

# AQA Psychology A-Level

## Option 2: Schizophrenia Essay Plans

This work by [PMT Education](https://www.pmt.education) is licensed under [CC BY-NC-ND 4.0](https://creativecommons.org/licenses/by-nc-nd/4.0/)



**Discuss issues of reliability and validity associated with the classification and or diagnosis of schizophrenia. (16 MARKS)**

<b>A01</b>	<ul style="list-style-type: none"> <li>• Reliability refers to a diagnosis being repeatable, clinicians must be able to reach the same conclusion at two separate points in time. According to Reiger, the diagnosis of schizophrenia has a Kappa Score of 0.46, which indicates sub par reliability.</li> <li>• Luhrmann et al (2015), found through interviewing schizophrenics from Ghana, India and the US that the 'harsh auditory hallucinations' that are characteristic of schizophrenia may not actually be a reliable method of classifying schizophrenia. This may be due to differences in cultural perception of the symptom.</li> <li>• Validity refers to a diagnosis being valid, an issue with validity is gender bias. This occurs when the diagnosis is dependent on the gender of an individual, Broverman (1970) found that clinicians in the US equate healthy behaviour as being healthy male behaviour. This results in females being more likely to be pathologised.</li> <li>• Another issue with validity is symptom overlap, with Eliason and Ross finding that people with dissociative identity disorder had more symptoms of schizophrenia than those with schizophrenia.</li> <li>• A final issue with validity is comorbidity, this refers to the extent that two or more conditions occur simultaneously in a patient. Buckley estimated that comorbid depression occurs in 50% of schizophrenic patient.</li> </ul>
<b>A03</b>	<ol style="list-style-type: none"> <li>1. Diagnosis of schizophrenia are often unreliable. This is due to discrepancies in classifying symptoms, with Mojabi and Nicholson (1995) finding that 50 psychiatrists failed to differentiate between bizarre and non-bizarre delusions. As reliability is low, patients may receive an incorrect diagnosis. This implies that modifications may need to be made to the methods of diagnosis and classification.</li> <li>2. Further evidence exists to suggest that diagnosis of schizophrenia are unreliable. Rosenhan conducted a study in which pseudo patients were all given consistently the same incorrect diagnosis of schizophrenia. But, after the premise of the study was revealed, new pseudo patients were sent to hospitals, but were not admitted despite presenting the same symptoms. This suggests that although the study is artificial, the diagnosis of schizophrenia is in real life may be subject to conditions.</li> <li>3. Furthermore, the diagnosis of schizophrenia has little predictive validity as patients do not share similar prognosis. Harrison (2001) found that 30% of patients showed improvement in some cases, but only 10% in others. It is therefore, impossible to predict with certainty whether someone will recover from schizophrenia.</li> <li>4. That being said, the idea of gender bias has been supported research. Loring and Powell found that 59% of case studies when male or no gender was given were given a diagnosis. This was in comparison to 20% when they were said to be female.</li> </ol>



## Discuss the biological explanations of schizophrenia. (16 MARKS)

<b>AO1</b>	<ul style="list-style-type: none"> <li>• It has been shown that schizophrenia has a tendency to run in families, with those being genetically related being more likely to develop the disorder than those related by marriage. A study conducted on twins by Joseph (2004) calculated that the concordance rate for m/z twin is 40.4% compared to 7.4% for d/z twins.</li> <li>• An alternative explanation is the dopamine hypothesis. Dopamine is a neurotransmitter, when in excess seemingly causes the positive symptoms of schizophrenia. Davis and Kahn proposed a revised dopamine hypothesis in which they suggested that the positive symptoms of schizophrenia are due to an excess of dopamine in subcortical regions of the brain. Conversely, they suggested that the negative symptoms are due to a lack of dopamine in the mesolimbic pathway.</li> <li>• Deficits to the hippocampus and its links to regions of the brain like the prefrontal cortex have been implicated in the onset of schizophrenia, with Gotto and Grace (2008) finding that hippocampal dysfunction influences a reduction in dopamine release. This in turn affects processing in the PFC, causing the cognitive symptoms of the disorder.</li> </ul>
<b>AO3</b>	<ol style="list-style-type: none"> <li>1. The difference in concordance found by Joseph may be due to m/z twins being treated more similarly. The effects of the environment are hard to remove from such studies, its as a result hard to distinguish the cause of the high concordance rate- genes, or the environment.</li> <li>2. Attempts to remove the effects of the environment come in the form of adoption studies. But for these to be acceptable, an assumption must be made that adoptees have not been selectively placed. Kringlen (1987) and Joseph (2004) found that prospective parents often receive information about the child prior to adoption.</li> <li>3. Noll (2009), however, claims there is evidence against the revised dopamine hypothesis. He argues that antipsychotics do not alleviate positive symptoms in a third of patients, as these drugs aim to normalise dopamine levels, their failure brings the link between dopamine and positive symptoms into question.</li> <li>4. The Neural explanations can allow for early detection and therefore treatment. Addington et al (2015) completed a longitudinal study in which brain scans were done, this suggests that brain tissue can be predicted, allowing patients to be treated before symptoms develop.</li> </ol>



