



Schizophrenia

A-level Revision Notes AQA(A)

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Exam Advice

You MUST revise everything - because the exam board could choose any question, however, it does make sense to spend more time on those topics which have not appeared for a while.

Exam Tip

With these particular questions there is a sizeable risk that people don't understand the difference between the questions, and then write about the wrong thing. Make sure you know which is which, for example do you understand the difference between "genetic explanation" and "neural correlates explanation", and do you have a model essay for each?

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

Section 1: Diagnosis and Classification of Schizophrenia

Classification is the process of organising symptoms into categories based on which symptoms cluster together in sufferers. Psychologists use the DSM and ICD to diagnose a patient with schizophrenia.

Diagnosis refers to the assigning of a label of a disorder to a patient. The ICD-10 (only

negative symptoms need to be present) is used worldwide and the DSM-5 (only positive symptoms need to be present) is used in America.

In order to diagnose Schizophrenia the Mental Health Profession developed the DSM (Diagnostic and Statistical Manual) still used today as a method of classifying mental disorders (particularly in the USA).

It is also used as a basis for the ICD (International Classification of Diseases) used by the World Health Organisation in classifying all disorders (mental and physical).

Note: you may come across the terms DSM-IV and ICD-10. These refer to the latest editions of the two classification systems.

Positive Symptoms

an excess or distortion of normal functions: including hallucinations and delusions.

Positive symptoms are an excess or distortion of normal functions, for example hallucinations, delusions and thought disturbances such as thought insertion.

AO1

- Hallucinations are usually auditory or visual perceptions of things that are not present. Imagined stimuli could involve any of the senses. Voices are usually heard coming from outside the person's head giving instructions on how to behave.
- Delusions are false beliefs. Usually the person has convinced him/herself that he/she is someone powerful or important, such as Jesus Christ, the Queen (e.g. Delusions of Grandeur). There are also delusions of being paranoid, worrying that people are out to get them.
- Psychomotor Disturbances: Stereotypical - Rocking backwards and forwards, twitches, & repetitive behaviors. Catatonia- staying in position for hours/days on end, cut off from the world.

Negative Symptoms

where normal functions are limited: including speech poverty and avolition.

Negative symptoms are a diminution or loss of normal functions such as psychomotor disturbances, avolition (the reduction of goal-directed behaviour), disturbances of mood and thought disorders.

AO1

- Thought disorder in which there are breaks in the train of thought and the person appears to make illogical jumps from one topic to another (loose association). Words may become confused and sentences incoherent (so called 'word salad'). Broadcasting is a thought disorder whereby a person believes their thoughts are being broadcast to others, for example over the radio or through TV. Alogia - aka speech poverty – is a thought disorder where correct words are used but with little meaning.
- Avolition: Lack of volition (i.e. desire): in which a person becomes totally apathetic and sits around waiting for things to happen. They engage in no self motivated behavior. Their get up and go has got up and gone!

Classification AO3

Slater & Roth (1969) say that hallucinations are the least important of all the symptoms, as they are not exclusive to schizophrenic people.

Classification and diagnosis does have advantages as it allows doctors to communicate more effectively about a patient and use similar terminology when discussing them. In addition, they can then predict the outcome of the disorder and suggest related treatment to help the patient.

Scheff (1966) points out that diagnosis classification labels the individual, and this can have many adverse effects, such as a self-fulfilling prophecy (patients may begin to act how they are expected to act), and lower self-esteem.

Ethics – do the benefits of classification (care, treatment, safety) outweigh the costs (possible misdiagnosis, mistreatment, loss of rights and responsibility, prejudice due to labelling).

Reliability and Validity in Diagnosis and Classification of Schizophrenia

with reference to co-morbidity, culture and gender bias and symptom overlap.

Reliability - AO1

For the classification system to be reliable, different clinicians using the same system (e.g. DSM) should arrive at the same diagnosis for the same individual.

Reliability is the level of agreement on the diagnosis by different psychiatrists across time and cultures; stability of diagnosis over time given no change in symptoms.

AO3

Diagnosis of schizophrenia is difficult as the practitioner has no physical signs but only symptoms (what the patient reports) to make a decision on.

