

# Schizophrenia revision & model answers

## Schizophrenia

In relation to their chosen disorder: **schizophrenia** or depression or anxiety disorders (either phobic disorders or obsessive compulsive disorder) candidates should be familiar with the following:

- Clinical characteristics of the chosen disorder Issues surrounding the classification and diagnosis of their chosen disorder, including reliability and validity
- Biological explanations of their chosen disorder, for example, genetics, biochemistry
- Psychological explanations of their chosen disorder, for example, behavioural, cognitive, psychodynamic and socio-cultural
- Biological therapies for their chosen disorder, including Psychological therapies for their chosen disorder, for example, behavioural, psychodynamic and cognitive-behavioural, including their evaluation in terms of appropriateness and effectiveness

This booklet contains revision notes (summaries) and model answers which have been written by students and edited to ensure that they are all grade A answers. Use them to plan your own essays! The essay are marked and the IDAs used in the essays are mentioned in the comments.

/ AO1 // AO2/AO3



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## Clinical characteristics

Schizophrenia (Sz) occurs in about 1% of the population across the world. The age of onset is commonly in late adolescence or early adulthood but it can develop in later life although rarely after the age of 45. It affects men and women equally but men tend to develop symptoms slightly earlier than women.

The symptoms (clinical characteristics) of Sz are divided in two categories:

**Positive symptoms:**

These are behaviours and features that non-schizophrenics do not experience

1. Delusions:
  - a. Delusions of grandeur: the sufferer believes he/she is someone important i.e. Jesus or has some special powers
  - b. Paranoid delusions: belief that others want to harm the sufferer
2. Hallucinations:
  - a. Auditory hallucinations: voices are heard which are mostly very critical or order the sufferer to do something.
  - b. Hallucinations can be from other senses: smell, visual
3. Thought insertion, withdrawal or broadcast
4. Incoherent speech: sufferers speech goes in every directions without following a logical and coherent thread, making it impossible for others to understand.
5. Catatonic behaviour: might be motionless for long periods of time. Repetitive and aimless behaviour (mannerism).

**Negative symptoms:**

Negative symptoms are associated with disruptions to normal emotions and behaviours.

1. Flatness of affect: the sufferer is apathetic (without enthusiasm) and talk without emotions.
2. Anhedonia: inability to enjoy pleasurable experiences.
3. Alogia: Poverty of speech which becomes less fluent. It is thought that this reflects "thought blocking" (when the person reports that the thought had been taken out of their head).
4. Avolition: Lack of ability to begin and sustain planned activities. This might lead a person to fail to take care of themselves i.e. lack of hygiene, poor diet.

January 2011

Outline clinical characteristics of schizophrenia. (5 marks)

This is a descriptive question (only AO1)

### Examiner's comments

A straightforward question, but many candidates wasted time scene setting, instead of outlining the clinical characteristics. Candidates should also be aware that evaluation **was not creditworthy**. When a maximum of 5 marks is available writing lengthy paragraphs is not a good strategy. However, in order to gain top marks, some degree of coherence is required and **candidates should avoid merely writing a list of the characteristics.**

## **Model answer**

The clinical characteristics of schizophrenia are divided into two groups: positive symptoms or negative symptoms. Positive symptoms are ones which are present in schizophrenics but not in 'normal' people, whereas negative symptoms can be found in people without schizophrenia but not in people with schizophrenia. /

Hallucinations are positive clinical symptoms of schizophrenia and can be auditory or visual. Visual hallucinations involve the person seeing things or people which other people don't see, so aren't actually real. / Auditory hallucinations are when voices can be heard inside the persons head or as if being said by an actual person. These voices may be threatening and abusive or order the sufferer to do something. Up to 70% of schizophrenics experience auditory hallucinations so it's a very common symptom. /

Another positive clinical characteristic is delusions of grandeur. It is when an individual has a strong and mistaken belief that they have an important mission, are in a position of power or are someone very important. For example, a schizophrenic may claim that he is Jesus. Schizophrenics refuse to take any evidence against their belief as disproving it. /

A final clinical characteristic is Anhedonia, this is a negative clinical characteristic. The person may no longer enjoy or look forward to doing activities they used to enjoy such as sports. This interest is unlikely to be diverted to another activity. The sufferer does not take any pleasure in what he/she used to feel were pleasurable experiences. /

**245 words. The symptoms are clearly explained. The explanation is structured around the division between the positive and the negative symptoms showing depth of knowledge and examples are given showing that the students can apply their knowledge.**

## Issues surrounding the diagnosis of schizophrenia

The issues discussed must relate to the validity and reliability of the diagnosis.

Two main systems are used to diagnose Sz:

The Diagnostic and Statistical Manual of Mental Disorder (Edition 5), was last published in 2013. The DSM is produced by the American Psychiatric Association. It is the most widely used diagnostic tool in psychiatric institutions around the world.

The International Statistical Classification of Diseases (known as ICD). It is produced by the World Health Organisation (WHO) and is currently in its 10th edition.

### Reliability:

**Inter-rater reliability:** if two psychiatrists see the same patients independently they should give the same diagnosis.

**Test-retest reliability:** are the tests used to diagnose the disorder consistent over time?

**Validity:** Does schizophrenia exist and is it a disorder separate from other disorders?

Validity and reliability are linked because if a diagnosis is not reliable then it cannot be valid.

## According to DSM 5

- To be diagnosed as schizophrenic symptoms must be present for six months and include at least one month of active symptoms.
- DSM-4 required one symptom whereas DSM 5 requires that an individual exhibit at least two of the specified symptoms. This presents an issue of time validity as an individual might have been diagnosed as Sz with DSM 4 but might not be diagnosed with the same disorder with DSM 5 (time validity)
- The subtypes are no longer used as it was found that symptoms changed, patients presented overlapping subtype symptoms and the division between the different subtypes was blurred therefore decreasing their validity.

## According the ICD 10

- The symptoms are similar but they only have to be present for 1 month
- ICD has 9 subtypes of schizophrenia

An individual might be diagnosed as schizophrenic when using the ICD 10 as he/she has presented symptoms for one month but not according to the DSM 5 as the period is less than 6 months. Furthermore the ICD requires only one symptom to be present whereas the DSM requires two symptoms.

A true diagnosis cannot be made until a patient is clinically interviewed. Psychiatrists rely on retrospective data, given by a person whose ability to recall much relevant information is unpredictable as some may be exaggerating the truth whereas others might be afraid of a diagnosis and not report some symptoms like hearing voices. However clear the diagnostic tool, the behaviour of an individual is always open to some interpretation. The process is subjective.

The psychiatrist uses his/her own life experience to interpret the patients' behaviour this might lead to misunderstanding because of cultural, social, sexual differences. This could explain why Afro-Caribbean individuals are more likely to be diagnosed schizophrenics than UK residents. It could also explain why people from lower social classes are also more likely to be diagnosed. However it could be that these people are more likely to develop Sz because they are more subject to stress because of discrimination or poverty.

This also could explain the low reliability of diagnosis i.e. Beck et al (1961) looked at the inter-rater reliability between 2 psychiatrists when considering the cases of 154 patients. The reliability was only 54% - this means they only agreed on a diagnoses for 54% of the 154 patients. However both the DSM and the ICD have changed many times since then (1961) so the findings might be different now (time validity). But Whaley (2001) found inter-rater reliability correlations in the diagnosis of Sz as low as 0.11. This suggests that the reliability of diagnosis has not improved in 40 years despite several updates of the two systems.

However Rosenhan questioned the validity of diagnosis as he showed that even sane individuals could be diagnosed as schizophrenic. However the classification systems have been updated many times since the study so the results might be different now. Furthermore psychiatrists do not expect people to present themselves faking symptoms and

they have a duty to protect patients from harming themselves and others so "better safe than sorry".

Diagnosing an individual with Sz has serious consequences and neither of the classifications take into account the severity of the symptoms.

The beliefs and biases of some might mean the unnecessary labelling of millions of people as sufferers of a mental disorder. Someone who has suffered a mental disorder has to disclose that information in situations such as job interviews, or they could face formal action and schizophrenics risk carrying the stigma of their condition for the rest of their lives. But labelling can be useful; the diagnosis can have positive consequences as the patient can then get support and treatment.

Meehl (1977) suggests that mental health professionals should be able to count on the diagnostic tools if they paid close attention to medical records, were serious about the process of diagnosis, took account of the very thorough descriptions presented by the major classificatory systems and considered all the evidence presented to them.

But in the real world there is limited time and resources available to many professionals working in the National Health Service and diagnoses are made by professionals that are rushed, and preoccupied with only admitting the most serious cases in order to safeguard the resources of the institution they are working for.

However the validity of the diagnosis can also be questioned because not only do schizophrenics rarely share the same symptoms but the outcome is also very varied. A review of 25 independent studies found that 10 years after an initial schizophrenic incident, 25% of schizophrenics were completely free of any symptoms, another 25% are "much improved" , living independently. After 30 years of the initial onset, the number of these "much improved" cases increases to 35%. 10 % with an initial diagnosis of schizophrenia still require hospitalisation after 30 years. The fact that the outcome can be so different puts in question the validity of the diagnosis. Are these people suffering from the same disorder?