## Discuss psychological explanations for schizophrenia. (16 MARKS)

<b>AO1</b>	<ul style="list-style-type: none"> <li>• The family dysfunction theory suggests that particular family styles can cause or exacerbate schizophrenia in vulnerable individuals. The Double Bind theory suggests that children who frequently receive contradictory information from their parents are likely to develop schizophrenia. This is because such messages prevent the development of an internal coherence construction.</li> <li>• Another family communication style is expressed emotion, in which psychiatric patients have members who refer to them in a hostile/critical way. Linzen (1997) says high levels of EE are a key factor in increasing relapse rates in schizophrenic patients.</li> <li>• Cognitive explanations suggest that delusions are formed when patients have their interpretations of their experiences controlled by inadequate processing of information.</li> <li>• Hallucinations, according to the cognitive explanation experience hypervigilance due to an excessive focus on auditory stimuli. This is not corrected as schizophrenics do not reality test as non-schizophrenics do.</li> </ul>
<b>AO3</b>	<ol style="list-style-type: none"> <li>1. However, not all individuals have the same susceptibility to the effects of a high EE environment. It was found by Altorfer et al (1998) found that a quarter of their patients showed no response to the stressful comments made by their relatives.</li> <li>2. Tienari et al (1994) found that adopted children with schizophrenic patients were only more likely to also receive a diagnosis if their family environment was described as disturbed. This provides evidence for family dysfunction being a causal factor in the onset of schizophrenia.</li> <li>3. The cognitive explanations have been criticised for not being able to explain all aspect of the disorder, like neurochemical changes in the brain. This suggests that cognitive explanations alone are not sufficient to explain the onset of schizophrenia.</li> <li>4. It was found by Sarin and Warin (2014) found through reviewing research that the positive symptoms of schizophrenia may be caused by faulty thinking. This provides evidence in support of the cognitive theory of schizophrenia.</li> </ol>



**Outline and evaluate drug therapies in the treatment of schizophrenia. (16 MARKS)**

<b>A01</b>	<ul style="list-style-type: none"> <li>• Typical antipsychotics are known as conventional antipsychotics, these are dopamine antagonists that bind to, but don't stimulate dopamine receptors. This is a result of binding to D2 receptors in the mesolimbic pathway, and are believed to minimise the positive symptoms of schizophrenia.</li> <li>• These bind to 60-70% of D2 receptors in the mesolimbic pathway, but also bind to a similar amount of these receptors in the brain, which leads to extrapyramidal side effects. As a result, the use of the typical antipsychotics is associated with a high cost.</li> <li>• Atypical antipsychotics carry a lower risk of extrapyramidal side effects and are believed to also have an impact on the negative and cognitive symptoms of schizophrenia. These have a lower affinity for dopamine receptors, and a higher affinity for serotonin receptors.</li> <li>• It is this lower affinity for dopamine receptors that is believed to cause the fewer side effects.</li> </ul>
<b>A03</b>	<ol style="list-style-type: none"> <li>1. A meta-analysis of 56 studies by Leucht et al (2015), showed that patients who had been stabilised and continued with both atypical and typical antipsychotics had lower relapse rates than those who were given placebos after their antipsychotics, showing their effectiveness.</li> <li>2. Antipsychotics are effective, but they are not without their risk, side effects such as extrapyramidal ones can prevent patients from persisting with their treatment. As a result, their overall effectiveness at treating schizophrenia is reduced.</li> <li>3. As atypical antipsychotics do not run the same risk of side effects, people are more likely to persist in their treatment. Crossley did find that they are just as effective as typical antipsychotics, but they had side effects like headaches which are far less severe than tardive dyskinesia associated with typical antipsychotics.</li> <li>4. Antipsychotics are a preferred method of treatment, due them being not only effective, but cheaper than other forms of treatment such as CBTp. In addition to this, they also require less effort on the part of the patient, increasing the chances of them persisting in the treatment.</li> </ol>