Jakobsen et al. (2005) tested the reliability of the ICD-10 classification system in diagnosing schizophrenia. A hundred Danish patients with a history of psychosis were assessed using operational criteria, and a concordance rate of 98% was obtained. This demonstrates the high reliability of the clinical diagnosis of schizophrenia using up-to-date classification.

Comorbidity describes people who suffer from two or more mental disorders. For example, schizophrenia and depression are often found together. This makes it more difficult to confidently diagnose schizophrenia. Comorbidity occurs because the symptoms of different disorders overlap. For example, major depression and schizophrenia both involve very low levels of motivation. This creates problems of

reliability. Does the low motivation reflect depression or schizophrenia, or both?

Gender bias: Loring and Powell (1988) found that some behavior which was regarded as psychotic in males was not regarded as psychotic in females.

Validity - AO1

Validity - the extent to which schizophrenia is a unique syndrome with characteristics, signs and symptoms.

For the classification system to be valid it should be meaningful and classify a real pattern of symptoms, which result from a real underlying cause.

AO3

The validity of schizophrenia as a single disorder is questioned by many. This is a useful point to emphasise in any essay on the disorder. There is no such thing as a 'normal' schizophrenic exhibiting the usual symptoms.

Since there are problems with the validity of diagnosis classification, unsuitable treatment may be administered, sometimes on an involuntary basis. This raises practical and ethical issues when selecting different types of treatment.

Problems of validity: Are we really testing what we think we are testing? In the USA only 20% of psychiatric patients were classed as having schizophrenia in the 1930s but this rose to 80% in the 1950's. In London the rate remained at 20%, suggesting neither group had a valid definition of schizophrenia.

Neuropsychologist Michael Foster Green suggests that neurocognitive deficits in basic functions such as memory, attention, central executive and problem solving skills may combine to have an outcome which we are labelling "Schizophrenia" as if it was the cause when in fact it is simply an umbrella term for a set of effects.

Predictive validity. If diagnosis leads to successful treatment, the diagnosis can be seen as valid. But in fact some Schizophrenics are successfully treated whereas others are not. Heather (1976) there is only a 50% chance of predicting what treatment a patient will receive based on diagnosis, suggesting that diagnosis is not valid.

Aetiological validity – for a diagnosis to be valid, all patients diagnosed as schizophrenic should have the same cause for their disorder. This is not the case with schizophrenia: The causes may be one of biological or psychological or both.

David Rosenhan (1972) famous experiment involving Pseudopatients led to 8 normal people being kept in hospital despite behaving normally. This suggests the doctors had no valid method for detecting schizophrenia. They assumed the bogus patients were schizophrenic with no real evidence. In a follow up study they rejected genuine patients whom they assumed were part of the deception.

Culture - One of the biggest controversies in relation to classification and diagnosis is to do with cultural relativism and variations in diagnosis. For example in some Asian countries people are not expected to show emotional expression, whereas in certain Arabic cultures public emotion is encouraged and understood. Without this knowledge a person displaying overt emotional behavior in a Western culture might be regarded as abnormal. Cochrane (1977) reported that the incidence of schizophrenia in the West Indies and the UK is 1 %, but that people of Afro-Caribbean origin are seven times more likely to be diagnosed as schizophrenic when living in the UK,

Cultural bias – African Americans and those of Afro-carribean descent are more likely to be diagnosed than their white counterparts but diagnostic rates in Africa and the West Indies is low - Western over diagnosis is a result of cultural norms and the diagnosis lacks validity.

Section 2: Biological Explanations for Schizophrenia

Genetics

Family studies find individuals who have schizophrenia and determine whether their biological relatives are similarly affected more often than non-biological relatives.

There are two types of twins – identical (monozygotic) and fraternal (dizygotic). To form identical twins, one fertilised egg (ovum) splits and develops two babies with exactly the

same genetic information.

AO1

- Gottesman (1991) found that MZ twins have a 48% risk of getting schizophrenia whereas DZ twins have a 27% risk rate. This is evidence that the higher the degree of genetic relatedness, the higher the risk of getting schizophrenia.
- Benzel et al. (2007) three genes: COMT , DRD4 , AKT1 - have all been associated with excess dopamine in specific D2 receptors, leading to acute episodes, positive symptoms which include delusions, hallucinations, strange attitudes.
- 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.
- 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.

