The specification

Classification of Schizophrenia	• Positive symptoms, including hallucinations and delusions • Negative symptoms, including speech poverty and avolition
Diagnosis and Classification	• Reliability and validity in diagnosis and classification of schizophrenia, including references to co-morbidity, culture and gender bias and symptom overlap
Biological explanations	• Genetics • Dopamine hypothesis • Neural correlates
Psychological explanations	• Family dysfunction • Cognitive explanations, including dysfunctional thought processing.
Therapy as used in the treatment of schizophrenia	• Drug therapy: typical and atypical antipsychotics • Cognitive behavioural therapy • Family therapy • Token economies as used in the management of schizophrenia
Interactionist approach	• The importance of an interactionist approach in explaining and treating schizophrenia; the diathesis-stress model.

What is schizophrenia?

Schizophrenia is a serious mental disorder suffered by about 1% of the world population. People from all cultures and levels of society develop schizophrenia- it is the most common mental disorder accounting for up to 50% of all mental patients.

Schizophrenia affects thoughts processes and the ability to determine reality. Degrees of severity varies between sufferers: some encounter only one episode, some have persistent episodes but live relatively normal lives through taking medication, while others have persistent episodes, are non-responsive to medication and remain severely disturbed. Schizophrenia may be a group of disorders, with different causes and explanations.

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 classification of mental disorders, 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).**

- You **do not** need to know all the symptoms of the ICD and DSM (listed below in the table).
- However, you **do need to understand that these differ slightly** in their classification of the disorder. This becomes important when we consider the reliability and validity of classification and diagnosis in the next section.

ICD-10	DSM-5
Symptoms should be present for **most of the time** during an episode of psychotic illness **lasting for at least one month** (or at some time during most of the days). At least **one of symptoms listed below:** • Thought echo, thought insertion or withdrawal, or thought broadcasting. • Delusions of control, influence or passivity, clearly referred to body or limb movements or specific thoughts, actions, or sensations; delusional perception. • Hallucinatory voices giving a running commentary on the patient's behaviour, or discussing him between themselves, or other types of hallucinatory voices coming from some part of the body. • Persistent delusions of other kinds that are culturally inappropriate and completely impossible (e.g. being able to control the weather, or being in communication with aliens from another world). OR at least **two of the symptoms listed below:** • Persistent hallucinations in any modality, when occurring every day for at least one month, when accompanied by delusions (which may be fleeting or half-formed) without clear affective content, or when accompanied by persistent over-valued ideas. • Neologisms, breaks or interpolations in the train of thought, resulting in incoherence or irrelevant speech. • Catatonic behaviour, such as excitement, posturing or waxy flexibility, negativism, mutism and stupor. • "Negative" symptoms such as marked apathy, paucity of speech, and blunting or incongruity of emotional responses (it must be clear that these are not due to depression or to neuroleptic medication). ICD-10 recognises a range of subtypes of schizophrenia e.g. *paranoid schizophrenia* (powerful delusions), *hebephrenic schizophrenia* (primarily negative symptoms), *catatonic schizophrenia* (disturbance to movement-immobile or overly active).	To meet the criteria for diagnosis of schizophrenia, the patient must have experienced **at least 2** of the following: Delusions Hallucinations Disorganized speech Disorganized or catatonic behaviour Negative symptoms **At least 1 of the symptoms must be the presence of delusions, hallucinations, or disorganized speech.** Continuous signs of the disturbance must **persist for at least 6 months**, -during which the patient must experience at **least 1 month of active symptoms** (or less if successfully treated), -with **social or occupational deterioration problems** occurring over a significant amount of time. These problems must not be attributable to another condition. The American Psychiatric Association (APA) removed schizophrenia subtypes from the *DSM-5*.

The boxes above <u>do NOT</u> need to be learnt for the exam. They are there to show you the differences between the two classification systems.

Positive symptoms

Atypical symptoms experienced *<u>in addition</u>* to normal experiences

Hallucinations

These are <u>unusual sensory experiences.</u>

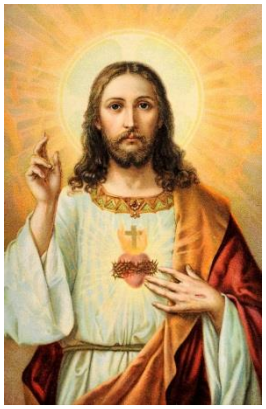
Some hallucinations are related to events in the environment whereas others bear no relationship to what the senses are picking up from the environment.

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 faces** or occasionally people or animals that are not there.

Delusions

Also known as <u>paranoia</u>, delusions are <u>irrational beliefs.</u> 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 or violence, some delusions can lead to aggression.

The DSM-5 places its emphasis on ***speech disorganisation*** in which speech becomes incoherent or the speaker changes topic mid-sentence.

Furthermore **disorganised thinking** can also be considered as a positive symptom for a sufferer of schizophrenia which can present as breaks or interpolations in the train of thought.
Catatonic behaviour may involve the sufferer performing strange positions and movements, or long periods of motionlessness. They may display rigidity or excessive movement. In cases where people experience excitability as a symptom, they may move in an erratic and extreme manner.

Negative Symptoms

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

Avolition

This can be defined as losing the will to perform the behaviours necessary to accomplish purposeful acts, such as activities of daily life, goals, and desires.

Can also 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 **very reduced motivation** to carry out a range of tasks and results in lowered activity levels, sometimes called 'apathy'.

Andreason (1982) identified 3 identifying signs of avolition;

- Poor hygiene and grooming
- Lack of persistence in work/education
- Lack of energy

Speech poverty (Alogia)

Schizophrenia is characterised by changes in patterns of speech.

Speech poverty can be defined as minimal verbal communication that lacks the additional unprompted content characteristic of normal 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.**

This is sometimes accompanied by a **delay in the sufferer's verbal responses** during conversation.

Characteristic of the symptom is the tendency only to speak when prompted, and to provide very limited answers.

For example, a person might respond to the question, "How did you feel when your mother yelled at you?" with "bad." When prompted to provide more

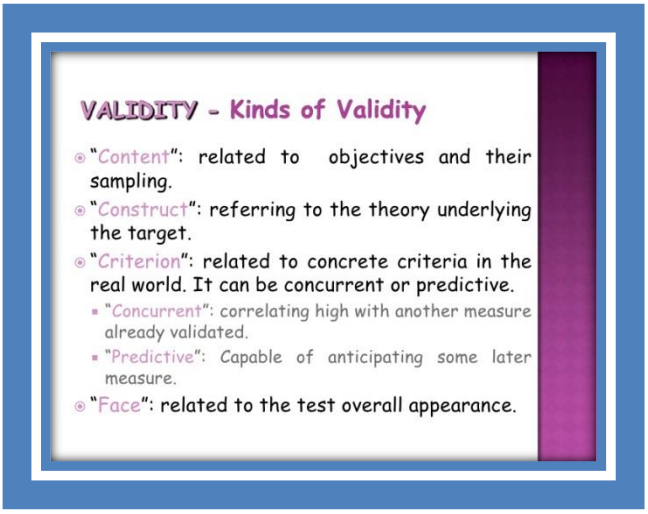
	information by a follow-up question, the responses would be similarly limited.

Reliability and validity in diagnosis and classification of schizophrenia

Reliability and validity in diagnosis and classification of schizophrenia

Classification systems such as DSM-V are worthless unless they're reliable. **Reliability** refers to the consistency of a classification system such as DSM e.g. to assess particular symptoms of schizophrenia. Reliability alone counts for nothing unless these systems and scales are also valid. **Validity** refers to the extent that a diagnosis represents something that is real and distinct from other disorders and the extent that a classification system such as DSM accurately diagnoses schizophrenia. Reliability and validity are inextricably linked because a diagnosis cannot be valid if it's not reliable.

Link to research methods (only need to know the last 3 types)



Validity

Validity is the extent to which we are measuring what we are intending to measure; in the case of schizophrenia it concerns how accurate the diagnosis is.

One standard way to assess validity of diagnosis is **concurrent validity (**amount of agreement between two different assessments)

Evidence investigating validity:

Cheniaux et al. (2009)

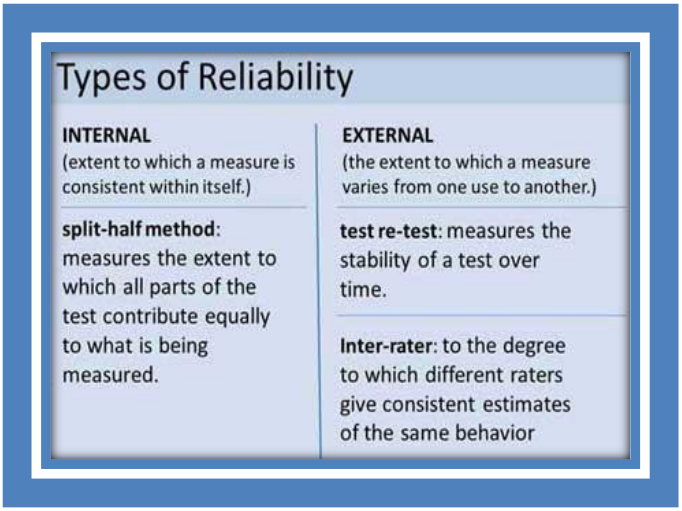
	Psychiatrist 1	Psychiatrist 2
DSM	26	13
ICD	44	24

Looking at the results from 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 highlights an issue with **concurrent validity**. Different assessment systems do not arrive at the same diagnosis.

