



Review Article

Overview and Treatments of Schizophrenia: A Recent Update

Monali Khatake^{*1}, Vinayak Gaware², Rahul Khaire², Charushila Bhangale²

¹Department of Pharmaceutical Quality Assurance, PRES College of Pharmacy (Women's), Chincholi, Nashik-422102

²Department of Pharmaceutical Chemistry, PRES College of Pharmacy (Women's), Chincholi, Nashik-422102

ARTICLE INFO

Received: 03 Mar. 2023

Revised: 05 Mar. 2023

Accepted: 08 Apr. 2023

Published: 20 Apr. 2023

Keywords:

Schizophrenia, Emotional responsiveness, Neurobiological theory, Neurotransmitter, Dopamine, Prefrontal cortex

DOI:

10.5281/zenodo.7847211

ABSTRACT

A mental illness called schizophrenia is characterised by disturbances in cognition, perception, emotional responsiveness, and social relations. Around 24 million individuals, or 1 in 300 persons (0.32%), worldwide suffer from schizophrenia. For men, this incidence is 1 in 222 (0.4%). Here, a person's capacity for thought, emotion, and behaviour is impacted. Memory loss and attention problems could also be present. According to neurological theory, imbalance of certain neurotransmitters, including: Gamma-aminobutyric acid (GABA) expression levels in the prefrontal cortex, an excess of dopamine, decreased glutamate levels in the cerebrospinal fluid, or decreased serotonin levels Schizophrenia is thus the most prevalent mental disease of our time, causing severe disruptions for those who have it and consuming a sizable percentage of the health system's limited resources. In this essay, the most current research on introduction, etiology, symptoms, and remedies is reviewed. Researching this disease ran into some methodological issues, which are addressed. The results of therapies available for this condition have improved, but whether a patient gets these treatments may rely on the services that the local community is willing to provide.

INTRODUCTION

Schizophrenia as a notion is fading. After being tormented by psychology for years, it now appears that psychiatry, the very field that once supported it, has killed it. Its demise won't be lamented. Today, receiving a diagnosis of schizophrenia is linked to a nearly two-decade decrease in life expectancy. Only one in seven individuals, according to some standards, heal. Surprisingly,

despite widely publicised improvements in therapies, the percentage of patients who heal hasn't grown over time. There is a serious problem. It comes out that the idea of schizophrenia itself is a part of the issue. The case for schizophrenia being a separate illness has been "fatally undermined." Psychosis (typically characterised by disturbing hallucinations, delusions, and

*Corresponding Author: Monali Khatake

Address: Dept. of Pharmaceutical Quality Assurance, PRES College of Pharmacy (Women's), Chincholi, Nashik-422102

Email ✉: khatakemonika@gmail.com

Relevant conflicts of interest/financial disclosures: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.



muddled ideas) is also asserted to occur along a continuum and in degrees, just as we now have the notion of autism spectrum disorder. The severe extremity of a range or continuum of events is schizophrenia. According to Jim van Os, a psychiatric professor at Maastricht University, we cannot transition to this new way of thinking without altering our vocabulary. As a result, he suggests that the word "schizophrenia" be eliminated. He proposes the idea of a psychosis spectrum disease in its stead. The perception of schizophrenia as a "hopeless chronic brain disease" is another issue. As a result, some people given this diagnosis, and some parents, have been told cancer would have been preferable, as it would be easier to cure. Yet this view of schizophrenia is only possible by excluding people who do have positive outcomes. For example, some who recover are effectively told that "it mustn't have been schizophrenia after all". Schizophrenia, when understood as a discrete, hopeless, and deteriorating brain disease, argues van Os, "does not exist" [1].

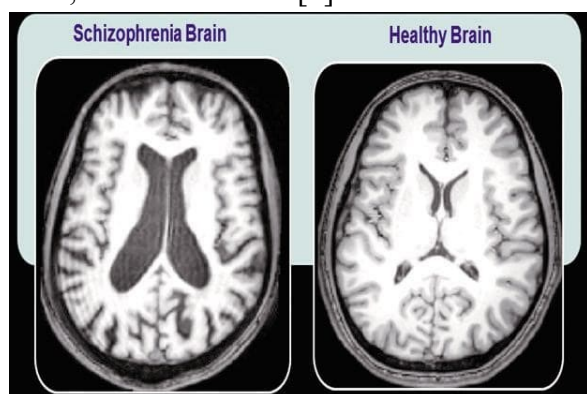


Figure 1: Comparison of Schizophrenial and Healthy Brain

Breaking down breakdowns

Instead, schizophrenia could end up being many distinct things. Sir Robin Murray, a renowned psychiatrist, says: "I anticipate the concept of schizophrenia being eliminated soon." The syndrome is already starting to subdivide, for instance, into instances brought on by substance misuse, societal disadvantage, copy number