**An essay on this topic must focus on the reliability and validity of the diagnosis**

*These are the points in the marking scheme however you can use others as long as you link them to the issues of reliability and validity,*

- The reliability of the major classification systems (ICD and DSM)
- The lack of homogeneity in schizophrenic symptoms
- Symptom overlap and the inclusion of mixed disorder categories (schizo - affective disorder ) by classification systems
- The problem of co-morbidity with depression
- The availability of other diagnostic criteria for schizophrenia eg Schneider criteria
- Cultural differences in symptom presentation
- The lack of objective tests for schizophrenia
- The difficulty of being able to predict outcome or response to treatment
- The question of whether schizophrenia is a mental disorder at all or a form of political control

January 12

**Discuss issues associated with the classification and/or diagnosis of schizophrenia.  
(8 marks + 16 marks)**

### **Examiner's comments**

AO1 credit was awarded for the identification/description of issues relating to classification and diagnosis, most of which can be placed under the headings of reliability and validity. Weaker answers often showed little evidence of organisation or planning with students producing long lists of clinical characteristics without identifying issues relating to classification or diagnosis. This approach gained rudimentary AO1 credit.

A lack of focus on the question was also notable for AO2/3 with weaker students. Many focussed almost exclusively on Rosenhan's 1973 study 'On being sane in insane places' often providing lengthy and detailed description without linking this to an issue related to classification or diagnosis. Weaker students also focussed on methodological evaluation of this research study, which was of limited relevance to the question. There was little recognition that Rosenhan's study is over 40 years old and changes have taken place to classification and diagnosis since then.

Stronger students approached the question by identifying an issue (such as the lack of reliability between ICD and DSM IV) then considering possible consequences of this and/or research evidence regarding reliability of diagnosis using the respective systems. There was some useful discussion of the problems of co-morbidity, cultural differences and Szasz's critique of the myth of mental illness in better answers. Higher AO2/3 marks went to students who evaluated each issue as they went through the essay. Those students who were able to consider a range of research evidence relating to reliability and validity were also rewarded.

### **Model answer**

The issues surrounding the diagnosis of schizophrenia focus on reliability and validity.

The DSM and the ICD are the two main diagnostic classification systems. The DSM (Diagnostic and Statistical Manual of Mental Disorders) was developed by the American Psychiatric Association, it is used throughout America and some parts of Europe, its aim is to help professionals with diagnosing mental disorders. The ICD (international Classification of Diseases) was developed by the World Health Organisation in order to collect health statistics from around the world. /

Both these systems are based on the assumption that mental disorders, like physical disorders, can be separated from each other by symptom patterns. / However the difficulty is that whereas the diagnosis of physical disorders can be based on objective tests like blood tests or imaging there are no such tests for mental disorders. The diagnosis is based on interviews and observations which means that they are likely to be subjective. //

Furthermore the two systems are slightly different in the criteria they use which means that in a country which uses one system an individual might be diagnosed with schizophrenia (Sz) but the same person might not be diagnosed in the same way in a country using the other system, this therefore shows a lack of reliability. //

The two systems use different criteria. The DSM requires that an individual presents at least two symptoms (i.e. hallucinations and delusions) for at least six months whereas the ICD requires the patients to present one symptom for only one month. This puts in question not only the reliability of the diagnostic but also its validity because if the criteria are so different are they diagnosing the same disorder? //

Additionally both classification systems are updated regularly which means that an individual might be diagnosed as schizophrenic at a point in time but might no longer be diagnosed as schizophrenic after an update. This again questions the validity and reliability of the diagnosis. //

Furthermore, it could be argued that the DSM is culturally biased as it was created by Americans for Americans. This may be an issue as behaviour in one culture may not be regarded as a symptom of schizophrenia but according to the DSM it is. For instance, hearing voices in some cultures is considered to be a message from the Gods and is regarded as an honour not a symptom of a mental disorder. This could be a problem for the reliability as an individual could be diagnosed as schizophrenic in one country but not in another country. //

The ICD however is less culturally biased than the DSM as it is published by the World Health Organisation and involves professionals from 193 countries and therefore various cultures are represented so it can be applied to more diverse cultures. //

The ICD classification system appears to offer some advantage over the DSM classification. Firstly, with the symptoms only needing to be present for one month as opposed to six with the DSM, sufferers do not have so much time in which they may be at risk to themselves and others. They also only have to live without help for one month before receiving diagnosis and therefore appropriate treatment. //

The validity of the subtypes of Sz described in the ICD is also questionable as the latest edition of the DSM abolished these classifications as patients did not seem to fit exactly the criteria and changed over time. //

One of the subtypes of Sz recognised by the ICD is undifferentiated schizophrenia, it is essentially a 'rag bag' for sufferers whose symptoms are hard to classify. Patients can have a wide range of symptoms and therefore two patients classified as undifferentiated schizophrenics may not have any symptoms in common. This puts in question the validity of this classification. //

However Jacobsen et al (2005) used the operational criteria checklist (OPCRIT) which is used for diagnosing schizophrenia. They found there was good agreement between the OPCRIT and the ICD when diagnosing patients. There was also good agreement between the DSM and the ICD and so there seems to be high reliability. //

Comorbidity is also an issue when diagnosing schizophrenia. Comorbidity is when a patient is suffering from two or more mental disorders at one time. Many Schizophrenics also suffer from other disorders such as depression and bi-polar disorder. Comorbidity occurs in part because the symptoms of different mental disorders often overlap with each other. /

Sim et al (2006) studied 142 hospitalized schizophrenics. 32% of whom had an additional mental disorder. Those with comorbidity had less awareness of their condition and poorer outcome than those without. The reliability can be questioned as we are unable to tell whether the low motivation reflects the existence of depression or schizophrenia or both and is a problem because comorbidity appears in such a large percentage of sufferers. // It also raises the questions as to why these are thought to be two different disorders when they have many common symptoms.

The continuum approach suggests that there's no sharp dividing line between individuals with schizophrenia and those without. It argues that we all experience symptoms which could be interpreted as symptoms of schizophrenia for example hearing the voice of someone close who has recently died. // It further suggests that what prompts a diagnosis is the severity of the symptoms rather than their existence. This then raises a question of validity as it suggests that schizophrenia is really a normal part of human experience rather than a mental disorder. //

This approach has been tested using questionnaires that include items alluding to some of the positive and negative symptoms. The questionnaires assess schizotypy which is a proneness to psychosis (especially schizophrenia). Chapman et al (1994) research supports this theory as they found that apparently normal individuals who had high scores of schizotypy were more likely to develop Sz, this suggests that there's no real dividing line between sufferers and non-sufferers. //

Once patients have been diagnosed they are then labelled as schizophrenic. Those who have suffered a mental disorder must disclose this information when applying for jobs as the label stays with a person throughout their life and they therefore risk carrying the stigma of their condition. / However one advantage to being diagnosed is the feeling of relief that patients may feel if they have been particularly scared or anxious as they finally have an explanation as to what has been going on in their lives. // This is also able to lead them to proper effective treatment that may enable them to live a normal functioning life. //

However, it can be damaging as sufferers have to live with the attached label for the rest of their lives. This may go against them and lead them to unemployment and poverty. // Furthermore labels may also lead to a self-fulfilling prophecy where people are treated in a certain way that elicit the expected behaviour. // Moreover their behaviour is interpreted in the light of the label and what would be considered eccentric will now be seen as evidence which confirm the label. //

Rosenhan's 1973 study has also thrown validity and reliability into question in his study "on being sane in insane places", he concluded that 'it is clear that we cannot distinguish the sane from the insane is psychiatric hospitals'.

Rosenhan used a range of different hospitals in his experiment using hospitals from all different states, both private and federal, old and new and so results can be generalised. // However the research is 40 years old and classification systems have been updated many times since then, for example the patient would have to present active symptoms for one month before being diagnosed

according to the ICD. Furthermore, Kety (1974) argued that just because someone is lying about their symptoms does not mean a psychiatrist does not know how to diagnose a patient if the portrayal of symptoms is convincing then there is no reason for doubt. // Despite their obvious weaknesses the classification systems have led to an improvement in the reliability of diagnosis which has benefitted the sufferers and society as it allows appropriate treatment and support which allow a great majority of schizophrenics to resume a fairly normal and productive life. //

*This essay is very well written but just over the word limit. (1370 words). There is more material than necessary with 26 AO1/AO2 when only 24 marks were available.*

*The points made are developed appropriately and examples are given to clarify the explanations.*

*IDAs: cultural bias in diagnosis, time validity, application of knowledge to real life (last paragraph)*

