13.2 Biological Explanations for Anorexia Nervosa

AQA 7182 · about 50 minutes · Write-in edition

Total Marks

Name: _____ Date: ____ / ____ / _____

Answer ALL questions.

1 Name and briefly explain one clinical implication of viewing anorexia nervosa as a biological disorder rather than as purely psychological.

(Total for Question 1 is 2 marks)

2 Outline one clear empirical criticism of evolutionary explanations (either adapted-to-flee-famine or reproductive suppression) for anorexia nervosa.

(Total for Question 2 is 3 marks)

3 Outline the role of serotonin in the biological account of anorexia nervosa. Refer to the neurotransmitter's broader functions and how altered serotonin function may influence symptoms.

(Total for Question 3 is 3 marks)

4 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 4 is 3 marks)

5 Outline twin-study evidence that supports a biological contribution to anorexia nervosa. Refer to concordance rates found in relevant research.

(Total for Question 5 is 3 marks)

6 Outline one piece of supporting empirical evidence for a neural explanation of anorexia nervosa, naming the study or type of study and its finding.

(Total for Question 6 is 3 marks)

7 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 7 is 16 marks)

8 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 8 is 6 marks)

Mark scheme · 13.2 Biological Explanations for Anorexia Nervosa

Question 1

names an implication, for example use of biological treatments such as pharmacotherapy or interventions targeting nutrition and hormonal restoration oe **B1**

briefly explains why, for example if biological factors maintain symptoms then medical approaches such as restoring weight and addressing neurotransmitter imbalance may be necessary alongside therapy oe **B1**

Answer: One implication is increased use of biological or medical interventions, for example focusing on restoring weight and hormonal balance and considering pharmacotherapy to address neurotransmitter dysfunction; this is because biological factors may maintain symptoms and need direct medical treatment as well as psychological therapy.

Question 2

identifies a relevant criticism, for example that evolutionary accounts are difficult to test empirically or that they rely on speculative ancestral scenarios oe **M1**

explains why this is a problem, for example lack of direct fossil or historical evidence and difficulty linking modern clinical cases to ancestral adaptive functions oe **M1**

may note that some empirical findings, such as the cultural variability and modern prevalence of anorexia, are hard to reconcile with a single adaptive explanation oe **A1**

Answer: A key empirical criticism is that evolutionary accounts are hard to test and rely on speculative assumptions about ancestral environments. There is limited direct evidence linking modern clinical anorexia to an ancestral adaptive foraging or reproductive strategy, and the cultural variability of anorexia makes it difficult to fit all cases to a single evolutionary explanation.

Question 3

states that serotonin is a neurotransmitter implicated in appetite regulation, mood and impulse control oe **M1**

states that altered serotonin function, for example in transporter or receptor activity, has been found in people with anorexia nervosa oe **M1**

links altered serotonin to symptoms by noting it may increase anxiety, rigid thinking or suppress appetite and so contribute to onset or maintenance of restrictive eating oe **A1**

Answer: Serotonin is involved in regulating appetite, mood and impulse control. Research indicates altered serotonin function in people with anorexia nervosa, such as differences in receptors or transporter activity. This dysregulation could increase anxiety and rigid eating patterns and/or reduce normal appetite signalling, contributing to the development or maintenance of restrictive eating.

Question 4

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 5

states that twin studies show higher concordance rates for anorexia nervosa in monozygotic (MZ) twins than dizygotic (DZ) twins oe **M1**

gives an approximate finding, for example that concordance in MZ twins is substantially higher than in DZ twins, supporting a genetic influence oe **M1**

links this to a biological contribution by noting higher MZ concordance implies heritable factors are involved in risk for anorexia nervosa oe **A1**

Answer: Twin studies have found higher concordance rates for anorexia nervosa in monozygotic than dizygotic twins. This pattern, where MZ concordance is substantially higher than DZ concordance, supports a genetic contribution to risk for anorexia nervosa because identical twins share more genetic material.

Question 6

names a relevant type of study or study, for example neuroimaging studies or twin studies oe **M1**

states the finding, for example neuroimaging shows altered activity in regions involved in appetite and reward, or twin studies show higher concordance in MZ than DZ twins oe **M1**

links the finding to neural explanation, for example altered brain activity supports involvement of neural circuits in appetite and reward that may underlie anorexia oe **A1**

Answer: An example is neuroimaging research showing altered activity in brain regions involved in appetite and reward processing in people with anorexia nervosa; this supports neural explanations by indicating that brain circuits that regulate hunger and reward are functioning differently in affected individuals.

Question 7

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.

Question 8

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.