FACTBOOK

AO1 and AO3

SCHIZOPHRENIA

Schizophrenia

Classification of schizophrenia

Schizophrenia is a psychotic disorder characterised by hallucinations and delusions.

Schizophrenia has a prevalence of 1% in the general population and can be diagnosed as acute or chronic, depending on the symptoms.

Schizophrenia can affect both men and women but is more commonly diagnosed in men.

The onset of schizophrenia in men tends to be around the ages of 18 to 25 years old.

The onset of schizophrenia in women tends to be around the ages of 25 to 30 years old.

The onset of schizophrenia tends to occur in early adulthood, with men experiencing symptoms around the ages of 18-25 and women around the ages of 25-30.

Acute (type I) schizophrenia has more positive symptoms and responds well to treatment.

Chronic (type II) schizophrenia has more of the negative symptoms and is less responsive to treatment.

Classification of mental disorder is the process of organising symptoms into categories based on which symptoms frequently cluster together.

The two main classification systems for mental health disorders are; the International Classification of Disease (ICD) outlined by the World Health Organisation, which is currently on version 11, and the Diagnostic and Statistical Manual version 5 which is outlined by the American Psychiatric Association.

The purpose behind designing a manual of mental disorders is to allow psychiatrists and medical professionals to diagnose people who have mental disorders appropriately. This means that they will subsequently receive the correct form of treatment and hopefully manage their symptoms to live a normal life.

A diagnostic system such as DSM or ICD is used to categorise the symptoms in order to make a formal diagnosis for treatment.

The ICD-11 and DSM-5 are the two main classification systems for mental health disorders, including schizophrenia.

Each diagnostic system has a set of criteria specific to every disorder which helps classify the symptoms so patients receive a reliable and valid diagnosis.

The Diagnostic Statistical Manual (DSM) is currently on version 5 and recognises groups of symptoms that make up a particular disorder.

According to Spitzer & Fleiss (1974) the use of diagnostic manuals and systems means more communication between clinicians, which is likely to increase reliability.

Positive symptoms are those are in addition to normal life experiences and are mostly concerned with losing touch of reality.

Positive symptoms of schizophrenia include hallucinations, delusions, and disorganised thinking.

Cognitive symptoms of schizophrenia include difficulties with attention, memory, and executive functioning.

Negative symptoms detract from normal life experiences and can include anhedonia (loss of pleasure in life), disturbances of affect like emotional flattening and disturbances in thought processing and psychomotor ability.

Studies have suggested that men are more likely to present with positive symptoms of schizophrenia whereas women are more likely to present with the negative symptoms.

The symptoms of schizophrenia can interfere severely with everyday tasks, so that many people with schizophrenia end up homeless or hospitalised.

To get a diagnosis, patients would typically self-report symptoms in a clinical interview with a psychiatrist. The observations of a family member or friend would also be taken into account.

During the diagnosis process questions would be asked about everyday functioning, early life and childhood.

The diagnosis process can take several months and depending on the onset, patients may need to be hospitalised beforehand.

Patients with sudden and severe mental health issues may be admitted to a hospital for their own safety and may be sectioned under the Mental Health Act (2007).

Diagnosis and classification are interlinked; in order to diagnose a specific clinical disorder we must distinguish one disorder from another.

To distinguish one disorder from another we identify clusters of symptoms that occur together and subsequently classify this as one disorder.

Diagnosis is possible by identifying symptoms and deciding based on the criteria which disorder a patient has.

The DSM-5 criteria for diagnosing schizophrenia include the presence of at least two of the following symptoms: delusions, hallucinations, disorganised speech, grossly disorganised or catatonic behaviour, and negative symptoms.

Symptoms of Schizophrenia

The symptoms of schizophrenia can be broken down into two categories; positive and negative.

Positive

Positive symptoms of schizophrenia are additional experiences that are beyond those of ordinary existence. Examples include hallucinations which can be visual or auditory, and delusions.

Hallucinations are sensory experiences that are not based in reality, such as hearing voices or seeing things that are not there.

Hallucinations are a positive symptom of schizophrenia and are sensory experiences that either have no basis in reality or are distorted perceptions of things that are there.

Hallucinations are sensory experiences that patients with schizophrenia experience such as seeing distortions in objects, apparitions or hearing voices.

Hallucinations are perceptual disturbances in schizophrenia.

Hallucinations that are auditory involve hearing voices.

Hallucinations that are visual involve seeing things that do not exist.

Hallucinations can be experienced in relation to any of the five senses.

Delusions are false beliefs that are not based in reality, such as believing that someone is trying to harm you or that you have special powers.

Delusions are distorted or irrational beliefs that patients with schizophrenia experience about themselves or the world, such as feelings of persecution or grandeur.

Delusions can include paranoia and irrational thought processes and these can take a range of forms.

Delusions are cognitive symptoms in schizophrenia.

Delusions of persecution are common in patients with schizophrenia.

Delusions of control involve patients thinking other people are controlling them.

Delusions of grandeur involve patients thinking they have a higher status than others, some may protest to be a king or queen.

Negative

Negative symptoms of schizophrenia are when patients experience a loss of normal experiences and abilities. Examples are avolition, speech poverty or an inability to communicate fluently.

Avolition which is sometimes called apathy is a negative symptom of schizophrenia. It is described as finding difficulty in goal directed activity.

Andreasen (1982) identified three signs of avolition; poor hygiene and grooming, lack of persistence in work or education and a lack of energy.

Avolition is a negative symptom of schizophrenia which involves a lack of motivation to carry out tasks and results in lowered activity levels.

Avolition is a fatigue like symptom where patients show a lack of personal care and an inability to make plans.

Affective flattening is a negative symptom of schizophrenia where patients show a lack of emotion or demonstrate inappropriate emotional responses.

Avolition and affective flattening are emotional characteristics of schizophrenia.

Schizophrenia is characterised by changes in patterns of speech.

Speech poverty is a negative symptom of schizophrenia which involves reduced frequency and quality of speech.

Speech poverty is seen as a negative symptom because the emphasis is on reduction in the amount and quality of speech in schizophrenia. This is sometimes accompanied by a delay in the person's verbal responses during a conversation.

With schizophrenia patients can experience speech disorganisation where they become incoherent or change topic mid-sentence.

Language impairments such as alogia and echolalia are negative symptoms of schizophrenia.

Alogia is the lessening of speech as described in speech poverty.

Echolalia is a negative symptom of schizophrenia where patients copy sounds from the environment around them. It can also be the meaningless repetition of words.

Some patients with schizophrenia show behavioural disturbances which affect their psychomotor ability. They may lose control of their muscles or hold posture positions for a long period of time (catatonic behaviour).

Reliability and Validity in Diagnosis

Reliability

Reliability and validity are two important factors when designing a test or any kind of diagnostic tool for mental illness.

Psychologists like Rosenhan (1973) highlighted the problems with diagnosis process and suggested that reliability and validity were essential factors in making sure patients received the treatment required.

Reliability is the extent to which something can produce consistent results through controlled replication.

Inter-rater reliability is when two or more psychiatrists agree on the same diagnosis for a patient.

According to Mattison et al (1979) the general inter-rater reliability of DSM-II (which was used in Rosenhan's study to diagnose the pseudo patients) was about 57%. This does not show a high level of agreement for the professionals using this to diagnose patients.

Goldstein (1988) found high levels of inter-rater reliability when patients were diagnosed in relation to schizophrenia.

Test re-test reliability is when a psychiatrist reaches the same diagnosis on more than one occasion with the same patient.

Test-retest reliability refers to the consistency of a diagnosis of schizophrenia when the same individual is assessed on two separate occasions.

Read et al (2004) found a 37% concordance rate in test re-test reliability for schizophrenia, between two clinicians.

Split-half reliability in schizophrenia diagnosis refers to the consistency of diagnoses made by splitting the diagnostic tool into two halves and comparing the results.

Reliability in diagnosis is important to ensure patients receive the correct treatment.

Söderberg et al (2005) found a concordance rate of 81% using the DSM-IV classification for schizophrenia.

Prior to DSM-5 reliability for the diagnosis of schizophrenia was low but Osorio et al (2019) report excellent reliability in over 180 schizophrenic patients using the criteria of the DSM-5. They found inter-rater reliability to be +0.97 and test re-test +0.92, illustrating high correlations. This tells us that we can be reasonably certain the diagnosis of schizophrenia is consistently applied.

Jakobsen et al (2005) found a concordance rate of 98% for schizophrenia from a sample of 100 patients in Denmark using the ICD-10 classification system.

Ward et al (1962) identified three factors which affect the reliability of diagnosis. First are client/ patient factors, second are clinician factors and third are classification factors.

Client/ patient factors which may affect the reliability of diagnosis could be as a result of the severity of the symptoms or atypical characteristics they present.

Clinician factors which may affect the reliability of diagnosis could be the result of how well trained or experienced they are, or how well they develop a rapport with their patient.

