8.6 The Nature-Nurture Debate and the Interactionist Approach

AQA 7182 · about 65 minutes · Write-in edition

Total Marks

Name: _____ Date: ____ / ____ / _____

Answer ALL questions.

1 Outline one reason why it is difficult to separate the effects of genes and environment when explaining behaviour. In your answer name a concept that links the two.

(Total for Question 1 is 2 marks)

2 Describe one ethical issue that arises when researching epigenetic effects of childhood adversity, and explain two ways a researcher could address this issue in their study design.

(Total for Question 2 is 6 marks)

3 Explain what is meant by the diathesis-stress model and give one brief example of how it links nature and nurture in explaining a psychological disorder.

(Total for Question 3 is 3 marks)

4 Explain briefly what epigenetics is and give one example of how an environmental factor might influence gene expression without changing DNA sequence.

(Total for Question 4 is 4 marks)

5 A school counsellor suggests that children who are naturally sociable will seek out friendship groups, clubs and other social situations and so gain more social practice, improving their social skills further. Which gene-environment interaction process does this describe, niche-picking or evocative interaction? Explain your answer.

(Total for Question 5 is 4 marks)

6 Explain two evaluation points that could be made about the diathesis-stress model as an interactionist account of mental disorder. Each point should be developed and state why it matters.

(Total for Question 6 is 6 marks)

7 Discuss the nature-nurture debate in psychology. In your answer, describe the interactionist approach, including the diathesis-stress model and epigenetics, and evaluate the usefulness of interactionist explanations compared with extreme nature or nurture accounts. Refer to theory, evidence and practical application where appropriate.

(Total for Question 7 is 16 marks)

8 A local mental health team proposes that some patients develop anxiety only after exposure to long-term work stress, despite having a family history of anxiety. Using the diathesis-stress model, outline how the team should explain why only some relatives became anxious.

(Total for Question 8 is 4 marks)

9 A researcher claims that exposure to urban pollution during childhood causes changes to gene expression that increase later risk of respiratory and some mental health problems. Identify two forms of evidence or research designs that could support an interactionist claim like this, and explain briefly why each would be appropriate.

(Total for Question 9 is 5 marks)

Mark scheme · 8.6 The Nature-Nurture Debate and the Interactionist Approach

Question 1

identifies a difficulty, e.g. gene-environment interaction where genes influence sensitivity to environments, or environmental effects on gene expression such as epigenetics oe	**B1**
briefly explains why that makes separation hard, e.g. genetic predispositions may only appear when particular environmental triggers are present oe	**B1**

Answer: Gene-environment interaction makes separation hard because genes and environment influence each other: for example, a genetic predisposition may only lead to a behaviour when an environmental trigger occurs.

Question 2

identifies an ethical issue, e.g. potential distress from recalling childhood trauma or breach of privacy due to sensitive biological data oe	**M1**
explains how it arises, e.g. asking participants about adversity may cause distress and collecting biological samples raises confidentiality concerns oe	**A1**
suggests a way to address it in design, e.g. use careful informed consent, offer support/referral, allow withdrawal, de-identify samples and store data securely oe	**M1**
explains why this is effective, e.g. de-identification protects privacy and debriefing reduces harm, increasing ethical acceptability and data quality oe	**A1**
additional point: consider special protections for vulnerable participants or use of archival/anonymous data where appropriate oe	**M1**
explains consequence, e.g. reduces need for intrusive questioning and limits risk while still allowing investigation of epigenetic markers oe	**A1**

Answer: An ethical issue is causing distress by asking about childhood adversity and handling sensitive biological data. Researchers should obtain fully informed consent, offer support and referral, allow withdrawal, de-identify and securely store samples, and consider using archival or anonymised data where possible to protect vulnerable participants and privacy.

Question 3

defines diathesis-stress model: a genetic or biological vulnerability (diathesis) interacts with an environmental stressor to produce a disorder or behaviour oe	**M1**
gives an example of interaction, e.g. a genetic predisposition to mood disorder may lead to depression only after a major life stress such as bereavement oe	**A1**
identifies this as linking nature (vulnerability) and nurture (trigger) oe	**A1**

Answer: The diathesis-stress model says a biological vulnerability plus an environmental stressor together produce a disorder; for example, someone with a genetic vulnerability to depression may develop the condition after severe life stress, showing both nature and nurture are needed.

Question 4

defines epigenetics: changes in gene expression caused by chemical modifications (e.g. methylation) that do not alter the DNA sequence oe	**M1**
gives an example environmental factor, e.g. nutrition, stress, or toxins affecting methylation patterns oe	**A1**
explains effect on gene expression, e.g. increased methylation can switch genes off, altering protein production and behaviour risk without changing DNA sequence oe	**A1**
links to nature-nurture, e.g. demonstrates how environment can influence biological processes and thus behaviour oe	**A1**

Answer: Epigenetics refers to chemical changes that alter gene expression without changing the DNA, such as methylation. For example, severe early-life stress might increase methylation of genes involved in stress regulation, reducing their expression and changing vulnerability to mental health problems, showing environment modifying biological functioning.

Question 5

identifies the process as niche-picking cao	**M1**
explains that the individual actively selects or creates environments matching their genetic tendency, e.g. seeking out social situations oe	**A1**
explains outcome: selection leads to further development of the trait through increased practice/opportunities oe	**A1**
contrasts with evocative interaction where others respond to a trait rather than the person actively selecting the environment oe	**A1**

Answer: Niche-picking: sociable children actively seek out social environments, which provides more practice and reinforces social skills; this differs from evocative interaction where others merely respond to a trait.

