Key point	Description	Evaluation (AO3)
Diagnosis and classification	• DSM-5 – positive symptoms must be present for diagnosis but in the ICD-10 2+ negative symptoms are needed • Positive symptoms include hallucinations (sensory experiences that have no basis in reality) and delusions (beliefs that have no basis in reality) • Negative symptoms include avolition (loss of motivation) and speech poverty (reduced frequency/quality of speech)	• Poor inter-rater reliability with different diagnostic tools to diagnose schizophrenia • Co-morbidity highlights the issue of diagnosing separate conditions • Symptom overlap with bipolar disorder questioning the validity of the diagnosis
Biological explanations for schizophrenia	• Gottesman found that there is a 48% concordance rate for schizophrenia in identical monozygotic twins • **Candidate genes** – In a genome wide study by Ripke et al (2014) they found that there are 108 separate genetic variations responsible for developing schizophrenia. These included controlling neurotransmitters. • **Dopamine hypothesis** – hyperdopaminergia in the subcortex (high levels of dopamine that has been linked to issues with the Brocas area) and hypodopaminergia in the cortex (low levels of dopamine responsible for negative symptoms) • **Neural correlates with negative symptoms** – negative correlation between ventral stratium and negative symptoms • **Neural correlates with positive symptoms** – lower levels in superior temporal gyrus and anterior cingulate gyrus associated with hallucinations.	• Supporting research e.g. Gottesman and Ripke • Antipsychotics work by reducing the level of dopamine therefore dopamine must be linked to the development of schizophrenia. • It's only correlational rather causational meaning validity cannot be established. • Other factors may play a role i.e. psychological explanations
Psychological explanations for schizophrenia • **SZ Mother** • **Double blind** • **Expressed emotion**	• Family dysfunction (The schizophregenic mother, double blind theory & expressed emotion) • **SZ mother** = Fromm-Reichmann (1948) states that the mother is cold, rejecting and controlling and creates a climate of tension and secrecy that leads to paranoid delusions that include SZ. • **Double blind** = the child receives mixed messages about their behaviour and the child is punished by the withdrawal of love. This makes them believe the world is dangerous and confusing and can create SZ symptoms such as disorganised thinking and paranoid delusions. • **Expressed emotion & SZ** = this is the level of emotion (mainly negative) expressed towards a patient by their carers. The high levels of stress can create a relapse in patients. This stress can also trigger the onset of SZ (diathesis stress model) It has 3 elements: 1) verbal criticism of the patient accompanied with violence 2) Hostility towards the patient including anger and rejection 3) emotional over-involvement in the life of the patient including needless self-sacrifice	• Read et al (2005) reviewed 46 studies of child abuse and SZ and found that 69% of adult women and 59% of adult males, with SZ had a history of physical abuse and/or sexual abuse in childhood. • Information from childhood is self reported therefore may be unreliable. Issues with sample sizes being unrepresentative. • Theories of family dysfunction are based on a judgment of the mother's personality = low validity. • Ethical issues with placing blame on parents for the development of SZ.
Psychological explanations for schizophrenia: **Cognitive explanations**	• Cognitive explanations focus on the mental processes involved with SZ • Frith et al (1992) identified 2 dysfunctional thought processing: • Metarepresentation = the ability to reflect on thoughts and behaviour. Patients with SZ have an inability to recognise internal thoughts as their own and instead believe it's others. This could explain the hallucinations and delusions • Central control = the ability to suppress automatic responses whilst we perform deliberate actions instead. Disorganised speech and thought disorder could result from the inability to stop automatic thoughts and speech triggered by thought e.g. SZ patients experience derailment in speech and thoughts because one word triggers associations and the patient cannot stop this automatic process.	• It's unclear whether SZ causes cognitive distortions or whether cognitive distortions cause SZ – causality is an issue which lowers internal validity. • Stirling et al (2006) found that 30 SZ patients took twice as long to name the colours of the ink in a stroop test. • Relies on introspection style methods - low valid

