Schizophrenia

Clinical characteristics:
Chronic: there are harmful and subtle changes in an apparently normal young person, who gradually loses drive and motivation then starts to drift away from friends. After **months or even years** of this decline in mental health, more obvious signs of disturbance such as delusional ideas or hallucinations appear.

Acute: obvious signs such as hallucinations can appear quite suddenly, usually after a stressful event. The individual shows very disturbed behaviour within a **few days.**

Symptoms of schizophrenia [ICD-10]

At least one symptom from section (1) or at least 2 symptoms from section (2). These have to be present for at least one month.

(1)

Thought control	**Thought withdrawal:** they believe their thoughts are extracted from their mind. **Thought insertion:** belief that thoughts are being inserted into their mind. **Thought broadcast:** belief that private thoughts have become accessible to other people.
Delusions of control, influence and passivity	A delusion is a distorted belief e.g. the schizophrenic may feel that they are not in control of their own thoughts, feelings and ability to make events happen.
Hallucinatory voices	These are voices that do not exist but feel real to the person hearing them.
Other persistent delusions	Belief that they have impossible powers and capabilities.

(2)

Persistent hallucinations	Hallucinations are distorted perceptions from the senses. They are perceptions which occur in the absence of stimuli and they most often take the form of auditory hallucinations such as hearing voices, however they can involve any of the senses.
Incoherent or irrelevant speech	Speech is jumbled so it becomes meaningless, where they often use made up words (called neologisms).
Catatonic behaviour	Unusual body movements, posture and uncontrolled limb movements.
Negative symptoms	Apathy and lack of motivation. **Flat affect:** reduced emotional response. Emotional displays are inappropriate.

Reliability issues concerning the diagnosis of schizophrenia

Differences between ICD-10 and DSM-TR-IV

•Duration: DSM requires symptoms to be present for 6 months. ICD requires 1 month.

•Emphasis: DSM emphasizes the course of the disorder and the accompanying functional impairment. ICD focuses more on first-rank symptoms (these are symptoms which are rarely found in disorders other than schizophrenia).

• Dimensions: DSM is multi-axial which means that various factors are taken into consideration before a diagnosis is made e.g. personality type.

Different types of schizophrenia: ICD-10 distinguishes 7 types, DSM has 5.

Paranoid schizophrenia:	Involves delusions of persecution and hallucinations. Reduction of negative symptoms such as lack of emotional expression and incoherent speech.
Hebephrenic schizophrenia	Behaviour is aimless and disorganised. Speech is incoherent. More lack of emotion/ display of emotion inappropriately.
Catatonic schizophrenia	Motor abnormality is the main characteristic. Strange postures and limb flailing. Negativism where they resist instructions to move.
Undifferentiated schizophrenia:	Individuals who have insufficient symptoms for any of the subtypes or too many symptoms to fit a particular category.
Post-schizophrenic depression:	Criteria for schizophrenia have been met in the last 12 months but are not currently present. Depressive symptoms are prolonged and severe.
Residual schizophrenia:	Criteria for schizophrenia have been met in the past but not at present. There have been many signs of negative symptoms in the past 12 months.
Simple schizophrenia:	Slow and progressive development of social withdrawal, apathy, poverty of speech and marked decline.

Type 1 and Type 2 syndromes: distinction made by Crow (1985)

Type 1 syndrome: an acute disorder characterised by positive symptoms e.g. hallucinations, delusions and disorganised speech.

Type 2 syndrome: a chronic disorder characterised by negative symptoms such as flattening of affect (emotions), apathy and poverty of speech.

Evaluating the use of **subtypes** in classification and diagnosis:

Type 1 and Type 2: Individuals can't easily be classified into one of these distinctions.

Blurred distinction between some subtypes: Reliability of diagnosis can be weakened where those distinguished as having one subtype of schizophrenia may later develop symptoms from another subtype.

Validity issues concerning the diagnosis of schizophrenia

Disorders similar to schizophrenia:
There are individuals who display symptoms similar to those of schizophrenia but don't meet the criteria e.g. schizoid personality disorder and schizotypal disorder. Validity is affected where these variations exist. It is difficult to diagnose a person displaying schizophrenia-like symptoms. The boundary between the classifications is blurred. This has led to criticisms concerning the portrayal by the diagnostic systems where schizophrenia is depicted as an absolute condition i.e. 'all-or-nothing'.