Reliability

Reliability means consistency of symptom measurement - an important measure being inter-rater reliability; this is the extent to which different clinicians agree on their assessments.

In the case of diagnosis inter-rater reliability means the extent to which two or more mental health professionals arrive at the same diagnosis for the same patients.



Evidence investigating reliability:

Cheniaux et al. (2009) had two psychiatrists independently diagnose 100 patients using both DSM and ICD criteria.

	Psychiatrist 1	Psychiatrist 2
DSM	26	13
	Psychiatrist 1	Psychiatrist 2
ICD	44	24

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 evidence highlights weaknesses in the use of classification systems to diagnose schizophrenia. This is an issue as the external reliability is low as the psychiatrists failed to diagnose the patients consistently. This poor reliability is an issue for the diagnosis of schizophrenia.

HOWEVER, *It is important to note that Cheniaux research was carried out using the DSM-IV and not DSM-5 and e*vidence generally suggests that the reliability and validity of diagnoses has improved as classification systems have been updated.

Jakobson et al (2005) tested the reliability of the ICD-10 classification system during the diagnosis of schizophrenia. 100 Danish patients with a history of psychosis were assessed using operational criteria, finding a concordance between clinicians of 98 per cent, demonstrating the **high inter-rater reliability** of clinical diagnosis of schizophrenia using up-to-date classifications.

Even if reliability and validity of diagnosis based on classification systems is not perfect, they do provide clinicians with a common language, permitting communication of research ideas and findings, which may ultimately lead to a better understanding of the disorder. They can then predict the outcome of the disorder and aid in the development of effective treatments.

Symptom Overlap

Symptom overlap is the perception that symptoms of schizophrenia are also symptoms of other mental disorders.

Schziophrenia only	Schiz and bipolar	Bipolar only	Bipolar + depression	Depression only	Depression, schiz, bipolar
Disorganised speech	Delusions	Periods of mania	Depressed mood most of the day	Depressed mood most of the day, no mania	Inability to do everyday tasks
Affective flattening	Hallucinations	Alternating moods between depression + mania	Significant weight loss/gain		Difficult concentrating
Poverty of speech	Psychomotor disturbance	Excessive involvement in pleasurable activities	Insomnia/hypersomnia		Inability to feel pleasure in normal pleasurable activities
	Subjective experience- thoughts racing		Feelings of worthlessness		
	Inflated self esteem		Recurrent thoughts of suicide		

Despite the claim that the classification of positive and negative symptoms would make for more valid diagnosis of schizophrenia, many of the symptoms of the disorder are often found with other disorders, which makes it difficult for clinicians to decide which particular disorder someone is suffering from.

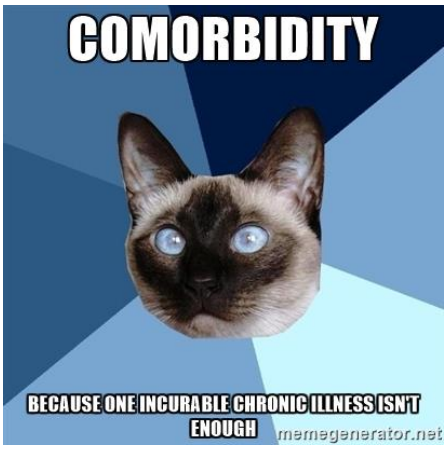
Read (2004) argued people diagnosed with schizophrenia have sufficient symptoms of other disorders that they could also receive at least one other diagnosis.

For example, **Symptom overlap** especially occurs with **bipolar disorder**, where negative symptoms e.g. depression and avolition are common symptoms, as well as positive symptoms e.g. hallucinations.

This highlights issues with the **validity** of trying to classify schizophrenia because a patient might be diagnosed as schizophrenic with the ICD, however, many of the same patients would receive a diagnosis of bipolar disorder according to DSM criteria.

A consequence of this issue could mean that individuals are misdiagnosed which can lead to years of delay in receiving relevant treatment, during which time suffering and further degeneration of symptoms can occur for the individual.

Co-morbidity



Co-morbidity is the phenomenon that 2 or more conditions occur together.

One issue which impacts the reliability and validity of a diagnosis is **comorbidity.** This is when two or more conditions occur together (e.g. Depression and Bipolar).

For example, Schizophrenia is commonly diagnosed with other conditions. **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%.

Therefore, comorbidity is an issue for the diagnosis and classification of schizophrenia. Different diagnoses could be given for the same person; in one instance they could be diagnosed with schizophrenia, diagnosed with bipolar in another instance or they could be diagnosed with both conditions. This issue could lead to inconsistencies in diagnoses between clinicians in relation to which disorder is diagnosed e.g. Schizophrenia or Depression, creating problems for the **reliability** of diagnosis.

Furthermore, comorbidity is also an issue for the classification of schizophrenia. Having simultaneous disorders suggests that schizophrenia may not actually be a separate disorder. A consequence is that it lowers the **(descriptive) validity** of schizophrenia, which can make effective treatment for schizophrenia difficult to achieve.

Culture bias in diagnosis

Culture bias concerns the tendency to over-diagnose members of other cultures as suffering from schizophrenia.

Culture bias is another problem which affects the **validity** of diagnosis. Although cross-cultural research of schizophrenia suggest a similar prevalence across races, research has shown that Schizophrenia, despite culturally formulated updates to diagnostic manuals, is repeatedly diagnosed at a higher rate in the African American population.

Research by **Cochrane (1977)** reported the incidence of schizophrenia in the West Indies and Britain to be similar, at around 1%, but that people of Afro-Caribbean

origin are 7 times more likely to be diagnosed with schizophrenia when living in Britain. Considering the incidence in both cultures is very similar this suggests that higher diagnosis rates are **not due to a genetic vulnerability**, but instead may be due to a cultural bias

Although there is not one explanation determining why African Americans are overrepresented. Two possible speculations are:

- Clinician bias - unconscious process stemming from stereotypes and biases which results in misdiagnosis (Schwartz, 2014).
- Under diagnosis of other disorders (Depression/Bipolar) in African Americans could contribute to the over-diagnosis of Schizophrenia.

Gara et al (2019) found that African American men with severe depression tend to be misdiagnosed with schizophrenia in comparison to other racial groups. The findings suggest that clinicians put more emphasis on psychotic than depressive symptoms in African-Americans, which skews diagnoses toward schizophrenia even when these patients show similar depressive and manic symptoms as white patients.

This is an issue as it suggests a lack of **validity** in diagnosing schizophrenia in people of African-American origin as differences in symptom expression are overlooked or misinterpreted by clinicians. The consequence of the misdiagnosis is that it prevents them receiving the optimal treatment for their disorder and puts them at risk of the side effects of medication taken for schizophrenia, such as diabetes and weight gain.

Gender Bias in diagnosis

The tendency for diagnostic criteria to be applied differently to male and females and for there to be differences in the classification of the disorder.

There is some disagreement between psychologists over the gender prevalence rate of schizophrenia. The accepted belief was that males and females were equally vulnerable to the disorder. However, some argue that clinicians (the majority of whom are men) have misapplied diagnostic criteria to women.

Long and Powell (1988) randomly selected 290 male and female psychiatrists to read cases studies of patients' behaviour and make a judgement on these people using standardised diagnostic criteria (e.g. DSM). When the patients were described as 'male' or no info about gender was given, 56% of psychiatrists have a schizophrenia diagnosis. When patients were

described as ‘female’, only 20% were given a diagnosis of schizophrenia. Interestingly, the gender bias was not as evident among the female psychiatrists, suggesting that diagnosis is influenced not only by gender of the patient but also the gender of the clinician.

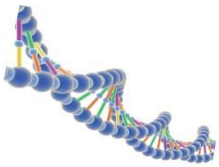
There is also gender bias in the fact that when making diagnoses, clinicians often fail to consider that males tend to suffer more negative symptoms than women (**Galderisi et al., 2012**) and women typically function better than men, being more likely to go to work and have good family relationships (**Cotton et al. 2009).**

This high functioning may explain why some women have not been diagnosed with schizophrenia when men with similar symptoms might have been; their better interpersonal functioning may bias clinicians to under-diagnose the disorder, 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.

These misconceptions could be affecting the **validity** of a diagnosis as clinicians are not considering all symptoms. This can be an issue and can lead to men and women who experience similar symptoms being diagnosed differently

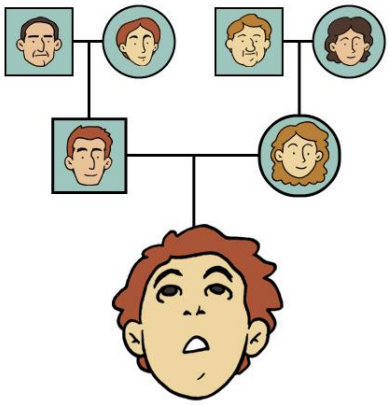
Clinicians also have tended to ignore the fact that there are different predisposing factors between males and females, which give them different vulnerability levels at different points of life, which may impact the validity of diagnosis. The first onset occurs in males between 18-25 years whereas, females between 25-35 years. This difference may be related to differences in the types of stressors both sexes experience at different ages and to age-related variations in female menstrual cycle, which tends to be overlooked during diagnosis.

