15.1 Biological Explanations of Aggression: Neural and Hormonal Mechanisms: 30-minute Pack

AQA 7182 · about 30 minutes

Total Marks

Name: _____ Date: ____ / ____ / _____

Answer ALL questions.

1 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 1 is 4 marks)

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2 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.

(Total for Question 2 is 16 marks)

3 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.

(Total for Question 3 is 6 marks)

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Mark scheme · 15.1 Biological Explanations of Aggression: Neural and Hormonal Mechanisms: 30-minute Pack

Question 1

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	**M1**
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	**A1**

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 2

Level 4 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.	**13-16**
Level 3 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.	**9-12**
Level 2 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.	**5-8**
Level 1 Knowledge is minimal and answers are descriptive or disjointed. Evaluation is barely present. Little or no use of studies and poor organisation.	**1-4**
Level 0 No creditable content.	**0**

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.

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Question 3

identifies that low serotonin is linked to reduced behavioural inhibition and poorer regulation of impulses oe **M1**

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 **A1**

identifies that reduced OFC activity means weaker inhibition of the amygdala and other limbic triggers of aggression oe **M1**

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 **A1**

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.

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