Dimensional or categorical disorder
Schizophrenia should be a dimensional disorder where its classification should relate to the **degree** to which problems are experience not the presence or absence of them e.g. some may experience hallucinations but have found ways to cope with them and so their functioning in life is not affected.

Schizophrenia as a multiple disorder
Individuals diagnosed with schizophrenia can present with different problems. This suggests there is no single underlying causal factor. Individuals don't respond in the same way to treatments. This suggests that there is a lack of validity in the diagnosis of schizophrenia. It is argued that each of the symptoms of schizophrenia should be seen as a disorder in its own right, with its own cause and treatment.

Differential diagnosis:
It is often difficult to distinguish between schizophrenia and seemingly unrelated syndromes e.g. those with temporal lobe epilepsy / experiences of drugs.

Dual diagnosis:
Co-morbidity is common, which is where one people diagnosed with one mental disorder simultaneously show symptoms of another. The multi-axial DSM encourages multiple diagnoses to be made where it might not always be appropriate.

Cultural variations:
Harrison et al. (1988): Schizophrenia is diagnosed more frequently in African Americans and African-Caribbean populations. It is difficult to determine whether this is due to greater genetic vulnerability or psychosocial factors (as being part of an ethnic minority). Misdiagnosis may occur where there is a misinterpretation of cultural differences in behaviour as being symptoms of schizophrenia.

Biological explanations of schizophrenia

Genetic explanation for schizophrenia:

•Evidence from family studies:

•First-degree relatives (offspring, parents and siblings) share 50% of their genes, second-degree relatives share 25%.

•Family studies involve the comparison of rates of schizophrenia in relatives of diagnosed cases compared with relatives of controls.

•There is evidence that the closer the biological relationship, the greater the risk of schizophrenia developing.

Support	Opposition
Kendler et al. (1985): first-degree relatives of those with schizophrenia are 18 times more at risk compared to the general population.	Family studies are conducted **retrospectively** when they are comparing a cross section of people who have already been diagnosed. Retrospective data is unreliable as problems in memory and records are likely. **Prospective studies** provide more reliable data as they follow people over time and can make comparisons before and after their condition occurs.
Key study: Kety et al. (1994) •High-risk group: 207 offspring of schizophrenic mothers. •Low-risk group: 104 control children with healthy mothers. •Children aged 10 to 18 and were matched on age/gender/socio-economic status. Follow up testing on children was conducted. • Schizophrenia was diagnosed in 16.2% of high-risk group. Only in 1.9% of low-risk group. **+** prospective study which follows the development of schizophrenia. **+** matching of risk factors in children	Can't differentiate between genetic and environmental influences as there is a shared environment.
	Many studies e.g. Kety et al., began before the more effective diagnostic systems were developed so it is possible that there was variation in symptoms for original diagnosis of schizophrenia.

Evidence from twin studies

•Twin studies compare the difference in concordance rates (the likelihood of both twins being affected with the disorder) between identical (Monozygotic- MZ) and non-identical (Dizygotic- DZ) twins.

•Only the MZ twins have identical genes.

• There is a higher concordance rate in MZ compared to DZ twins.

• MZ twins reared apart can be used to distinguish the effects of genetics and environment.

Support	Opposition
Gottesman and Shields (1982): 58% of twins who were reared apart, were concordant for schizophrenia.	Even MZ twins that are rear apart share the same **womb environment** before birth. The contribution of environmental factors can't be entirely discounted.
Fischer (1971): 9.4% of offspring from a non-affected MZ twin developed schizophrenia compared to 1% of general population.	Where twins are reared apart, such separation may be due to **family problems** which can influence their mental health.
Cardno et al. (1999): 40% of concordance rate in MZ twins compared with 5.3% in DZ twins.	

Evidence from adoption studies

•Adopted children who develop schizophrenia can be compared to their biological and adoptive parents.

•If schizophrenia has a genetic component, the development of the disorder should be maintained even if there is a change in environment, such as being raised by non-biological parents.

•Adoption studies attempt to highlight such genetic influence.