Biological Explanations for Schizophrenia



There are several biological explanations for schizophrenia, which see the disorder as determined by **physiological means**. The biological factors focused on here are genetics, abnormal dopamine functioning and neural correlates. Although causes of schizophrenia are not fully understood, research does indicate that biological factors play a role in the development of the disorder.

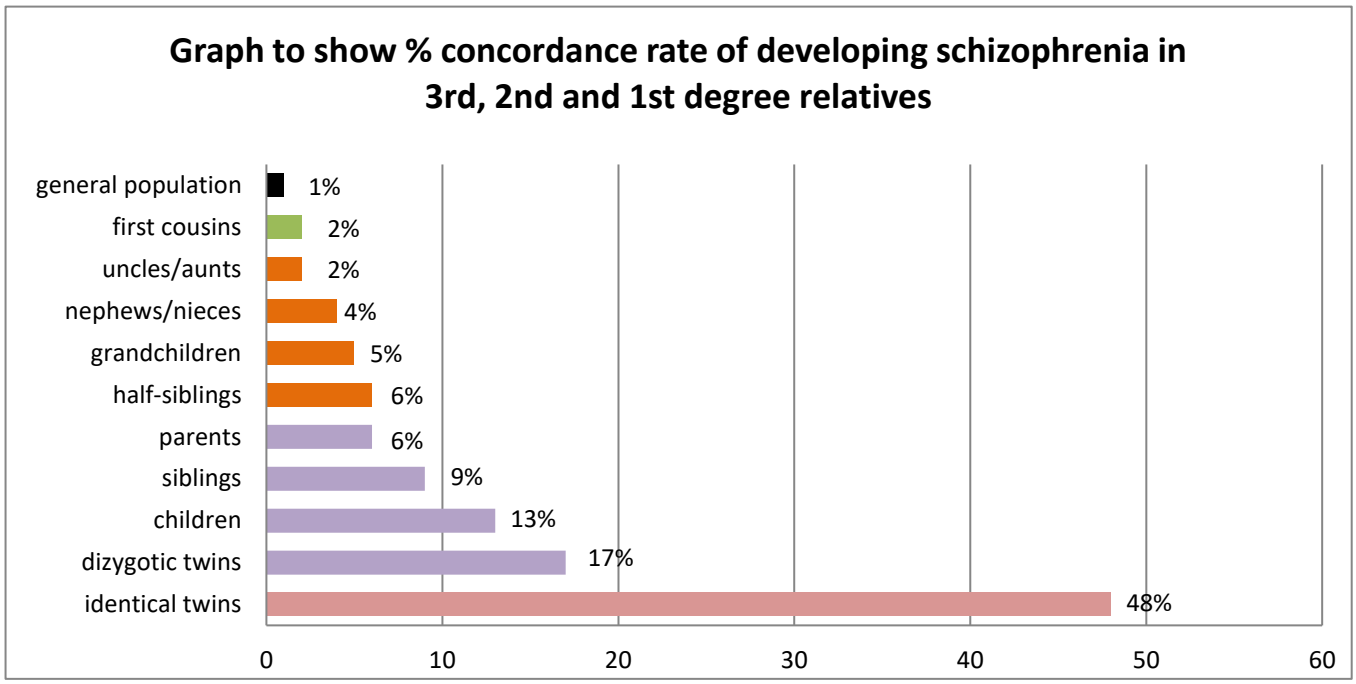
General Genetic Link Theory



The genetic explanation sees schizophrenia as transmitted through genes passed on to individuals from their families. We share a different % of genetics with our relatives depending on how genetically similar we are to them. For example, we share 50% of our genetics with 1st degree relatives e.g. parents, siblings (purple on graph). We share 25% with 2nd degree relatives e.g. grandparents, aunts/uncles (pink on graph) We share 12.5% with 3rd degree relatives e.g. cousins, great grandparents (green).

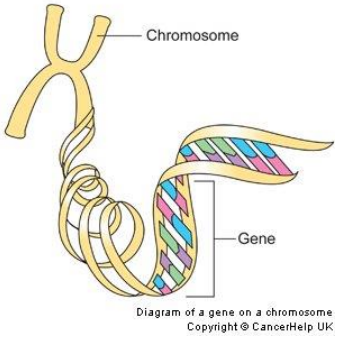
Investigations that look at the genetic similarity between family members and how it is associated with the likelihood of developing schizophrenia are good evidence for understanding the influence that genes play. However, we have to be careful when using this evidence as showing a genetic link because family members tend to share aspects of their environment as well as many of their genes (see evaluation).

Gottesman (1991) conducted a large-scale family study and found a strong relationship between the degree of genetic similarity and shared risk of schizophrenia. For example, 48% concordance rate in MZ twins in comparison to 17% in DZ twins.



More specific genetic explanation

It is not believed that there is a single 'schizophrenic gene', but that several genes are involved, which increase an individual's overall vulnerability to developing schizophrenia- this is a **polygenic** approach to schizophrenia 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 heterogenous,** i.e. *different* combinations of factors can lead to the condition.



Ripke et al. (2014) carried out a huge study combining all previous data from genome-wide studies (i.e. those looking at the whole 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 in the brain and in tissues with an important role in immunity, as well as those coding for functioning of a number of neurotransmitters including dopamine. This supports the overall idea of a biological causation in the disorder.

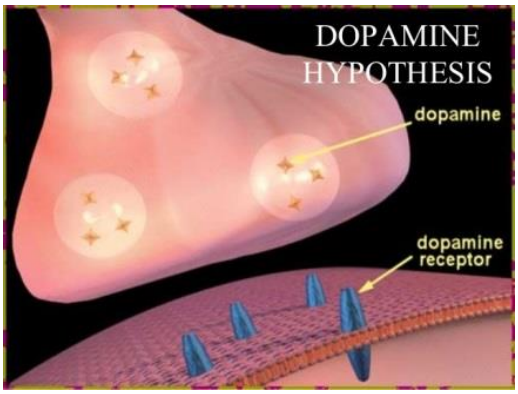
Evaluation of genetic explanations

Supporting evidence 	There is overwhelming evidence for the idea that genetic factors make some people more vulnerable to developing schizophrenia than others. **Kety and Ingraham (1992)** found that prevalence rates of schizophrenia were 10 x higher among genetic than adoptive relatives of schizophrenics, suggesting that genetics play a greater role than environmental factors. This is because the role of environment has been eliminated by looking at individuals who grew up away from their biological parents. So if the individual still develops schizophrenia this must be due to genes and not due to living with parents whose behaviour may have had an impact on development of the disorder.
Issues with research investigating biological explanations 	The research conducted to assess the relative contribution of genetics to the development of schizophrenia could be criticised for a number of reasons. For example, a crucial assumption underlying all twin studies is that the environment of MZ twins and DZ twins is equivalent. It's assumed, therefore, that the greater concordance for schizophrenia between MZ twins is a product of greater genetic similarity rather than greater environmental similarity.

	However, as Joseph (2004) points out, it's widely accepted that MZ twins are treated more similarly than DZ twins, encounter more similar environments (more likely to do things together) and experience more 'identity confusion' (frequently being treated as twins rather than 2 separate identities). As a result, Joseph argues there is reason to believe that the differences in concordance rates between MZ and DZ twins reflect nothing more than the environmental differences that distinguish the two types of twin. The issues we encounter in this research means the contribution of genes to schizophrenia can never truly be established.
Nature-nurture	P - The nature nurture debate is highly relevant in the discussion of the causes of schizophrenia. E - Genetic explanations would fall under the nature side of the debate, implying that schizophrenia is solely caused by genes inherited from the parents and therefore fails to consider the involvement or contribution from environmental factors e.g. family dysfunction or abnormal cognitive processes. E - However, this argument may be faulty as schizophrenia development cannot be entirely genetic in basis. Demonstrated by the evidence that concordance rates between MZ twins would be 100% if it was entirely genetic, which they are not. L - The **diathesis-stress model** may be a better way to explain the development of schizophrenia, where individuals inherit different levels of genetic predisposition, but ultimately it is environmental triggers that determine whether individuals go on to develop schizophrenia.

The dopamine hypothesis

The dopamine hypothesis



Dopamine is a neurotransmitter involved in initiating movement and has a major role in reward motivated behaviour

Dopamine (a neurotransmitter) is widely believed to work differently in the brain of a patient with schizophrenia and this may result in the symptoms observed in sufferers. It is probable that genetic factors are linked to faulty dopaminergic systems.

