PSY.OPT17 Biological Explanations of Aggression: Neural and Hormonal Mechanisms

Total Marks

AQA 7182 · Calculators not allowed · about 75 minutes

Name: _____ Date: ____ / ____ / _____

Answer ALL questions. Show all your working.

1 Outline the role of the amygdala in aggressive responses, referring to neural mechanisms of aggression and the limbic system in the brain.

Outline the role of the amygdala in aggressive responses, referring to neural mechanisms of aggression and the limbic system in the brain.

(Total for Question 1 is 2 marks)

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2 Explain how the orbitofrontal cortex (OFC) interacts with the limbic system to regulate aggressive impulses, including the effect of OFC damage.

Explain how the orbitofrontal cortex (OFC) interacts with the limbic system to regulate aggressive impulses, including the effect of OFC damage.

(Total for Question 2 is 3 marks)

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3 Outline the role of serotonin in inhibiting aggression via cortical control, and explain how low serotonin is linked to impulsive aggressive behaviour.

Outline the role of serotonin in inhibiting aggression via cortical control, and explain how low serotonin is linked to impulsive aggressive behaviour.

(Total for Question 3 is 3 marks)

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4 Outline one piece of correlational evidence linking testosterone levels to aggressive behaviour in humans, naming a researcher or year where appropriate, and note a limitation of such correlational evidence.

Outline one piece of correlational evidence linking testosterone levels to aggressive behaviour in humans, naming a researcher or year where appropriate, and note a limitation of such correlational evidence.

(Total for Question 4 is 4 marks)

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5 Outline the role of cortisol in moderating the relationship between testosterone and aggressive behaviour, referring to how cortisol levels may influence testosterone effects.

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(Total for Question 5 is 2 marks)

6 A 28-year-old male, Kwame, has a history of sudden impulsive aggressive outbursts that appear disproportionate to the triggers. A hospital records his low cerebrospinal fluid serotonin metabolite levels and finds reduced orbitofrontal cortex activity on neuroimaging. Using these details, explain how low serotonin and reduced OFC activity could account for Kwame's impulsive aggression. Refer specifically to the mechanisms involved.

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(Total for Question 6 is 6 marks)

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7 Outline one quasi-experimental piece of evidence that links testosterone to aggressive behaviour in humans and explain one limitation of interpreting such quasi-experimental findings.

Outline one quasi-experimental piece of evidence that links testosterone to aggressive behaviour in humans and explain one limitation of interpreting such quasi-experimental findings.

(Total for Question 7 is 4 marks)

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8 Outline one reason why animal studies of testosterone and aggression may not generalise fully to humans, referring to biological or ethical differences.

Outline one reason why animal studies of testosterone and aggression may not generalise fully to humans, referring to biological or ethical differences.

(Total for Question 8 is 2 marks)

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9 Outline how serotonin and testosterone might interact to influence aggressive behaviour, giving a brief mechanism for their joint effects.

Outline how serotonin and testosterone might interact to influence aggressive behaviour, giving a brief mechanism for their joint effects.

(Total for Question 9 is 2 marks)

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10 Identify one ethical issue raised by human hormonal manipulation studies of aggression (for example trials administering testosterone), and suggest how researchers can address this issue.

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(Total for Question 10 is 2 marks)

11 Discuss neural and hormonal explanations of aggression. In your answer, evaluate the strengths and limitations of these biological accounts, including methodological issues such as correlational designs, problems of extrapolating from animal studies, and the risk of biological reductionism. Use real study references where appropriate and show balanced AO1, AO2 and AO3 material.

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(Total for Question 11 is 16 marks)

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12 Explain one methodological difference between correlational hormone studies and experimental manipulations of neural function, and state one implication of that difference for causal inference about aggression.

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(Total for Question 12 is 4 marks)

Mark scheme · PSY.OPT17 Biological Explanations of Aggression: Neural and Hormonal Mechanisms

Question 1

- **B1** states that the amygdala is part of the limbic system and is involved in processing emotions such as fear and anger oe
- **B1** states that increased amygdala activity is associated with heightened threat detection and can trigger aggressive responses oe
- **Answer: The amygdala is a limbic system structure involved in processing emotions such as fear and anger; increased amygdala activity is linked to stronger threat responses and can trigger aggression.**

Question 2

- **M1** states that the OFC is involved in impulse control and social decision making oe
- **A1** explains that the OFC exerts top down inhibitory control over limbic structures such as the amygdala, helping to suppress aggressive impulses oe
- **A1** notes that damage or reduced activity in the OFC is linked to poorer inhibition and increased impulsive aggression oe
- **Answer: The OFC supports impulse control and social decision making and exerts top down inhibition over limbic areas like the amygdala to suppress aggressive impulses; damage or reduced OFC activity impairs this inhibition and is linked to increased impulsive aggression.**

