6.2 Applying Nature-Nurture and Gene-Environment Interaction to Biopsychology: 30-minute Pack

AQA 7182 · about 30 minutes

Total Marks

Name: _____ Date: ____ / ____ / _____

Answer ALL questions.

1 Identify which gene-environment correlation is shown when genotype relates to selection of environments that change neurobiology.

(Total for Question 1 is 4 marks)

2 Evaluate two biopsychological explanations for why the same genetic marker yields different outcomes depending on childhood adversity.

(Total for Question 2 is 6 marks)

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3 Discuss the nature-nurture debate with specific reference to biopsychological research, GxE, epigenetics and empirical evidence.

(Total for Question 3 is 16 marks)

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Mark scheme · 6.2 Applying Nature-Nurture and Gene-Environment Interaction to Biopsychology: 30-minute Pack

Question 1

identifies the form: active gene-environment correlation (niche-picking) cao	**M1**
explains that individuals select environments or activities consistent with their genetically influenced traits, here choosing exercise routines oe	**A1**
explains the consequence: the selected environment then alters neurobiology (increased striatal dopamine responsivity), showing genes influence exposure to environmental effects oe	**A1**
link back to biopsychology: this shows a pathway where genotype shapes behaviour which shapes brain function, an interactionist process oe	**A1**

Answer: Active gene-environment correlation, or niche-picking. Genetically influenced traits lead individuals to select environments (high-intensity exercise) that then modify neurobiology (striatal dopamine responsivity), showing gene-driven environmental selection produces biological change.

Question 2

supporting point for diathesis-stress: evidence that adversity acts as trigger interacting with genetic vulnerability to produce dysfunction oe	**M1**
explains why this matters: it accounts for differential outcomes despite identical genotype, showing environment determines whether vulnerability is expressed and guiding interventions to reduce stress exposure oe	**A1**
supporting point for epigenetic account: evidence that childhood adversity produces persistent epigenetic changes that alter gene expression related to prefrontal function oe	**M1**
explains why this matters: it provides a biological mechanism by which early adversity can produce long-term changes in inhibitory control even if genotype is the same, suggesting treatments might need to target molecular or neuroplastic processes oe	**A1**
evaluative comparison: notes a limitation of each account, e.g. diathesis-stress is descriptive and may not explain persistence, while epigenetic evidence in humans is correlational and may not prove causation oe	**M1**
explains consequence: this means clinicians should treat findings as evidence for interaction but be cautious about assuming simple causal pathways, and consider both psychosocial and biological interventions oe	**A1**

Answer: Support for diathesis-stress: childhood adversity can trigger expression of genetic vulnerability, explaining why only some carriers show poor prefrontal control, which suggests reducing adversity could prevent problems. Support for epigenetics: adversity can produce persistent epigenetic changes that alter gene expression related to inhibitory control, offering a biological mechanism for long-term effects and implying interventions may need to address neural/plasticity processes. Limitation: diathesis-stress can be descriptive without mechanistic detail, while human epigenetic studies are often correlational, so both accounts are plausible but require cautious interpretation and combined intervention approaches.

Question 3

Level 4 Knowledge of relevant biopsychological concepts and evidence is accurate, detailed and well developed. The discussion is analytic and evaluative, considering gene-environment interaction, epigenetic mechanisms and empirical studies, and clearly argues the extent to which interactionism is supported over biological determinism. Arguments are well supported with examples and limitations, and the answer is coherent and uses specialist terminology appropriately.	**13-16**
Level 3 Knowledge and understanding of key biopsychological concepts and evidence is good, though some points may lack depth. The discussion evaluates interactionist and deterministic accounts with relevant examples such as twin studies, plasticity findings and epigenetic research, and shows a clear but not fully developed judgement on the balance of evidence. Organisation is good and terminology is generally appropriate.	**9-12**
Level 2 Knowledge of concepts and evidence is present but basic. Discussion offers some evaluation of nature-nurture in biopsychology, but analysis is limited or one-sided, and application of empirical evidence is superficial. The answer may be descriptive and lacks a sustained, balanced conclusion. Specialist terms are used inconsistently.	**5-8**
Level 1 Very limited knowledge and understanding. Discussion is largely descriptive or fragmented, with little or no evaluation and minimal use of relevant evidence. Conclusion, if present, is unsupported. Specialist terminology is absent or used incorrectly.	**1-4**
Level 0 No creditable content.	**0**

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Indicative content:

- AO1: Define the nature-nurture debate in the context of biopsychology: the extent to which biological factors (genes, neurochemistry) versus environmental experience shape behaviour and brain function.
- AO1: Gene-environment interaction types: active, evocative and passive gene-environment correlations; gene-by-environment (GxE) interactions where genotype moderates environmental effects; diathesis-stress as a common GxE model.
- AO1: Epigenetic mechanisms such as DNA methylation and histone modification alter gene expression without changing DNA sequence and provide a molecular pathway linking early environment to long-term changes in neural function.
- AO1: Empirical biopsychological evidence supporting interactionism: twin studies showing heritability of neurotransmitter measures but lower concordance for psychiatric outcomes; Maguire et al. style findings where intensive spatial experience changes hippocampal structure via plasticity; human and animal studies showing early-life stress alters HPA axis function via epigenetic changes.
- AO3: Evaluation points supporting interactionism: many biopsychological findings show neither genes nor environment alone fully determine outcomes; GxE findings and epigenetic data provide mechanisms; plasticity demonstrates environment-driven neural change within a genetic framework.
- AO3: Evaluation points qualifying the strength of the evidence: twin and GxE studies can be correlational and subject to confounds; epigenetic data in humans is often correlational and causation is difficult to prove; measures of heritability refer to populations and can be misunderstood as fixed for individuals.
- AO3: Consideration of biological determinism criticisms: historical tendency to overstate genetic causation has ethical and social implications; deterministic interpretations may underplay the role of social and therapeutic interventions which biopsychological evidence often supports as effective.
- AO3: Application to practice: interactionist evidence supports combined approaches, e.g. pharmacological treatments plus psychotherapy or environmental interventions, and suggests early intervention to prevent adverse epigenetic programming.
- AO3: Weighing evidence: conclude by assessing that biopsychological research predominantly supports an interactionist account, because mechanisms and data show reciprocal influence between genes and environment, but some deterministic claims remain in public discourse and some empirical limitations warrant cautious interpretation.

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