Original hypothesis	The revised dopamine hypothesis:
High levels of the neurotransmitter dopamine (**hyperdopaminergic activity**)	**Low** levels of the neurotransmitter dopamine (**hypodopaminergic activity**)
Found…. **mesolimbic pathway** and subcortex (central areas of the brain e.g. **Broca's area).**	Found…. **Mesocortical pathway** particularly the frontal lobes (pre-frontal cortex)
May be associated with the…. **Positive symptoms** of schizophrenia e.g. auditory hallucinations as well as poverty of speech	May be associated with the…. **Negative symptoms.** e.g. decision making
Why? This could be because a person has an increased number of **D2 receptors** in the brain (especially in subcortical areas such as the limbic system) or it is thought that messages from neurons that transmit dopamine either fire to readily or too often leading to the cha *The hypothesis that D2 receptors are somehow altered in schizophrenia is supported by genetics' findings that have shown a clear association between the DRD2 gene and schizophrenia. (Ripke et al., 2014).*	

Evaluation of dopamine hypothesis

Supporting evidence 	There is support from a number of sources for abnormal dopamine functioning in schizophrenia. **Curren et al. (2004)** found that when amphetamines, which activate dopamine production (agonists), are given to non-sufferers it can produce schizophrenia-like symptoms and make symptoms worse in those already suffering from schizophrenia. Equally, **Kessler et al. (2003)** used PET and MRI scans to compare people with schizophrenia with non-sufferers, finding that schizophrenics had elevated dopamine receptor levels in certain brain areas and differences in levels of dopamine in the cortex were also found. Both types of experimental research suggest an important role for dopamine in the onset in schizophrenia.
Issues with causation 	However, evidence for the dopamine hypothesis is still inconclusive and there are issues with establishing causation. The differences in the biochemistry of schizophrenics could just as easily be an effect rather than a cause of the disorder. **Lloyd et al. (1984)** believe that if dopamine is a causative factor, it may be an indirect factor mediated through environmental factors, because abnormal family circumstances can lead to high levels of dopamine, which in turn trigger schizophrenic symptoms. This research illustrates that we should be cautious in drawing firm conclusions about the direct role of dopamine in the development of schizophrenia.
Biological reductionism  	P - This theory can be criticised for being biologically reductionist. This is because it simplifies the complex development of schizophrenia to a single biological component, in this case the neurotransmitter dopamine. E - It could be the case that many other neurotransmitters are also involved in the development of the disorder. For example, much of the attention in current research has shifted to the role of a neurotransmitter called glutamate **(Moghaddam and Javitt, 2012),** as well as newer anti-psychotic drugs that also implicate serotonin's involvement too. E - This reductionist approach can be problematic because a variety of factors that may be involved in the development of the schizophrenia are being overlooked by isolating a single biological cause. However, taking a reductionist approach can also be very beneficial, in that it has helped to inform the development of drug treatments to treat schizophrenia. For example, anti-psychotic drugs that affect dopamine levels are the principal treatment offered to patients experiencing a schizophrenic episode and have been shown to be effective in reducing severity of symptoms. **(Thorney et al. 2003).** L -Thus, illustrating the usefulness of this explanation of schizophrenia, despite its reductionist nature.

Neural correlates of schizophrenia

The neural correlates explanation suggests that abnormalities within specific brain areas may be associated with the development of schizophrenia. Research uses non-invasive scanning techniques, such as fMRI, to compare the brain functioning of sufferers of schizophrenics and non-sufferers, to identify brain areas that may be linked to the disorder.

Participants are often given tasks associated with types of functioning known to be abnormal in sufferers, for example social cognition, thought processing and working memory tasks.

Negative symptoms

Early research (**Johnstone et al., 1976**) focused on schizophrenics having enlarged ventricles (fluid-filled gaps between brain areas).

Enlarged ventricles are especially associated with damage to central brain areas and the pre-frontal cortex, which more recent scanning studies have also linked to the disorder.

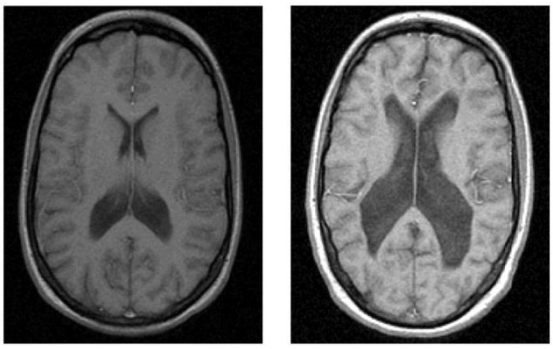


Fig. 1 Ventriculomegaly in discordant monozygotic twins seen on T_1-weighted MRI scans. Healthy twin (left) compared with twin with schizophrenia (right). With permission of Dr M. Picchioni.

Tomoyuki (2008) conducted a 10-year longitudinal MRI study on 15 patients and 12 controls (matched as closely as possible by age, gender and the duration of education) and found progressive ventricular enlargements in patients with schizophrenia but not in controls. Such damage has often been associated with the worsening of **negative symptoms** such as avolition and speech poverty but cannot explain all symptoms and incidences of schizophrenia.

Positive symptoms

Allen et al. (2007) found that positive symptoms also have neural correlates. They 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.

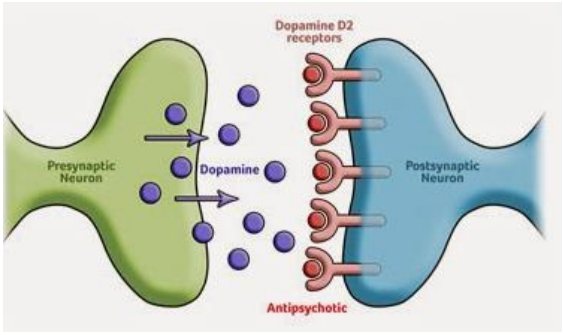
Evaluation of neural correlates

Supporting evidence 	There are a number of neural correlates of schizophrenia symptoms, including both positive and negative symptoms. The research helps to identify particular brain systems that might not be working normally. For example, **Tilo et al. (2001)** used fMRI scans to investigate the level of activity in the Wernicke brain area (an area associated with coherent speech) when schizophrenic and non-schizophrenic patients were asked to talk about a Rorschach ink-blot. They found that in schizophrenic patients the severity of their though disorder was negatively correlated with the level of activity in Wernicke's area. This supports the idea of abnormal functioning in specific brain areas being related to schizophrenic symptoms e.g. speech disorganisation.
Issue with causation 	A major limitation of the correlational research in this area of study is that we cannot establish **causation**; does the unusual activity in that region *cause* the symptoms of schizophrenia or does the disorder itself *cause* these brain differences? For example, it appears to be that people who have severe symptoms of schizophrenia and who do not respond to medication are the individuals who mainly exhibit enlarged ventricles (not all sufferers' do). This could mean that the physical brain damage (enlarged ventricles) is an effect of suffering from schizophrenia over a long period rather than brain damage leading to schizophrenia in the first place. The existence of neural correlates in schizophrenia therefore tell us relatively little in itself.
Challenging evidence 	Furthermore, there is scientific evidence to support this issue with causation conducted by **Ho et al. (2003)** They performed MRI scans on recent-onset schizophrenics and re-scanned them 3 years later. They found evidence of brain damage in the recent-onset patients, which worsened over time, especially in the frontal lobes, which correlated with an increase in the severity of their symptoms. **This suggests brain damage does increase in schizophrenics over time and may not be the initial cause of the disorder.** Consideration also needs to be given to the possible role of a third variable impacting on the relationships we see between brain abnormalities and the development of schizophrenia. For example, environmental factors such as substance abuse and stress levels may also be having a damaging influence upon brain tissue. More longitudinal research that assesses whether damage progressively worsens as the disorder continues is needed.

Biological therapies- Antipsychotic Drug treatment

The most common treatment for schizophrenia involves the use of **antipsychotic drugs.** Antipsychotics can be taken as tablets, in the form of syrup or by injection. Anti-psychotics may be required in the short-term or long term. Some people can take a short course then stop them without the return of symptoms and some people may be required to take them for life or risk recurrence of symptoms. They are divided into **typical** (traditional) and newer **atypical** drugs.

Typical antipsychotics- around since the 1950s



Typical antipsychotics e.g. **Chlopromazine** work by acting as **antagonists** in the dopamine system, in other words they **reduce the action** of a neurotransmitter.

Dopamine **antagonists** work by **blocking D2 receptors** in the synapses of the brain that absorb dopamine, thus reducing **positive symptoms** of the disorder, such as **hallucinations and delusions.**

Chlorpromazine is also an effective sedative and is often used to calm patients when they are very anxious, this may be because it **affects histamine receptors** (but it is not fully understood why it has this effect).

Typical antipsychotics tend to block all types of dopamine activity, (in other parts of the brain as well) and this causes side effects and may be harmful.

Atypical antipsychotics-used since the 1970s

The aim of developing new antipsychotics was to improve upon the effectiveness of drugs in suppressing symptoms and also to minimise **extrapyramidal side effects** (**EPSE**) (drug-induced movement disorders). There are a range of atypical antipsychotics and they work in different ways.

Atypical antipsychotics, such as ***Clozapine*** also acts on **dopamine receptors reducing positive symptoms**. In addition it acts as an **antagonist** for **serotonin** and an **agonist** (increasing the release of) for **glutamate** receptors and it is believed that this action helps **improve mood** and **reduce negative symptoms** in patients e.g **avolition** this may also **improve cognitive functioning** by reducing **disorganized thinking**. These benefits mean that it is sometimes prescribed when a patient is considered at high risk of suicide.