AO3

Very important to note genetics are only partly responsible, otherwise identical twins would have 100% concordance rates.

One weakness of the genetic explanation of schizophrenia is that there are methodological problems. 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 demand characteristics.

A second weakness is the problem of nature-v-Nurture. 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 cannot give a full explanation of the disorder.

A final weakness of the genetic explanation of schizophrenia is that it is biologically reductionist. 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 behavior. 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 oversimplistic as schizophrenia is not due to a single gene.

The Dopamine Hypothesis

AO1

- 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.
- High dopamine activity leads to acute episodes, and positive symptoms which include: delusions, hallucinations, confused thinking.
- 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 behavior which is very similar to paranoid schizophrenia. Small doses given to people already suffering from schizophrenia tend to worsen their symptoms.
- 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).

AO3

One criticism of the dopamine hypothesis is there is a problem with the chicken and egg. Is the raised dopamine levels the cause of the schizophrenia, or is it the raised dopamine level the result of schizophrenia? It is not clear which comes first. This suggests that one needs to be careful when establishing cause and effect relationships in schizophrenic patients.

One of the biggest criticisms of the dopamine hypothesis came when Farde et al found no difference between schizophrenics' levels of dopamine compared with 'healthy' individuals in 1990.

Noll (2009) also argues around one third of patients do not respond to drugs which block dopamine so other neurotransmitters may be involved.

A final weakness of the dopamine hypothesis is that it is biologically deterministic. 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 freewill.

Neural Correlates

AO1

- Neural correlates are patterns of structure or activity in the brain that occur in conjunction with schizophrenia
- People with schizophrenia have abnormally large ventricles in the brain. Ventricles

are fluid filled cavities (i.e. holes) in the brain that supply nutrients and remove waste. This means that the brains of schizophrenics are lighter than normal. The ventricles of a person with schizophrenia are on average about 15% bigger than normal (Torrey, 2002).

AO3

A strength is that the research into enlarged ventricles and neurotransmitter levels have high reliability. 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 tested and re-tested the same results would be achieved.

Supporting evidence for the brain structure explanation comes from further empirical support 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 wider academic credibility for enlarged ventricles determining the likelihood of schizophrenia developing.

A second weakness of the neuroanatomical explanations is that it is biologically deterministic. 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 freewill.

Section 3: Psychological Explanations for Schizophrenia

Family Dysfunction

Family Dysfunction refers to any forms of abnormal processes within a family such as conflict, communication problems, cold parenting, criticism, control and high levels of expressed emotions.

These may be risk factors for the development and maintenance of schizophrenia.

AO1

- Laing and others rejected the medical / biological explanation of mental disorders. They did not believe that schizophrenia was a disease. They believed that schizophrenia was a result of social pressures from life. Laing believed that schizophrenia was a result of the interactions between people, especially in families.
- Bateson et al. (1956) suggested the double bind theory, which suggests that children who frequently receive contradictory messages from their parents are more likely to develop schizophrenia. For example parents who say they care whilst appearing critical or who express love whilst appearing angry. They did not believe that schizophrenia was a disease. They believed that schizophrenia was a result of social pressures from life.
- Prolonged exposure to such interactions prevents the development of an internally coherent construction of reality; in the long run, this manifests itself as typically schizophrenic symptoms such as flattening affect, delusions and hallucinations, incoherent thinking and speaking, and in some cases paranoia.
- Another family variable associated with schizophrenia is a negative emotional climate, or more generally a high degree of expressed emotion (EE). EE is a family communication style that involves criticism, hostility and emotional over-involvement. The researchers concluded that this is more important in maintaining schizophrenia than in causing it in the first place, (Brown et al 1958). Schizophrenics returning to such a family were more likely to relapse into the disorder than those returning to a family low in EE. The rate of relapse was particularly high if returning to a high EE family was coupled with no medication.

AO3

One strength of the double bind explanation comes from further empirical support provided by Berger (1965). They found that schizophrenics reported a higher recall of double bind statements by their mothers than non-schizophrenics. However, evidence may not be reliable as patient's recall may be affected by their schizophrenia. This suggests that there is wider academic credibility for the idea of contradictory messages causing schizophrenia.

