11.2 SCHIZOPHRENIA

TYPES, SYMPTOMS AND CHARACTERISTICS

ASK YOURSELF
How would you diagnose schizophrenia in someone? What behaviours and characteristics would you look for?

Schizophrenia was first called "dementia praecox" (premature dementia) as it affects people's thoughts, emotions and behaviours. Below are the types of schizophrenia currently recognised and the characteristics that need to be shown for people to be diagnosed.

Types

Schizophrenia is an umbrella term used to outline a range of different psychotic disorders that affect thoughts, emotions and behaviours. These are the main diagnostic types:

- Simple – when people gradually withdraw themselves from reality.
- Paranoid – when people have delusional thoughts and hallucinations and may experience delusions of grandeur.
- Catatonic – when people have motor activity disturbances that may involve them sitting or standing in the same position for hours.
- Disorganised – when people have disorganised behaviour, thoughts and speech patterns. They may also experience auditory hallucinations.
- Undifferentiated – when an individual does not fit into one of the types above but is still experiencing affected thoughts and behaviours.

Characteristics

For a diagnosis of schizophrenia, the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM*) outlines the following:

- The person shows two of the following for at least one month: delusions, hallucinations, disorganised speech, disorganised or catatonic behaviour, flattening of emotions; or continual voices in the head giving a running commentary of what is happening.
- The person must show social and/or occupational functioning that has declined.
- There must be no evidence that medical factors are causing the behaviours.

Symptoms can be split into positive and negative:

- Positive refers to the *addition* of certain behaviours. For example, hallucinations, delusions of grandeur or control and insertion of thoughts are all positive.
- Negative refers to the *removal* of certain behaviours. For example, poverty of speech, withdrawal from society and flattening of mood are all negative.

Case studies

CHALLENGE YOURSELF
Find two real-life case studies of people being either diagnosed with schizophrenia or living with schizophrenia.

EXPLANATIONS OF SCHIZOPHRENIA

Below we will examine a range of potential causes of schizophrenia, giving evidence.

Genetic

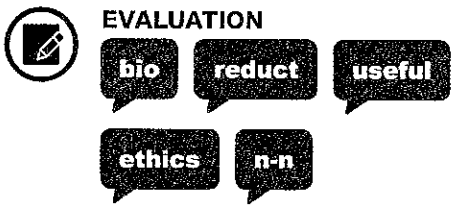
This idea states that there is a link between schizophrenia and inherited genetic material. If this is the case then the closer our genetic link is to someone diagnosed with schizophrenia, the more likely we are to be diagnosed ourselves. Gottesman (1991) examined over 40 studies conducted in Europe to pool data on research focused on genetics and schizophrenia. The results are shown in Table 11.2.1.

Relative	Percentage risk
Nephews or nieces	4
Children	13
Non-identical (dyzgotic) twins	17
Identical (monozygotic) twins	48
In contrast: general population	1

Table 11.2.1 Link between genetics and schizophrenia (Gottesman, 1991)

It seems that the data support the idea of schizophrenia being inherited because the more genetic material people shared, the more likely they were to be diagnosed too. However, the highest risk was 48 per cent (not 100 per cent, indicating a wholly genetic trait) so it looks as if people may be born with a predisposition to develop schizophrenia and it is some environmental influence that ultimately causes it.

Yang *et al* (2013) analysed ten "candidate" genes that could be responsible for schizophrenia in a sample of 1 512 participants. While there was no single gene that appeared to be associated with schizophrenia, the DAO gene was strongly associated with schizophrenia in comparison to all of the other candidate genes. In addition, Roofeh *et al* (2013) noted that the human leukocyte antigen region of a genome could well be a plausible cause for some types of schizophrenia. It is interesting to note in the Yang *et al* (2013) study the DAO gene may interact with another called RASD2 which may affect dopamine production (the next cause we will look at).

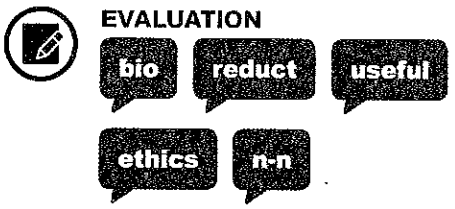


Biochemical (dopamine hypothesis)

This idea is based around the idea that schizophrenia is caused by an excess of dopamine in the brain. This involves two main ideas:

- When people experience amphetamine psychosis it resembles certain types of schizophrenia. This is caused by an excess of dopamine.
- Drug treatment (e.g. prescribing phenothiazines) does help to treat some of the symptoms of schizophrenia *but* these drugs can bring about symptoms similar to Parkinson's disease which is cause by *low* levels of dopamine.

Linstroem *et al* (1999) used a PET scan to test out the dopamine hypothesis. Ten schizophrenics and ten healthy controls were injected with a radioactively labelled chemical called L-DOPA. This is used in the production of dopamine. The PET scan could trace its usage in all participants. The L-DOPA was taken up significantly faster in the schizophrenics, pointing towards them producing more dopamine. Also, Arakawa *et al* (2010) noted that a drug called perospirone, which has a high affinity to D2 dopamine receptors, had an average 75 per cent usage rate which then blocked the further production of dopamine in schizophrenics. Seeman (2011) reviewed the field and noted that animal models of schizophrenia pointed toward elevation in levels of D2 receptors and that antipsychotics do reverse the elevation in D2 receptors but should only be used in the short term to stop other side effects.