## Outline and evaluate cognitive behavioural therapy as used in the treatment of schizophrenia. (16 MARKS)

<b>A01</b>	<ul style="list-style-type: none"> <li>• CBT has been adapted into Cognitive Behavioural Therapy for Psychosis, this works by correcting the distorted beliefs that influence their feelings, causing maladaptive behaviour. Schizophrenics have faulty interpretations, and CBTp is used to try and correct these faulty interpretations. It is recommended by NICE, that patients complete 10 sessions of CBTp.</li> <li>• CBTp involves:             <ul style="list-style-type: none"> <li>○ Encouraging patients to find the origins of their symptoms.</li> <li>○ They are also taught to evaluate the content of their delusions, using the ABC model.</li> <li>○ Behavioural assignments may be set to improve their level of functioning.</li> <li>○ The patient also develops alternatives to their previous maladaptive beliefs, using mechanisms already in their mind.</li> </ul> </li> </ul>
<b>A03</b>	<ol style="list-style-type: none"> <li>1. CBTp has been found to be more effective in reducing the rate of rehospitalisation for up to 18 months. These comparisons were made against standard care- drug therapy alone. This provides evidence for their success.</li> <li>2. The effectiveness of the treatment seems to be dependent on the stage of the disorder. It was found by Addington and Addington (2005) claim that during the more acute stages of the disorder, CBTp is no effective- it is only after drugs have been taken, is it useful. This suggests that it's not a viable form of treatment for all patients, limiting its effectiveness.</li> <li>3. This therapy has been criticised for being time consuming and for requiring a great deal of effort on the part of the clients. As a result, they are less likely to persist in it, reducing its overall effectiveness.</li> <li>4. This effectiveness is also limited by its lack of availability, with only 1 in 10 of people in the UK who could potentially benefit it from getting access to it. Its lack of availability is not a procedural fault of CBTp, as research has shown it to be effective in improving the prognosis of schizophrenic individuals.</li> </ol>



**Outline and evaluate family therapy as used in the treatment of schizophrenia.  
(16 MARKS)**

<b>A01</b>	<ul style="list-style-type: none"> <li>● Family therapy is offered for a duration of 3-12 months for at least 10 sessions, in an attempt to reduce the level of expressed emotion within the family. It is estimated by Garrety et al (2005) that the relapse rate is reduced from 50% to 25% when family therapy is used, instead of standard care alone.</li> <li>● Family therapy typically involves:           <ul style="list-style-type: none"> <li>○ Psychoeducation- informing family members about schizophrenia.</li> <li>○ Alliance Formation- giving family members information about how to support someone with schizophrenia.</li> <li>○ Maintaining reasonable expectations- giving family more information about the prognosis of their relative.</li> <li>○ Encouraging more effective communication amongst family, to reduce EE.</li> </ul> </li> <li>● In a study by Pharaoh et al (2010) in which RCTs were used, family therapy was shown to improve the mental state of patients, as well as increase their compliance with medication.</li> <li>● There was also a reduction in the risk of relapse after treatment for at least 24 months.</li> </ul>
<b>A03</b>	<ol style="list-style-type: none"> <li>1. The studies analysed by Pharaoh claimed to have used RCTs, but subsequent analysis by Wu et al (2006) noted that 53 of these studies showed that random allocation was not used. This discredits the study, bringing into question whether or not family therapy is actually effective.</li> <li>2. But family therapy is a cost effective form of treatment, especially in comparison to standard care alone. In addition to this, the cost of hospitalisation is reduced as family therapy has been associated with the reduction of relapse rates in schizophrenia.</li> <li>3. Family therapy has also been linked to improving the prognosis of schizophrenia, as well as being beneficial to the family of the schizophrenic individuals. Cobban et al (2013), analysed the results of 50 family studies and 60% of these reported a significant positive impact on the family following the family intervention.</li> <li>4. The quality of the evidence used to support family therapies is often questionable, small-scale studies are used often to support the benefits of psychological therapies. These studies lack randomised controls, meaning the effectiveness of these therapies may be overestimated by the studies.</li> </ol>