A second strength of the research into expressed emotion (EE) is that it has practical applications. For example Hogarty (1991) produced a type of therapy session, which reduced social conflicts between parents and their children which reduced EE and thus relapse rates. This suggests that gaining an insight into family relationships allows psychiatric professionals to help improve the quality of patient's lives.

Individual differences – EE is associated with relapse but not all patients who live in high EE families relapse and not all patients in low EE families avoid relapse – Family dysfunction is an incomplete explanation for schizophrenia.

A weakness of the family relationships approach is that there is a problem of cause and effect. Mischler & Waxler (1968) found significant differences in the way mothers spoke to their schizophrenic daughters compared to their normal daughters, which suggests that dysfunctional communication may be a result of living with the schizophrenic rather than the cause of the disorder. This suggests that there is a problem of the chicken and egg scenario in relation to expressed emotion causing schizophrenia.

A second weakness of the double bind theory is that there are ethical issues. There are serious ethical concerns in blaming the family, particularly as there is little evidence upon which to base this. Gender bias is also an issue as the mother tends to be blamed the most, which means such research is highly socially sensitive. This suggests that the research therefore does not protect individuals from harm.

Cause and effect – It remains unclear whether cognitive factors cause schizophrenia or if schizophrenia causes these cognitions – Family dysfunction may not be a valid explanation for schizophrenia.

Cognitive explanations

including dysfunctional thought processing.

Cognitive approaches examine how people think, how they process information. Researchers have focused on two factors which appear to be related to some of the experiences and behaviors of people diagnosed with schizophrenia.

First, cognitive deficits which are impairments in thought processes such as perception, memory and attention. Second, cognitive biases are present when people notice, pay attention to, or remember certain types of information better than other.

AO1

Cognitive Deficits

- There is evidence that people diagnosed as schizophrenic have difficulties in processing various types of information, for example visual and auditory information. Research indicates their attention skills may be deficient – they often appear easily distracted.
- A number of researchers have suggested that difficulties in understanding other people's behavior might explain some of the experiences of those diagnosed as schizophrenic. Social behavior depends, in part, on using other people's actions as clues for understanding what they might be thinking. Some people who have been diagnosed as schizophrenic appear to have difficulties with this skill.
- Cognitive deficits have been suggested as possible explanations for a range of behaviors associated with schizophrenia. These include reduced levels of emotional expression, disorganised speech and delusions.

Cognitive Biases

- Cognitive biases refer to selective attention. The idea of cognitive biases has been used to explain some of the behaviors which have been traditionally regarded as 'symptoms' of 'schizophrenia'.

- - Delusions: The most common delusion that people diagnosed with schizophrenia report is that others are trying to harm or kill them – delusions of persecution. Research suggests that these delusions are associated with specific biases in reasoning about and explaining social situations. Many people who experience feelings of persecution have a general tendency to assume that other people cause the things that go wrong with their lives.

AO3

A strength of the cognitive explanation is that it has practical applications. Yellowless et al. (2002) developed a machine that produced virtual hallucinations, such as hearing the television telling you to kill yourself or one person's face morphing into another's. The intention is to show schizophrenics that their hallucinations are not real. This suggests that understanding the effects of cognitive deficits allows psychologists to create new initiatives for schizophrenics and improve the quality of their lives.

A final strength is that it takes on board the nurture approach to the development of schizophrenia. For example, it suggests that schizophrenic behavior is the cause of environmental factors such as cognitive factors.

One weakness of the cognitive explanation is that there are problems with cause and effect. Cognitive approaches do not explain the causes of cognitive deficits – where they come from in the first place. Is it the cognitive deficits which causes the schizophrenic behavior or is the schizophrenia that causes the cognitive deficits? This suggests that there are problems with the chicken and egg problem.

A second weakness of the cognitive model is that it is reductionist. The reason for this is because the approach does not consider other factors such as genes. It could be that the problems caused by low neurotransmitters creates the cognitive deficits. This suggests that the cognitive approach is oversimplistic when consider the explanation of schizophrenia.

Section 4: Drug Therapy: typical and atypical

antipsychotics

Drug therapy is a biological treatment for schizophrenia. Antipsychotic drugs are used to reduce the intensity of symptoms (particularly positive symptoms).