Risperidone is believed to bind to dopamine receptors more strongly than *clozapine* and is therefore more effective in much smaller doses than most antipsychotics and may lead to **fewer side effects.**

Notes:

Evaluation of drug therapy

Effectiveness – supporting evidence for typical anti-psychotics ✓	**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. There was also evidence from three trials that relapse rates were also lower when chlorpromazine was taken. Thus supporting the use of typical antipsychotics.
Effectiveness and appropriateness – supporting evidence for atypical anti-psychotics ✓	In addition, there is support for the benefits of atypical antipsychotics, particularly clozapine. **Herbert Meltzer (2012)** concluded that clozapine is more effective than typical antipsychotics and other atypical anti-psychotics, and that it is effective in 30-50% of treatment-resistant cases where typical antipsychotics have failed. This suggests atypical antipsychotics could be seen as a more effective drug therapy in comparison to typical antipsychotics as well as being a more appropriate drug treatment for certain individuals who do not respond well to other types of anti-psychotics.
Economic implications ✓	Drug therapies can also have positive economic implications. People who suffer from schizophrenia are often prevented from going to work and sometimes have to be hospitalised which has significant implications on the economy. Therefore, if anti-psychotics lead to symptom reduction they could enable individuals to return to work and/or could prevent them from having to be admitted to hospital which reduces the negative impact these factors have on the economy.
Appropriateness – side effects ✗	A problem with antipsychotic drugs is the likelihood of side effects, ranging from mild to serious. Typical antipsychotics are associated with a number of side effects including, dry mouth, constipation, lethargy and confusion, and long-term use can result in involuntary muscle movement, often facial muscles. This is called *tardive dyskinesia*, and is caused by dopamine oversensitivity. Atypical antipsychotics were developed to reduce the frequency of side effects and this has generally succeeded. However, side effects do still exist and are likely to include weight gain, cardiovascular problems, and an increased chance of developing diabetes. This can be a problem for two reasons, the first being it could lead to a reduction in quality of life if the symptoms are severe enough and secondly it can lead to sufferers stopping the treatment and therefore experiencing relapse.

Psychological explanations for schizophrenia

Please note: Psychological explanations include Behavioural and cognitive explanations.

Explanations of Schizophrenia include the following theories

- Family dysfunction, including double bind and expressed emotion
- Cognitive explanations, including dysfunctional thought processing

Family Dysfunction

Much evidence has now accumulated to suggest that like other mental health problems schizophrenia can be a reaction to stressful events and life circumstances. The family dysfunction explanation identifies sources of stress within families, which can cause or influence the development of schizophrenia.

These include:

- Maladaptive patterns of communication
- Experience of conflict
- High levels of criticism
- Controlling behaviours

Double Bind theory

This theory was proposed by **Bateson et al. (1972),** who believes that family climate is important in the development of schizophrenia and emphasises 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. The words spoken can present different meanings but also **the message** (words spoken) and the **meta-message** (way in which the message is transmitted through tone of voice and body language) can have different meanings. This double bind situation is also referred to a 'no-win' situation. When they 'get it wrong' (which is often) the child is punished by withdrawal of love. Prolonged exposure to such interactions may leave an individual with an understanding of the world as confusing and dangerous. The individual **loses touch with reality** and this is reflected in schizophrenic symptoms like **disorganised thinking** and **speech** and **paranoid delusions.** In some cases this type of treatment may result in auditory hallucinations for example a sufferer may hear voices telling them they are worthless

The role of family dysfunction in the onset of symptoms: Double bind

Certain families use maladaptive patterns of communication

↓

In a double bind (or 'no win') situation the message (words spoken) and the meta-message (transmitted in tone of voice and body language) have different meanings.

↓

For example a parent who says they love their child but appears constantly critical leaves their child in over where they stand leading to a false sense of reality

↓

These conflicts caused by the use of the double bind lead to schizophrenic symptoms of:

-, disorganised thought, paranoid delusions and hallucinations

↓

Bateson suggests that disorganised speech of the schizophrenic represents an attempt to not communicate in order to keep the social world at bay.

High Expressed emotion

Another feature of family dysfunction considers the role of **Expressed Emotion (EE)** where families may exhibit **criticism**, **hostility** **exaggerated involvement** and/or **control** and exert a negative influence on the sufferer. This is primarily an explanation for **relapse** in recovering schizophrenics. 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 due to their genetic make-up (diathesis-stress model). Where families show high expressed emotion including exaggerated involvement, control or criticism this has been found to increase the likelihood of **relapse.**

High expressed emotion from caregivers can lead to a child experiencing overwhelming emotion that can affect how the individual may respond to future stress and interpret new and challenging experiences and thus result in **paranoid thinking**. Furthermore individuals may dissociate or "mentally leave" as a result of high stress and trauma which can explain negative symptoms such as **speech poverty** and **avolition**. An unhealthy level of involvement and control could explain why the sufferer experiences **paranoia** about the world**.**

Vaughn and Leff (1976) showed that relapse rates were higher amongst patients who had been discharged into home environments which were higher in expressed emotion (EE).They distinguished between high EE families, where relapse rates were 51% compared to low EE families (13%). They also found that in high EE families the

likelihood of relapse correlated with the amount of time spent in contact with family members.

Evaluation of family dysfunction

Research support 	P- There is research evidence to support the role of family dysfunction in childhood and an increased risk of developing schizophrenia in adulthood. E **Tienari et al. (2004)** found that the level of schizophrenia diagnosed in adopted children of schizophrenic mothers was 5.8% for those adopted into healthy family environments. This increased to 36.8% for those children raised in dysfunctional families. This **supports** not only **the family dysfunction explanation** but also the idea that individuals with high genetic vulnerability to schizophrenia are more affected by **environmental stressors.** E **Butzlaff & Hooley (1998)** performed a meta-analysis of 26 studies to find that schizophrenics returning to a family environment of high expressed emotion experienced more than twice the average **relapse rate** of symptoms. L This research provides valuable support for the role of family dysfunction in the **onset** and **relapse** of those suffering with schizophrenia
Useful contribution 	The family dysfunction explanation highlights the importance of considering how social and **environmental factors** may **contribute** to the onset of the condition. The theory **emphasises the role** families can play in the onset and in relapse of Schizophrenia, which other explanations fail to consider. As a result, the theory has **contributed to an interactionist approach** to explaining the cause of the disorder which adopts a **more holistic** approach highlighting the complexity of the onset of symptoms and providing an explanation for **high relapse rates**. The theory and research also has **useful application** as it confirms that people's friends and families can be very important in helping their loved ones recover and has informed methods of **family therapy** which has been found to be successful in preventing relapse. People whose home atmosphere is supportive, calm and tolerant tend to do better. This demonstrates the theories value and the **useful contributions** the explanation has made to our understanding and explanation of schizophrenia.
Issues with causation 	The support for the role of family dysfunction can be criticised due to its correlational nature and its inability to establish the true **cause of schizophrenia.** It is very difficult to establish the direction of the relationship between environment and behaviour. Family dysfunction including **maladaptive communication may be the result** of the child's symptomatic behaviour rather than the cause of the illness. Furthermore there is ample theory and evidence to suggest there is a genetic and or neural cause to the disorder and that family dysfunction might act as a **contributing factor** or **trigger** for the condition; not be at the **root cause of the condition**. This **challenges** the support for the theory of family dysfunction and its **ability** to **explain the cause** of schizophrenia.
Social Sensitivity 	Adopting the family dysfunction explanation may have negative implications as it can be interpreted as **blaming the parents** of sufferer's for their child's development of the disorder. The theory suggests the cause of schizophrenia is the families' maladaptive communication and the home environment. As a result of this explanation parents of sufferers may then **feel responsible** for their child's illness. Furthermore, responsibility being placed on parents for their child's illness can cause even greater levels of stress and anxiety in the family which may in turn then trigger off or **exacerbate the illness.**

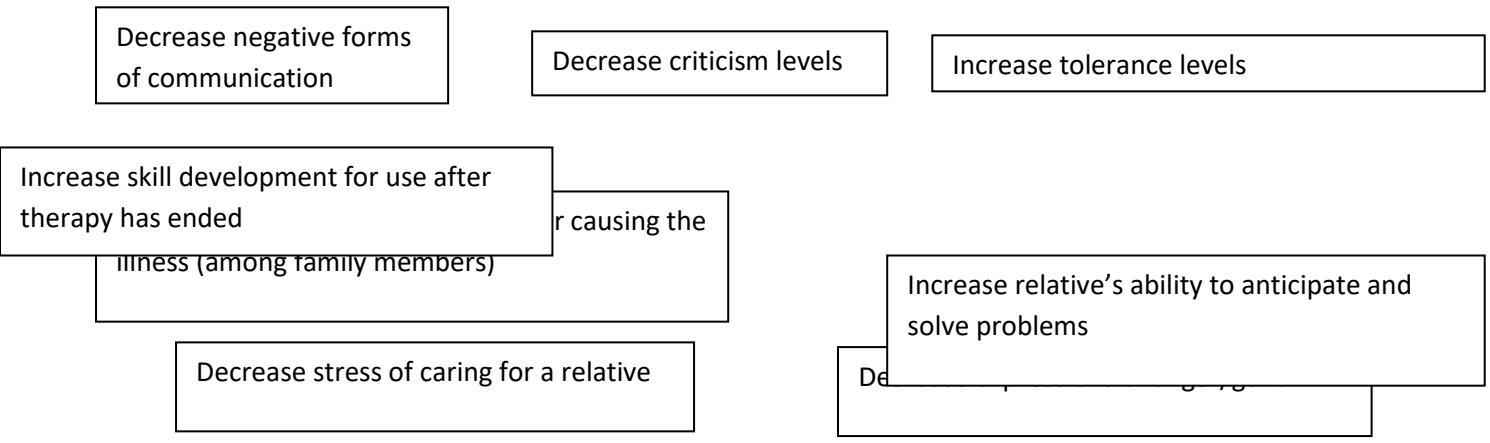
	A disadvantage of adopting this explanation therefore is its potential **negative implications** and furthermore the **social sensitivity** of the theory may mean that the theory is not widely researched or accepted by society.