(genetic) variations, etc. This process will presumably quicken, and the word "schizophrenia" will pass into obscurity like "dropsy." Many of the symptoms associated with schizophrenia, such as hallucinations, delusions, disordered thinking and behavior, indifference, and flat feeling, are now being studied from various perspectives. It's true that in the past, people have erred by mistaking a path for the path or, more frequently, a secondary road for a motorway. For instance, researchers E. Fuller Torrey and Robert Yolken have contended that "the most important etiological agent (cause of schizophrenia) may turn out to be a contagious cat" based on their research on the parasite *Toxoplasma gondii*, which is spread to humans through cats. The percentage of individuals who heal hasn't risen over time, so it won't. There is a serious problem. It comes out that the idea of schizophrenia itself is a part of the issue. The case for schizophrenia being a separate illness has been "fatally undermined." Psychosis (typically characterised by disturbing hallucinations, delusions, and muddled ideas) is also asserted to occur along a continuum and in degrees, just as we now have the notion of autism spectrum disorder. The severe extremity of a range or continuum of events is schizophrenia. According to Jim van Os, a psychiatric professor at Maastricht University, we cannot transition to this new way of thinking without altering our vocabulary. As a result, he suggests that the word "schizophrenia" be eliminated. He proposes the idea of a psychosis spectrum disease in its stead. The perception of schizophrenia as a "hopeless chronic brain disease" is another issue. Because cancer is more treatable than this condition, some individuals who have received this diagnosis and some parents have been advised that cancer would have been better. Yet this view of schizophrenia is only possible by excluding people who do have positive outcomes (Figure 2) For example, some who

recover are effectively told that “it mustn’t have been schizophrenia after all”. Schizophrenia, when understood as a discrete, hopeless and deteriorating brain disease, argues van Os, “does not exist” [1]. Their Evidence does suggest that exposure to T. Gondii when young can increase the odds of someone being diagnosed with schizophrenia. However, the size of this effect involves less than a twofold increase in the odds of someone being diagnosed with schizophrenia. At best, this can be compared to other risk variables, and it is almost certainly much smaller. For instance, the likelihood of developing a psychotic condition (like schizophrenia) increases by about two to three times when a person uses weed or experiences early viral infections of the central nervous system. Much larger figures are revealed by more in-depth analyses. Daily use of high-potency, skunk-like weed is linked to a fivefold rise in the risk of getting psychosis when compared to non-users. The likelihood of getting insanity is more than fifty times higher for those who have experienced five distinct kinds of trauma (including sexual and physical abuse) than for those who have not. There are also other ways to get "schizophrenia." About 1% of instances, or 22q11.2 deletion syndrome, are thought to be caused by the loss of a brief segment of chromosome 22. Although this is still debatable, it's also conceivable that a small minority of individuals with a schizophrenia diagnosis may have their symptoms rooted in brain inflammation brought on by autoimmune diseases like anti-NMDA receptor encephalitis. All of the aforementioned risk factors may result in comparable experiences that we, in our immaturity, have categorised as schizophrenia. The experiences of one individual may be the product of a brain disease with a strong genetic component, possibly resulting from an exaggeration of the typical adolescent process of pruning links between brain cells. The experiences

of another individual might be the result of a sophisticated posttraumatic response. Such internal and exterior elements might also interact. In any case, it appears that the two opposing groups in the schizophrenia debate—those who think it is a neurodevelopmental disease with a hereditary basis and those who think it is a reaction to psychosocial variables like adversity—both had a piece of the solution. This tension was exacerbated by the notion that schizophrenia was a unique entity with a single path to manifestation [2].

SYMPTOMS

When the illness is prevalent, there may be periods when it is difficult for the patient to tell the difference between actual and imagined experiences. The intensity, length, and frequency of symptoms can differ depending on the disease, but in people with schizophrenia, the frequency of serious psychotic symptoms frequently declines with age. Stressful circumstances, drinking or illicit substance use, and improper medication use frequently cause symptoms to worsen. There are three main groups for symptoms: Positive signs: Hallucinations, such as hearing voices or seeing things that don't exist, anxiety, and exaggerated or skewed perceptions, beliefs, and behaviours are examples of (those unusually present) hallucinations.

ailment symptoms: (those abnormally absent) a reduction or lack of the capacity to make decisions, talk, communicate emotions, or enjoy oneself. Unorganized signs: Problems with reasoning, confusion, and disordered thoughts, as well as occasionally strange behaviour or abnormal motions. Another aspect of functioning that is impacted by schizophrenia is cognition, which can result in issues with focus, recollection, and attention as well as poor academic achievement. Schizophrenia symptoms typically first manifest in early adulthood and must last for at least six months in order to be diagnosed. Men



typically begin to exhibit symptoms in their late teens or early 20s, whereas women typically begin to exhibit symptoms in their 20s and early 30s. Early warning signs may include less drive, strained relationships, and subpar academic achievement. However, a therapist should perform a comprehensive medical evaluation before making a diagnosis to rule out drug abuse or other neurological or medical conditions whose symptoms resemble schizophrenia [3].