Typical Antipsychotics

AO1

- First generation Antipsychotics are called “Typical Antipsychotics” Eg. Chlorpromazine and Haloperidol.
- Typical antipsychotic drugs are used to reduce the intensity of positive symptoms, blocking dopamine receptors in the synapses of the brain and thus reducing the action of dopamine.
- They arrest dopamine production by blocking the D2 receptors in synapses that absorb dopamine, in the mesolimbic pathway thus reducing positive symptoms, such as auditory hallucinations.
- But they tended to block ALL types of dopamine activity, (in other parts of the brain as well) and this caused side effects and may have been harmful.

Atypical Antipsychotics

- Newer drugs, called “atypical antipsychotics” attempt to target D2 dopamine activity in the limbic system but not D3 receptors in other parts of the brain.
- Atypical antipsychotics such as Clozapine bind to dopamine, serotonin and glutamate receptors.
- Atypical antipsychotic drugs work on negative symptoms, improving mood, cognitive functions and reducing depression and anxiety.

- They also have some effect on other neurotransmitters such as serotonin. They generally have fewer side effects eg. less effect on movement Eg. Clozapine, Olanzapine and Risperidone.

AO3

Since the mid-1950s antipsychotic medications have greatly improved treatment. Medications reduce positive symptoms particularly hallucinations and delusions; and usually allow the patient to function more effectively and appropriately.

Antipsychotic drugs are highly effective as they are relatively cheap to produce, easy to administer and have a positive effect on many sufferers. However they do not "cure" schizophrenia, rather they dampen symptoms down so that patients can live fairly normal lives in the community.

Kahn et al. (2008) found that antipsychotics are generally effective for at least one year, but second- generation drugs were no more effective than first-generation ones.

Some sufferers only take a course of antipsychotics once, while others have to take a regular dose in order to prevent symptoms reappearing. There is a sizeable minority who do not respond to drug treatment. Pills are not as helpful with other symptoms, especially emotional problems. Older antipsychotics like haloperidol or chlorpromazine may produce side effects Sometimes when people with schizophrenia become depressed, so it is common to prescribe anti-depressants at the same time as the anti-psychotics.

All patients are in danger of relapsing but without medication the relapses are more common and more severe which suggests the drugs are effective.

Clozapine targets multiple neurotransmitters, not just dopamine, and has been shown to be more effective than other antipsychotics, although the possibility of severe side effects – in particular, loss of the white blood cells that fight infection Even newer antipsychotic drugs, such as risperidone and olanzapine are safer, and they also may be better tolerated. They may or may not treat the illness as well as clozapine, however.

Meta – Analysis by Crossley Et Al (2010) suggested that Atypical antipsychotics are no more effective, but do have less side effects.

Recovery may be due to psychological factors – The placebo effect is when patients'

symptoms are reduced because they believe that it should – However, Thornley et al carried out a meta-analysis comparing the effects of Chlorpromazine to placebo conditions and found Chlorpromazine to be associated with better overall functioning – Drug therapy is an effective treatment for SZ.

RWA – Offering drugs can lead to an enhanced quality of life as patients are given independence – Positive impact on the economy as patients can return to work and no longer need to be provided with institutional care.

Ethical issues – Antipsychotics have been used in hospitals to calm patients and make them easier for staff to work with rather than for the patients' benefit – Can lead to the abuse of the Human Rights Act (no one should be subject to degrading treatment).

Severe side effects – Long term use can result in tardive dyskinesia which manifests as involuntary facial movements such as blinking and lip smacking – While they may be effective, the severity of the side effects mean the costs outweigh the benefits therefore they are not an appropriate treatment.

Exam Tip

In most cases the original “typical antipsychotics” have more side effects, so if the exam paper asks for two biological therapies you can write about typical antipsychotics and emphasise the side effects, then you can write about the atypical antipsychotics and give them credit for having less side effects.

Section 5: Psychological Therapies for Schizophrenia

Family Therapy

Family therapy is a form of therapy carried out with members of the family with the aim of

improving their communication and reducing the stress of living as a family.

Family Therapy aims to reduce levels of expressed emotion, and reduced the likelihood of relapse.