Key point	Description	Evaluation (AO3)
Biological therapies: **Drug therapy**	• **Typical antipsychotics** e.g. **chlorpromazine** (tablets/injection, taken daily, up to 1000mg) • The older version of antipsychotics – more side effects • Typical antipsychotics act as antagonists (chemicals that reduce the action of a neurotransmitter) in the dopamine system. They block the dopamine receptors and therefore reduce dopamine. According to the dopamine hypothesis, this antagonistic effect normalizes neurotransmission in key areas reducing symptoms like hallucinations. • **Atypical anti-psychotics** e.g. Clozapine (tablet form, 300mg to 450mg per day) or Risperidone (tablets/injections etc that last for up to 2 weeks, built up to 12mg) • The newer version of antipsychotics - less serious side effects but more potent • **Clozapine** = blinds to dopamine receptors in the same way as chlorpromazine but acts on seretonin and glutamate receptors. This helps to boost mood and lower depression which aims to help cognitive functioning. • **Resperidone** = most recent drug to be developed (1990's). This binds more strongly to dopamine and seretonin receptors and therefore doesn't need to be taken daily.	• Thornley (2003) reviewed studies comparing the effects of chlorpromazine to placebos and found that p's on chlorpromazine showed better overall functioning and less symptom severity. It also led to low relapse rates. • Side effects can range from mild to even fatal. Typical anti-psychotics include dizziness, stiff jaw and itchy skin. It can result in neuroleptic malignant syndrome (NMS) resulting in high temp and even a coma. Atypical anti-psychotics have less side effects. • Relient on the dopamine hypothesis being the cause of SZ. For some patient's this does work therefore their SZ may have a biological cause, but SZ can be the result of multiple factors and drug therapy alone cannot treat this.
Psychological therapies: **CBT** **Family therapy** **Token economy**	• CBT helps patients to identify irrational thoughts in a bid to change them. It helps patients to make sense of how their delusions and hallucinations impact them and how they can be challenged. • Family therapy takes place with families and improves the communication and interactions and aim to reduce the chance of a relapse. Pharoah et al (2010) found that it helps to create a therapeutic alliance, reduce the stress of caring for a SZ patient, reduction in anger and guilt from family members etc. • Token economies are reward systems used to manage the behaviour of SZ patients. When a SZ patient performs a desirable behaviour they are awarded with a token (secondary reinforcer) that can be exchanged for a reward (primary reinforcer). This is based on operant conditioning principles.	• Jauhar et al (2014) reviewed 34 studies of CBT and SZ and found that CBT had a significant but small effects on both positive and negative symptoms. • Pharoah et al (2010) family therapy can reduce hospital readmission over a year and boosts quality of life. • McMonagle and Sultana (2009) found that only 1 out of 3 studies showed improvement in symptoms for token economies. • Treatments improve quality of life but cannot fully cure SZ. Therefore other treatments must be considered in a holistic approach to treatment. • Ethical issues arise from token economy programmes because SZ patients have privelleges removed in order to be rewarded.
Interactionist approach to schizophrenia	• Interactionist approach = recognizes there are biological, psychological and societal factors in the development of SZ. • **Diathesis stress model** = both a vulnerability to SZ (diathesis) and a stress-trigger (stress) are needed to develop SZ. Genes and trauma are diathesis and stress can be biological or psychological in nature. • **Meehl's model** = if a person does not have the schizogene, then no amount of stress would lead to SZ. Carriers of the gene and chronic stress in childhood (schizophregenic mother) could result in SZ. • **Modern understanding of diathesis =** Many genes increase the likelihood of SZ, there is no schizogene. Diathesis can be biological and psychological e.g. psychological trauma as this can alter the developing brain and the HPA system can become over-active making p's more vulnerable to stress. • **Modern understanding of stress =** This involves any factor that can induce stress. E.g. Cannabis is a stressor because it can increase the likelihood of SZ up to 7 times. Cannabis can interfere with the dopamine system. • **Treatments =** both biological and psychological treatments are needed e.g. antipsychotics with CBT. These treatments can target all factors involved with the development of SZ and can ease symptoms	• Tienari et al (2004) found that a child-rearing style with high levels of criticism and conflict and low levels of empathy was implicated in the development of SZ but only for children with high genetic risk – more susceptible to parenting styles • The original diathesis-stress model is outdated and there are multiple genes involved rather than one 'schizogene'. This leads to increased awareness and developments. • Studies have shown an advantage with multiple treatments e.g. antipsychotics, CBT, token economies etc. This is only possible when an interactionist approach is taken. • Psychologists are not fully aware of the mechanisms that underlie 'diathesis' and 'stress' • Treatment-causation fallacy = whilst treatments help e.g. CNT, it does not mean that SZ is a direct result of cognitive factors.

