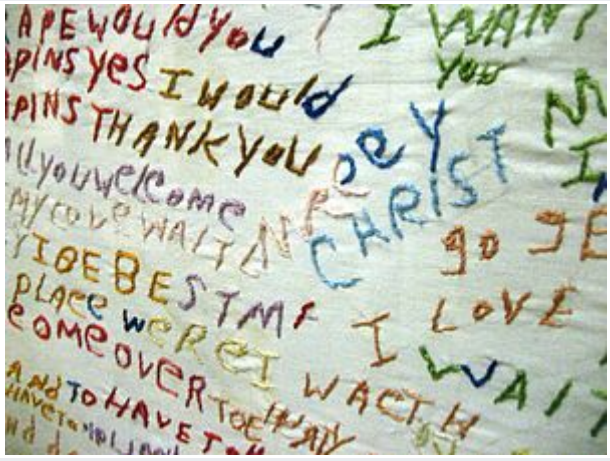
Schizophrenia

Schizophrenia is a mental illness characterized by episodes of psychosis with hearing voices, delusions (false beliefs), and disordered thinking.[12][2] Other symptoms may include social withdrawal, decreased emotional expression, and lack of motivation.[2][3] Symptoms typically come on gradually, begin in young adulthood, and in many cases never resolve.[3] As of 2019, there is no objective diagnostic test;[13] diagnosis is based on observed behavior, and a history that includes the person's reported experiences, and reports of others familiar with the person. To be diagnosed with schizophrenia, symptoms and functional impairment need to be present for six months.[6][12] People with schizophrenia often have other mental health problems like anxiety disorders such as obsessive-compulsive disorder, depression, or a substance-use disorder.[14]

About 0.3% to 0.7% of people are affected by schizophrenia during their lifetimes.[10] In 2017, there were an estimated 1.1 million new cases and a total of 19.8 million cases globally.[15] Males are more often affected and onset is on average earlier in age.[2] The causes of schizophrenia include environmental and genetic factors.[5] Possible environmental factors include being raised in a city, cannabis use during adolescence, certain infections, the ages of a person's parents, and poor nutrition during pregnancy.[5][16] Genetic factors include a variety of common and rare genetic variants.[17]

About 20% of people with schizophrenia have a favorable outcome, and some individuals recover completely.[6] About 50% have lifelong impairment.[18] Social problems, such as long-term unemployment, poverty, and homelessness, are common.[6][19] Compared to the general population, people with schizophrenia have a higher suicide rate (about 5% overall) and more physical health problems,[10][20] leading to an average decreased life

Schizophrenia



Cloth embroidered by a person diagnosed with schizophrenia

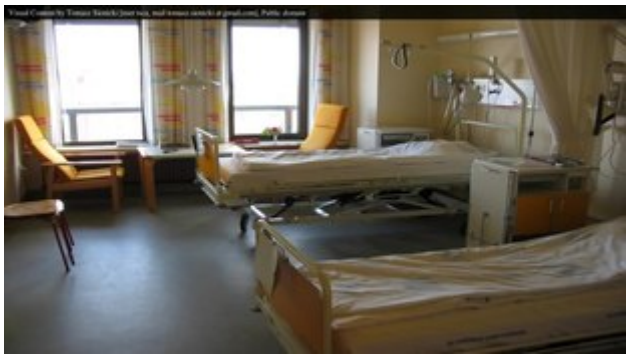
Pronunciation	/ˌskɪtsəˈfriːniə/, UK also /ˌskɪdzə-/, US also /-ˈfrɛniə/[1]
Specialty	Psychiatry
Symptoms	delusions, confused thinking, hearing voices others do not[2][3]
Complications	Suicide, heart disease, lifestyle diseases[4]
Usual onset	Ages 16 to 30[3]
Duration	Chronic[3]
Causes	Environmental and genetic factors[5]
Risk factors	Family history, cannabis use, problems during pregnancy, being raised in a city, older father[5]
Diagnostic method	Based on observed behavior, reported experiences, and reports of others familiar with the person[6]
Differential diagnosis	Substance abuse, Huntington's disease, mood disorders (bipolar disorder),

expectancy of 20 years.[9] In 2015, an estimated 17,000 people worldwide died from behavior related to, or caused by, schizophrenia.[11]

The mainstay of treatment is an antipsychotic medication, along with counselling, job training, and social rehabilitation.[5] In those who do not improve with other antipsychotics, clozapine may be tried.[5] In situations where there is a risk of harm to self or others, involuntary hospitalization may be necessary, although hospital stays are shorter and less frequent than they once were.[21]

	autism[7] borderline personality disorder[8]
Treatment	Counseling, job training[2][5]
Medication	Antipsychotics[5]
Prognosis	20 years shorter life expectancy[9][4]
Frequency	~0.5%[10]
Deaths	~17,000 (2015)[11]

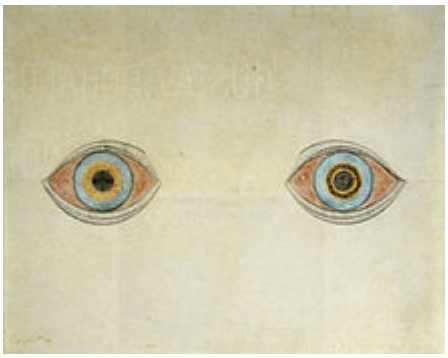
Contents



Play media

Video summary (script)

Signs and symptoms



My Eyes at the Moment of the Apparitions by German artist August Natterer, who had schizophrenia

Schizophrenia is characterized by episodes of psychosis. Psychoses can occur in several conditions and are often transient making early diagnosis of schizophrenia problematic. Psychosis noted for the first time in a person who is later diagnosed with schizophrenia is referred to as a first-episode psychosis (FEP). Another well referenced term is duration of untreated psychosis (DUP) that optimally needs to be as short as possible.[12][22]

A person with schizophrenia may experience hallucinations (most commonly hearing voices), delusions (often bizarre or persecutory in nature), and thought disorders that include disorganized thinking and speech, speech that is not understandable known as word salad, and thought blocking.[3][23] Other common sysmptoms are social withdrawal, self-neglect particularly in hygiene, and self-care, and loss of motivation and judgment.[24]

Distortions of self-experience such as feeling as if one's thoughts or feelings are not really one's own to believing thoughts are being inserted into one's mind, sometimes termed passivity phenomena, are also common.[25] There is often an observable pattern of emotional difficulty, for example lack of responsiveness.[26] Impairment in social cognition is associated with schizophrenia,[27][28] as are symptoms of paranoia. Social isolation commonly occurs.[29] Difficulties in working and long-term memory, attention, executive functioning, and speed of processing also commonly occur.[10] People with schizophrenia often find facial emotion perception to be difficult.[30]

Schizophrenia is often described in terms of positive, and negative symptoms, and cognitive symptoms.[3][31] The severity of the positive and negative symptoms can be assessed using the Positive and Negative Syndrome Scale (PANSS).[32]

Positive symptoms

Positive symptoms are those symptoms that are not normally experienced, but are present in people with schizophrenia. They can include delusions, disordered thoughts and speech, and hallucinations, typically regarded as manifestations of psychosis.[12] Hallucinations can involve any of the senses including taste, sight, touch, and hearing.[33] They are also typically related to the content of the delusional theme.[34] Positive symptoms generally respond well to medication.[34]

Negative symptoms

Negative symptoms are deficits of normal emotional responses or of other thought processes.[24] They commonly include flat expressions or little emotion, poverty of speech, inability to experience pleasure, lack of desire to form relationships, and lack of motivation. Negative symptoms appear to contribute more to poor quality of life, functional ability, and the burden on others than positive symptoms do.[18][35] People with greater negative symptoms often have a history of poor adjustment before the onset of illness. Negative symptoms are less responsive to medication.[24][36]

Cognitive symptoms

Cognitive symptoms are deficits in cognition, and are the earliest and most constantly found symptoms in schizophrenia.[37][38] The presence and degree of cognitive dysfunction in people with schizophrenia has been reported to be a better indicator of functionality than the presentation of positive or negative symptoms.[39] The deficits impacting the cognitive function are found in a large number of areas: working memory, long-term memory,[40][41] verbal declarative memory,[42] semantic processing,[43] episodic memory,[44] attention,[18] learning (particularly verbal learning).[40] Deficits in verbal memory are the most pronounced in someone with schizophrenia, and are not accounted for by deficit in attention. Verbal memory impairment has been linked to a decreased ability in those with schizophrenia to semantically encode (process information relating to meaning), which is cited as a cause for another known deficit in long-term memory.[40] From a list of words people usually tend to remember positive words, but in schizophrenia all words are remembered equally, suggesting that anhedonia impairs the semantic encoding of words.[40] These deficits have been found in people before the onset of the illness to some extent.[39][45][46] First-degree relatives of those with schizophrenia and other high-risk people also show a degree of deficit in cognitive abilities, and specifically in working memory.[46] A review of the literature on cognitive deficits in people with schizophrenia shows that the deficits may be present in early adolescence, or as early as childhood.[39] The deficits which a person with schizophrenia presents tend to remain the same over time in most patients, or follow an identifiable course based upon environmental variables.[39][40]

Although the evidence that cognitive deficits remain stable over time is reliable and abundant,[40][44] much of the research in this domain focuses on methods to improve attention and working memory.[40][41] Efforts to improve learning ability in people with schizophrenia using a high- versus low-reward condition and an instruction-absent or instruction-present condition revealed that increasing reward leads to poorer performance while providing instruction leads to improved performance, highlighting that some treatments may exist to increase cognitive performance.[40] Training people with schizophrenia to alter their thinking, attention, and language behaviors by verbalizing tasks, engaging in cognitive rehearsal, giving self-instructions, giving coping statements to the self to handle failure, and providing self-reinforcement for success, improves performance on recall tasks.[40] This type of training, known as self-instructional (SI) training, produced benefits such as lower number of nonsense verbalizations and improved recall when distracted.[40]

Onset

Late adolescence and early adulthood are peak periods for the onset of schizophrenia,[10] critical years in a young adult's social and vocational development.[47] In 40% of men and 23% of women diagnosed with schizophrenia, the condition manifested itself before the age of 19.[48] The most general symptoms of schizophrenia tend to appear between ages 16 and 30.[3][6] The onset of the disorder is usually between ages 18 and 25 for men and between 25 and 35 for women.[49] To minimize the developmental disruption associated with schizophrenia, much work has been done to identify and treat the prodromal (pre-onset) phase of schizophrenia, which has been detected up to 30 months before the onset of symptoms.[47] Those who go on to develop schizophrenia may experience transient or self-limiting psychotic symptoms[50] and the non-specific symptoms of social withdrawal, irritability, dysphoria,[51] and clumsiness before the onset of the disease.[52] Children who go on to develop schizophrenia may also demonstrate decreased intelligence, decreased motor development (reaching milestones such as walking slowly), isolated play preference, social anxiety, and poor school performance.[53][54][55]

Causes

A combination of genetic and environmental factors play a role in the development of schizophrenia.[10][56] People who have a transient psychosis, who also have a family history of schizophrenia have a 20–40% chance of being diagnosed one year later.[57]

Genetic

Estimates of the heritability of schizophrenia are around 80%, which implies that 80% of the individual differences in risk to schizophrenia is associated with genetics.[58][59] These estimates vary because of the difficulty in separating genetic and environmental influences and some have labeled these estimates inaccurate.[60][61] The greatest single risk factor for developing schizophrenia is having a first-degree relative with the disease (risk is 6.5%); more than 40% of identical twins of those with schizophrenia are also affected.[56] If one parent is affected the risk is about 13% and if both are affected the risk is nearly 50%.[58] Results of candidate gene studies of schizophrenia have generally failed to find consistent associations,[62] and the genetic loci identified by genome-wide association studies as associated with schizophrenia explain only a small fraction of the variation in the disease.[63]

Many genes are known to be involved in schizophrenia, each with small effect and unknown transmission and expression.[17][64] The summation of these effect sizes into a polygenic risk score can explain at least 7% of the variability in liability for schizophrenia.[65] Around 5% of cases of schizophrenia are understood to be at least partially attributable to rare copy number variants (CNVs), including 22q11, 1q21 and 16p11.[66] These rare CNVs increase the risk of someone developing the disorder by as much as 20-fold, and are frequently comorbid with autism and intellectual disabilities.[66]

The question of how schizophrenia could be primarily genetically influenced, given that people with schizophrenia have lower fertility rates, is a paradox. It is expected that genetic variants that increase the risk of schizophrenia would be selected against due to their negative effects on reproductive fitness. A number of potential explanations have been proposed, including that alleles associated with schizophrenia risk confers a fitness advantage in unaffected individuals.[67][68] While some evidence has not supported this idea,[61] others propose that a large number of alleles each contributing a small amount can persist.[69]

Environment

Environmental factors associated with the development of schizophrenia include the living environment, drug use, and prenatal stressors.[10]

Prenatal maternal stress has been associated with an increased risk of schizophrenia, possibly in association with reelin.[70] Maternal nutritional deficiencies, as well as maternal obesity have also been identified as possible risk factors for schizophrenia. Both maternal stress and infection have been demonstrated to alter fetal neurodevelopment through pro-inflammatory proteins such as IL-8 and TNF.[71][72]

Parenting style seems to have no major effect, although people with supportive parents do better than those with critical or hostile parents.[56] Childhood trauma, death of a parent, and being bullied or abused increase the risk of psychosis.[73][74] Living in an urban environment during childhood or as an adult has consistently been found to increase the risk of schizophrenia by a factor of two,[10][56] even after taking

into account drug use, ethnic group, and size of social group.[75] Other factors that play an important role include social isolation and immigration related to social adversity, racial discrimination, family dysfunction, unemployment, and poor housing conditions.[56][76]

It has been hypothesized that in some people, development of schizophrenia is related to intestinal tract dysfunction such as seen with non-celiac gluten sensitivity or abnormalities in the intestinal flora.[77] A subgroup of persons with schizophrenia present an immune response to gluten differently from that found in people with celiac, with elevated levels of certain serum biomarkers of gluten sensitivity such as anti-gliadin IgG or anti-gliadin IgA antibodies.[78]

Substance use

About half of those with schizophrenia use recreational drugs, including cannabis, nicotine, and alcohol excessively.[79][80] Recreational drugs include stimulants such as amphetamine, and cocaine, that can lead to a temporary stimulant psychosis, that presents very similarly to schizophrenia. Alcohol use can also result in a similar alcohol-related psychosis.[56][81]

Cannabis may be a contributory factor in schizophrenia, potentially increasing the risk of the disease in those who are already at risk.[16] The increased risk may require the presence of certain genes within an individual.[16] Among those who are at risk of psychosis, it is associated with twice the rate.[82]

Drugs may be also be used as coping mechanisms by people who have schizophrenia, to deal with depression, anxiety, boredom, and loneliness.[79][83]

Developmental factors

Factors such as oxygen deprivation, infection, prenatal maternal stress, and malnutrition in the mother during fetal development, may result in a slight increase in the risk of schizophrenia later in life.[10] There is a slighter risk associated with being born in the winter or spring possibly due to a prenatal viral infection.[56] Other infections during pregnancy or around the time of birth that have been linked to an increased risk include infections by *Toxoplasma gondii* and *Chlamydia*.[84] The increased risk is about five to eight percent.[85] Viral infections of the brain during childhood are also linked to a risk of schizophrenia during adulthood.[86]

Mechanisms

While the mechanisms of schizophrenia are unknown, a number of attempts have been made to explain the link between altered brain function and schizophrenia.[10] One of the most common is the dopamine hypothesis, which attributes psychosis to the mind's faulty interpretation of the misfiring of dopaminergic neurons.[10] Other possible mechanisms include glutaminergic neurotransmission and neurodevelopment. Frameworks have hypothesized links between these biological abnormalities and symptoms.[87]

Abnormal dopamine signalling has been implicated in schizophrenia based on the usefulness of medications that effect the dopamine receptor and the observation that dopamine levels are increased during acute psychosis.[88][89] Abnormalities in dopamine signalling have been hypothesized to underlie delusions.[90][91][92] A decrease in D_1 receptors in the prefrontal cortex may also be responsible for deficits in working memory.[93][94][95][96]

Reduced NMDA receptor signalling is suggested by multiple lines of evidence. Studies demonstrate reduced NMDA receptor expression and NMDA receptor blockers mimic both schizophrenia symptoms and the physiological abnormalities associated with schizophrenia.[97][98][99] Post-mortem studies consistently find that a subset of these neurons fail to express GAD67,[100] in addition to abnormalities in morphology. The subsets of interneurons that are abnormal in schizophrenia are responsible for the synchronizing of neural ensembles that is necessary during working memory tasks, a process that is electrophysiologically reflected in gamma frequency (30–80 Hz) oscillations. Both working memory tasks and gamma oscillations are impaired in schizophrenia, which may reflect abnormal interneuron functionality.[100][101][102][103]

Evidence suggest that schizophrenia has a neurodevelopmental component. Before the onset of schizophrenia there is often impairments in cognition, social functioning, and motor skills.[104] Furthermore, problems before birth such as maternal infection,[105][106] maternal malnutrition and complications during pregnancy all increase risk for schizophrenia.[71] Schizophrenia usually emerges 18-25, an age period that overlaps with certain stages of neurodevelopment that are implicated in schizophrenia.[107]

