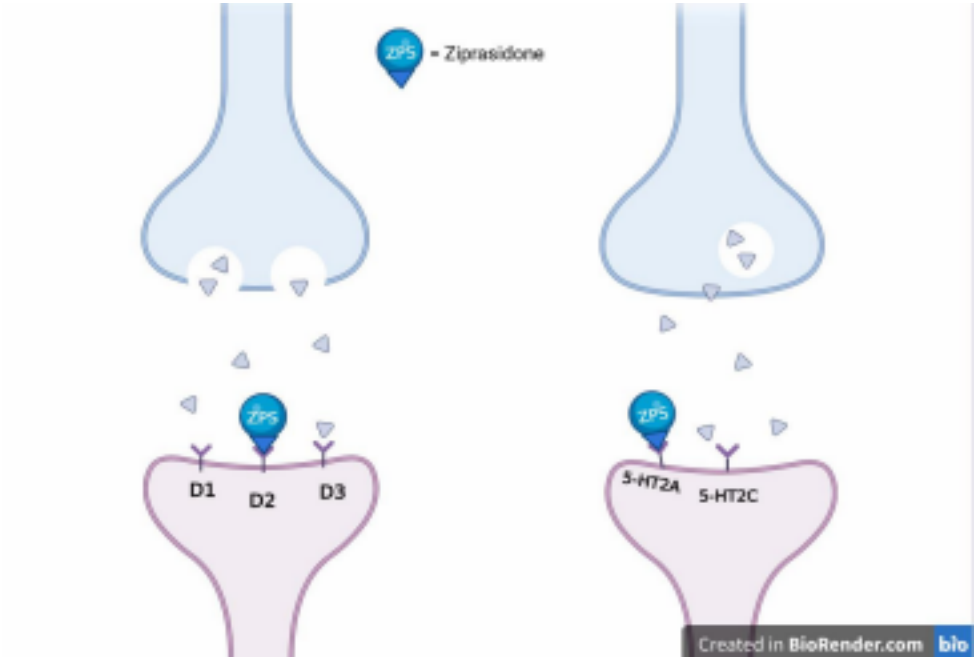

Figure 3. Ziprasidone Effect on Receptors**.** The D2 and 5-HT2A receptors are the most affected receptors when an individual develops schizophrenia. Ziprasidone attaches to the binding sites in the receptors, restricting as well as regulating the flow of dopamine and serotonin neurotransmitters. Source. Made with BioRender

Differences in Side-Effects between Adolescents and Adults on Ziprasidone

Ziprasidone is used for treating schizophrenia for a large number of reasons. If a patient begins to show signs of agitation, paranoia, or inability to sleep, they have a large chance of being put on ziprasidone. Due to the ongoing development of the prefrontal cortex, adolescents exhibit altered pharmacodynamic responses compared to adults. The symptoms of schizophrenia within the pathways tend to fluctuate depending on the individual. Some people may experience hypoactivity in the D2 and 5-HT2A receptors rather than hyperactivity, in which other medications might have to be used, such as aripiprazole (Abilify), brexpiprazole (Rexulti), and cariprazine (Vraylar). As ziprasidone binds to the receptors and successfully manages to restrict the binding of dopamine and serotonin transmitters to their respective receptors, it promotes a safer volume of release of the two hormones.[13] Ziprasidone effectively reduces muscle spasms, manic episodes, as well as most positive symptoms within schizophrenia. Although it does reduce the symptoms, the drug is prone to causing multiple side effects as well.

Drowsiness is one of the more standard side effects of the drug, especially prominent within adolescent users.[1] During adolescence, the brain is still in the process of development, meaning that certain receptors and synapses have not fully developed yet either. One of the main receptors that are in the process of development is the D1 and D2 receptors, which are the main receptors that are even more unstable in schizophrenic patients. So when ziprasidone suppresses these receptors, it could lead to a much larger drop in effective dopamine signaling than what is usually required. Meanwhile, the receptors in most adults are fully developed during the time they are put on the medication, so their receptors can handle the suppression of the receptors better. Along with drowsiness, other side effects that occur persistently in adolescents include dizziness, headache, nausea, and constipation. These side effects occur more prominently in adolescents due to the fact that multiple receptors are being altered simultaneously, affecting communication in the neurochemical pathways and causing such effects to occur.[1]