**Biological explanation of schizophrenia**

<b>Biological explanation of schizophrenia</b>			
<b>GENETIC EXPLANATIONS</b>	<b>A01 Explanations/Research</b>	<b>Strengths</b>	<b>Weaknesses</b>
	<p>Research by Miyakawa et al. (2003) studied DNA from human families affected by schizophrenia and found that those with the disease were more likely to have a defective version of a gene, called PPP3CC which is associated with the production of calcineurin which regulates the immune system. Also, research by Sherrington et al. (1988) has found a gene located on chromosome 5 which has been linked in a small number of extended families where they have the disorder. Murry (2012) argues that Sz involves a large number of genes, each of which make only a small contribution.</p> <p><b>Family studies:</b> First degree relatives share an average of 50% of their genes, and second degree relatives share approx 25%. To investigate genetic transmission of schizophrenia, studies compare rates of schizophrenia in relatives of diagnosed cases compared to relatives of controls. Evidence suggests that the closer the biological relationship, the greater the risk of developing schizophrenia. Kendler (1985) has shown that first-degree relatives of those with schizophrenia are 18 times more at risk than the general population. Gottesman (1991) has found that schizophrenia is more common in the biological relatives of a schizophrenic, and that the closer the degree of genetic relatedness, the greater the risk.</p> <p><b>Twin studies:</b> Twin studies offer an alternative way of establishing genetic links, by comparing the difference in concordance rates. Both share the same environment, but only the MZ twins have identical make up. Many studies have been conducted and they all show a much higher concordance rate in MZ twins than DZ twins. To separate out genetics conclusively from the environment, researchers have sought out MZ twins reared apart where one twin has been diagnosed with schizophrenia. Gottesman and Shields (1982) used the Maudsley twin register and found 58% (7 out of 12 MZ TWIN PAIRS REARED APART) were concordant with schizophrenia. If the genetic hypothesis is correct, then the offspring of a non-affected discordant MZ twin should be high risk. Fischer (1971) found that 9.4% of such offspring developed schizophrenia, which is a much higher incidence than in the general population. A study in London using the Maudley Twin Register by Cardno et al. (1999) found a 40% concordance rate in MZ twins, compared to 5.3% in DZ twins.</p> <p><b>Adoption studies:</b> A more effective way of separating out the effects of environmental and genetic factors is to look at adopted children who later develop schizophrenia and care them with their biological and adoptive parents. The Finish Adoption Study, which Tienari began in 1969 identified adopted away offspring of biological mothers who had been diagnosed with schizophrenia (112 index cases), plus a matched control group of 135 adopted offspring of mothers who had not been diagnosed with any mental disorder. Adoptees ranged from 5-7 years at the start of the study and all had begun separation from their mother before the age of 4. The study reported that 7% of the index adoptees developed schizophrenia, compared to 1.5% of the controls. A Danish Adoption Study, reported by Kety et al. (1994), taking a national sample from across Denmark, found high rates of diagnosis for chronic schizophrenia in adoptees whose biological parents had the same diagnosis, even though they had been adopted by 'healthy' parents. The data provided by these prospective studies have indicated a strong genetic link for schizophrenia.</p>	<p>⊙ One strength of the genetic explanation of schizophrenia is that there is <b>further empirical support</b> provided by Kety et al. (1975). On one of the largest adoption studies, Kety used two groups of adoptees who were identified as: (a) 33 who had schizophrenia, and (b) a matched group who didn't. Rates of the disorder were compared in the biological and adoptive families of the two groups of adoptees – the rate was greater among biological relatives of the schizophrenic adoptees than among those of the controls, a finding which supports the genetic explanation. Further, the rate of schizophrenia wasn't increased among couples who adopted the schizophrenic adoptees, suggesting that environmental factors weren't of crucial importance (Gelder, 1989). This suggests that there is <b>wider academic credibility</b> for the notion that genetics play an influential role in the development of schizophrenia.</p>	<p>⊙ One weakness of the genetic explanation of schizophrenia is that there are <b>methodological problems</b>. Family, twin and adoption studies must be considered cautiously because they are retrospective, and diagnosis may be biased by knowledge that other family members who may have been diagnosed. This suggests that there may be problems of <b>demand characteristics</b>.</p> <p>⊙ A second weakness is the problem of <b>nature-v-Nurture</b>. It is very difficult to separate out the influence of nature-v-nurture. The fact that the concordance rates are not 100% means that schizophrenia cannot wholly be explained by genes and it could be that the individual has a pre-disposition to schizophrenia and simply makes the individual more at risk of developing the disorder. This suggests that the biological account <b>cannot give a full explanation</b> of the disorder.</p> <p>⊙ A final weakness of the genetic explanation of schizophrenia is that it is <b>biologically reductionist</b>. The Genome Project has increased understanding of the complexity of the gene. Given that a much lower number of genes exist than anticipated, it is now recognised that genes have multiple functions and that many genes behaviour. Schizophrenia is a multi-factorial trait as it is the result of multiple genes and environmental factors. This suggests that the research into gene mapping is <b>oversimplistic</b> as schizophrenia is not due to a single gene. A better explanation could be <b>epigenetics</b> which explains that it is not enough for a gene to be present it needs to be switched on or off by environmental factors to become active and lead to the development of characteristics such as Sz.</p> <p>⊙ Another weakness of the family studies is that they <b>lack population validity</b>. The reason for this is because the samples that are used are small in numbers and only a select number of families are used. This suggests that there are issues with the findings being <b>generalised to the whole population</b>.</p>
<b>DOPAMINE HYPOTHESIS</b>	<b>A01 Explanations/Research</b>	<b>Strengths</b>	<b>Weaknesses</b>
	<p>Dopamine is a neurotransmitter. It is one of the chemicals in the brain which causes neurons to fire. The original dopamine hypothesis stated that schizophrenia suffered from an excessive amount of dopamine. This causes the neurons that use dopamine to fire too often and transmit too many messages. This message 'over load' may produce many of the symptoms of schizophrenia. Evidence for this comes from that fact that amphetamines increase the amounts of dopamine. Large doses of amphetamine given to people with no history of psychological disorders produce behaviour which is very similar to paranoid schizophrenia. Small doses given to people already suffering from schizophrenia tend to worsen their symptoms. Further evidence points to the connection between dopamine and schizophrenia comes from antipsychotic drugs to treat the disorder. They work by blocking dopamine receptors, so preventing dopamine-receiving neurons from firing. In addition, L-Dopa, which is used to treat Parkinson's Disease (caused by a lack of dopamine), can produce schizophrenic symptoms in individuals with no history of the disorder (Grilly, 2002).</p> <p>A second explanation developed, which suggests that it is not excessive dopamine but that fact that there are more dopamine receptors. More receptors lead to more firing and an over production of messages. Autopsies have found that there are generally a large number of dopamine receptors (Owen et al., 1987) and there was an increase in the amount of dopamine in the left amygdale (Falkai et al. 1988) and increased dopamine in the caudate nucleus and putamen (Owen et al, 1978).</p>	<p>⊙ A strength of the research into schizophrenia is that it has <b>practical applications</b>. From the research using schizophrenics new drugs have been developed such as Clozapine, which is much more effective than neuroleptics at relieving schizophrenic behaviour. These drugs <b>improve the patients' quality of life</b>.</p>	<p>⊙ Is the raised dopamine levels the cause of the schizophrenia, or is it the raised dopamine level the result of schizophrenia? (<b>Brain plasticity</b>) It is not clear which comes first. This suggests that one needs to be careful when establishing <b>cause and effect relationships</b> in schizophrenic patients.</p> <p>⊙ A second criticism is that there is <b>contradictory evidence</b> for the biochemical explanation from Kasper et al. (1999). He suggests there are a number of problems with the dopamine hypothesis. First, antipsychotic drugs are effective for only positive symptoms. Therefore, excessive dopamine can at best explain only some types of schizophrenia. Second, newer atypical antipsychotic drugs (e.g., clozapine) have proved more effective than traditional ones in successfully treating the symptoms of schizophrenia despite blocking fewer dopamine receptors for a shorter period of time. This suggests that there is <b>refuting evidence</b> for the notion of dopamine being the main contributing factor associated with schizophrenia.</p> <p>⊙ A final weakness of the dopamine hypothesis is that it is <b>biologically deterministic</b>. The reason for this is because if the individual does have excessive amounts of dopamine then does it really mean that they will develop schizophrenia? This suggests that the dopamine hypothesis does not account for <b>freewill</b>.</p>

BRAIN STRUCTURE	A01 Explanations/Research	Strengths	Weaknesses
	<p>Using PET, MRI and Cat scans researchers have discovered that many schizophrenics have enlarged ventricles, cavities in the brain that supply nutrients and remove waste. The ventricles of a person with schizophrenia are on average about 15% bigger than normal (Torrey, 2002).</p> <p>Brown et al. (1986) found decreased brain weight and enlarged ventricles, which are the cavities in the brain that hold cerebrospinal fluid. Flaum et al. (1995) also found enlarged ventricles, along with smaller thalamic hippocampal and superior temporal volumes. Buchsbaum (1990) found abnormalities in the frontal and pre-frontal cortex, the basil ganglia, the hippocampus and the amygdale. As more MRI studies are being undertaken, more abnormalities are being identified. Structural abnormalities have been found more often in those with negative/chronic symptoms, rather than positive/acute symptoms, lending support to the belief that there are two types of schizophrenia: Type 1 (acute) and Type 2 (chronic).</p>	<p>⊗ A strength is that the research into enlarged ventricles and neurotransmitter levels have <b>high reliability</b>. The reason for this is because the research is carried out in highly controlled environments, which specialist, high tech equipment such as MRI and PET scans. These machines take accurate readings of brain regions such as the frontal and pre-frontal cortex, the basil ganglia, the hippocampus and the amygdale. This suggests that if this research was <b>tested and re-tested</b> the same results would be achieved.</p> <p>⊗ Supporting evidence for the brain structure explanation comes from <b>further empirical support</b> from Suddath et al. (1990). He used MRI (magnetic resonance imaging) to obtain pictures of the brain structure of MZ twins in which one twin was schizophrenic. The schizophrenic twin generally had more enlarged ventricles and a reduced anterior hypothalamus. The differences were so large the schizophrenic twins could be easily identified from the brain images in 12 out of 15 pairs. This suggests that there is <b>wider academic credibility</b> for enlarged ventricles determining the likelihood of schizophrenia developing.</p>	<p>⊗ One weakness of the neuroanatomical explanation is the problem of <b>cause and effect</b>. Causation cannot be inferred as associations have only been identified. The brain dysfunction may be a symptom of the disorder rather than the cause, as the plasticity (flexibility) of the brain means that it may change as a result of abnormality. This suggests that there is a problem with the issue of <b>chicken and egg</b>.</p> <p>⊗ A second weakness of the neuroanatomical explanations is that it is <b>biologically deterministic</b>. The reason for this is because if the individual does have large ventricles then does it really mean that they will develop schizophrenia? This suggests that the dopamine hypothesis does not account for <b>freewill</b>.</p> <p>⊗ Beng-Choon Ho (2010) in a longitudinal correlational study of 211 schizophrenics found that antipsychotic drugs have measurable influence on brain tissue loss over time. However this was a correlational study so it does not show cause and effect. However the findings were supported by Lewis (2009) who administered antipsychotic drugs to primates and found a brain volume loss of 10%. This study was carried out on animals so we cannot extrapolate to humans without caution.</p> <p>⊗ if the reduction in brain volume is the cause of the schizophrenic symptoms then it cannot explain why after 30 years of the initial onset, 35% of the schizophrenics are classified as "much improved".</p>

**In contrast**, the psychological explanations **rejects** the view that schizophrenia is caused by genetics and brain chemistry. **Instead** it **favours** the idea that the disorder is caused by life events - the environment, upbringing, family etc. For example, research shows that dysfunctional family interaction, where there is a lot of "expressed emotion", can lead to schizophrenia. Also many schizophrenics come from lower social classes which implies that poverty and poor housing are involved. **This suggests** that the biological account does not provide a full explanation of schizophrenia.