Aims of Family Therapy

- To educate relatives about schizophrenia.
- To stabilize the social authority of the doctor and the family.
- To improve how the family communicated and handled the situation.
- To teach patients and carers more effective stress management techniques.

Methods used in Family Therapy

- Pharoah identified examples of how family therapy works: It helps family members achieve a balance between caring for the individual and maintaining their own lives, it reduces anger and guilt, it improves their ability to anticipate and solve problems and forms a therapeutic alliance.
- Families taught to have weekly family meetings solving problems on family and individual goals, resolve conflict between members, and pinpoint stressors.
- Preliminary analysis: Through interviews and observation the therapist identifies strengths and weaknesses of family members and identifies problem behaviors.
- Information transfer – teaching the patient and the family the actual facts about the illness, it's causes, the influence of drug abuse, and the effect of stress and guilt.
- Communication skills training – teach family to listen, to express emotions and to discuss things. Additional communication skills are taught, such as “compromise and negotiation,” and “requesting a time out” . This is mainly aimed at lowering expressed emotion.

A study by Anderson et al. (1991) found a relapse rate of almost 40% when patients had drugs only, compared to only 20 % when Family Therapy or Social Skills training were used and the relapse rate was less than 5% when both were used together with the medication.

Pharaoh et al. (2003) meta - analysis found family interventions help the patient to understand their illness and to live with it, developing emotional strength and coping skills, thus reducing rates of relapse.

Pharaoh identified examples of how family therapy works: It helps family members achieve a balance between caring for the individual and maintaining their own lives, it reduces anger and guilt, it improves their ability to anticipate and solve problems and forms a therapeutic alliance.

Economic Benefits: Family therapy is highly cost effective because it reduces relapse rates, so the patients are less likely to take up hospital beds and resources. The NICE review of family therapy studies demonstrated that it was associated with significant cost savings when offered to patients alongside the standard care – Relapse rates are also lower which suggests the savings could be even higher.

Lobban (2013) reports that other family members felt they were able to cope better thanks to family therapy. In more extreme cases the patient might be unable to cope with the pressures of having to discuss their ideas and feelings and could become stressed by the therapy, or over-fixated with the details of their illness.

Token Economy

AO1

- Token economies aim to manage schizophrenia rather than treat it.
- They are a form of behavioural therapy where desirable behaviours are encouraged by the use of selective reinforcement and is based on operant conditioning.
- When desired behaviour is displayed eg. Getting dressed, tokens (in the form of coloured discs) are given immediately as secondary reinforcers which can be

exchanged for rewards eg. Sweets and cigarettes. • This manages schizophrenia because it maintains desirable behaviour and no longer reinforces undesirable behaviour.

- The focus of a token economy is on shaping and positively reinforcing desired behaviors and NOT on punishing undesirable behaviors. The technique alleviates negative symptoms such as poor motivation, and nurses subsequently view patients more positively, which raises staff morale and has beneficial outcomes for patients.
- It can also reduce positive symptoms by not rewarding them, but rewarding desirable behavior instead. Desirable behavior includes self-care, taking medication, work skills, and treatment participation.

AO3

Paul and Lentz (1977) Token economy led to better overall patient functioning and less behavioral disturbance, More cost effective (lower hospital costs)

Upper and Newton (1971) found that the weight gain associated with taking antipsychotics was addressed with token economy regimes. Chronic schizophrenics achieved 3lbs of weight loss a week.

McMonagle and Sultana (2000) reviewed token economy regimes over a 15-year period, finding that they did reduce negative symptoms, though it was unclear if behavioral changes were maintained beyond the treatment programme.

It is difficult to keep this treatment going once the patients are back at home in the community. Kazdin et al. Found that changes in behavior achieved through token economies do not remain when tokens are with-drawn, suggesting that such treatments address effects of schizophrenia rather than causes. It is not a cure.

There have also been ethical concerns as such a process is seen to be dehumanising, subjecting the patient to a regime which takes away their right to make choices. In the 1950s and 60s nurses often “rewarded” patients with cigarettes. Due to the pivotal role of dopamine in schizophrenia this led to a culture of heavy smoking and nicotine addiction in psychiatric hospitals of the era.