Adolescents are quite often not diagnosed with schizophrenia even though the symptoms of the disorder are present due to the social stigma around schizophrenia. Since individuals who are diagnosed with schizophrenia are all perceived as violent and insane, even when on proper medication, it can cause discrimination in the workplace, job market, or other social surroundings. The discrimination could potentially trigger episodes within the individual due to environmental stress, aggravating the symptoms of the disorder even more; instead, they are labeled with a "psychotic disorder" or as "unspecified psychosis."[1] The adolescent brain is also still in its developing stage, as stated before, and schizophrenia can often be mistaken for other disorders such as bipolar disorder, substance-use psychosis, or even brain tumors.[1]

More caution must be taken when encountering adolescents with schizophrenia, as their cerebral functionality is not at the correct stage for proper diagnosis. Another factor to consider is lifestyle choices. The use of cannabis, specific steroids, and other hallucinogens can severely disorient an adolescent due to the sensitivity of their serotonin and glutamate receptors, which are the main receptors that these drugs disrupt.[1] The disruption within the brain causes paranoia and hallucinations that impact an adolescent much more vigorously. The side effects of the hallucinogens are nearly parallel to the positive symptoms in schizophrenia, which can lead to a false diagnosis.

Most Effective Medication for Adolescents Compared to Adults

Many doctors tend to prescribe ziprasidone after the use of another antipsychotic, even though it is an atypical antipsychotic, because it shows a much lower risk of weight gain during the prescription period than other second-generation antipsychotics. Even though ziprasidone does bind to H1 receptors, it binds with minimal affinity in comparison to other antipsychotics. Since the H1 receptors are in charge of producing histamine, which controls food intake and appetite, minimal suppression of the receptors enables more control over the quantity of food consumed by the prescribed patient. Ziprasidone also does not fluctuate blood sugar levels or cholesterol, for the same reasons as lower weight gain (minimal H1 receptor suppression), showing low risks of metabolic side effects that would otherwise be present in other medications.[1]

Interview with Psychiatrist

Dr. Mahamaya Bhattacharyya, a certified psychiatrist, explains the process of diagnosis for schizophrenia as well as the precautions taken beforehand in order to properly diagnose an adolescent patient. When a patient with symptoms of schizophrenia comes in, it's usually during or after a psychotic break. The patient is given olanzapine, which is a more effective alternative to ziprasidone. Olanzapine has a broader binding range, suppressing more receptors in which hyperactivity could commence. Along with that, olanzapine also has a higher continuation rate in patients, although the medication has higher adverse reactions. While the patient is given antipsychotics in intervals, they are heavily monitored and tested for any organic causes for the psychotic break.

The medication a patient is put on also depends on the severity of their schizophrenia. Residual schizophrenia is considered the least severe form of schizophrenia, as it only showcases the negative effects of schizophrenia, such as bad hygiene, poor memory, and attention deficit. These patients are given olanzapine, as it is the standard medication for treatment. For more severe cases where an adolescent patient becomes suicidal and violent—often the case for severe paranoid-type schizophrenia—clozapine will be prescribed, and the patient will be heavily monitored. Ziprasidone is normally used for less severe cases of paranoid-type schizophrenia for patients who have been suffering from excessive weight gain and cholesterol level increases due to other antipsychotic medications. However, ziprasidone is not the go-to medication, as it requires regular monitoring as well as constant testing and data collection.

Possible Modifications

Ziprasidone can potentially be further improved to have a faster effect as well as higher efficiency. The two main problems with ziprasidone are that the drug does not work as quickly and efficiently as other drugs, and that it requires regular monitoring within the hospital. Due to recent developments within artificial intelligence, results can be very quickly synthesized and written with little to no human error. AI can be used to monitor the symptoms of patients and send alerts whenever it spots a complication or any anomalies. This method allows psychiatrists to treat other patients as well without having to closely monitor the patient. Another limitation of the drug includes the efficacy of the drug, which is one of the reasons that many patients drop the antipsychotic fairly quickly. Potential solutions for this could be the addition of lifestyle changes within the individual.

Since schizophrenia is often passed down genetically, if an individual has any family history of schizophrenia, it is important they take the proper precautions to prevent psychotic breaks that could potentially cause the disorder to develop.[2] Refraining from the use of cannabis as well as other hallucinogens prevents the risks of development, as hallucinogens overstimulate serotonin receptors, causing hallucinations, paranoia, and restlessness, all of which can trigger the development of schizophrenia later on. Although antipsychotic medication tremendously aids the modulation of schizophrenia, diet and lifestyle also impact its severity. It is important to maintain a regular sleep schedule of at least eight hours a day, as it allows regulation of neurotransmitter functionality within the system. Without an adequate amount of sleep each night, the brain is not able to reset.