Classification factors in the reliability of diagnosis could be related to the criteria used, depending on which classification system is used (DSM or ICD) and how they define the disorders.

If a clinician gives a diagnosis to one person presenting with a set of symptoms and then gives another person presenting with the same issues a different diagnosis, this shows unreliability in the diagnosis.

Validity

To receive a valid diagnosis patients must present with symptoms that are true of that disorder. If they do not describe symptoms that are true of the disorder, they cannot receive a formal diagnosis.

A valid diagnosis is one where the symptoms match the label so if someone suffering from hallucinations and thought insertion are diagnosed as schizophrenia then this is valid.

Descriptive validity refers to the symptoms of a disorder being unique and distinct from another disorder.

Aetiological validity refers to all the people with a disorder appearing to experience it in the same way.

Face validity in schizophrenia diagnosis refers to the extent to which the diagnostic criteria appear to be valid and relevant to the symptoms and characteristics of schizophrenia.

Construct validity in schizophrenia diagnosis refers to the extent to which the diagnostic criteria accurately measure the underlying construct of schizophrenia; does it measure what it claims to.

Criterion validity in schizophrenia diagnosis refers to the extent to which we can predict future outcomes or behaviours related to schizophrenia using the criteria in the classification system.

There are two different types of criterion validity: concurrent and predictive.

Concurrent validity in schizophrenia diagnosis refers to the extent to which the diagnostic criteria align with other established measures or diagnoses of schizophrenia.

Predictive validity in schizophrenia diagnosis refers to the extent to which the diagnostic criteria accurately predict future outcomes or behaviours related to schizophrenia, such as treatment response or long-term prognosis.

Mason et al (1997) followed 99 patients with schizophrenia over 13 years to see if the predictive validity of the main classification systems worked. They found that if they used the symptoms that were present over six months, there was good predictive validity.

Rosenhan (1973) found that the diagnosis of schizophrenia lacked validity, and that patients were treated as a reflection of the label they were given, not on their individual symptoms or characteristics.

Rosenhan (1973) found that DSM-III was not a valid diagnostic tool as all of the pseudopatients were diagnosed incorrectly with having a mental disorder.

Jablensky (2010) conducted a review and found that for the time being, the clinical concept of schizophrenia is valid and is supported by empirical evidence.

DSM-5 reflects the shift in the opinion of clinicians that it may not be possible to diagnose schizophrenia as a separate disorder because clients present with such different symptoms. This was supported by Jansson & Parnas (2007), who reviewed 92 studies that applied different definitions of schizophrenia to the same patient samples. They found that both ICD-10 and DSM-

IV had reasonable reliability but low validity, which suggests schizophrenia may not be a separate condition.

Co-morbidity in Schizophrenia

Comorbidity refers to the coexistence of two separate conditions or disorders at the same time. This adds to the difficulty of making valid and reliable diagnoses.

It is common for someone with schizophrenia to also experience depression and/or OCD.

Mood disorders, such as depression or bipolar disorder, are often seen alongside schizophrenia.

Anxiety disorders, such as generalised anxiety disorder or panic disorder, can co-occur with schizophrenia.

Substance use disorders, such as alcohol or drug abuse, are commonly co-morbid with schizophrenia.

Depression shares some of the negative symptoms of schizophrenia, this means there is symptom overlap.

Buckley et al (2008) reported that around 50% of patients with schizophrenia also had depression.

Jeste et al (1996) claim that patients with comorbid symptoms are in the majority and yet are left out of many clinical studies.

The high levels of comorbid symptoms seen in schizophrenia suggest it is not a clearly defined mental illness and there can be problems with the validity of the diagnosis.

DSM-5 has moved schizophrenia to a spectrum like disorder as patients present with varying degrees from mild to severe.

Comorbidity is a limitation with the diagnosis of schizophrenia. If disorders occur together this calls into question the validity of their individual diagnosis.

Schizophrenia is commonly diagnosed with other mental health conditions. This is a problem for the classification because it means schizophrenia may not exist as a distinct condition.

Symptom Overlap in Schizophrenia

Symptom overlap occurs when the symptoms of two mental illnesses are very similar. It is very problematic between schizophrenia and bipolar disorder.

Konstantareas & Hewitt (2001) compared 14 people with autism and 14 people with schizophrenia. None of those with schizophrenia had symptoms of autism but seven (50%) of those with autism reported symptoms of schizophrenia.

Symptom overlap between schizophrenia and autism can complicate the process of distinguishing between the two disorders.

Identifying and understanding the specific symptoms that differentiate schizophrenia from autism is crucial for accurate diagnosis and appropriate treatment.

Ripke et al (2011) looked at 50,000 patients' genetic material. They found that of seven gene locations on the genome associated with schizophrenia, three were also associated with bipolar disorder. This suggests there was a genetic overlap between the two disorders.

Genetic overlap between disorders suggests that clusters of genes may be responsible for the conditions which opens up the possibility of gene therapy.

Neuroscience is showing clear differences in the brains of sufferers with schizophrenia which are not shown in other disorders. Scanning techniques may help in the diagnosis and subsequently may result in less symptom overlap overall.

Ketter (2005) points out that misdiagnosis due to symptom overlap could lead to the wrong treatment and needless suffering.

The stickiness of labels and the negative association with a diagnosis of schizophrenia may make clinicians especially cautious. It is not uncommon for a patient to have two possible diagnoses for a couple of months, until the picture becomes clearer.

Symptom overlap is a limitation of schizophrenia diagnosis.

Both schizophrenia and bipolar disorder can involve positive symptoms such as delusions and negative symptoms such as avolition.

Misdiagnosis due to symptom overlap between schizophrenia and bipolar disorder can lead to incorrect treatment and unnecessary suffering.

Gender & Culture Bias in Schizophrenia

Gender Bias

Schizophrenia was originally believed to have equal incidence in males and females. However, it is thought that gender bias could have masked the fact that women have a very different experience of schizophrenia than men.

Castle et al (1991) argue that there are gender differences in the experience of schizophrenia. Females typically have a less severe experience but are more likely to show negative symptoms. Whereas males have a more severe experience and typically describe more positive symptoms.

Castle et al (1993) found that the male incidence of schizophrenia was twice that of females using diagnostic criteria from DSM-IV.

Lewine et al (1984) found that if clearer criteria was used, fewer females were diagnosed with schizophrenia, suggesting that there may have been an earlier bias towards women.

Women with schizophrenia are more likely to experience affective symptoms, such as depression and anxiety, while men are more likely to experience negative symptoms, such as social withdrawal and apathy.

Women with schizophrenia tend to have better overall functioning and a more favourable prognosis compared to men.

Men with schizophrenia tend to have an earlier age of onset compared to women.

There are clear differences in the age of onset for schizophrenia, with males showing typical symptoms around their late teens and females in the mid to late 20s.

Men with schizophrenia tend to have a higher prevalence of positive symptoms, such as hallucinations and delusions, compared to women.

Men with schizophrenia tend to have a higher prevalence of substance abuse comorbidity compared to women.

Women with schizophrenia tend to have a higher likelihood of having a later age of onset compared to men.

The fact that there are clear differences in the age of onset for schizophrenia, and how the disorder is experienced by men and women suggest that there are problems with the validity of diagnosis.

If women are diagnosed with schizophrenia in their late 50s it is likely to be more chronic than acute. This could be linked to the menopause and may indicate that oestrogen may protect women until the levels drop at menopause.

Kulkarni et al (2001) found that oestradiol (from oestrogen) added into antipsychotic medication helped women more than the antipsychotic alone.

A limitation of schizophrenia diagnosis is the existence of gender bias.

Gender bias is the tendency to ignore or exaggerate symptoms in both men and women, so that the true picture is often distorted.

The misdiagnosis of women with schizophrenia is a gender bias and indicates that women may not be receiving the treatment they need.

The differences between men and women in their experience of schizophrenia have been ignored until recently. It is now clear that women respond better to the medication and have a better outcome because they are typically in relationships and have support at the onset of the disorder.

Angermeyer & Kuhn (1988) reviewed 50 studies of schizophrenia and found women had fewer re-hospitalisations, fewer admissions and shorter hospital stays.

Goldstein (1988) agrees that women have a better prognosis when it comes to schizophrenia than men.

Seeman (1986) reviewed literature from the 1980s concerning gender differences in the social outcome of people with schizophrenia. Seeman concluded that women with schizophrenia live better lives than men with schizophrenia.

Cultural Bias

Cultural bias refers to the tendency to over diagnose members of other ethnic groups as having schizophrenia because their typical behaviours might be seen as abnormal by clinicians.

Problems with cultural bias could be the result of communication issues in how the patient describes their symptoms and lifestyle.

Cultural bias is a limitation of schizophrenia diagnosis.