Support	Opposition
Tienari et al. (2000): the risk for developing schizophrenia was 4 times greater in adopted children with schizophrenic biological mothers compared to adopted children from biological mothers without schizophrenia.	**Wahlberg et al. (2000):** re-examined the Tiernari et al. data (2000) data and found a strong effect of environmental factors where those at risk of developing schizophrenia were adopted into families with poor communication.
Kety et al. (1994): study of a national sample from Denmark. High rates of schizophrenia were found in adopted children whose biological parents were schizophrenic.	**Longitudinal research:** the diagnostic criteria for schizophrenia are continually changing.
Tienari et al. (1987): adopted children who had schizophrenic mothers had a 7% chance of developing schizophrenia compared to 1.5% of the controls (who were also adopted children, but had non-schizophrenic mothers).	

Overall evaluation for genetic explanation of schizophrenia:

Support	Opposition
There is strong evidence that genetics are a risk factor for schizophrenia.	Even if a MZ twin has the disorder, the risk for the other twin is less than 50%, suggesting that genetic influences don't offer a complete explanation.
	89% of people with schizophrenia don't have a relative who has been diagnosed with the disorder.
	Research into the location of specific genes has not produced definitive results. It is impossible to understand the underlying mechanism that leads from the genetic risk to the disorder.

Biochemical explanations of schizophrenia

The dopamine hypothesis:

- Dopamine is a neurotransmitter (chemical that acts as a messenger to transmit impulses from one nerve cell to another across a synapse) that is found in the limbic system.
- Over-activity in the dopamine-controlled parts of the brain can result in schizophrenia.
- Phenothiazines which inhibit dopamine activity can reduce the symptoms of schizophrenia.
- L-dopa, a dopamine releasing drug, can cause schizophrenic symptoms in non-psychotic people.
- LSD/amphetamines increase dopamine activity and induce schizophrenic symptoms.

Support	Opposition
Falkai et al. (1998): examinations of brains from dead schizophrenic patients show that there is an excess of dopamine in the left amygdala.	It is difficult to determine if increases in dopamine found in brain regions are the result of schizophrenia or the cause of it.
Wong et al. (1986): PET scans show greater dopamine receptor density in the caudate nuclei in those with schizophrenia.	Drugs tend to be effective at alleviating positive symptoms of schizophrenia, but not negative symptoms, suggesting that all of the symptoms of schizophrenia may not be directly related to dopamine.
	Clozapine is effective in treating schizophrenia; however it changes levels in serotonin and not dopamine.
	Dopamine is unlikely to be the only factor in schizophrenia because it has also been implicated in mania and other disorders.
	The biochemical explanation is **reductionist** where stress / irrational thought processes are not taking into consideration.

Neuroanatomical explanations

Magnetic resonance imaging:

• A non-invasive technique where radio waves are recorded from the brain.
• The recordings are computerised and assembled into a 3D image of brain structures.
• These studies show definite structural abnormalities in the brains of patients with schizophrenia.

Support	Oppositional
Earlier research depended on post-mortem studies where it was difficult to determine if structural damage was a **causal** factor or whether it was the result of drug therapy/ natural progression of the disorder. The examination of living patients provides a clearer understanding of the causal relationship.	Brain imaging in relation to schizophrenia has been restricted mainly to those who have already been diagnosed so the direction of causality is difficult to determine.
Wood et al. (2005): 79 males who were considered to be high risk for schizophrenia were compared to 49 healthy males for their hippocampus size. The high risk individuals had significantly smaller hippocampi than the control group. The brain abnormality existed before the condition.	**Weinberger (1987):** there is inconclusive evidence concerning whether brain changes are before the onset of schizophrenia or whether they follow the clinical symptoms.
Buchsbaum (1990): abnormalities in the frontal and pre-frontal cortex, the basal ganglia, the hippocampus and the amygdala for patients.	**Flaum et al. (1995):** found no abnormalities in the temporal lobe regions of those with schizophrenia
Woodruff et al. (1997): significant reductions in the temporal lobe compared to controls.	
Castner et al. (1998): exposed monkeys to brain-damaging x-rays during foetal development. No ill effects were found during childhood, but during adolescence they developed symptoms of schizophrenia.	