**Diathesis Stress Model:** suggests that stress, through its effects on cortisol production, acts upon a preexisting vulnerability to trigger and/or worsen the symptoms of schizophrenia (there is a genetic vulnerability to a disorder but this is triggered when an individual has been exposed to a stressful life events). Both of these factors are necessary for a disorder to develop. This could explain why the concordance rate for mental disorders for MZ twins is nothing like 100%.

June 11

**'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. (9 marks + 16 marks)**

### **Examiner's comments**

This was the most popular question attempted by over half of the cohort. Instances of partial performance were rare and many candidates considered three or even four relevant biological explanations of schizophrenia, the most common being genetics, biochemistry and neuroanatomy. Most candidates outlined the genetic explanation using research and were able to achieve reasonable AO1 marks by doing so. There were some impressive descriptions of the dopamine hypothesis, many including specific details relating to D2 receptors.

There were two main problems with AO2/AO3 in weaker candidates. Some focused on methodological evaluation of research studies without making the implications of their criticism clear in relation to the biological explanation presented (eg genetics). Many candidates attempted generic points on reductionism and determinism. Some weaker candidates covered three or more explanations which limited AO2/AO3 and the development and elaboration of argument. Many candidates did not follow a clear line of argument and this limited their marks.

Higher AO2/AO3 marks went to candidates who evaluated each explanation as they went through the essay. Those candidates who were able to consider the inter relationships between biological explanations (for example genetics and biochemistry) were also rewarded.

### **Model answer**

The assumption made by the biological approach is that mental disorders are caused by physiological factors, in the same way physical disorders are. When considering schizophrenia, the biological approach focuses on genetic, biochemical and structural factors. /

Genes code for a person's characteristics and neuroanatomy. The argument that genes are the cause of schizophrenia, predicts that the rate of schizophrenia will be higher between relatives due to the offspring of a schizophrenic inheriting this gene. / The claim is supported by the prevalence statistic being the same around the world at 1% of the population having schizophrenia, showing that the prevalence doesn't vary with the environment. // Furthermore Kendler showed that relatives who share 50% of their genes with someone who is schizophrenic, i.e. a parent, are eighteen times more at risk of developing schizophrenia. //

One disadvantage of using family studies such as Kendler's is that most children also share the same environment as their parents and spend more time together so will influence each other's behaviour. This means we cannot rule out the influence the environment may have on the development of schizophrenia as it is difficult to separate the influence of nature and nurture in a family. //

Despite this criticism, there are many studies which do provide strong evidence for a genetic cause such as Gottesman's. He found that 46% of children with two schizophrenic parents will develop schizophrenia.

This likelihood decreases to 16% if only one parent is schizophrenic. Gottesman shows that the concordance rate is much higher between relatives, supporting the influence of genetic factors. //

However, another issue with family studies is that it is a very small sample due to the difficulty had finding families with two schizophrenic parents and a schizophrenic child. Only a select number of these families will be used for the study, decreasing the number of participants available even more. For this reason, samples like Kendler's and Gottesman's have low population validity meaning the findings cannot be generalised to the whole population. //

Twin studies can also be used to establish the links between the genes of relatives. The majority of twin studies show a higher concordance rate of schizophrenia in monozygotic twins than in dizygotic twins who are not identical. Torrey carried out a reassessment of eight twin studies which had already been carried out. His conclusion was that the concordance rate for monozygotic twins was 26%, compared to the 6%

concordance rate for dizygotic twins. / Torrey's work supports the view that schizophrenia is genetically inherited because with no genetic influence, there wouldn't be a difference in the concordance rate between monozygotic and dizygotic twins. However, because the concordance rate wasn't 100%, other factors such as the environment must also be considered. //

The problem with using monozygotic twins in the twin studies is that an assumption is made that they were born identical and have been raised in exactly the same way. This may not be the case as often one twin has taken more nutrients than the other so is born bigger or more developed. This could affect the development of the central nervous system. //

Using adoption studies is a more effective way of looking at schizophrenia as it allows the researcher to separate the influence of genetic factors from the influence of environmental factors. Kety et al were interested in whether the concordance rate of schizophrenia differed between adopted adults with a schizophrenic biological mother and adopted adults without a schizophrenic relative. They found that the rate of schizophrenia was much higher in people whose biological parent had been diagnosed as schizophrenic, despite being raised by non-schizophrenic adults. This shows that the genetic influence is more significant than environmental factors. /

The trouble with using adoption studies is that it is hard to access the reasons behind the child's adoption but it's possible that the child suffered some type of trauma which has led to their development of schizophrenia as the diathesis –stress model suggests that stress can trigger the onset of Sz. // Similarly, the time they had spent with their schizophrenic parent may have influenced the way they behave. For example children often copy what they see their parents doing, so imitation of the schizophrenic behaviour could have increased their likelihood of developing the disorder themselves. //

All of the research done using samples of adopted schizophrenics show the same results. Studies such as Tienari's where 10% of adopted participants with schizophrenic mothers also developed schizophrenia, compared to the 1% of participants with non-schizophrenic mothers, support Kety's findings. All these studies show that the concordance rate is higher between children and their biological parents, providing strong evidence for the claim of schizophrenia being inherited. //

Even though there are a lot of studies which suggest that there is a strong genetic influence on developing schizophrenia, no gene has yet been discovered which is responsible for schizophrenia. This makes it hard to rely on genetic factors being the main cause of schizophrenia. However, the biological approach does recognise that genes can be turned on and off by environmental factors, epigenetics, so is not completely deterministic. //

The dopamine hypothesis is another explanation used in the biological approach. This explains that schizophrenia sufferers have an excessive amount of dopamine, a neurotransmitter. This causes the neurons to fire too often, therefore transmitting more messages which is believed to cause the schizophrenic symptoms. / Other causes of the symptoms may be the reuptake of dopamine to the presynaptic neuron is disturbed, there are too many dopamine receptors or the receptors are too sensitive to dopamine. // Evidence supporting this hypothesis comes from the research done in to antipsychotic drugs which reduce schizophrenic symptoms by blocking the dopamine receptors. This suggests that it is overactive dopamine receptors which cause the symptoms. // L-dopa is a chemical which an increase in dopamine levels and is used to treat people with a lack of dopamine such as with Parkinson's disease. Grilly found that when L-dopa is given to patients with Parkinson's disease, it can produce positive schizophrenic symptoms in the patients; this would indicate that dopamine levels are related to schizophrenic symptoms. //

A weakness with the dopamine hypothesis is that it is unclear whether the high dopamine levels cause schizophrenia, or if having schizophrenia causes an increase in the level of dopamine. This uncertainty makes it difficult to establish a cause and effect relationship between dopamine levels and schizophrenia. //

A second weakness is that if antipsychotic drugs block the dopamine receptors, and the sensitivity of dopamine receptors are the cause of the symptoms, when the drugs are taken there should be a rapid improvement of all the schizophrenic symptoms displayed as antipsychotics block the dopamine receptors.

However, this is not what happens and only positive symptoms tend to improve. As well as this, it can be up to two months before the antipsychotic drugs start working and an improvement is shown. //

The biological explanation also claims that abnormalities in the structure of the brain can also cause schizophrenia. Research such as Johnstone's found that schizophrenics have enlarged ventricles in the brain compared to non-schizophrenics suggesting that schizophrenia is linked to a loss of brain tissue. There are also abnormalities in their frontal lobe which could be the cause of their disorganised thoughts, identified by the use of MRI scanning. /

This research is highly reliable as it has to be carried out in very controlled environments using specialist researchers and equipment as the study of the brain is very vital. The equipment used is very accurate. All these factors suggest that the findings of studies such as Johnstone's would be found by other researchers who carried out the same study. //

A limitation of stating that abnormalities such as enlarged ventricles in the brain is the cause of schizophrenia is that, if this was the case, no one would be able to be cured or re-diagnosed as being non-schizophrenic. This is because the brain's cortex does not grow back to fill the space of the enlarged ventricles so no one would be able to make any improvements in their condition. Considering this factor, 35% of schizophrenics are classified as being 'much improved' later on in life, this shouldn't be possible if the basis of schizophrenia is the loss of brain matter. //

*This essay is very well written and clearly structured but just over the word limit. (1300 words).*

*The points made are developed appropriately and examples are given to clarify the explanations.*

*IDAs: lack of representativeness of samples, reliability of scans and evaluation of the studies used to support/challenge the theories.*

## Biological therapies



The evaluation of therapies should be done in terms of their **appropriateness** and **effectiveness**

### Antipsychotics

It is thought that schizophrenics produce too much dopamine or have more dopamine receptors than non-schizophrenics. We cannot decrease the amount of dopamine or reduce the number of receptors so the next best thing is to block the receptors.