Social skills, stigma, and rejection

Others frequently stigmatise and avoid those who have schizophrenia. Undoubtedly, societal misunderstanding about schizophrenia contributes to this. However, it is also conceivable that some of the social deficits that are associated with those who have this condition pose challenges for both those individuals and those they engage with. This might eventually result in more pessimism, social isolation, and exclusion from other people [4]. Nisenson, Berenbaum, and Good (2001) requested student research aides (all of whom had been chosen because they had pleasant personalities) to develop short friendships with schizophrenia patients as an empirical proof of this [5]. The research aides' behaviour evolved over the course of the 2-week investigation [6]. What was most striking was how much more disrespect the pupils displayed for the patients [7]. But do people's aversion to marrying, interacting with, or hiring someone with schizophrenia have anything to do with their lack of social skills? The response seems to be yes, at least in part. Clinically stable outpatients with schizophrenia participated in a 3-minute roleplay discussion with a partner, which Penn, Kohlmaier, and Corrigan (2000) recorded. The patients' social skills were then evaluated by trained study aides, who took into account things like how well the patients made eye contact, spoke, and whether or not there were any pauses or stutters in their speech [8]. After 41 students saw the recorded role-plays, they were asked how

much social distance they would prefer to maintain from each of the cases they had seen [9]. How "strange" the patient was described as being by the students was the greatest indicator of whether they would show a wish to avoid interacting with them [10].

In turn, the patient's general social abilities anticipated this. Simply put, this research shows that we tend to view individuals as strange when they have poor social skills, and that we naturally want to avoid them [11].

ETIOLOGY

According to several studies, abnormalities in numerous neurotransmitters, such as glutaminergic and GABA hypoactivity or dopaminergic, serotonergic, and alpha-adrenergic hyperactivity, contribute to the onset of schizophrenia. A major contributing factor is genetics; monozygotic twins have a 46% agreement rate and are 40% more likely to acquire schizophrenia if both parents have the disorder. Along with Dysbindin (DTNBP1), which promotes glutamate release, and Catecholamine O-Methyl Transferase (COMT) mutation, which controls dopamine function, these genes have been linked to glutamate signalling and brain growth. As stated above, a number of environmental factors have been linked to an increased chance of contracting the illness, including:

- Abnormal fetal development and low birth weight.
- Gestational diabetes.
- Preeclampsia.
- Emergency cesarean section and other birthing complications.
- Maternal malnutrition and vitamin D deficiency.
- Winter births - associated with a 10% higher relative risk.
- Urban residence increases the risk of developing schizophrenia by 2% to 4%.



According to a research done in Britain [12], the incidence is also up to ten times higher in children of African and Caribbean migrants than it is in European children. Numerous studies have examined the link between cannabis use and psychosis, and new longitudinal studies indicate a 40% higher risk as well as a dose-effect connection between cannabis use and the risk of getting schizophrenia [13].

PATHOPHYSIOLOGY

Regarding how schizophrenia develops, there are three major theories. According to the neurochemical abnormality theory, an imbalance of dopamine, serotonin, glutamate, and GABA causes the disease's mental symptoms. It suggests that the four principal dopaminergic networks play a role in the onset of schizophrenia. According to this dopamine theory, the mesolimbic pathway's excessive stimulation of D2 receptors is what causes the disease's positive symptoms, whereas the extrapyramidal system's effects on low amounts of dopamine in the nigrostriatal pathway are thought to be what causes the disease's motor symptoms. The mesocortical pathway is believed to cause low amounts of dopamine in the mesocortical region, which in turn causes the disease's negative effects. Other symptoms like amenorrhea and diminished desire may be brought on by elevated prolactin levels as a result of decreased tuberoinfundibular dopamine availability as a result of tuberoinfundibular pathway obstruction. While serotonergic hyperactivity has also been shown to contribute to the development of schizophrenia, evidence suggests that glutaminergic hypoactivity may also play a role. NMDA receptor blockers have been shown to exacerbate both positive and negative symptoms in schizophrenia [14]. The presence of anomalies in the brain structure, the lack of gliosis, which suggests in utero changes, and the finding that motor and cognitive deficits in patients occur prior to the start of the disease are additional

reasons that schizophrenia is a neurodevelopmental disorder. The disconnect theory, on the other hand, concentrates on the neuroanatomical alterations detected in PET and fMRI images. (Figure 3). Schizophrenia results in a decrease in grey matter volume that affects both the frontal and temporal regions. There are also differences in the frontal regions and hippocampus, which may be a factor in the disease's various cognitive and memory problems.

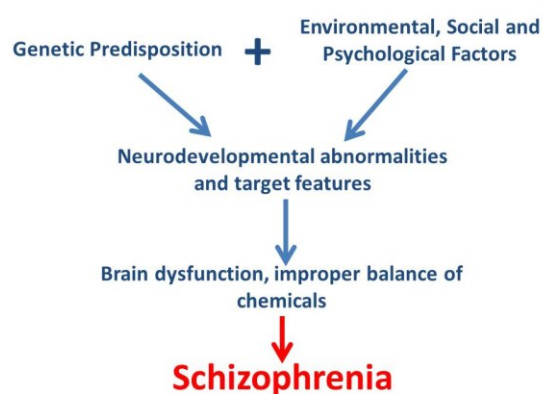


Figure 2: Pathophysiology of Schizophrenia

WEB DIFFERENTIAL DIAGNOSIS

Differential diagnoses that need to be considered are as follows:

- Bipolar I Disorder with psychotic features.
- Delusional disorders.
- Schizoaffective disorder.
- Brief psychotic disorder.
- Psychosis NOS.
- Certain personality disorders.
- Drug and medication-induced psychosis.

