### 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.

Notes:
<table>
<thead>
<tr>
<th>ICD-10</th>
<th>DSM-5</th>
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| 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 do NOT 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 in addition to normal experiences

<table>
<thead>
<tr>
<th>Hallucinations</th>
<th>Delusions</th>
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<tbody>
<tr>
<td>These are unusual sensory experiences.</td>
<td>Also known as paranoia, delusions are irrational beliefs. These can take a range of forms.</td>
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<tr>
<td>Some hallucinations are related to events in the environment whereas others bear no relationship to what the senses are picking up from the environment.</td>
<td>Common delusions involve being an important historical, political or religious figure, such as Jesus or Napoleon.</td>
</tr>
<tr>
<td>For example, voices heard either talking to or commenting on the sufferer, often criticising them.</td>
<td>Delusions also commonly involve being persecuted, perhaps by government or aliens or of having superpowers.</td>
</tr>
<tr>
<td>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.</td>
<td>Another class of delusions concerns the body. Sufferers may believe that they or part of them is under external control.</td>
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<td></td>
<td>Delusions can make a sufferer of schizophrenia behave in ways that make sense to them but seem bizarre to others.</td>
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<tr>
<td></td>
<td>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.</td>
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</tbody>
</table>

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.

Notes:

**Negative Symptoms**

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

<table>
<thead>
<tr>
<th>Avolition</th>
<th>Speech poverty (Alogia)</th>
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<tbody>
<tr>
<td><strong>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.</strong></td>
<td>Schizophrenia is characterised by changes in patterns of speech.</td>
</tr>
<tr>
<td>Can also be described as finding it difficult to begin or keep up with <strong>goal-directed activity</strong> i.e. actions performed in order to achieve a result.</td>
<td><strong>Speech poverty can be defined as minimal verbal communication that lacks the additional unprompted content characteristic of normal speech.</strong></td>
</tr>
<tr>
<td>Sufferers of schizophrenia often have <strong>very reduced motivation</strong> to carry out a range of tasks and results in lowered activity levels, sometimes called ‘apathy’.</td>
<td>The ICD-10 recognises speech poverty as a negative symptom. This is because the <strong>emphasis is on reduction in the amount and quality of speech.</strong></td>
</tr>
<tr>
<td><em>Andreason (1982) identified 3 identifying signs of avolition;</em></td>
<td></td>
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<tr>
<td>• Poor hygiene and grooming</td>
<td></td>
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<tr>
<td>• Lack of persistence in work/education</td>
<td></td>
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<tr>
<td>• Lack of energy</td>
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<td><img src="image" alt="Image" /></td>
<td><img src="image" alt="Image" /></td>
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<tr>
<td>This is sometimes accompanied by a <strong>delay in the sufferer’s verbal responses</strong> during conversation.</td>
<td>Characteristic of the symptom is the tendency only to speak when prompted, and to provide very limited answers.</td>
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<tr>
<td>For example, a person might respond to the question, “How did you feel when your mother yelled at you?” with “bad.”</td>
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Reliability and validity in diagnosis and classification of schizophrenia

Reliability

Reliability means consistency of symptom measurement - an important measure being inter-rater reliability; this is the extent to which different assessors 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:

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

<table>
<thead>
<tr>
<th></th>
<th>Psychiatrist 1</th>
<th>Psychiatrist 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>DSM</td>
<td>26</td>
<td>13</td>
</tr>
<tr>
<td>ICD</td>
<td>44</td>
<td>24</td>
</tr>
</tbody>
</table>

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 shows 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.

Soderberg et al. (2005) reported a concordance rate of 81% using the DSM classification system. The DSM classification system is regarded as more reliable than the ICD because the symptoms outlined for each category are more specific. For
example, **Nilsson et al. (2000)** found only a 60% concordance rate between practitioners using the ICD classification system, implying the DSM system is more reliable as the consistency is greater for the DSM than it is for the ICD.

Evidence generally suggests that the reliability of diagnoses has improved as classification systems have been updated. In addition, the reliability of diagnosis for schizophrenia is seen as superior to other disorders e.g. schizophrenia 81% concordance vs. 63% for anxiety disorders.

