SCHIZOPHRENIA

THE SPECIFICATION SAYS...

Classification of schizophrenia. Positive symptoms of schizophrenia, including hallucinations and delusions. Negative symptoms of schizophrenia, including speech poverty and avolition.

Reliability and validity in diagnosis and classification of schizophrenia, including reference to co-morbidity, culture and gender bias and symptom overlap.

This spread is concerned with the symptoms and diagnosis of schizophrenia. There are many issues surrounding the diagnosis of schizophrenia, including its reliability and validity.

KEY TERMS

Schizophrenia – A severe mental illness where contact with reality and insight are impaired, an example of psychosis.

Classification of mental disorder – The process of organising symptoms into categories based on which symptoms cluster together in sufferers.

Positive symptoms of schizophrenia – Atypical symptoms experienced *in addition* to normal experiences. They include hallucinations and delusions.

Hallucinations – A positive symptom of schizophrenia. They are sensory experiences of stimuli that have either no basis in reality or are distorted perceptions of things that are there.

Delusions – A positive symptom of schizophrenia. They involve beliefs that have no basis in reality, for example, that the sufferer is someone else or that they are the victim of a conspiracy.

Negative symptoms of schizophrenia – Atypical experiences that represent the *loss* of a usual experience such as clear thinking or 'normal' levels of motivation.

Speech poverty – A negative symptom of schizophrenia. It involves reduced frequency and quality of speech.

Avolition – A negative symptom of schizophrenia. It involves loss of motivation to carry out tasks and results in lowered activity levels.

Co-morbidity – The occurrence of two illnesses or conditions together, for example a person has both schizophrenia and a personality disorder. Where two conditions are frequently diagnosed together it calls into question the validity of classifying the two disorders separately.

Symptom overlap – Occurs when two or more conditions share symptoms. Where conditions share many symptoms this calls into question the validity of classifying the two disorders separately.

Auditory hallucinations can be distracting and make it difficult to focus on other tasks requiring an auditory channel like speaking on a phone.

Diagnosis and classification of schizophrenia

Schizophrenia is a serious mental disorder suffered by about 1% of the world population. It is more commonly diagnosed in men than women, more commonly diagnosed in cities than the countryside and in working-class rather than middle-class people. The symptoms of schizophrenia can interfere severely with everyday tasks, so that many sufferers end up homeless or hospitalised.

Classification of schizophrenia

Schizophrenia does not have a single defining characteristic. It is a cluster of symptoms some of which appear to be unrelated. The two major systems for the classification of mental disorder, are the World Health Organisation's *International Classification of Disease* edition 10 (**ICD-10**) and the American Psychiatric Association's *Diagnostic and Statistical Manual* edition 5 (**DSM-5**, sometimes written as DSM-V). These differ slightly in their classification of schizophrenia. For example, in the DSM-5 system one of the so-called positive symptoms – delusions, hallucinations or speech disorganisation – must be present for diagnosis whereas two or more negative symptoms are sufficient under ICD.

ICD-10 recognises a range of subtypes of schizophrenia. *Paranoid schizophrenia* is characterised by powerful delusions and hallucinations but relatively few other symptoms. *Hebephrenic schizophrenia*, on the other hand, involves primarily negative symptoms. The defining characteristic of *catatonic schizophrenia* is disturbance to movement, leaving the sufferer immobile or alternatively overactive. Previous editions of the DSM system also recognised subtypes of schizophrenia but this has been dropped in DSM-5.

Positive symptoms

Positive symptoms of schizophrenia are additional experiences beyond those of ordinary existence. They include hallucinations and delusions.

Hallucinations These are unusual sensory experiences. Some hallucinations are related to events in the environment whereas others bear no relationship to what the senses are picking up from the environment, for example, voices heard either talking to or commenting on the sufferer, often criticising them. Hallucinations can be experienced in relation to any sense. The sufferer may, for example, see distorted facial expressions or occasionally people or animals that are not there.

Delusions Also known as paranoia, delusions are irrational beliefs. These can take a range of forms. Common delusions involve being an important historical, political or religious figure, such as Jesus or Napoleon. Delusions also commonly involve being persecuted, perhaps by government or aliens or of having superpowers. Another class of delusions concerns the body. Sufferers may believe that they or part of them is under external control. Delusions can make a sufferer of schizophrenia behave in ways that make sense to them but seem bizarre to others. Although the vast majority of sufferers are not aggressive and are in fact more likely to be victims than perpetrators of violence, some delusions can lead to aggression.

Negative symptoms

Negative symptoms of schizophrenia involve the loss of usual abilities and experiences. Examples include avolition and speech poverty.

Avolition Sometimes called 'apathy', can be described as finding it difficult to begin or keep up with goal-directed activity, i.e. actions performed in order to achieve a result. Sufferers of schizophrenia often have sharply reduced motivation to carry out a range of activities. Andreason (1982) identified three identifying signs of avolition; poor hygiene and grooming, lack of persistence in work or education and lack of energy.

Speech poverty Schizophrenia is characterised by changes in patterns of speech. The ICD-10 recognises speech poverty as a negative symptom. This is because the emphasis is on reduction in the amount and quality of speech in schizophrenia. This is sometimes accompanied by a delay in the sufferer's verbal responses during conversation.

Nowadays, however, the DSM system places its emphasis on speech *disorganisation* in which speech becomes incoherent or the speaker changes topic mid-sentence. This is classified in DSM-5 as a positive symptom of schizophrenia, whilst speech poverty remains as a negative symptom.

Practical activity on page 214

Evaluation

Reliability

Reliability means consistency. An important measure of reliability is **inter-rater reliability**, the extent to which different assessors agree on their assessments. In the case of diagnosis this means the extent to which two or more mental health professionals arrive at the same diagnosis for the same patients. Elie Cheniaux *et al.* (2009) had two psychiatrists independently diagnose 100 patients using both DSM and ICD criteria. Inter-rater reliability was poor, with one psychiatrist diagnosing 26 with schizophrenia according to DSM and 44 according to ICD, and the other diagnosing 13 according to DSM and 24 according to ICD. This poor reliability is a weakness of diagnosis of schizophrenia.

Validity

Validity is the extent to which we are measuring what we are intending to measure. In the case of a mental disorder like schizophrenia there are a number of validity issues to consider. One standard way to assess validity of a diagnosis is **criterion validity**; do different assessment systems arrive at the same diagnosis for the same patient? Looking at the figures in the Cheniaux *et al.* study above we can see that schizophrenia is much more likely to be diagnosed using ICD than DSM. This suggests that schizophrenia is either over-diagnosed in ICD or under-diagnosed in DSM. Either way, this is poor validity – a weakness of diagnosis.

Co-morbidity

'Morbidity' refers to a medical condition or how common it is (hence we talk about morbidity rates). Co-morbidity is the phenomenon that two or more conditions occur together – hence we speak of co-morbidity rates. If conditions occur together a lot of the time then this calls into question the validity of their diagnosis and classification because they might actually be a single condition. Schizophrenia is commonly diagnosed with other conditions. In one review Peter Buckley *et al.* (2009) concluded that around half of patients with a diagnosis of schizophrenia also have a diagnosis of **depression** (50%) or substance abuse (47%). **Post-traumatic stress disorder** also occurred in 29% of cases and OCD in 23%. This poses a challenge for both classification and diagnosis of schizophrenia. In terms of diagnosis, if half the schizophrenia patients are also diagnosed with depression, maybe we are just quite bad at telling the difference between the two conditions. In terms of classification, it may be that, if very severe depression looks a lot like schizophrenia and vice versa, then they might be better seen as a single condition. This confusing picture is a weakness of diagnosis and classification.

Symptom overlap

There is considerable overlap between the symptoms of schizophrenia and other conditions. For example, both schizophrenia and **bipolar disorder** involve positive symptoms like delusions and negative symptoms like avolition. This again calls into question the validity of both the classification and diagnosis of schizophrenia.

Under ICD a patient might be diagnosed as a schizophrenic; however, many of the same patients would receive a diagnosis of bipolar disorder according to DSM criteria. This is unsurprising given the overlap of symptoms. It even suggests that schizophrenia and bipolar disorder may not be two different conditions but one.

Evaluation eXtra

Gender bias in diagnosis

Julia Longenecker *et al.* (2010) reviewed studies of the prevalence of schizophrenia and concluded that since the 1980s men have been diagnosed with schizophrenia rather more often than women (prior to this there appears to have been no difference).