In the relationship between online and offline action, online is given a greater precedence than basic web access [15]. Overall, the writing's current state paints an intriguing image of how web-based amusement is used in general activity. The internet is typically used for emancipatory activities such as bringing issues to light, mobilising people, establishing extremist plans, discussing and evaluating activities, but it is also

unjustly used (by groups and experts) to entrap, misdirect, and suppress undesirable activities. Without a doubt, internet entertainment helps minority groups communicate and understand one another more successfully [16]. Online amusement is incredibly dynamic and pluralistic thanks to this, but it may also alienate and ensnare social orders. Progressively, online activism and disconnected activism are essential social-mental tools for mobilization, debate, and conflict [17].

INTERVENTIONS FOR SCHIZOPHRENIA

This part will cover the various therapies that are currently available for managing the symptoms of schizophrenia as well as issues that patients with the condition encounter, like unemployment, illiteracy, and loss of social connections [18].

Pharmacological intervention

After a single incident of psychosis, it has been found that 6% of people with schizophrenia experience a complete return from their schizophrenia symptoms [19]. Deterioration of symptoms has been recorded in 39% of the cases [19]. According to estimates, one in seven people with schizophrenia fully heal [20]. Table 1 lists the problems with and potential solutions for treating schizophrenia holistically. Several antipsychotic medications are frequently used in the early therapy of schizophrenia. Antipsychotic medicines typically target the symptoms of schizophrenia rather than its underlying causes, such as stress and substance misuse. (see above). As shown in Table 1, the majority of antipsychotic medications reduce disturbances and delusions, though some also aim to treat the unfavourable signs of schizophrenia. Typically, the only therapy choice for schizophrenia is antipsychotic medication. By inhibiting dopamine receptors, the majority of antipsychotic medications lessen the positive signs of schizophrenia [21].

160 people with schizophrenia were randomly assigned to receive therapy for up to 2 years with either clozapine or chlorpromazine in one trial by

Girgis et al. [22]. It was discovered that clozapine had a greater retention rate than chlorpromazine. It was discovered that clozapine had no advantages over traditional antipsychotics in another research that involved 34 schizophrenia patients [23]. According to McEvoy et al.'s research, many people with schizophrenia stopped receiving therapy because some antipsychotic medications were ineffective [24]. For the therapy of both positive and negative symptoms in schizophrenia patients, a daily dose of clozapine between 523 mg and 600 mg is helpful [24]. According to Sanz-Fuentenebro et al.'s research, people with schizophrenia taking clozapine maintained their initial therapy for a lot longer than those taking risperidone [25]. Particularly, risperidone had a recall rate of 82 point eight percent compared to clozapine's 93 point four percent. Patients taking clozapine typically experience greater weight increase than those taking risperidone [26]. A total of 63 patients were chosen and arbitrarily assigned to receive either clozapine or risperidone in one research by Sahni et al. [27]. In terms of sociodemographic factors like age, sex, schooling, employment, salary, family structure, and marital status, the two groups were comparable. In the clozapine group, the mean sickness length was 19 points 39 months, while it was 18 points 63 months in the risperidone group. Positive effects were significantly reduced by both medications. It was discovered that risperidone and clozapine decreased positive symptoms similarly, but clozapine significantly outperformed risperidone in reducing negative symptoms. Accordingly, it has been observed that giving clozapine to patients who are chronically psychotic reduces suicidal thoughts [28]. Clozapine has been found to lessen suicidal ideation in people with schizophrenia. The likelihood of suicidal behaviours was found to be reduced by three times with long-term clozapine therapy. Furthermore, metformin (500 mg twice daily) is frequently given to clozapine individuals

who want to reduce weight. To control weight and enhance metabolic metrics, aripiprazole and clozapine may occasionally be administered together [29]. In comparison to clozapine alone, aripiprazole and clozapine administration had a favourable impact on the positive and overall symptoms of people with schizophrenia, according to one research. Antipsychotic medications also aid in the improvement of confused behaviour in daily life. Additionally, they are employed to lessen brain decline, which enhances relationships and helps people pursue education and employment. Antipsychotic medications aid in reducing confused behaviour in daily life. Additionally, they are employed to advance work [32], schooling [30, 31], and relationships [30, 31]. The function of pharmaceutical intervention in the total treatment of schizophrenia is summarised in Table 1 [33].

TABLE 1. Abbreviations & Acronyms

OCD	Obsessive Compulsive Disorder
T. Gondii	Toxoplasma Gondii
DNA	Deoxyribose Nucleic Acid
Anti-NMDA Receptors	N-methyl-D-aspartate Receptors
GABA	Gamma-aminobutyric Acid
NGR1	Negative Growth Regulatory Protein 1
DTNBP1	Dystrobrevin Binding Protein 1
COMT	Catecholamine O-Methyl Transferase
PET	Positron Emmision Tomography
FMRI	Functional Magnetic Resonance Imaging
NOS	Not Specified Otherwise
CBT	Cognitive Behaviour Therapy
UK	United Kingdoms
NHS	National Health Services
PORT	Patient Outcomes Research Teams