Deficits in executive functions, such as planning, inhibition, and working memory, are pervasive in schizophrenia. Although these functions are dissociable, their dysfunction in schizophrenia may reflect an underlying deficit in the ability to represent goal related information in working memory, and to utilize this to direct cognition and behavior.[108][109] These impairments have been linked to a number of neuroimaging and neuropathological abnormalities. For example, functional neuroimaging studies report evidence of reduced neural processing efficiency, whereby the dorsolateral prefrontal cortex is activated to a greater degree to achieve a certain level of performance relative to controls on working memory tasks. These abnormalities may be linked to the consistent post-mortem finding of reduced neuropil, evidenced by increased pyramidal cell density and reduced dendritic spine density. These cellular and functional abnormalities may also be reflected in structural neuroimaging studies that find reduced grey matter volume in association with deficits in working memory tasks.[110]

Positive and negative symptoms have been linked to reduced cortical thickness in the superior temporal lobe,[111] and orbitofrontal cortex, respectively.[112] Anhedonia, traditionally defined as a reduced capacity to experience pleasure, is frequently reported in schizophrenia. However, a large body of evidence suggests that hedonic responses are intact in schizophrenia,[113] and that what is reported to be anhedonia is a reflection of dysfunction in other processes related to reward.[114] Overall, a failure of reward prediction is thought to lead to impairment in the generation of cognition and behavior required to obtain rewards, despite normal hedonic responses.[115]

Bayesian models of brain functioning have been utilized to link abnormalities in cellular functioning to symptoms.[116][117] Both hallucinations and delusions have been suggested to reflect improper encoding of prior expectations, thereby causing expectation to excessively influence sensory perception and the formation of beliefs. In approved models of circuits that mediate predictive coding, hypoactive NMDA receptor activation, similar to that seen in schizophrenia, could theoretically result in classic symptoms of schizophrenia such as delusions and hallucinations.[118][119]

Diagnosis

Schizophrenia is characterised by episodes of psychosis.[12] Psychoses can occur in several conditions and are often transient making early diagnosis of schizophrenia problematic. Psychosis noted for the first time in a person that is later diagnosed with schizophrenia is referred to as a first-episode psychosis (FEP). Diagnosis of schizophrenia needs a period of six months where symptoms are noted, however a different diagnosis of schizophreniform disorder can be made before the six months needed for schizophrenia.[120] The time between the onset of symtoms to being given treatment – the duration of untreated psychosis (DUP) needs to be as short as possible.[22]

Schizophrenia is diagnosed based on criteria in either the American Psychiatric Association's (APA) fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5) or the World Health Organization's International Statistical Classification of Diseases and Related Health Problems (ICD-10). These criteria use the self-reported experiences of the person and reported abnormalities in behavior, followed by a psychiatric assessment. The mental status examination is an important part of the assessment.[121] Symptoms associated with schizophrenia occur along a continuum in the population and must reach a certain severity and level of impairment before a diagnosis is made.[56] An established tool for assessing the severity of positive and negative symptoms is the Positive and Negative Syndrome Scale (PANSS).[32] As of 2019, there is no objective test or biomarker to confirm diagnosis.[13]

Criteria

In 2013, the APA released the fifth edition of the DSM (DSM-5). To be diagnosed with schizophrenia, two diagnostic criteria have to be met over the period of one month, with a significant impact on social or occupational functioning for at least six months. The person had to be suffering from delusions, hallucinations, or disorganized speech. A second symptom could be negative symptoms, or severely disorganized or catatonic behaviour.[120] The definition of schizophrenia remains essentially the same as that specified by the 2000 version of DSM (DSM-IV-TR), but DSM-5 makes a number of changes.

- Subtype classifications – such as catatonic and paranoid schizophrenia – are removed. These were retained in previous revisions largely for reasons of tradition, but had subsequently proved to be of little worth.[122]
- Catatonia is no longer so strongly associated with schizophrenia.[123]
- In describing a person's schizophrenia, it is recommended that a better distinction be made between the current state of the condition and its historical progress, to achieve a clearer overall characterization.[122]
- Special treatment of Schneider's first-rank symptoms is no longer recommended.[122]
- Schizoaffective disorder is better defined to demarcate it more cleanly from schizophrenia.[122]
- An assessment covering eight domains of psychopathology – such as whether hallucination or mania is experienced – is recommended to help clinical decision-making.[124]

The ICD-10 criteria are typically used in European countries; the DSM criteria are used in the United States and some other countries, and are prevailing in research studies. The ICD-10 criteria put more emphasis on Schneiderian first-rank symptoms. In practice, agreement between the two systems is high.[125] The current proposal for the ICD-11 criteria for schizophrenia recommends adding self-disorder as a symptom.[25]

If signs of disturbance are present for more than a month but less than six months, the diagnosis of schizophreniform disorder is applied. Psychotic symptoms lasting less than a month may be diagnosed as brief psychotic disorder, and various conditions may be classed as psychotic disorder not otherwise

specified; schizoaffective disorder is diagnosed if symptoms of mood disorder are substantially present alongside psychotic symptoms. If the psychotic symptoms are the direct physiological result of a general medical condition or a substance, then the diagnosis is one of a psychosis secondary to that condition.[120] Schizophrenia is not diagnosed if symptoms of pervasive developmental disorder are present unless prominent delusions or hallucinations are also present.[120]

Subtypes

With the publication of DSM-5, the APA removed all sub-classifications of schizophrenia.[126] The five sub-classifications included in DSM-IV-TR were:[127][128]

- Paranoid type: Delusions or auditory hallucinations are present, but thought disorder, disorganized behavior, or affective flattening are not. Delusions are persecutory and/or grandiose, but in addition to these, other themes such as jealousy, religiosity, or somatization may also be present. (DSM code 295.3/ICD code F20.0)
- Disorganized type: Named *hebephrenic schizophrenia* in the ICD. Where thought disorder and flat affect are present together. (DSM code 295.1/ICD code F20.1)
- Catatonic type: The subject may be almost immobile or exhibit agitated, purposeless movement. Symptoms can include catatonic stupor and waxy flexibility. (DSM code 295.2/ICD code F20.2)
- Undifferentiated type: Psychotic symptoms are present but the criteria for paranoid, disorganized, or catatonic types have not been met. (DSM code 295.9/ICD code F20.3)
- Residual type: Where positive symptoms are present at a low intensity only. (DSM code 295.6/ICD code F20.5)

The ICD-10 defines additional subtypes:[127]

- Post-schizophrenic depression: A depressive episode arising in the aftermath of a schizophrenic illness where some low-level schizophrenic symptoms may still be present. (ICD code F20.4)
- Simple schizophrenia: Insidious and progressive development of prominent negative symptoms with no history of psychotic episodes. (ICD code F20.6)
- Other schizophrenia include cenesthopathic schizophrenia and schizophreniform disorder NOS (ICD code F20.8).[129]

Differential diagnosis

Psychotic symptoms may be present in several other mental disorders, including bipolar disorder,[7] borderline personality disorder,[8] substance intoxication, and substance-induced psychosis. Non-bizarre delusions are also present in delusional disorder, and social withdrawal in social anxiety disorder, avoidant personality disorder and schizotypal personality disorder. Schizotypal personality disorder has symptoms that are similar but less severe than those of schizophrenia.[6] Schizophrenia occurs along with obsessive-compulsive disorder (OCD) considerably more often than could be explained by chance, although it can be difficult to distinguish obsessions that occur in OCD from the delusions of schizophrenia.[130] Benzodiazepine withdrawal syndrome when severe, can resemble schizophrenia and be misdiagnosed as such.[131]

A more general medical and neurological examination may be needed to rule out medical illnesses which may rarely produce psychotic schizophrenia-like symptoms, such as metabolic disturbance, systemic infection, syphilis, AIDS dementia complex, epilepsy, limbic encephalitis, and brain lesions. Stroke,

multiple sclerosis, hyperthyroidism, hypothyroidism, and dementias such as Alzheimer's disease, Huntington's disease, frontotemporal dementia, and the Lewy body dementias may also be associated with schizophrenia-like psychotic symptoms.[132] It may be necessary to rule out a delirium, which can be distinguished by visual hallucinations, acute onset and fluctuating level of consciousness, and indicates an underlying medical illness. Investigations are not generally repeated for relapse unless there is a specific *medical* indication or possible adverse effects from antipsychotic medication. In children hallucinations must be separated from typical childhood fantasies.[6]

Prevention

Prevention of schizophrenia is difficult as there are no reliable markers for the later development of the disorder.[133] There is tentative evidence for the effectiveness of early interventions to prevent schizophrenia.[134] There is some evidence that early intervention in those with a psychotic episode may improve short-term outcomes, but there is little benefit from these measures after five years.[10] Attempting to prevent schizophrenia in the prodrome phase is of uncertain benefit and therefore as of 2009 is not recommended.[135] Cognitive behavioral therapy may reduce the risk of psychosis in those at high risk after a year[136] and is recommended in this group, by the National Institute for Health and Care Excellence (NICE).[137] Another preventive measure is to avoid drugs that have been associated with development of the disorder, including cannabis, cocaine, and amphetamines.[56]

Management

The primary treatment of schizophrenia is antipsychotic medications, often in combination with psychological and social supports.[10] Community support services including drop-in centers, visits by members of a community mental health team, supported employment[138] and support groups are common. Hospitalization may occur for severe episodes either voluntarily or (if mental health legislation allows it) involuntarily. Long-term hospitalization is uncommon since deinstitutionalization beginning in the 1950s, although it still occurs.[21]

Exercise therapy has been shown to improve positive and negative symptoms, cognition, and improve quality of life.[139] Aerobic exercise has been shown to improve cognitive deficits of working memory and attention.[140] Exercise has also been shown to increase the volume of the hippocampus in those with schizophrenia. A decrease in hippocampal volume is one of the factors linked to the development of the disease.[139] However, there still remains the problem of increasing motivation for, and maintaining participation in physical activity.[141] Supervised sessions are recommended.[140]

Medication

The first-line treatment for schizophrenia is an antipsychotic medication. Antipsychotics block the effects of dopamine a neurotransmitter. The first-generation antipsychotics, now called typical antipsychotics, only affect dopamine levels. Those brought out later, the second-generation antipsychotics known as atypical antipsychotics, can also have effect on another neurotransmitter serotonin. Antipsychotics can reduce the symptoms of anxiety within hours of their use but for other symptoms they may take several days or weeks to reach their full effect.[142][12] They have little effect on negative and cognitive symptoms, which may be helped by additional psychotherapies and medications.[143]

Risperidone (trade name Risperdal) is a common atypical antipsychotic medication.

After a first-episode psychosis where there has been a full recovery with no symptoms for twelve months, stopping medication may be considered. Up to 40% in these cases remain well though some may need to continue on low doses. Where there has been a second relapse but with no further symptoms after a full year, antipsychotics may be reduced. Repeated psychotic episodes worsen the long-term outlook and the risk of relapse following a second episode is high, and long term treatment is usually recommended.[144][145] Antipsychotics may need to be stopped or switched, if a person fails to improve adequately or has associated adverse effects. This needs to be closely monitored, and should take place over weeks or months, except in urgent situations such as the development of agranulocytosis from the use of clozapine. In this type of abrupt stoppage a more severe rebound psychosis can occur.[144]

There is no single antipsychotic suitable for first-line treatment for everyone, as responses and tolerances vary between people.[146] Atypical antipsychotics such as amisulpride, olanzapine, risperidone, and clozapine may be more effective but are associated with greater side effects.[147] There is a good response in 40–50%, a partial response in 30–40%, and treatment resistance (failure of symptoms to respond satisfactorily after six weeks to two or three different antipsychotics) in 20% of people.[36] Clozapine is an effective treatment for those who respond poorly to other drugs ("treatment-resistant" or "refractory" schizophrenia),[148] but it has the potentially serious side effect of agranulocytosis (lowered white blood cell count) in less than 4% of people.[10][56][149]

Most people on antipsychotics have side effects. People on typical antipsychotics tend to have a higher rate of extrapyramidal side effects as movement disorders; some atypicals are associated with considerable weight gain, diabetes and risk of metabolic syndrome. This is most pronounced with olanzapine; risperidone and quetiapine are also associated with weight gain.[147] Risperidone has a similar rate of extrapyramidal symptoms to haloperidol.[147] It remains unclear whether the newer antipsychotics reduce the chances of developing neuroleptic malignant syndrome or tardive dyskinesia, a rare but serious neurological disorder.[150]

About 30 to 50 percent of people with schizophrenia fail to accept that they have an illness or comply with their recommended treatment.[151] For those who are unwilling or unable to take medication regularly, long-acting depot preparations of antipsychotics may be used to achieve control.[152] They reduce the risk of relapse to a greater degree than oral medications.[153] When used in combination with psychosocial interventions, they may improve long-term adherence to treatment.[152]

Supplements

Disruption of the gut microbiota has been linked to inflammation, and disorders of the central nervous system. This includes schizophrenia, and probiotic supplementation has been proposed to improve its symptoms. A review found no evidence to support this but it concludes that probiotics may be of benefit in regulating bowel movements and lessening the metabolic effects of antipsychotics.[154]

Interventions

A number of interventions that include several types of psychotherapy may be useful in the treatment of schizophrenia such as: family therapy,[155] group therapy, cognitive remediation therapy,[156] cognitive behavioral therapy, and metacognitive training.[157] Skills training, and help with substance use, and weight management– often needed as a side effect of an antipsychotic, are also offered.[158] In the US, interventions for first episode psychosis have been brought together in an overall approach known as coordinated speciality care (CSC) and also includes support for education.[12] In the UK *care across all phases* is a similar approach that covers many of the treament guidelines recommended.[137] The aim is to reduce the number of relapses and stays in hospital.[155]

Other support services for education, employment, and housing are usually offered. For people suffering from severe schizophrenia, and discharged from a stay in hospital, these services are often brought together in an integrated approach to offer support in the community away from the hospital setting. In addition to medicine management, housing, and finances, assistance is given for more routine matters such as help with shopping and using public transport. This approach is known as assertive community treatment (ACT) and has been shown to achieve positive results in symptoms, social functioning and quality of life.[159][160] Another more intense approach is known as *intensive care management* (ICM). ICM is a stage further than ACT and emphasises support of high intensity in smaller caseloads, (less than twenty). This approach is to provide long-term care in the community. Studies show that ICM improves many of the relevant outcomes including social functioning.[161]

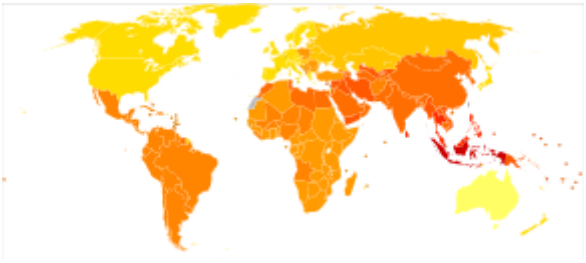
Some studies have shown little evidence for the effectiveness of cognitive behavioral therapy (CBT) in either reducing symptoms or preventing relapse.[162][163] Other studies have found that CBT improves overall psychotic symptoms, but has no effect on social function, relapse, or quality of life.[164] In the UK it is recommended as an add-on therapy in the treatment of schizophrenia, but is not supported for use in treatment resistant schizophrenia.[163][165] Arts therapies are seen to improve negative symptoms in some people, and are recommended by NICE in the UK.[166][167] This approach however, is criticised as having not been well-researched, and arts therapies are not recommended in Australian guidelines for example.[167][168][169] Peer support, in which people with personal experience of schizophrenia, provide help to each other, is of unclear benefit.[170]

Prognosis

Schizophrenia has great human and economic costs.[10] It results in a decreased life expectancy of 20 years.[9][4] This is primarily because of its association with obesity, poor diet, sedentary lifestyles, and smoking, with an increased rate of suicide playing a lesser role.[10][9][171] Antipsychotic medications may also increase the risk.[9] These differences in life expectancy increased between the 1970s and 1990s.[172] Primary polydipsia, or excessive fluid intake, is relatively common in people with chronic schizophrenia.[173][174] This may lead to hyponatremia which can be life-threatening. Antipsychotics can lead to a dry mouth, but there are several other factors that may contribute to the disorder. It is suggested to lead to a reduction in life expectancy by 13 per cent.[174]

Schizophrenia is a major cause of disability, with active psychosis ranked as the third-most-disabling condition after quadriplegia and dementia and ahead of paraplegia and blindness.[175] Approximately three-fourths of people with schizophrenia have ongoing disability with relapses[36] and 16.7 million people globally are deemed to have moderate or severe disability from the condition.[176] Some people do recover completely and others function well in society.[177] Most people with schizophrenia live independently with community support.[10] About 85% are unemployed.[5] In people with a first episode of psychosis a good long-term outcome occurs in 42%, an intermediate outcome in 35% and a poor

outcome in 27%.[178] Outcomes for schizophrenia appear better in the developing than the developed world.[179] These conclusions have been questioned.[180][181]



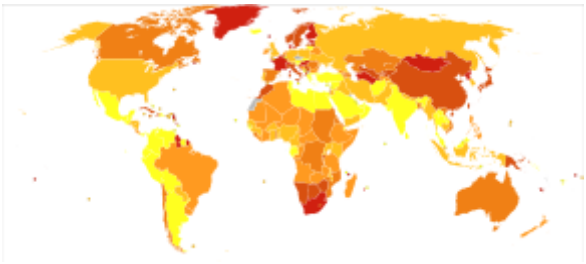
Disability-adjusted life years lost due to schizophrenia per 100,000 inhabitants in 2004.