Psychological explanations of schizophrenia

Psychosocial factors:

• Socioeconomic status: schizophrenia occurs more in lower socio-economic groups. This could be due to the group status being a risk factor for schizophrenia. Alternatively people with schizophrenia may no longer cope adequately with jobs and relationships and so they become part of lower socio-economic groups.
•Migrant populations: schizophrenia occurs more frequently in migrant populations especially African-Caribbean individuals. This may be due to psychosocial adversity and stress of living with racial discrimination.

Support	Opposition
Fox (1990): it is more likely that factors associated with living in poorer conditions e.g. stress may trigger the onset of schizophrenia compared to individuals with schizophrenia moving down in social status.	Racial bias may occur in the diagnosis of migrant populations so more of such people are diagnosed with schizophrenia.
	Unlikely that social class or economic statuses are anything more than contributory factors.

Family relationships:

•Disturbed patterns of communication within families might be a factor in the development of schizophrenia.

• However, the family may play a more important role in the course of a disorder rather than being the cause.

• Expressed emotion (EE) may be an important factor in maintaining schizophrenia.

• EE includes hostility, criticism, over-involvement and over-concern.

Support	Opposition
Vaughn and Leff (1976): 51% relapse in those of high-EE homes compared to 13% relapse for those in low-EE homes. Specifically, relapse rates increased with more face-to-face contact with high-EE relatives. Those who spent less time in the high EE environment were significantly less likely to relapse.	**Goldstein (1988):** Many patients with schizophrenia are away from their families and they are not less prone to relapse despite the lack of EE.
Brown (1972): patients with schizophrenia who returned to homes with high levels of EE showed a greater tendency to relapse than those returning to low-EE homes.	High EE may develop in the family as a response to the burdens of living with a person suffering from schizophrenia.
Miklowitz (2004): EE is a well-established maintenance model of schizophrenia. Treatment often includes programmes to educate family members to control levels of EE.	
Bateson et al. (1956): double-bind hypothesis: children are given conflicting messages from parents who express care, yet simultaneously are critical of the children. This leads to confusion, self-doubt and eventual withdrawal.	
Lidz et al. (1965): discord between parents was associated with schizophrenia in offspring.	

Cognitive explanations of schizophrenia

Attention:

The mechanisms that filter and process incoming stimuli are defective in the brains of schizophrenic patients.

•Individuals with schizophrenia can't focus attention selectively and this inability to filter means they attempt to process too much irrelevant information.

•They are inundated by external stimuli which they are unable to interpret appropriately.

Failure to activate schemas:

Hemsley (1993): In schizophrenia there is a breakdown in the relationship between information from memory and new information.

• Schemas are not activated so schizophrenics are subjected to sensory overload and they can't ignore which aspects of a situation to attend to and which to ignore on the basis of a schema.

•Superficial incidents are seen as highly relevant and significant.

•Internal thoughts are often not recognised as arising from memory and so are thought to be from an external source and experienced as auditory hallucinations.

Faulty cognitive processes

Frith (1992):

• Schizophrenia patients are unable to distinguish between actions that are brought about externally or generated internally.

• The symptoms of schizophrenia can be explained as the result of:

1) Inability to generate voluntary action
2) Inability to monitor voluntary action
3) Inability to monitor the beliefs and intentions of others

•These 3 factors are part of meta-representation which allows us to understand the beliefs and intentions of others.

• Faulty operation is due to functional disconnection between frontal areas of the brain concerned with action and more posterior areas of the brain that control perception.

Support	Opposition
Frith (1992): evidence where changes in cerebral blood flow in the brains of people with schizophrenia when they are engaged in specific cognitive tasks	Frith has been criticised as being reductionist where his theory fails to take into account the role of environmental factors.
Hemsley (1993): tentative research in animals has found a neurological relationship between problems in the hippocampus and schema activation failure.	Cognitive theories don't explain the causes, only the symptoms of schizophrenia.
Genetic support has been found where **Park et al. (1995)** found working memory deficits in people with schizophrenia and in their first-degree non-schizophrenic relatives.	

Biological therapies for schizophrenia

Drug therapy

• Antipsychotic drugs are mainly typical (affect dopamine) or atypical (affect serotonin levels).

• Dopamine plays an important role in schizophrenia development.

• Antipsychotic drug therapy attempts to reduce the number of dopamine receptor sites by blocking them.