Family therapy

Family dysfunction is known to play a role in the relapse rates of individuals with schizophrenia and potentially contribute to the development of the disorder as well. Family therapy is a form of psychotherapy that involves the whole family, including the family member with schizophrenia (if it is practical). A characteristic of schizophrenia is that individuals are often suspicious about their treatment and thus the benefit of involving the individual more actively in their treatment helps to overcome this problem and **reduce symptoms of paranoia.**

Family therapy's **main aim is to reduce the stress levels** in families to aid recovery for schizophrenia sufferers. Therapists meet regularly with the patient and family members, for usually between 9 months and a year.

Aims of the therapy



Linked directly to symptoms

- ➔ Altering **relationships and communication patterns** within dysfunctional families can help to **reduce instances of double bind** and the accompanying **stress** this can result in a **reduction in symptoms e.g. disorganised speech/thoughts.**

- ➔ **Lowering levels of expressed emotion** can help with reducing relapse rates. For example, during family therapy the relatives might be asked to set reasonable expectations for the individual with schizophrenia and set appropriate levels of involvement. This **reduces levels of exaggerated control** and involvement by the family, which in turn **decreases the stress** for the sufferer and **reduces symptoms like paranoid thinking** associated with high EE environments.

➔ The reduction in stress levels within the family can also **increase the chances of the patient complying with medication**. This combination of benefits tends to result in a reduced likelihood of relapse and re-admission to hospital.

Evaluation of family therapy

Effectiveness 	There is **reliable support** from research into the effectiveness of using family therapy to help reduce symptoms and prevent relapse in sufferers of Schizophrenia. **McFarlane et al. (2003)** reviewed available evidence to find that family therapy improved family relationships resulting in **symptom reduction** and **reduced relapse rates**, among family members, which leads to increased well-being for patients. This suggests that family therapy is **an effective treatment**, with an indication that **better family relationships** play a role in **symptom reduction**. Furthermore **Pharoah et al. (2010)** 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. This further demonstrates the therapies effectiveness in not only symptom reduction **but reducing the likelihood of relapse** and improving the quality of life for individuals suffering from the disorder.
Appropriateness  	Family therapy may be a more appropriate treatment for some sufferers than for others. Family interventions in **early psychosis** have been found to significantly reduced relapse and readmission rates. This treatment is particularly useful for **younger patients** who still live at home with their families who are also undergoing medical treatment and require support and for patients who lack insight into their illness or cannot speak coherently about it, as family members may be able to assist here and act as an advocate. Family therapy however may not always be an appropriate form of treatment for all sufferers and families. First, attending a family program conveys a series of **demands** to the sufferer and caregivers, such as transportation (which also implies money), time, **motivation**, and **energy**. Stigma can sometimes cause relatives to quit. **Severity of symptoms** may also prevent some sufferers from participating in family therapy and lead to high dropout rates. Therefore family therapy should be carefully considered in relation to the appropriateness for each individual and their family.
Economic implications 	An advantage of this therapy is the considerable **economic benefits.** **Family therapy** is often not widely available due to its time consuming and costly nature however the NICE review of family studies demonstrated that when implemented it was associated with **significant cost savings** when offered to service users in **combination** with other treatments such as drug therapy. The extra cost of the resource required for family therapy is offset by a reduction in cost through preventing the need for further and long lasting treatment. Family therapy has been found to reduce relapse rates and therefore prevent the cost for **further care which may require** e.g family therapy may prevent the individual requiring medication or in extreme cases hospitalisation.

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Cognitive explanations

A cognitive explanation for any phenomenon is one which focuses on the role of **mental processes**. According to this approach, the **cognitive impairments** shown by people with schizophrenia (e.g. poor attentional control; language deficits; disorganised thinking) play an important role in the development and maintenance of schizophrenia

Attentional Bias

Bentall (1994) proposed that people with schizophrenia have **deficits** and **biases** in the way they **process information**. This means there is an unusual **attentional bias** to stimuli of a threatening and/or emotional nature. For example, the content of **hallucinations** and the **delusions** regarding their origin may be understood in terms of **biased information processing**. **Paranoid delusions** may be a result of an individual **misinterpreting** an event as **threatening** due to an **exaggerated amount of processing** surrounding that experience or specific stimuli. For example if a person was cutting a cake with a knife... the knife is given too much focus and the individual becomes paranoid that the knife will be used as a weapon to harm them.

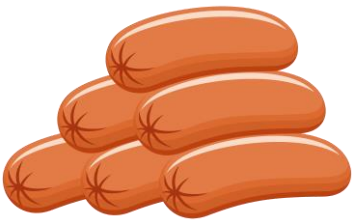
Dysfunctional thought processing

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

Metarepresentation is the cognitive ability to **identify** and reflect on **our own thoughts**, **behaviours,** emotions and experiences. A lack of or **dysfunction** in this **self-monitoring tool** would disrupt our ability to recognise our **own thoughts** and actions and **distinguish** them from the thoughts actions being carried out by others and therefore our own thoughts and ideas may be attributed to external sources. Frith suggested that this can explain the experience of auditory hallucinations. Also commonly experienced delusions such as of being controlled or persecuted can be explained by failures in metarepresentation and inability to make judgements about peoples intentions. Thought insertion (the experience of having thoughts projected into the mind by others) is a common delusion experienced by suffers which can also be explained through faulty meta-representation**.**

Central control is the cognitive ability to **suppress undesired automatic responses** while we **perform deliberate actions** that reflect our wishes and intentions. To be able to fit in to society's norms and expectations around public behaviour, central control enables us to suppress **stimulus driven behaviour** and **activate willed behaviour** and a dysfunction in central control may result in the display of behaviour seen as abnormal. If an individual has impaired central control then they are unable to control their **automatic response to any stimuli**. Specific language communicated to sufferers through conversation can trigger associations and memories that they would be unable to suppress their automatic responses to and **disorganised speech** and **derailment of thought** could present as a result. The inability to suppress automatic thoughts and speech can even be triggered by other thoughts explaining the experience of **paranoia** and **delusions**.

For example, if you lived with the symptoms of schizophrenia and you were having a conversation about what you did at the weekend but then you suddenly had a thought about forgetting to buy sausages, you would say that out loud because you cannot suppress the thought. For example, 'I had a really nice time with Harry last night. I must remember to buy sausages. He came over with some beers. There is a fly on the wall by the TV...' etc. The inability to suppress those thoughts leads to disorganised speech.



Evaluation of Cognitive Explanations

✓ **Research support**	P- There is strong support for the idea that information is processed differently in the mind of a sufferer of schizophrenia. Accounting for both positive and negative symptoms. E- **Stirling et al. (2006)** compared 30 patients with schizophrenia with 18 non-patients on a range of cognitive tasks including the **Stroop Test**, in which participants had to name the ink colours of colour words, suppressing the impulse to read the words in order to do this task. Sufferers took **over twice as long** as the control group to name the ink colour which would suggest that that the sufferers are therefore presenting with central control dysfunction. This research **supports Frith's theory** that dysfunctional thought processes including central control have a role in the cause of schizophrenia supporting cognitive explanation for the disorder.
✗ **Issues with causation**	Despite a large body of research supporting the link between symptoms and faulty cognition (proximal causes), the cognitive theories do not tell us anything about the **origins of those faulty cognitions** (distal causes). It may be the case that **structural brain abnormalities** lead to the differences in thought processes seen in symptoms of the disorder. For example research has found that some schizophrenics have **enlarged ventricles** in the prefrontal cortex and also that sufferers experiencing hallucinations have lower activation levels in the superior temporal gyrus. This would suggest that there is a **neural basis to cognitive symptoms** such as derailment of thought and language. The cognitive approach to schizophrenia therefore may be criticised as it does provide us with understanding about the underlying causes of dysfunctional cognitive processing and the symptoms experienced by sufferers. This would suggest Interactionist explanations using theories of cognitive neuroscience that consider biological and cognitive contributions to the disorder would be more effective in explaining onset. The **cognitive explanation** alone therefore can be found to be **limited** in its ability to provide a **complete explanation** of schizophrenia.