Question 3

- **M1** states that serotonin is a neurotransmitter involved in mood regulation and behavioural inhibition oe
- **A1** explains that adequate serotonin supports functioning of prefrontal regions such as the OFC, which inhibit aggressive impulses from the limbic system oe
- **A1** states that low serotonin levels are associated with reduced impulse control and increased impulsive or reactive aggression oe
- **Answer: Serotonin is a neurotransmitter linked to mood and behavioural inhibition; sufficient serotonin supports prefrontal control, including the OFC, which inhibits limbic aggressive impulses, whereas low serotonin is associated with poorer impulse control and increased impulsive aggression.**

Question 4

- **M1** identifies a correlational finding, for example that higher baseline testosterone is associated with higher levels of aggression or dominance behaviours in samples of men oe
- **A1** provides a named example or plausible reference, e.g. research finding positive correlations between salivary testosterone and aggression in young adult males oe
- **A1** explains a methodological limitation: correlation does not establish causation, the relationship may be bidirectional or caused by a third variable oe
- **A1** notes that such studies typically rely on hormone sampling and behavioural measures or self reports, which may have reliability issues oe
- **Answer: Correlational studies report that higher baseline testosterone is linked to greater aggression or dominance behaviour in samples of men; for example studies using salivary testosterone measures have found positive correlations with aggressive acts, but correlation cannot prove causation and the association may reflect bidirectional effects or a third variable, with measures also subject to reliability limits.**

Question 5

- **B1** states that cortisol is a stress hormone that can interact with testosterone to influence aggression oe
- **B1** states that high cortisol can suppress the behavioural effects of testosterone, so testosterone may predict aggression more strongly when cortisol is low oe
- **Answer: Cortisol, a stress hormone, moderates testosterone's effects: high cortisol can inhibit testosterone-related aggression, so testosterone is more likely to predict aggressive behaviour when cortisol levels are low.**

Question 6

- **M1** identifies that low serotonin is linked to reduced behavioural inhibition and poorer regulation of impulses oe
- **A1** explains that serotonin supports prefrontal regions like the OFC, so low serotonin can impair OFC functioning and weaken top down control of limbic aggression responses oe
- **M1** identifies that reduced OFC activity means weaker inhibition of the amygdala and other limbic triggers of aggression oe
- **A1** explains this leads to more rapid, less controlled responses to perceived provocation, accounting for impulsive outbursts in Kwame oe
- **A1** applies the case details explicitly, linking low serotonin result and imaging findings to the behavioural pattern of sudden disproportionate aggression in Kwame oe
- **A1** may mention that this pattern fits the model of serotonergic modulation of cortical inhibition over limbic aggression, not necessarily implying a purely environmental cause oe
- **Answer: Low serotonin reduces behavioural inhibition and impairs prefrontal regions such as the OFC; combined with reduced OFC activity, top down inhibition of limbic structures like the amygdala is weakened, producing less control over threat-driven responses and leading to rapid, disproportionate aggressive outbursts as seen in Kwame. This links the biochemical and neural imaging findings to his impulsive aggression via impaired serotonergic support for cortical inhibition.**

Question 7

- **M1** identifies a quasi-experimental example, for instance studies of changes in testosterone following competitions that show winners' testosterone increases are linked to increases in dominance or aggression-related behaviours oe
- **A1** explains the quasi-experimental nature: testosterone changes are measured in naturalistic situations where participants are not randomly assigned to hormone levels oe
- **M1** identifies a limitation such as lack of random assignment, so causal conclusions are limited and confounding variables (e.g. prior aggression, social status) may explain results oe
- **A1** explains the limitation in context, noting this weakens claims that testosterone alone causes observed increases in aggressive or dominant behaviour oe
- **Answer: Quasi-experimental studies, such as those measuring testosterone changes after competitive wins and linking increased testosterone to greater dominance or aggression-related actions, suggest an association in natural contexts. However, because participants are not randomly assigned to hormone levels and contexts vary, confounding variables may explain the effect, limiting causal interpretation of testosterone as the sole cause of increased aggression.**

Question 8

- **B1** states a limitation, e.g. differences in social complexity between humans and animals, or different hormonal regulation across species oe
- **B1** explains why this matters, for example human aggression is influenced by complex cognition and culture which animal models do not capture, reducing external validity oe
- **Answer: Animal study findings may not generalise because humans have more complex social cognition and cultural influences on aggression and because hormonal systems can differ across species; this reduces the external validity of animal evidence for human aggression.**

Question 9

- **B1** states that serotonin primarily supports cortical inhibition of aggression while testosterone is linked to increased dominance and aggression tendencies oe
- **B1** explains that testosterone effects on aggression may be more likely when serotonin-mediated inhibition is low, so low serotonin can allow testosterone-related aggression to be expressed oe
- **Answer: Serotonin supports cortical inhibition of aggressive impulses while testosterone promotes dominance and aggression; when serotonin-mediated inhibition is low, testosterone's effects are more likely to be expressed, producing higher likelihood of aggressive behaviour.**

