PSY.BIO10 Applying Nature-Nurture and Gene-Environment Interaction to Biopsychology

Total Marks

AQA 7182 · Calculators not allowed · about 75 minutes

Name: _____ Date: ____ / ____ / _____

Answer ALL questions. Show all your working.

1 Define heritability in the context of biopsychology, and give one example of what a heritability estimate might refer to in neurotransmitter functioning research.

Define heritability and give an example applied to neurotransmitter functioning.

(Total for Question 1 is 2 marks)

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2 Maggie is a monozygotic twin whose co-twin developed depression after a stressful divorce. Maggie has the same genetic vulnerability but has not experienced similar stressors. Explain, using the diathesis-stress model, why Maggie has not developed depression.

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 Kwame shows stronger synaptic plasticity in hippocampal circuits after intensive spatial training as a delivery driver. Explain how this observation supports an interactionist view rather than strict biological determinism.

Explain how training-induced hippocampal plasticity supports interactionism.

(Total for Question 3 is 4 marks)

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4 A twin study finds higher concordance for serotonin transporter gene variants in monozygotic twins than dizygotic twins, but concordance for clinical depression is lower than for the gene variant. Apply this pattern to explain gene-environment interaction in depression vulnerability.

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

(Total for Question 4 is 4 marks)

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5 Researchers measure DNA methylation levels in the promoter region of a stress-related gene in adults who experienced childhood neglect and in matched controls. The neglected group shows increased methylation and reduced gene expression. Explain how this result supports the role of epigenetics in biopsychology.

Explain how increased DNA methylation after childhood neglect supports epigenetic mechanisms.

(Total for Question 5 is 4 marks)

6 A study reports that people with a particular allele associated with reduced dopamine transporter efficiency are more likely to take up high-intensity exercise routines, which in turn increase striatal dopamine responsivity. Identify and explain which form of gene-environment correlation this illustrates.

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

(Total for Question 6 is 4 marks)

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7 Oliwia and her brother are dizygotic twins raised together. A study measures their cortisol responses to an acute stressor and finds moderate concordance. Explain two reasons, using biopsychological knowledge, why concordance for a physiological stress response might be moderate rather than very high.

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

(Total for Question 7 is 4 marks)

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8 A drug company claims a new medication 'corrects' a genetic abnormality linked to anxiety by increasing expression of a particular receptor protein. A critic argues this ignores environmental contributions. Apply the concept of epigenetics to give one critique the critic could make that is specific to biopsychology.

Apply epigenetics to critique a claim that a drug 'corrects' genetic abnormality by ignoring environment.

(Total for Question 8 is 3 marks)

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9 Apply the concept of evocative gene-environment interaction to this case: Yusuf has a genetic tendency to high reactivity to social stimuli, which makes him behave in a way that elicits harsher feedback from peers, increasing his amygdala responsivity over time. Explain how this is an evocative interaction and its biopsychological consequence.

Explain how Yusuf's genetically influenced behaviour evokes environmental responses that alter his brain.

(Total for Question 9 is 4 marks)

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10 A longitudinal study measures hippocampal volume in individuals from childhood to adulthood and finds that those who engaged in intensive navigational practice in adolescence show larger adult hippocampal volumes, but only if they carried a variant of a gene associated with efficient synaptic remodelling. Identify which interaction this illustrates and briefly explain why it supports an interactionist view.

Identify the gene-environment interaction when practice affects brain only in carriers of a facilitating gene.

(Total for Question 10 is 3 marks)

11 A clinic reports that among patients with the same genetic marker linked to impulsivity, those who experienced childhood adversity show altered prefrontal inhibitory control on tasks, whereas those without adversity do not. The clinic asks you to evaluate two biopsychological explanations for this pattern: (i) the diathesis-stress model, and (ii) persistent epigenetic modification. Provide two evaluative points, one supporting the diathesis-stress account and one supporting the epigenetic account, and explain how each point matters for interpreting the clinic's findings.

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

(Total for Question 11 is 6 marks)

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12 Discuss the nature-nurture debate in relation to biopsychological research. In your answer refer to gene-environment interaction, epigenetics and relevant empirical evidence from biopsychology, and evaluate the extent to which biopsychological evidence supports an interactionist account over strict biological determinism.