- no data
- ≤ 185
- 185–197
- 197–207
- 207–218
- 218–229
- 229–240
- 240–251
- 251–262
- 262–273
- 273–284
- 284–295
- ≥ 295

There is a higher than average suicide rate associated with schizophrenia. This had been cited at 10%, but has been revised to an estimate of 4.9%, most often occurring in the period following onset or first hospital admission.[20][182] Several times more (20 to 40%) attempt suicide at least once.[6][183] There are a variety of risk factors, including male gender, depression, and a high intelligence quotient.[183]

Schizophrenia and smoking have shown a strong association in studies worldwide.[184][185] Use of cigarettes is especially high in those diagnosed with schizophrenia, with estimates ranging from 80 to 90% being regular smokers, as compared to 20% of the general population.[185] Those who smoke tend to smoke heavily, and additionally smoke cigarettes with high nicotine content.[186] Some propose that this is in an effort to improve symptoms.[187] Among people with schizophrenia use of cannabis is also common.[79]

Epidemiology



Deaths per million persons due to schizophrenia in 2012.

- 0–0
- 1–1
- 2–2
- 3–3
- 4–6
- 7–20

In 2017, the Global Burden of Disease Study estimated there were 1.1 million new cases and a total of 19.8 million cases globally.[15] Schizophrenia affects around 0.3–0.7% of people at some point in their life.[10] It occurs 1.4 times more frequently in males than females and typically appears earlier in men[56]—the peak ages of onset are 25 years for males and 27 years for females.[188] Onset in childhood is much rarer,[189] as is onset in middle or old age.[190]

Despite the prior belief that schizophrenia occurs at similar rates worldwide, its frequency varies across the world,[6][191] within countries,[192] and at the local and neighborhood level.[193] This variation has been estimated to be fivefold.[5] It causes approximately one percent of worldwide disability adjusted life years[56] and resulted in 20,000 deaths in 2010.[194] The rate of schizophrenia varies up to threefold depending on how it is defined.[10]

In 2000, the World Health Organization found the percentage of people affected and the number of new cases that develop each year is roughly similar around the world, with age-standardized prevalence per 100,000 ranging from 343 in Africa to 544 in Japan and Oceania for men, and from 378 in Africa to 527 in Southeastern Europe for women.[195] About 1.1% of adults have schizophrenia in the United States.[196]

History

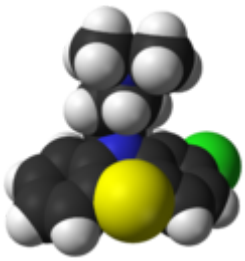
The history of schizophrenia is complex and does not lend itself easily to a linear narrative.[197] Accounts of a schizophrenia-like syndrome are rare in records before the 19th century. The earliest cases detailed were reported in 1797, and 1809.[198] *Dementia praecox*, meaning premature dementia was used by German psychiatrist Heinrich Schüle in 1886, and then in 1891 by Arnold Pick in a case report of hebephrenia. In 1893 Emil Kraepelin used the term in making a distinction, known as the Kraepelinian dichotomy, between the two psychoses – praecox dementia, and manic depression.[199] Kraepelin believed that *dementia praecox* was probably caused by a long-term, smouldering systemic or "whole body" disease process that affected many organs and peripheral nerves in the body but which affected the brain after puberty in a final decisive cascade.[200] It was thought to be an early form of dementia, a degenerative disease.[201] When it became evident that the disorder was not degenerative it was renamed schizophrenia by Eugen Bleuler in 1908.

The term "schizophrenia" was coined by Eugen Bleuler.

The word *schizophrenia*—which translates roughly as "splitting of the mind" and comes from the Greek roots *schizein* (σχίζειν, "to split") and *phrēn, phren-* (φρήν, φρεν-, "mind")[202] and was intended to describe the separation of function between personality, thinking, memory, and perception. American and British interpretations of Bleuler led to the claim that he described its main symptoms as four *A*'s: flattened *affect*, *autism*, impaired *association* of ideas, and *ambivalence*.[203][204]

The term schizophrenia used to be associated with *split personality* by the general population but that usage went into decline when it became known as a separate disorder, first as *multiple identity disorder* , and later as dissociative identity disorder.[205]

In the early 20th century, the psychiatrist Kurt Schneider listed the forms of psychotic symptoms that he thought distinguished schizophrenia from other psychotic disorders. [206] These are called first-rank symptoms that are mostly covered in the updated use of positive symptoms. They are no longer included in DSM-5. First-rank symptoms are seen to be of limited use in detecting schizophrenia but may be of help in differential diagnosis.[207]



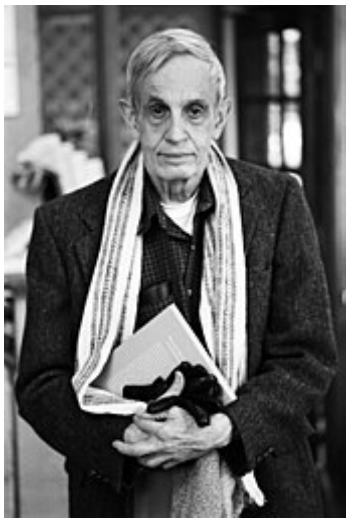
A molecule of chlorpromazine, the first antipsychotic developed in the 1950s.

Treatment was revolutionized in the mid-1950s with the development and introduction of the first typical antipsychotic, chlorpromazine.[208]

In the early 1970's in the US, the diagnostic model used for schizophrenia was broad and clinically-based using DSM II. It had been noted that schizophrenia was diagnosed far more in the US than in Europe which had been using the ICD-9 criteria. The US model was criticised for failing to demarcate clearly those people with a mental illness, and those without. In 1980 DSM III was published and showed a shift in focus from the clinically-based biopsychosocial model to a reason-based medical model.[209] DSM IV showed an increased focus to an evidence-based medical model.[210] DSM-5 was published in 2013 and introduced changes to DSM IV.

Society and culture

In 2002, the term for schizophrenia in Japan was changed from *seishin-bunretsu-byō* (精神分裂病, lit. "mind-split disease") to *tōgō-shitchō-shō* (統合失調症, lit. "integration-dysregulation syndrome") to reduce stigma.[10] The new name also interpreted as "integration disorder" was inspired by the biopsychosocial model; it increased the percentage of people who were informed of the diagnosis from 37 to 70% over three years.[211] A similar change was made in South Korea in 2012.[212] A professor of psychiatry, Jim van Os, has proposed changing the English term to "psychosis spectrum syndrome".[213]



John Nash, an American mathematician and joint recipient of the 1994 Nobel Prize for Economics, who had schizophrenia. His life was the subject of the 1998 book, A Beautiful Mind by Sylvia Nasar.

In the United States, the cost of schizophrenia—including direct costs (outpatient, inpatient, drugs, and long-term care) and non-health care costs (law enforcement, reduced workplace productivity, and unemployment)—was estimated to be $62.7 billion in 2002.[214]

The book *A Beautiful Mind* chronicled the life of John Forbes Nash who had been diagnosed with schizophrenia but who went on to win the Nobel Prize for Economics. This was later made into the film with the same name.

In 1964 a lengthy case study of three males diagnosed with paranoid schizophrenia who each had the delusional belief that they were Jesus Christ was published as a book. This has the title of *The Three Christs of Ypsilanti*, and has later (2017) been made into a film called *Three Christs*.

Violence

People with severe mental illness, including schizophrenia, are at a significantly greater risk of being victims of both violent and non-violent crime.[215] Schizophrenia has been associated with a higher rate of violent acts, but most appear to be related to associated substance abuse.[216] Rates of homicide linked to psychosis are similar to those linked to substance misuse, and parallel the overall rate in a region.[217] What role schizophrenia has on violence independent of drug misuse is controversial, but certain aspects of individual histories or mental states may be factors.[218] About 11% of people in prison for homicide have schizophrenia and 21% have mood disorders.[219] Another study found about 8-10% of people with schizophrenia had committed a violent act in the past year compared to 2% of the general population.[219]

Media coverage relating to violent acts by people with schizophrenia reinforces public perception of an association between schizophrenia and violence.[216] In a large, representative sample from a 1999 study, 12.8% of Americans believed that those with schizophrenia were "very likely" to do something violent against others, and 48.1% said that they were "somewhat likely" to. Over 74% said that people with schizophrenia were either "not very able" or "not able at all" to make decisions concerning their treatment, and 70.2% said the same of money-management decisions.[220] The perception of people with psychosis as violent has more than doubled in prevalence since the 1950s, according to one meta-analysis.[221]

Research directions

Schizophrenia is not believed to occur in non-human species[222] but it may be possible to develop a pharmacologically induced nonhuman primate model of schizophrenia.[223]

Research has found a tentative benefit in using minocycline, a broad-spectrum antibiotic, to treat schizophrenia.[224] Nidotherapy or efforts to change the environment of people with schizophrenia to improve their ability to function, is also being studied but there is not enough evidence yet to make conclusions about its effectiveness.[225] Negative symptoms have proven a challenge to treat, as they are generally not improved by medication. Various agents have been explored for possible benefits in this area.[226] There have been trials on medications with anti-inflammatory activity, based on the premise that inflammation might play a role in the pathology of schizophrenia.[227]

Brain stimulation

Various brain stimulation techniques are being studied to treat the positive symptoms of schizophrenia, in particular auditory verbal hallucinations (AVHs).[228][229] A 2015 Cochrane review found unclear evidence of benefit.[230] Most studies focus on transcranial direct-current stimulation (tDCM), and repetitive transcranial magnetic stimulation (rTMS).[229] Techniques based on focused ultrasound for deep brain stimulation could provide insight for the treatment of AVHs.[229]

References

1. Jones D (2003) [1917]. Roach P, Hartmann J, Setter J (eds.). *English Pronouncing Dictionary*. Cambridge: Cambridge University Press. ISBN 978-3-12-539683-8.
2. "Schizophrenia Fact sheet N°397" (http://www.who.int/mediacentre/factsheets/fs397/en/). *WHO*. September 2015. Archived (https://web.archive.org/web/20161018051507/http://www.who.int/mediacentre/factsheets/fs397/en/) from the original on 18 October 2016. Retrieved 3 February 2016.
3. "Schizophrenia" (http://www.nimh.nih.gov/health/topics/schizophrenia/index.shtml). *National Institute of Mental Health*. January 2016. Archived (https://web.archive.org/web/20161125145206/https://www.nimh.nih.gov/health/topics/schizophrenia/index.shtml) from the original on 25 November 2016. Retrieved 3 February 2016.
4. "Medicinal treatment of psychosis/schizophrenia" (http://www.sbu.se/en/publications/sbu-assesses/schizophrenia--pharmacological-treatments-patient-involvement-and-organization-of-care/). *www.sbu.se*. Swedish Agency for Health Technology Assessment and Assessment of Social Services (SBU). 21 November 2012. Archived (https://web.archive.org/web/20170629092226/http://www.sbu.se/en/publications/sbu-assesses/schizophrenia--pharmacological-treatments-patient-involvement-and-organization-of-care/) from the original on 29 June 2017. Retrieved 26 June 2017.
5. Owen MJ, Sawa A, Mortensen PB (July 2016). "Schizophrenia" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4940219). *Lancet*. **388** (10039): 86–97. doi:10.1016/S0140-6736(15)01121-6 (https://doi.org/10.1016%2FS0140-6736%2815%2901121-6). PMC 4940219 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4940219). PMID 26777917 (https://pubmed.ncbi.nlm.nih.gov/26777917).
6. American Psychiatric Association (2013). *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.). Arlington: American Psychiatric Publishing. pp. 101–05. ISBN 978-0-89042-555-8.

7. Ferri FF (2010). *Ferri's differential diagnosis : a practical guide to the differential diagnosis of symptoms, signs, and clinical disorders* (2nd ed.). Philadelphia, PA: Elsevier/Mosby. p. Chapter S. ISBN 978-0-323-07699-9.
8. Paris J (December 2018). "Differential Diagnosis of Borderline Personality Disorder". *The Psychiatric clinics of North America*. **41** (4): 575–582. doi:10.1016/j.psc.2018.07.001 (https://doi.org/10.1016%2Fj.psc.2018.07.001). PMID 30447725 (https://pubmed.ncbi.nlm.nih.gov/30447725).
9. Laursen TM, Nordentoft M, Mortensen PB (2014). "Excess early mortality in schizophrenia". *Annual Review of Clinical Psychology*. **10**: 425–48. doi:10.1146/annurev-clinpsy-032813-153657 (https://doi.org/10.1146%2Fannurev-clinpsy-032813-153657). PMID 24313570 (https://pubmed.ncbi.nlm.nih.gov/24313570).
10. van Os J, Kapur S (August 2009). "Schizophrenia" (https://web.archive.org/web/20130623065810/http://xa.yimg.com/kq/groups/19525360/611943554/name/Schizophrenia+-+The+Lancet.pdf) (PDF). *Lancet*. **374** (9690): 635–45. doi:10.1016/S0140-6736(09)60995-8 (https://doi.org/10.1016%2FS0140-6736%2809%2960995-8). PMID 19700006 (https://pubmed.ncbi.nlm.nih.gov/19700006). Archived from the original (http://xa.yimg.com/kq/groups/19525360/611943554/name/Schizophrenia+-+The+Lancet.pdf) (PDF) on 23 June 2013. Retrieved 23 December 2011.
11. GBD 2015 Mortality and Causes of Death Collaborators (October 2016). "Global, regional, and national life expectancy, all-cause mortality, and cause-specific mortality for 249 causes of death, 1980-2015: a systematic analysis for the Global Burden of Disease Study 2015" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5388903). *Lancet*. **388** (10053): 1459–1544. doi:10.1016/s0140-6736(16)31012-1 (https://doi.org/10.1016%2Fs0140-6736%2816%2931012-1). PMC 5388903 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5388903). PMID 27733281 (https://pubmed.ncbi.nlm.nih.gov/27733281).
12. "NIMH » RAISE Questions and Answers" (https://www.nimh.nih.gov/health/topics/schizophrenia/raise/raise-questions-and-answers.shtml#4). *www.nimh.nih.gov*. Retrieved 29 December 2019.
13. de Filippis R, Carbone EA, Gaetano R, et al. (2019). "Machine learning techniques in a structural and functional MRI diagnostic approach in schizophrenia: a systematic review" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6590624). *Neuropsychiatr Dis Treat*. **15**: 1605–1627. doi:10.2147/NDT.S202418 (https://doi.org/10.2147%2FNDT.S202418). PMC 6590624 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6590624). PMID 31354276 (https://pubmed.ncbi.nlm.nih.gov/31354276).
14. Buckley PF, Miller BJ, Lehrer DS, Castle DJ (March 2009). "Psychiatric comorbidities and schizophrenia" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2659306). *Schizophrenia Bulletin*. **35** (2): 383–402. doi:10.1093/schbul/sbn135 (https://doi.org/10.1093%2Fschbul%2Fsbn135). PMC 2659306 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2659306). PMID 19011234 (https://pubmed.ncbi.nlm.nih.gov/19011234).
15. James SL, Abate D (November 2018). "Global, regional, and national incidence, prevalence, and years lived with disability for 354 diseases and injuries for 195 countries and territories, 1990–2017: a systematic analysis for the Global Burden of Disease Study 2017" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6227754). *The Lancet*. **392** (10159): 1789–1858. doi:10.1016/S0140-6736(18)32279-7 (https://doi.org/10.1016%2FS0140-6736%2818%2932279-7). PMC 6227754 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6227754). PMID 30496104 (https://pubmed.ncbi.nlm.nih.gov/30496104).
16. Parakh P, Basu D (August 2013). "Cannabis and psychosis: have we found the missing links?". *Asian Journal of Psychiatry* (Review). **6** (4): 281–7. doi:10.1016/j.ajp.2013.03.012 (https://doi.org/10.1016%2Fj.ajp.2013.03.012). PMID 23810133 (https://pubmed.ncbi.nlm.nih.gov/23810133). "Cannabis acts as a component cause of psychosis, that is, it increases the risk of psychosis in people with certain genetic or environmental vulnerabilities, though by itself, it is neither a sufficient nor a necessary cause of psychosis."