There are two types:

- **First generation antipsychotics** also called neuroleptics such as **chlorpromazine**: these block the dopamine receptors.  
They have been in use since the 1950s and are still used today.  
They reduce the positive symptoms in about 75% of the patients however they do not reduce the negative symptoms.  
They have serious side-effects:
  1. Patients complain of feeling apathetic (no enthusiasm).
  2. Increased risk of depression.
  3. Affect sexual function
  4. Tardive dyskinesia which is a very disabling disorder involving tremors and lack of control of the motor function to the point where patients might be unable to look after

themselves, they have problems breathing, sleeping and feeding themselves. . After 25 years of treatment T.D is present in up to 68% of the patients (Glenmullen,2000)

5. Because of the severe side-effects and the lack of organised thinking there is a problem with compliance (the patients do not take their drugs regularly) in many cases the drugs are given by injection every 2-4 weeks to prevent patients from being hospitalised.
  6. Ethical problems: can we force patients to take these drugs when there is the possibility of serious side-effects?
- **The second generation antipsychotics also called atypical antipsychotics** such as **risperidone**: these block both the dopamine and the serotonin receptors.
    1. They seem to be more effective against the negative symptoms. They are reported to be more effective for the 25% of patients who were not helped by the first generation of antipsychotics.
    2. Apart from Clozapine they also give to motor problems like the first generation antipsychotics.
    3. Clozapine can lead to **Agranulocytosis** , a disorder of the blood in which the number of granulocytes (a type of white blood cells) is reduced. This can be fatal. Monitoring by regular blood tests is necessary.
    4. Weight gain of 5kg or more is common
    5. Sexual dysfunction

Evaluation common for the two types of drugs:

- They control the symptoms but do not cure the condition so if the patients stop taking the drugs the symptoms recur.
- Suicide remains high in schizophrenics (1/10000 general population Vs 10% after 10 years with Sz)
- Severe side-effects
- Focus on biology and ignore other social and psychological factors which could lead to improvement
- Allows most schizophrenics to live and work fairly normally
- Cost effective for society
- The patients are seen as passive recipients of the treatment not as partners, this leads to disempowerment which in itself can lead to helplessness
- Drugs allow patients to be stable and have enough insight to take part in psychological therapies like CBT
- It is difficult to evaluate the outcome of drug treatments as most schizophrenics are involved in both biological and psychological therapies at the same time.

## ECT

Involves passing a short (0.5 second) electric shock (0.6 AMPS) through the brain in order to induce a seizure it is thought that it 'restart' it and its functions. Prior to the procedure a patient is made unconscious and is given muscle relaxants to prevent fractures caused by the very powerful muscle contractions.

ECT was largely abandoned as a treatment for schizophrenia after the discovery of the antipsychotic drugs in the 1950s but has recently been re-introduced in the USA. In the UK, the use of ECT is not recommended

by NICE except in very particular cases and as a last resort when other treatments have proved ineffective (mainly for catatonic schizophrenia).

Serious side effects: Temporary memory loss however it can be permanent

Death in 4 in 100 000 cases

Ethics: Patients who suffer from schizophrenia may be willing to try anything in order to improve their symptoms; if they feel scared, under constant threat and maybe even suicidal. Although a patient may give informed consent they may not be necessarily fully aware of what they are signing up for!



**Make the description explicitly relevant to schizophrenia and show awareness of the fact that this is not a mainstream therapy.**

**January 10**

**“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.**

**(9 marks + 16 marks)**

### **Examiner’s comments**

Some candidates wasted time in their answers by including detailed descriptions of schizophrenia or explanations of its origin. This was only creditworthy where it provided clear underpinning for the explanation of the therapy. There were some excellent well-detailed answers, especially on drugs, where students showed an impressive knowledge of the relevant drugs and their mode of action. There were also some detailed accounts of CBT showing good understanding of how it works in the context of schizophrenia. Other psychological therapies were often less well done. For example, where candidates selected token economy, the description and evaluation lacked any understanding of how it is used with this particular client group. Many candidates were not well prepared for this question and wrote generally about therapies without making them explicitly relevant to schizophrenia. Psychoanalysis, in particular, is difficult to make relevant to schizophrenia except in the rare instances when candidates referred specifically to the work of Sullivan. Some candidates, having given a general description of psychoanalysis then actually acknowledged that 'Freud said it wouldn't work'. ECT was accepted but, given that NICE does not recommend it as a first-line therapy for most cases of schizophrenia in the UK and that the APA in the USA suggest it only when other treatments have been tried and failed, it was disappointing to see so many answers in which it was offered uncritically as a mainstream therapy for schizophrenia. Similarly, 70 year-old psychosurgery techniques were often described in long and gory detail without any apparent understanding of current practice. In some answers, the evaluation was actually better than the description and some candidates made good use of outcome studies to support their arguments. However, in many answers AO2/3 was generic and unsupported by evidence. The same points were repeated for each type of therapy. In particular, 'drop-in' references to issues and debates such as 'It is reductionist' or 'It is determinist' showed weak understanding and gained little or no credit.

Candidates would have benefited from taking a little more time to plan their answer in order to achieve coherent elaboration and a clear line of argument.

Given the new organisation of the specification which requires candidates to have studied therapies in the context of a particular disorder, the lack of understanding and ability to make material explicitly relevant to schizophrenia shown in a large number of answers was rather disappointing.

## **Model answer**

Drug therapy is the main biological therapy used to treat schizophrenia (Sz). There are two types of drugs the first generation (conventional) and the second generation (atypical) antipsychotics.

First generation antipsychotics such as chlorpromazine were first used as an anaesthetic and in the 1950s were found to decrease the positive symptoms of schizophrenia. Their mode of action is based on the dopamine hypothesis (the theory that schizophrenics produce too much dopamine or have too many or too sensitive D2 receptors). As we cannot control the amount of dopamine produced or change the number or sensitivity of the receptors, antipsychotics bind to the receptors in order to prevent stimulation them and thus, suppress the symptoms. /

The appropriateness of this treatment is debatable. While drug therapy is a much more humane approach to treating schizophrenia than electro convulsive therapy or the restraints that were used before the drugs and allow the patients to lead a relatively normal life instead of being confined to a mental hospital, // it is questionable whether it is a treatment suiting all schizophrenics.

Antipsychotic treatment rely on the patients remembering take their medication which is difficult as schizophrenics have disorganised minds (this affects their memory, and their ability to plan and think logically). // This can lead to the "the revolving door" syndrome where the patients forget to take their medication, are re-admitted to hospital where they are stabilised on medication and discharged again. This suggests that the treatment might not be appropriate for patients with severe cognitive impairment. // Although the therapy can be adapted to this type of situation as the drugs can be given by injection in a slow-release form every four weeks. This therefore makes it more appropriate for patients with cognitive difficulties. //

However conventional antipsychotics have serious side-effects, these include apathy, increased risk of depression and tardive dyskinesia- an irreversible motor disorder which affect 68% of patients after 25 years of treatment. / Therefore injecting drugs with such severe side-effects raise ethical questions as once injected the action of the drugs cannot be stopped unlike tablets taken daily. This is particularly true as patients with cognitive deficits are not in a fit state to give informed consent.//

The increase of risk of depression is a serious side-effect which also raise ethical issues as schizophrenics have a high risk of suicide-14% commit suicide within ten years of diagnosis. This puts in question the appropriateness of the treatment.//

The side-effects of the conventional antipsychotics could affect the effectiveness of the treatment as patients might stop the treatment if they are worried about developing a disorder such as tardive dyskinesia. // Furthermore the treatment is not a cure as if the treatment is stopped the symptoms return in over 80% of the patients so it seems that drugs just control the symptoms. //

Furthermore, Beng-Choon Ho (2010) in a longitudinal correlational study of 211 schizophrenics found that antipsychotic drugs have measurable influence on brain tissue loss over time. However this was a correlational study so it does not show cause and effect. // These findings were supported by Lewis (2009) who administered antipsychotic drugs to primates and found a brain volume loss of 10%. This study was carried out on animals so we cannot extrapolate to humans without caution. //

Conventional antipsychotics are effective in controlling only 75% the positive symptoms, they have been found to be ineffective on negative symptoms. // This lead researchers to hypothesise that negative and positive symptoms had different causes and the discovery of second generation (atypical) antipsychotics. These drugs bind temporarily to the dopamine receptors but also to the serotonin receptors in an attempt to control both positive and negative symptoms. /

Kapur and Remington (2000) found that they are more effective for the negative symptoms than the conventional drugs which suggests that they are more appropriate and effective for a wider range of patients. //

Because the drugs bind to the dopamine receptors for a shorter time they have less serious side-effects than the conventional drugs and Jest et al. found that the incidence rate of tardive dyskinesia is 25% lower than with first generation antipsychotics, which suggests that this treatment is more appropriate. // However atypical antipsychotics can cause agranulocytosis (a white blood cell deficiency) which can be fatal in 2% of the patients. This is monitored by frequent blood tests and these drugs cannot be injected in a slow-release form. //