In the UK people are more likely to be diagnosed with schizophrenia if they are of African-Caribbean descent.

Pinto & Jones (2008) claim that British people of African-Caribbean origin are up to 9 times as likely to receive a diagnosis of schizophrenia as white British people.

Sugarman & Craufurd (1994) found that as successive generations came and settled in the UK from the Caribbean, their risk of being diagnosed with schizophrenia increased.

Cochrane (1983) suggests that mothers in immigrant families to Western Europe may have caught influenza when they became pregnant, as flu is not a common illness in the Caribbean.

Kirkbride et al (2008) contest the idea of bias because of the consistent pattern that has been found over successive generations. They conclude that migratory factors may be important.

Whaley (2004) believes that cultural bias may affect diagnosis as each culture has a different way of expressing their symptoms, which could lead to misinterpretation by clinicians from another culture.

The DSM 5 has a section the acknowledges there has been cultural bias in diagnosis in the past and brings attention to the understanding that different cultures describe their illnesses in different ways.

In many cultures it is normal to claim to have heard voices or seen people who have recently died. Rack (1982) suggests that people showing this behaviour in Western society are more likely to be perceived as psychotic and diagnosed with schizophrenia.

Biological Explanations for Schizophrenia

Genetic

The genetic explanation of schizophrenia suggests that innate, inherited characteristics are responsible for the vulnerability in developing the disorder.

Evidence suggests that the closer the biological relationship, the greater the risk of developing schizophrenia.

Kendler (1985) has shown that first-degree relatives of those with schizophrenia are 18 times more at risk than the general population.

Gottesman (2010) found a strong link for schizophrenia between parents and their offspring. The research shows that offspring are 31.7 times more likely to develop schizophrenia when both parents have been diagnosed with it.

Concordance rates are calculated to show the level of genetic agreement in twin and family studies. The higher the concordance rate, the stronger the genetic link to schizophrenia.

Varma & Sharma (1993) reported a concordance rate of 35% in first degree relatives of people with schizophrenia, compared to only 9% in a control group.

Twin studies and family studies are used to show the likelihood of genetic vulnerability in schizophrenia.

There are two types of twins – identical (monozygotic) and fraternal (dizygotic). MZ twins share 100% of their DNA, whereas DZ twins only share 50% of their DNA.

Gottesman & Shields (1976) document a concordance rate of 58% in MZ twins and 12% in DZ twins, suggesting a strong genetic link for schizophrenia.

Gottesman (1991) found that MZ twins have a 48% risk of getting schizophrenia whereas DZ twins have a 17% risk rate. This is evidence that the higher the degree of genetic relativeness, the higher the risk of getting schizophrenia.

A recent twin study by Hilker et al (2018) found a concordance rate of 33% for identical twins (MZ) and 7% for non-identical twins (DZ).

Some research into the genetic explanation of schizophrenia aims to identify candidate genes. Early research in this area looked for a single genetic variation in the belief that one faulty gene could explain schizophrenia.

Recent research claims that a number of different genes are involved in schizophrenia (polygenic).

Ripke et al (2014) combined previous data from genome wide studies of schizophrenia and found 108 separate genetic variations that were associated with an increased risk of schizophrenia.

As different studies have identified various candidate genes it appears that schizophrenia is aetiologically heterogeneous.

Benzel et al (2007) identified three genes associated with schizophrenia. These genes are COMT, DRD4 and AKT1. All have been associated with excess dopamine in specific D2 receptors, leading to acute episodes of psychosis including positive symptoms such as delusions and hallucinations.

Miyakawa et al (2003) studied DNA from families affected by schizophrenia and found that those with the disorder were more likely to have a defective version of a gene, called PPP3CC which is associated with the production of calcineurin which regulates the immune system.

Sherrington et al (1988) found a gene located on chromosome 5 which has been linked in a small number of extended families where they have schizophrenia.

One genetic explanation for schizophrenia is based on a mutation in parental DNA which can be caused by radiation, poison or viral infection.

Brown et al (2002) found a positive correlation between paternal age and risk of schizophrenia. They suggest a correlation of 0.7% in fathers under 25 and 2% in fathers over 50.

Adoption studies can show the biological influence of schizophrenia. If adopted children of biological parents with schizophrenia, also develop the disorder it provided support for the genetic explanation.

Kety et al (1988) found 14% of biological siblings of adoptees were classified as schizophrenic whereas only 2.7% of adoptive siblings were found to be schizophrenic.

Heston (1966) reported that the offspring of severely schizophrenic mothers who were removed from them during the first 3 days of life still grew up to have schizophrenia at the same rate as those reared by their schizophrenic mothers at home.

Tienari et al (2004) show that biological children of parents with schizophrenia are at heightened risk even if they grow up in an adoptive family.

Evaluation

Family, twin and adoption studies must be considered cautiously because they are based on retrospective information and diagnosis may be biased by knowledge that other family members have also been diagnosed.

It is important to recognise that genetics are only partly implicated in the development of schizophrenia as concordance rates are never 100%. If genes were the only cause identical twins would have 100% concordance rates.

One limitation of the genetic explanation of schizophrenia is the problem of nature versus nurture. It is very difficult to separate out the influence of nature versus nurture. As concordance rates are not 100%, it cannot be assumed that schizophrenia is wholly explained by genes. It could be that the individual has a predisposition to schizophrenia, making them more at risk of developing the disorder but other environmental factors may play a part.

The UK Genome Project (2012) has increased our understanding of the complexity of the genetic influence on behaviour. It is now recognised that genes have multiple functions and it is suggested that schizophrenia is a multi-factorial trait. This means that the research into gene mapping is too simplistic and schizophrenia may not be due to a single gene.

When considering the genetic explanation of schizophrenia it is important to recognise issues of generalisation with twin studies.

The interactionist approach suggests that a combination of nature and nurture may be responsible for the development of disorders. The diathesis stress model claims that people are born with a biological predisposition which is triggered by environmental stressors which results in subsequent disorders.

In family studies members share aspects of their environment as well as many of their genes which makes it difficult to identify the underlying cause of schizophrenia.

One limitation of the genetic explanation for schizophrenia is that there is clear evidence to show that environmental factors also increase the risk of developing the disorder.

Biological risk factors include birth complications as studied by Morgan et al (2017), as well as smoking THC-rich cannabis in teenage years (Di Forti et al, 2015).

Psychological risk factors such as childhood trauma can leave people more vulnerable to adult mental health problems and the development of schizophrenia.

Morkved et al (2017) found 67% of people with schizophrenia and related psychotic disorders reported at least one childhood trauma compared to 38% in a control group.

One practical application of our understanding of the role of genes in schizophrenia is genetic counselling. Potential parents with a history of schizophrenia or relatives with schizophrenia can receive advice for family planning.

Alternative explanations such as those suggested by Frith (1992), claim that schizophrenia is not biological in nature but is the result of specific difficulties in cognitive processing.

Alternative cognitive explanations are also supported by Baron-Cohen (1985) who suggests that schizophrenia is explained by having an impaired 'Theory of Mind', which is the ability to see something from someone else's perspective.

An alternative biological explanation is the dopamine hypothesis which suggests that an overactivity of dopamine in certain brain regions may contribute to the development of schizophrenia.

Dopamine Hypothesis

Dopamine is a neurotransmitter. It is one of the chemicals in the brain which causes neurons to fire. The original dopamine hypothesis stated that schizophrenia suffered from an excessive amount of dopamine. This causes the neurons that use dopamine to fire too often and transmit too many messages.

Dopamine is a neurotransmitter that is active in the limbic system, which is the part of the brain that deals with emotion and drives, including fear and reward.

Dopamine works in the brain to stimulate the action of neurons at the synapse. It is believed that too much dopamine causes the onset of schizophrenia.

Dopamine is synthesised from the amino acid tyrosine. Tyrosine is converted into DOPA by the enzyme tyrosine hydroxylase.

Dopamine is stored in synaptic vesicles until its release across the synapse during transmission.

When dopamine is released during synaptic transmission it acts on five types of postsynaptic receptors (DI-D5).

The original dopamine hypothesis was put forward by Van Rossum (1967) and stated that there was hyperactivity of dopamine transmission, which resulted in symptoms of schizophrenia and drugs that blocked dopamine reduced psychotic symptoms.

In 1967 Jacques Van Rossum proposed that "overstimulation of dopamine receptors could be part of the aetiology" of schizophrenia (Baumeister & Francis, 2002).

In schizophrenia a negative feedback mechanism exists through the presynaptic D2 receptor which regulates the release of dopamine from the presynaptic neuron.

It is suggested that antipsychotic drugs which block dopamine via D2 receptors are effective in reducing the positive symptoms of schizophrenia.

Most antipsychotic drugs will act on the overfiring D2 receptor on the presynaptic neuron, as well as block the receptor sites on the postsynaptic neuron preventing them from absorbing excess dopamine.