Conclusion

This research's focus is to examine how neurochemical imbalances in schizophrenia, particularly involving dopamine and serotonin pathways, affect brain function, and how medication such as ziprasidone differs in effectiveness and side effects between adolescents compared to adults. The central focus of the paper was to understand the differences in the structural effects and receptor dysregulation in adolescents versus adults contributing to symptoms, and how treatment outcomes may vary depending on age and severity of the disorder. The literature review explained how schizophrenia is strongly linked to disruptions in the mesolimbic pathway, including hyperactivity of D2 and 5-HT2A receptors, as well as reductions in gray and white matter. These biological changes help explain the wide range of positive, negative, and cognitive symptoms observed in patients. Ziprasidone was shown to mitigate these effects by acting as an antagonist to the dopamine and serotonin receptors, helping regulate neurotransmitter levels. However, its impact differs by age group, with adolescents experiencing more pronounced side effects due to ongoing brain development, while adults tend to tolerate the medication more effectively.

Despite these insights, the explanation does not fully account for the complexity of schizophrenia. The disorder cannot be understood solely through neurochemical activity or medication response, as factors such as environmental stress, substance use, diagnostic challenges, and social stigmas play significant roles in the development of the disorder as well. Additionally, variability in patient responses to different antipsychotic medications highlights the limitations of a single treatment approach. Therefore, schizophrenia is better understood as a multifactorial disorder influenced by an interaction of biological, psychological, and environmental factors. A more comprehensive perspective that includes neurodevelopment, lifestyle influences, and individualized treatment strategies is necessary to fully address the condition.

Neurochemical disruptions in areas of the brain—predominantly in areas containing the majority of serotonin and dopamine receptors—are the cause for the majority of positive, negative, and cognitive symptoms. Ziprasidone works as a suppressor and an antagonist, decreasing neurotransmission activity within synapses and receptors in order to treat symptoms; however, the suppression of these receptors affects adolescents considerably more than adults. This is due to the fact that their brain is still in its early development stages, so the suppression of these receptors can cause significant fluctuations in how the adolescent is affected. Some solutions can help decrease the gap in side effects between adolescents and adults, such as regular monitoring with AI to decrease the labor from psychologists, along with balanced amounts of sleep and food consumed throughout the day.

Ultimately, advancing treatment of schizophrenia requires an integrated approach that goes beyond neurotransmitter regulation alone. Future research as well as clinical practices should prioritize personal care, earlier and more accurate diagnosis, and the overall development of treatments that are both effective and better suited to both adolescents and adults.

Acknowledgements

I would like to thank Professor Varkey for guiding me through my research as well as Professor Jen for helping me convert my ideas into well-constructed paragraphs. I would also like to thank my family, who has supported me throughout this entire journey and pushed me to challenge myself with this research.

References

1. Wu EQ, Shi L, Birnbaum H, Hudson T, Kessler R. Annual prevalence of diagnosed schizophrenia in the USA: a claims data analysis approach. *Psychol Med*. **2006**;36(11):1535-1540. doi:10.1017/S0033291706008191

2. Yue, Y.; Kong, L.; Wang, J.; Li, C.; Tan, L.; Su, H.; Xu, Y. Regional Abnormality of Grey Matter in Schizophrenia: Effect from the Illness or Treatment? *PLoS One* **2016**, *11* (1), e0147204. DOI: 10.1371/journal.pone.0147204.

3. Cleveland Clinic. Grey Matter. https://my.clevelandclinic.org/health/body/24831-grey-matter (accessed June 24, 2026).

4. Elbe, D.; Carandang, C. G. Focus on ziprasidone: A review of its use in child and adolescent psychiatry. *J. Can. Acad. Child Adolesc. Psychiatry* **2008**, *17* (4), 220–229.

5. Findling, R. L.; Cavuş, I.; Pappadopulos, E.; Vanderburg, D. G.; Schwartz, J. H.; Gundapaneni, B. K.; DelBello, M. P. Individual changes in antipsychotic-related weight gain following ziprasidone treatment. *J. Clin. Psychiatry* **2013**, *70* (4), 555–560. DOI: 10.4088/JCP.08m04374.

6. National Center for Biotechnology Information. *Antipsychotics*; StatPearls Publishing, 2024. https://www.ncbi.nlm.nih.gov/books/NBK448157/ (accessed June 24, 2026).