Atypical drugs can also lead to weight gain and a decrease in libido which can impact negatively on the patients' self-esteem and social support and could lead to them stopping the medication. //

Atypical drugs were found by Awad and Voruganti (1999) to be effective for 85% of the patients compared to conventional drugs which were found to be effective in only 65%. // However Leucht et al found that, despite being much more expensive than the conventional drugs, two atypical drugs were only slightly more effective than their conventional counterpart and two were no more effective. //

The effectiveness of drug treatment is questionable in most cases as they are very often use in conjunction with psychological therapy, for example cognitive behavioural therapy so it is difficult to discern whether the improvements observed are the result of the drugs or of the psychological therapy. //

However the drugs act faster than psychological therapies which can get the patients to a state where they can respond to psychological therapies. //

Also as the drugs are faster acting than psychological therapies make them more appropriate for patients who are very distressed and at risk of suicide or a danger to others. //

Despite these limitations, drugs have improved the quality of life of schizophrenics by allowing them to lead a fairly normal life it has also been very cost-effective for society as patients can now be treated outside hospital setting. //

**This is well within the word limit: 900 words**

**This essay is clearly structured: explanation and evaluation of both conventional and atypical antipsychotics and an evaluation of drug treatment as a whole at the end.**

**IDAs: ethics, application to real life.**

## Psychological explanations of schizophrenia

You could choose any psychological explanations out of behavioural, cognitive, psychodynamic and socio-cultural explanations. In this booklet I have used behavioural and cognitive explanations.

See table page 19

## Psychological explanations of schizophrenia

Psychological explanations of schizophrenia			
Behavioural explanation	<b>A01 Explanations/Research</b> Assumption: schizophrenia like any other behaviour is learnt from the environment. The learning occurs through 2 processes: Operant conditioning: if the behaviour is positively reinforced then the behaviour will be repeated. If it is punished then the frequency of the behaviour will decrease. Social learning: If an individual observes the schizophrenic behaviour being reinforced in another person i.e. parent (vicarious reinforcement) the individual might reproduce the behaviour to obtain the same reinforcements. These two processes combine An individual starts imitating schizophrenic behaviour observed in another then this is maintained and developed through operant conditioning.	<b>Strengths</b> ☉ it could explain why the disorder tends to run in families so it is possible that people may learn to exhibit symptoms through observing other people who do and want to imitate these people to get the same reinforcements. ☉ The behaviour could be reinforced by getting more attention from family, friends and the medical profession. ☉ The approach has given rise to therapies which seem to improve some aspects of schizophrenic behaviour <i>i.e. token economy</i> . These therapies unlike the biological therapies do not have side-effects	<b>Weaknesses</b> ☹ This approach cannot really account for the core features of schizophrenia: hallucinations, delusions and disorganization of thinking. ☹ It cannot explain why so many people with schizophrenia exhibit similar symptoms regardless of where they originate. ☹ It cannot explain why an individual who has never had contact with a schizophrenic display the same symptoms of schizophrenia. ☹ Experiments with behaviour modification for schizophrenia have indicated that, whilst symptoms can be modified (disorganised speech), the accompanying experiences (hallucinations) tend to persist, which suggests that the cause cannot be only the learning of the behaviour. ☹ It cannot explain why the suicide rate is 10 times higher in schizophrenic than in the normal population. Suicide cannot obviously be reinforced. ☹ Reductionist as does not account for the neurological factors involved. However these could be the consequence rather than the cause of the disorder (brain plasticity) furthermore does not take into account psychological factors i.e. conflicts in early childhood or social factors i.e. stress and urbanicity.
	<b>A01 Explanations/Research</b> The cognitive approach focuses on how people think, how they process information. <b>Assumption: mental disorders including schizophrenia are caused by "faulty" thinking.</b> We are normally able to filter incoming stimuli and process them to extract meaning it is thought that these filtering mechanisms and processing systems are defective in the brains of schizophrenics. Cognitive theorists assume that these cognitive deficits are due to underlying physiological abnormalities. Frith's model Frith distinguishes between conscious and preconscious processing. <ul style="list-style-type: none"> <li>• Conscious processing is where the highest level of cognitive functioning takes place, we are aware of this level. This has a limited capacity so we can only carry out one task at a time.</li> <li>• The preconscious processing, takes place without our awareness, this an automatic process and we can carry out many tasks at any one time</li> </ul> Usually only important information arrives into conscious awareness, in schizophrenics, Frith proposes that the attentional filter breaks down and the unimportant information also gets in the conscious so it is seen as something important which needs to be acted upon. Frith believes that it is how delusions happen. Frith explains auditory hallucinations in a similar way. We are bombarded with sounds constantly and preconscious mechanisms interpret these sounds and only the significant sound reach the conscious level of processing. If the conscious/preconscious filter is defective, it is possible that we misinterpret non-speech sounds as speech and experience them as voices. Hemsley's model: Non-schizophrenic individuals give meaning to new sensory input by using subconsciously previously stored knowledge (schemas). Hemsley proposes that this process breaks down in schizophrenics and that the schemas are not activated, therefore they are subjected to an overload of sensory information and do not know which to attend to. Internal speech and thoughts are not recognised as such but as experienced as coming from an external source and experienced as auditory hallucinations.	<b>Strengths</b> ☉ Meyer-Lynderberg et al. (2000) found a link between excess level of dopamine in the prefrontal cortex and dysfunctions of the working memory. Working memory dysfunction is associated with cognitive disorganisation typical of schizophrenics. This supports the idea that underlying biological factors are involved in schizophrenia. ☉ It has given rise to CBT which seems to improve the outcome for many schizophrenics (see therapy) and has no side-effects ☉ Bental et al. (1991) found that Sz's struggled to identify words belonging to a certain category, such as birds, that they had seen before, created themselves or had not seen before, supporting Frith's theory that people with Sz had metarepresentation problems.	<b>Weaknesses</b> ☹ The models cannot explain why the voices heard are so negative and abusive or suggest reprehensible acts. ☹ The cognitive approach explains mainly positive symptoms. ☹ It could explain why delusions are often influenced by cultural factors i.e. a French schizophrenic might think he is Napoleon whereas a British one might think he is Nelson ☹ It is reductionist because despite the fact that it takes into account physiological factors, it does not take into account the influence of early childhood conflicts (psychodynamic explanation) or social factors such as stress or urbanicity ☹ the cognitive approach explains some symptoms rather than the cause of Sz, however it gives us some insight in the disorder and this has been used to create therapies which have shown to be helpful in the treatment of Sz.

**In contrast**, the biological explanation **rejects** the view that schizophrenia is caused by psychological factors. **Instead it favours** the idea that the disorder is caused by excessive levels of dopamine, enlarged ventricles and genetics. **This suggests** that the biological account does not provide a full explanation of schizophrenia.

In conclusion the **Diathesis Stress Model**, which suggests that there is a genetic vulnerability to a disorder (Diathesis), but this is triggered when an individual has been exposed to a stressful life event (stress). Both of these factors are necessary for a disorder to develop. This is why not all the children with schizophrenia develop the disorder, and why the concordance rate for mental disorders for MZ twins is nothing like 100%.

## Discuss two psychological explanations of schizophrenia (8+16 marks)

### **Model answer**

The assumption of the behavioural approach is that schizophrenia like any other mental disorder is learnt from the environment. It proposes that behaviour is learnt through two processes: operant conditioning and social learning.

Social learning is learning by imitation. An individual observes the behaviour being displayed by another person, if the behaviour is reinforced in the other person (vicarious reinforcements) then the individual is likely to perform the behaviour to obtain the same reinforcements. For example a child could observe one parent display schizophrenic behaviour and get attention because of it so the child learns the behaviour and performs it to get the same attention. *L*

Operant conditioning is learning through consequences, if a behaviour has positive outcomes then the behaviour is likely to be repeated. For example an individual reports hallucinations and gets attention from medical staff, he/she finds this pleasant and repeats the behaviour. *L*

The two processes are combined the individual learns the behaviour through imitation this gets reinforced so the individual repeats the behaviour again hoping for the same consequence. *L*

This could explain why schizophrenia “runs” in family as people are likely to see the behaviour modelled by a relative. Duck argues that the best models are people of the same age, same sex and of similar status but they can also be significant persons in the child’s life like a parent. *//* However it could be argued that the schizophrenic behaviour is punished by society rather than reinforced as schizophrenics find it difficult to get jobs, they are likely also to have difficulties in finding housing, they are also likely to have very few friends so the reinforcement through gaining attention does not seem to outweigh the difficulties schizophrenics face because of their condition. *//*

Furthermore it could also be argued that many people displaying symptoms of schizophrenia have never seen schizophrenic behaviour, if schizophrenia is learnt then we cannot explain where they learnt it from. *//* Additionally the symptoms of schizophrenia do not vary between cultures, the content of hallucinations and delusions do vary but not the fact that the sufferers display these symptoms, if schizophrenia was learnt from the environment then we would expect variations between cultures. *//*

The behavioural approach focuses on observable behaviour however the main symptoms of schizophrenia are internal experiences such as hallucinations, delusions and disorganised thinking and it would be difficult to explain these experiences in terms of learning without considering that the sufferers are making up the symptoms. *//* This seems very unlikely as the rate of suicide in schizophrenics is 10% after 10 years with Sz compared with 1/10000 general population, this suggests a deep suffering and a feeling of helplessness which cannot be explained by the behavioural approach. *//*

