6.2 Applying Nature-Nurture and Gene-Environment Interaction to Biopsychology: Test Pack

AQA 7182 · about 15 minutes

Total Marks

Name: _____ Date: ____ / ____ / _____

Answer ALL questions.

1 Define heritability and give an example applied to neurotransmitter functioning.

(Total for Question 1 is 2 marks)

2 Apply the diathesis-stress model to explain different outcomes in genetically identical twins following different life events.

(Total for Question 2 is 2 marks)

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3 Explain two biopsychological reasons why dizygotic twins raised together show only moderate concordance for cortisol response.

(Total for Question 3 is 4 marks)

4 Apply twin concordance pattern to explain gene-environment interaction in depression.

(Total for Question 4 is 4 marks)

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Mark scheme · 6.2 Applying Nature-Nurture and Gene-Environment Interaction to Biopsychology: Test Pack

Question 1

definition: the proportion of observed variation in a trait within a population that can be attributed to genetic differences between individuals oe — **B1**

example: e.g. a twin study might estimate that 50% of individual differences in dopamine receptor density are due to genetic variation oe — **B1**

Answer: Heritability is the proportion of variance in a trait within a population attributable to genetic differences. Example: a twin study estimating that around 50% of differences in dopamine receptor availability are genetic.

Question 2

states that diathesis-stress posits a genetic vulnerability (diathesis) plus an environmental trigger (stress) are needed for disorder onset oe — **M1**

applies to Maggie: although genetically vulnerable, she did not experience the environmental trigger (stressful divorce), so the disorder did not emerge oe — **A1**

Answer: Diathesis-stress holds that genes create a vulnerability but an environmental trigger is required. Maggie shares genetic vulnerability with her twin but lacks the stressful trigger, so she did not develop depression.

Question 3

reason 1: dizygotic twins share only about 50% of segregating genes, so genetic influence on cortisol regulation will not produce very high concordance oe — **M1**

explains why this matters: genetic variants affecting HPA axis functioning differ between DZ twins, limiting concordance oe — **A1**

reason 2: non-shared environmental factors and epigenetic differences can alter cortisol responses, even in twins raised together oe — **M1**

explains that different personal experiences, illnesses, or stress exposures can change HPA axis sensitivity via plasticity or epigenetic mechanisms, reducing concordance oe — **A1**

Answer: Moderate concordance arises because DZ twins share only about half their genes, so genetic effects on HPA axis regulation differ, and because non-shared environmental experiences and epigenetic changes can alter cortisol reactivity even when twins are raised together.

Question 4

identifies the pattern: gene variant concordance is higher in MZ twins than DZ twins but depression concordance is lower, implying genes are not the sole determinant oe — **M1**

explains that environmental factors must influence whether genetic vulnerability leads to disorder, consistent with gene-environment interaction oe — **A1**

applies diathesis-stress: the gene increases vulnerability but environmental triggers such as life stress determine which twins develop depression oe — **A1**

concludes this pattern supports interactionism rather than simple genetic determinism because shared genotype does not produce identical outcomes without differing environments oe — **A1**

Answer: Higher concordance for the gene but lower concordance for depression suggests genetic variants increase vulnerability but environmental triggers determine disorder onset. This pattern fits gene-environment interaction, for example the diathesis-stress model, and argues against strict genetic determinism.

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