Dopamine agonists are drugs that increase the amount of dopamine activity in the brain. Examples include amphetamines and L-DOPA, which is a treatment for Parkinson's disease.

L-DOPA is a drug which converts to dopamine and is often given to patients with Parkinson's disease but has found to create schizophrenia like symptoms. Dopamine itself does not cross the blood-brain barrier and therefore cannot be used to treat Parkinson's instead L-DOPA, which does cross the blood-brain barrier is used.

Amphetamines and similar drugs increase levels of dopamine in the brain and can cause symptoms which resemble those present in psychosis.

Hallucinogenic drugs such as LSD create psychotic type symptoms and work on the dopamine circuit.

Antipsychotic drugs such as clozapine has been found to antagonise dopamine activity and reduce positive psychotic symptoms.

High dopamine activity leads to acute episodes of schizophrenia, and positive symptoms which include; delusions, hallucinations, confused thinking.

Hyperdopaminergia is the term used to describe high levels of dopamine in the brain.

An excess of dopamine receptors in pathways from the sub cortex to Broca's area, which is responsible for speech production, may explain symptoms of schizophrenia such as speech poverty or auditory hallucinations.

Davis et al (1991) proposed the addition of cortical hypodopaminergia, which is abnormally low levels of dopamine in the brain's cortex.

Low levels of dopamine in the prefrontal cortex, which is responsible for thinking, could explain the cognitive deficits in people with schizophrenia.

Post-mortem studies like Owen et al (1987) have found that there are generally a large number of dopamine receptors in people with schizophrenia.

Owen et al (1987) found there was increased dopamine in the caudate nucleus and putamen of patients with schizophrenia.

Falkai et al (1988) found an increased amount of dopamine in the left side of the amygdala in the limbic system of patients with schizophrenia.

Wong et al (1986) used pet scans to show a two-fold increase in the density of postsynaptic dopamine receptors in a group of schizophrenic patients who had never been treated with drugs.

Post-mortem analysis of schizophrenic patients brains have shown increased dopamine receptor density in parts of the limbic system.

The dopamine hypothesis of schizophrenia claims that hyperactivity of dopamine D2 receptor neurotransmission in subcortical and limbic brain regions contributes to positive symptoms of schizophrenia (Toda & Abi-Dargham, 2007).

The dopamine hypothesis of schizophrenia states that there is dysregulation of neurotransmission in brain dopaminergic circuits with excessive dopaminergic signalling in the mesolimbic pathway (causing positive symptoms) and reduced signalling in the mesocortical pathway (resulting in negative symptoms) (Miyamoto et al., 2012).

Howes & Shatalina (2022) suggest that presynaptic dopamine dysfunction results in increased availability and release of dopamine, which has been associated with symptoms of schizophrenia.

Dopaminergic transmission in the prefrontal cortex is mediated by D1 receptors, and D1 dysfunction has been linked to cognitive impairment and negative symptoms of schizophrenia.

The brain's pleasure pathway is known as the mesocorticolimbic pathway. Dopamine is released when we do something that makes us feel good, this flood of dopamine can lead to euphoria. This could link with some of the symptoms of schizophrenia.

The mesolimbic pathway is the site of the rewards pathway which mediates pleasure and reward. Antipsychotics can block D2 receptors in this pathway reducing pleasure effects.

Davis et al (1991) suggested an increase in dopamine in the mesolimbic pathway is associated with positive symptoms of schizophrenia.

Davis et al (1991) suggested an increase in dopamine in the mesocortical pathway is associated with negative symptoms of schizophrenia.

Hyperactivity of dopamine in the mesolimbic pathway is linked to an increase in positive symptoms of schizophrenia.

Decreased dopamine in the mesocortical pathway is said to be responsible for the negative and depressive symptoms of schizophrenia.

It is also reported that upregulation of D2 receptors in the caudate nucleus of patients with schizophrenia directly correlates with poorer performance in cognitive tasks involving corticostriatal pathways (Hirvonen et al., 2004).

Studies have shown an increased density of the dopamine D2 receptor in post-mortem brain tissue of schizophrenia sufferers (Seeman et al., 2000).

A dopamine antagonist (anti-dopaminergic) is a type of drug which blocks dopamine receptors by receptor antagonism. This means that they stop the neurons from releasing excessive dopamine into the system which results in the severe positive symptoms of schizophrenia.

Most antipsychotics are dopamine antagonists, and as such they have found use in treating schizophrenia, bipolar disorder, and stimulant psychosis.

A dopamine agonist is a drug that activates receptors in the brain which produce dopamine, a chemical that helps regulate movement and mood.

An agonist is a substance that acts like another substance and therefore stimulates an action at the synapse.

Clinical trials suggests that some dopamine agonists may produce a mild improvement in negative symptoms of schizophrenia like affective flattening, depressed mood, alogia and avolition.

Lindstroem et al (1999) radioactively labelled a chemical called L-DOPA, which is used by the brain to produce dopamine. They administered the L-DOPA to 10 untreated patients with schizophrenia as well as 10 people in a control group. Using a PET scan they were able to trace what happened to the L-DOPA. The L-DOPA was taken up more quickly in the patients with schizophrenia, suggesting that they were producing more dopamine than the control group.

Javitt (2000) found that glycine, a glutamate receptor agonist, reversed drug induced psychosis in rats and improved the symptoms of people with schizophrenia.

Evaluation

The dopamine hypothesis has generated huge amounts of research and driven drug treatments that have had success in treating people with schizophrenia.

As demonstrated by Lindstroem et al (1999) it is now possible to indirectly measure dopamine levels through scanning techniques, for example using radio labelled L-DOPA to compare healthy people with those with schizophrenia.

Support for the dopamine hypothesis comes from the evidence in drug therapy. Antipsychotic drugs reduce dopamine activity and also reduce the intensity of symptoms (Tauscher et al, 2014).

Curran et al (2004) found that amphetamines increase dopamine activity and worsen symptoms in people with schizophrenia.

Tenn et al (2003) supports the dopamine hypothesis after inducing schizophrenia like symptoms in rats using amphetamines. They then relieved the symptoms using drugs that reduce dopamine activity.

The dopamine hypothesis has been criticised for being too simplistic and inconclusive. Excess dopamine has been associated with the positive symptoms of schizophrenia but does not explain the negative symptoms.

Healy (2000) suggested that drug companies have a personal stake in promoting the dopamine hypothesis because they stand to make huge profits from antipsychotics.

Davis et al (1991) suggested that high levels of dopamine are not found in all individuals with schizophrenia.

Noll (2009) argues around one third of patients do not respond to drugs which block dopamine so other neurotransmitters may be involved.

One criticism of the dopamine hypothesis is that it is a correlational idea. This suggests that caution needs to be taken when establishing cause and effect relationships in schizophrenic patients. Is the raised dopamine levels the cause of the schizophrenia, or is it the raised dopamine level the result of schizophrenia? It is not clear which comes first.

Farde et al (1990) found no difference between schizophrenics' levels of dopamine compared with 'healthy' individuals.

The dopamine hypothesis is biologically deterministic. The reason for this is because if the individual does have excessive amounts of dopamine, then does it mean that they will develop schizophrenia? This suggests that the dopamine hypothesis does not account for freewill.

One limitation of the dopamine hypothesis is evidence for the central role of glutamate. There is some belief that glutamate levels are lowered in people with schizophrenia (Carlsson et al, 2000).

In contrast, McCutcheon et al (2020) suggest that post-mortem and live scanning studies have consistently found raised levels of the neurotransmitter glutamate in several brain regions of people with schizophrenia.

Neural Correlates

The neural correlates explanation suggests that structural differences and brain abnormalities are responsible for schizophrenia.

New advances in brain scanning technology have allowed the brains of individuals with schizophrenia to be investigated and compared to the brains of healthy individuals.

Many of the supporting studies for the neural correlates explanation show a correlation between differences in brain structure and schizophrenia.

Young et al (1990) used MRI scans to investigate the brain structures in schizophrenia patients and found structural differences compared with 'normal' brains.

Warner (1994) suggested that early brain trauma, for example a viral infection during pregnancy, may relate to structural abnormalities in the brains of schizophrenic patients.

Post-mortem research has identified differences in the ventricles of the brains in people with schizophrenia. They show differences in the size of these ventricles with schizophrenics having enlarged ventricles.

Enlarged ventricles suggest damage to the central brain areas and prefrontal cortex. This could account for the negative symptoms of people with schizophrenia.

Andreasen et al (1990) conducted a controlled study using CAT scans. They found a significant enlargement of the ventricles in patients with schizophrenia compared to a control group.

There is some research to show that in schizophrenic patients there is reduced symmetry in the temporal, frontal and occipital lobes. This reduced symmetry is believed to originate during brain lateralisation in foetal development.

Li et al (2010) conducted a meta-analysis and highlighted the bilateral amygdala and right fusiform gyri were less active in schizophrenia patients. This area of the brain is used for processing faces and could explain why many schizophrenic patients suffer with this.