Complementary intervention and diet

Although the consumption of unsaturated fat was found to be comparable in both groups, it was discovered that the diets of schizophrenia patients contained more total fat and less fibre than the diets of a control group matched for age, gender, and education. In different research, the nutritional consumption of 30 people with schizophrenia who were residing in assisted-living institutions in Scotland was compared to that of a control group that was matched for sex, age, smoking, and job status [34]. The bulk of schizophrenia sufferers were overweight or obese, and their consumption of saturated fat was greater than that advised in diets for people with schizophrenia [35]. A study of the dietary habits of 102 people with schizophrenia with a focus on fruit and vegetable intake and smoking behaviour revealed that people with schizophrenia consumed less total fiber, retinol, carotene, vitamin C, vitamin E, fruit, and vegetables than the control group [36, 37]. The research came to the conclusion that the patients' dietary options were bad, particularly for the men. Graham et al. [38] hypothesised that giving vitamin D to people with schizophrenia lessens their unpleasant symptoms. The eating patterns of 146 adult community-dwelling schizophrenic patients were examined in another research by [39]. In comparison to a control group, it was found that patients ingested more food containing protein, carbohydrates, and lipids. In people with schizophrenia, these behaviours can result in systemic inflammation, type II diabetes, and cardiovascular illnesses [40]. The short lifespan of people with schizophrenia is linked to these illnesses [41]. High-fiber diets may strengthen the immune and cardiovascular systems, avoiding early death in schizophrenia, according to a research study by Joseph et al. [42].

Cognitive behavior therapy

CBT is a therapy approach that aids in changing unfavourable thought, emotion, and behaviour



patterns. Practical self-help techniques are used in CBT, and it has been discovered that these techniques reduce positive signs of schizophrenia. CBT blends "cognitive therapy" and "behavioral therapy," two different types of treatments. The patient can frequently have healthy ideas and behaviours thanks to the use of these two methods together. In order to treat both the main symptoms of the disease and social impairments, Morrison summarises the use of CBT in schizophrenia patients [43]. Morrison noted that many signs of schizophrenia are resistant to pharmacological therapy and proposed that adding CBT to the administration of antipsychotics may be more successful than doing so alone. For instance, a number of studies have discovered that cognitive therapy and CBT can improve cognitive impairments, which in turn can reduce positive symptoms [44, 45]. CBT can be used with a variety of methods to change beliefs and behaviours. Various CBT techniques can be used successfully in schizophrenia, according to a research paper that outlined the fundamentals of CBT for schizophrenia [46]. One method, referred to as cognitive restructuring, involves asking the patient to provide proof to support their views. The customer is helped by this method to recognise their delusions. With the help of this method, the patient can develop the ability to recognize, question, and replace unhelpful thoughts with more constructive and realistic ones. CBT has been proven to be successful in treating homelessness. CBT improves relationships, reduces cognitive impairment, and has a beneficial impact on enjoyment. The goal of behavioural treatment is to help the sufferer learn how to change their behaviour. So that they can apply their recently acquired skills in social settings, they might practise speaking skills, for instance. CBT helps patients interact socially, which influences companionship and relationships. Over the past 15 years, research have been conducted to validate

CBT for schizophrenia. In the UK, CBT is one of the most widely used treatments for schizophrenia. (generally in addition to medications). In reality, the UK National Health Service (NHS) has suggested CBT as the first line of treatment for those with schizophrenia. Similar to this, CBT was advised for those with schizophrenia by the American Psychiatric Association [47]. CBT has recently been advised for patients who continue to experience delusional symptoms by the US Schizophrenia Patient Outcomes Research Team (PORT).

Yoga therapy

In many cases, yoga treatment is used in conjunction with pharmaceutical drugs to treat the signs of schizophrenia [48]. Pharmacological treatment alone may not have all the desired benefits in treating negative signs of schizophrenia [49]. Yoga, when combined with psychotropic drugs, is more effective at treating both positive and negative effects than drugs alone. Additionally, pharmaceutical treatments for schizophrenia frequently result in fat [49]. Antipsychotic drug administration has been shown to cause weight increase; however, yoga therapy has been found to help decrease this. Pharmacological treatments may result in endocrinological and menstruation disorders, which yoga therapy has been shown to be effective in treating [49]. Two groups of patients taking antipsychotic drugs were investigated in a study by Gangadhar et al. [49]. Yoga treatment was applied to one group. A regimen of bodily activities were used on the other group. For a month, both groups received training. (at least 12 sessions). The yoga group outperformed the other group in terms of bad symptom ratings. Similar to how the other group performed, yoga treatment had greater impacts on social dysfunction. In keeping with this, Vancampfort et al. discovered that yoga practise lowers metabolic risk, enhances mental and physical well-being, and decreases depressive



symptoms [50]. The body's creation of oxytocin is the most likely cause of yoga therapy's efficacy. A hormone that promotes happiness is oxytocin. In one trial, oxytocin and antipsychotic medications were given to 40 individuals [51]. It was discovered that in those individuals, both negative and positive symptoms changed. There are many benefits to yoga treatment. Yoga treatment has been shown to enhance memory, decrease depressive and psychotic symptoms, and improve quality of life. The problems with holistic schizophrenia treatment are listed in Table 1, along with possible yoga interventions [51].