17. van de Leemput J, Hess JL, Glatt SJ, Tsuang MT (2016). "Genetics of Schizophrenia: Historical Insights and Prevailing Evidence". *Advances in Genetics*. **96**: 99–141. doi:10.1016/bs.adgen.2016.08.001 (https://doi.org/10.1016%2Fbs.adgen.2016.08.001). PMID 27968732 (https://pubmed.ncbi.nlm.nih.gov/27968732).
18. Lawrence RE, First MB, Lieberman JA (2015). "Chapter 48: Schizophrenia and Other Psychoses". In Tasman A, Kay J, Lieberman JA, First MB, Riba MB (eds.). *Psychiatry* (fourth ed.). John Wiley & Sons, Ltd. pp. 798, 816, 819. doi:10.1002/9781118753378.ch48 (https://doi.org/10.1002%2F9781118753378.ch48). ISBN 978-1-118-84547-9.
19. Foster A, Gable J, Buckley J (September 2012). "Homelessness in schizophrenia". *The Psychiatric Clinics of North America*. **35** (3): 717–34. doi:10.1016/j.psc.2012.06.010 (https://doi.org/10.1016%2Fj.psc.2012.06.010). PMID 22929875 (https://pubmed.ncbi.nlm.nih.gov/22929875).
20. Hor K, Taylor M (November 2010). "Suicide and schizophrenia: a systematic review of rates and risk factors" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2951591). *Journal of Psychopharmacology*. **24** (4 Suppl): 81–90. doi:10.1177/1359786810385490 (https://doi.org/10.1177%2F1359786810385490). PMC 2951591 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2951591). PMID 20923923 (https://pubmed.ncbi.nlm.nih.gov/20923923).
21. Becker T, Kilian R (2006). "Psychiatric services for people with severe mental illness across western Europe: what can be generalized from current knowledge about differences in provision, costs and outcomes of mental health care?". *Acta Psychiatrica Scandinavica. Supplementum*. **113** (429): 9–16. doi:10.1111/j.1600-0447.2005.00711.x (https://doi.org/10.1111%2Fj.1600-0447.2005.00711.x). PMID 16445476 (https://pubmed.ncbi.nlm.nih.gov/16445476).
22. Marshall M (September 2005). "Association between duration of untreated psychosis and outcome in cohorts of first-episode patients: a systematic review". *Archives of general psychiatry*. **62** (9): 975–83. doi:10.1001/archpsyc.62.9.975 (https://doi.org/10.1001%2Farchpsyc.62.9.975). PMID 16143729 (https://pubmed.ncbi.nlm.nih.gov/16143729).
23. "National Institute of Mental Health" (http://www.nimh.nih.gov/health/publications/schizophrenia/complete-index.shtml). Archived (https://web.archive.org/web/20110928024705/http://www.nimh.nih.gov/health/publications/schizophrenia/complete-index.shtml) from the original on 28 September 2011. Retrieved 2 October 2011.
24. Carson VB (2000). *Mental Health Nursing: The Nurse-patient Journey* (https://books.google.com/books?id=QM5rAAAAMAAJ). W.B. Saunders. p. 638. ISBN 978-0-7216-8053-8. Archived (https://web.archive.org/web/20160513011255/https://books.google.com/books?id=QM5rAAAAMAAJ) from the original on 13 May 2016.
25. Heinz A, Voss M, Lawrie SM, et al. (September 2016). "Shall we really say goodbye to first rank symptoms?". *European Psychiatry*. **37**: 8–13. doi:10.1016/j.eurpsy.2016.04.010 (https://doi.org/10.1016%2Fj.eurpsy.2016.04.010). PMID 27429167 (https://pubmed.ncbi.nlm.nih.gov/27429167).
26. Hirsch SR, Weinberger DR (2003). *Schizophrenia* (https://books.google.com/books?id=x3fmsV55rigC&pg=PA21). Wiley-Blackwell. p. 21. ISBN 978-0-632-06388-8. Archived (https://web.archive.org/web/20150320131542/http://books.google.com/books?id=x3fmsV55rigC&pg=PA21) from the original on 20 March 2015.
27. Le Gall, E; Iakimova, G (December 2018). "[Social cognition in schizophrenia and autism spectrum disorder: Points of convergence and functional differences]". *L'Encephale*. **44** (6): 523–537. doi:10.1016/j.encep.2018.03.004 (https://doi.org/10.1016%2Fj.encep.2018.03.004). PMID 30122298 (https://pubmed.ncbi.nlm.nih.gov/30122298).
28. Brunet-Gouet E, Decety J (December 2006). "Social brain dysfunctions in schizophrenia: a review of neuroimaging studies". *Psychiatry Research*. **148** (2–3): 75–92. doi:10.1016/j.pscychresns.2006.05.001 (https://doi.org/10.1016%2Fj.pscychresns.2006.05.001). PMID 17088049 (https://pubmed.ncbi.nlm.nih.gov/17088049).

29. Hirsch SR, Weinberger DR (2003). *Schizophrenia* (https://books.google.com/books?id=x3fmsV55rigC&pg=PA21). Wiley-Blackwell. p. 481. ISBN 978-0-632-06388-8.
30. Kohler CG, Walker JB, Martin EA, Healey KM, Moberg PJ (September 2010). "Facial emotion perception in schizophrenia: a meta-analytic review" (http://schizophreniabulletin.oxfordjournals.org/content/36/5/1009.full). *Schizophrenia Bulletin*. **36** (5): 1009–19. doi:10.1093/schbul/sbn192 (https://doi.org/10.1093%2Fschbul%2Fsbn192). PMC 2930336 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2930336). PMID 19329561 (https://pubmed.ncbi.nlm.nih.gov/19329561). Archived (https://web.archive.org/web/20150725211920/http://schizophreniabulletin.oxfordjournals.org/content/36/5/1009.full) from the original on 25 July 2015.
31. Sims A (2002). *Symptoms in the mind: an introduction to descriptive psychopathology*. Philadelphia: W. B. Saunders. ISBN 978-0-7020-2627-0.
32. Lindenmayer JP (1 December 2017). "Are Shorter Versions of the Positive and Negative Syndrome Scale (PANSS) Doable? A Critical Review" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5788254). *Innovations in Clinical Neuroscience*. **14** (11–12): 73–76. PMC 5788254 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5788254). PMID 29410940 (https://pubmed.ncbi.nlm.nih.gov/29410940).
33. Kneisl C, Trigoboff E (2009). *Contemporary Psychiatric-Mental Health Nursing* (2nd ed.). London: Pearson Prentice Ltd. p. 371.
34. American Psychiatric Association. Task Force on DSM-IV. (2000). *Diagnostic and statistical manual of mental disorders: DSM-IV-TR*. American Psychiatric Pub. p. 299. ISBN 978-0-89042-025-6.
35. Velligan DI, Alphs LD (1 March 2008). "Negative Symptoms in Schizophrenia: The Importance of Identification and Treatment" (http://www.psychiatrictimes.com/schizophrenia/article/10168/1147581). *Psychiatric Times*. **25** (3). Archived (https://web.archive.org/web/20091006015627/http://www.psychiatrictimes.com/schizophrenia/article/10168/1147581) from the original on 6 October 2009.
36. Smith T, Weston C, Lieberman J (August 2010). "Schizophrenia (maintenance treatment)". *American Family Physician*. **82** (4): 338–9. PMID 20704164 (https://pubmed.ncbi.nlm.nih.gov/20704164).
37. Sekar A, et al. (11 February 2016). "Schizophrenia risk from complex variation of complement component 4" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4752392). *Nature*. **530** (7589): 177–83. Bibcode:2016Natur.530..177. (https://ui.adsabs.harvard.edu/abs/2016Natur.530..177.). doi:10.1038/nature16549 (https://doi.org/10.1038%2Fnature16549). PMC 4752392 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4752392). PMID 26814963 (https://pubmed.ncbi.nlm.nih.gov/26814963).
38. Dauvermann MR, Whalley HC, Schmidt A, et al. (1 January 2014). "Computational neuropsychiatry - schizophrenia as a cognitive brain network disorder" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3971172). *Frontiers in Psychiatry*. **5**: 30. doi:10.3389/fpsyt.2014.00030 (https://doi.org/10.3389%2Ffpsyt.2014.00030). PMC 3971172 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3971172). PMID 24723894 (https://pubmed.ncbi.nlm.nih.gov/24723894).
39. Bozikas VP, Andreou C (February 2011). "Longitudinal studies of cognition in first episode psychosis: a systematic review of the literature". *The Australian and New Zealand Journal of Psychiatry*. **45** (2): 93–108. doi:10.3109/00048674.2010.541418 (https://doi.org/10.3109%2F00048674.2010.541418). PMID 21320033 (https://pubmed.ncbi.nlm.nih.gov/21320033).
40. Kurtz MM, Moberg PJ, Gur RC, Gur RE (December 2001). "Approaches to cognitive remediation of neuropsychological deficits in schizophrenia: a review and meta-analysis". *Neuropsychology Review*. **11** (4): 197–210. doi:10.1023/A:1012953108158 (https://doi.org/10.1023%2FA%3A1012953108158). PMID 11883669 (https://pubmed.ncbi.nlm.nih.gov/11883669).

41. Tan BL (August 2009). "Profile of cognitive problems in schizophrenia and implications for vocational functioning". *Australian Occupational Therapy Journal*. **56** (4): 220–8. doi:10.1111/j.1440-1630.2008.00759.x (https://doi.org/10.1111%2Fj.1440-1630.2008.00759.x). PMID 20854522 (https://pubmed.ncbi.nlm.nih.gov/20854522).
42. Cirillo MA, Seidman LJ (June 2003). "Verbal declarative memory dysfunction in schizophrenia: from clinical assessment to genetics and brain mechanisms". *Neuropsychology Review*. **13** (2): 43–77. doi:10.1023/A:1023870821631 (https://doi.org/10.1023%2FA%3A1023870821631). PMID 12887039 (https://pubmed.ncbi.nlm.nih.gov/12887039).
43. Pomarol-Clotet E, Oh TM, Laws KR, McKenna PJ (February 2008). "Semantic priming in schizophrenia: systematic review and meta-analysis". *The British Journal of Psychiatry*. **192** (2): 92–7. doi:10.1192/bjp.bp.106.032102 (https://doi.org/10.1192%2Fbjp.bp.106.032102). PMID 18245021 (https://pubmed.ncbi.nlm.nih.gov/18245021).
44. Goldberg TE, Keefe RS, Goldman RS, Robinson DG, Harvey PD (April 2010). "Circumstances under which practice does not make perfect: a review of the practice effect literature in schizophrenia and its relevance to clinical treatment studies" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3055399). *Neuropsychopharmacology*. **35** (5): 1053–62. doi:10.1038/npp.2009.211 (https://doi.org/10.1038%2Fnpp.2009.211). PMC 3055399 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3055399). PMID 20090669 (https://pubmed.ncbi.nlm.nih.gov/20090669).
45. Shah JN, Qureshi SU, Jawaid A, Schulz PE (June 2012). "Is there evidence for late cognitive decline in chronic schizophrenia?". *The Psychiatric Quarterly*. **83** (2): 127–44. doi:10.1007/s11126-011-9189-8 (https://doi.org/10.1007%2Fs11126-011-9189-8). PMID 21863346 (https://pubmed.ncbi.nlm.nih.gov/21863346).
46. Barch DM (1 August 2003). "Cognition in Schizophrenia Does Working Memory Work?". *Current Directions in Psychological Science*. **12** (4): 146–150. doi:10.1111/1467-8721.01251 (https://doi.org/10.1111%2F1467-8721.01251). ISSN 0963-7214 (https://www.worldcat.org/issn/0963-7214).
47. Addington J, Cadenhead KS, Cannon TD, et al. (May 2007). "North American Prodrome Longitudinal Study: a collaborative multisite approach to prodromal schizophrenia research" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2526151). *Schizophrenia Bulletin*. **33** (3): 665–72. doi:10.1093/schbul/sbl075 (https://doi.org/10.1093%2Fschbul%2Fsbl075). PMC 2526151 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2526151). PMID 17255119 (https://pubmed.ncbi.nlm.nih.gov/17255119).
48. Cullen KR, Kumra S, Regan J, et al. (2008). "Atypical Antipsychotics for Treatment of Schizophrenia Spectrum Disorders" (http://www.psychiatrictimes.com/schizophrenia/article/10168/1147536). *Psychiatric Times*. **25** (3). Archived (https://web.archive.org/web/20081228153056/http://www.psychiatrictimes.com/schizophrenia/article/10168/1147536) from the original on 28 December 2008.
49. Ochoa S, Usall J, Cobo J, Labad X, Kulkarni J (2012). "Gender differences in schizophrenia and first-episode psychosis: a comprehensive literature review" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3420456). *Schizophrenia Research and Treatment* (Review). **2012**: 1–9. doi:10.1155/2012/916198 (https://doi.org/10.1155%2F2012%2F916198). PMC 3420456 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3420456). PMID 22966451 (https://pubmed.ncbi.nlm.nih.gov/22966451).
50. Amminger GP, Leicester S, Yung AR, et al. (May 2006). "Early-onset of symptoms predicts conversion to non-affective psychosis in ultra-high risk individuals". *Schizophrenia Research*. **84** (1): 67–76. doi:10.1016/j.schres.2006.02.018 (https://doi.org/10.1016%2Fj.schres.2006.02.018). PMID 16677803 (https://pubmed.ncbi.nlm.nih.gov/16677803).
51. Parnas J, Jorgensen A (November 1989). "Pre-morbid psychopathology in schizophrenia spectrum". *The British Journal of Psychiatry*. **155** (5): 623–7. doi:10.1192/s0007125000018109 (https://doi.org/10.1192%2Fs0007125000018109). PMID 2611591 (https://pubmed.ncbi.nlm.nih.gov/2611591).

52. Coyle J (2006). "Chapter 54: The Neurochemistry of Schizophrenia". In Siegal GJ, et al. (eds.). *Basic Neurochemistry: Molecular, Cellular and Medical Aspects* (7th ed.). Burlington, MA: Elsevier Academic Press. pp. 876–78. ISBN 978-0-12-088397-4.
53. Khandaker GM, Barnett JH, White IR, Jones PB (November 2011). "A quantitative meta-analysis of population-based studies of premorbid intelligence and schizophrenia" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3485562). *Schizophrenia Research*. **132** (2–3): 220–7. doi:10.1016/j.schres.2011.06.017 (https://doi.org/10.1016%2Fj.schres.2011.06.017). PMC 3485562 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3485562). PMID 21764562 (https://pubmed.ncbi.nlm.nih.gov/21764562).
54. Welham J, Isohanni M, Jones P, McGrath J (May 2009). "The antecedents of schizophrenia: a review of birth cohort studies" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2669575). *Schizophrenia Bulletin*. **35** (3): 603–23. doi:10.1093/schbul/sbn084 (https://doi.org/10.1093%2Fschbul%2Fsbn084). PMC 2669575 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2669575). PMID 18658128 (https://pubmed.ncbi.nlm.nih.gov/18658128).
55. Dickson H, Laurens KR, Cullen AE, Hodgins S (April 2012). "Meta-analyses of cognitive and motor function in youth aged 16 years and younger who subsequently develop schizophrenia" (https://www.researchgate.net/publication/51623569). *Psychological Medicine*. **42** (4): 743–55. doi:10.1017/s0033291711001693 (https://doi.org/10.1017%2Fs0033291711001693). PMID 21896236 (https://pubmed.ncbi.nlm.nih.gov/21896236). Archived (https://web.archive.org/web/20170202022622/https://www.researchgate.net/publication/51623569_Meta-analyses_of_cognitive_and_motor_function_in_youth_aged_16_years_and_younger_who_subsequently_develop_schizophrenia) from the original on 2 February 2017.
56. Picchioni MM, Murray RM (July 2007). "Schizophrenia" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1914490). *BMJ*. **335** (7610): 91–5. doi:10.1136/bmj.39227.616447.BE (https://doi.org/10.1136%2Fbmj.39227.616447.BE). PMC 1914490 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1914490). PMID 17626963 (https://pubmed.ncbi.nlm.nih.gov/17626963).
57. Drake RJ, Lewis SW (March 2005). "Early detection of schizophrenia". *Current Opinion in Psychiatry*. **18** (2): 147–50. doi:10.1097/00001504-200503000-00007 (https://doi.org/10.1097%2F00001504-200503000-00007). PMID 16639167 (https://pubmed.ncbi.nlm.nih.gov/16639167).
58. Combs DR, Mueser KT, Gutierrez MM (2011). "Chapter 8: Schizophrenia: Etiological considerations" (https://books.google.com/books?id=iJtzm1KfU5oC&pg=PT282). In Hersen M, Beidel DC (eds.). *Adult psychopathology and diagnosis* (6th ed.). John Wiley & Sons. ISBN 978-1-118-13884-7.
59. van de Leemput J, Hess JL, Glatt SJ, Tsuang MT (2016). "Genetics of Schizophrenia: Historical Insights and Prevailing Evidence". *Advances in Genetics*. **96**: 99–141. doi:10.1016/bs.adgen.2016.08.001 (https://doi.org/10.1016%2Fbs.adgen.2016.08.001). PMID 27968732 (https://pubmed.ncbi.nlm.nih.gov/27968732).
60. O'Donovan MC, Williams NM, Owen MJ (October 2003). "Recent advances in the genetics of schizophrenia". *Human Molecular Genetics*. 12 Spec No 2: R125–33. doi:10.1093/hmg/ddg302 (https://doi.org/10.1093%2Fhmg%2Fddg302). PMID 12952866 (https://pubmed.ncbi.nlm.nih.gov/12952866).
61. Torrey EF, Yolken RH (August 2019). "Schizophrenia as a pseudogenetic disease: A call for more gene-environmental studies". *Psychiatry Research*. **278**: 146–150. doi:10.1016/j.psychres.2019.06.006 (https://doi.org/10.1016%2Fj.psychres.2019.06.006). PMID 31200193 (https://pubmed.ncbi.nlm.nih.gov/31200193).
62. Farrell MS, Werge T, Sklar P, et al. (May 2015). "Evaluating historical candidate genes for schizophrenia" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4414705). *Molecular Psychiatry*. **20** (5): 555–62. doi:10.1038/mp.2015.16 (https://doi.org/10.1038%2Fmp.2015.16). PMC 4414705 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4414705). PMID 25754081 (https://pubmed.ncbi.nlm.nih.gov/25754081).