Research has found grey matter differences in schizophrenic brains over time. Grey matter deficiencies can decrease by up to 20% after just five years.

Huffman & Hampson (2012) attempted to explain auditory verbal hallucinations (AVHs). They suggest that excessive activity seen in Wernicke's area could be the reason for the overabundance of potentially conscious language representations, explaining the voices.

Evaluation

Ho et al (2003) have shown by rescanning schizophrenic patients, that brain differences increase overtime as symptoms worsen. This is despite being on medication and helps establish a stronger causal relationship.

Brain scans are used to support the argument for the neural correlates explanation, and they provide reliable data from which conclusions can be drawn.

Not all people with schizophrenia show enlarged ventricles which raises doubt on this explanation.

As this explanation is based on correlational data, it is difficult to make causal conclusions.

Brain scanning studies may lack reliability if the patients investigated are not medication free. Antipsychotics may interfere with the outcome of many brain scans.

Neural correlates does not consider comorbid factors such as stress or addiction. These will affect brain tissue and make cause and effect conclusions more difficult.

The neural correlates explanation must also consider the role of dopamine and it is action in a schizophrenics brain. Dopamine is important in the functioning of several brain systems related to symptoms of schizophrenia.

This explanation is reductionist as the ignores the individual experiences of the patient and just focuses on structural or neural brain differences.

Psychological Explanations of Schizophrenia

Family Dysfunction

Many studies have attempted to link schizophrenia to childhood and adult experiences of living in a dysfunctional family.

Froom-Reichmann (1948) proposed a psychodynamic explanation for schizophrenia based on accounts she heard from her patients. Many reported a particular type of parent which she called a 'schizophrenogenic mother'.

Froom-Reichmann (1948) suggested a schizophrenogenic mother is cold, rejecting and controlling. These mothers create a family climate characterised by tension and secrecy which leads to distrust and can develop into paranoid delusions and ultimately schizophrenia.

Bateson et al (1972) agreed that family climate is important in the development of schizophrenia but emphasised the role of communication style within the family.

The family dysfunction explanation is concerned with sources of stress, which can bring on problems with people who have a genetic predisposition to schizophrenia. This is known as the diathesis-stress model.

Sometimes the source of stress is within the family but it is difficult to identify the exact underlying cause of this stress.

Some psychologists suggest that family relationships with abnormal communication styles may play a part in schizophrenia by creating highly stressful environments.

Expressed emotion (EE) is the level of emotion, in particular negative emotion which is expressed towards a person with schizophrenia by their carers who are often family members.

Linszen et al (1997) suggested that expressed emotion (EE) is a communication style the involves criticism, hostility and emotional over involvement. They suggest a patient returning to a family with high-EE is about four times more likely to relapse than a patient whose family is low in EE.

McGlashan (1994) proposed that expressed emotion (EE) which is overly critical or highly emotive may be involved in the cause of schizophrenia.

Research into expressed emotion (EE) reveals that family dynamics are an important predictor of relapse of positive symptoms with schizophrenia.

Brown et al (1972) and Vaughn et al (1976) both established the detrimental effects of ineffective medication and high face-to-face contact on relapse rates of patients living in high-EE families.

Kavanagh (1992) conducted a meta-analysis and reported a 48% relapse rate in high-EE environments, versus 21% in low-EE environments.

It is not known as whether or not high EE exists as a true cause of relapse in schizophrenia or whether it reflects the stress within the family who are living with a patient with a chronic mental illness.

Smith et al (1993) found that high-EE relatives report higher levels of stress and have more difficulty coping then low-EE relatives.

Eva et al (1995) found that relatives who score highly on EE assessments tend to listen effectively and talk more in family interviews.

Morkved et al (2017) found that adults with schizophrenia reported at least one childhood trauma, mostly abuse. This strongly suggests but family dysfunction makes people more vulnerable to schizophrenia.

Read et al (2005) reported that 69% of women and 59% of men with schizophrenia had a history of physical and/or sexual abuse.

Patino et al (2005) conducted a cross-sectional study with 3426 children and adolescents looking at family dysfunction and migration history. They found that both factors were associated with a four times risk of developing psychotic symptoms.

Stress releases cortisol, which is a hormone that causes multiple effects within the body including its role in the fight or flight response. In the long-term these effects are harmful physically and mentally.

Tienari et al (2004) found that 37% of high-genetic risk adoptees living in a dysfunctional family environment developed schizophrenia compared to only 5% of those in healthy family environments.

Bateson (1972) devised the double-bind hypothesis, which claims but if there is a contradictory position there will be negative consequences. Bateson hypothesised that growing up in a high negative-emotion environment could lead to psychosis.

Kavanagh (1992) supports Bateson's double-bind hypothesis, as it was found that schizophrenic patients returning home to high-emotion families had higher relapse rates than those returning to low-emotion families.

Evaluation

The theory that high levels of EE cause schizophrenic relapse led to an effective therapy where high-EE relatives are shown how to reduce their negatively expressed emotion.

Terkelsen et al (1983) argues that blaming the family creates an atmosphere of adversity and mistrust, with poorer outcomes for the patient.

Research linking family dysfunction to schizophrenia is highly socially sensitive because it can lead to parent blaming, in particular mothers.

Mari & Streiner (1994) found that family therapy significantly reduced expressed emotion and hospitalisation and also increased medication compliance.

Family therapy has been shown to be effective in achieving a more positive outcome in patients with schizophrenia, supporting the family dysfunction explanation.

Indicators of family dysfunction include insecure attachment and exposure to childhood trauma, especially abuse. Read et al (2005) offer support following a review on adults with schizophrenia who were disproportionately likely to have insecure attachment types.

Living with a family member who suffers from schizophrenia is very stressful and can create high emotional states and conflicts. Therefore it is difficult to establish which comes first, the dysfunctional family or the dysfunctional child.

This explanation fails to consider that not all children living in dysfunctional families go on to develop schizophrenia.

There is limited evidence supporting the idea of the schizophrenogenic mother and the double-bind hypothesis.

Cognitive Explanations of Schizophrenia

Cognitive psychologists study mental processes such as memory, emotion, perception, attention and language. They describe how disruption of these processes may lead to some of the symptoms of schizophrenia.

O'Farrell (2000) suggests that cognitive impairments accompany schizophrenia in 75% of cases.

Cognitive impairments are often visible before the onset of schizophrenia, suggesting they are not a result of the disorder.

Cognitive explanations of schizophrenia focus on faulty thinking and distorted beliefs.

Cognitive processes include thinking, memory, attention and perception.

People with schizophrenia are believed to have faulty mental processes and cognitive deficits.

Hallucinations and delusions are problems with information processing.

Many research studies have investigated the idea of cognitive deficits underlying the difficulties faced by schizophrenic patients, with regard to their memory, attention and executive functioning.

Frith (1992) explained the symptoms of schizophrenia in terms of difficulty with information processing, specifically two difficulties; metarepresentation (ability to reflect on our thoughts, behaviour and experiences) and central control (ability to suppress our automatic responses to stimuli while we perform actions that reflect our wishes or intentions).

Frith (1992) believed that positive symptoms (e.g. delusions or hallucinations) can be explained by metarepresentations, whereas negative symptoms can be explained in terms of central control.

Bentall et al (1991) found that schizophrenics cannot distinguish between words they had heard and words they had produced. This suggests that there are problems with metarepresentation.

Stirling et al (2006) compared performance on a range of cognitive tasks in 30 people with schizophrenia and 30 people in a control group. Those with schizophrenia were found to struggle more suggesting that cognitive processes are impaired.

According to Elvevag & Goldberg (2000) schizophrenia is better explained by cognitive deficits rather than symptoms. Memory and attention are the main core deficits, which provides support for this idea.

Takahashi et al (2013) got people with and without schizophrenia to listen to tones and to try to tell them apart. They found that schizophrenic patients struggled with this task.

Beck & Rector (2005) proposed a cognitive model in which there is an interaction of environmental, neurobiological, behavioural and cognitive factors that lead to cognitive deficits.

Bowie & Harvey (2006) found that cognitive impairments were a core feature of schizophrenia, mainly affecting attention, working memory, verbal learning an executive function.

Dysfunctional thought processing is an important part of the cognitive explanation for schizophrenia.

Schizophrenia is characterised by disruption to normal thought processing and this can be seen in many of the symptoms.

According to Simon et al (2015) reduced thought processing in the ventral striatum is associated with negative symptoms of schizophrenia, while reduced processing in the temporal and cingulate gyri is associated with hallucinations.

Simon et al (2015) suggests that lower than usual levels of information processing in schizophrenic patients indicate that cognition is likely to be impaired.

Healthy individuals are able to use metacognition to guide their thinking and problem solving. This is their awareness of how they are thinking and feeling, as well as their knowledge of when they have made a mistake.