Question 6

evaluation point 1: model is useful clinically, leading to matched interventions targeting stressors and vulnerability oe	**M1**
explains why it matters: it supports prevention and personalised treatment, e.g. reducing stressors can prevent disorder in those at risk oe	**A1**
evaluation point 2: the model can be vague about specifying the exact biological diathesis and threshold, making precise prediction difficult oe	**M1**
explains why it matters: limited predictive power reduces the model's utility for identifying who will develop a disorder and raises issues for testing the model empirically oe	**A1**
further development: mention empirical support or limitation, e.g. some studies show gene by environment interactions but measurement issues and replication problems remain oe	**M1**
explains consequence: supports cautious use of the model and need for more precise biomarkers and measures of stress to improve application and testing oe	**A1**

Answer: The diathesis-stress model is clinically useful because it suggests interventions that target environmental stressors as well as biological vulnerability, supporting prevention and personalised treatment. However, it is often vague about the specific diathesis and the threshold for disorder, limiting prediction and empirical testing; this matters because without precise markers we cannot reliably identify who will develop a disorder and replication of interaction findings is often challenging.

Question 7

Level 4 Knowledge of the nature-nurture debate and interactionist approaches is accurate and detailed, covering relevant concepts such as diathesis-stress and epigenetics. Evaluation is thorough, well developed and balanced, integrating empirical evidence, methodological critique and practical implications. The answer is coherent, well structured and uses specialist terminology correctly.	**13-16**
Level 3 Knowledge is clear and generally accurate, covering key interactionist ideas and some evidence. Evaluation is evident and competent, with some balance between strengths and limitations, and some application to practice. The answer is mostly well organised with appropriate use of terminology.	**9-12**
Level 2 Knowledge is present but limited in depth or range. Evaluation is superficial or one-sided, with limited integration of evidence or application. The answer may lack organisation or precise use of specialist terms.	**5-8**
Level 1 Limited knowledge and minimal evaluation. Responses may be list-like, unclear or largely irrelevant. Little or no use of evidence or application. Specialist terminology is largely absent or misused.	**1-4**
Level 0 No creditable content.	**0**

Indicative content:

- AO1: Define nature-nurture debate: the extent to which behaviour is determined by genetic inheritance versus environmental influences.
- AO1: Describe extreme positions: nativist/biological determinism emphasises genes, empiricist/environmentalist emphasises learned experience.
- AO1: Describe interactionist approach: genes and environment interact in complex ways; cannot be separated cleanly.
- AO1: Explain diathesis-stress model: genetic vulnerability plus environmental stressor produce disorder; includes threshold idea and protective factors.
- AO1: Explain gene-environment interaction processes such as niche-picking and evocative interaction.
- AO1: Explain epigenetics: environment-induced chemical modifications of gene expression, e.g. methylation, that can alter risk without changing DNA sequence.
- AO3: Evaluate theoretical usefulness: interactionist accounts avoid biological determinism and permit multiple causal pathways; better fit to complex human behaviour.
- AO3: Evaluate empirical support and limitations: cite that some studies find gene by environment interactions but replication is variable and measurement of stress and diathesis is challenging; longitudinal and mechanistic studies strengthen claims but causal inference is sometimes limited.
- AO3: Methodological critiques: difficulties measuring genes, epigenetic markers, and environmental exposures accurately; confounds and reverse causality possible; ethical limits on experimental manipulation.
- AO3: Practical applications: interactionist models inform prevention and treatment by targeting environment and resilience, and suggest personalised interventions; epigenetics raises possibilities for biomarkers but also ethical issues around stigma and determinism.
- AO3: Compare with extreme accounts: pure nature struggles to explain environmental effects and individual differences in identical genetics; pure nurture cannot account for inherited tendencies and biological constraints. Interactionist approach offers a more comprehensive explanatory framework.
- AO3: Conclude with balanced judgement: interactionist approach currently offers the most useful scientific and clinical framework, but requires rigorous research, careful communication to avoid deterministic misinterpretation, and attention to ethical implications.

Question 8

applies diathesis-stress: family members may share a genetic vulnerability (diathesis) to anxiety oe	**M1**
explains environmental trigger: long-term work stress acts as the stressor that can activate the diathesis in some individuals oe	**A1**
explains individual differences: relatives differ in the level of genetic vulnerability and exposure to stress, so only some reach the threshold for developing anxiety oe	**A1**
may mention protective factors reducing risk in some relatives, e.g. social support or coping skills, preventing disorder despite similar family history oe	**A1**

Answer: Family members may share genetic vulnerability, but only those who experienced sufficient long-term work stress had that vulnerability activated; differences in genetic susceptibility, stress exposure, or protective factors like social support explain why only some relatives developed anxiety.

Question 9

identifies a suitable research design or evidence type, e.g. longitudinal study tracking pollution exposure and later gene expression/health outcomes oe	**M1**
explains why appropriate, e.g. longitudinal design shows temporal order and links early exposure to later outcomes, supporting causal inference oe	**A1**
identifies a second suitable design, e.g. experimental or quasi-experimental animal studies or natural experiments measuring epigenetic markers after controlled exposure oe	**M1**
explains why appropriate, e.g. controlled exposure allows examination of biological mechanisms such as methylation changes that could mediate effects oe	**A1**
alternative valid evidence allowed, e.g. twin or sibling-comparison studies controlling genetic background while differing in exposure, with explanation of control of confounds oe	**A1**

Answer: Examples: a longitudinal cohort study measuring childhood pollution exposure and later epigenetic markers and health outcomes, because it links early exposure with later changes over time; and controlled animal or natural-experiment studies that assess epigenetic changes after exposure, because they allow investigation of biological mechanisms. Twin or sibling comparisons that differ in exposure could also help control genetic confounds.