63. Schulz, S. Charles; Green, Michael Foster; Nelson, Katharine J. (2016). *Schizophrenia and Psychotic Spectrum Disorders* (https://books.google.com/books?id=7ukmDAAAQBAJ). Oxford University Press. pp. 124–5. ISBN 9780199378067.
64. Schork AJ, Wang Y, Thompson WK, Dale AM, Andreassen OA (February 2016). "New statistical approaches exploit the polygenic architecture of schizophrenia--implications for the underlying neurobiology" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5380793). *Current Opinion in Neurobiology*. **36**: 89–98. doi:10.1016/j.conb.2015.10.008 (https://doi.org/10.1016%2Fj.conb.2015.10.008). PMC 5380793 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5380793). PMID 26555806 (https://pubmed.ncbi.nlm.nih.gov/26555806).
65. Kendler KS (March 2016). "The Schizophrenia Polygenic Risk Score: To What Does It Predispose in Adolescence?". *JAMA Psychiatry*. **73** (3): 193–4. doi:10.1001/jamapsychiatry.2015.2964 (https://doi.org/10.1001%2Fjamapsychiatry.2015.2964). PMID 26817666 (https://pubmed.ncbi.nlm.nih.gov/26817666).
66. Lowther C, Costain G, Baribeau DA, Bassett AS (September 2017). "Genomic Disorders in Psychiatry-What Does the Clinician Need to Know?". *Current Psychiatry Reports*. **19** (11): 82. doi:10.1007/s11920-017-0831-5 (https://doi.org/10.1007%2Fs11920-017-0831-5). PMID 28929285 (https://pubmed.ncbi.nlm.nih.gov/28929285).
67. Bundy H, Stahl D, MacCabe JH (February 2011). "A systematic review and meta-analysis of the fertility of patients with schizophrenia and their unaffected relatives: Fertility in schizophrenia". *Acta Psychiatrica Scandinavica*. **123** (2): 98–106. doi:10.1111/j.1600-0447.2010.01623.x (https://doi.org/10.1111%2Fj.1600-0447.2010.01623.x). PMID 20958271 (https://pubmed.ncbi.nlm.nih.gov/20958271).
68. van Dongen J, Boomsma DI (March 2013). "The evolutionary paradox and the missing heritability of schizophrenia". *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics*. **162** (2): 122–136. doi:10.1002/ajmg.b.32135 (https://doi.org/10.1002%2Fajmg.b.32135). PMID 23355297 (https://pubmed.ncbi.nlm.nih.gov/23355297).
69. Owen MJ, Sawa A, Mortensen PB (2 July 2016). "Schizophrenia" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4940219). *Lancet*. **388** (10039): 86–97. doi:10.1016/S0140-6736(15)01121-6 (https://doi.org/10.1016%2FS0140-6736%2815%2901121-6). PMC 4940219 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4940219). PMID 26777917 (https://pubmed.ncbi.nlm.nih.gov/26777917).
70. Negrón-Oyarzo I, Lara-Vásquez A, Palacios-García I, Fuentealba P, Aboitiz F (March 2016). "Schizophrenia and reelin: a model based on prenatal stress to study epigenetics, brain development and behavior" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4787713). *Biological Research*. **49**: 16. doi:10.1186/s40659-016-0076-5 (https://doi.org/10.1186%2Fs40659-016-0076-5). PMC 4787713 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4787713). PMID 26968981 (https://pubmed.ncbi.nlm.nih.gov/26968981).
71. Brown AS (January 2011). "The environment and susceptibility to schizophrenia" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3521525). *Progress in Neurobiology*. **93** (1): 23–58. doi:10.1016/j.pneurobio.2010.09.003 (https://doi.org/10.1016%2Fj.pneurobio.2010.09.003). PMC 3521525 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3521525). PMID 20955757 (https://pubmed.ncbi.nlm.nih.gov/20955757).
72. Le Charpentier, Y.; Hoang, C.; Mokni, M.; Finet, J. F.; Biaggi, A.; Saguin, M.; Plantier, F. (1992). "Histopathology and ultrastructure of opportunistic infections of the digestive tract in acquired immunodeficiency syndrome". *Archives d'Anatomie et de Cytologie Pathologiques*. **40** (2–3): 138–49. PMID 1280409 (https://pubmed.ncbi.nlm.nih.gov/1280409).
73. Dvir Y, Denietolis B, Frazier JA (October 2013). "Childhood trauma and psychosis". *Child and Adolescent Psychiatric Clinics of North America*. **22** (4): 629–41. doi:10.1016/j.chc.2013.04.006 (https://doi.org/10.1016%2Fj.chc.2013.04.006). PMID 24012077 (https://pubmed.ncbi.nlm.nih.gov/24012077).

74. Misiak B, Krefft M, Bielawski T, Moustafa AA, Sąsiadek MM, Frydecka D (April 2017). "Toward a unified theory of childhood trauma and psychosis: A comprehensive review of epidemiological, clinical, neuropsychological and biological findings". *Neuroscience and Biobehavioral Reviews*. **75**: 393–406. doi:10.1016/j.neubiorev.2017.02.015 (https://doi.org/10.1016%2Fj.neubiorev.2017.02.015). PMID 28216171 (https://pubmed.ncbi.nlm.nih.gov/28216171).
75. van Os J (April 2004). "Does the urban environment cause psychosis?". *The British Journal of Psychiatry*. **184** (4): 287–8. doi:10.1192/bjp.184.4.287 (https://doi.org/10.1192%2Fbjp.184.4.287). PMID 15056569 (https://pubmed.ncbi.nlm.nih.gov/15056569).
76. Selten JP, Cantor-Graae E, Kahn RS (March 2007). "Migration and schizophrenia". *Current Opinion in Psychiatry*. **20** (2): 111–5. doi:10.1097/YCO.0b013e328017f68e (https://doi.org/10.1097%2FYCO.0b013e328017f68e). PMID 17278906 (https://pubmed.ncbi.nlm.nih.gov/17278906).
77. Nemani K, Hosseini Ghomi R, McCormick B, Fan X (January 2015). "Schizophrenia and the gut-brain axis". *Progress in Neuro-Psychopharmacology & Biological Psychiatry*. **56**: 155–60. doi:10.1016/j.pnpbp.2014.08.018 (https://doi.org/10.1016%2Fj.pnpbp.2014.08.018). PMID 25240858 (https://pubmed.ncbi.nlm.nih.gov/25240858).
78. Lachance LR, McKenzie K (February 2014). "Biomarkers of gluten sensitivity in patients with non-affective psychosis: a meta-analysis". *Schizophrenia Research* (Review). **152** (2–3): 521–7. doi:10.1016/j.schres.2013.12.001 (https://doi.org/10.1016%2Fj.schres.2013.12.001). PMID 24368154 (https://pubmed.ncbi.nlm.nih.gov/24368154).
79. Gregg L, Barrowclough C, Haddock G (May 2007). "Reasons for increased substance use in psychosis". *Clinical Psychology Review*. **27** (4): 494–510. doi:10.1016/j.cpr.2006.09.004 (https://doi.org/10.1016%2Fj.cpr.2006.09.004). PMID 17240501 (https://pubmed.ncbi.nlm.nih.gov/17240501).
80. Sagud M, Mihaljević-Peles A, Mück-Seler D, et al. (September 2009). "Smoking and schizophrenia". *Psychiatria Danubina*. **21** (3): 371–5. PMID 19794359 (https://pubmed.ncbi.nlm.nih.gov/19794359).
81. Larson M (30 March 2006). "Alcohol-Related Psychosis" (http://www.emedicine.com/med/topic3113.htm). *EMedicine*. Archived (https://web.archive.org/web/20081109011819/http://www.emedicine.com/MED/topic3113.htm) from the original on 9 November 2008. Retrieved 27 September 2006.
82. Ortiz-Medina MB, Perea M, Torales J, Ventriglio A, Vitrani G, Aguilar L, Roncero C (November 2018). "Cannabis consumption and psychosis or schizophrenia development". *The International Journal of Social Psychiatry*. **64** (7): 690–704. doi:10.1177/0020764018801690 (https://doi.org/10.1177%2F0020764018801690). PMID 30442059 (https://pubmed.ncbi.nlm.nih.gov/30442059).
83. Leweke FM, Koethe D (June 2008). "Cannabis and psychiatric disorders: it is not only addiction". *Addiction Biology*. **13** (2): 264–75. doi:10.1111/j.1369-1600.2008.00106.x (https://doi.org/10.1111%2Fj.1369-1600.2008.00106.x). PMID 18482435 (https://pubmed.ncbi.nlm.nih.gov/18482435).
84. Arias I, Sorlozano A, Villegas E, et al. (April 2012). "Infectious agents associated with schizophrenia: a meta-analysis". *Schizophrenia Research*. **136** (1–3): 128–36. doi:10.1016/j.schres.2011.10.026 (https://doi.org/10.1016%2Fj.schres.2011.10.026). PMID 22104141 (https://pubmed.ncbi.nlm.nih.gov/22104141).
85. Yolken R (June 2004). "Viruses and schizophrenia: a focus on herpes simplex virus". *Herpes*. 11 Suppl 2 (Suppl 2): 83A–88A. PMID 15319094 (https://pubmed.ncbi.nlm.nih.gov/15319094).

86. Khandaker GM (August 2012). "Childhood infection and adult schizophrenia: a meta-analysis of population-based studies" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3485564). *Schizophr. Res*. **139** (1–3): 161–8. doi:10.1016/j.schres.2012.05.023 (https://doi.org/10.1016%2Fj.schres.2012.05.023). PMC 3485564 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3485564). PMID 22704639 (https://pubmed.ncbi.nlm.nih.gov/22704639).
87. Insel TR (November 2010). "Rethinking schizophrenia". *Nature*. **468** (7321): 187–93. Bibcode:2010Natur.468..187I (https://ui.adsabs.harvard.edu/abs/2010Natur.468..187I). doi:10.1038/nature09552 (https://doi.org/10.1038%2Fnature09552). PMID 21068826 (https://pubmed.ncbi.nlm.nih.gov/21068826).
88. Fusar-Poli P, Meyer-Lindenberg A (January 2013). "Striatal presynaptic dopamine in schizophrenia, part II: meta-analysis of [(18)F/(11)C]-DOPA PET studies" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3523905). *Schizophrenia Bulletin*. **39** (1): 33–42. doi:10.1093/schbul/sbr180 (https://doi.org/10.1093%2Fschbul%2Fsbr180). PMC 3523905 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3523905). PMID 22282454 (https://pubmed.ncbi.nlm.nih.gov/22282454).
89. Howes OD, Kambeitz J, Kim E, et al. (August 2012). "The nature of dopamine dysfunction in schizophrenia and what this means for treatment" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3730746). *Archives of General Psychiatry*. **69** (8): 776–86. doi:10.1001/archgenpsychiatry.2012.169 (https://doi.org/10.1001%2Farchgenpsychiatry.2012.169). PMC 3730746 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3730746). PMID 22474070 (https://pubmed.ncbi.nlm.nih.gov/22474070).
90. Broyd A, Balzan RP, Woodward TS, Allen P (June 2017). "Dopamine, cognitive biases and assessment of certainty: A neurocognitive model of delusions" (https://kclpure.kcl.ac.uk/portal/en/publications/dopamine-cognitive-biases-and-assessment-of-certainty-a-neurocognitive-model-of-delusions(138513dd-3a44-4deb-bac1-be45767ef51b).html). *Clinical Psychology Review*. **54**: 96–106. doi:10.1016/j.cpr.2017.04.006 (https://doi.org/10.1016%2Fj.cpr.2017.04.006). PMID 28448827 (https://pubmed.ncbi.nlm.nih.gov/28448827).
91. Howes OD, Murray RM (May 2014). "Schizophrenia: an integrated sociodevelopmental-cognitive model" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4127444). *Lancet*. **383** (9929): 1677–1687. doi:10.1016/S0140-6736(13)62036-X (https://doi.org/10.1016%2FS0140-6736%2813%2962036-X). PMC 4127444 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4127444). PMID 24315522 (https://pubmed.ncbi.nlm.nih.gov/24315522).
92. Grace AA (August 2016). "Dysregulation of the dopamine system in the pathophysiology of schizophrenia and depression" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5166560). *Nature Reviews. Neuroscience*. **17** (8): 524–32. doi:10.1038/nrn.2016.57 (https://doi.org/10.1038%2Fnrn.2016.57). PMC 5166560 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5166560). PMID 27256556 (https://pubmed.ncbi.nlm.nih.gov/27256556).
93. Goldman-Rakic PS, Castner SA, Svensson TH, Siever LJ, Williams GV (June 2004). "Targeting the dopamine D1 receptor in schizophrenia: insights for cognitive dysfunction". *Psychopharmacology*. **174** (1): 3–16. doi:10.1007/s00213-004-1793-y (https://doi.org/10.1007%2Fs00213-004-1793-y). PMID 15118803 (https://pubmed.ncbi.nlm.nih.gov/15118803).
94. Arnsten AF, Girgis RR, Gray DL, Mailman RB (January 2017). "Novel Dopamine Therapeutics for Cognitive Deficits in Schizophrenia" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4949134). *Biological Psychiatry*. **81** (1): 67–77. doi:10.1016/j.biopsych.2015.12.028 (https://doi.org/10.1016%2Fj.biopsych.2015.12.028). PMC 4949134 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4949134). PMID 26946382 (https://pubmed.ncbi.nlm.nih.gov/26946382).
95. Abi-Dargham A, Moore H (October 2003). "Prefrontal DA transmission at D1 receptors and the pathology of schizophrenia". *The Neuroscientist*. **9** (5): 404–16. doi:10.1177/1073858403252674 (https://doi.org/10.1177%2F1073858403252674). PMID 14580124 (https://pubmed.ncbi.nlm.nih.gov/14580124).

96. Maia TV, Frank MJ (January 2017). "An Integrative Perspective on the Role of Dopamine in Schizophrenia" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5486232). *Biological Psychiatry*. **81** (1): 52–66. doi:10.1016/j.biopsych.2016.05.021 (https://doi.org/10.1016%2Fj.biopsych.2016.05.021). PMC 5486232 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5486232). PMID 27452791 (https://pubmed.ncbi.nlm.nih.gov/27452791).
97. Catts VS, Lai YL, Weickert CS, Weickert TW, Catts SV (April 2016). "A quantitative review of the post-mortem evidence for decreased cortical N-methyl-D-aspartate receptor expression levels in schizophrenia: How can we link molecular abnormalities to mismatch negativity deficits?". *Biological Psychology*. **116**: 57–67. doi:10.1016/j.biopsycho.2015.10.013 (https://doi.org/10.1016%2Fj.biopsycho.2015.10.013). PMID 26549579 (https://pubmed.ncbi.nlm.nih.gov/26549579).
98. Michie PT, Malmierca MS, Harms L, Todd J (April 2016). "The neurobiology of MMN and implications for schizophrenia". *Biological Psychology*. **116**: 90–7. doi:10.1016/j.biopsycho.2016.01.011 (https://doi.org/10.1016%2Fj.biopsycho.2016.01.011). PMID 26826620 (https://pubmed.ncbi.nlm.nih.gov/26826620).
99. Pratt J, Dawson N, Morris BJ, et al. (February 2017). "Thalamo-cortical communication, glutamatergic neurotransmission and neural oscillations: A unique window into the origins of ScZ?" (http://eprints.gla.ac.uk/132874/1/132874.pdf) (PDF). *Schizophrenia Research*. **180**: 4–12. doi:10.1016/j.schres.2016.05.013 (https://doi.org/10.1016%2Fj.schres.2016.05.013). PMID 27317361 (https://pubmed.ncbi.nlm.nih.gov/27317361).
100. Marín O (January 2012). "Interneuron dysfunction in psychiatric disorders". *Nature Reviews. Neuroscience*. **13** (2): 107–20. doi:10.1038/nrn3155 (https://doi.org/10.1038%2Fnrn3155). PMID 22251963 (https://pubmed.ncbi.nlm.nih.gov/22251963).
101. Lewis DA, Hashimoto T, Volk DW (April 2005). "Cortical inhibitory neurons and schizophrenia". *Nature Reviews. Neuroscience*. **6** (4): 312–24. doi:10.1038/nrn1648 (https://doi.org/10.1038%2Fnrn1648). PMID 15803162 (https://pubmed.ncbi.nlm.nih.gov/15803162).
102. Senkowski D, Gallinat J (June 2015). "Dysfunctional prefrontal gamma-band oscillations reflect working memory and other cognitive deficits in schizophrenia". *Biological Psychiatry*. **77** (12): 1010–9. doi:10.1016/j.biopsych.2015.02.034 (https://doi.org/10.1016%2Fj.biopsych.2015.02.034). PMID 25847179 (https://pubmed.ncbi.nlm.nih.gov/25847179). "Several studies that investigated perceptual processes found impaired GBR in ScZ patients over sensory areas, such as the auditory and visual cortex. Moreover, studies examining steady-state auditory-evoked potentials showed deficits in the gen- eration of oscillations in the gamma band."
103. Reilly TJ, Nottage JF, Studerus E, et al. (July 2018). "Gamma band oscillations in the early phase of psychosis: A systematic review" (https://kclpure.kcl.ac.uk/portal/en/publications/gamma-band-oscillations-in-the-early-phase-of-psychosis(e2f500b7-f358-433d-9f9c-7f6b9c52bb2f).html). *Neuroscience and Biobehavioral Reviews*. **90**: 381–399. doi:10.1016/j.neubiorev.2018.04.006 (https://doi.org/10.1016%2Fj.neubiorev.2018.04.006). PMID 29656029 (https://pubmed.ncbi.nlm.nih.gov/29656029). "Decreased gamma power in response to a task was a relatively consistent finding, with 5 out of 6 studies reported reduced evoked or induced power."
104. Birnbaum R, Weinberger DR (December 2017). "Genetic insights into the neurodevelopmental origins of schizophrenia". *Nature Reviews. Neuroscience*. **18** (12): 727–740. doi:10.1038/nrn.2017.125 (https://doi.org/10.1038%2Fnrn.2017.125). PMID 29070826 (https://pubmed.ncbi.nlm.nih.gov/29070826).
105. Khandaker GM, Zimbron J, Lewis G, Jones PB (February 2013). "Prenatal maternal infection, neurodevelopment and adult schizophrenia: a systematic review of population-based studies" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3479084). *Psychological Medicine*. **43** (2): 239–57. doi:10.1017/S0033291712000736 (https://doi.org/10.1017%2FS0033291712000736). PMC 3479084 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3479084). PMID 22717193 (https://pubmed.ncbi.nlm.nih.gov/22717193).

