13.4 Biological and Psychological Explanations for Obesity

AQA 7182 · about 60 minutes · Write-in edition

Total Marks

Name: _____ Date: ____ / ____ / _____

Answer ALL questions.

1 Explain briefly how social and cultural norms around food can influence obesity rates in a population.

(Total for Question 1 is 2 marks)

2 Explain one strength and one limitation of using genetic explanations (such as FTO associations) to account for obesity, with brief reasoning for why each matters for psychology.

(Total for Question 2 is 4 marks)

3 Outline evidence from twin, family and adoption studies that supports a genetic contribution to obesity.

(Total for Question 3 is 3 marks)

4 Outline how restraint theory can explain episodes of overeating and weight gain in people dieting for weight control, applied to obesity rather than anorexia.

(Total for Question 4 is 3 marks)

5 Outline one neural explanation that involves dysfunction of hypothalamic hunger/satiety signalling (other than leptin/ghrelin already covered).

(Total for Question 5 is 3 marks)

6 Outline and evaluate the boundary model and its relevance to overeating and obesity, keeping the focus brief and applied specifically to obesity rather than eating disorders.

(Total for Question 6 is 6 marks)

7 Discuss biological, psychological and social explanations for obesity. In your answer consider the relative contribution of biological versus psychological and social factors, and reach a supported judgement.

(Total for Question 7 is 16 marks)

8 Explain the roles of leptin and ghrelin as neural/hormonal mechanisms involved in hunger and satiety related to obesity.

(Total for Question 8 is 4 marks)

9 Case: Kwame is a 38-year-old man who has steadily gained 25 kg over five years despite several attempts at dieting. He reports strong hunger between meals, frequent cravings for high-fat snacks, and that strict diets often end with episodes of unplanned overeating. His family history includes two close relatives who are obese from an early age. His GP found no medical condition but noted his resting appetite-related hormone profile showed reduced sensitivity to satiety signals. Using the information in this case, explain TWO factors, one biological and one psychological or social, that could together account for Kwame's weight gain.

(Total for Question 9 is 6 marks)

Mark scheme · 13.4 Biological and Psychological Explanations for Obesity

Question 1

states that social and cultural norms shape typical portion sizes, dietary choices and attitudes to eating occasions oe — **M1**

explains that norms promoting large portions, energy-dense celebratory foods, or high consumption of convenience foods can increase average calorie intake across a population, raising obesity prevalence oe — **A1**

Answer: Cultural norms that promote large portion sizes, regular consumption of calorie-dense convenience foods or social eating occasions encourage higher average calorie intake; when many people adopt these norms, population obesity rates can rise.

Question 2

states a valid strength, e.g. genetic explanations are supported by empirical evidence such as twin/adoption studies and molecular genetics oe — **M1**

explains why this matters, e.g. provides objective, biologically based predictors which can inform medical interventions and personalised prevention oe — **A1**

states a valid limitation, e.g. genetic explanations can be overly deterministic and may underplay environmental/behavioural influences oe — **M1**

explains why this matters, e.g. ignoring modifiable social and psychological causes could hinder behavioural interventions and stigmatise individuals oe — **A1**

Answer: Strength: genetic explanations are supported by twin/adoption studies and gene-association findings, offering objective predictors that can guide medical or personalised approaches. Limitation: they risk genetic determinism and may underplay environmental and behavioural causes; this could reduce emphasis on effective social and psychological interventions and increase stigma.

Question 3

mentions that monozygotic (identical) twins show higher concordance for BMI than dizygotic twins, suggesting a genetic influence oe — **M1**

states that family studies show relatives of obese individuals are at greater risk of obesity oe — **M1**

notes that adoption studies find adoptees' BMI correlates more strongly with biological parents than with adoptive parents, supporting heritability oe — **A1**

Answer: Twin studies show higher BMI concordance for monozygotic than dizygotic twins, family studies show higher obesity risk among biological relatives, and adoption studies find adoptees resemble biological rather than adoptive parents in BMI, all supporting a genetic contribution to obesity.