This may simply be because men are more **genetically** vulnerable to developing schizophrenia than women. However, another possible explanation is **gender bias** in the diagnosis of schizophrenia. It appears that female patients typically function better than men, being more likely to work and have good family relationships (Cotton *et al.* 2009). This high functioning may explain why some women have not been diagnosed with schizophrenia where men with similar symptoms might have been; their better interpersonal functioning may bias practitioners to under-diagnose schizophrenia, either because symptoms are masked altogether by good interpersonal functioning, or because the quality of interpersonal functioning makes the case seem too mild to warrant a diagnosis.

Consider: *What does possible under-diagnosis of schizophrenia in women suggest about the validity of the diagnosis?*

Cultural bias in diagnosis

African Americans and English people of Afro-Caribbean origin are several times more likely than white people to be diagnosed with schizophrenia. Given that rates in Africa and the West Indies are not particularly high, this is almost certainly *not* due to genetic vulnerability. Instead diagnosis seems to be beset with issues of **culture bias**.

There may be several factors at work here. One issue is that positive symptoms such as hearing voices may be more acceptable in African cultures because of cultural beliefs in communication with ancestors, and thus people are more ready to acknowledge such experiences. When reported to a psychiatrist from a different cultural tradition these experiences are likely to be seen as bizarre and irrational. In addition, Javier Escobar (2012) has pointed out that (overwhelmingly white) psychiatrists may tend to over-interpret symptoms and distrust the honesty of black people during diagnosis.

Consider: *What does the over-diagnosis of schizophrenia in Black British and Americans suggest about the validity of the diagnosis?*

Apply it

Concepts: Agwe's story

Jamaican-born Agwe has been referred to a psychiatrist because he has told people that he has recently heard his dead grandfather talking to him.

Questions

1. With reference to the terms 'positive symptoms' and 'hallucinations' explain why Agwe might receive a diagnosis of schizophrenia.
2. How might issues of culture bias affect this potential diagnosis?

Artist Louis Wain reportedly captured the visual distortions of his descent into schizophrenia in these pictures of a cat.

Apply it

Methods: Buckley *et al.*

According to Buckley *et al.*, 50% of patients with a diagnosis of schizophrenia also have depression, whilst 47% have one of substance abuse. For PTSD the figure is 29% and for OCD it is 23%.

Questions

1. Estimate how many people from a sample of 1637 patients with a diagnosis of schizophrenia also have OCD. *(1 mark)*
2. What is the ratio of patients with co-morbid OCD to those with co-morbid substance abuse? *(2 marks)*

CHECK IT

1. Explain what is meant by the *posit symptoms of schizophrenia.* *[4 ma*
2. Explain the term *avolition.* *[2 ma*
3. Explain the issues of culture bias and gender bias in the diagnosis schizophrenia. *[8 ma*
4. Describe and evaluate the classification and diagnosis of schizophrenia. *[16 ma*

BIOLOGICAL EXPLANATIONS FOR SCHIZOPHRENIA

THE SPECIFICATION SAYS...

Biological explanations for schizophrenia: genetics, the dopamine hypothesis and neural correlates

Most modern mental health professionals believe that schizophrenia is at least partly biological in origin. This spread is concerned with genetic vulnerability to schizophrenia, the possible role of the neurotransmitter dopamine and the neural correlates of schizophrenia. These three explanations are inter-related because, if schizophrenic is genetic, then those genes lead to biological differences such as abnormal levels of dopamine and/or abnormal structure of the brain (neural correlates).

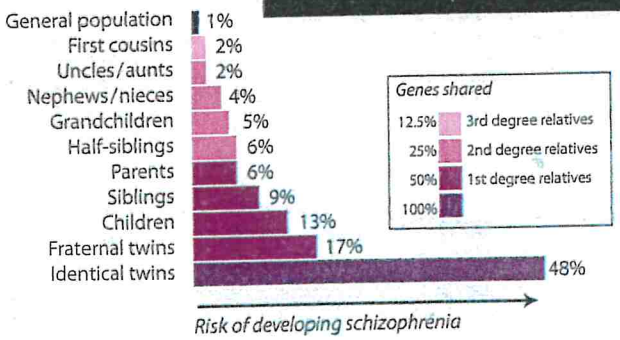
KEY TERMS

Genetics – Genes consist of DNA strands. DNA produces 'instructions' for general physical features of an organism (such as eye colour, height) and also specific physical features (such as neurotransmitter levels and size of brain structures). These may impact on psychological features (such as intelligence and mental disorder). Genes are transmitted from parents to offspring, i.e. inherited.

Dopamine – A neurotransmitter that generally has an excitatory effect and is associated with the sensation of pleasure. Unusually high levels are associated with schizophrenia and unusually low levels are associated with Parkinson's disease.

Neural correlates – Patterns of structure or activity in the brain that occur in conjunction with an experience and may be implicated in the origins of that experience.

As genetic similarity increases so does the probability of sharing schizophrenia. Source: Gottesman (1991)



Apply it Concepts: Gene's genes

Gene and his partner Kary are considering having children. However, Gene is concerned that, as his own father has suffered severely with schizophrenia for many years, he may pass on 'the gene' for schizophrenia to his own children. Gene and Kary attend genetic counselling in order to learn about the risks of their children developing schizophrenia.

Questions

1. What is the probability of Gene and Kary's child developing schizophrenia?
2. What might the genetic counsellor tell them about their understanding of 'the gene' for schizophrenia?

Biological explanations

The genetic basis of schizophrenia

Schizophrenia runs in families It has been noted for many years that **schizophrenia** runs in families. This is quite weak evidence in itself for a genetic link because family members tend to share aspects of their environment as well as many of their genes. However, there have been systematic investigations of the extent to which greater genetic similarity between family members is associated with the likelihood of both developing schizophrenia. For example, we share 100% of our genes with an identical twin, 50% with a sibling or parent and so on. There is a strong relationship between the degree of genetic similarity and shared risk of schizophrenia. This is shown in the graph below left, which presents the findings from Irving Gottesman's (1991) large-scale family study.

Candidate genes Individual genes are believed to be associated with risk of inheritance. Because a number of genes each appear to confer a small increased risk of schizophrenia it appears that schizophrenia is polygenic, i.e. it requires a number of factors to work in combination. Because different studies have identified different candidate genes it also appears that schizophrenia is aetiologically heterogeneous, i.e. *different* combinations of factors can lead to the condition. Stephen Ripke *et al.* (2014) carried out a huge study combining all previous data from **genome**-wide studies (i.e. those looking at the whole human genome as opposed to particular genes) of schizophrenia. The genetic make-up of 37,000 patients was compared to that of 113,000 controls; 108 separate genetic variations were associated with increased risk of schizophrenia. Genes associated with increased risk included those coding for the functioning of a number of **neurotransmitters** including dopamine.

The dopamine hypothesis

Neurotransmitters The brain's chemical messengers appear to work differently in the brain of a patient with schizophrenia. In particular dopamine (or DA) is widely believed to be involved. Dopamine is important in the functioning of several brain systems that may be implicated in the symptoms of schizophrenia.

Hyperdopaminergia in the subcortex The original version of the dopamine hypothesis focused on the possible role of high levels or activity of dopamine (*hyper*dopaminergia) in the **subcortex**, i.e. the central areas of the brain. For example, an excess of dopamine receptors in **Broca's area** (which is responsible for speech production) may be associated with poverty of speech and/or the experience of auditory hallucinations.

Hypodopaminergia in the cortex More recent versions of the dopamine hypothesis have focused instead on abnormal dopamine systems in the brain's **cortex**. Goldman-Rakic *et al.* (2004) have identified a role for low levels of dopamine (*hypo*dopaminergia) in the **prefrontal cortex** (responsible for thinking and decision making) in the negative symptoms of schizophrenia.

It may be that both hyper- and hypodopaminergia are correct explanations – both high and low levels of dopamine in different brain regions are involved in schizophrenia.

Neural correlates of schizophrenia

Neural correlates are measurements of the structure or function of the brain that correlate with an experience, in this case schizophrenia. Both **positive** and **negative symptoms** have neural correlates.

Neural correlates of negative symptoms One negative symptom **avolition** involves the loss of motivation. Motivation involves the anticipation of a reward, and certain regions of the brain, for example, the **ventral striatum**, are believed to be particularly involved in this anticipation. It therefore follows that abnormality of areas like the ventral striatum may be involved in the development of avolition. Juckel *et al.* (2006) have measured activity levels in the ventral striatum in schizophrenia and found lower levels of activity than those observed in controls. Moreover, they observed a **negative correlation** between activity levels in the ventral striatum and the severity of overall negative symptoms. Thus activity in the ventral striatum is a neural correlate of negative symptoms of schizophrenia.

