12.2 Psychological Explanations for Schizophrenia: Family and Cognitive Theory

AQA 7182 · about 50 minutes · Write-in edition

Total Marks

Name: _____ Date: ____ / ____ / _____

Answer ALL questions.

1 Identify and explain a methodological problem for family-based theories of schizophrenia.

(Total for Question 1 is 2 marks)

2 Outline a cognitive deficit and the symptom of schizophrenia it explains.

(Total for Question 2 is 3 marks)

3 Outline Bateson et al. double bind theory in the context of schizophrenia explanations.

(Total for Question 3 is 3 marks)

4 Outline expressed emotion as an explanation for relapse in schizophrenia.

(Total for Question 4 is 3 marks)

5 Outline impaired metarepresentation as a cognitive explanation for positive symptoms in schizophrenia.

(Total for Question 5 is 2 marks)

6 Outline central control dysfunction as a cognitive explanation for disorganised speech in schizophrenia.

(Total for Question 6 is 3 marks)

7 Outline lowered cognitive processing as a cognitive explanation for schizophrenia.

(Total for Question 7 is 3 marks)

8 Outline evidence linking expressed emotion to relapse in schizophrenia.

(Total for Question 8 is 3 marks)

9 Discuss psychological explanations for schizophrenia and compare them with biological accounts.

(Total for Question 9 is 16 marks)

Mark scheme · 12.2 Psychological Explanations for Schizophrenia: Family and Cognitive Theory

Question 1

identifies a methodological problem, e.g. reliance on retrospective reports, observer bias or confusing cause and effect (correlational data) oe — **M1**

explains limitation: retrospective or biased reporting may reflect current illness effects not causal family patterns, and correlational designs cannot establish causation oe — **A1**

Answer: A problem is reliance on retrospective reports and correlational data: parents or patients may recall family interactions differently after illness, and such designs cannot prove family patterns caused the disorder rather than result from it.

Question 2

identifies a specific cognitive deficit, e.g. impaired central control, impaired metarepresentation, or reduced processing speed oe — **M1**

states the specific symptom it explains, e.g. central control dysfunction explains disorganised speech; metarepresentation impairment explains auditory hallucinations or thought insertion oe — **M1**

briefly links how the deficit produces the symptom, e.g. impaired central control produces loose associations, leading to incoherent speech oe — **A1**

Answer: E.g. impaired central control is linked to disorganised speech because difficulties suppressing automatic associations lead to loose connections between ideas; impaired metarepresentation is linked to auditory hallucinations or thought insertion by causing misattribution of inner speech.

Question 3

identifies a double bind as repeated contradictory messages where a child cannot comment on the contradiction and cannot resolve the conflict oe — **M1**

states that Bateson et al. proposed such communication patterns may lead to confusing thinking and disordered communication oe — **M1**

links the double bind to the development of odd thought patterns or delusional beliefs as a proposed causal mechanism oe — **A1**

Answer: A double bind involves repeated contradictory messages a child cannot challenge or escape, and Bateson et al. suggested such family communication can produce confusing thinking and symptoms associated with schizophrenia.

Question 4

identifies typical EE features: critical comments, hostility and emotional overinvolvement from carers oe — **M1**

states EE is associated with a greater risk of relapse for someone with schizophrenia compared with low EE families oe — **M1**

explains that the stress of living in a high EE environment is proposed to trigger symptom relapse or worsen outcomes oe — **A1**

Answer: High expressed emotion, characterised by criticism, hostility and emotional overinvolvement from family carers, is associated with increased stress and a higher risk of relapse for people with schizophrenia.

Question 5

states metarepresentation is the ability to reflect on and represent one own thoughts and intentions oe — **M1**

explains that impairment could lead to misattributing inner speech to external sources, producing symptoms like thought insertion or auditory hallucinations oe — **A1**

Answer: Metarepresentation is the capacity to reflect on one own thoughts; if impaired, people may misattribute their own inner speech or thoughts to external sources, which can explain thought insertion and auditory hallucinations.

Question 6

identifies central control as the cognitive ability to suppress automatic responses and coordinate thoughts and behaviour oe — **M1**

states that dysfunction in central control leads to disrupted organisation of thought and speech, and to loose associations oe — **M1**

links this dysfunction to observable symptoms such as disorganised speech and difficulty maintaining a goal-directed sequence of thought oe — **A1**

Answer: Central control is the ability to suppress automatic responses and coordinate thought processes; if it is impaired, someone may show disorganised speech, loose associations and difficulty maintaining coherent, goal-directed thought.