Cognitive

This idea states that schizophrenia is caused by faulty information processing. Frith (1992) noted that schizophrenics might have a deficient "metarepresentation" system – the system that makes people able to reflect on thoughts, emotions and behaviours. It could also be linked to theory of mind (see Core study 3.3 for AS level on page 21) as it controls self-awareness and how we interpret the actions of others. These are characteristics that are lacking in some schizophrenics.

Also, those showing more negative symptoms might have a dysfunctional supervisory attention system. This system is responsible for generating self-initiated actions. Frith & Done (1986) reported that when participants were asked to do things such as name as many different fruits as possible, or generate as many designs for something as possible,

those with schizophrenia (with negative symptoms predominant) had great difficulty in managing this.

Frith (1992) also examined a central monitoring system. This allows us to be able to understand and label actions that we do as being controlled by ourselves. Frith had noticed that in some schizophrenics inner speech may not be recognised as being self-generated. Therefore, when they hear "voices" it is their own voice but they are unaware that it is themselves producing inner speech and believe it is someone else.

Finally, Johnson *et al* (2013) tested the cognitive abilities of 99 schizophrenics and 77 healthy controls on a battery of cognitive tests. It was seen that the schizophrenics performed worse across all cognitive tests including those for working memory (which involves tasks such as dealing with inner speech) and that this might be the core determinant of overall cognitive impairment in schizophrenics.

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CHALLENGE YOURSELF

Argue that the cognitive theory is either a cause of schizophrenia or an effect of the condition.

TREATMENTS FOR SCHIZOPHRENIA

There are four different treatments that will be covered below.

Biochemical

This treatment centres on using drugs to alleviate the symptoms of schizophrenia. Davison & Neale (1997) noted that, from the 1950s onwards, drugs classed as phenothiazines were commonly used to treat schizophrenia. They were effective as they block dopamine receptors in the brain. However, many had "extrapyramidal side effects" which resemble symptoms of neurological diseases such as Parkinsonian-type tremors, dystonia (muscular rigidity), dyskinesia (chewing movements) and akasthesia (the inability to keep still). Second-generation antipsychotics were developed to also block dopamine receptors *but* produce fewer side effects and there are now third-generation antipsychotics that reportedly produce even fewer side effects.

Contemporary research still shows the effectiveness of antipsychotics in treating schizophrenia. Sarkar & Grover (2013) conducted a meta-analysis on 15 randomised controlled studies testing the effectiveness of antipsychotics on children and adolescents diagnosed with schizophrenia. It was seen that both first- and second-generation antipsychotic drugs were superior to the placebo in alleviating symptoms. Second-generation drugs were superior overall with chlozapine being the most effective of all drugs. Extrapyramidal side effects were seen more in first-generation antipsychotics while side effects that affected metabolism were seen more often in second-generation drugs.

Ehret, Sopko & Lemieux (2010) noted that a third-generation drug called lurasidone had been shown to be effective in four separate clinical trials, reducing both positive and negative symptoms. Noted side effects had only been nausea, vomiting and dizziness (they noted that drugs like clozapine were now showing more metabolic dysfunction side effects plus bone marrow toxicity so newer drugs needed to be developed).

Keating (2013) noted that a first-generation drug called loxapine was now being used again as an effective treatment for agitation in schizophrenic patients by getting them to inhale it as a powder. This meant a rapid onset of effect (usually around 10 minutes) by using a non-invasive method that showed few side effects.

Finally, Motiwala, Siscoe & El-Mallakh (2013) reported on the use of depot aripiprazole for schizophrenia. Depot injections are usually given deep into a muscle and allow the administration of a sustained-action drug formulation for slow release and gradual absorption, so that the active agent can act for much longer periods than is possible with standard injections. Only one study had been published in a peer review journal but it was positive in terms of effectiveness and safety so in the future this method of antipsychotic drug delivery may gain momentum.

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Electro-convulsive therapy (ECT)

ECT is basically a procedure where a person receives a brief application of electricity to induce a seizure. Early attempts at this were not pleasant but nowadays patients are anaesthetised and given muscle relaxants. Electrodes are fitted to specific areas of the head and a small electrical current is passed through them for no longer than 1 second. The seizure may last up to 1 minute and the patient regains consciousness in around 15 minutes. There will always be debate about whether ECT should be used for any mental health issue as clinicians and psychologists are divided on the severity of the therapy itself and the longer-term side effects. ECT is now mainly used for depression (we will come back to its effectiveness with this on page 220), but there has been research conducted on the use of ECT with schizophrenics.