Neural correlates of positive symptoms Positive symptoms also have neural correlates. Allen *et al.* (2007) scanned the brains of patients experiencing auditory **hallucinations** and compared them to a **control group** whilst they identified pre-recorded speech as theirs or others. Lower activation levels in the **superior temporal gyrus** and **anterior cingulate gyrus** were found in the hallucination group, who also made more errors than the control group. We can thus say that reduced activity in these two areas of the brain is a neural correlate of auditory hallucination.

Evaluation

Multiple sources of evidence for genetic susceptibility

There is now very strong evidence for genetic vulnerability to schizophrenia from a variety of sources. The Gottesman (1991) study (facing page) clearly shows how genetic similarity and shared risk of schizophrenia are closely related. **Adoption studies** such as that by Pekka Tienari *et al* (2004), discussed on page 210, clearly show that children of schizophrenia sufferers are still at heightened risk of schizophrenia if adopted into families with no history of schizophrenia. There is also evidence from studies conducted at the molecular level showing that particular genetic variations significantly increase the risk of schizophrenia (Ripke *et al*. 2014, facing page).

There is thus overwhelming evidence for the idea that genetic factors make some people much more vulnerable to developing schizophrenia than others. This does not of course mean that schizophrenia is entirely genetic. There are a number of factors in the environment associated with risk of schizophrenia, but the available evidence suggests that genetic susceptibility is very important.

Mixed evidence for the dopamine hypothesis

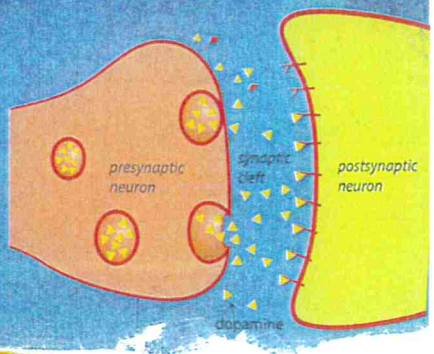
There is support from a number of sources for abnormal dopamine functioning in schizophrenia. Dopamine **agonists** like amphetamines that increase the levels of dopamine make schizophrenia worse and can produce schizophrenia-like symptoms in non-sufferers (Curran *et al*. 2004). **Antipsychotic drugs**, on the other hand, work by reducing dopamine activity (Tauscher *et al*. 2014). Both kinds of drug study suggest an important role for dopamine in schizophrenia. Radioactive labelling studies such as that by Lindstroem *et al*. (1999) have found that chemicals needed to produce dopamine are taken up faster in the brains of schizophrenia sufferers than controls, suggesting that they produce more dopamine.

There is also evidence to suggest that dopamine does not provide a complete explanation for schizophrenia. Some of the genes identified in the Ripke *et al*. study code for the production of other neurotransmitters, so it appears that although dopamine is likely to be one important factor in schizophrenia, so are other neurotransmitters. Much of the attention in current research has shifted to the role of a neurotransmitter called **glutamate** (Moghaddam and Javitt 2012). Evidence for the dopamine hypothesis can perhaps be best described as mixed.

The correlation-causation problem

There are a number of neural correlates of schizophrenia symptoms, including both positive and negative symptoms. Although studies like those on the facing page are useful in flagging up particular brain systems that may not be working normally, this kind of evidence leaves some important questions unanswered. Most importantly, does the unusual activity in a region of the brain *cause* the symptom? Logically there are other possible explanations for the correlation.

Take, for example, the correlation between levels of activity in the ventral striatum and negative symptoms of schizophrenia. It may be that something wrong in the striatum is causing negative symptoms. However, it is just as possible that the negative symptoms themselves mean that less information passes through the striatum, resulting in the reduced activity. A third possibility is that another factor influences both the negative symptoms and the ventral striatum activity. The existence of neural correlates in schizophrenia therefore tells us relatively little in itself.



The action of dopamine at a synapse. This appears to be disrupted in schizophrenia.

Apply it **Concepts:**

Parkinson's and the dopamine hypothesis

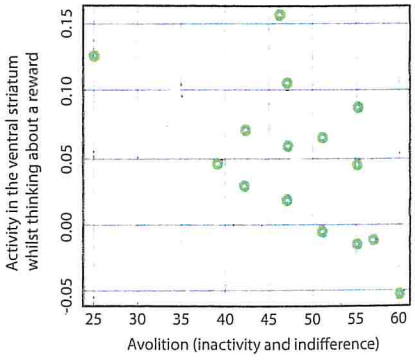
Parkinson's disease is a degenerative condition in which cells in a region of the brain called the *substantia nigra* die, resulting in a reduction in dopamine levels. This in turn affect the brain's ability to control movement. Parkinson's is treated with drugs that help the brain produce more dopamine. However, these drugs worsen the symptoms of schizophrenia.

Questions

1. What does this suggest about the dopamine hypothesis as an explanation for schizophrenia?
2. Now imagine that a new drug for treating Parkinson's also worked by raising dopamine levels only in the cortex, and that this *reduced* the symptoms of schizophrenia. What would this suggest about the dopamine hypothesis?

Apply it **Methods: A correlation**

The following graph shows the correlation between avolition and activity in the brain's reward system in schizophrenia (fro Simon *et al*. 2015).



Questions

1. Estimate the **correlation coefficient** shown in the picture. *(2 marks)* (See page 63)
2. Explain what this graph shows. *(2 marks)*
3. What **statistical test** would you use to test the significance of these results? Explain the reasons for your choice. *(3 marks)* (See page 70)

Evaluation eXtra

The role of mutation

Schizophrenia can take place in the absence of a family history of the disorder. One explanation for this is **mutation** in parental DNA, for example, in paternal sperm cells. This can be caused by radiation, poison or viral infection. Evidence for the role of mutation comes from a study showing a **positive correlation** between paternal age (associated with increased risk of sperm mutation) and risk of schizophrenia, increasing from around 0.7% with fathers under 25 to over 2% in fathers over 50 (Brown *et al*. 2002).

Consider: *Does this link between mutations in paternal sperm and risk of schizophrenia support the importance of genetic factors in the development of schizophrenia? Explain your answer.*

The role of the psychological environment is important but unclear

The evidence supporting the role of biological factors in schizophrenia is overwhelming. However, there is also evidence to suggest an important role for environmental factors, including psychological ones such as family functioning during childhood. After all, the probability of developing schizophrenia even if your identical twin has it is less than 50%. Psychological explanations are further explored on page 206.

Consider: *To what extent should we see schizophrenia as a biological condition?*

CHECK IT

1. Explain the role of genetics in the development of schizophrenia. *[4 mar*
2. Evaluate the role of genetics in the developmen of schizophrenia. *[8 mar*
3. What is meant by *neural correlates*? *[2 mar*
4. Outline the dopamine hypothesis. *[4 mar*
5. Describe and evaluate biological explanations for schizophrenia. *[16 mar*

PSYCHOLOGICAL EXPLANATIONS FOR SCHIZOPHRENIA

THE SPECIFICATION SAYS...

Psychological explanations for schizophrenia: family dysfunction and cognitive explanations, including dysfunctional thought processing.

Although there is little doubt that biological processes are important in both the origins and symptoms of schizophrenia, this does not mean that psychological processes are not also important. This spread is concerned with the role of family dysfunction and cognitive explanations in schizophrenia.

KEY TERMS

Family dysfunction – Abnormal processes within a family such as poor family communication, cold parenting and high levels of expressed emotion. These may be risk factors for both the development and maintenance of schizophrenia.

Cognitive explanations – Explanations that focus on mental processes such as thinking, language and attention.

Dysfunctional thought processing – A general term meaning information processing that is not functioning normally and produces undesirable consequences.

Apply it **Concepts: Raj**

Raj, a patient with schizophrenia, and his mother are planning for Raj's release from a psychiatric hospital with Raj's psychiatrist. Raj's mother says that she fears that he will not cope living on his own but also that his living with her will require sacrifices on her part. She points to Raj's record of coping without her in the past. The psychiatrist notes that Raj's family functioning appears to be dysfunctional.

Question

In what ways does this interaction suggest that Raj's family functioning is dysfunctional?

The schizophrenogenic mother is cold and rejecting.

Psychological explanations

There are a range of psychological explanations for **schizophrenia**. Some of these have focused on the psychological environment, in particular the family, and its role in making individuals particularly vulnerable to schizophrenia. Others have focused more on the mind of the sufferer and emphasising the role of abnormal cognition in the experience of schizophrenia.