Question 7

states lowered processing speed or efficiency means cognitive tasks take longer or are performed less accurately than typical oe — **M1**

links lowered processing to problems with divided attention, working memory or executive function in people with schizophrenia oe — **M1**

explains that slower or less efficient processing could contribute to disorganised thinking, poor problem solving or difficulties in goal-directed behaviour oe — **A1**

Answer: Lowered cognitive processing speed or efficiency means tasks are performed more slowly or less accurately; this is linked to problems with attention, working memory and executive function, and may contribute to disorganised thinking and poor goal-directed behaviour in schizophrenia.

Question 8

identifies an empirical finding, e.g. studies have found higher relapse rates among patients returning to high EE families compared with low EE families oe	**M1**
gives a basic statistic or comparative statement, e.g. relapse rates are substantially higher, typically reported as around 2 to 3 times greater in high EE families in some studies oe	**M1**
briefly explains why this supports the EE account, e.g. high EE may increase stress which triggers symptom exacerbation or relapse oe	**A1**

Answer: Research shows that patients returning to high EE families have higher relapse rates than those returning to low EE families; some studies report relapse rates around two to three times higher in high EE settings, supporting the idea that family stress contributes to relapse.

Question 9

Level 4 Knowledge of family and cognitive psychological explanations is accurate and detailed. Evaluation is thorough, balanced and explicit, including methodological critique, empirical support and comparison with biological explanations. The answer shows clear judgement about explanatory power and limitations, and uses relevant examples and research. Specialist terminology is used accurately and the response is coherent and well structured.	**13-16**
Level 3 Knowledge of the psychological explanations is clear with some detail. Evaluation is developed and shows awareness of strengths and limitations, and makes some comparison with biological accounts. The answer is mostly organised and uses appropriate terminology, though some points may be less well developed.	**9-12**
Level 2 Knowledge of psychological explanations is present but limited or partially accurate. Evaluation is superficial or one-sided, and comparison with biological explanations is limited. The answer may lack organisation or detail and use terminology inconsistently.	**5-8**
Level 1 Knowledge is minimal or inaccurate. Evaluation is very limited or absent, with little or no comparison to biological accounts. The answer lacks structure and uses few specialist terms.	**1-4**
Level 0 No creditable content.	**0**

Indicative content:

- AO1: Family dysfunction explanations include the schizophrenogenic mother (cold, rejecting, domineering parenting creates a climate that may contribute to schizophrenia), Bateson et al. double bind communication (repeated contradictory messages that the child cannot resolve lead to confusing thinking and disordered communication), and expressed emotion (high levels of criticism, hostility and emotional overinvolvement from carers are linked to higher relapse rates).
- AO1: Cognitive explanations include impaired metarepresentation (reduced ability to reflect on and recognise own thoughts, leading to misattribution of inner speech as external, explaining hallucinations and thought insertion), central control dysfunction (difficulty suppressing automatic responses and organising thought, explaining disorganised speech and thought disorder), and lowered processing efficiency (slower or less efficient attention and executive function contributing to disorganisation).
- AO3: Strengths of family accounts: draw attention to psychosocial and environmental contributors, help explain relapse and family interactions that affect course of illness, and have informed family intervention strategies. Weaknesses: risk of blaming families, many studies are correlational or retrospective so causality is uncertain, and findings have not shown consistent causal mechanisms across populations.
- AO3: Strengths of cognitive accounts: offer mechanistic, symptom-level explanations linking specific cognitive deficits to particular symptoms, are supported by cognitive testing showing deficits in attention and executive function, and can integrate with psychological therapies targeting thinking. Weaknesses: cognitive accounts may describe processes but not fully explain why deficits arise; some cognitive findings overlap with effects of medication or illness chronicity.
- AO3: Empirical support: EE studies show higher relapse risk in high EE families; cognitive research finds impairments in central control, working memory and source monitoring in patients with schizophrenia. Limitations include heterogeneity of findings and difficulties separating cause from effect.
- AO3: Comparison with biological explanations: biological accounts (genetic vulnerability, dopamine hypothesis, neural correlates) explain some aspects such as onset, heritability and some neurobiological correlates, whereas psychological accounts explain symptom phenomenology and environmental maintenance factors like relapse. Interactionist perspectives integrate both, but psychological explanations alone may struggle to explain the strong genetic and neurobiological findings.
- AO3: Overall evaluation: psychological explanations provide useful, clinically relevant accounts for specific symptoms and for environmental influences on relapse, but are limited by methodological problems and by not accounting for biological risk factors; the most persuasive position is that psychological and biological factors together provide a fuller explanation, with psychological models contributing important proximate mechanisms and treatment targets.