Notes:

**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 **criterion validity (predictive and concurrent)**.

Evidence investigating validity:

**Cheniaux et al. (2009)**

<table>
<thead>
<tr>
<th></th>
<th>Number of Sz diagnoses made:</th>
</tr>
</thead>
<tbody>
<tr>
<td>DSM</td>
<td>39</td>
</tr>
<tr>
<td>ICD</td>
<td>68</td>
</tr>
</tbody>
</table>

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. This shows that both the diagnostic systems lack concurrent validity meaning it is likely to lead to inaccurate diagnoses.

Validity can be also assessed using **predictive validity**- if diagnosis leads to successful treatment, then diagnosis is seen as valid.
Mason et al. (1997) tested the ability of 4 different classification systems of diagnosis to predict the outcome of the disorder (over a 13 year period) in 99 schizophrenic patients. They found more modern classification systems had high predictive validity, especially if only symptoms that lasted at least 6 months were considered. This suggests that predictive diagnosis has improved over time, as classification systems have been updated.

Notes:
The symptoms of schizophrenia are often found shared with other disorders, which makes it difficult for clinicians to decide which particular disorder someone is suffering from.

<table>
<thead>
<tr>
<th>Schizophrenia only</th>
<th>Schiz and bipolar</th>
<th>Bipolar only</th>
<th>Bipolar + depression</th>
<th>Depression only</th>
<th>Depression, schiz, bipolar</th>
</tr>
</thead>
<tbody>
<tr>
<td>Disorganised speech</td>
<td>Delusions</td>
<td>Periods of mania</td>
<td>Depressed mood most of the day</td>
<td>Depressed mood most of the day, no mania</td>
<td>Inability to do everyday tasks</td>
</tr>
<tr>
<td>Affective flattening</td>
<td>Hallucinations</td>
<td>Alternating moods between depression + mania</td>
<td>Significant weight loss/gain</td>
<td></td>
<td>Difficult concentrating</td>
</tr>
<tr>
<td>Poverty of speech</td>
<td>Psychomotor disturbance</td>
<td>Excessive involvement in pleasurable activities</td>
<td>Insomnia/hypersomnia</td>
<td></td>
<td>Inability to feel pleasure in normal pleasurable activities</td>
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<td></td>
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</table>

**Symptom overlap** especially occurs with **bipolar disorder**, where negative symptoms e.g. depression and avolition is a common symptom, as well as positive symptoms e.g. hallucinations.

Under the ICD a patient might be diagnosed with schizophrenia, however, the same patients may receive a diagnosis of bipolar disorder according to DSM criteria.

Therefore, we cannot be sure that a correct diagnosis is being made if the symptoms of schizophrenia overlap with other disorders. This is likely to lead to an inaccurate diagnosis.

In addition, this can lead to inconsistencies in diagnosis as different clinicians may diagnose schizophrenia and others may diagnose a different disorder which reduces the reliability of diagnosis.
Co-morbidity

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

This can create problems with reliability of diagnosis, as there may be confusion over which actual disorder is being diagnosed.

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%. This illustrates the difficulties in diagnosing schizophrenia.

In terms of validity, an individual could be given a diagnosis of schizophrenia when in fact they are suffering from schizophrenia and another condition. Also, as schizophrenia is commonly diagnosed with other conditions, this raises the question as to whether schizophrenia is actually a separate disorder. The issue of comorbidity could also lead to inconsistent diagnoses between clinicians in relation to which disorder is diagnosed e.g. Schizophrenia or Depression or both.

Extended evaluation- impact on treatment:

Jeste et al. (1996) state that schizophrenics with co-morbid conditions are excluded from research and yet they form the majority of patients, which suggests that research findings into the causes of schizophrenia cannot be generalised to most sufferers. This also has a knock-on effect as to which treatments patients should receive.

Alcohol, cannabis and cocaine are substances frequently abused by schizophrenics and not only does such co-morbid substance abuse make reliable and valid diagnoses of schizophrenia difficult to achieve, it also leads to lower levels of functioning, increased hospitalisations and lower compliance with medication, which makes effective treatment difficult to achieve.
Culture bias in diagnosis

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

In Britain, for example people of Afro-Caribbean descent are much more likely than white people to be diagnosed as schizophrenia. They are also more likely to be confined in secure hospitals than white schizophrenics.