Schizophrenia diagnosis

Biological explanations

Psychological explanations

Biological therapies: Drug therapy

Psychological therapies

Interactionist approach to schizophrenia

Links to other topics

Issues & Debates
Nature/Nurture, Determinism/freewill

Approaches
Biological, cognitive, psychodynamic

Attachment
Upbringing and

Key researchers/studies		Counter-arguments (GRAVE)
Gottesman (1991)	Found that identical twins have a 48% chance of developing schizophrenia if the other twin has it.	Good validity as the researchers used different family members as a comparison
Ripke et al (2014)	Found that genes associated with increased risk included those responsible for dopamine production	The relationship is a correlation and the researchers cannot conclude that these genes cause schizophrenia
Rakic et al (2004)	Found that low dopamine levels (hypodopaminergia) in the prefrontal cortex in negative symptoms	The relationship is a correlation and the researchers cannot conclude that this causes schizophrenia
Juckel et al (2006)	Found lower activity levels in the ventral stratium therefore activity in this area is a neural correlate	Good validity as the researchers used brain scans to measure brain and neural activity.
Allen et al (2007)	Found lower activation levels in the superior temporal gyrus and the anterior cingulate gurus in SZ p's.	Good validity as the researchers used brain scans to measure brain and neural activity.
Fromm-Reichmann (1972)	Patients with SZ often had a schizophregenic mother who is cold, rejecting and controlling.	The relationship is a correlation and the researchers cannot conclude that this causes schizophrenia
Frith (1992)	Identified 2 types of dysfunctional thought processing: Metarepresentation and central control	Researchers are unable to know whether cognitive factors cause or are a result of schizophrenia.
Pharoah et al (2010)	Suggests that family therapy strategies reduce stress and expressed emotion	Not a successful treatment if schizophrenia is caused by other factors e.g. genetics/neural correlates
Read et al (2001)	Early trauma to the brain e.g. through child abuse can affect brain development e.g. HPA system	Difficult to generalize the results as it's difficult to find people with schizophrenia with child abuse
Houston et al (2008)	It's possible to use CBT to relieve psychological symptoms even with a biological cause	Supported by further research

Key terms

Schizophrenia	A severe mental illness where contact with reality and insight is impaired
Positive symptoms	Atypical symptoms experienced in addition to normal experiences e.g. hallucinations and delusion
Negative symptoms	Atypical experiences that represent a loss of a usual experience e.g. clear thinking or 'normal' levels of motivation.
Co-mordbidity	The occurrence of two illnesses or conditions together e.g. schizophrenia can be co-morbid with personality disorders
Symptom overlap	Occurs when two or more conditions share symptoms. Where a condition shares many symptoms, this questions the validity of classifying the two disorders separately.
Dopamine	A neurotransmitter that is associated with pleasure and has an excitatory effect.
Neural correlates	Pattern of structure/activity in the brain that occur with experiences
Family dysfunction	Abnormal processes within a family e.g. poor communication or cold parenting
Cognitive explanations	Explanations that focus on mental processes e.g. thinking, language etc.
Dysfunctional thought processing	Information processing is not functioning 'normally' and produces undesirable consequences
Typical antipsychotics	The first generation of antipsychotic drugs e.g. Chlorpromazine
Atypical antipsychotics	The second generation of antipsychotic drugs e.g. Clozapine or Risperidone
Family therapy	A therapy carried out with the family
The interactionist approach	An approach that views biological and psychological factors in schizophrenia.