However the approach has led to therapies which seem to have improved some of the symptoms for example Wilder et al. (2001) reported a research in which Every time a Sz patient made a statement unrelated to the topic being discussed, the therapist would ignore the statement and ask to take a break, then look away for 30 seconds. The sessions lasted for 10 mn and took place 2-3 times a week. The patients' bizarre vocalisations reduced dramatically over 30 sessions. / This suggests that the lack of reinforcement leads to an improvement of disorganised speech but it could also be argued that it might not necessarily indicate an improvement of the internal experiences of the patient, it might have been to the fact that the patient felt he/she could not talk about these disturbing experiences. //

Furthermore this research took place in hospital setting so the patients were most likely to be taking antipsychotic medication at the same time as being involved in this therapy so it is impossible to say whether the improvement was due to the drugs or to the sessions with the therapist. //

Furthermore the behavioural is reductionist. It does not take into account the biological symptoms observed in most schizophrenics for example enlarged ventricles but it could be argued that this is an effect rather than the cause of the disorder as the brain is a plastic organ which can change in its structure and functioning depending on the way we use it. //

Another psychological explanation is the cognitive approach. It assumes that schizophrenia like all mental disorders is caused by faulty thinking, however it also assumes that these cognitive deficits are due to underlying neurophysiological abnormalities. /

We are normally able to filter incoming stimuli and process them to extract meaning it is thought that these filtering mechanisms and processing systems are defective in the brains of schizophrenics. Frith argues that we usually filter information at a preconscious level and that only what is relevant reaches the conscious level where it is processed. In schizophrenics this filter is "faulty" and all information reaches the conscious level so irrelevant information is seen as relevant. / For example Frith explains auditory hallucinations in the following way: we are bombarded with sounds constantly and preconscious mechanisms interpret these sounds and only the significant sounds reach the conscious level of processing. In schizophrenics, the conscious/preconscious filter is defective, so they misinterpret non-speech sounds as speech and experience them as voices. /

Furthermore Hemsley proposes that non-schizophrenic individuals give meaning to new sensory input by using subconsciously previously stored knowledge (schemas). He suggests that this process breaks down in schizophrenics and that the schemas are not activated, therefore they are subjected to an overload of sensory information and do not know which to attend to. For example internal speech and thoughts are not recognised as such but as experienced as coming from an external source and experienced as auditory hallucinations. /

However it does not explain why the voices heard by schizophrenics are mostly critical, abusive and command them to commit reprehensible acts. We all have internal speech but although it might be critical at times it does not usually pretend to be God or command us to rape or kill. //

Bental et al. (1991) found that SzS struggled to identify words belonging to a certain category, such as birds, that they had seen before, created themselves or had not seen before, supporting Frith's theory that people with Sz had metarepresentation problems. //

The idea that underlying biological factors are involved in schizophrenia is supported by Meyer-Lynderberg et al. (2000) found a link between excess level of dopamine in the prefrontal cortex and dysfunctions of the working memory. Working memory dysfunction is associated with cognitive disorganisation typical of schizophrenics. //

The cognitive approach could explain symptoms such as hallucinations which are related to cognitive functioning but it cannot explain symptoms such as flat affect and mannerism and other negative symptoms. So it cannot explain the whole complexity of the disorder//

By focusing on the cognitive symptoms the approach is reductionist however this is useful as it has helped design therapies such as CBT which has shown to be helpful in the treatment of schizophrenia however this therapy is mostly used in conjunction with drug treatment so it is difficult to ascertain what has lead to the improvement. //

This approach cannot explain why close relatives have higher risks of developing schizophrenia than the general population or why people living in urban areas or of foreign origin are also more likely to be diagnosed with the disorder. //

In general the biological approach has better research support than the psychological explanations. For example Gottesman (1991) is one of many studies which support the influence of genetic factors in schizophrenia. However the psychological explanations have made important contributions to the treatment of the disorder and the therapies derived from the psychological explanations have no side-effects. //

**1282 words**

**Both explanations were given about the same weight (behavioural explanation has 9 AO2/AO3 whereas the cognitive explanation has 7 AO2/AO3)**

**Clear structure.**

**IDAs cultural variations, applications to real life, evaluation of sample and implications of the lack of representativeness.**

## Psychological therapies

### Behavioural approach-Token economy

The assumption of the learning approach is that all mental disorders including schizophrenia are learnt through operant conditioning and social learning therefore the therapies based on this approach aim at unlearning the behaviour and learning a more adaptive behaviour using the same processes.

Token economy is based on operant conditioning, it is a behaviour modification techniques which aim at modifying observable behaviour.

It occurs in three stages:

- Identifying the undesirable or maladaptive behaviour this can be not eating or displaying aggressive behaviour
- Identifying the reinforcers that maintain such behaviour for example nursing staff or the patient's family can be reinforcing the aggressive behaviour by giving more attention to the patient. This attention according to the behavioural approach makes it more likely to be repeated.
- Restructuring the environment so that the undesirable behaviour is no longer reinforced. This involves making the family/ nursing staff that how they are inadvertently reinforcing the behaviour and stopping the reinforcement for example not giving more attention for not being aggressive. However if the desired behaviour is displayed the patient receive a token which can be exchanged for privileges such as extra sport activities. However from an ethical point of view it is important that basic commodities such as food, water and sleep cannot be used as privileges, they are considered to be human rights.

The effectiveness of the treatment is supported by Ayllon and Azrin (1968) they studied female patients with SZ who had been hospitalised for an average of 16 years. They were all rewarded with plastic token for actions such as making their bed; they exchanged these tokens for pleasant activities such as seeing a film or additional visits to the canteen. They found that this was a successful therapy and the number of chores the patients performed each day increased to 5 to over 40.

This therapy is used in many psychiatric hospital to change the behaviour of patients and although it could be argued that it is in fact "manipulating" the patient it can be very useful for schizophrenics who have little insight in their condition as it might succeed in making them perform behaviours which benefits their health such as eating and taking care of their own hygiene. It is particularly useful for negative symptoms such as catatonia as it provides motivation.

However it also has limits as it focuses only on observable behaviours but the most distressing symptoms such as hallucinations and delusions are not improved by this therapy.

Furthermore Kazdin & Bootzin (1972) have claimed that the token economy does not lead to permanent behavioural change, and that once the reinforcement is removed, the undesirable behaviours return to their initial level which means that the change is temporary and does not address the cause of the disorder.

Another limitation is that it requires an environment where the therapist is in total control so that the patient has no access to the privileges without the consent of the therapist. It might not work when the patient goes back in the community or in their family. This means that the behaviour might be improved enough for the patient to be discharged but then needs readmitting because the change of behaviour is not maintained. This is a problem as the aim of the treatment is to reintegrate the patients in the community.

Dickerson et al. (2005) carried out a meta-analysis and found that token economy is probably most appropriate when used in combination with other forms of therapy which are designed to treat positive symptoms and most patient taking part in token economy also take antipsychotic drugs. However it makes it difficult then to ascertain whether it is effective on its own

Furthermore it requires the staffs to receive training and also to be consistent with each other which is difficult to achieve. If one staff gave a token too easily and another one required more from the patient then the message would be mixed and the change of behaviour might not occur.

Another difficulty with this therapy is that it is time consuming as the staffs have to be vigilant and observe the patients closely; this is difficult in a busy hospital especially as the token economy system is only effective if the tokens are given immediately after the desired behaviour has occurred. The longer the interval between the behaviour and the token the less likely it is that learning will take place.

Additionally there are ethical problems with this therapy as it could be argued that the patient should have to right to determine his/her behaviour even if this appears to go against his/her best interest. Furthermore the undesired behaviour might be punished by depriving the patients who might already be distressed by their condition of things they like such as playing their musical instruments.

### **Cognitive approach-CBT**

The cognitive approach assumes that mental disorders including schizophrenia are caused by "faulty" thinking. Furthermore it argues that the cognitive deficits observed in schizophrenic such as disorganised thinking and hallucinations are due to underlying physiological abnormalities. Therefore Cognitive Behavioural Therapy (CBT), the therapy derived from this approach aims at modifying the faulty thinking.

The form of CBT used with schizophrenics is called personal therapy. The therapist first aims to develop a trusting and respectful relationship with the patient. Then the patient explains his/her confusing, distressing experiences i.e. abusive voices.

Together they work at uncovering any patterns or triggers in the client's distressing experiences the therapist might ask questions such as "How long do you think others have been talking about you?" .

The therapist then challenges gently the client's beliefs i.e. that people want to harm him/her by asking questions such as "Are there any other possible explanations for what happened?" .

Then they try to develop strategies to deal with the distressing experiences.

The effectiveness of CBT is supported by Tarrrier et al (2000) found that people with Sz receiving 20 sessions of Personal therapy in 10 weeks along with drug therapy, followed by 4 booster sessions the following year, did

better than the Sz on drug alone, 1/3 of patients achieved 50% reduction of psychotic experiences, 15% were free of positive symptoms. None of the patients on drugs alone were completely free of positive symptoms. The difference in outcome was still present a year later.

However the sample used in the study was carefully selected: the patients included in the sample were patients who took their drugs (so excluded patients who refuse medication). Furthermore patients who were too confused, agitated or paranoid were not included as they would not have benefitted from CBT so CBT might be useful only for a certain type of patients not for all Sz. So although this shows a significant improvement in the patients, the sample is not representative of all schizophrenics so we cannot generalise the findings.

Furthermore it shows a limitation of the therapy, the patients who have little insight in their condition were excluded from the sample because they could not take part in the therapy, being unable to participate in a fairly rational discussion.