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.

Alternatively, it may be that Afro-Caribbean people living in Britain experience heightened stress levels, from poverty and racism for instance, and that it is these factors that may contribute to higher levels of schizophrenia in such cultural groups.

Whaley (2004) believes the main reason for the incidence of schizophrenia among Black Americans (2.1%) being greater than among white Americans (1.4%) (incidence rates found by Keith et al, 1991) is cultural bias, where ethnic differences in symptom expression are overlooked or misinterpreted by practitioners. This suggests that the classification and diagnosis of schizophrenia lacks validity when being used cross-culturally.
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 two cases of ‘patients behaviour’ When the patient was described as male (or no gender) – 56% gave Sz diagnosis. When the patient was described as female - 20% diagnosis. When the psychiatrist was female no gender bias was found.

- 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.

- 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.

Notes:
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

<table>
<thead>
<tr>
<th>Supporting evidence</th>
<th>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.</th>
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<tbody>
<tr>
<td>Issues with research investigating biological explanations</td>
<td>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, family studies fail to consider the contribution of shared environmental influences on development of the disorder. It is logical to assume that 1st degree relatives living in the same household share similarities about their environment and therefore we cannot conclude that it is their genetic similarity that is causing higher concordance rates in family members. This argument can also apply to twin studies as MZ twins are often</td>
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</table>
treated much more similarly than DZ twins e.g. parents and schools interacting with them in the same way. This means that the higher concordance we see in MZ twins might not just be because they share 100% of their genetics but that they have also been treated in exactly the same way in their environments. The issues we encounter in this research means the contribution of genes to schizophrenia can never truly be established.

<table>
<thead>
<tr>
<th>Nature-nurture</th>
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<tbody>
<tr>
<td>P - The nature nurture debate is highly relevant in the discussion of the causes of schizophrenia.</td>
</tr>
<tr>
<td>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.</td>
</tr>
<tr>
<td>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.</td>
</tr>
<tr>
<td>L - The <strong>diathesis-stress model</strong> 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.</td>
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</table>
The dopamine hypothesis

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. Dopamine acts to increase the rate of firing in the synapse of neurons, which enhances the communication between neurons.

It is probable that genetic factors are linked to faulty dopaminergic systems.

<table>
<thead>
<tr>
<th>Hyperdopaminergic explanations (High levels)</th>
<th>Hypodopaminergic explanations (low levels)</th>
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<tr>
<td>The original version of the dopamine hypothesis focused on the possible role of high levels/activity of dopamine in the subcortex (central areas of the brain). For example, an excess of dopamine receptors in Broca’s area (responsible for speech production) may be associated with poverty of speech and/or the experience of auditory hallucinations.</td>
<td>The theory was updated because high levels of dopamine are not found in all schizophrenics. More recent versions of the dopamine hypothesis have focused instead on abnormal dopamine systems in the brains cortex. Goldman-Rakic et al. (2004) have identified a role for low levels of dopamine in the prefrontal cortex (responsible for thinking and decision-making) in the negative symptoms of schizophrenia.</td>
</tr>
</tbody>
</table>

It may be the case that both high and low levels of dopamine in different brain regions are involved in schizophrenia.

Notes:
## 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.

Such damage has often been associated with 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 thought 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 antipsychotics**

> **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 antipsychotics**

> 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 antipsychotics, 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 antipsychotics.

**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**

- Decrease negative forms of communication
- Decrease criticism levels
- Increase tolerance levels
- Decrease feelings of responsibility for causing the illness (among family members)
- Increase skill development for use after therapy has ended
- Increase relative’s ability to anticipate and solve problems
- Decrease stress of caring for a relative
- Decrease expressions of anger/guilt

**Linked directly to symptoms**

- **Alter**ing **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. |
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 sufferers 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; for example to carry on holding a hot plate whilst we carry it to the table. 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 e.g. shouting in the street. 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.