Family dysfunction

Psychologists have attempted to link schizophrenia to childhood and adult experiences of living in a dysfunctional family (family dysfunction).

The schizophrenogenic mother Frieda Fromm-Reichmann (1948) proposed a psychodynamic explanation for schizophrenia based on the accounts she heard from her patients about their childhoods. Fromm-Reichm an noted that many of her patients spoke of a particular type of parent, which she called the **schizophrenogenic mother**. 'Schizophrenogenic' literally means 'schizophrenia-causing'. According to Fromm-Reichmann the schizophrenogenic mother is cold, rejecting and controlling, and tends to create a family climate characterised by tension and secrecy. This leads to distrust that later develops into paranoid delusions (i.e. the belief that one is being persecuted by another person), and ultimately schizophrenia.

Double-bind theory Gregory Bateson *et al.* (1972) agreed that family climate is important in the development of schizophrenia but emphasised the role of communication style within a family. The developing child regularly finds themselves trapped in situations where they fear doing the wrong thing, but receive mixed messages about what this is, and feel unable to comment on the unfairness of this situation or seek clarification. When they 'get it wrong' (which is often) the child is punished by withdrawal of love. This leaves them with an understanding of the world as confusing and dangerous, and this is reflected in symptoms like disorganised thinking and paranoid delusions. Bateson was clear that this was neither the main type of communication in the family of schizophrenia-sufferers nor the only factor in developing schizophrenia, just a risk factor.

Expressed emotion and schizophrenia Expressed emotion (or EE) is the level of emotion, in particular negative emotion, expressed towards a patient by their carers. EE contains several elements:

- Verbal criticism of the patient, occasionally accompanied by violence.
- Hostility towards the patient, including anger and rejection.
- Emotional over-involvement in the life of the patient, including needless self-sacrifice.

These high levels of expressed emotion in carers directed towards the patient are a serious source of stress for the patient. This is primarily an explanation for relapse in patients with schizophrenia. However, it has also been suggested that it may be a source of stress that can trigger the onset of schizophrenia in a person who is already vulnerable, for example, due to their genetic make-up (the **diathesis-stress model** discussed fully on page 212).

Cognitive explanations

A cognitive explanation for any phenomenon is one which focuses on the role of mental processes. Schizophrenia is associated with several types of abnormal information processing, and these can provide possible explanations for schizophrenia as a whole.

Schizophrenia is characterised by disruption to normal thought processing. We can see this in many of its symptoms. We have already seen that reduced processing in the **ventral striatum** is associated with negative symptoms, whilst reduced processing of information in the **temporal** and **cingulate gyri** are associated with **hallucinations** (see page 202). This lower than usual level of information processing suggests that cognition is likely to be impaired.

Christopher Frith *et al.* (1992) identified two kinds of dysfunctional thought processing that could underlie some symptoms:

- *Metarepresentation* is the cognitive ability to reflect on thoughts and behaviour. This allows us insight into our own intentions and goals. It also allows us to interpret the actions of others. Dysfunction in metarepresentation would disrupt our ability to recognise our own actions and thoughts as being carried out by ourselves rather than someone else. This would explain hallucinations of voices and delusions like thought insertion (the experience of having thoughts projected into the mind by others).
- *Central control* is the cognitive ability to suppress automatic responses while we perform deliberate actions instead. Disorganised speech and thought disorder could result from the inability to suppress automatic thoughts and speech triggered by other thoughts. For example, sufferers with schizophrenia tend to experience derailment of thoughts and spoken sentences because each word triggers associations, and the patient cannot suppress automatic responses to these.

Practical activity on page 215

Evaluation

Support for family dysfunction as a risk factor

There is evidence to suggest that difficult family relationships in childhood are associated with increased risk of schizophrenia in adulthood. For example, Read *et al.* (2005) reviewed 46 studies of child abuse and schizophrenia and concluded that 69% of adult women in-patients with a diagnosis of schizophrenia had a history of physical abuse, sexual abuse or both in childhood. For men the figure was 59%. Adults with **insecure attachments** to their primary carer are also more likely to have schizophrenia (Berry *et al.* 2008).

There is thus a large body of evidence linking family dysfunction to schizophrenia. However, most of this evidence shares a weakness. Information about childhood experiences was gathered after the development of symptoms, and the schizophrenia may have distorted patients' recall of childhood experiences. This creates a serious problem of **validity**. A much smaller number of studies (e.g. Tienari *et al.*, see page 213) have been carried out prospectively, i.e. they followed up children following childhood experiences to see if the childhood experience predicted any adult characteristics. There *is* prospective evidence linking family dysfunction to schizophrenia but not a huge amount and results have been inconsistent.

Weak evidence for family-based explanations

Although there is plenty of evidence supporting the broad principle that poor childhood experiences in the family are associated with adult schizophrenia, there is almost none to support the importance of the schizophrenogenic mother or double bind. Both these theories are based on clinical observation of patients, and early evidence involved assessing the personality of the mothers of patients for 'crazy-making characteristics' – an approach that makes many modern psychiatrists wince (Harrington 2012).

Another problem with dysfunctional family explanations for schizophrenia is that they have led historically to parent-blaming. Parents, who have already suffered at seeing their child's descent into schizophrenia and who are likely to bear lifelong responsibility for their care, underwent further trauma by receiving the blame for the condition. This is literally adding insult to injury. In fact the shift in the 1980s from hospital to community care, often involving parental care, may be one of the factors leading to the decline of the schizophrenogenic mother and double bind theories – parents no longer tolerated them.

Strong evidence for dysfunctional information processing

There is strong support for the idea that information is processed differently in the mind of the schizophrenia sufferer. In one study Stirling *et al.* (2006) compared 30 patients with a diagnosis of schizophrenia with 18 non-patient **controls** on a range of cognitive tasks including the Stroop Test (see right), in which participants have to name the ink colours of colour words, suppressing the impulse to read the words in order to do this task. In line with Frith's theory of central control dysfunction, patients took over twice as long to name the ink colours as the control group.

Although there is a mass of evidence like this to show that information processing is different in the mind of schizophrenia sufferer, there is a problem with cognitive explanations for schizophrenia. Links between symptoms and faulty cognition are clear; however, this does not tell us anything about the *origins* of those cognitions or of schizophrenia. Cognitive theories can explain the proximal causes of schizophrenia, i.e. what causes current symptoms but not the distal causes, i.e. the origins of the condition.

Evaluation eXtra

Evidence for biological factors is not adequately considered

In their pure forms at least, psychological explanations (particularly family dysfunction) for schizophrenia can be hard to reconcile with the biological explanations we looked at on the previous spread. It could be that both biological and psychological factors can separately produce the same symptoms, which raises the question of whether both outcomes are really schizophrenia. Alternatively, we can view this in terms of the diathesis-stress model where the diathesis may be biological (as discussed on the previous spread) or psychological (as discussed on this spread).

Consider: *Given the strength of evidence for biological factors in schizophrenia, what is the place of psychological explanations?*

Direction of causality

We have a mass of information concerning abnormal cognitions as well as a mass of information about abnormal biology in schizophrenia. However, it remains unclear what causes what, including whether cognitive factors are a cause or are a result of the neural correlates and abnormal neurotransmitter levels seen in schizophrenia.

Consider: *What does this mean for the validity of psychological and biological explanations for schizophrenia?*

Apply it

Concepts: A case of speech poverty

Melanie is having a psychiatric assessment after reporting to her GP that she is hearing voices and believes that someone else is projecting thoughts into her mind. During the interview Melanie finds it hard to keep her attention on what she is saying and frequently her conversation goes off 'on a tangent'. The psychiatrist notes this as 'derailment'.

Question

How might the psychiatrist explain each of these symptoms using the idea of dysfunctional thought processing?

Apply it

Methods: Stirling *et al.*

Stirling *et al.* (2006) studied the performance of people with schizophrenia and non-patient groups on the Stroop test. The results were as follows:

Time (s)	Mean	Standard deviation
schizophrenia	123.20	65.52
controls	58.12	11.26

Questions

1. What is meant by **standard deviation**? (*2 marks*)
2. What do the standard deviations in this table of results show? (*2 marks*)
3. Taken together with the **mean**, what do the results tell us? (*2 marks*)
4. Explain *one* strength of using the standard deviation over other **measures of dispersion**. (*2 marks*)

Sufferers of schizophrenia typically struggle with the Stroop task where you are required to name the colour that the word is written in (see text), suggesting difficulty in suppressing automatic processing.

