12.1 Biological Explanations for Schizophrenia: Genetics and Neural Correlates: Test Pack

AQA 7182 · about 15 minutes

Total Marks

Name: _____ Date: ____ / ____ / _____

Answer all questions. For questions worth 4 marks or more write in full sentences. Total time guidance 15 minutes.

1 Define what is meant by a neural correlate in the study of schizophrenia and give one concise example in your answer, naming the symptom and brain region.

(Total for Question 1 is 2 marks)

2 Outline one neural correlate that has been associated with positive symptoms of schizophrenia, for example auditory verbal hallucinations. Name the brain area and the typical finding.

(Total for Question 2 is 3 marks)

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3 Explain two methodological or interpretative limitations of using genetic and neural correlate evidence to claim a purely biological cause of schizophrenia. Give one point about genetics and one about neural correlates, and explain why each weakens a simple biological causal claim.

(Total for Question 3 is 4 marks)

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4 Explain the revised dopamine hypothesis, distinguishing hyperdopaminergia and hypodopaminergia and stating which brain regions are implicated for positive and negative symptoms respectively.

(Total for Question 4 is 4 marks)

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Mark scheme · 12.1 Biological Explanations for Schizophrenia: Genetics and Neural Correlates: Test Pack

Question 1

defines neural correlate, for example a brain structure or activity pattern reliably associated with a particular symptom or behaviour oe	**B1**
gives a concise example, for example reduced ventral striatum activity linked with avolition, or altered Broca's area activity linked with auditory hallucinations oe	**B1**

Answer: A neural correlate is a brain structure or activity pattern reliably associated with a particular symptom or behaviour; for example, reduced ventral striatum activity has been linked with avolition, and altered Broca's area activity has been linked with auditory hallucinations.

Question 2

identifies a relevant neural correlate, for example reduced activity or altered activation in Broca's area or superior temporal gyrus during auditory verbal hallucinations oe	**M1**
describes the typical finding, for example increased spontaneous activity or decreased task-related activation in language production areas linked to hearing voices oe	**M1**
briefly explains why this is relevant to positive symptoms, for example disruption in language regions may lead to misattribution of inner speech as external voices oe	**A1**

Answer: One neural correlate of positive symptoms, such as auditory verbal hallucinations, is altered activity in language-related areas like Broca's area or the superior temporal gyrus. Studies report increased spontaneous activation or atypical task-related activity in these regions, which could lead to misattribution of inner speech as external voices.

Question 3

identifies a limitation of genetic evidence, for example high heritability estimates do not prove determinism because shared environment or gene-environment interaction may be important oe	**M1**
explains why this weakens a simple biological cause claim, for example adoption and twin studies show association but cannot fully exclude environmental confounds or epigenetic mechanisms oe	**A1**
identifies a limitation of neural correlate evidence, for example correlation does not imply causation, brain differences may be a consequence of illness, medication or chronic stress rather than the cause oe	**M1**
explains why this weakens a simple biological cause claim, for example altered brain activity may reflect symptom expression or effects of antipsychotic treatment rather than being the primary cause of schizophrenia oe	**A1**

Answer: Genetic evidence, while showing increased risk, does not prove determinism because gene-environment interactions and shared environmental factors can contribute; adoption and twin studies reduce but do not eliminate these confounds. Neural correlate evidence shows associations between brain activity and symptoms, but correlation does not imply causation, and brain differences could be consequences of the disorder, of medication, or of prolonged stress, so they do not by themselves establish a purely biological cause.

Question 4

states the revision, for example the dopamine story was refined to say there may be too much dopamine in some regions and too little in others oe	**M1**
defines hyperdopaminergia in subcortical regions, such as the mesolimbic pathway, and links it to positive symptoms oe	**M1**
defines hypodopaminergia in the prefrontal cortex, such as the mesocortical pathway, and links it to negative and cognitive symptoms oe	**M1**
explains the functional implication, for example excess subcortical dopamine may cause sensory salience and hallucinations while low prefrontal dopamine may impair motivation and executive function oe	**A1**

Answer: The revised dopamine hypothesis recognises that dopamine abnormalities are region specific: hyperdopaminergia, too much dopamine activity, in subcortical regions such as the mesolimbic pathway is linked to positive symptoms, while hypodopaminergia, too little dopamine activity, in the prefrontal cortex, is linked to negative and cognitive symptoms. Excess subcortical dopamine may cause abnormal signalling of importance leading to hallucinations, while low prefrontal dopamine may impair motivation and planning.

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