Ethical issues – Severely ill patients can't get privileges because they are less able to comply with desirable behaviours than moderately ill patients – They may suffer from discrimination

Cognitive Behavioral Therapy

AO1

In CBT, patients may be taught to recognise examples of dysfunctional or delusional thinking, then may receive help on how to avoid acting on these thoughts. This will not get rid of the symptoms of schizophrenia but it can make patients better able to cope with them.

Central idea: Patients problems are based on incorrect beliefs and expectations. CBT aims to identify and alter irrational thinking including regarding:

- General beliefs.
- Self image.
- Beliefs about what others think.
- Expectations of how others will act.
- Methods of coping with problems.

In theory, when the misunderstandings have been swept away, emotional attitudes will also improve.

Assessment: The therapist encourages the patient to explain their concerns.

- describing delusions
- reflecting on relationships
- laying out what they hope to achieve through the therapy.

Engagement:

The therapist wins the trust of the patient, so they can work together. This requires honesty, patience and unconditional acceptance. The therapist needs to accept that

the illusions may seem real to the patient at the time and should be dealt with accordingly.

ABC: Get the patients to understand what is really happening in their life:

A: Antecedent - what is triggering your problem ?

B: behavior – how do you react in these situations ?

C: Consequences – what impact does that have on your relationships with others?

Normalisation:

Help the patient realise it is normal to have negative thoughts in certain situations. Therefore there is no need to feel stressed or ashamed about them.

Critical Collaborative Analysis:

Carrying on a logical discussion till the patient begins to see where their ideas are going wrong and why they developed. Work out ways to recognise negative thoughts and test faulty beliefs when they arise, and then challenge and re-think them.

Developing Alternative Explanations:

Helping the patient to find logical reasons for the things which trouble them Let the patient develop their own alternatives to their previous maladaptive behavior by looking at coping strategies and alternative explanations.

Another form of CBT: **Coping Strategy Enhancement (CSE)**

- Tarrier (1987) used detailed interview techniques, and found that people with schizophrenia can often identify triggers to the onset of their psychotic symptoms, and then develop their own methods of coping with the distress caused. These might include things as simple as turning up the TV to drown out the voices they were hearing!

- At least 73% of his sample reported that these strategies were successful in managing their symptoms.
- CSE aims to teach individuals to develop and apply effective coping strategies which will reduce the frequency, intensity and duration of psychotic symptoms and alleviate the accompanying distress. There are two components:
 1. Education and rapport training: therapist and client work together to improve the effectiveness of the client's own coping strategies and develop new ones.
 2. Symptom targeting: a specific symptom is selected for which a particular coping strategy can be devised. Strategies are practised within a session and the client is helped through any problems in applying it. They are then given homework tasks to practice, and keep a record of how it worked.

AO3

CBT does seem to reduce relapses and readmissions to hospital (NICE 2014) However the fact that these people were on medication and having regular meetings with doctors would be expected to have that effect anyway.

Turkington et al. (2006) CBT is highly effective and should be used as a mainstream treatment for schizophrenia wherever possible.

Tarrier (2005) reviewed trials of CBT, finding evidence of reduced symptoms, especially positive ones, and lower relapse rates.

Requires self-awareness and willingness to engage – Held back by the symptoms schizophrenics encounter – It is an ineffective treatment likely to lead to disengagement.

Lengthy – It takes months compared to drug therapy that takes weeks which leads to disengaged treatment as they don't see immediate effects - A patient who is very distressed and perhaps suicidal may benefit better in the short term from antipsychotics.

Addington and Addington (2005) claim that CBT is of little use in the early stages of an acute schizophrenic episode, but perhaps more useful when the patient is more calm and beginning to worry about how life will be after they recover. In other words, it doesn't cure schizophrenia, it just helps people get over it.

Research in Hampshire, by Kingdon and Kirschen (2006) found that CBT is not suitable for all patients, especially those who are too thought disorientated or agitated, who refuse medication, or who are too paranoid to form trusting alliances with practitioners.

As there is strong evidence that relapse is related to stress and expressed emotion within the family, it seems likely that CBT should be employed alongside family therapy in order to reduce the pressures on the individual patient.