Patients with schizophrenia lose their awareness of metacognition which results in cognitive dysfunction. This often presents itself in positive symptoms such as hallucinations.

Joshua et al (2009) used the Hayling sentence completion task to compare people with schizophrenia and bipolar disorder against healthy individuals. They found that people with schizophrenia were slower to respond and slower to suppress inappropriate responses.

Joshua et al (2009) found that performance of people with schizophrenia is associated with higher ratings of cognitive disorganisation.

Evans et al (1997) used the Behavioural Assessment of the Dysexecutive Syndrome (BADS) test which is a series of tasks the assess the skills and demands of everyday life. They found that

individuals with schizophrenia had impaired executive functioning, as well as problems with their memory.

Brune et al (2011) found evidence in a meta-analysis over 20 years of research this supported the idea the schizophrenia impaired metacognition, leaving patients with impairments in social functioning, self-reflection and empathy.

Evaluation

Cognitive theories do not explain the cause of schizophrenia or what has led to the deficits.

If the problems with metacognition can be established patients can be treated and helped to overcome these deficits.

Metacognition is a hypothetical concept and can only be investigated by observing behaviour on various tests or assessments. This raises issues about the reliability and validity of this idea.

If cognitive therapies are used alongside biological interventions it can lead to successful outcomes for patients with schizophrenia.

Cognitive explanations focus on individual experiences and patients feelings and beliefs, rather than relying on the interpretations of others.

Cognitive explanations are reductionist as they ignore any biological causes of schizophrenia.

Cognitive explanations of schizophrenia on their own only provide partial explanations for the disorder.

Drug Therapy: Typical and Atypical Antipsychotics

People with schizophrenia are usually offered antipsychotic medication to help reduce their positive symptoms.

Antipsychotic drugs may be required in the short-term or the long-term. Some patients may require antipsychotic medication for life.

Many antipsychotic drugs are used as the first form of treatment for patients with schizophrenia.

Antipsychotic drugs fall into two types; typical drugs (first generation) which were developed in the 1950s and atypical drugs (second generation) which are newer and developed around the 1990s.

Kahn et al (2008) found that antipsychotics are generally effective for at least one year, but second- generation drugs were no more effective than first-generation ones.

All patients are in danger of relapsing but without medication the relapses are more common and more severe which suggests the drugs are effective.

A meta-analysis by Crossley et al (2010) suggested that atypical antipsychotics are no more effective but do have less side effects.

Typical antipsychotics, such as chlorpromazine, primarily target positive symptoms of schizophrenia, such as hallucinations and delusions.

Atypical antipsychotics, such as risperidone, are newer medications that also target negative symptoms of schizophrenia, such as social withdrawal and lack of motivation.

Typical antipsychotics can be taken as tablets, syrup or by injection.

Typical antipsychotics stop dopamine production by blocking the D2 receptors in synapses that absorb dopamine, in the mesolimbic pathway thus reducing positive symptoms, such as auditory hallucinations.

Typical antipsychotics tended to block all types of dopamine activity, (in other parts of the brain as well) and this caused side effects and may have been harmful.

Typical antipsychotic drugs work by blocking D2 receptors in parts of the limbic system in the brain.

Many typical antipsychotics are known as dopamine antagonists. Antagonists are chemicals which reduce the action of neurotransmitter at the synapse.

An antagonist is a chemical substance that binds to and blocks the activation of certain receptors on cells, preventing a biological response.

Dopamine antagonists work by blocking dopamine receptors in the synapses of the brain, reducing the action of dopamine.

Typical antipsychotic drugs block dopamine receptors and reduce levels of excitation via dopamine at the synapse.

Examples of typical antipsychotic drugs include Chlorpromazine, Fluphenazine and Haloperidol.

Typical antipsychotics work to reduce the levels of hallucinations and delusions but tend not to be effective on the negative symptoms of schizophrenia.

Many typical antipsychotics also act as a sedative and can help calm patients and reduce their anxiety.

Atypical antipsychotics were developed to improve upon the effectiveness of drug therapy in suppressing the symptoms of psychosis but also to minimise the side effects.

Atypical antipsychotic drugs are targeted and act on specific dopamine receptor sites, usually D2 receptors.

Atypical antipsychotics work on both positive and negative symptoms of schizophrenia.

Examples of atypical antipsychotic drugs are Clozapine, Quetiapine and Olanzapine.

Clozapine binds to dopamine receptors but also acts on serotonin and glutamate receptors. It is believed that this action improves mood and helps to reduce anxiety and depression in patients.

Meltzer (2012) concluded that clozapine is more effective than typical antipsychotics and other atypical antipsychotics.

Risperidone is a more recently developed atypical antipsychotic and can be taken as a tablet, syrup or injection lasting up to two weeks.

Risperidone is believed to bind to dopamine and serotonin receptors, and results in fewer side effects than other antipsychotics.

Chlorpromazine is a typical antipsychotic which is often used to help calm individuals down, as it has a sedative effect.

Thornley et al (2003) reviewed studies comparing the effects of chlorpromazine with a placebo. Data from 13 trials with a total of 1121 participants showed that chlorpromazine was associated with better overall functioning and reduced symptom severity.

Comer et al (2001) claim that typical antipsychotic drugs reduce schizophrenic symptoms in the majority of patients but are less successful at treating negative symptoms such as flattened affect.

Typical antipsychotics have more side effects such as dry mouth, blocked nose, urinary problems and in some case tardive dyskinesia (facial twitching).

Atypical antipsychotics have less side effects but patients can still be at risk of weight loss and sleep disruption.

Smith et al (2010) suggest that antipsychotic drugs can reduce the positive symptoms of psychosis in 8-15 days.

Drug therapy is individualised and is based on benefits, risks and costs for each specific patient.

Both typical and atypical antipsychotics have similar dropout rates and symptom relapse rates when used at low to moderate dosages.

Clozapine is an effective treatment for patients who respond poorly to other drugs, but there is a higher risk of a serious side effect called agranulocytosis (reduced white blood cells).

Evaluation

Davis et al (1989) found that antipsychotics were more effective than placebos in a meta-analysis of over 100 studies. They found that 75% of patients improved against 25% in the placebo group.

Marder (1996) found that clozapine was as effective as typical antipsychotics for positive symptoms, helping 30 to 61% of patients who were resistant to typical antipsychotics.

Drug therapy is usually faster acting and more cost effective than other psychological therapies.

Lieberman (2005) found that 74% of 1432 individuals discontinued treatment within 18 months because of side effects. Typical drugs caused muscular disorders whereas atypical drugs caused more weight gain.

Some antipsychotic drugs have a wide variety of actions on the brain and behaviour than they are intended to treat. This can lead to a range of side effects.

Unwanted side effects from antipsychotic medication can be very serious and can cause distress and more serious health complications.

Research into the effectiveness of drug therapy is very socially sensitive. Some people argue that these drugs are used as a form of social control, making patients easier to manage.

Moncrieff (2013) suggest that antipsychotics are often used in hospitals to calm patients with schizophrenia and make them easier for staff to work with, rather than for the benefits of the patients themselves.

Ethical issues arise when people are forced to take drugs. This may be the case in patients who are hospitalised with schizophrenia.

Drug treatments may control the symptoms of schizophrenia but do not cure the underlying problem which may be causing the disorder.

Healy (2012) suggested that most studies investigating the effectiveness of drug therapy are often only focused on the short-term effects.

Long-term use of antipsychotic drugs can result in serious side effects such as tardive dyskinesia, which is caused by dopamine supersensitivity and causes involuntary facial movements such as grimacing, blinking and lip smacking.

The most serious side effect of antipsychotic drugs (mainly typical antipsychotics) is neuroleptic malignant syndrome (NMS). NMS results in high temperature, delirium and can be fatal. This means the antipsychotics can do harm as well as good.

Compliance is a major problem with drug therapy for schizophrenia. It is estimated that 40-50% of people with the disorder have problems sticking to their prescribed medication.

Smith et al (2010) suggest that antipsychotic drugs often fail to significantly improve the negative symptoms and cognitive dysfunction associated with schizophrenia.

Cognitive Behaviour Therapy for Schizophrenia

Cognitive Behavioural Therapy (CBT) is commonly used to treat people with schizophrenia. It usually takes place over a period of 5-20 sessions either in groups or on an individual basis.

CBT aims to deal with both thoughts (cognitions) and behaviour.

Cognitive Behavioural Therapy (CBT) involves both cognitive and behavioural elements.

The cognitive element of CBT aims to identify irrational and negative thoughts and replace these negative thoughts with more positive ones.

The behavioural element of CBT encourages patients to test their beliefs through behavioural experiments and homework.

As schizophrenia is associated with cognitive problems such as attention, memory and perception, CBT can be a useful therapy.

With CBT for schizophrenia it is believed that patients can be helped to see logical ways for dealing with their hallucinations and delusions.