7. Tufts University School of Medicine. Teen Behavior Explained by a Neuroscientist. https://medicine.tufts.edu/news-events/news/teen-behavior-explained-neuroscientist (accessed June 24, 2026).

8. Filley, C. M.; Fields, R. D. Ziprasidone for schizophrenia and bipolar disorder: A review of the clinical trials. *Neuropsychiatr. Dis. Treat.* **2016**, *12*, 1925–1935. DOI: 10.2147/NDT.S102321.

9. Mercadante, A. A.; Tadi, P. Neuroanatomy, Gray Matter. In *StatPearls*; StatPearls Publishing, 2026. https://www.ncbi.nlm.nih.gov/books/NBK553239/ (accessed February 22, 2026).

10. Substance Abuse and Mental Health Services Administration. Table 3.22, DSM-IV to DSM-5 Schizophrenia. In *Impact of the DSM-5 on Therapeutic Decisions and Outcomes*; Substance Abuse and Mental Health Services Administration, **2016**.
https://www.ncbi.nlm.nih.gov/books/NBK519704/table/ch3.t22/ (accessed June 24, 2026).

11. McCutcheon, R. A.; Abi-Dargham, A.; Howes, O. D. Schizophrenia, Dopamine and the Striatum: From Biology to Symptoms. *Trends Neurosci.* **2019**, *42* (3), 205–220. DOI: 10.1016/j.tins.2018.12.004.

12. Rawani, N.S.; Chan, A.W.; Dursun, S.M.; Baker, G.B. The Underlying Neurobiological Mechanisms of Psychosis: Focus on Neurotransmission Dysregulation, Neuroinflammation, Oxidative Stress, and Mitochondrial Dysfunction. Antioxidants 2024, 13, 709. https://doi.org/10.3390/antiox13060709

13. Brisch, R.; Saniotis, A.; Wolf, R.; Bielau, H.; Bernstein, H. G.; Steiner, J.; Bogerts, B.; Braun, K.; Jankowski, Z.; Kumaratilake, J.; Henneberg, M.; Gos, T. The role of dopamine in schizophrenia from a neurobiological and evolutionary perspective: old fashioned, but still in vogue. *Front. Psychiatry* **2014**, *5*, 47. DOI: 10.3389/fpsyt.2014.00047.

14. Schmidt, A. W.; Lebel, L. A.; Howard, H. R., Jr.; Zorn, S. H. Ziprasidone: A novel antipsychotic agent with a unique human receptor binding profile. *Eur. J. Pharmacol.* **2001**, *425* (3), 197–201. DOI: 10.1016/S0014-2999(01)01188-7.

15. MedlinePlus. Ziprasidone. U.S. National Library of Medicine. https://medlineplus.gov/druginfo/meds/a699062.html (accessed June 24, 2026).

16. Sturman, D. A.; Moghaddam, B. The neurobiology of adolescence: changes in brain architecture, functional dynamics, and behavioral tendencies. *Neurosci. Biobehavioral Rev.* **2011**, *35* (8), 1704–1712. DOI: 10.1016/j.neubiorev.2011.04.003.

17. van Zelst, C. Stigmatization as an environmental risk in schizophrenia: a user perspective. *Schizophr. Bull.* **2009**, *35* (2), 293–296. DOI: 10.1093/schbul/sbn184.

18. Paul, T.; Javed, S.; Karam, A.; Loh, H.; Ferrer, G. F. A Misdiagnosed Case of Schizoaffective Disorder With Bipolar Manifestations. *Cureus* **2021**, *13* (7), e16686. DOI: 10.7759/cureus.16686.

19. National Institute on Drug Abuse. Psychedelic and dissociative drugs. National Institutes of Health. https://nida.nih.gov/research-topics/psychedelic-dissociative-drugs (accessed June 24, 2026).

20. Greenberg, W. M.; Citrome, L. Ziprasidone for schizophrenia and bipolar disorder: a review of the clinical trials. *CNS Drug Rev.* **2007**, *13* (2), 137–177. DOI: 10.1111/j.1527-3458.2007.00010.x.

21. National Health Service. Causes - Schizophrenia. NHS. https://www.nhs.uk/mental health/conditions/schizophrenia/causes/ (accessed June 24, 2026).

22. National Center for Biotechnology Information. PubChem Compound Summary for CID 60854, Ziprasidone. https://pubchem.ncbi.nlm.nih.gov/compound/Ziprasidone (accessed May 8, 2026).

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