RED YELLOW GREEN
ORANGE BLUE GREEN
YELLOW BLUE RED
ORANGE RED GREEN
RED YELLOW GREEN
ORANGE BLUE GREEN
YELLOW BLUE RED
ORANGE RED GREEN

CHECK IT

1. Outline what is meant by *dysfunctional thought processing*. Use examples in your answer. [3 ma
2. Evaluate family dysfunction as an explanation fo schizophrenia. [8 ma
3. Explain **one** limitation of family dysfunction as a explanation of schizophrenia. [4 ma
4. Describe and evaluate psychological theories of schizophrenia. [16 ma

BIOLOGICAL THERAPIES FOR SCHIZOPHRENIA: DRUG THERAPY

THE SPECIFICATION SAYS...

Drug therapy: typical and atypical antipsychotics

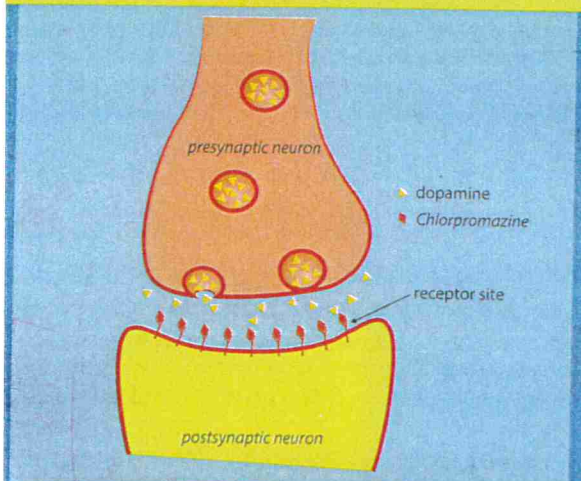
This spread is concerned with antipsychotic medication. This is the most commonly used treatment for schizophrenia. Antipsychotic drugs can be split into older typical antipsychotics and newer atypical antipsychotics.

KEY TERMS

Antipsychotics – Drugs used to reduce the intensity of symptoms, in particular the positive symptoms, of psychotic conditions like schizophrenia.

Typical antipsychotics – The first generation of antipsychotic drugs, having been used since the 1950s. They work as dopamine antagonists and include *Chlorpromazine*.

Atypical antipsychotics – Drugs for schizophrenia (a psychotic disorder) developed after typical antipsychotics. They typically target a range of neurotransmitters such as dopamine and serotonin. Examples include *Clozapine* and *Risperidone*.



Chlorpromazine acts as a dopamine antagonist, i.e. it acts against dopamine, in this case by blocking dopamine receptors at the postsynaptic neuron.

Apply it **Concepts:**

Explaining it to Brendan

Brendan has recently been diagnosed with schizophrenia with accompanying depression and suicidal thoughts. Brendan is a scientist and is curious about how antipsychotics work. He is also curious about his psychiatrist's choice of *Clozapine* as his antipsychotic.

Questions

1. How might Brendan's psychiatrist explain the effects of antipsychotics. Refer to the dopamine hypothesis in your answer.
2. Why might Brendan's psychiatrist have prescribed *Clozapine*?

Drug therapies

The most common treatment for **schizophrenia** involves the use of antipsychotic drugs. Antipsychotics can be taken as tablets or in the form of syrup. For those particularly at risk of failing to take their medication regularly some antipsychotics are available as injections given every 2–4 weeks.

Antipsychotics may be required in the short or long term. Some patients can take a short course of antipsychotics then stop their use without the return of symptoms. Other patients may require antipsychotics for life or face the likelihood of a recurrence of schizophrenia. Antipsychotics can be divided into typical (traditional) and newer atypical or second-generation drugs.

Typical antipsychotics

These have been around since the 1950s and include *Chlorpromazine*. *Chlorpromazine* can be taken as tablets, syrup or by injection. If taken orally it is administered daily up to a maximum of 1000mg, although initially doses are much smaller and for most patients the dosage is gradually increased to a maximum of 400 to 800 mg. Typical prescribed doses have declined over the last 50 years (Liu and de Haan 2009).

There is a strong association between the use of typical antipsychotics like *Chlorpromazine* and the **dopamine hypothesis**. Typical antipsychotics like *Chlorpromazine* work by acting as **antagonists** in the dopamine system. Antagonists are chemicals which reduce the action of a **neurotransmitter**. Dopamine antagonists work by blocking dopamine receptors in the synapses of the brain, reducing the action of dopamine. Initially when a patient begins taking *Chlorpromazine* dopamine levels build up, but then its production is reduced. According to the dopamine hypothesis of schizophrenia this dopamine-antagonist effect normalises neurotransmission in key areas of the brain, reducing symptoms like **hallucinations**.

As well as having antipsychotic properties *Chlorpromazine* is also an effective sedative. This is believed to be related to its effect on histamine receptors but it is not fully understood how this leads to sedation. *Chlorpromazine* is often used to calm patients not only with schizophrenia but also with other conditions. This has often been done when patients are first admitted to hospitals and are very anxious. Syrup is absorbed faster than tablets so it tends to be used when *Chlorpromazine* is used for its sedative properties.

Atypical antipsychotics

These drugs have been used since the 1970s. The aim in developing newer antipsychotics was to maintain or improve upon the effectiveness of drugs in suppressing the symptoms of psychosis and also minimise the side effects. There are a range of atypical antipsychotics and they do not all work in the same way. In fact we do not know how some of them work.

Clozapine *Clozapine* was developed in the 1960s and first trialled in the early 1970s. It was withdrawn for a while in the 1970s following the deaths of some patients from a blood condition called *agranulocytosis*. However, in the 1980s when it was discovered to be more effective than typical antipsychotics *Clozapine* was remarketed as a treatment for schizophrenia to be used when other treatments failed. It is still used in this way today, and people taking it have regular blood tests to ensure they are not developing agranulocytosis. Because of its potentially fatal side effects *Clozapine* is not available as an injection. Daily dosage is a little lower than for *Chlorpromazine*, typically 300 to 450mg a day.

Clozapine binds to dopamine receptors in the same way that *Chlorpromazine* does, but in addition it acts on **serotonin** and **glutamate** receptors. It is believed that this action helps improve mood and reduce **depression** and anxiety in patients, and that it may improve cognitive functioning. The mood-enhancing effects of *Clozapine* mean that it is sometimes prescribed when a patient is considered at high risk of suicide. This is important as 30 to 50% of people suffering from schizophrenia attempt suicide at some point.

Risperidone *Risperidone* is a more recently developed atypical antipsychotic, having been around since the 1990s. It was developed in an attempt to produce a drug as effective as *Clozapine* but without its serious side effects. Like *Chlorpromazine*, *Risperidone* can be taken in the form of tablets, syrup or an injection that lasts for around two weeks. In common with other antipsychotics a small dose is initially given and this is built up to a typical daily dose of 4–8mg and a maximum of 12mg.

Like *Clozapine*, *Risperidone* is believed to bind to dopamine and serotonin receptors. *Risperidone* binds more strongly to dopamine receptors than *Clozapine* and is therefore effective in much smaller doses than most antipsychotics. There is some evidence to suggest that this leads to fewer side effects than is typical for antipsychotics.

Evaluation

Evidence for effectiveness

There is a large body of evidence to support the idea that both typical and atypical antipsychotics are at least moderately effective in tackling the symptoms of schizophrenia. Ben Thornley *et al.* (2003) reviewed studies comparing the effects of *Chlorpromazine* to control conditions in which patients received a **placebo** so their experiences were identical except for the presence of *Chlorpromazine* in their medication. Data from 13 trials with a total of 1121 participants showed that *Chlorpromazine* was associated with better overall functioning and reduced symptom severity. Data from three trials with a total of 512 participants showed that relapse rate was also lower when *Chlorpromazine* was taken.

In addition there is support for the benefits of atypical antipsychotics. In a review Herbert Meltzer (2012) concluded that *Clozapine* is more effective than typical antipsychotics and other atypical psychotics, and that it is effective in 30–50% of treatment-resistant cases where typical antipsychotics have failed. A number of studies have compared the effectiveness of *Clozapine* and other atypical antipsychotics like *Risperidone* but results have been inconclusive, perhaps because some patients respond better to one drug or the other. It does seem though that antipsychotics in general are reasonably effective, and this is a strength.