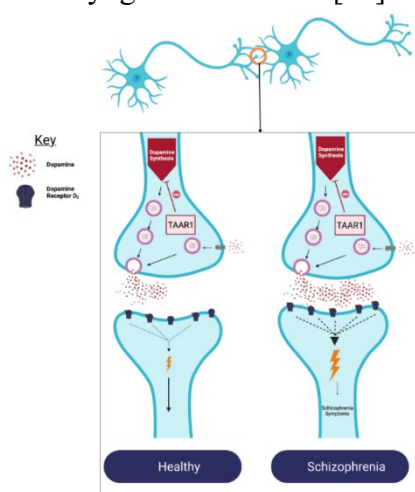


Figure 3: Neurotransmitter dopamine release in schizophrenia condition

COMORBIDITIES AND THEIR MANAGEMENT IN SCHIZOPHRENIA

The commoner co morbidities and their management are as follows:

Alcohol, cannabis, and cocaine are the most frequently misused drugs, and their use significantly affects the trajectory of the disease in people with schizophrenia who also have substance use problems. In addition, between 50% and 90% of people with schizophrenia smoke, which raises the risk of death from disease. Some psychotropic medications become less successful when a person smokes. Higher rates of psychotic recurrence, worsening function, and increased social failure are all linked to co-morbid drug use

disorder in schizophrenia. Furthermore, higher suicidal thoughts and victimisation are linked to the dual diagnosis.

Due to patients with dual illnesses not adhering to their treatment regimens as well, the use of longer-acting oral medicines and depot shots has also been shown to be beneficial. Treatment with clozapine appears to be most successful in decreasing alcohol and drug misuse in schizophrenic patients. In individuals with dual diagnoses, there is an increased risk of negative consequences from combining prescription drugs with illicit drugs. Patients frequently use the anti-obesity drug sibutramine to reduce their weight [52].

melancholy in schizophrenia: Between 25% and 81% of people with schizophrenia report having melancholy. Schizophrenic patients who experience depressive symptoms have a worse quality of life and are at higher risk for harm to oneself or others (including suicide), psychosis relapse, drug abuse issues, and psychiatric hospitalisation. In conclusion, schizophrenia frequently includes concomitant depressive symptoms, which are linked to a substantially worse long-term functional result. Standard of care should include the active management of particular symptoms of melancholy.

OCD in schizophrenia

Sexual, somatic, religious, aggressive, and somatic contamination are frequent motifs, with or without associated compulsions. These symptoms show inflated ideations and delusional appearance, which coincide with the underlying psychosis. Recent data point to a worse clinical trajectory, longer-term prognosis, and increased cognitive impairment.

As obsessive thoughts during recovery or the remission phase, as a de novo OC syndrome connected to therapy with atypical antipsychotics, or as a concomitant separate OC disorder, the syndrome may present during the prodromal phase

or during active psychotic disease. Treatment involves using antipsychotics like haloperidol as an additional form of anti-OC medication. Another option is to use cognitive behavioural therapy [53].

Eating disorder in Schizophrenia

Aggressive behaviour in schizophrenia must be controlled. Epidemiology has shown that people with schizophrenia are more likely to act violently if they also misuse drugs or alcohol. According to some research, 10% of patients who are admitted to institutions assault someone else within 24 hours. Temporary aggression is linked to both favourable psychotic signs and environmental variables. Patients with consistently violent psychoses may be treated with a variety of medications, such as traditional neuroleptics, atypical neuroleptics, mood stabilisers like sodium valproate, and rarely lithium carbonate. According to a new research, clozapine reduces violent behaviour in schizophrenia patients [54].

Schizophrenia and persistent aggressive behavior

Aggressive behaviour in schizophrenia must be controlled. Epidemiology has shown that people with schizophrenia are more likely to act violently if they also misuse drugs or alcohol. According to some research, 10% of patients who are admitted to institutions assault someone else within 24 hours. Temporary aggression is linked to both favourable psychotic signs and environmental variables. Atypical neuroleptics, conventional neuroleptics, mood stabilisers like sodium valproate, and rarely lithium carbonate are all options for treating patients with consistently violent psychosis [55]. According to a new research, clozapine reduces violent behaviour in schizophrenia patients.

CONCLUSION

The exact causes of schizophrenia are not fully understood, but researchers believe that a combination of genetic, environmental, and neurobiological factors may contribute to its

development. There is currently no cure for schizophrenia, but a range of treatments can help manage symptoms and improve quality of life. Antipsychotic medications are the most commonly used treatment for schizophrenia. They work by blocking dopamine receptors in the brain, which can reduce hallucinations, delusions, and other positive symptoms. However, these medications may also cause side effects such as weight gain, sedation, and movement disorders. Psychosocial interventions, such as cognitive-behavioral therapy and family therapy, can also be beneficial in treating schizophrenia. These interventions can help people with schizophrenia learn coping skills, improve communication, and reduce stress.

Overall, treatment for schizophrenia is most effective when it is tailored to the individual's specific needs and preferences. With appropriate treatment and support, many people with schizophrenia can lead productive and fulfilling lives.