Section 6: Interactionist Approach

The Interactionist approach acknowledges that there are a range of factors (including biological and psychological) which are involved in the development of schizophrenia.

The Diathesis-stress Model

AO1

- The DSM model states that both a vulnerability to SZ and a stress trigger are necessary to develop the condition.
- Zubin and Spring suggest that a person may be born with a predisposition towards schizophrenia which is then triggered by stress in everyday life. But if they have a supportive environment and/or good coping skills the illness may not develop.
- Concordance rates are never 100% which suggests that environmental factors must also play a role in the development of SZ. MZ twins may have the same genetic vulnerability but can be triggered by different stressors.
- Tienari Et. A. (2004): Adopted children from families with schizophrenia had more chance of developing the illness than children from normal families. This supports a genetic link. However, those children from families schizophrenia were less likely to develop the illness if placed in a “good” family with kind relationships, empathy, security, etc. So environment does play a part in triggering the illness.

AO3

Holistic – Identifies that patients have different triggers, genes etc. – Patients can receive different treatments for their SZ which will be more effective.

Falloon et al (1996) stress – such as divorce or bereavement, causes the brain to be flooded with neurotransmitters which brings on the acute episode.

Brown and Birley (1968) 50% people who had an acute schizophrenic episode had experienced a major life event in 3 weeks prior.

Substance abuse: Amphetamine and Cannabis and other drugs have also been identified as triggers as they affect serotonin and glutamate levels.

Vasos (2012) Found the risk of schizophrenia was 2.37 times greater in cities than it was in the countryside, probably due to stress levels. Hickling (1999) the stress of urban living made African-Caribbean immigrants in Britain 8 to 10 times more likely to experience schizophrenia. Faris and Dunham (1939) found clear pattern of correlation between inner city environments and levels of psychosis. Pederson and Mortensen (Denmark 2001) found Scandinavian villages have very LOW levels of psychosis, but 15 years of living in a city increased risk.

Fox (1990): It is more likely that factors associated with living in poorer conditions (e.g. stress) may trigger the onset of schizophrenia, rather than individuals with schizophrenia moving down in social status.

Bentall's meta analysis (2012) shows that stress arising from abuse in childhood increases the risk of developing schizophrenia.

Toyokawa, Et. Al (2011) Suggest many aspects of urban living – ranging from life stressors to the use of drugs, can have an effect on human epigenetics. So the stressors of modern living could cause increased schizophrenia in future generations.

About the Author

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2017 Exam Paper

2017 Mark Scheme

2017 Examiner Report

Assessment Objectives

AO1

Demonstrate knowledge

(a) demonstrate knowledge and understanding of scientific ideas, processes, techniques and procedures.

(b) show a knowledge and understanding of psychological theories, terminology, concepts, studies and methods.

AO2

Application of knowledge

(a) apply knowledge and understanding of scientific ideas, processes, techniques and procedures:

in a theoretical context

in a practical context

when handling qualitative data

when handling quantitative data

This skill area tests knowledge of research design and data analysis, and applying theoretical understanding of psychology to everyday/real-life examples.

AO3

Analyse, interpret and evaluate

(a) analyse, interpret and evaluate scientific information, ideas and evidence, including in relation to issues, to:

make judgements and reach conclusions

develop and refine practical design and procedures.

Examples of how you can score AO3 marks

Whether or not theories are supported or refuted by valid research evidence.

After describing a theory go on to describe a piece of research evidence saying, 'X's study supports/refutes this theory...' and then describe the research study.

Contextualising how the topic in question relates to broader debates and approaches in Psychology.

For example, would they agree or disagree with a theory or the findings of the study?

Animal Research - This raises the issue of whether it's morally and/or scientifically right to use animals.

The main criterion is that benefits must outweigh costs.

Animal research also raises the issue of extrapolation. Can we generalize from studies on animals to humans as their anatomy & physiology is different from humans?

General criticisms and/or strengths of theories and studies.

E.g. 'Bandura's Bobo Doll studies are laboratory experiments and therefore criticisable on the grounds of lacking ecological validity'.

To gain marks for criticising study's methodologies the criticism must be contextualised: i.e. say why this is a problem in this particular study.

'Therefore, the violence the children witnessed was on television and was against a doll not a human'.