• Phenothiazines are drugs which function by blocking dopamine receptors e.g. chlorpromazine.

• These drugs reduce the acute positive symptoms of schizophrenia, where maximum benefits are found within the first 6 months of use.

• Some drugs that block serotonin receptors are also used to treat schizophrenia e.g. clozapine.

Effectiveness

Support	Opposition
Julien (2005) clozapine has been found as an effective treatment of schizophrenia.	Antipsychotic drugs lack effectiveness in treating negative symptoms e.g. loss of motivation.
Antipsychotics produce a sedative effect and significantly reduce psychotic symptoms of hallucinations and delusions.	**Rzewuska (2002):** symptoms often return if the patient stops taking the drugs.
deLima et al. (2005): serotonin moderating drugs can alleviate negative symptoms	

Appropriateness

Support	Opposition
More recently developed drugs are more effective and cause fewer side effects	**Side effects**: symptoms similar to those of Parkinson's disease e.g. stiffness, immobility and tremors. This occurs in 30% of patients using antipsychotics (Gualtieri, 1991).
Kemp et al. (1998): motivation interviewing techniques improve compliance to therapy.	Antipsychotics that affect serotonin levels result in damage to the immune system
	Compliance: Davis et al. (1993): if antipsychotic drugs are stopped abruptly then symptoms recur. Patients often refuse to comply with treatment because of side effects.

Psychological therapies for schizophrenia

Family intervention/ therapy

- These aim to reduce levels of negative expressed emotion in a family.
- An educational element is involved where family members are given information about the disorder and ways of managing it e.g. improving communication styles, lowering expressed emotion, adjusting expectations and expanding social networks.

Support	Opposition
Effectiveness: Pharaoh et al. (2003) meta-analysis in which family interventions were found to be effective in reducing rates of relapse.	The meta-analysis revealed a wide range of outcomes so the results are not conclusive.
Appropriateness: It can improve compliance for taking medication.	The therapy can only be used for those who are in close contact with their families, which many schizophrenic patients aren't.
Pilling et al. (2002): Family intervention had preventative effects on psychotic relapse and medication compliance.	

Social skills training

- Individuals with schizophrenia tend to have problems with social skills such as interaction, self-care, coping with stressful situations and appraising social situations. Social skills training involves the teaching of such interpersonal skills.

Support	Opposition
It works appropriately with other therapies. Hogarty (2002): patients on medication that received social skills training relapse less than those on each treatment separately.	Gains are not maintained after the treatment has ended

Cognitive-behavioural therapy (CBT)

•The goal of CBT is to adjust the thinking patterns and alter the irrational beliefs of schizophrenia patients.

• Belief modification is a particular type of cognitive intervention: it teaches strategies to counter delusional beliefs and hallucinations. It involves cognitive challenge.

•Clients are taught to consider their negative responses to situations as hypotheses (ideas) rather than reality.

•They then learn to challenge their negative interpretations.

Effectiveness

Support	Opposition
Drury et al. (2000): immediate short-term gains were found with the use of belief modification.	**Drury et al. (2000):** the benefits of treatment were not maintained in the long term, as discovered in the follow-up 5 years later.
Jones et al. (2000): meta-analysis of trials of belief modification: it reduced the frequency and intensity of hallucinations.	
Turkington et al. (2000): CBT has a significant effect on positive and negative symptoms of schizophrenia. CBT can also be delivered effectively by psychiatric nurses in brief intervention programmes.	
Pilling et al. (2002): meta-analysis which found that CBT produced positive effects and had low dropout rates.	

Appropriateness

Support	Opposition
CBT is a collaborative therapy and involves active cooperation. This is more ethical than drug therapy where the patient becomes passive.	CBT can be seen as only normalising symptoms rather than curing them.

Specification: need to know

• Clinical characteristics of schizophrenia

• Issues concerning reliability and validity when diagnosing schizophrenia

• Biological explanations of schizophrenia e.g. genetic, biochemical and neuroanatomical

• Psychological explanations of schizophrenia e.g. psychosocial factors, family relationships, cognitive explanations

• Biological therapies for schizophrenia, including their evaluation in terms of appropriateness and effectiveness.

• Psychological therapies for schizophrenia including their evaluation in terms of appropriateness and effectiveness