CBT starts with an initial assessment, in which the patient and therapist identify the patient's problems. Thereafter, the patient and therapist agree on a set of goals, and plan of action to achieve these goals.

If CBT is using Beck's cognitive triad, the therapist will help the patient to identify negative thoughts in relation to themselves, their world and their future.

In CBT, the patient and therapist will work together to challenge the irrational thoughts, by discussing evidence for and against them.

In CBT, the patient will be encouraged to assess the validity of their negative thoughts and may be set homework, to challenge and test their negative thoughts.

The ABC model of treatment was first created by Ellis (1957) for people suffering from depression. However it has since being applied to people with schizophrenia.

When the ABC model is used in CBT there are six main stages; assessment, engagement, ABC model, goal setting, normalising and critical collaborative analysis.

In Ellis's CBT, during the assessment patients express their thoughts and feelings about their experience with schizophrenia whilst the therapist listens actively.

During the engagement stage of CBT the use of Socratic questioning and empathy helps build a rapport between the patient and the therapist.

Socratic questioning is a method of inquiry that seeks to explore complex ideas, concepts, and beliefs by asking questions that challenge assumptions, clarify meaning, and reveal underlying principles.

In Ellis's approach to CBT therapists will work though the ABC model and include D (dispute) and E (effect). Like Beck, the main idea is to challenge irrational thoughts, however, with Ellis's theory this is achieved through 'dispute' (argument).

In CBT there are different types of dispute which can be used. Logical dispute is where the therapist questions the logic of a person's thoughts and empirical dispute where the therapists seeks evidence for a person's thoughts.

In CBT, an example of logical dispute is where the therapist may ask the patient, 'does the way you think about that situation make any sense?'.

In CBT, an example of empirical dispute is where the therapist may ask the patient, 'where is the evidence that your beliefs are true?'.

During the goal setting stage of CBT patients work with the therapist to set realistic goals which are measurable and achievable.

In CBT, the therapist will help the patient normalise their psychotic experiences. This is done through discussion and dispute.

In CBT once trust is formed, gentle questioning is used to help the patient appreciate their maladaptive beliefs. This is the critical collaborative analysis stage.

In CBT following a session, the therapist may set their patient homework to do at home, with the idea that the patient is encouraged to identify their own irrational beliefs and find alternative ways to stop or change them.

Sensky et al (2000) compared CBT with nonspecific befriending interventions. They used a group of 97 patients in the UK who were nonresponsive to medication. After an average of 19 sessions patient showed improvements in both positive and negative symptoms.

Sensky et al (2000) found that CBT has significant and lasting benefits even after nine months, for both positive and negative symptoms of schizophrenia.

Coping strategy enhancement (CSE) is a technique which aims to help schizophrenics find the best strategies to deal with their symptoms.

In a recent form of CBT patients create an avatar to represent the voices they hear in their mind. In this avatar therapy patients practise strategies for challenging and overcoming the threats made by negative voices.

Bradshaw (1998) followed a single patient over four years. Over the period of treatment her symptoms improved dramatically, and it was suggested that CBT contributed to this.

Turkington et al (2004) demonstrated how CBT can be used to challenge positive symptoms of schizophrenia such as paranoid delusions.

Jauhar et al (2014) reviewed 34 studies of using CBT with schizophrenia, concluding that there is clear evidence for small but significant effects on both positive and negative symptoms.

Pontillo et al (2016) found reductions in frequency and severity of auditory hallucinations when using CBT.

Clinical evidence from NICE (2019) which is the National Institute for Health and Care Excellence, recommends CBT as a beneficial treatment for schizophrenia.

Evaluation

CBT works in combination with drug therapy and is more effective than either treatment on its own. NICE guidelines recommend the combination of CBT and drug therapy for patients with schizophrenia.

CBT requires a skilled therapist who is trained to work with patients with psychosis.

CBT is a very time-consuming process and several sessions are required before a patient sees any improvement in their symptoms.

CBT is not suitable for all patients. People with extreme agitation or anxiety may not be able to rationalise or empathise with a therapist.

CBT is not suitable for acutely psychotic patients who are too ill to engage with the demanding therapy.

Trower et al (2004) suggest that CBT provides strategies for dealing with schizophrenia rather than treating the symptoms.

CBT may improve the quality of life for people with schizophrenia but may not actually cure them.

CBT as a treatment fails to consider the biological nature of schizophrenia. It does not reduce excessive dopamine levels.

Thomas (2015) points out that different studies have involved the use of different CBT techniques on people with varying degrees of severity for schizophrenia. This makes it more difficult to say CBT will be successful for all patients.

Family Therapy for Schizophrenia

The importance of family, particularly in schizophrenic relapse has led to the development of numerous family interventions.

Family interventions use a variety of therapeutic techniques to educate and inform the patient and their family and develop strategies for managing the illness.

Family therapy helps to overcome the issues associated with dysfunctional families and shows them more positive ways of dealing with the stress of a life with a family member with schizophrenia.

Family therapy takes place with families as well as the identified patient. The therapy aims to improve the quality of communication and interaction between family members.

Pharoah et al (2010) identified a range of strategies that family therapists use to try and improve the functioning of the family that has a member with schizophrenia.

Family therapy aims to reduce levels of expressed emotion (EE), which is often felt through negative emotions such as anger and guilt.

Returning home to high expressed emotion (EE) environments causes higher relapse rates in patients with schizophrenia.

Family therapy can help reduce stress which in turn lowers the likelihood of patients having a relapse.

The goals of family therapy are to give the family all the information they need to understand the illness.

Family therapy encourages discussion about what it is like to live with a family member with schizophrenia. This discussion is carried out to allow family members an opportunity to share their support.

Family therapy can help the family understand that they are not to blame for the illness and give them ways to help the individual with the disorder move forward.

Family therapy allows all members to voice their opinions and provides a safe space to discuss their feelings about living with a family member with schizophrenia.

Family therapy takes place over many sessions and is usually carried out in the patient's home. Group sessions can also take place where several families share their experiences.

In family therapy the therapist encourages the family members to form a therapeutic alliance whereby they all agree on the aims of the therapy.

In family therapy the therapist tries to improve the family's beliefs, attitudes and behaviours towards schizophrenia.

Another aim of family therapy is to ensure that family members achieve a balance between caring for the individual with schizophrenia and maintaining their own lives.

Burbach (2018) proposed a model for working with families dealing with schizophrenia. This model of practise enables the family to move through a series of phases to offer emotional and practical support to their family member.

Pilling et al (2002) compared family therapy to other types of treatment in a meta-analysis of 18 different studies. They found patients who experienced family therapy had fewer relapses and less hospital admissions.

A meta-analysis by Bird et al (2010) showed that family interventions in early psychosis significantly reduced relapse and re-admission rates.

A review article by Caqueo-Urizar et al (2015) highlighted the burden placed on the family when caring for a patient with schizophrenia. Without support they frequently experience stress and isolation but will have more positive outcomes with family intervention.

One form of family therapy, known as the Optimal Treatment Project, combines training for patients and their families in coping with stress, social skills and crisis management.

The Optimal Treatment Project (family therapy) has been successfully used in more than 20 countries worldwide.

Family therapy has been found to have a positive influence on many outcomes including drug compliance.

The National Institute for Health and Care Excellence (NICE) suggest that family intervention should be used for patients who have persistent and relapsing conditions and who are supported by a caregiver.

Falloon et al (1985) looked at relapse rates for patients receiving family therapy where 11% were re-hospitalised within one year compared with those receiving only individual therapy where 50% were re-hospitalised within a year.

Pharoah et al (2003) found that relapse rates and hospitalisations were significantly reduced following family interventions.

Evaluation

During family therapy individual family members can provide valuable information about how the patient is dealing with the disorder on a day-to-day basis.

Younger patients with schizophrenia will still be living at home with their parents as caregivers and will benefit from the support of family therapy.

McFarlane (2016) conducted a review of studies and concluded that family therapy was one of the most consistently effective treatments available for schizophrenia.

McFarlane (2016) found that relapse rates were reduced by 50-60% in patients with schizophrenia following family therapy.

Lobban & Barrowclough (2016) concluded family therapy was just as important for the identified patient as it is for the families themselves.

If family therapy is conducted in the homes of patients, it is likely to have less of an economic impact on the institutionalised healthcare system.

Family therapy does not work in all cases as each family member has to be prepared to engage with the therapy.

As with CBT, family therapy is usually used in conjunction with medication.

Patients with schizophrenia come from a wide variety of family backgrounds, so family intervention may not always be appropriate.

Family therapy is time consuming and can be a costly process which may explain why it is not as successful as drug therapy.

Token Economies as used in the Management of Schizophrenia

Token economy programmes (TEPs) are based on the principle of operant conditioning which suggests that behaviour can be shaped by positive and negative reinforcers.

