13.2 Biological Explanations for Anorexia Nervosa: 30-minute Pack

AQA 7182 · about 30 minutes

Total Marks

Name: _____ Date: ____ / ____ / _____

Answer ALL questions.

1 Outline Guisinger's adapted-to-flee-famine hypothesis as an evolutionary explanation for anorexia nervosa. Explain the proposed adaptive mechanism and how it might become maladaptive in modern contexts.

(Total for Question 1 is 3 marks)

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2 Discuss biological explanations for anorexia nervosa. In your answer include neural explanations (hypothalamic regulation, appetite hormones, neurotransmitters) and evolutionary explanations (the adapted-to-flee-famine hypothesis and reproductive suppression hypothesis). Evaluate the explanations using supporting evidence and limitations such as reductionism, testability and real-world application. You should reach a reasoned judgement.

(Total for Question 2 is 16 marks)

3 Case: Erin, a 20-year-old undergraduate, has lost a large amount of weight over six months, is amenorrhoeic and reports high levels of anxiety. Blood tests show low leptin levels. Using biological explanations, explain how low leptin and hypothalamic signalling could account for both Erin's low weight and amenorrhoea. Refer specifically to physiological mechanisms.

(Total for Question 3 is 6 marks)

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Mark scheme · 13.2 Biological Explanations for Anorexia Nervosa: 30-minute Pack

Question 1

identifies the hypothesis: anorexia is an evolved response that may have promoted migration away from famine or depleted resources oe	**M1**
explains the proposed mechanism, for example reduced appetite and increased activity would encourage individuals to leave a famine area and forage or migrate rather than remain where food is scarce oe	**M1**
notes that in modern environments this foraging/migration response may be triggered inappropriately, producing pathological restriction despite food being available oe	**A1**

Answer: Guisinger's adapted-to-flee-famine hypothesis proposes that behaviours seen in anorexia, such as reduced appetite and increased activity, could be extreme activations of an evolved foraging or migration response that helped ancestors leave famine zones. In modern environments this response may be misfired, producing harmful restriction even when food is available.

Question 2

Level 4 Knowledge and understanding of biological explanations is accurate and well developed. Discussion and evaluation are thorough, showing a good range of relevant supporting and challenging evidence, and arguments are evaluated in depth. The answer integrates neural and evolutionary accounts and considers practical implications. There is a clear, well-supported judgement.	**13-16**
Level 3 Knowledge and understanding is generally accurate with reasonable detail. Evaluation is clear and includes both strengths and limitations, though some points may be less developed. There is some integration of evidence and a conclusion is reached, but the judgement may be less fully supported.	**9-12**
Level 2 Knowledge is present but limited or partially accurate. Evaluation is superficial or one-sided, missing key counterarguments or evidence. Little synthesis of neural and evolutionary explanations. Conclusion is brief or not well supported.	**5-8**
Level 1 Knowledge is very limited, largely descriptive or inaccurate. Little or no evaluation. No coherent conclusion. Answer may be disorganised.	**1-4**
Level 0 No creditable content.	**0**

Indicative content:

- AO1: Neural explanations include hypothalamic regulation via lateral and ventromedial nuclei, integration of hormonal and neural signals, and disruption of appetite control.
- AO1: Appetite-regulating hormones such as leptin (satiety) and ghrelin (hunger) signal energy status to the hypothalamus; abnormalities in these hormones are reported in anorexia nervosa and can disrupt hunger and reproductive signalling.
- AO1: Neurotransmitters, particularly serotonin, are implicated in appetite, mood and impulse control; altered serotonin function is found in people with anorexia and may increase anxiety and rigid eating behaviours.
- AO1: Evolutionary explanations include Guisinger's adapted-to-flee-famine hypothesis, proposing that reduced appetite and increased activity promoted migration from famine zones, and the reproductive suppression hypothesis, proposing adaptive suppression of reproduction during poor conditions, evidenced by amenorrhoea.
- AO3: Supporting evidence includes twin studies showing higher MZ concordance, neuroimaging demonstrating altered activity in appetite/reward circuits, and biological markers such as low leptin linked to amenorrhoea and energy deficit.
- AO3: Limitations include biological reductionism, which overlooks psychological, social and cultural contributors relevant to onset and maintenance; this matters for treatment because biological-only approaches may be insufficient.
- AO3: Evolutionary hypotheses face testability problems and rely on speculative ancestral scenarios; cultural variation in anorexia prevalence challenges a single adaptive account.
- AO3: Practical application is mixed: biological findings support medical interventions such as nutritional rehabilitation and possible pharmacotherapy, but research translation is limited because biological correlates can be consequences rather than causes of starvation.
- AO3: Consideration of interactionist perspectives: biological vulnerabilities may interact with psychological triggers and cultural pressures, producing a more complete explanation and informing multimodal treatment.
- AO3: Reasoned judgement might weigh the strength of objective biological evidence for contribution to risk and maintenance, but conclude that biological explanations are necessary but not sufficient, and best understood as part of an integrated biopsychosocial model.

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Question 3

identifies that leptin signals energy sufficiency to the hypothalamus and normally promotes satiety and supports reproductive hormone function oe	**M1**
explains that low leptin indicates low energy stores, reducing satiety signalling and altering hypothalamic regulation of appetite and metabolism oe	**M1**
explains that low leptin can suppress the hypothalamic-pituitary-gonadal axis, reducing gonadotrophin-releasing hormone (GnRH) release and leading to amenorrhoea oe	**M1**
applies these mechanisms to Erin: low leptin from weight loss disrupts appetite and energy regulation and causes menstrual suppression, accounting for both low weight maintenance and amenorrhoea oe	**A1**
links high anxiety to altered neurotransmitter/hormone function, for example that serotonin dysregulation often co-occurs and can maintain restrictive behaviour, showing an interaction between hormones and neurotransmitters oe	**A1**
offers a concise clinical implication, for example that restoring weight and leptin levels would be expected to reverse amenorrhoea and help normalise appetite signalling oe	**A1**

Answer: Leptin signals energy sufficiency to the hypothalamus and normally promotes satiety and supports reproductive function via the hypothalamic-pituitary-gonadal axis. Low leptin, as seen in Erin, signals depleted energy stores, altering hypothalamic appetite and metabolic regulation and helping to maintain low weight. Low leptin also suppresses GnRH release, reducing LH/FSH and causing amenorrhoea. Anxiety may be linked to co-occurring neurotransmitter dysregulation such as serotonin, which can reinforce restrictive eating. Clinically, restoring weight and leptin would be expected to restore menstrual function and appetite regulation.

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