106. Brown AS, Derkits EJ (March 2010). "Prenatal infection and schizophrenia: a review of epidemiologic and translational studies" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3652286). *The American Journal of Psychiatry*. **167** (3): 261–80. doi:10.1176/appi.ajp.2009.09030361 (https://doi.org/10.1176%2Fappi.ajp.2009.09030361). PMC 3652286 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3652286). PMID 20123911 (https://pubmed.ncbi.nlm.nih.gov/20123911).
107. Cannon TD (December 2015). "How Schizophrenia Develops: Cognitive and Brain Mechanisms Underlying Onset of Psychosis" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4673025). *Trends in Cognitive Sciences*. **19** (12): 744–756. doi:10.1016/j.tics.2015.09.009 (https://doi.org/10.1016%2Fj.tics.2015.09.009). PMC 4673025 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4673025). PMID 26493362 (https://pubmed.ncbi.nlm.nih.gov/26493362).
108. Lesh TA, Niendam TA, Minzenberg MJ, Carter CS (January 2011). "Cognitive control deficits in schizophrenia: mechanisms and meaning" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3052853). *Neuropsychopharmacology*. **36** (1): 316–38. doi:10.1038/npp.2010.156 (https://doi.org/10.1038%2Fnpp.2010.156). PMC 3052853 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3052853). PMID 20844478 (https://pubmed.ncbi.nlm.nih.gov/20844478).
109. Barch DM, Ceaser A (January 2012). "Cognition in schizophrenia: core psychological and neural mechanisms" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3860986). *Trends in Cognitive Sciences*. **16** (1): 27–34. doi:10.1016/j.tics.2011.11.015 (https://doi.org/10.1016%2Fj.tics.2011.11.015). PMC 3860986 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3860986). PMID 22169777 (https://pubmed.ncbi.nlm.nih.gov/22169777).
110. Eisenberg DP, Berman KF (January 2010). "Executive function, neural circuitry, and genetic mechanisms in schizophrenia" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2794926). *Neuropsychopharmacology*. **35** (1): 258–77. doi:10.1038/npp.2009.111 (https://doi.org/10.1038%2Fnpp.2009.111). PMC 2794926 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2794926). PMID 19693005 (https://pubmed.ncbi.nlm.nih.gov/19693005).
111. Walton E, Hibar DP, van Erp TG, et al. (May 2017). "Positive symptoms associate with cortical thinning in the superior temporal gyrus via the ENIGMA Schizophrenia consortium" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5399182). *Acta Psychiatrica Scandinavica*. **135** (5): 439–447. doi:10.1111/acps.12718 (https://doi.org/10.1111%2Facps.12718). PMC 5399182 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5399182). PMID 28369804 (https://pubmed.ncbi.nlm.nih.gov/28369804).
112. Walton E, Hibar DP, van Erp TG, et al. (Karolinska Schizophrenia Project Consortium (KaSP)) (January 2018). "Prefrontal cortical thinning links to negative symptoms in schizophrenia via the ENIGMA consortium" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5826665). *Psychological Medicine*. **48** (1): 82–94. doi:10.1017/S0033291717001283 (https://doi.org/10.1017%2FS0033291717001283). PMC 5826665 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5826665). PMID 28545597 (https://pubmed.ncbi.nlm.nih.gov/28545597).
113. Cohen AS, Minor KS (January 2010). "Emotional experience in patients with schizophrenia revisited: meta-analysis of laboratory studies" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2800132). *Schizophrenia Bulletin*. **36** (1): 143–50. doi:10.1093/schbul/sbn061 (https://doi.org/10.1093%2Fschbul%2Fsbn061). PMC 2800132 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2800132). PMID 18562345 (https://pubmed.ncbi.nlm.nih.gov/18562345).
114. Strauss GP, Gold JM (April 2012). "A new perspective on anhedonia in schizophrenia" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3732829). *The American Journal of Psychiatry*. **169** (4): 364–73. doi:10.1176/appi.ajp.2011.11030447 (https://doi.org/10.1176%2Fappi.ajp.2011.11030447). PMC 3732829 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3732829). PMID 22407079 (https://pubmed.ncbi.nlm.nih.gov/22407079).

115. Young J, Anticevic A, Barch D (2018). "Cognitive and Motivational Neuroscience of Psychotic Disorders". In Charney D, Buxbaum J, Sklar P, Nestler E (eds.). *Charney & Nestler's Neurobiology of Mental Illness* (5th ed.). New York: Oxford University Press. pp. 215, 217. ISBN 9780190681425. "Several recent reviews (e.g., Cohen and Minor, 2010) have found that individuals with schizophrenia show relatively intact self-reported emotional responses to affect-eliciting stimuli as well as other indicators of intact response(215)...Taken together, the literature increasingly suggests that there may be a deficit in putatively DA-mediated reward learning and/ or reward prediction functions in schizophrenia. Such findings suggest that impairment in striatal reward prediction mechanisms may influence “wanting” in schizophrenia in a way that reduces the ability of individuals with schizophrenia to use anticipated rewards to drive motivated behavior.(217)"
116. Friston KJ, Stephan KE, Montague R, Dolan RJ (July 2014). "Computational psychiatry: the brain as a phantastic organ". *The Lancet. Psychiatry*. **1** (2): 148–58. doi:10.1016/S2215-0366(14)70275-5 (https://doi.org/10.1016%2FS2215-0366%2814%2970275-5). PMID 26360579 (https://pubmed.ncbi.nlm.nih.gov/26360579).
117. Griffin JD, Fletcher PC (May 2017). "Predictive Processing, Source Monitoring, and Psychosis" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5424073). *Annual Review of Clinical Psychology*. **13**: 265–289. doi:10.1146/annurev-clinpsy-032816-045145 (https://doi.org/10.1146%2Fannurev-clinpsy-032816-045145). PMC 5424073 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5424073). PMID 28375719 (https://pubmed.ncbi.nlm.nih.gov/28375719).
118. Fletcher PC, Frith CD (January 2009). "Perceiving is believing: a Bayesian approach to explaining the positive symptoms of schizophrenia" (https://pure.au.dk/ws/files/48560345/fletcher_frithNRN.pdf) (PDF). *Nature Reviews. Neuroscience*. **10** (1): 48–58. doi:10.1038/nrn2536 (https://doi.org/10.1038%2Fnrn2536). PMID 19050712 (https://pubmed.ncbi.nlm.nih.gov/19050712).
119. Corlett PR, Taylor JR, Wang XJ, Fletcher PC, Krystal JH (November 2010). "Toward a neurobiology of delusions" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3676875). *Progress in Neurobiology*. **92** (3): 345–69. doi:10.1016/j.pneurobio.2010.06.007 (https://doi.org/10.1016%2Fj.pneurobio.2010.06.007). PMC 3676875 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3676875). PMID 20558235 (https://pubmed.ncbi.nlm.nih.gov/20558235).
120. American Psychiatric Association (2013). *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.). Arlington: American Psychiatric Publishing. ISBN 978-0-89042-555-8.
121. Soltan M, Girguis J (8 May 2017). "How to approach the mental state examination" (https://www.bmj.com/content/357/sbmj.j1821). *BMJ*. **357**: j1821. doi:10.1136/sbmj.j1821 (https://doi.org/10.1136%2Fsbmj.j1821). PMID 31055448 (https://pubmed.ncbi.nlm.nih.gov/31055448). Retrieved 9 January 2020.
122. Tandon R, Gaebel W, Barch DM, et al. (October 2013). "Definition and description of schizophrenia in the DSM-5". *Schizophrenia Research*. **150** (1): 3–10. doi:10.1016/j.schres.2013.05.028 (https://doi.org/10.1016%2Fj.schres.2013.05.028). PMID 23800613 (https://pubmed.ncbi.nlm.nih.gov/23800613).
123. As referenced from PMID 23800613 (https://pubmed.ncbi.nlm.nih.gov/23800613), Heckers S, Tandon R, Bustillo J (March 2010). "Catatonia in the DSM--shall we move or not?" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2833126). *Schizophrenia Bulletin* (Editorial). **36** (2): 205–7. doi:10.1093/schbul/sbp136 (https://doi.org/10.1093%2Fschbul%2Fsbp136). PMC 2833126 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2833126). PMID 19933711 (https://pubmed.ncbi.nlm.nih.gov/19933711).
124. Barch DM, Bustillo J, Gaebel W, et al. (October 2013). "Logic and justification for dimensional assessment of symptoms and related clinical phenomena in psychosis: relevance to DSM-5". *Schizophrenia Research*. **150** (1): 15–20. doi:10.1016/j.schres.2013.04.027 (https://doi.org/10.1016%2Fj.schres.2013.04.027). PMID 23706415 (https://pubmed.ncbi.nlm.nih.gov/23706415).

125. Jakobsen KD, Frederiksen JN, Hansen T, et al. (2005). "Reliability of clinical ICD-10 schizophrenia diagnoses". *Nordic Journal of Psychiatry*. **59** (3): 209–12. doi:10.1080/08039480510027698 (https://doi.org/10.1080%2F08039480510027698). PMID 16195122 (https://pubmed.ncbi.nlm.nih.gov/16195122).
126. American Psychiatric Association DSM-5 Work Groups (2010) Proposed Revisions – Schizophrenia and Other Psychotic Disorders (http://www.dsm5.org/ProposedRevisions/Pages/SchizophreniaandOtherPsychoticDisorders.aspx) Archived (https://web.archive.org/web/20110130153913/http://www.dsm5.org/PROPOSEDREVISIONS/Pages/SchizophreniaandOtherPsychoticDisorders.aspx) 30 January 2011 at the Wayback Machine. Retrieved 17 February 2010.
127. "The ICD-10 Classification of Mental and Behavioural Disorders" (http://www.who.int/classifications/icd/en/GRNBOOK.pdf) (PDF). World Health Organization. p. 26. Archived (https://web.archive.org/web/20160618131606/http://www.who.int/classifications/icd/en/GRNBOOK.pdf) (PDF) from the original on 18 June 2016.
128. "DSM-5 Changes: Schizophrenia & Psychotic Disorders" (http://pro.psychcentral.com/dsm-5-changes-schizophrenia-psychotic-disorders/004336.html). 29 May 2014. Archived (https://web.archive.org/web/20160501001901/http://pro.psychcentral.com/dsm-5-changes-schizophrenia-psychotic-disorders/004336.html) from the original on 1 May 2016. Retrieved 8 January 2016.
129. "ICD-10 Version:2016" (http://apps.who.int/classifications/icd10/browse/2016/en#/F20.8). *apps.who.int*. Archived (https://web.archive.org/web/20170208033519/http://apps.who.int/classifications/icd10/browse/2016/en#/F20.8) from the original on 8 February 2017. Retrieved 1 December 2017.
130. Bottas A (15 April 2009). "Comorbidity: Schizophrenia With Obsessive-Compulsive Disorder" (http://www.psychiatrictimes.com/display/article/10168/1402540). *Psychiatric Times*. **26** (4). Archived (https://web.archive.org/web/20130403064649/http://www.psychiatrictimes.com/display/article/10168/1402540) from the original on 3 April 2013.
131. Gabbard GO (15 May 2007). *Gabbard's Treatments of Psychiatric Disorders, Fourth Edition (Treatments of Psychiatric Disorders)* (https://books.google.com/?id=-T2aEqfwLW4C&pg=PA209). American Psychiatric Publishing. pp. 209–11. ISBN 978-1-58562-216-0.
132. Murray ED, Buttner N, Price BH (2012). "Depression and Psychosis in Neurological Practice". In Bradley WG, Daroff RB, Fenichel GM, Jankovic J (eds.). *Bradley's neurology in clinical practice*. **1** (6th ed.). Philadelphia, PA: Elsevier/Saunders. pp. 92–111. ISBN 978-1-4377-0434-1.
133. Cannon TD, Cornblatt B, McGorry P (May 2007). "The empirical status of the ultra high-risk (prodromal) research paradigm" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2526144). *Schizophrenia Bulletin*. **33** (3): 661–4. doi:10.1093/schbul/sbm031 (https://doi.org/10.1093%2Fschbul%2Fsbm031). PMC 2526144 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2526144). PMID 17470445 (https://pubmed.ncbi.nlm.nih.gov/17470445).
134. Marshall M, Rathbone J (June 2011). "Early intervention for psychosis" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4163966). *The Cochrane Database of Systematic Reviews* (6): CD004718. doi:10.1002/14651858.CD004718.pub3 (https://doi.org/10.1002%2F14651858.CD004718.pub3). PMC 4163966 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4163966). PMID 21678345 (https://pubmed.ncbi.nlm.nih.gov/21678345).
135. de Koning MB, Bloemen OJ, van Amelsvoort TA, et al. (June 2009). "Early intervention in patients at ultra high risk of psychosis: benefits and risks" (http://dare.ubvu.vu.nl/bitstream/1871/17133/2/Koning%2c%20de%20Acta%20Psychiatrica%20Scandinavica%20119%286%29%202009%20u.pdf) (PDF). *Acta Psychiatrica Scandinavica*. **119** (6): 426–42. doi:10.1111/j.1600-0447.2009.01372.x (https://doi.org/10.1111%2Fj.1600-0447.2009.01372.x). hdl:1871/17133 (https://hdl.handle.net/1871%2F17133). PMID 19392813 (https://pubmed.ncbi.nlm.nih.gov/19392813).

136. Stafford MR, Jackson H, Mayo-Wilson E, Morrison AP, Kendall T (January 2013). "Early interventions to prevent psychosis: systematic review and meta-analysis" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3548617). *BMJ*. **346**: f185. doi:10.1136/bmj.f185 (https://doi.org/10.1136%2Fbmj.f185). PMC 3548617 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3548617). PMID 23335473 (https://pubmed.ncbi.nlm.nih.gov/23335473).
137. "Psychosis and schizophrenia in adults: treatment and management" (https://web.archive.org/web/20140420070946/http://www.nice.org.uk/nicemedia/live/14382/66534/66534.pdf) (PDF). *NICE*. March 2014. p. 7. Archived from the original (http://www.nice.org.uk/nicemedia/live/14382/66534/66534.pdf) (PDF) on 20 April 2014. Retrieved 19 April 2014.
138. McGurk SR, Mueser KT, Feldman K, Wolfe R, Pascaris A (March 2007). "Cognitive training for supported employment: 2-3 year outcomes of a randomized controlled trial". *The American Journal of Psychiatry*. **164** (3): 437–41. doi:10.1176/appi.ajp.164.3.437 (https://doi.org/10.1176%2Fappi.ajp.164.3.437). PMID 17329468 (https://pubmed.ncbi.nlm.nih.gov/17329468).
139. Girdler SJ, Confino JE, Woesner ME (15 February 2019). "Exercise as a Treatment for Schizophrenia: A Review" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6386427). *Psychopharmacology Bulletin*. **49** (1): 56–69. PMC 6386427 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6386427). PMID 30858639 (https://pubmed.ncbi.nlm.nih.gov/30858639).
140. Firth J (1 May 2017). "Aerobic Exercise Improves Cognitive Functioning in People With Schizophrenia: A Systematic Review and Meta-Analysis" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5464163). *Schizophrenia Bulletin*. **43** (3): 546–556. doi:10.1093/schbul/sbw115 (https://doi.org/10.1093%2Fschbul%2Fsbw115). PMC 5464163 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5464163). PMID 27521348 (https://pubmed.ncbi.nlm.nih.gov/27521348).
141. Tréhout M, Dollfus S (December 2018). "[Physical activity in patients with schizophrenia: From neurobiology to clinical benefits]". *L'Encephale*. **44** (6): 538–547. doi:10.1016/j.encep.2018.05.005 (https://doi.org/10.1016%2Fj.encep.2018.05.005). PMID 29983176 (https://pubmed.ncbi.nlm.nih.gov/29983176).
142. "Psychosis - Treatment" (https://www.nhs.uk/conditions/psychosis/treatment/). *nhs.uk*. 3 October 2018. Retrieved 12 January 2020.
143. Ortiz-Orendain, J (12 December 2019). "Modafinil for people with schizophrenia or related disorders". *The Cochrane database of systematic reviews*. **12**: CD008661. doi:10.1002/14651858.CD008661.pub2 (https://doi.org/10.1002%2F14651858.CD008661.pub2). PMID 31828767 (https://pubmed.ncbi.nlm.nih.gov/31828767).
144. Keks N, Schwartz D, Hope J (October 2019). "Stopping and switching antipsychotic drugs". *Australian prescriber*. **42** (5): 152–157. doi:10.18773/austprescr.2019.052 (https://doi.org/10.18773%2Faustprescr.2019.052). PMID 31631928 (https://pubmed.ncbi.nlm.nih.gov/31631928).
145. Harrow M, Jobe TH (September 2013). "Does long-term treatment of schizophrenia with antipsychotic medications facilitate recovery?" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3756791). *Schizophrenia Bulletin*. **39** (5): 962–5. doi:10.1093/schbul/sbt034 (https://doi.org/10.1093%2Fschbul%2Fsbt034). PMC 3756791 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3756791). PMID 23512950 (https://pubmed.ncbi.nlm.nih.gov/23512950).
146. Lally J, MacCabe JH (2015). "Antipsychotic medication in schizophrenia: a review". *British Medical Bulletin*. **114** (1): 169–179. doi:10.1093/bmb/ldv017 (https://doi.org/10.1093%2Fbmb%2Fldv017). PMID 25957394 (https://pubmed.ncbi.nlm.nih.gov/25957394).
147. Barry SJ, Gaughan TM, Hunter R (June 2012). "Schizophrenia" (https://archive.is/20140911114812/http://www.clinicalevidence.bmj.com/x/systematic-review/1007/archive/06/2012.html). *BMJ Clinical Evidence*. **2012**. PMC 3385413 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3385413). PMID 23870705 (https://pubmed.ncbi.nlm.nih.gov/23870705). Archived from the original (http://www.clinicalevidence.bmj.com/x/systematic-review/1007/archive/06/2012.html) on 11 September 2014.