Additionally Lewis et al. (2002) CBT is used during the first episode it shortens the length of that episode but 18 months later the patients suffer the same relapse rate as patients who did not have CBT. This suggests that CBT might be more effective at the onset of the disorder.

However a meta-analysis carried out by NICE (2009) on 2118 patients did not show statistically significant differences between CBT and standard care for outcomes related to mortality (suicide), relapse or treatment adherence. This cast doubt on the effectiveness of CBT in the treatment of schizophrenia.

The evidence for the effectiveness of CBT as a treatment of schizophrenia is mixed at best but it has been argued that it is more effective for the positive symptoms such as auditory hallucinations than for negative symptoms such as catatonia.

Furthermore it could be argued that it might not be CBT itself which causes an improvement but the relationship between the therapist and the client. Schizophrenics are often rejected and when they talk about their symptoms people tend to feel uncomfortable but the therapist is interested and is willing to help, this in itself might be helpful and lead to improvement.

This treatment is not a cure and in Tarrier's research only 15% of patients was free of positive symptoms however it helps some patients and has not negative physical side-effects.

June 2012

**Outline and evaluate one biological therapy for schizophrenia and one psychological therapy for schizophrenia. (8 marks + 16 marks)**

### **Examiner's comments**

Schizophrenia remains the most popular option in Section A and was attempted by around 60% of students. In contrast to the usual pattern shown on Section A, many students provided insufficient descriptive detail of therapies for AO1 often achieving basic marks for this component of the question. Many students showed real lack of understanding of the nature of schizophrenia or awareness of current treatments which was worrying. The best route to good AO1 and AO2/AO3 marks was to focus on treatments which are currently used for schizophrenia (anti psychotic drugs and CBT) and to include appropriate reference to outcomes studies.

Many students focused on drug therapy and there is evidence that some schools and colleges are teaching this area well, with impressive detail regarding modes of action of conventional and atypical anti psychotics. Weaker students simply named different types of drugs with little reference to mode of action which achieved basic AO1 credit.

Students who selected ECT or psychosurgery were less successful in both description and evaluation of the techniques. Descriptions were largely generic, often adding little detail beyond that covered at AS. Such descriptions gained basic or rudimentary AO1 credit. Many students appeared to have little appreciation that ECT and psychosurgery have been largely abandoned as a treatment for schizophrenia, except under very rare conditions (eg treatment of severe catatonic states by ECT).

The description of the psychological therapies was also mixed in quality. There were a range of options for students to choose from and most focused on applications of CBT and its derivatives such as coping strategy enhancement or family based interventions. Better answers shaped descriptions of CBT specifically to the symptoms of schizophrenia (eg logical disputing to challenge delusional beliefs). Weaker students provided generic descriptions of CBT with little attempt to apply these to the unique symptoms/features of schizophrenia and were awarded basic marks.

Students who selected psychodynamic therapies were less successful in both their description and evaluation of these techniques. Descriptions were largely generic, often adding little detail beyond that covered at AS with little understanding that psychodynamic therapies are no longer considered generally suitable for schizophrenia or recommended by NICE. This approach gained rudimentary AO1 credit. Many became sidetracked into generic evaluations of psychodynamics, losing the focus on therapy.

The AO2 tended to be more thorough on the biological therapies and there were some useful discussions of outcomes research of different generations of anti-psychotics. In better answers, evaluation was clearly organised around three main areas, appropriateness, effectiveness and ethical issues. Weaker students often struggled to get beyond the level and type of evaluation required at AS level when discussing drug treatments. Many made statements, for example regarding side effects or costs of treatment that were imprecise and lacked elaboration or evidence. Weaker students showed little realisation that treatment is free at the point of delivery in the UK. Statements of this nature are classed as basic or rudimentary commentary and attract minimal credit. Outcome studies were few and far between in Question 01. Students should be encouraged to include outcomes data when discussing treatments on all topics for Section A.

### **Model answer**

The most commonly used biological treatments used are antipsychotics. One of the biological explanations of the schizophrenia is that the level of dopamine is higher in schizophrenics than in non-schizophrenics or that

they have too many dopamine receptors or that these receptors are too sensitive. We cannot modify the amount of dopamine present in the synaptic gaps, the number of receptors or their sensitivity so the aim of antipsychotic drugs is to block these receptors. /

There are two types of antipsychotic drugs: the neuroleptics drugs, first generation antipsychotics, for example chlorpromazine which bind to the dopamine receptors without stimulating them. By reducing the stimulation of the dopamine system they reduce or even eliminate the hallucinations and delusions experienced by schizophrenics. / The second generation antipsychotics also called atypical antipsychotics such as risperidone also bind to the dopamine receptors but then rapidly dissociate to allow normal dopamine transmission, they also act on the serotonin receptors. /

The effectiveness of neuroleptics has been shown by Sampath et al. (1992). They used patients who had been taking neuroleptics for 5 years. One group switched to a placebo drug whilst the other group continued to take the drugs, they found that 75% of the patients taking the placebo drugs relapsed within 1 year compared with only 33% of patients who continued taking the drug. //

However it also shows that these drugs are not a cure as 33% of the patients who carried on taking the drugs relapsed. A further limitation of these drugs is that they are not effective for negative symptoms such as catatonia. //

Atypical antipsychotics seem to be more effective against the negative symptoms. They are reported to be more effective for the 25% of patients who were not helped by the first generation of antipsychotics. Leuch et al (1999) conducted a meta-analysis and found that the atypical antipsychotics were superior to neuroleptics. //

In term of appropriateness, both types of antipsychotics can have severe side-effects. Neuroleptics can lead to tardive dyskinesia (uncontrollable movements of face, tongue, hands and feet). According to Glenmullen,2000 after 25 years of treatment T.D is present in up to 68% of the patients. This is irreversible in 75% of the cases. // Atypical antipsychotics can lead to agranulocytosis, a disorder in which the number of granulocytes (white blood cells) is dangerously reduced. This can be fatal. //

Furthermore drug treatment require compliance from patients who have cognitive deficits and might find it difficult to keep to the prescribed schedule which could lead to a cycle of rehospitalisation which is expensive for society and distressing for the patient and his family, the "revolving door syndrome". The drugs can be given by slow release injection but considering the possibility of severe side-effects the appropriateness of this type of treatment is questionable. //

Furthermore the appropriateness of the treatment could be questioned in situations where the disorder has been triggered by social factors for example such as high stress as it places the focus on the patient but does not address the difficulties which have lead to the development of the disorder so when the patient improves and goes back to his environment he/she is likely to relapse. //

Cognitive behavioural therapy (CBT) is a psychological therapy used in the treatment of schizophrenia. It is derived from the cognitive explanation. It assumes that schizophrenia like any other mental disorders is due to

faulty thinking therefore the aim of the therapy is to challenge the schizophrenic distorted thinking and beliefs such as hallucinations and delusions and modify this thinking. /

The form of CBT used with schizophrenics is called personal therapy. The therapist first aims to develop a trusting and respectful relationship with the patient. Then the patient explains his/her confusing, distressing experiences i.e. abusive voices.

Together they work at uncovering any patterns or triggers in the client's distressing experiences the therapist might ask questions such as "How long do you think others have been talking about you?" . /

The therapist then challenges gently the client's beliefs i.e. that people want to harm him/her by asking questions such as "Are there any other possible explanations for what happened?".

Then, in partnership, they try to develop strategies to deal with the distressing experiences. /

CBT takes place in one hour long sessions and 16-20 sessions are usually thought to be necessary although some patients will require much longer. /

The effectiveness of CBT is supported by Tarrrier et al (2000) found that people with Sz receiving 20 sessions of Personal therapy in 10 weeks along with drug therapy, followed by 4 booster sessions the following year, did better than the Sz on drug alone, 1/3 of patients achieved 50% reduction of psychotic experiences, 15% were free of positive symptoms. None of the patients on drugs alone were completely free of positive symptoms. The difference in outcome was still present a year later. //

However the sample used in the study was carefully selected: the patients included in the sample were patients who took their drugs (so excluded patients who refuse medication). Furthermore patients who were too confused, agitated or paranoid were not included as they would not have benefitted from CBT so CBT might be appropriate only for a certain type of patients not for all Sz. //

Lewis et al.( 2002) also support the effectiveness of CBT as he found that if it is used during the first episode it shortens the length of that episode but 18 months later the patients suffer the same relapse rate as patients who did not have CBT. This suggests that CBT might be more effective at the onset of the disorder. //

However a meta-analysis carried out by NICE (2009) on 2118 patients did not show statistically significant differences between CBT and standard care for outcomes related to mortality (suicide), relapse or treatment adherence. This cast doubt on the effectiveness of CBT in the treatment of schizophrenia. //

The evidence for the effectiveness of CBT as a treatment of schizophrenia is mixed at best but it has been argued that it is more effective for the positive symptoms such as auditory hallucinations than for negative symptoms such as catatonia. //

Furthermore it could be argued that it might not be CBT itself which causes an improvement but the relationship between the therapist and the client. Schizophrenics are often rejected and when they talk about their symptoms people tend to feel uncomfortable but the therapist is interested and is willing to help, this in itself might be helpful and lead to improvement. //

This treatment is not a cure and in Tarrrier's research only 15% of patients was free of positive symptoms however it helps some patients and can help reduce the amount of drugs necessary to reduce the symptoms and has not negative physical side-effects. //

It is often used in conjunction with drugs which make it difficult to assess its effectiveness however Kuipers et al. (1999) found a significant reduction in the positive symptoms when CBT and drug treatment were combined //.

**1170 words**