REFERENCES

1. Social elements in schizophrenia. Hooley JM. *Current Directions in Psychological Science*, August 2010, 19, 238–242.
2. Emotion processing and its link to social performance in people with schizophrenia, Hooker C et al. *Research in psychiatry*. 2002 Sep 15;112(1):41-50.
3. "Social skills and social functioning," by Mueser et al. (1998)
4. "Prevalence and stability of social skill deficits in schizophrenia," by Mueser et al. *Research on schizophrenia* 5.2 (1991): 167–176
5. "Gender, social competence, and symptomatology in schizophrenia: a longitudinal analysis," Mueser et al. 138 in *Journal of Abnormal Psychology* 99.2 (1990).
6. The evolution of interpersonal interactions in people with schizophrenia, Nisenson et al. 111–125 in *Psychiatry* 64.2 (2001).



7. Social skills, perceived beauty, and symptoms are interpersonal variables that contribute to the stigma of schizophrenia, according to Penn et al. *Research on schizophrenia* 45.1-2 (2000): 37–45
8. "Neurocognitive and social cognitive predictors of interpersonal skill in schizophrenia," by Pinkham et al. 167–178 in *Psychiatry Research* 143.2-3 (2006).
9. Zhu, et al. Social performance and recognition of social signals are impaired in Chinese individuals with schizophrenia. *Clinical Neurosciences in Psychiatry*, 61, 149–158
10. E.L. Messias et al. schizophrenia epidemiology: a summary of research and urban legends. 2007 Sep 1;30(3):323–338 in *Psychiatric Clinics of North America*.
11. A review of schizophrenia susceptibility and risks: Beyond the two strike theory, Davis et al. (2016) 185–194 in *Neuroscience & Biobehavioral Reviews* 65.
12. "Schizophrenia: overview and treatment options," *Pharmacy and Therapeutics* 39.9 (2014): 638, by Patel et al.
13. "Diagnostic and statistical manual of mental disorders," Carter, Marcia Jean. *Journal of therapeutic leisure* 48.3 (2014): 275.
14. "Ferri's Differential Diagnosis: A Practical Guide to the Differential Diagnosis of Symptoms, Signs, and Clinical Disorders," by Jane K. Case, 14. Elsevier, 2006. *Mayo Clinic Proceedings*, Vol. 81, No. 10.
15. Differential diagnostic of borderline personality disorder, Joel Paris. *Clinical Psychiatry* 41.4 (2018): 575–582.
16. The diagnostic notion of schizophrenia: its background, development, and possibilities, by Assen Jablensky, 16. *Clinical neurobiology discussions* (2022).
17. "Psychosis prevalence and physical, metabolic, and cognitive co-morbidity: data from the second Australian national survey of psychosis," by Morgan, V. A., et al. 2146-2176 in *Psychological Medicine* 44.10 (2014).
18. E. Jääskeläinen et al., "A Systematic Review and Meta-Analysis of Recovery in Schizophrenia." 39.6 (2013): 1296–1306 *Schizophrenia Bulletin*.
19. Self-reported physical activity levels and exercise recommendation practises of conference attendees at the 2017 Royal Australian and New Zealand College of Psychiatrists (RANZCP) meeting, by Fibbins et al. 2020: 565-572 in *Journal of Mental Health*, 29.5.
20. "Clozapine v. chlorpromazine in treatment-naive, first-episode schizophrenia: 9-year outcomes of a randomised clinical trial," by Girgis et al. 281-288 in *The British Journal of Psychiatry*, Vol. 199.4, 2011.
21. "Clozapine as a first treatment for schizophrenia," by Woerner et al. (2003): 1514–1516 in *American Journal of Psychiatry* 160.8.
22. "Effectiveness of clozapine versus olanzapine, quetiapine, and risperidone in patients with chronic schizophrenia who did not respond to prior atypical antipsychotic treatment," McEvoy, Joseph P., et al. (2006): 600–610 in *American Journal of Psychiatry* 163.4.
23. Javier Sanz-Fuentenebro et al. Results of a one-year random comparison of clozapine and risperidone in the treatment-naive first incident of schizophrenia. *Research on schizophrenia* 149.1-3 (2013): 156–161.
24. Atypical antipsychotics and weight gain: a comprehensive review, Taylor, D. M. and McAskill, 24. (2000): 416-432 in *Acta Psychiatrica Scandinavica* 101.6.
25. 25. Sahni S, et al. A pilot trial comparing clozapine and risperidone in the management of first-episode schizophrenia. 2016