Question 4

states restraint theory proposes deliberate cognitive control of eating to restrict intake, termed dietary restraint oe — **M1**

explains that when restraint is broken, for example after a 'forbidden' food is eaten, this can lead to disinhibited overeating or bingeing oe — **A1**

applies to obesity by noting repeated cycles of restraint and disinhibition can produce net weight gain over time oe — **A1**

Answer: Restraint theory proposes that cognitive efforts to restrict intake make people vulnerable to disinhibited overeating when cognitive control fails; for people aiming to lose weight, repeated cycles of strict restraint followed by loss of control can produce overall excess calorie intake and weight gain, contributing to obesity.

Question 5

identifies a neural mechanism, e.g. dysfunction in arcuate nucleus signalling, melanocortin pathway abnormality, or altered neuropeptide Y activity oe — **M1**

explains how such dysfunction can affect appetite control, e.g. impaired anorexigenic signalling leads to reduced satiety and increased food intake oe — **A1**

applies to obesity by stating that chronic disruption of hypothalamic signalling can promote persistent overeating and weight gain oe — **A1**

Answer: For example, dysfunction in hypothalamic pathways such as impaired melanocortin signalling or elevated neuropeptide Y activity can reduce anorexigenic signals and increase orexigenic drive, leading to weaker satiety responses, persistent overeating and gradual weight gain contributing to obesity.

Question 6

outlines the boundary model idea that eating is regulated between upper and lower boundaries influenced by physiological hunger and satiety and by cognitive factors like restraint oe	**M1**
applies the model to obesity, e.g. repeated cognitive restraint can lower the threshold for disinhibition so people overshoot the upper boundary and overeat, contributing to weight gain oe	**A1**
evaluates with a strength, e.g. integrates biological and cognitive factors so is useful for explaining interactions and guiding combined interventions oe	**M1**
explains why this strength matters, e.g. shows why treatments should target both physiological signals and cognitive restraint patterns to reduce relapse into overeating oe	**A1**
evaluates with a limitation, e.g. empirical support is mixed and the model is descriptive rather than mechanistic about how boundaries shift in different individuals oe	**M1**
explains why this limitation matters, e.g. without precise mechanisms it is harder to design targeted biological treatments and to predict who will benefit from cognitive approaches oe	**A1**

Answer: The boundary model proposes eating is regulated between physiological lower and upper boundaries, with cognitive restraint and external cues determining where those boundaries lie; in obesity, chronic restraint and cue-rich environments can make people repeatedly exceed the upper boundary leading to overeating. Strength: it integrates biological and cognitive explanations and supports combined interventions. Limitation: empirical tests are mixed and the model does not specify exact mechanisms for boundary shifts, limiting precise predictive use.

Question 7

Level 4 Knowledge of biological, psychological and social explanations is accurate and detailed. Discussion and evaluation are thorough and well balanced, considering evidence supporting and challenging each explanation. There is clear synthesis about the relative contributions with a reasoned and defended judgement. Application and use of terminology are precise and the answer is coherent.	**13-16**
Level 3 Knowledge is generally accurate with some detail. There is a balanced discussion with evaluation, though some points may be less developed. The relative contribution is considered and a conclusion is reached but may be less strongly defended. Application is present and terminology is used appropriately.	**9-12**
Level 2 Knowledge is limited or partial. Discussion and evaluation are present but superficial. Consideration of relative contribution is attempted but weak. Answers may be descriptive rather than analytical and application may be limited.	**5-8**
Level 1 Very limited knowledge, little or no relevant evaluation. Discussion lacks coherence and there is no meaningful judgement on relative contribution. Answer may be brief or list-like.	**1-4**
Level 0 No creditable content.	**0**

Indicative content:

- AO1: Biological explanations include genetic influences such as FTO gene variants and high heritability estimates from twin/adoption studies; neural/hormonal mechanisms include leptin resistance, ghrelin effects and hypothalamic pathway dysfunction affecting hunger and satiety.
- AO1: Psychological explanations include restraint theory and the boundary model which link cognitive control and disinhibition to overeating, and behavioural accounts such as operant conditioning and habit formation via reinforcement of palatable food consumption.
- AO1: Social explanations include cultural norms, portion size, food availability, socioeconomic status and marketing that shape eating behaviours and exposure to energy-dense foods.
- AO3: Evaluation of biological: strengths include objective, replicable genetic and physiological findings that can inform medical treatments; limitations include risk of genetic determinism and that genes interact with environment so genetic accounts alone cannot predict obesity outcomes.
- AO3: Evaluation of psychological: strengths include explanatory power for dieting cycles and binge episodes and clear intervention targets (CBT, habit change); limitations include some models being descriptive and individual differences in response to restraint or cues.
- AO3: Evaluation of social: strengths include good explanatory power for population-level trends and clear policy implications (portion control, regulation); limitations include variability between cultures and difficulty establishing causal direction.
- AO3: Integration and interaction: evidence often shows gene-environment interaction, for example genetic predisposition is expressed more strongly in obesogenic environments; psychological factors such as restraint may interact with biological hunger signals, producing cycles of weight gain.
- AO3: Consideration of relative contribution: a balanced view argues biological factors set vulnerability while social and psychological factors determine expression and course; a purely biological or purely social account is insufficient.
- AO3: A reasoned judgement could argue that the most useful approach is integrative, recognising biological predispositions but prioritising environmental and psychological interventions for public health impact, while still using biological insights for personalised treatment where appropriate.

Question 8

identifies leptin as a hormone produced by adipose tissue that signals satiety to the hypothalamus oe	**M1**
identifies ghrelin as a gut-derived hormone that stimulates appetite and signals hunger to the hypothalamus oe	**M1**
explains that leptin resistance or impaired leptin signalling can reduce satiety signalling, promoting overeating and weight gain oe	**A1**
explains that elevated ghrelin or exaggerated ghrelin responses can increase hunger and food intake, contributing to obesity oe	**A1**

Answer: Leptin, produced by fat cells, normally signals satiety to the hypothalamus; if leptin signalling is impaired or resistance develops, satiety is reduced and overeating may follow. Ghrelin, produced by the stomach, stimulates hunger and increases food intake; high ghrelin activity can promote increased eating and weight gain.

Question 9

identifies a biological factor from the case, e.g. family history of obesity or reduced sensitivity to satiety signalling (leptin resistance or hypothalamic dysfunction) oe	**M1**
explains how the biological factor could lead to weight gain, e.g. genetic predisposition via FTO or impaired satiety signalling reduces feeling of fullness, causing increased calorie intake oe	**A1**
identifies a psychological or social factor from the case, e.g. dieting and restraint leading to disinhibited overeating, or cravings for high-fat snacks influenced by reinforcement and availability oe	**M1**
explains how this factor contributes to weight gain, e.g. cycles of strict restraint and subsequent bingeing increase net calorie intake; cravings and reinforced eating of palatable foods maintain overeating oe	**A1**
links the two factors interactively, showing how biological vulnerability and psychological/social processes combine oe	**M1**
explains the interaction, e.g. genetic or neural reduction in satiety makes dieting harder, increasing susceptibility to disinhibition and reinforced snack consumption, so both factors together accelerate weight gain oe	**A1**

Answer: Biological: reduced sensitivity to satiety signalling and a family history of obesity suggest a genetic or hypothalamic vulnerability, meaning Kwame feels less full and so eats more. Psychological/social: repeated strict dieting and resultant restraint followed by disinhibited overeating, together with cravings for high-fat snacks that are readily available and rewarding, increase calorie intake. Interaction: the biological tendency to weak satiety makes dieting more likely to fail and increases the impact of cravings and reinforcement, so the combination explains sustained weight gain.