Question 10

- **B1** identifies an ethical issue, e.g. risk of causing psychological or behavioural harm by increasing aggression through hormone administration oe
- **B1** suggests a way to address it, e.g. rigorous screening, careful informed consent, close monitoring, and stopping rules with support available for participants oe
- **Answer: An ethical issue is the risk of inducing harmful increases in aggression through hormone administration; researchers can address this by careful screening, full informed consent, close monitoring, predefined stopping criteria and provision of support to participants.**

Question 11

- **Level 4** (13-16): Knowledge of neural and hormonal explanations is accurate and detailed. Discussion and evaluation are thorough and balanced, considering methodological strengths and limitations, issues of causation, generalisability from animal studies, and reductionism. Real studies or examples are used appropriately and the answer considers implications. The answer is coherent, well-structured and uses appropriate specialist terminology.
- **Level 3** (9-12): Knowledge is clear with some detail. Evaluation is effective but not exhaustive, addressing key issues such as correlational evidence and animal studies. Some use of studies or examples to support points. The answer is generally well organised and applies material to evaluation.
- **Level 2** (5-8): Knowledge is present but limited in depth. Evaluation is superficial or partial, for example listing strengths and limitations without full development or application. Limited reference to studies. The answer may lack clear organisation.
- **Level 1** (1-4): Knowledge is minimal and answers are descriptive or disjointed. Evaluation is barely present. Little or no use of studies and poor organisation.
- **Level 0** (0): No creditable content.
- **Indicative content:**
  - AO1: Neural explanations include the role of the limbic system, particularly the amygdala, in detecting threat and generating aggressive responses, and the role of prefrontal regions, notably the orbitofrontal cortex, in inhibiting impulsive aggression via top down control.
  - AO1: Hormonal explanations emphasise testosterone's association with dominance and aggressive behaviours and cortisol's moderating influence, and serotonergic systems that support behavioural inhibition via prefrontal circuitry.
  - AO2: Real evidence examples: amygdala hyperactivity observed in some violent offenders; neuroimaging linking reduced OFC activity with impulsive aggression; correlational studies linking salivary testosterone to aggression; quasi-experiments showing testosterone changes after competition associated with dominance behaviours. Animal studies show causal hormonal effects on aggression, e.g. testosterone manipulations in rodents.
  - AO3 methodological critique: many human hormone studies are correlational so causation cannot be assumed, bidirectional explanations and third variables (e.g. social status) are possible, and quasi-experimental naturalistic designs lack random assignment. Hormone measurement timing and reliability can be problematic.
  - AO3 generalisability issues: strong animal evidence exists but extrapolating to humans is limited by differences in social complexity and cognition; ethical constraints restrict experimental hormone manipulation in humans.
  - AO3 theoretical critique: biological reductionism may oversimplify aggression by downplaying social, cognitive and situational influences; an integrative biopsychosocial approach may be more explanatory.
  - AO3 strengths and applications: biological explanations can identify mechanisms that suggest interventions, for example serotonergic drugs or behavioural strategies to strengthen prefrontal control, and can inform risk assessment when combined with psychosocial data.
  - AO3 balance: note individual differences, gene-environment interactions and interaction between hormones (testosterone and cortisol) and neurotransmitters (serotonin) which complicate simple biological accounts.
  - Conclusion: A considered judgement that neural and hormonal explanations provide important mechanistic insights and useful applications, but their limitations in causal inference, generalisability and potential reductionism mean they are best integrated with psychological and social explanations.

Question 12

- **M1** identifies a methodological difference, e.g. correlational hormone studies measure natural variation in hormone levels without manipulation, whereas experimental neural manipulations (e.g. transcranial magnetic stimulation or lesion studies in animals) involve active manipulation of brain activity oe
- **A1** explains why this difference matters: experimental manipulation allows stronger causal inference because researchers control the independent variable and use randomisation or counterbalancing, while correlational studies cannot rule out reverse causation or confounds oe
- **M1** states an implication for causal inference, e.g. correlational hormone studies cannot establish that hormones cause aggression, only that they are associated, while well-controlled experiments provide stronger evidence of causation oe
- **A1** applies this implication to aggression research, noting ethical limits on experimental manipulation in humans may constrain how far causation can be established directly in people oe
- **Answer: Correlational hormone studies measure natural hormone variation without manipulation, whereas experimental neural manipulations actively alter brain activity. This matters because experiments allow stronger causal inference by controlling the independent variable, while correlational studies cannot rule out reverse causation or confounding, so they only show association; ethical limits on human experiments further constrain causal claims about aggression in people.**