Serious side effects

A problem with antipsychotic drugs is the likelihood of side effects, ranging from the mild to the serious and even fatal. Typical antipsychotics are associated with a range of side effects including dizziness, agitation, sleepiness, stiff jaw, weight gain and itchy skin. Long-term use can result in *tardive dyskinesia*, which is caused by dopamine supersensitivity and manifests as involuntary facial movements such as grimacing, blinking and lip smacking.

The most serious side effect of typical antipsychotics is *neuroleptic malignant syndrome* (NMS). This is believed to be caused because the drug blocks dopamine action in the **hypothalamus**, an area of the brain associated with the regulation of a number of body systems. NMS results in high temperature, delirium and coma, and can be fatal. As typical doses of antipsychotics have declined NMS has become rarer. Estimates of its frequency range from less than 0.1% to just over 2%.

Atypical antipsychotics were developed to reduce the frequency of side effects and generally this has succeeded (Meltzer 2012). However, side effects still exist and patients taking *Clozapine* have to have regular blood tests to alert doctors to early signs of *agranulocytosis*. Side effects are thus still a significant weakness of antipsychotic drugs.

Use of antipsychotics depends on the dopamine hypothesis

This is more a theoretical issue than a practical one. Our understanding of the mechanism of antipsychotic drugs is strongly tied up with the dopamine hypothesis in its original form. Remember this is the idea that there are higher than usual levels of dopamine activity in the **subcortex** of the brain. There is, however, quite a bit of evidence to show that this original dopamine hypothesis is not a complete explanation for schizophrenia, and that in fact dopamine levels in parts of the brain other than the subcortex are too low rather than too high. If this is true then it is not clear how antipsychotics, which are dopamine antagonists, can help with schizophrenia when they reduce dopamine activity. In fact our modern understanding of the relationship between dopamine and psychosis suggests that antipsychotics *shouldn't* work. This has undermined the faith of some people that antipsychotics do in fact work.

Critics have compared antipsychotics to straitjackets, describing them as human rights violations.

Apply it **Methods: Clozapine**

Clozapine is estimated to be successful in 30–50% of cases where typical antipsychotics have failed.

Questions

1. Outcome studies like this generally use **independent groups designs**. Explain why this is a good idea. (*2 marks*)
2. If you were to design an experimental study to test the effects of *Clozapine* on schizophrenia how could you control for **confounding variables**? (*3 marks*)
3. (a) Explain what is meant by the term **demand characteristics**. (*2 marks*)
   (b) How could you eliminate the effect of demand characteristics in this study? (2 marks)

Apply it

Concepts: Questioning antipsychotics

You are a mental health professional and a family friend of Cally. Cally has been prescribed antipsychotics, but is refusing to take them because she has heard that they have serious side effects and that they do not work.

Question

Cally's family ask your advice. What might you tell them about Cally's concerns?

Evaluation eXtra

Problems with the evidence for effectiveness

Although there is an impressive mass of evidence to support the effectiveness of antipsychotics, there have been some vigorous challenges to their usefulness. David Healy (2012) has suggested that some successful trials have had their data published multiple times, exaggerating the evidence for positive effects. Healy also suggests that because antipsychotics have powerful calming effects, it is easy to demonstrate that they have some positive effect on patients. This is not the same as saying they really reduce the severity of psychosis. To make matters worse, most published studies assess short-term benefits rather than long-term benefits and compare patients who keep taking antipsychotics with those suffering withdrawal having just stopped taking them.

Consider: *What do these issues suggest about the effectiveness of antipsychotics?*

The chemical cosh argument

It is widely believed that antipsychotics have been used in hospital situations to calm patients and make them easier for staff to work with, rather than for the benefits to the patients themselves. Although short-term use of antipsychotics to calm agitated patients is recommended by the National Institute for Health and Clinical Excellence (NICE), this practice is seen by some as a human rights abuse (Moncrieff 2013)

Consider: *How serious are the **ethical issues** associated with the use of antipsychotics to calm agitated patients?*

STUDY TIP

Don't shy away from using technical terms and concepts as really contribute to the quality of your answers. You may w reread the spread on synaptic transmission in our Year 1 bo fully understand the processes discussed on this spread.

CHECK IT

1. Explain what is meant by an *atypical antipsychotic* drug. [2 m
2. Outline the use of typical antipsychotics. [6 m
3. Evaluate the use of antipsychotic drugs to treat schizophrenia. [10 m
4. Describe and evaluate antipsychotics as a treatment for schizophrenia. [16 m

PSYCHOLOGICAL THERAPIES FOR SCHIZOPHRENIA

THE SPECIFICATION SAYS...

Cognitive behaviour therapy and family therapy as used in the treatment of schizophrenia. Token economies as used in the management of schizophrenia.

This spread is concerned with psychological approaches to treating schizophrenia. The conventional wisdom has been that these should be used alongside antipsychotic drugs.

KEY TERMS

Cognitive behaviour therapy (CBT) – A method for treating mental disorders based on both cognitive and behavioural techniques. From the cognitive viewpoint the therapy aims to deal with thinking, such as challenging negative thoughts. The therapy also includes behavioural techniques.

Family therapy – A psychological therapy carried out with all or some members of a family with the aim of improving their communication and reducing the stress of living as a family.

Token economies – A form of behavioural therapy, where desirable behaviours are encouraged by the use of selective reinforcement. For example, patients are given rewards (tokens) as secondary reinforcers when they engage in correct/socially desirable behaviours. The tokens can then be exchanged for primary reinforcers – favourite foods or privileges.

Family therapy aims to reduce stress and expression of negative emotion in families.

Apply it Concepts: Bronwyn

Bronwyn has been an in-patient in her local psychiatric hospital for the last year. She was admitted with a diagnosis of schizophrenia after developing a strong belief that she was being controlled by the government. After a year of medication and a course of CBT her symptoms are now under control. However, Bronwyn has become institutionalised in the hospital setting and she no longer takes good care of her appearance as she used to.

Questions

1. How might CBT have been used to help Bronwyn with her belief that she was being controlled by the government?
2. How might a token economy be used to improve Bronwyn's grooming behaviour?

Psychological therapies

Cognitive behaviour therapy (CBT)

Cognitive behaviour therapy is now commonly used to treat patients with schizophrenia. It usually takes place for between five and twenty sessions, either in groups or on an individual basis. The aim of CBT in general involves helping patients identify **irrational thoughts** and trying to change them. This may involve argument or a discussion of how likely the patient's beliefs are to be true, and a consideration of other less threatening possibilities – see case example below. This will not get rid of the symptoms of schizophrenia but it can make patients better able to cope with them.

How CBT helps Patients can be helped to make sense of how their **delusions** and **hallucinations** impact on their feelings and behaviour. Just understanding where symptoms come from can be hugely helpful for some patients. If, for example, a patient hears voices and believes the voices are demons, they will naturally be very afraid. Offering psychological explanations for the existence of hallucinations and delusions can help reduce this anxiety. Delusions can also be challenged so that a patient can come to learn that their beliefs are not based on reality.

A case example Turkington *et al.* (2004) describe an example of CBT used to challenge where a paranoid patient's delusions come from:

Paranoid patient: The Mafia are observing me to decide how to kill me.
Therapist: You are obviously very frightened . . . there must be a good reason for this.
Paranoid patient: Do you think it's the Mafia?
Therapist: It's a possibility, but there could be other explanations. How do you know that it's the Mafia?

Family therapy

Family therapy takes place with families rather than individual patients, aiming to improve the quality of communication and interaction between family members. There is a range of approaches to family therapy for schizophrenia. In keeping with psychological therapies like the **double bind** and the **schizophrenogenic mother**, some therapists see the family as the root cause of the condition. Nowadays though, most family therapists are more concerned with reducing stress within the family that might contribute to a patient's risk of relapse. In particular, family therapy aims to reduce levels of **expressed emotion** (EE).

How family therapy helps Fiona Pharoah *et al.* (2010) identify a range of strategies by which family therapists aim to improve the functioning of a family with a member suffering from schizophrenia:

- Forming a therapeutic alliance with all family members.
- Reducing the stress of caring for a relative with schizophrenia.
- Improving the ability of the family to anticipate and solve problems.
- Reduction of anger and guilt in family members.
- Helping family members achieve a balance between caring for the individual with schizophrenia and maintaining their own lives.
- Improving families' beliefs about and behaviour towards schizophrenia.

Pharoah *et al.* suggest that these strategies work by reducing levels of stress and expressed emotion, whilst increasing the chances of patients' complying with medication. This combination of benefits tends to result in a reduced likelihood of relapse and re-admission to hospital.