RED	GREEN	BLUE	YELLOW	PINK
ORANGE	BLUE	GREEN	BLUE	WHITE
GREEN	YELLOW	ORANGE	BLUE	WHITE
BROWN	RED	BLUE	YELLOW	GREEN
PINK	YELLOW	GREEN	BLUE	RED

 **Predictive validiy Positive contribution to effective treatment**	Cognitive treatments have been found to be **effective** which would further support the **validity of the explanation.** Research findings demonstrate that Cognitive Behaviour Therapy has a **significant effect in reducing both positive and negative symptoms** of schizophrenia through brief intervention programmes (Tarrier et al., 2005). For example CBT can help develop the functioning of meta representation through the sufferer challenging the origin of delusions and recognise the source of hallucinations. As such strategies adopted in the **therapy have been found to improve symptoms** this suggests the cognitive dysfunction is the cause of such symptoms The effectiveness of Cognitive treatments demonstrates the **predictive validity** of the cognitive **explanation** for schizophrenia

Cognitive behavioural therapy

CBTp (Cognitive behavioural therapy for psychosis) is a **structured talking therapy** for schizophrenia. The National Institute for Health and Care excellence (NICE) currently recommends CBTp for everyone with Psychosis. It usually takes place for between 5-20 sessions, either in **groups or an individual basis.**

The basic assumption of CBTp is that people often have distorted beliefs, which influence the feelings and behaviours in maladaptive ways, for example, someone with schizophrenia may believe that their behaviours is being controlled by someone or someone else. **Delusions** are thought to result from faulty interpretations of events, and CBTp is used to help the patient identify and correct these faulty interpretations.

The aim of CBTp is to help the patient make links between their cognition, emotions and behaviours and their symptoms. By monitoring this, they are better able to consider alternative ways of explaining why they feel and behave the way they do which reduces **distress and improves functioning** (e.g. how a particularly traumatic event has contributed to their **paranoid delusions)**

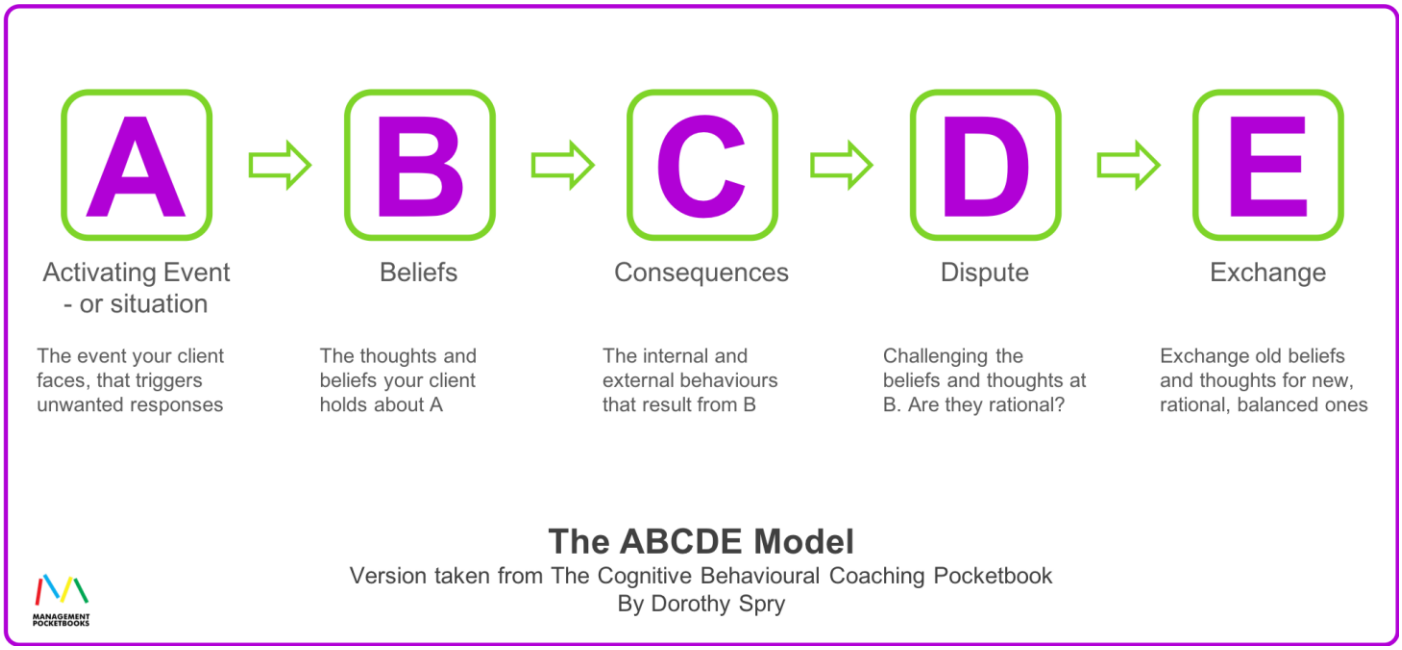
CBTp strategies include challenging beliefs (including origin of 'voices') and reality testing (to reduce distress) via the ABCDE framework.

Cognitive restructuring via ABCDE framework.

- Initial **Assessment** of patient's experiences.
- **Engagement** from the therapist
- **ABC model**
    - the patient gives their explanation of the activating events (A) that appear to cause their emotions, behaviours and beliefs (B).
    - Their beliefs which are actually the cause of the consequences (C) can then be rationalised, disputed (D) and changed.

For example through **reality testing,** the patient is encouraged to evaluate the content of their delusions and voices and consider ways to test their validity. The therapist will challenge the patient's **delusions** so that they can come to learn their beliefs are not based on reality. For example if they believe that they are being watched or followed the individual can identify that this is their internal belief, there is no evidence of this and control the way they respond to this future. This can also reduce negative symptoms like **avolition** as the sufferer is less like to **demonstrate avoidant behaviour.**

The therapist can also use strategies such as **normalisation** (placing psychotic experiences on a continuum with normal experiences (many people have unusual experiences such as hallucinations/delusions under different circumstances) to reduce alienation and stigma) and **critical collaborative analysis** (gentle questioning to help patient understand illogical deductions and conclusions) with the goal of helping the patient develop **alternative explanations** for their previously unhealthy assumptions.



Evaluation of CBT

Effectiveness – Supporting evidence ✓	There is evidence for the effectiveness of using CBT to treat schizophrenia For example, **Tarrier (2005)** reviewed 20 controlled trials of CBT using 739 patients, showing consistent evidence that CBT reduces persistent positive symptoms in chronic patients and may have modest effects in speeding recovery in acutely ill patients. This suggest CBT is viable treatment for schizophrenia, particularly for reducing positive symptoms such as delusions and hallucinations.
Effectiveness – Evidence could highlight the need for combination treatments 	However, **Jauhar et al. (2014)** performed a meta-analysis of 34 studies of CBT for schizophrenia. They concluded that CBT has a significant but fairly small effect on positive and negative symptoms. A potential reason for the small effect found could be due to CBT being investigated as a lone treatment. **Tarrier et al, (2000)** suggests that CBT plus antipsychotics is effective in treating schizophrenia and more effective than drugs or CBT alone. With this in mind, it may be more beneficial for CBT to be used as part of a combination treatment for schizophrenia.
Appropriateness – sufferers may not be able to engage with therapy ✗	CBT may not be an appropriate therapy for all sufferers of schizophrenia. This is because it relies on the individual to engage with the therapy and therapist, which may be especially difficult for those who are experiencing paranoia, or who are too disorientated or agitated to form trusting alliances with practitioners. Therefore, it is important to consider the individual sufferer when suggesting CBT as a treatment option as it may only be appropriate when the sufferer is in a position to engage in the process of CBT.
Appropriateness- Reduces distress ✓	Although psychological therapies like CBT do not help everyone, for others they can make a huge difference to their lives. Even where they do not reduce the frequency or intensity of their experiences of delusions or hallucinations, they often help reduce distress. They can also help people to find ways of achieving their goals and getting on with their lives even if their experiences (for example hearing voices) continue. Therefore, it is important to consider CBT as a treatment option for schizophrenia.

Token economies

Token economies are a **behaviourist** approach to the management of schizophrenia, where tokens are rewarded for demonstrations of desired behavioural change.

The technique is mainly used with long-term hospitalised patients to enable them to leave hospital and live relatively independently within the community. Similar programs have also been used in outpatient facilities.

Token economies are particularly **aimed at changing negative symptoms**, such as low motivation, poor attention and social withdrawal.

The technique uses **operant conditioning** principles, where patients receive **reinforcements** in the form of tokens **immediately** after producing a desired behaviour e.g. getting dressed in the morning, making the bed. The tokens can then later be exchanged for goods or privileges e.g. sweets, cigarettes, or a walk outside the hospital. The reward acts as the **primary reinforcer** and the token acts as the **secondary reinforcer.**

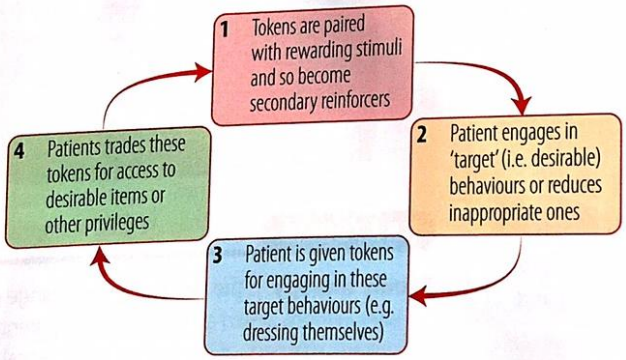
How does it work?

The behavioural principles employed in token systems are based mainly on the theory of operant conditioning. The principles of operant conditioning describe the relationship between a behaviour and environmental events. Key within this relationship is the idea of positive reinforcement, i.e. an increase in the frequency of a particular behaviour when it is followed by a desirable event.