148. Taylor DM, Duncan-McConnell D (2000). "Refractory schizophrenia and atypical antipsychotics". *Journal of Psychopharmacology*. **14** (4): 409–18. doi:10.1177/026988110001400411 (https://doi.org/10.1177%2F026988110001400411). PMID 11198061 (https://pubmed.ncbi.nlm.nih.gov/11198061).
149. Essali A, Al-Haj Haasan N, Li C, Rathbone J (January 2009). "Clozapine versus typical neuroleptic medication for schizophrenia". *The Cochrane Database of Systematic Reviews* (1): CD000059. doi:10.1002/14651858.CD000059.pub2 (https://doi.org/10.1002%2F14651858.CD000059.pub2). PMID 19160174 (https://pubmed.ncbi.nlm.nih.gov/19160174).
150. Ananth J, Parameswaran S, Gunatilake S, Burgoyne K, Sidhom T (April 2004). "Neuroleptic malignant syndrome and atypical antipsychotic drugs" (https://semanticscholar.org/paper/59befa5208c42771cd65150de9656e6a1fd2dc83). *The Journal of Clinical Psychiatry*. **65** (4): 464–70. doi:10.4088/JCP.v65n0403 (https://doi.org/10.4088%2FJCP.v65n0403). PMID 15119907 (https://pubmed.ncbi.nlm.nih.gov/15119907).
151. Baier M (August 2010). "Insight in schizophrenia: a review". *Current Psychiatry Reports*. **12** (4): 356–61. doi:10.1007/s11920-010-0125-7 (https://doi.org/10.1007%2Fs11920-010-0125-7). PMID 20526897 (https://pubmed.ncbi.nlm.nih.gov/20526897).
152. McEvoy JP (2006). "Risks versus benefits of different types of long-acting injectable antipsychotics". *The Journal of Clinical Psychiatry*. 67 Suppl 5: 15–8. PMID 16822092 (https://pubmed.ncbi.nlm.nih.gov/16822092).
153. Leucht S, Tardy M, Komossa K, et al. (June 2012). "Antipsychotic drugs versus placebo for relapse prevention in schizophrenia: a systematic review and meta-analysis". *Lancet*. **379** (9831): 2063–71. doi:10.1016/S0140-6736(12)60239-6 (https://doi.org/10.1016%2FS0140-6736%2812%2960239-6). PMID 22560607 (https://pubmed.ncbi.nlm.nih.gov/22560607).
154. Ng QX, Soh AY, et al. (2019). "A Systematic Review of the Effect of Probiotic Supplementation on Schizophrenia Symptoms". *Neuropsychobiology*. **78** (1): 1–6. doi:10.1159/000498862 (https://doi.org/10.1159%2F000498862). PMID 30947230 (https://pubmed.ncbi.nlm.nih.gov/30947230).
155. Pharoah F, Mari J, Rathbone J, Wong W (December 2010). "Family intervention for schizophrenia" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4204509). *The Cochrane Database of Systematic Reviews*. **12** (12): CD000088. doi:10.1002/14651858.CD000088.pub2 (https://doi.org/10.1002%2F14651858.CD000088.pub2). PMC 4204509 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4204509). PMID 21154340 (https://pubmed.ncbi.nlm.nih.gov/21154340).
156. Medalia A, Choi J (September 2009). "Cognitive remediation in schizophrenia" (http://www.brown.uk.com/schizophrenia/medalia.pdf) (PDF). *Neuropsychology Review*. **19** (3): 353–64. doi:10.1007/s11065-009-9097-y (https://doi.org/10.1007%2Fs11065-009-9097-y). PMID 19444614 (https://pubmed.ncbi.nlm.nih.gov/19444614). Archived (https://web.archive.org/web/20161023084359/http://www.brown.uk.com/schizophrenia/medalia.pdf) (PDF) from the original on 23 October 2016.
157. Philipp R, Kriston L, Lanio J, et al. (2019). "Effectiveness of metacognitive interventions for mental disorders in adults-A systematic review and meta-analysis (METACOG)". *Clin Psychology & Psychotherapy*. **26** (2): 227–240. doi:10.1002/cpp.2345 (https://doi.org/10.1002%2Fcpp.2345). PMID 30456821 (https://pubmed.ncbi.nlm.nih.gov/30456821).
158. Dixon LB, Dickerson F, Bellack AS, et al. (January 2010). "The 2009 schizophrenia PORT psychosocial treatment recommendations and summary statements" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2800143). *Schizophrenia Bulletin*. **36** (1): 48–70. doi:10.1093/schbul/sbp115 (https://doi.org/10.1093%2Fschbul%2Fsbp115). PMC 2800143 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2800143). PMID 19955389 (https://pubmed.ncbi.nlm.nih.gov/19955389).
159. Bond GR, Drake RE (June 2015). "The critical ingredients of assertive community treatment". *World psychiatry : official journal of the World Psychiatric Association (WPA)*. **14** (2): 240–2. doi:10.1002/wps.20234 (https://doi.org/10.1002%2Fwps.20234). PMID 26043344 (https://pubmed.ncbi.nlm.nih.gov/26043344).

160. Smeerdijk M (2017). "[Using resource groups in assertive community treatment; literature review and recommendation]". *Tijdschrift voor psychiatrie*. **59** (8): 466–473. PMID 28880347 (https://pubmed.ncbi.nlm.nih.gov/28880347).
161. Dieterich M (6 January 2017). "Intensive case management for severe mental illness". *The Cochrane database of systematic reviews*. **1**: CD007906. doi:10.1002/14651858.CD007906.pub3 (https://doi.org/10.1002%2F14651858.CD007906.pub3). PMID 28067944 (https://pubmed.ncbi.nlm.nih.gov/28067944).
162. Jauhar S, McKenna PJ, Radua J, et al. (January 2014). "Cognitive-behavioural therapy for the symptoms of schizophrenia: systematic review and meta-analysis with examination of potential bias". *The British Journal of Psychiatry* (Review). **204** (1): 20–9. doi:10.1192/bjp.bp.112.116285 (https://doi.org/10.1192%2Fbjp.bp.112.116285). PMID 24385461 (https://pubmed.ncbi.nlm.nih.gov/24385461).
163. Jones C, Hacker D, Meaden A, et al. (November 2018). "Cognitive behavioural therapy plus standard care versus standard care plus other psychosocial treatments for people with schizophrenia" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6516879). *The Cochrane Database of Systematic Reviews*. **11**: CD008712. doi:10.1002/14651858.CD008712.pub3 (https://doi.org/10.1002%2F14651858.CD008712.pub3). PMC 6516879 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6516879). PMID 30480760 (https://pubmed.ncbi.nlm.nih.gov/30480760).
164. "Cognitive Behavioural Therapy for Psychosis: A Health Technology Assessment" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6235075). *Ontario Health Technology Assessment Series*. **18** (5): 1–141. 2018. PMC 6235075 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6235075). PMID 30443277 (https://pubmed.ncbi.nlm.nih.gov/30443277).
165. "CBT and treatment resistant schizophrenia" (https://discover.dc.nihr.ac.uk/content/signal-000678/cbt-and-treatment-resistant-schizophrenia). *discover.dc.nihr.ac.uk*. 20 November 2018. Retrieved 10 January 2020.
166. "Schizophrenia - Treatment" (https://www.nhs.uk/conditions/schizophrenia/treatment/). *nhs.uk*. 23 October 2017. Retrieved 8 January 2020.
167. Bastiampillai, Tarun; Allison, Stephen; Gupta, Arun (1 November 2016). "NICE guidelines for schizophrenia: can art therapy be justified?" (https://www.thelancet.com/journals/lanpsy/article/PIIS2215-0366(16)30322-4/fulltext). *The Lancet Psychiatry*. **3** (11): 1016–1017. doi:10.1016/S2215-0366(16)30322-4 (https://doi.org/10.1016%2FS2215-0366%2816%2930322-4). PMID 27794367 (https://pubmed.ncbi.nlm.nih.gov/27794367).
168. Ruddy R, Milnes D (October 2005). "Art therapy for schizophrenia or schizophrenia-like illnesses" (http://www.cochrane.org/reviews/en/ab003728.html). *The Cochrane Database of Systematic Reviews* (4): CD003728. doi:10.1002/14651858.CD003728.pub2 (https://doi.org/10.1002%2F14651858.CD003728.pub2). PMID 16235338 (https://pubmed.ncbi.nlm.nih.gov/16235338). Archived (https://web.archive.org/web/20111027132811/http://www2.cochrane.org/reviews/en/ab003728.html) from the original on 27 October 2011.
169. Ruddy RA, Dent-Brown K (January 2007). "Drama therapy for schizophrenia or schizophrenia-like illnesses" (http://www.cochrane.org/reviews/en/ab005378.html). *The Cochrane Database of Systematic Reviews* (1): CD005378. doi:10.1002/14651858.CD005378.pub2 (https://doi.org/10.1002%2F14651858.CD005378.pub2). PMID 17253555 (https://pubmed.ncbi.nlm.nih.gov/17253555). Archived (https://web.archive.org/web/20110825024045/http://www2.cochrane.org/reviews/en/ab005378.html) from the original on 25 August 2011.
170. Chien WT, Clifton AV, Zhao S, Lui S (4 April 2019). "Peer support for people with schizophrenia or other serious mental illness" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6448529). *The Cochrane Database of Systematic Reviews*. **4**: CD010880. doi:10.1002/14651858.CD010880.pub2 (https://doi.org/10.1002%2F14651858.CD010880.pub2). PMC 6448529 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6448529). PMID 30946482 (https://pubmed.ncbi.nlm.nih.gov/30946482).

171. Erlangsen A, Eaton WW, Mortensen PB, Conwell Y (February 2012). "Schizophrenia--a predictor of suicide during the second half of life?" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3266451). *Schizophrenia Research*. **134** (2–3): 111–7. doi:10.1016/j.schres.2011.09.032 (https://doi.org/10.1016%2Fj.schres.2011.09.032). PMC 3266451 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3266451). PMID 22018943 (https://pubmed.ncbi.nlm.nih.gov/22018943).
172. Saha S, Chant D, McGrath J (October 2007). "A systematic review of mortality in schizophrenia: is the differential mortality gap worsening over time?". *Archives of General Psychiatry*. **64** (10): 1123–31. doi:10.1001/archpsyc.64.10.1123 (https://doi.org/10.1001%2Farchpsyc.64.10.1123). PMID 17909124 (https://pubmed.ncbi.nlm.nih.gov/17909124).
173. Goroll AH, Mulley AG (2011). *Primary Care Medicine: Office Evaluation and Management of The Adult Patient: Sixth Edition* (https://books.google.com/books?id=fXpKo1afC2YC&pg=PT1766). Lippincott Williams & Wilkins. p. Chapter 101. ISBN 978-1-4511-2159-9.
174. Rizvi S, Gold J, Khan AM (5 August 2019). "Role of Naltrexone in Improving Compulsive Drinking in Psychogenic Polydipsia" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6777931). *Cureus*. **11** (8): e5320. doi:10.7759/cureus.5320 (https://doi.org/10.7759%2Fcureus.5320). PMC 6777931 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6777931). PMID 31598428 (https://pubmed.ncbi.nlm.nih.gov/31598428).
175. Ustün TB, Rehm J, Chatterji S, et al. (July 1999). "Multiple-informant ranking of the disabling effects of different health conditions in 14 countries. WHO/NIH Joint Project CAR Study Group". *Lancet*. **354** (9173): 111–5. doi:10.1016/S0140-6736(98)07507-2 (https://doi.org/10.1016%2FS0140-6736%2898%2907507-2). PMID 10408486 (https://pubmed.ncbi.nlm.nih.gov/10408486).
176. World Health Organization (2008). *The global burden of disease : 2004 update* ([Online-Ausg.] ed.). Geneva, Switzerland: World Health Organization. p. 35. ISBN 9789241563710.
177. Warner R (July 2009). "Recovery from schizophrenia and the recovery model". *Current Opinion in Psychiatry*. **22** (4): 374–80. doi:10.1097/YCO.0b013e32832c920b (https://doi.org/10.1097%2FYCO.0b013e32832c920b). PMID 19417668 (https://pubmed.ncbi.nlm.nih.gov/19417668).
178. Menezes NM, Arenovich T, Zipursky RB (October 2006). "A systematic review of longitudinal outcome studies of first-episode psychosis" (https://semanticscholar.org/paper/8539923c1b4f6bacb128d478b9ec7f6e95559210). *Psychological Medicine*. **36** (10): 1349–62. doi:10.1017/S0033291706007951 (https://doi.org/10.1017%2FS0033291706007951). PMID 16756689 (https://pubmed.ncbi.nlm.nih.gov/16756689).
179. Isaac M, Chand P, Murthy P (August 2007). "Schizophrenia outcome measures in the wider international community". *The British Journal of Psychiatry. Supplement*. **50**: s71–7. doi:10.1192/bjp.191.50.s71 (https://doi.org/10.1192%2Fbjp.191.50.s71). PMID 18019048 (https://pubmed.ncbi.nlm.nih.gov/18019048).
180. Cohen A, Patel V, Thara R, Gureje O (March 2008). "Questioning an axiom: better prognosis for schizophrenia in the developing world?" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2632419). *Schizophrenia Bulletin*. **34** (2): 229–44. doi:10.1093/schbul/sbm105 (https://doi.org/10.1093%2Fschbul%2Fsbm105). PMC 2632419 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2632419). PMID 17905787 (https://pubmed.ncbi.nlm.nih.gov/17905787).
181. Burns J (August 2009). "Dispelling a myth: developing world poverty, inequality, violence and social fragmentation are not good for outcome in schizophrenia". *African Journal of Psychiatry*. **12** (3): 200–5. doi:10.4314/ajpsy.v12i3.48494 (https://doi.org/10.4314%2Fajpsy.v12i3.48494). PMID 19894340 (https://pubmed.ncbi.nlm.nih.gov/19894340).
182. Palmer BA, Pankratz VS, Bostwick JM (March 2005). "The lifetime risk of suicide in schizophrenia: a reexamination". *Archives of General Psychiatry*. **62** (3): 247–53. doi:10.1001/archpsyc.62.3.247 (https://doi.org/10.1001%2Farchpsyc.62.3.247). PMID 15753237 (https://pubmed.ncbi.nlm.nih.gov/15753237).