Zervas, Theleritis & Soldatos (2012) conducted a review of the use of ECT in schizophrenia. They looked at four issues: symptom response, technical application, continuation/maintenance ECT and its combination with medication. It would appear that ECT can be quite effective with catatonic schizophrenics and in reducing paranoid delusions. There was also evidence that it may improve a person's responsivity to medication. Lengthier courses worked well with catatonic schizophrenics. When combined with medication, ECT worked better than when only ECT was used. Phutane *et al* (2011) also noted that in a sample of 202 schizophrenics who had undergone ECT, the common reason why they had the ECT was to "augment pharmachotherapy" and that the main target was catatonia. Thirthalli *et al* (2009) reported that in a sample of schizophrenics split into catatonic and non-catatonic people, those who were catatonic required fewer ECT sessions to help control their symptoms. Finally, Flamarique *et al* (2012) reported that adolescents who received ECT in conjunction with clozapine had a lower re-hospitalisation rate (7.1 per cent) compared to a group who received ECT and a different antipsychotic (58.3 per cent).

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Token economy

As we saw in sections 8.5 and 9.6, token economies are based on the idea of operant conditioning (rewards and learning by consequence). Behaviour is shaped towards something desired by giving out tokens (e.g. plastic chips or a stamp) every time a relevant behaviour is shown. Patients can accrue these tokens and exchange them for something they would like (e.g. money or food vouchers). Therefore, patients continue to show desired behaviours as they want to earn tokens to exchange for primary reinforcers that fulfil a direct biological need such as hunger or enjoyment.

Ayllon & Azrin (1968) introduced a token economy to a psychiatric hospital in a ward for long-stay female patients. Patients were rewarded for behaviours such as brushing their hair, making their bed and having a neat appearance. Their behaviour rapidly improved and staff morale was raised as staff were seeing more positive behaviours.

Gholipour *et al* (2012) tested out the effectiveness of a token economy versus an exercise programme in helping people with schizophrenia. A total of 45 patients were randomly split into three groups – two treatments and a control – (therefore, there were 15 patients per group). All participants were male, had been diagnosed for at least 3 years, were between 20 and 50 years old and had no other mental health illness. Negative symptoms of schizophrenia were measured pre- and post-treatment. The average symptom scores pre- and post-treatment are shown in Table 11.2.2.

Group	Pre-treatment score	Post-treatment score
Exercise	71.07	50.47
Token economy	76.73	41.20
Control	84.67	84.87

Table 11.2.2 Average symptom scores pre- and post-treatment (Gholipour *et al*, 2012)

As Table 11.2.2 shows, the largest reduction on negative symptom scores was in the token economy group.

Prior to this study, Dickerson, Tenhula & Green-Paden (2005) conducted a review of the field. They found 13 studies and it appeared that there was evidence for the effectiveness of a token economy in increasing the adaptive behaviours of patients with schizophrenia. They noted that many studies had methodological issues that could cast doubt on findings and that long-term follow ups were rare.

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CBT

This type of therapy aims to change or modify people's thoughts and beliefs and also change the way that they process information. A therapist will challenge irrational and faulty thoughts as well as behaviours that are not helping. Patients may be set tasks outside the face-to-face therapy to help challenge faulty thoughts and beliefs. For schizophrenia, the intention of CBT would be to help patients make sense of the psychotic experiences and reduce the negative effects of the condition plus any distress they may be feeling. Patients may also be given help to understand that views, thoughts and interpretations are not facts, then given help to deal with assessing them.

Bechdolf *et al* (2005) assessed the effectiveness of CBT versus group psychoeducation on re-hospitalisation and medication compliance up to 24 months after treatment. A total of 88 patients were randomly assigned to either group and they received 8 weeks' therapy. When followed up six months later, the CBT group were less likely to be hospitalised and be taking their medication. At 24 months post-treatment, the CBT group had 71 days fewer in hospital. In a further study, Bechdolf *et al* (2010) analysed the data collected from their first study but on quality of life measures taken at six months post-treatment. Both groups reported improved quality of life but there was no significant difference between the two treatment groups.

Ng, Hui & Pau (2008) assessed the introduction of a CBT programme in a hostel for people who had become treatment-resistant to schizophrenia (drug therapy) in Hong Kong. Measures of schizophrenic symptoms, mood, insight and self-esteem were taken pre- and post-treatment. Six months after treatment there was a significant reduction in the symptoms of schizophrenia alongside an increase in self-esteem. Mood and insight remained unchanged.

Davis *et al* (2008) noted that there had been little research into patients evaluating CBT for schizophrenia. Their study used 44 patients with schizophrenia who either underwent CBT or a support-group programme. The study lasted for six months. Irrespective of group, all patients were satisfied with the intervention they had taken part in, rating it either good or excellent. However, those in the CBT group reported higher levels of satisfaction overall especially with the quality of service and the assistance given for problem solving.

Finally, Sarin, Wallin & Widerlöv (2011) conducted a meta-analysis on the use of CBT with schizophrenics. They concluded that there was strong evidence for CBT affecting positive, negative and general symptoms of schizophrenia compared to all other therapies. They also stated that the effects of CBT can be delayed and having 20 sessions or more is better than shorter programmes that are available.

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CHALLENGE YOURSELF

You have been asked to choose the most effective programme for dealing with schizophrenia at a local clinic. Which therapy would you choose and how would you run the programme? Justify your choices.