Evaluation of Cognitive Explanations
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 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.

### Predictive validity

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

CBT for psychosis is a structured talking therapy which looks at the way that people interpret, make sense of and react to their experiences. The National Institute for Health and Care excellence (NICE) currently recommends CBT for everyone with Psychosis. It usually takes place for between 5-20 sessions, either in groups or an individual basis. CBT helps sufferers understand their symptoms including their cognitions, emotions and behaviours and involves helping sufferers to identify and make links with previous experiences and how they have impacted the way they interpret and respond to the world e.g how being a particularly traumatic event has contributed to their paranoid delusions.

The aim and process of CBT will depend on the individual’s symptoms and their goals. The therapist will help the service user to identify what symptoms they want to explore.

Stage 1- Formulation

The therapist will work with the service user to create a formulation which is a diagram showing how events or experiences lead to thinking patterns and specific behavioural responses. Just understanding where symptoms come from can be hugely helpful for some as it can reduce the stress and anxiety associated with their experiences which in turn can help reduce positive and negative symptoms of schizophrenia.

Stage 2- Intervention

Service users are encouraged to actively engage with their psychotic experiences. Therapists help them interpret their delusions and hallucinations, their potential origin and how they are impacting on their feelings, thoughts and behaviours. They are encouraged to evaluate the content of their delusions and voices and consider ways to test their validity. Delusions are also challenged so that a patient 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 in the future. This can also reduce negative symptoms like avolition as the sufferer is less like to demonstrate avoidant behaviour.

Stage 3- Identification/ Consolidation of Skill

The service user can work with the therapist to identify their behavioural responses to their symptoms and challenge or adapt these to become more positive coping skills. Maladaptive coping strategies e.g avoidant behaviour or drug use behaviour to suppress hallucinations can be identified, challenged and replaced with
more **positive behavioural responses** e.g contacting a friend, going to the gym or writing the hallucinations down.

### Evaluation of CBT

<table>
<thead>
<tr>
<th>Effectiveness – Supporting evidence</th>
<th>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.</th>
</tr>
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<tbody>
<tr>
<td>Effectiveness – Evidence could highlight the need for combination treatments</td>
<td>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.</td>
</tr>
<tr>
<td>Appropriateness – suffers may not be able to engage with therapy</td>
<td>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.</td>
</tr>
<tr>
<td>Appropriateness – Reduces distress</td>
<td>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.</td>
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</table>
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**.
## Evaluation of token economies

<table>
<thead>
<tr>
<th>Effectiveness</th>
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<tbody>
<tr>
<td><strong>– limited evidence</strong></td>
<td>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.</td>
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<table>
<thead>
<tr>
<th>Effectiveness</th>
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<tr>
<td><strong>X</strong></td>
<td>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.</td>
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<table>
<thead>
<tr>
<th>Ethical concerns</th>
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<tr>
<td><strong>X</strong></td>
<td>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.</td>
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</table>
### 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.**

<table>
<thead>
<tr>
<th>Diathesis-Stress Model</th>
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<tbody>
<tr>
<td>Diathesis: Vulnerability to Psychological Disorders</td>
</tr>
<tr>
<td>Genetic inheritance</td>
</tr>
<tr>
<td>Biological processes, such as brain abnormalities or neurotransmitter problems</td>
</tr>
<tr>
<td>Early learning experiences</td>
</tr>
<tr>
<td>Environmental Stressors</td>
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<tr>
<td>Noxious physical stressors</td>
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<tr>
<td>Relationship problems</td>
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<tr>
<td>Trauma, abuse, neglect</td>
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</tbody>
</table>

| Psychological Disorders |

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 stress.

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**

<table>
<thead>
<tr>
<th>The interactionist approach can be seen as a more appropriate and complete explanation of schizophrenia</th>
<th>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</th>
</tr>
</thead>
<tbody>
<tr>
<td>The interactionist approach has highlighted the necessity for combination treatments.</td>
<td>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.</td>
</tr>
<tr>
<td>The original diathesis-stress model could be argued to be too simple.</td>
<td>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.</td>
</tr>
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</table>
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)