There are two types of positive reinforcer:

- Primary reinforcers are anything that give pleasure (e.g. food or comfort) or remove unpleasant states (e.g. that alleviate boredom). Primary reinforcers do not depend on learning in order to acquire their reinforcing value.
- Secondary reinforcers initially have no value to the individual, but acquire their reinforcing properties as a result of being paired with primary reinforcers. In a token economy, the tokens given out when a patient engages in a target behaviour (e.g. taking care over their personal appearance or helping tidy up after a meal) are secondary reinforcers.

To be maximally effective, a reinforcer needs to be delivered immediately after the performance of the target behaviour. If the token does not follow immediately, then another behaviour (e.g. arguing with a fellow patient) may have been performed in the intervening period. It would then be this behaviour that is reinforced, not the target behaviour.



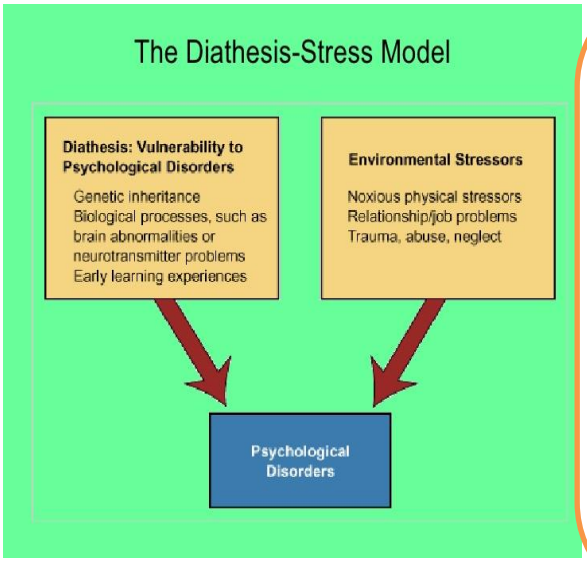
▲ The token economy cycle.

Evaluation of token economies

Effectiveness – limited evidence 	There is some evidence to support the effectiveness of token economy within a care setting. For example, one small study looking at token economy being used in a psychiatric hospital favoured the token economy approach with improvement in negative symptoms at three months. Therefore, this partially supports token economy as a way of managing schizophrenia and its ability to reduce symptoms. However, overall there is limited evidence to support the effectiveness of token economy at treating symptoms long term.
Effectiveness 	A problem with token economy is that the effects may not be maintained beyond the care setting. This is because desirable behaviour becomes dependent on being reinforced which means these rewards stop when individuals with schizophrenia are no longer under the care of the provision. This could lead to a relapse of symptoms for example, the sufferer may lose motivation which could lead to avolition reoccurring and may lead to high re-admittance rates.
Ethical concerns 	A criticism of using token economy in psychiatric institutions is that it raises ethical concerns. This is because token economies work on the principles of rewarding patients with every day pleasures, such as watching television and this could be seen as unethical as they are denying people with schizophrenia these pleasurable activities until they behave in a way the institution finds desirable whereas these activities are freely available outside of the institution. This raises serious ethical and moral questions as to whether token economy should be used as a way of managing symptoms of schizophrenia within a hospital setting.

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

The interactionist approach 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. **One example of an interactionist approach is the diathesis-stress model.**



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 the disorder but the onset of the condition is triggered by

In early diathesis-stress models, diathesis was seen as entirely genetic the result of a single 'schziogene'. However, it is now clear that many genes appear to increase genetic vulnerability slightly (**Ripke et al. 2014).** Also, modern views on diathesis also include a range of factors beyond genetic, including psychological trauma (Ingram and
Luxton 2005) so trauma becomes the diathesis rather than the stressor. Read et al (2001) proposed that early trauma alters the developing brain. Early and severe trauma, such as child abuse, can seriously affect aspects of brain development for example the HPA system (involved in biological stress response) can become overactive, making a person much more vulnerable to stress later on in their life.

In the original diathesis-stress model of schizophrenia, stress was seen as psychological in nature, in particular related to parenting. For example, family dysfunction of high expressed emotion where families exhibit criticism and hostility which can trigger the onset of schizophrenia for someone who is already vulnerable. However, modern definitions of stress include anything that is a risk for triggering schizophrenia e.g. substance abuse, psychological trauma.

In relation to treating schizophrenia, research indicates **combination treatments**, where more than one treatment is administered simultaneously to patients, are

generally most effective. This is reflective of the interactionist model of using both biological and psychological treatments.

Evaluation

The interactionist approach can be seen as a more appropriate and complete explanation of schizophrenia	**Tienari et al. (2004)** found that the level of schizophrenia diagnosed in adopted children of schizophrenic mothers was 5.8% for those adopted into healthy family environments. This increased to 36.8% for those children raised in dysfunctional families. This suggests individuals with high genetic vulnerability to schizophrenia are more affected by environmental stressors thus supporting the importance of using an interactionist approach to explain schizophrenia
The interactionist approach has highlighted the necessity for combination treatments.	**Tarrier et al. (2004)** randomly allocated 315 patients to a medication+ CBT group, medication+ supportive counselling group or a control group (medication only). Patients in the two combination groups showed lower symptom levels than those in the control group after 18 months, although there was no difference in rates of hospital readmission. This suggests using just biological treatments alone will lead to less successful treatment outcomes compared to combination treatments. This demonstrates the importance of adopting an interactionist approach in order to achieve superior long term treatment outcomes.
The original diathesis-stress model could be argued to be too simple.	This because it focused on a single gene (schizogene) as the diathesis and dysfunctional parenting as the major source of stress. However, multiple genes can increase vulnerability to schizophrenia rather than a single 'schizogene' and research suggests psychological trauma can also make some vulnerable to stress triggers. Also, stress can come in many forms e.g. parenting, family dysfunction, substance misuse. This suggests that there are a number of vulnerabilities and stressors that could be involved in the onset of schizophrenia therefore it is important to adopt an interactionist approach that considers the complexity of diathesis-stress.

Design a study exam question

It appears from previous research that students who have not studied psychology do not have a good understanding of schizophrenia.

Design an experiment to investigate students understanding of schizophrenia.

(12 marks)

Include in your answer:

- Variables and a suitable Operationalised hypothesis
- Sampling method- justification
- Data collection technique- justification
- How to analyse the data: the most appropriate descriptive and inferential statistics and justification

Practice short answer exam questions

1) Janelle has been diagnosed as suffering from schizophrenia. It began when she started hearing voices in her head criticising her behaviour and she became convinced that she'd been chosen by alien beings for a special purpose. Friends noticed that it became increasingly difficult to make sense of Janelle's speech and she would give only brief answers to their questions. She also became untidy and unenthusiastic about life in general, spending hours pacing up and down her room.

   Make reference to the scenario above concerning Janelle to identify negative and positive symptoms of schizophrenia. (4 marks)

2) Outline the neural correlates explanation for schizophrenia (4 marks).
3) Briefly outline the cognitive explanation of schizophrenia and explain one limitation with this approach (6 marks)
4) Briefly outline how cognitive behavioural therapy (CBT) is used to treat schizophrenia and explain one limitation of using CBT to treat schizophrenia. (4 marks)
5) Discuss the use of CBT to treat schizophrenia (6 marks)
6) Outline family therapy as used in the treatment of schizophrenia (6 marks)
7) Evaluate the use of family therapy as used in the treatment of schizophrenia, making reference to one other method of treatment (6 marks)
8) Outline two limitations of using token economies to manage schizophrenia (4 marks)
9) outline the diathesis-stress model for explaining schizophrenia' (6 marks)
10) Read the item and then answer the questions that follow.
    Louise comes from a family with a history of schizophrenia, as both her grandfather and an aunt have been diagnosed with the disorder. Louise's father has recently died from cancer and she has just moved out of the family home to start a university course. Although she has always been healthy in the past, she has just begun to experience symptoms of schizophrenia, such as delusions and hallucinations.

Using your knowledge of schizophrenia, explain why Louise is now showing symptoms of schizophrenia. (Total 4 marks)

11) Apart from effectiveness, briefly explain one limitation of drug therapy for schizophrenia. (Total 2 marks)

12) Discuss drug therapy as a method used in the management of schizophrenia. (Total 6 marks)

Essay questions

- Discuss token economies as a method used in the management of schizophrenia. (Total 8 marks)
- Outline and compare two treatments for schizophrenia. (Total 16 marks)
- 'There is considerable evidence that schizophrenia is caused by biological factors. These can be genetic, neuroanatomical, biochemical, viral or a combination of such factors'. Discuss biological explanations of schizophrenia. (Total 16 marks)
- 'Therapies can be time-consuming and, in some cases, uncomfortable for the client. It is, therefore, very important to offer the most appropriate and effective type of treatment.' Outline and evaluate two or more therapies used in the treatment of schizophrenia. (Total 16 marks)
- Outline and evaluate the dopamine hypothesis of schizophrenia (8 marks)

Essay planning

Outline and evaluate culture and gender bias in the diagnosis and classification of schizophrenia (8 marks)

Discuss reliability and/or validity in relation to the diagnosis and classification of schizophrenia. [16 marks]

Outline and evaluate one biological explanation for schizophrenia (8 marks).

Outline and evaluate the family dysfunction explanation of schizophrenia (16 marks)