Token economies

Token economies are reward systems used to manage the behaviour of patients with schizophrenia, in particular those who have developed patterns of maladaptive behaviour through spending long periods in psychiatric hospitals (referred to as 'institutionalised'). Under these circumstances it is common for patients to develop bad hygiene or perhaps to remain in pyjamas all day. Modifying these bad habits does not cure schizophrenia but it improves the patient's quality of life and makes it more likely that they can live outside a hospital setting.

Tokens The idea is that tokens – for example, in the form of coloured discs – are given immediately to patients when they have carried out a desirable behaviour that has been targeted for **reinforcement**. This may be getting dressed in the morning, making a bed, etc., according to the patient's individual behaviour issues. This immediacy of reward is important because it prevents 'delay discounting', the reduced effect of a delayed reward.

Rewards Although the tokens have no value in themselves they can be swapped later for more tangible rewards. Token economies are a kind of behavioural therapy based on **operant conditioning**. Tokens are **secondary reinforcers** because they only have value once the patient has learned that they can be used to obtain rewards. These rewards might be in the form of materials such as sweets, cigarettes or magazines or rather in the form of services such as having a room cleaned or privileges such as a walk outside the hospital.

Evaluation

Evidence for effectiveness

There is some support for the benefits of psychological treatment for schizophrenia. Sameer Jauhar *et al.* (2014) reviewed the results of 34 studies of CBT for schizophrenia. They concluded that CBT has a significant but fairly small effect on both positive and negative symptoms.

Pharoah *et al.* reviewed the evidence for the effectiveness of family therapy for families of schizophrenia sufferers. They concluded that there is moderate evidence to show that family therapy significantly reduces hospital readmission over the course of a year and improves quality of life for patients and their families. However, they also noted that results of different studies were inconsistent and that there were problems with the quality of some evidence. Overall then the evidence base for family therapy is fairly weak.

A review of the evidence for token economies (McMonagle and Sultana 2009) found only three studies where patients had been **randomly allocated** to conditions, with a total of only 110 patients. Random allocation is important in matching patients to treatment and **control groups**. Only one of the three studies showed improvement in symptoms and none yielded useful information about behaviour change.

Overall there is only modest support for the effectiveness of psychological treatments and schizophrenia remains one of the harder mental health problems to treat. This is a limitation of psychological treatments.

Treatments improve quality of life but do not cure

All the psychological treatments for schizophrenia discussed here aim to make schizophrenia more manageable and in some way improve patients' quality of life. CBT helps by allowing patients to make sense of and in some cases challenge some of their symptoms. Family therapy helps by reducing the stress of living with schizophrenia in a family, both for the patient themselves and other family members. Token economies help by making patients' behaviour more socially acceptable so that they can better re-integrate into society. These things are all worth doing, but should not be confused with *curing* schizophrenia. Of course biological treatments do not cure schizophrenia either but they do reduce the severity of some symptoms.

This failure to cure schizophrenia is a weakness of psychological treatments.

Ethical issues

Although psychological treatments for schizophrenia do not have the serious side effects or medical risks of drug treatments, they can raise ethical issues. In particular token economy systems have proved controversial. The major issue is that privileges, services, etc., become more available to patients with mild symptoms and less so for those with more severe symptoms of schizophrenia that prevent them complying with desirable behaviours. This means that the most severely ill patients suffer discrimination in addition to other symptoms, and some families of patients have challenged the legality of this. This has in turn reduced the use of token economies in the psychiatric system.

Other psychological therapies can raise additional issues. CBT may involve, for example, challenging a person's paranoia, but at what point does this interfere with an individual's freedom of thought? If, for example, CBT challenged a patient's beliefs in a highly controlling government, this can easily stray into modifying their politics.

Ethical issues like this are a weakness of psychological treatments for schizophrenia.

Token economies raise ethical issues.

Apply it — Concepts: A case of high EE

Randolph has just come out of hospital after an episode of schizophrenia. His symptoms are under control as long as he takes his medication and he has returned to live with his mother and two sisters. Randolph's psychiatrist is concerned, however, by the high levels of expressed emotion in Randolph's family. She recommends family therapy for Randolph's family.

Questions

1. What might be the benefits of family therapy for Randolph and his family?
2. If Randolph's family ask his psychiatrist about the effectiveness of family therapy what might she tell them?

Apply it — Methods: Meta-analysis

Jauhar *et al.* (2014) carried out a meta-analysis on the results of 34 studies assessing the effectiveness of CBT in the treatment of schizophrenia. They concluded that patients in CBT were better off for both positive and negative symptoms than controls ($p<0.001$).

Questions

1. Explain what $p<0.001$ means. *(2 marks)*
2. Typically we say that a difference between conditions is significant when $p<0.05$. How significant is $p<0.001$ compared to $p<0.05$? *(2 marks)*

Evaluation eXtra

Quality of the evidence for effectiveness

Many small-scale studies in which mental health professionals have compared patients before and after psychological treatments have found more positive results than those described above However, these studies have problems; there is often a lack of a control group, or, if there is one then patients are not randomly allocated to treatment and control conditions. Where these studies are included in reviews, conclusions are generally more optimistic than those that strictly control which studies are included. The other way of looking at this of course is that reviews with tight controls on what studies are included may be too pessimistic in their conclusions

Consider: *What does this disparity in conclusions suggest about the effectiveness of psychological treatments?*

Alternative psychological treatments

There are other psychological therapies that can be helpful for some people with schizophrenia that are less well-known and less likely to be available to patients. For example, the National Institute for Health and Clinical Excellence (NICE) recommends art therapy, provided a qualified art therapist with experience of working with schizophrenia sufferers is available

Consider: *Is there a case for making a wider variety of psychological treatments available for sufferers of schizophrenia?Quality of the evidence for effectiveness*

STUDY TIP

All three therapies on this spread are named in the specificatio which means you need to be able to describe each of them sep and offer some evaluation. We don't have space here for every question but you should be prepared for all possibilities.

CHECK IT

1. Explain how cognitive behaviour therapy is in the treatment of schizophrenia. *[3 n*
2. Outline the use of family therapy to treat schizophrenia. *[6 n*
3. Evaluate the use of cognitive behaviour therapy to treat schizophrenia. *[6 r*
4. 'Cognitive behaviour therapy, family therap token economies are all used in the treatm and management of schizophrenia.' Discus use of these methods. Include comparisons drug therapies in your answer. *[16 r*

THE INTERACTIONIST APPROACH TO SCHIZOPHRENIA

THE SPECIFICATION SAYS...

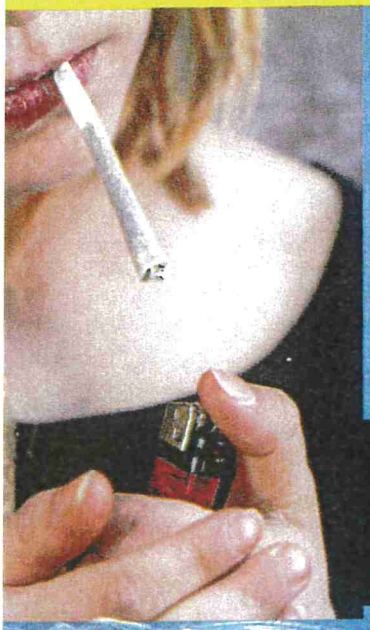
The importance of an interactionist approach in explaining and treating schizophrenia: the diathesis-stress model.

This spread is concerned with the interactionist approach to schizophrenia. This involves taking account of both biological and psychological factors in the development of schizophrenia. Treatment involves combining medication with psychological therapies.

KEY TERMS

The interactionist approach – A broad approach to explaining schizophrenia, which acknowledges that a range of factors, including biological and psychological factors, are involved in the development of schizophrenia.

The diathesis-stress model – An interactionist approach to explaining behaviour. For example schizophrenia is explained as the result of both an underlying vulnerability (diathesis) and a trigger, both of which are necessary for the onset of schizophrenia. In early versions of the model, vulnerability was genetic and triggers were psychological. Nowadays both genes and trauma are seen as diatheses, and stress can be psychological or biological in nature.



High doses of cannabis can trigger schizophrenia in people with a pre-existing vulnerability.

Apply it **Concepts: Alison**

Alison has a family history of schizophrenia and has spent time in foster care following childhood abuse. At the age of 17 she has recently been in a serious accident and is considering smoking cannabis for pain relief and to help her relax.

Questions

Referring to the diathesis-stress model of schizophrenia, how could you explain to Alison that she would be unwise to smoke cannabis?