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

(Total for Question 12 is 16 marks)

Mark scheme · PSY.BIO10 Applying Nature-Nurture and Gene-Environment Interaction to Biopsychology

Question 1

- **B1** 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
- **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

- **M1** states that diathesis-stress posits a genetic vulnerability (diathesis) plus an environmental trigger (stress) are needed for disorder onset oe
- **A1** applies to Maggie: although genetically vulnerable, she did not experience the environmental trigger (stressful divorce), so the disorder did not emerge oe
- **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

- **M1** identifies that environmental experience (spatial training) produced neurobiological change (increased plasticity) oe
- **A1** explains this shows genes set potential but environment shapes brain structure/function via experience-dependent plasticity oe
- **A1** links to biopsychological mechanism: synaptic plasticity and changes in hippocampal circuits are biology mediating experience effects oe
- **A1** concludes this is interactionist because both biological substrate and environmental input were needed to produce the observed change oe
- **Answer: Intensive spatial training led to measurable hippocampal plasticity, showing environmental experience can modify brain structure. Genes provide mechanisms and capacity for plasticity but environmental input activates and shapes those mechanisms, so this supports interactionism rather than strict biological determinism.**

Question 4

- **M1** 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
- **A1** 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
- **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.**

Question 5

- **M1** identifies the finding: increased DNA methylation and reduced gene expression in those with childhood neglect oe
- **A1** explains that DNA methylation can alter gene expression without changing the nucleotide sequence, which is the definition of an epigenetic mechanism oe
- **A1** applies to biopsychology by linking reduced expression of a stress-related gene to altered neurobiological functioning and later stress responses oe
- **A1** concludes the result shows how early environment can produce long-term biological effects via epigenetic changes, supporting a nurture-influences-biology account oe
- **Answer: Increased methylation and reduced expression after childhood neglect shows that environmental experience can modify gene expression without changing DNA sequence. This epigenetic change can alter neurobiology related to stress, providing a biological pathway by which early environment has lasting effects.**

Question 6

- **M1** identifies the form: active gene-environment correlation (niche-picking) cao
- **A1** 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
- **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 7

- **M1** reason 1: dizygotic twins share only about 50% of segregating genes, so genetic influence on cortisol regulation will not produce very high concordance oe
- **A1** explains why this matters: genetic variants affecting HPA axis functioning differ between DZ twins, limiting concordance oe
- **M1** reason 2: non-shared environmental factors and epigenetic differences can alter cortisol responses, even in twins raised together oe
- **A1** explains that different personal experiences, illnesses, or stress exposures can change HPA axis sensitivity via plasticity or epigenetic mechanisms, reducing concordance oe
- **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 8

- **M1** identifies that epigenetic marks can alter gene expression independently of DNA sequence, so receptor expression may be influenced by environment oe
- **A1** applies to critique: the drug may change protein levels short term but persistent epigenetic modifications from stress or learning could continue to alter expression, limiting the drug's effectiveness oe
- **A1** adds that biopsychological interventions addressing environmental triggers or stressors may be needed alongside pharmacological treatment oe
- **Answer: Epigenetic modifications from environmental experiences can alter receptor expression independently of sequence. A drug that raises receptor levels may be limited if persistent epigenetic changes from stress continue to suppress expression, so environmental interventions may also be needed.**

Question 9

- **M1** identifies evocative gene-environment interaction: genetically influenced traits evoke particular responses from the environment oe
- **A1** applies to Yusuf: his genetic high social reactivity leads him to behave in ways that elicit harsher peer feedback oe
- **A1** explains biopsychological consequence: repeated harsher feedback acts as an environmental input that can increase amygdala responsivity via plasticity or stress-related neurochemical changes oe
- **A1** concludes this shows a two-way pathway where genes shape social niche which then shapes neural function, illustrating interactionism oe
- **Answer: This is evocative interaction: Yusuf's genetic reactivity produces behaviours that elicit harsher peer feedback, and that environmental feedback then increases amygdala responsivity through plasticity and stress-related changes. Genes thus shape the environment they experience, which in turn alters brain function.**

Question 10

- **M1** identifies gene-environment interaction, specifically a gene-by-environment (GxE) interaction where environment effect depends on genotype cao
- **A1** explains that the environmental input (navigational practice) produced structural brain change only in those with the facilitating gene, showing the effect of practice is moderated by genotype oe
- **A1** concludes this supports interactionism because neither gene nor practice alone fully determines the outcome, their combination does oe
- **Answer: This is a gene-by-environment interaction: the effect of navigational practice on hippocampal volume depends on genotype. It supports interactionism because both the facilitating gene and practice are required to produce larger hippocampal volume.**

Question 11

- **M1** supporting point for diathesis-stress: evidence that adversity acts as trigger interacting with genetic vulnerability to produce dysfunction oe
- **A1** 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
- **M1** supporting point for epigenetic account: evidence that childhood adversity produces persistent epigenetic changes that alter gene expression related to prefrontal function oe
- **A1** 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
- **M1** 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
- **A1** 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
- **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 12

- **Level 4** (13-16): 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.
- **Level 3** (9-12): 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.
- **Level 2** (5-8): 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.
- **Level 1** (1-4): 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.
- **Level 0** (0): No creditable content.
- **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.