12.1 Biological Explanations for Schizophrenia: Genetics and Neural Correlates: 30-minute Pack

AQA 7182 · about 30 minutes

Total Marks

Name: _____ Date: ____ / ____ / _____

Answer all questions. For questions worth 4 marks or more write in full sentences. Total time guidance 30 minutes.

1 Outline the evidence from twin and adoption studies that is used to support genetic explanations for schizophrenia. Mention how each design helps separate genetic and environmental influences.

(Total for Question 1 is 3 marks)

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2 Discuss biological explanations for schizophrenia, including genetic explanations, the dopamine hypothesis and neural correlates. Refer to supporting evidence and to evidence or arguments that challenge biological accounts. Use the material above where relevant, and consider methodological and theoretical strengths and limitations.

(Total for Question 2 is 16 marks)

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3 Explain the concept of polygenic inheritance in schizophrenia and define what is meant by candidate genes in this context, giving an example of a gene often investigated.

(Total for Question 3 is 4 marks)

4 Case application, Dopamine hypothesis. Noah reports recent onset paranoid delusions and frequent auditory hallucinations. A clinician considers the dopamine explanation. Explain, using the revised dopamine hypothesis, how dopamine abnormalities could account for Noah's positive symptoms. Refer to brain regions and mechanisms in your answer.

(Total for Question 4 is 4 marks)

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Mark scheme · 12.1 Biological Explanations for Schizophrenia: Genetics and Neural Correlates: 30-minute Pack

Question 1

identifies twin study evidence, for example higher concordance in monozygotic MZ twins than dizygotic DZ twins oe	**M1**
identifies adoption study evidence, for example adopted-away children of parents with schizophrenia show elevated risk even when raised apart oe	**M1**
briefly explains why these findings support genetic influence, because MZ twins share 100% of genes while DZ twins share about 50%, and adoption separates genetic from family environment oe	**A1**

Answer: Twin studies find higher concordance for schizophrenia in monozygotic than dizygotic twins, suggesting genetic influence, and adoption studies show adopted-away children of parents with schizophrenia still have an elevated risk, which supports a genetic contribution because these designs help separate genes from shared environment.

Question 2

Level 4 Knowledge of biological explanations is accurate and detailed, showing clear understanding of genetics, dopamine hypotheses and neural correlates. Evaluation is thorough and balanced, using a wide range of relevant evidence and methodological critique, including discussion of gene-environment interaction and issues of causation. Argument is coherent and applied to the topic throughout, with appropriate use of specialist terminology.	**13-16**
Level 3 Knowledge is clear and generally accurate, covering main biological explanations. Evaluation is present and mostly effective, with some relevant evidence and methodological points. Discussion addresses strengths and limitations, but may not fully integrate evidence or consider alternative explanations in depth. Specialist terminology is used appropriately.	**9-12**
Level 2 Some relevant knowledge of biological explanations is present but may be partial or simplified. Evaluation is limited or descriptive rather than analytical. References to evidence or methods may be brief or one-sided. Answer may lack clear organisation or full development of points.	**5-8**
Level 1 Knowledge is minimal or inaccurate. Evaluation is very limited or missing. Answer may be largely a list of points with little coherence or development.	**1-4**
Level 0 No creditworthy content.	**0**

Indicative content:

- AO1: Genetic explanations, including family, twin and adoption study findings, for example higher concordance in MZ than DZ twins, and increased risk in first degree relatives; Gottesman is commonly cited for twin concordance figures.
- AO1: The concept of polygenic inheritance and candidate genes such as COMT and DRD2, and the idea that risk arises from many alleles each with small effect.
- AO1: The dopamine hypothesis, original and revised. Original claim that excess dopamine activity, particularly via D2 receptors, underlies schizophrenia, supported by observation that dopamine-blocking antipsychotics reduce positive symptoms. Revised account distinguishes hyperdopaminergia in mesolimbic/subcortical regions linked to positive symptoms and hypodopaminergia in prefrontal cortex linked to negative and cognitive symptoms.
- AO1: Neural correlates associated with positive symptoms, such as altered Broca area and superior temporal gyrus activity linked to auditory hallucinations, and correlates associated with negative symptoms, such as reduced ventral striatum or prefrontal activation linked to avolition and flattened affect.
- AO3: Strengths, including biological explanations being supported by converging evidence from multiple methods, for example genetic epidemiology, molecular genetics and neuroimaging, and explanatory power for some clinical observations including drug effects.
- AO3: Methodological limitations, for example twin and family studies may be confounded by shared environment and assortative mating, genome wide association studies show many loci each with tiny effect, and candidate gene findings have often failed to replicate robustly.
- AO3: Issues of causation and directionality for neural correlates, imaging findings are correlational and may reflect consequences of illness, chronic stress or medication effects rather than primary causes; longitudinal and high risk studies complicate simple causal claims.
- AO3: The role of gene-environment interaction and epigenetics, showing that genetic vulnerability may require environmental triggers, which weakens strong biological determinism and supports more interactionist models even while biological mechanisms remain important.
- AO3: Clinical and practical implications, for example biological research has led to drug targets and improved understanding of neural mechanisms, but treatment response heterogeneity and side effects show limits to a purely biological approach.
- AO3: Alternative or complementary explanations, for example psychological and social factors that interact with biology, and the need for integrative models that account for developmental timing, stress, cannabis use and social adversity.
- AO3: Evaluation of the evidence quality, including replication issues in genetic associations, sample sizes, population stratification, and differences between groups and individuals, which limit generalisability of some biological findings.

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Question 3

defines polygenic inheritance, for example schizophrenia is influenced by many genes each contributing a small effect oe	**M1**
explains implication, for example there is no single schizophrenia gene but many loci increase risk, accounting for variability in presentation oe	**M1**
defines candidate genes, for example specific genes investigated for association with schizophrenia such as COMT or DRD2 oe	**M1**
explains how candidate genes fit into the polygenic model, for example particular candidate genes may increase risk modestly as part of a larger set of risk alleles oe	**A1**

Answer: Polygenic inheritance means schizophrenia is influenced by many genes, each contributing a small increase in risk rather than one single gene causing the disorder. Candidate genes are particular genes studied for an association with schizophrenia, for example COMT or DRD2; these candidate genes are thought to contribute modestly to risk as part of a wider polygenic architecture.

Question 4

applies hyperdopaminergia to Noah's positive symptoms, stating excess dopamine in subcortical regions such as the mesolimbic pathway oe	**M1**
links excess subcortical dopamine to abnormal salience or misattribution processes, explaining how this could produce delusions or hallucinations oe	**M1**
refers to specific regions relevant to hallucinations, for example increased activity in auditory/language regions combined with dopamine dysregulation oe	**M1**
integrates these points to explain plausibly how Noah's paranoid delusions and hearing voices could result from regional dopamine excess producing aberrant signalling and altered perception of internal thoughts as external oe	**A1**

Answer: The revised dopamine hypothesis would explain Noah's delusions and hallucinations by hyperdopaminergia in subcortical regions, such as the mesolimbic pathway, causing abnormal assignment of salience to internal thoughts and perceptions. This excess dopamine can lead to aberrant signalling that helps form delusional beliefs, while altered activity in auditory and language regions may combine with dopamine dysregulation to make inner speech seem like external voices.

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