The interactionist approach

Put simply the interactionist approach (also sometimes called the 'biosocial approach') is an approach that acknowledges that there are biological, psychological and societal factors in the development of **schizophrenia**. Biological factors include genetic vulnerability and neurochemical and neurological abnormality. Psychological factors include stress, for example, resulting from **life events** and **daily hassles**, including poor quality interactions in the family.

Explaining the interactionist approach: The diathesis-stress model

Diathesis means vulnerability. In this context *stress* simply means a negative psychological experience. The diathesis-stress model says that both a vulnerability to schizophrenia and a stress-trigger are necessary in order to develop the condition. One or more underlying factors make a person particularly vulnerable to developing schizophrenia but the onset of the condition is triggered by stress.

Meehl's model In the original diathesis-stress model (Meehl 1962) diathesis (vulnerability) was entirely **genetic**, the result of a single 'schizogene'. This led to the development of a biologically based *schizotypic personality*, one characteristic of which is sensitivity to stress. According to Paul Meehl, if a person does not have the schizogene then no amount of stress would lead to schizophrenia. However, in carriers of the gene, chronic stress through childhood and adolescence, in particular the presence of a **schizophrenogenic mother** (see page 208), could result in the development of the condition.

The modern understanding of diathesis One way in which our understanding of diathesis has changed is that it is now clear that many genes each appear to increase genetic vulnerability slightly; there is no single 'schizogene' (Ripke *et al*. 2014). Modern views of diathesis also include a range of factors beyond the genetic, including psychological trauma (Ingram and Luxton 2005) – so trauma becomes the diathesis rather than the stressor. Read *et al*. (2001) proposed a neurodevelopmental model in which early trauma alters the developing brain. Early and severe enough trauma, such as child abuse, can seriously affect many aspects of brain development. For example the **hypothalamic-pituitary-adrenal** (HPA) system can become over-active, making the person much more vulnerable to later stress.

The modern understanding of stress In the original diathesis-stress model of schizophrenia, stress was seen as psychological in nature, in particular related to parenting. Although psychological stress, including that resulting from parenting may still be considered important, a modern definition of stress (in relation to the diathesis-stress model) includes anything that risks triggering schizophrenia (Houston *et al*. 2008). Much of the recent research into factors triggering an episode of schizophrenia has concerned cannabis use. In terms of the diathesis-stress model cannabis is a stressor because it increases the risk of schizophrenia by up to seven times according to dose. This is probably because cannabis interferes with the **dopamine** system. However, most people do not develop schizophrenia after smoking cannabis so it seems there must also be one or more vulnerability factors.

Treatment according to the interactionist model

The interactionist model of schizophrenia acknowledges both biological and psychological factors in schizophrenia and is therefore compatible with both biological and psychological treatments. In particular the model is associated with combining **antipsychotic** medication and psychological therapies, most commonly **CBT**.

Douglas Turkington *et al*. (2006) point out that it is perfectly possible to believe in biological causes of schizophrenia and still practise CBT to relieve psychological symptoms. However, this requires adopting an interactionist model; it is not possible to adopt a purely biological approach and tell patients that their condition is purely biological and that there is no psychological significance to symptoms, and to simultaneously treat them with CBT.

In Britain it is increasingly standard practice to treat patients with a combination of antipsychotic drugs and CBT. In the USA there is more of a history of conflict between psychological and biological models of schizophrenia and this may have led to slower adoption of an interactionist approach. Thus medication without an accompanying psychological treatment is more common than in the UK.

It is unusual to treat schizophrenia using psychological therapies alone. CBT, family therapy and the use of token economies with sufferers of schizophrenia are usually carried out with patients taking antipsychotics.

Evaluation

Evidence for the role of vulnerability and triggers

There is evidence to support the dual role of vulnerability and stress in the development of schizophrenia. Pekka Tienari *et al.* (2004) investigated the combination of genetic vulnerability and parenting style (the trigger). Children adopted from 19,000 Finnish mothers with schizophrenia between 1960 and 1979 were followed up. Their adoptive parents were assessed for child-rearing style, and the rates of schizophrenia were compared to those in a **control group** of adoptees without any genetic risk. A child-rearing style characterised by high levels of criticism and conflict and low levels of empathy was implicated in the development of schizophrenia but only for the children with high genetic risk but not in the control group. This suggests that both genetic vulnerability and family-related stress are important in the development of schizophrenia – genetically vulnerable children are more sensitive to parenting behaviour.

This is very strong direct support for the importance of adopting an interactionist approach to schizophrenia, including hanging on to the idea that poor parenting is a possible source of stress.

The original diathesis–stress model is over-simple

The classic model of a single schizogene and schizophrenic parenting style as the major source of stress is now known to be very over-simple. Multiple genes increase vulnerability to schizophrenia, each having a small effect on its own; there is no single schizogene. Also stress can come in many forms, including but not limited to dysfunctional parenting. Therefore vulnerability and stress do not have one single source.

In fact it is now believed that vulnerability can be the result of early trauma as well as genetic make-up, and that stress can come in many forms including biological. In one recent study by James Houston *et al.* (2008) childhood sexual trauma emerged as a vulnerability factor whilst cannabis use was a trigger. This shows that the old idea of diathesis as biological and stress as psychological has turned out to be overly simple.

This is a problem for the old idea of diathesis-stress but not for newer models.

Support for the effectiveness of combinations of treatments

There is support for the usefulness of adopting an interactionist approach from studies comparing the effectiveness of combinations of biological and psychological treatments for schizophrenia versus biological treatments alone. As Turkington *et al.* (2006) point out it is not really possible to use combination treatments without adopting an interactionist approach.

Studies show an advantage to using combinations of treatments for schizophrenia. For example, in one study by Nicholas Tarrier *et al.* (2004) 315 patients were **randomly allocated** to a medication + CBT group, medication + supportive counselling or a control group. Patients in the two combination groups showed lower symptom levels than those in the control group (medication only) although there was no difference in rates of hospital readmission.

Studies like this show that there is a clear practical advantage to adopting an interactionist approach in the form of superior treatment outcomes, and therefore highlight the importance of taking an interactionist approach.

A combination of genetic vulnerability and growing up in a dysfunctional family increases the risk of schizophrenia. Each on their own may not be problematic but they interact in such a way to produce mental health problems.

Apply it

Concepts: The interactionist approach

Whitney is in hospital having recently been diagnosed with schizophrenia. Her family are confused at the doctor's explanation that schizophrenia is an illness and his recommendation that she be treated by both antipsychotics and CBT.

Questions

Referring to the interactionist approach to schizophrenia, explain why Whitney's psychiatrist takes this view and recommends both medication and a psychological treatment.

Apply it

Methods: A survey of diathesis-stress

In the Houston *et al.* (2008) study 5877 participants responded to a survey; 543 reported a childhood sexual trauma and 643 reported using cannabis before the age of

Questions

1. Is the data above **quantitative** or **qualitative data**? Exp your answer. (*2 marks*)
2. Surveys can involve **questionnaires** or **interviews**. Expl *one* advantage of using a questionnaire to gather this d (*2 marks*)
3. (*a*) What is a **closed question**? (1 mark)
   (b) Why might closed questions be used here? (2 marks)
4. Explain *one* **ethical issue** with this study. (*2 marks*)

Evaluation eXtra

We don't know exactly how diathesis and stress work

There is strong evidence to suggest that some sort of underlying vulnerability coupled with stress can lead to schizophrenia. We also have well-informed suggestions for how vulnerabilities and stress might lead to symptoms. However, we do not yet fully understand the mechanisms by which the symptoms of schizophrenia appear and how both vulnerability and stress produce them.

Consider: *How does this incomplete understanding affect our evaluation of the diathesis-stress model?*

The treatment-causation fallacy

Turkington *et al.* argue that there is a good logical fit between the interactionist approach and using combination treatments. However, the fact that combined biological and psychological treatments are more effective than either on their own does not necessarily mean the interactionist approach to schizophrenia is correct. Similarly the fact that drugs help does not mean that schizophrenia is biological in origin. This error of logic is called the treatment-causation fallacy.

Consider: *Why does this matter? How does it affect our evaluation of the interactionist approach to explaining schizophrenia?*

CHECK IT

1. Explain the importance of adopting an interactionist approach to schizophrenia. [4 n
2. Outline the diathesis-stress model of schizophrenia. [6 n
3. Evaluate the diathesis-stress model of schizophrenia. [10 n
4. Describe and evaluate the interactionist approach to both explaining and treating schizophrenia. [16 n