**Discuss token economies as used in the management of schizophrenia. (16 MARKS)**

<b>AO1</b>	<ul style="list-style-type: none"> <li>• Token economy was developed by Aylon &amp; Azrin (1968) and was originally used on a ward with female schizophrenic patients. They were given tokens after they had completed tasks like making their beds, the tokens were exchanged for rewards like watching a movie. The use of tokens improved the number of desirable behaviours performed daily.</li> <li>• The tokens are given value by being presented alongside the primary reinforcing stimulus- the reward. Through this, the neutral tokens acquire the same reinforcing qualities as the stimulus, making them secondary reinforcers.</li> <li>• The exchange of tokens for rewards is the most important part in token economy, it was found by Kazdin (1977) that the effectiveness of the tokens decreases the more time passes between the target behaviour and the presentation of the token and the exchange of reinforcers.</li> </ul>
<b>AO3</b>	<ol style="list-style-type: none"> <li>1. Token economy is supported by research by Dickerson (2015), 11 of the 13 studies reported beneficial effects of the use of token economy. It was concluded that these do provide evidence to support effectiveness of the use of token economy, particularly in increasing the adaptive behaviour of schizophrenic patients.</li> <li>2. Studies on token economy tend to be uncontrolled, as found by Comer (2013). This failing causes misleading results, as patient's past improvements can only be compared with their past behaviours, rather than a control group, there could be other factors improving their behaviour.</li> <li>3. Token economy only appears to be effective at reducing negative symptoms, only appears to be as such within a hospital setting. Outpatients, can only be monitored for a few hours a day, as a result, their positive results cannot be maintained after the patients leave the psychiatric ward.</li> <li>4. The use of token economy has been criticised for being unethical, for it to work, psychiatrists have to be in control over food, privacy and other things that act as primary reinforcers. This contradicts the notion that all humans have rights to these things without having to earn them, bringing into question whether such a treatment should be used when it violates basic human rights.</li> </ol>





## Outline and evaluate the diathesis-stress model of schizophrenia. (16 MARKS)

<p><b>AO1</b></p>	<ul style="list-style-type: none"> <li>• The diathesis-stress model views schizophrenia as being the result of an interaction between biological (diathesis) and environmental (stress) influences.</li> <li>• Support for this theory comes from family studies, with Gottesman reporting that identical twins are more likely to develop schizophrenia than non identical twins.</li> <li>• But concordance is not 100%, even for identical twins, indicating that there must be other factors- environmental factors that may cause the onset of schizophrenia.</li> <li>• Environmental factors include living in urbanised environments and suffering childhood traumas. Varese et al (2012) found children who had experienced severe trauma prior to turning 16 were more likely to develop schizophrenia.</li> <li>• Similarly, Vassos et al (2012) found that those living in urbanised environments were 2.57 times more likely to develop schizophrenia- both studies noted though that this was dependent on genetic factors.</li> <li>• A study by Tienari et al (2014) provides evidence in support of the diathesis-stress model. Adopted children of biological schizophrenic mothers were over three times more likely to develop schizophrenia the the control group of adopted children with non-schizophrenic mothers.</li> <li>• Tienari did find that being adopted into low OPAS scoring families acted as a protective measure against schizophrenia- even in the high risk group.</li> </ul>
<p><b>AO3</b></p>	<ol style="list-style-type: none"> <li>1. This study is often cited as research to support the diathesis-stress model, but the study has methodological limitations that discredit its use as support. The OPAS assessment was only conducted at one point in time, which Tienari noted could have resulted in a failing to accurately reflect developmental changes in family functioning.</li> <li>2. Determining causal stress is difficult to do, with Hammen believing that maladaptive methods of coping with stress in childhood meant that children fail to develop effective coping skills compromising their resilience. As a result, they have stressful experiences throughout their childhood, making it difficult to determine causal stress.</li> <li>3. Roman-Clarkson (1990), found no urban-rural differences in mental health amongst women in New Zealand. But, Paykei (2000) found a difference and that these differences disappeared after adjusting for the socioeconomic differences for the groups, suggesting that urbanisation as a causal factor schizophrenia is an oversimplification.</li> <li>4. This model has implications for treatment, as although genetic vulnerability is hard to control, other factors can be controlled so that individuals with a genetic vulnerability do not develop schizophrenia.</li> </ol>