183. Carlborg A, Winnerbäck K, Jönsson EG, Jokinen J, Nordström P (July 2010). "Suicide in schizophrenia". *Expert Review of Neurotherapeutics*. **10** (7): 1153–64. doi:10.1586/ern.10.82 (https://doi.org/10.1586%2Fern.10.82). PMID 20586695 (https://pubmed.ncbi.nlm.nih.gov/20586695).
184. de Leon J, Diaz FJ (July 2005). "A meta-analysis of worldwide studies demonstrates an association between schizophrenia and tobacco smoking behaviors". *Schizophrenia Research*. **76** (2–3): 135–57. doi:10.1016/j.schres.2005.02.010 (https://doi.org/10.1016%2Fj.schres.2005.02.010). PMID 15949648 (https://pubmed.ncbi.nlm.nih.gov/15949648).
185. Keltner NL, Grant JS (November 2006). "Smoke, smoke, smoke that cigarette". *Perspectives in Psychiatric Care*. **42** (4): 256–61. doi:10.1111/j.1744-6163.2006.00085.x (https://doi.org/10.1111%2Fj.1744-6163.2006.00085.x). PMID 17107571 (https://pubmed.ncbi.nlm.nih.gov/17107571).
186. *Diagnostic and statistical manual of mental disorders : DSM-IV-TR* (4 ed.). American Psychiatric Association. 2000. p. 304. ISBN 978-0-89042-025-6.
187. Kumari V, Postma P (January 2005). "Nicotine use in schizophrenia: the self medication hypotheses". *Neuroscience and Biobehavioral Reviews*. **29** (6): 1021–34. doi:10.1016/j.neubiorev.2005.02.006 (https://doi.org/10.1016%2Fj.neubiorev.2005.02.006). PMID 15964073 (https://pubmed.ncbi.nlm.nih.gov/15964073).
188. Cascio MT, Cella M, Preti A, Meneghelli A, Cocchi A (May 2012). "Gender and duration of untreated psychosis: a systematic review and meta-analysis". *Early Intervention in Psychiatry* (Review). **6** (2): 115–27. doi:10.1111/j.1751-7893.2012.00351.x (https://doi.org/10.1111%2Fj.1751-7893.2012.00351.x). PMID 22380467 (https://pubmed.ncbi.nlm.nih.gov/22380467).
189. Kumra S, Shaw M, Merka P, Nakayama E, Augustin R (December 2001). "Childhood-onset schizophrenia: research update". *Canadian Journal of Psychiatry*. **46** (10): 923–30. doi:10.1177/070674370104601004 (https://doi.org/10.1177%2F070674370104601004). PMID 11816313 (https://pubmed.ncbi.nlm.nih.gov/11816313).
190. Hassett A, Ames D, Chiu E, eds. (2005). *Psychosis in the Elderly* (https://books.google.com/?id=eLaMOJ9oj28C&printsec=frontcover&dq=Psychosis+in+the+Elderly). London: Taylor and Francis. p. 6. ISBN 978-1-84184-394-0.
191. Jablensky A, Sartorius N, Ernberg G, et al. (1992). "Schizophrenia: manifestations, incidence and course in different cultures. A World Health Organization ten-country study" (https://semanticscholar.org/paper/4b383519bc499ad88c08e57602856ff964233dde). *Psychological Medicine. Monograph Supplement*. **20**: 1–97. doi:10.1017/S0264180100000904 (https://doi.org/10.1017%2FS0264180100000904). PMID 1565705 (https://pubmed.ncbi.nlm.nih.gov/1565705).
192. Kirkbride JB, Fearon P, Morgan C, et al. (March 2006). "Heterogeneity in incidence rates of schizophrenia and other psychotic syndromes: findings from the 3-center AeSOP study". *Archives of General Psychiatry*. **63** (3): 250–8. doi:10.1001/archpsyc.63.3.250 (https://doi.org/10.1001%2Farchpsyc.63.3.250). PMID 16520429 (https://pubmed.ncbi.nlm.nih.gov/16520429).
193. Kirkbride JB, Fearon P, Morgan C, et al. (June 2007). "Neighbourhood variation in the incidence of psychotic disorders in Southeast London". *Social Psychiatry and Psychiatric Epidemiology*. **42** (6): 438–45. doi:10.1007/s00127-007-0193-0 (https://doi.org/10.1007%2Fs00127-007-0193-0). PMID 17473901 (https://pubmed.ncbi.nlm.nih.gov/17473901).
194. Lozano R, Naghavi M, Foreman K, et al. (December 2012). "Global and regional mortality from 235 causes of death for 20 age groups in 1990 and 2010: a systematic analysis for the Global Burden of Disease Study 2010". *Lancet*. **380** (9859): 2095–128. doi:10.1016/S0140-6736(12)61728-0 (https://doi.org/10.1016%2FS0140-6736%2812%2961728-0). hdl:10536/DRO/DU:30050819 (https://hdl.handle.net/10536%2FDRO%2FDU%3A30050819). PMID 23245604 (https://pubmed.ncbi.nlm.nih.gov/23245604).

195. Ayuso-Mateos JL. "Global burden of schizophrenia in the year 2000" (http://www.who.int/healthinfo/statistics/bod_schizophrenia.pdf) (PDF). World Health Organization. Archived (https://web.archive.org/web/20160304075113/http://www.who.int/healthinfo/statistics/bod_schizophrenia.pdf) (PDF) from the original on 4 March 2016. Retrieved 27 February 2013.
196. "Schizophrenia" (http://www.nimh.nih.gov/health/statistics/prevalence/schizophrenia.shtml). Archived (https://web.archive.org/web/20161004192420/https://www.nimh.nih.gov/health/statistics/prevalence/schizophrenia.shtml) from the original on 4 October 2016. Retrieved 29 December 2015.
197. Yuhas D. "Throughout History, Defining Schizophrenia Has Remained a Challenge" (http://www.scientificamerican.com/article.cfm?id=throughout-history-defining-schizophrenia-has-remained-challenge). Scientific American Mind (March/April 2013). Archived (https://web.archive.org/web/20131105011048/http://www.scientificamerican.com/article.cfm?id=throughout-history-defining-schizophrenia-has-remained-challenge) from the original on 5 November 2013. Retrieved 3 March 2013.
198. Heinrichs RW (2003). "Historical origins of schizophrenia: two early madmen and their illness". *Journal of the History of the Behavioral Sciences*. **39** (4): 349–63. doi:10.1002/jhbs.10152 (https://doi.org/10.1002%2Fjhbs.10152). PMID 14601041 (https://pubmed.ncbi.nlm.nih.gov/14601041).
199. Noll R (2011). *American madness: the rise and fall of dementia praecox*. Cambridge, MA: Harvard University Press. ISBN 978-0-674-04739-6.
200. Noll R (2012). "Whole body madness" (http://www.psychiatrictimes.com/blog/couchincrisis/content/article/10168/2104852). *Psychiatric Times*. **29** (12): 13–14. Archived (https://web.archive.org/web/20130111095522/http://www.psychiatrictimes.com/blog/couchincrisis/content/article/10168/2104852) from the original on 11 January 2013.
201. Hansen RA, Atchison B (2000). *Conditions in occupational therapy: effect on occupational performance*. Hagerstown, MD: Lippincott Williams & Wilkins. ISBN 978-0-683-30417-6.
202. Kuhn R, Cahn CH (September 2004). "Eugen Bleuler's concepts of psychopathology". *History of Psychiatry*. **15** (59 Pt 3): 361–6. doi:10.1177/0957154X04044603 (https://doi.org/10.1177%2F0957154X04044603). PMID 15386868 (https://pubmed.ncbi.nlm.nih.gov/15386868).
203. Stotz-Ingenlath G (2000). "Epistemological aspects of Eugen Bleuler's conception of schizophrenia in 1911" (http://www.kluweronline.com/art.pdf?issn=1386-7423&volume=3&page=153) (PDF). *Medicine, Health Care and Philosophy*. **3** (2): 153–9. doi:10.1023/A:1009919309015 (https://doi.org/10.1023%2FA%3A1009919309015). PMID 11079343 (https://pubmed.ncbi.nlm.nih.gov/11079343).
204. McNally K (May 2009). "Eugene Bleuler's four As". *History of Psychology*. **12** (2): 43–59. doi:10.1037/a0015934 (https://doi.org/10.1037%2Fa0015934). PMID 19831234 (https://pubmed.ncbi.nlm.nih.gov/19831234).
205. McNally K (2016). "The Split Personality". *A Critical History of Schizophrenia*. Palgrave Macmillan UK. pp. 21–38. doi:10.1057/9781137456816_3 (https://doi.org/10.1057%2F9781137456816_3). ISBN 978-1-349-55226-9.
206. Schneider K (1959). *Clinical Psychopathology* (https://books.google.com/?id=ofzOAAAAMAAJ) (5 ed.). New York: Grune & Stratton.
207. Picardi A (2019). "The Two Faces of First-Rank Symptoms". *Psychopathology*. **52** (4): 221–231. doi:10.1159/000503152 (https://doi.org/10.1159%2F000503152). PMID 31610542 (https://pubmed.ncbi.nlm.nih.gov/31610542).
208. Turner T (January 2007). "Chlorpromazine: unlocking psychosis". *BMJ*. 334 Suppl 1 (suppl): s7. doi:10.1136/bmj.39034.609074.94 (https://doi.org/10.1136%2Fbmj.39034.609074.94). PMID 17204765 (https://pubmed.ncbi.nlm.nih.gov/17204765).

209. Wilson M (March 1993). "DSM-III and the transformation of American psychiatry: a history". *The American Journal of Psychiatry*. **150** (3): 399–410. doi:10.1176/ajp.150.3.399 (https://doi.org/10.1176%2Fajp.150.3.399). PMID 8434655 (https://pubmed.ncbi.nlm.nih.gov/8434655).
210. Fischer BA (December 2012). "A review of American psychiatry through its diagnoses: the history and development of the Diagnostic and Statistical Manual of Mental Disorders". *The Journal of Nervous and Mental Disease*. **200** (12): 1022–30. doi:10.1097/NMD.0b013e318275cf19 (https://doi.org/10.1097%2FNMD.0b013e318275cf19). PMID 23197117 (https://pubmed.ncbi.nlm.nih.gov/23197117).
211. Sato M (February 2006). "Renaming schizophrenia: a Japanese perspective" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1472254). *World Psychiatry*. **5** (1): 53–5. PMC 1472254 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1472254). PMID 16757998 (https://pubmed.ncbi.nlm.nih.gov/16757998).
212. Lee YS, Kim JJ, Kwon JS (August 2013). "Renaming schizophrenia in South Korea". *Lancet*. **382** (9893): 683–4. doi:10.1016/S0140-6736(13)61776-6 (https://doi.org/10.1016%2FS0140-6736%2813%2961776-6). PMID 23972810 (https://pubmed.ncbi.nlm.nih.gov/23972810).
213. van Os J (February 2016). " "Schizophrenia" does not exist". *BMJ*. **352**: i375. doi:10.1136/bmj.i375 (https://doi.org/10.1136%2Fbmj.i375). PMID 26837945 (https://pubmed.ncbi.nlm.nih.gov/26837945).
214. Wu EQ, Birnbaum HG, Shi L, et al. (September 2005). "The economic burden of schizophrenia in the United States in 2002". *The Journal of Clinical Psychiatry*. **66** (9): 1122–9. doi:10.4088/jcp.v66n0906 (https://doi.org/10.4088%2Fjcp.v66n0906). PMID 16187769 (https://pubmed.ncbi.nlm.nih.gov/16187769).
215. Maniglio R (March 2009). "Severe mental illness and criminal victimization: a systematic review". *Acta Psychiatrica Scandinavica*. **119** (3): 180–91. doi:10.1111/j.1600-0447.2008.01300.x (https://doi.org/10.1111%2Fj.1600-0447.2008.01300.x). PMID 19016668 (https://pubmed.ncbi.nlm.nih.gov/19016668).
216. Fazel S, Gulati G, Linsell L, Geddes JR, Grann M (August 2009). "Schizophrenia and violence: systematic review and meta-analysis" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2718581). *PLoS Medicine*. **6** (8): e1000120. doi:10.1371/journal.pmed.1000120 (https://doi.org/10.1371%2Fjournal.pmed.1000120). PMC 2718581 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2718581). PMID 19668362 (https://pubmed.ncbi.nlm.nih.gov/19668362).
217. Large M, Smith G, Nielssen O (July 2009). "The relationship between the rate of homicide by those with schizophrenia and the overall homicide rate: a systematic review and meta-analysis". *Schizophrenia Research*. **112** (1–3): 123–9. doi:10.1016/j.schres.2009.04.004 (https://doi.org/10.1016%2Fj.schres.2009.04.004). PMID 19457644 (https://pubmed.ncbi.nlm.nih.gov/19457644).
218. Bo S, Abu-Akel A, Kongerslev M, Haahr UH, Simonsen E (July 2011). "Risk factors for violence among patients with schizophrenia". *Clinical Psychology Review*. **31** (5): 711–26. doi:10.1016/j.cpr.2011.03.002 (https://doi.org/10.1016%2Fj.cpr.2011.03.002). PMID 21497585 (https://pubmed.ncbi.nlm.nih.gov/21497585).
219. Valença AM, de Moraes TM (October 2006). "[Relationship between homicide and mental disorders]". *Revista Brasileira de Psiquiatria*. 28 Suppl 2: S62–8. doi:10.1590/s1516-44462006000600003 (https://doi.org/10.1590%2Fs1516-44462006000600003). PMID 17143446 (https://pubmed.ncbi.nlm.nih.gov/17143446).
220. Pescosolido BA, Monahan J, Link BG, Stueve A, Kikuzawa S (September 1999). "The public's view of the competence, dangerousness, and need for legal coercion of persons with mental health problems" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1508769). *American Journal of Public Health*. **89** (9): 1339–45. doi:10.2105/AJPH.89.9.1339 (https://doi.org/10.2105%2FAJPH.89.9.1339). PMC 1508769 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1508769). PMID 10474550 (https://pubmed.ncbi.nlm.nih.gov/10474550).

221. Phelan JC, Link BG, Stueve A, Pescosolido BA (June 2000). "Public Conceptions of Mental Illness in 1950 and 1996: What Is Mental Illness and Is It to be Feared?" (https://semanticscholar.org/paper/eba39c2a85b5823239d3ea6f8e4bc3dc75cdddf1). *Journal of Health and Social Behavior*. **41** (2): 188–207. doi:10.2307/2676305 (https://doi.org/10.2307%2F2676305). JSTOR 2676305 (https://www.jstor.org/stable/2676305).
222. Stetka, Bret (24 March 2015). "Why Don't Animals Get Schizophrenia (and How Come We Do)?" (https://www.scientificamerican.com/article/why-don-t-animals-get-schizophrenia-and-how-come-we-do/). *Scientific American*.
223. Gil-da-Costa R, Stoner GR, Fung R, Albright TD (September 2013). "Nonhuman primate model of schizophrenia using a noninvasive EEG method" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3780912). *Proceedings of the National Academy of Sciences of the United States of America*. **110** (38): 15425–30. Bibcode:2013PNAS..11015425G (https://ui.adsabs.harvard.edu/abs/2013PNAS..11015425G). doi:10.1073/pnas.1312264110 (https://doi.org/10.1073%2Fpnas.1312264110). PMC 3780912 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3780912). PMID 23959894 (https://pubmed.ncbi.nlm.nih.gov/23959894).
224. Dean OM, Data-Franco J, Giorlando F, Berk M (May 2012). "Minocycline: therapeutic potential in psychiatry". *CNS Drugs*. **26** (5): 391–401. doi:10.2165/11632000-000000000-00000 (https://doi.org/10.2165%2F11632000-000000000-00000). PMID 22486246 (https://pubmed.ncbi.nlm.nih.gov/22486246).
225. Chamberlain IJ, Sampson S (March 2013). Chamberlain IJ (ed.). "Nidotherapy for people with schizophrenia" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6492500). *The Cochrane Database of Systematic Reviews*. **3** (3): CD009929. doi:10.1002/14651858.CD009929.pub2 (https://doi.org/10.1002%2F14651858.CD009929.pub2). PMC 6492500 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6492500). PMID 23543583 (https://pubmed.ncbi.nlm.nih.gov/23543583).
226. Chue P, Lalonde JK (2014). "Addressing the unmet needs of patients with persistent negative symptoms of schizophrenia: emerging pharmacological treatment options" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4020880). *Neuropsychiatric Disease and Treatment*. **10**: 777–89. doi:10.2147/ndt.s43404 (https://doi.org/10.2147%2Fndt.s43404). PMC 4020880 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4020880). PMID 24855363 (https://pubmed.ncbi.nlm.nih.gov/24855363).
227. Keller WR, Kum LM, Wehring HJ, et al. (April 2013). "A review of anti-inflammatory agents for symptoms of schizophrenia" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3641824). *Journal of Psychopharmacology*. **27** (4): 337–42. doi:10.1177/0269881112467089 (https://doi.org/10.1177%2F0269881112467089). PMC 3641824 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3641824). PMID 23151612 (https://pubmed.ncbi.nlm.nih.gov/23151612).
228. Nieuwdorp, W; Koops, S; Somers, M; Sommer, IE (May 2015). "Transcranial magnetic stimulation, transcranial direct current stimulation and electroconvulsive therapy for medication-resistant psychosis of schizophrenia". *Current Opinion in Psychiatry*. **28** (3): 222–8. doi:10.1097/YCO.0000000000000156 (https://doi.org/10.1097%2FYCO.0000000000000156). PMID 25768083 (https://pubmed.ncbi.nlm.nih.gov/25768083).
229. Nathou C, Etard O, Dollfus S (2019). "Auditory verbal hallucinations in schizophrenia: current perspectives in brain stimulation treatments" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6662171). *Neuropsychiatric Disease and Treatment*. **15**: 2105–2117. doi:10.2147/NDT.S168801 (https://doi.org/10.2147%2FNDT.S168801). PMC 6662171 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6662171). PMID 31413576 (https://pubmed.ncbi.nlm.nih.gov/31413576).
230. Dougall, N; Maayan, N; Soares-Weiser, K; McDermott, LM; McIntosh, A (20 August 2015). "Transcranial magnetic stimulation (TMS) for schizophrenia". *The Cochrane Database of Systematic Reviews*. **2015** (8): CD006081. doi:10.1002/14651858.CD006081.pub2 (https://doi.org/10.1002%2F14651858.CD006081.pub2). hdl:1893/22520 (https://hdl.handle.net/1893%2F22520). PMID 26289586 (https://pubmed.ncbi.nlm.nih.gov/26289586).

External links

- Media related to Schizophrenia at Wikimedia Commons
- Schizophrenia (https://curlie.org/Health/Mental_Health/Disorders/Schizophrenia/) at Curlie

Classification	**ICD-10**: F20 (http://apps.who.int/classifications/icd10/browse/2016/en#/F20) · **ICD-9-CM**: 295 (http://www.icd9data.com/getICD9Code.ashx?icd9=295) · **OMIM**: 181500 (https://omim.org/entry/181500) · **MeSH**: D012559 (https://www.nlm.nih.gov/cgi/mesh/2015/MB_cgi?field=uid&term=D012559) · **DiseasesDB**: 11890 (http://www.diseasesdatabase.com/ddb11890.htm)
External resources	**MedlinePlus**: 000928 (https://www.nlm.nih.gov/medlineplus/ency/article/000928.htm) · **eMedicine**: med/2072 (https://emedicine.medscape.com/med/2072-overview) emerg/520 (http://www.emedicine.com/emerg/topic520.htm#) · **Patient UK**: Schizophrenia (https://patient.info/doctor/schizophrenia-pro) · **Scholia**: Q41112 (https://tools.wmflabs.org/scholia/topic/Q41112)