Token economy programmes are behaviourist programmes that work best with institutionalised patients in long-term psychiatric care.

Token economy programmes proposed by behaviourist psychologists assume that dysfunctional behaviour is learned and can be unlearned.

Token economies are reward systems used to manage the behaviour of people with schizophrenia, in particular those who have developed patterns of maladaptive behaviour.

Token economy is a system of behaviour management based on the systematic reinforcement of target behaviour. The reinforces are rewards which can be exchanged for other reinforces.

Primary reinforcers are things that motivate behaviour because they satisfy an individual's basic survival needs. Primary reinforcers are distinguished from other types of reinforcers because they have innate biological value.

Secondary reinforcers are stimuli, objects, or events that become reinforcing based on their association with a primary reinforcer.

In token economy programmes (TEPs) secondary reinforces such as tokens are exchanged for primary reinforces, which are rewards that people want to receive.

Token economy programmes are used in hospitals to help patients with schizophrenia gain more control over their daily lives and increase positive behaviours.

Token economy programmes provide structured point systems which emphasise and reinforce positive behaviour.

In a token economy programme patients would be rewarded with tokens or points which can later be exchanged for genuine rewards.

Token economies usually involve schedules of reinforcement and are used to modify the behaviour of those suffering from schizophrenia.

McMonagle & Sultana (2000) conducted a meta-analysis of token economy programmes and found some evidence for progress with the negative symptoms of schizophrenia.

Dickerson et al (2005) offer support for token economies in helping patients to become more independent when they are used with other treatments such as drug therapy.

Paul & Lentz (1977) compared the effectiveness of token economy against other forms of therapy. They studied 84 chronic and hospitalised psychiatric patients all receiving drug treatment and followed them for a period a 4.5 years.

Paul & Lentz (1977) found that patients receiving token economy were more likely to reduce their antipsychotic drugs and more than 10% were able to leave the hospital.

Glowacki et al (2016) reviewed seven high-quality studies examining the effectiveness of token economies for schizophrenia. All the studies showed a reduction in negative symptoms and a decline in unwanted behaviours.

Evaluation

Token economy programmes provide a positive means for motivating and at times controlling behaviour.

Token economy programmes do not require specialist training as all staff can award tokens to patients in hospital.

Token economy programmes can provide psychiatrists with a way of monitoring the progress of the patient. The patient may also get some indirect intrinsic satisfaction with their progress.

Behavioural models such as token economy can help people change their behaviour.

Some psychologists argue that people with schizophrenia may only get the chance to live outside hospital if their personal care and social interaction is improved. In some cases this is achieved using token economy.

Token economy programmes can be very staff intensive and demand resources and time to fully establish their use.

One problem with token economy programmes is that patients may become dependent upon receiving points or tokens to perform daily tasks. This will not change their behaviour in the long-term.

Token economy programmes require consistency and cooperation at all times. This may be difficult to achieve within the hospital setting.

Token economy programmes have issues with generalisation as they do not work effectively outside of the hospital setting.

One problem with token economies is they are very difficult to continue once a person is outside the hospital setting. This is because target behaviours cannot be monitored closely and tokens cannot be administered immediately.

Token economy programmes work more successfully when used in combination with other treatment programmes.

There are issues of social sensitivity when carrying out token economy programmes with patients suffering from schizophrenia. As vulnerable individuals there is an element of free will which is removed.

There are ethical issues in token economies raised by restricting rewards to vulnerable people with mental disorders.

Token economy programmes can also be seen as a form of social control. The power to control and withhold rewards lies with those who are administering the points or tokens.

Token economies involving reinforces that withhold a basic human right such as food, clothing or privacy are unethical and have been ruled illegal in the USA.

One criticism of token economies is that the principle of conditioning does not cure schizophrenia, it only helps manage behaviour.

Alternative therapies such as art therapy do not raise ethical issues and according to Chiang et al (2019) are just as effective as token economies.

Interactionist Approach

The interactionist approach combines both biological and psychological explanations for schizophrenia.

The interactionist approach is sometimes called the biosocial approach.

The interactionist approach acknowledges that there are biological, psychological and social factors in the development of schizophrenia.

Biological factors include genetic vulnerability, neurochemical and neurological abnormality.

Psychological factors include stress from external sources such as life events or daily hassles.

Social factors include family dysfunction and poor-quality interactions within the family.

Brown & Birley (1968) investigated the life events of schizophrenic patients in the 12 weeks prior to their psychotic episode. They found that 50% of patients experienced a stressful event in the three weeks prior to the episode. This suggests that the stressful experience may have triggered the schizophrenic episode.

Hirsch et al (1996) conducted a longitudinal study looking at the life events experienced by 71 schizophrenic patients over a period of four years. They found that the impact of stressful life events in the 12 months prior to a schizophrenic episode had a significant effect.

Many studies have suggested that stress in the environment has impacted on the development of schizophrenic symptoms.

An idea to explain the combination of biology and environment is called the diathesis-stress model.

The diathesis-stress model suggests that behaviour is predetermined by biological factors and triggered by environmental influences.

The diathesis-stress model suggests that both a biological vulnerability to schizophrenia and external stress triggers are necessary to develop the disorder.

In the diathesis-stress model one or more underlying factors may make a person more vulnerable to developing schizophrenia, but the onset of the condition is triggered by stress.

Meehl (1962) suggested that in the original diathesis-stress model the vulnerability to schizophrenia was entirely genetic, the result of a single ‘schizogene’.

According to Meehl (1962) if a person does not have the schizogene then no amount of stress would lead to schizophrenia.

Ripke et al (2014) argue that there is no single gene for schizophrenia but that many genes appear to increase genetic vulnerability.

On their own, biological risk factors do not trigger a psychotic episode, but with an environmental stressor such as family dysfunction or substance misuse, schizophrenia can be triggered.

Ingram & Luxton (2005) have a modern view of the diathesis-stress model which includes a range of factors beyond the genetic such as psychological trauma.

Barlow & Durand (2009) offer support for the diathesis-stress model, in that some people have a genetic vulnerability which can be triggered by the stress of a dysfunctional family. This interaction leads to the development of schizophrenia.

Read et al (2001) proposed a neurodevelopmental model which claims that early trauma physically alters the developing brain. This can lead to an increased vulnerability for schizophrenia.

The diathesis-stress model can be used to explain schizophrenia as it acknowledges an interaction between biology and the environment.

The interactionist approach has been used to explain schizophrenia but can also be used when considering the treatment of schizophrenia.

Adopting an interactionist approach for the treatment of schizophrenia combines biological intervention such as antipsychotics, with psychological therapy such as CBT.

Hogarty et al (1986) looked at relapse rates of schizophrenia patients and found a rate of 41% with drug therapy alone, but with family therapy it improved to 19%.

Sudak (2011) found that compliance with antipsychotic medication improved when patients were also given CBT. This highlights the benefit of an interactionist approach.

Evaluation

Many research studies support the interaction of both antipsychotics and CBT for the treatment of schizophrenia.

One strength of the interactionist approach is in the combination of biological and psychological treatments in the real-world application for schizophrenia.

Studies show that combining treatments enhances their effectiveness in patients with schizophrenia.

Sensky et al (2000) found that CBT has significant and lasting benefits even after nine months, for both positive and negative symptoms of schizophrenia. They found that CBT worked best when combined with antipsychotic medication.

Turkington et al (2006) argue that it is perfectly possible to believe in biological causes of schizophrenia and still practise CBT to relieve psychological symptoms.

In the UK it is standard practise to treat people with schizophrenia using a combination of antipsychotic drugs and CBT.

In the USA it is less common to use an interactionist approach for treating schizophrenia. Medication is usually prescribed without accompanying psychological treatment.

Tarrier et al (2004) randomly allocated 315 participants to either a medication and CBT group, a medication and counselling group, or a control group (medication only). They found that participants in the two combination groups showed lower symptoms than the control group.

Tarrier et al (2004) suggest that there is a clear practical advantage to adopting an interactionist approach to schizophrenia in terms of more positive treatment outcomes.

Tienari et al (2004) investigated the impact of both genetic vulnerability and a psychological trigger (dysfunctional parenting). They followed 19,000 children in Finland whose biological mothers had been diagnosed with schizophrenia. In adulthood this high genetic risk group were found to have a stronger association with the development of schizophrenia.

One limitation of the diathesis-stress model is over simplicity. There are so many environmental factors associated with the onset of schizophrenia as well as all the multiple genes which have been identified.

Jarvis & Okami (2019) suggest caution when considering the interactionist approach and highlight an error called the treatment-causation fallacy. This is the mistake that just because a treatment is successful does not mean it justifies a particular explanation.

By taking an interactionist approach, we can avoid issues such as the treatment causation fallacy. This is the assumption, that when using one treatment such as drugs, if symptoms are reduced, it is assumed that the cause is biological. This may not be the case.