- November;144(5):697 The Indian Journal of Medical Research.
26. "Suicidal risk during treatment with clozapine: a meta-analysis," by John Hennen and Ross J. Baldessarini. *Research on schizophrenia* 73.2-3 (2005): 139–145.
 27. "Effect of aripiprazole augmentation of clozapine in schizophrenia: a double-blind, placebo-controlled study," Muscatello, Maria Rosaria A., et al. *Research on schizophrenia* 127.1-3 (2011): 93–99.
 28. "Effects of cognitive behavioural therapy on work outcomes in vocational rehabilitation for participants with schizophrenia spectrum disorders," by Paul H. Lysaker et al., 28. *Research on schizophrenia* 107.2-3 (2009): 186–191.
 29. Individual placement and support: an evidence-based strategy for assisted work. Drake et al. 2012; Oxford University Press.
 30. "Supported employment for adults with severe mental illness," Kinoshita et al. *Database of Systematic Reviews Cochrane* 9 (2013).
 31. The unhealthy living of individuals with schizophrenia, by Steve Brown et al. *Medical psychology* 29.3 (1999): 697–701.
 32. "Dietary intake of schizophrenic patients in Nithsdale, Scotland: case-control study," by Robin McCreadie et al. *Bmj* 317.7161 (1998): 784-785.
 33. Weight gain in male teenage schizophrenic inpatients treated with olanzapine is correlated with increased food consumption and poor habitual exercise levels, according to Gothelf et al. 159.6 (2002): 1055–1057 in the *American Journal of Psychiatry*.
 34. The gluten connection: the link between celiac disease and schizophrenia, 34 Kalaydjian, A. E., et al. 82–90 are included in *Acta Psychiatrica Scandinavica* 113.2 (2006).
 35. In the postmortem orbitofrontal cortex of individuals with bipolar disorder, there are deficiencies in docosahexaenoic acid and related elevations in the metabolism of arachidonic acid and saturated fatty acids. *Research in psychiatry* 160.3 (2008): 285-299.
 36. Relationship of low vitamin D levels with positive, negative, and cognitive symptom categories in individuals with first-episode schizophrenia, Graham, K. A., et al. *Early Intervention in Psychiatry*, 9, 3, 397–405, 2015.
 37. *Psychiatry (Edgmont)* 2.2 (2005): 31. Strassnig, "Dietary intake of patients with schizophrenia."
 38. Schizophrenia, gluten, and low-carb, ketogenic diets: a case report and literature survey, 38. Kraft. 2009: 1-3; *Nutrition & Metabolism* 6.1.
 39. "Mortality in people with schizophrenia in rural China," by Mao-Sheng, Ran, et al. (2007) 237–242 *The British Journal of Psychiatry*, 190.3.
 40. Jamie Joseph et al. published a study titled "Modified Mediterranean diet for enrichment of short chain fatty acids: potential adjunctive therapeutic to target immune and metabolic dysfunction in schizophrenia?" 40. *Neuroscience Frontiers* 11 (2017): 155.
 41. "Holistic management of schizophrenia symptoms using pharmacological and non-pharmacological treatment," by Ganguly et al. (2018). *Frontiers in Public Health* 6: 166.
 42. Results of a 2-year randomised study on the effects of cognitive enhancement treatment on job outcomes in early schizophrenia, Eack et al. 42. *Social Work Practice Research* 21.1 (2011): 32–42.
 43. "Computerized cognitive training restores neural activity within the reality monitoring



- network in schizophrenia," by Subramaniam et al. 43. (2012) 842–885 in *Neuron* 73.4.
44. Gumley, A., et al. 44. Iacp president's message stefan g. Hofmann, phd primum non necera 47 (2015): 11 "quiet narcissism continued from pg. 9."
45. "Suicides during pregnancy and 1 year postpartum in Sweden, 1980-2007," by Esscher et al. 462-469 in *British Journal of Psychiatry* 208.5 (2016).
46. "Yoga therapy for schizophrenia," by Arun Jha, 46. (2008): 397-397, *Acta Psychiatrica Scandinavica* 117.5.
47. 47. "Development of the beliefs about yoga scale," by Sohl et al. 21.1 (2011): 85–91 *International Journal of Yoga Therapy*.
48. "Systematic review of the advantages of physical therapy within a multidisciplinary care approach for people with schizophrenia," Vancampfort et al. 48. 2012;92(1):11–23; *Physical Therapy*.
49. David Feifel 49. Is oxytocin a potentially effective therapy for schizophrenia? *Neurotherapeutics expert review* 11.2 (2011): 157–159.
50. 50. "Substance use among schizophrenic outpatients: prevalence, course, and relation to functional status." Chouljian, Tandy L., et al. 1995: 19–24, *Annals of Clinical Psychiatry* 7.1.
51. The trajectory of schizophrenia over 13 years: a study from the World Health Organization-led International Study on Schizophrenia (ISoS), by Mason et al. 1996: 580-586 in *The British Journal of Psychiatry*, 169.5.
52. "Violent and destructive behaviour among the severely mentally ill in rural areas: evidence from Arkansas' community mental health system," by Brian J. Cuffel, 52. *Journal of Community Mental Health* 30.5 (1994): 495–504.
53. "Improving treatment adherence in patients with schizophrenia," M. Kane John, 53. *Psychiatry Research and Practice* 72.9 (2011): 27738.
54. Wang, among others. "Tobacco increases protection against oxidative stress by overexpressing an Arabidopsis peroxisomal ascorbate peroxidase gene." 725–732, 1999, *Plant and Cell Physiology* 40.7.
55. Alcohol and weed use in schizophrenia: impacts of clozapine vs. risperidone, Green, Alan I., et al. *Research on schizophrenia* 60.1 (2003): 81–85.

HOW TO CITE: Monali Khatake*, Vinayak Gaware, Rahul Khaire, Charushila Bhangale, Overview and Treatments of Schizophrenia: A Recent Update, *Int. J. in Pharm. Sci.*, 2023, Vol 1, Issue 4, 153-165. <https://doi.org/10.5281/zenodo.7847211>

