

Paper 3 · Section C option · Eating Behaviour

A-level topic mock · 2026 · Maximum mark: 48

Eating Behaviour is A-level only (AQA spec 4.3.6) — it is a Paper 3 option and is not assessed at AS. Indicative content is not exhaustive; credit any other valid points. Levels-based questions (Q10 and Q11) require holistic judgement using the descriptors — match the answer to the band that best fits, then fine-tune within it. Specialist vocabulary (neophobia, taste aversion, hypothalamus, ghrelin, leptin, satiety, anorexia nervosa, enmeshment, autonomy, modelling, restraint theory, disinhibition, the boundary model) follows AQA's 2025 wording. **Note (2025 spec):** "explanations for the success and failure of dieting" has been **removed**; restraint theory, disinhibition and the boundary model are retained as psychological explanations for *obesity*.

C Eating Behaviour**0 1**AO1 · 1 mark multiple choice

| Which one of the following best describes taste aversion?

Answer: B — Learning to avoid a food after it has been followed by illness.

A describes neophobia; C describes the evolved preference for sweet/energy-rich foods; D describes social learning (modelling).

0 2AO1 · 1 mark multiple choice

| Which one of the following hormones is released by fat cells and signals satiety to the brain?

Answer: B — Leptin.

A (ghrelin) is the "hunger hormone", secreted by the stomach to stimulate appetite; C (insulin) regulates blood glucose; D (serotonin) is a neurotransmitter linked to mood and satiety signalling but is not released by fat cells.

0 3

AO1 · 1 mark multiple choice

| Which one of the following best describes disinhibition in explanations of obesity?

Answer: C — The abandonment of dietary restraint, leading to overeating.

A describes a genetic explanation; B describes the thrifty-gene / evolutionary fat-storage idea; D describes a neural factor. Only C captures disinhibition (the "what-the-hell" collapse of restraint in the boundary model).

0 4

AO1 · 3 marks short answer

| Outline the role of the hypothalamus in the control of eating behaviour.

Marks for this question: AO1 = 3 marks

- **1–2 marks** for the **lateral hypothalamus (LH)** as the "hunger/feeding centre": activated by falling glucose / ghrelin (and neuropeptide Y); stimulation triggers eating, and damage causes **aphagia** (failure to eat).
- **1–2 marks** for the **ventromedial hypothalamus (VMH)** as the "satiety centre": activated when full; stimulation stops eating, and damage causes **hyperphagia** (overeating) and weight gain.
- Credit the idea of **homeostasis** — the hypothalamus monitors blood glucose and hormonal signals (ghrelin, leptin) to keep energy balance stable (the dual-centre model).

Award up to 3 marks. Full marks possible from a detailed account of the LH/VMH dual-centre model.

Use your knowledge of the role of learning to explain Tom's food preferences. Refer to social and cultural influences.

Marks for this question: AO2 = 4 marks

- **4 marks** — Clear, coherent application of at least two learning mechanisms to the stem, using accurate terminology.
- **3 marks** — Effective but one mechanism less developed.
- **2 marks** — One mechanism applied to the stem.
- **1 mark** — Brief/partial; minimal use of the stem.

Indicative content:

- **Operant conditioning / reinforcement:** chocolate was repeatedly given as a **reward** for good behaviour, so it became positively associated and preferred (reward → preference).
- **Social learning / modelling:** Tom **observed his older brothers** (models) enthusiastically eating spicy food and imitated them — vicarious reinforcement from seeing their enjoyment.
- **Cultural influence:** spicy food is "a popular part of his family's culture", so social norms and repeated exposure shaped his preference.

Top-band answers name the mechanisms (reinforcement, modelling, cultural norms) AND tie each to a specific detail of Tom's experience.

Use restraint theory and/or the boundary model to explain Nadia's eating behaviour.

Marks for this question: AO2 = 4 marks

- **4 marks** — Clear explanation linking the theory to the stem, using accurate terminology (restraint, diet boundary, disinhibition).
- **3 marks** — Effective but slightly underdeveloped.
- **2 marks** — Theory applied but link to the stem limited.
- **1 mark** — Brief/partial.

Indicative content:

- **Restraint theory:** deliberately restricting intake (Nadia's "strict daily calorie limit") paradoxically makes overeating *more* likely, because the restriction cannot be sustained.
- **Boundary model (Herman & Polivy):** restrained eaters set a cognitive "diet boundary" below their physiological satiety boundary. Eating the biscuit pushes Nadia past her diet boundary, so restraint collapses.
- **Disinhibition / the "what-the-hell" effect:** her thought "I've broken my diet now anyway" releases the restraint, and she overeats up to (or beyond) her satiety boundary for the rest of the evening.

Full marks require the disinhibition idea to be explicitly tied to Nadia's quoted thought and subsequent overeating.

Outline biological explanations for anorexia nervosa. Refer to genetic and/or neural explanations.

Marks for this question: AO1 = 4 marks

- **Genetic** (up to 4): anorexia nervosa (AN) runs in families; **higher concordance in MZ than DZ twins** (e.g. Holland et al.); first-degree relatives of sufferers are at substantially greater risk; AN is polygenic, with candidate genes affecting serotonin systems. Adoption/family evidence points to heritability.
- **Neural** (up to 4): **serotonin** and **dopamine** dysfunction — disturbed serotonin activity affecting anxiety and appetite; dopamine abnormalities in the reward system (restriction becomes reinforcing). Structural/functional differences (e.g. in the insula, which integrates taste and body signals).

Award up to 4 marks across genetic and/or neural. Full marks are available from one area if sufficiently detailed.

0 8

AO1 · 3 marks short answer

Outline a biological explanation for obesity. Refer to genetic and/or neural factors.

Marks for this question: AO1 = 3 marks

- **Genetic:** BMI is highly heritable; twin and adoption studies show a child's weight tracks their **biological** parents'; candidate genes such as the **FTO gene** are associated with raised obesity risk.
- **Neural: leptin resistance** — the brain stops registering leptin's satiety signal, so eating continues; dysfunction of the **ventromedial hypothalamus**; set-point theory (the body defends a higher weight).

Award up to 3 marks. Either genetic or neural detail can reach full marks.

0 9

AO1 · 3 marks short answer

Outline the role of ghrelin and leptin in the control of eating behaviour.

Marks for this question: AO1 = 3 marks

- **Ghrelin** (the "hunger hormone"): secreted by the **stomach** when it is empty; levels rise before meals and fall after eating; it acts on the **hypothalamus** to stimulate appetite.
- **Leptin:** secreted by **adipose (fat) cells**; signals long-term energy stores and **satiety**; high leptin acts on the hypothalamus to suppress appetite. (Leptin resistance is linked to obesity.)

Award at least 1 mark for the accurate role of each hormone; 3rd mark for additional detail (site of release, action on the hypothalamus, timing).

Discuss the evolutionary explanation for food preferences. Refer to at least one strength and one limitation.

Marks for this question: AO1 = 4 marks, AO3 = 4 marks

Level	Marks	Descriptor
4	7–8	Knowledge of the evolutionary explanation is accurate and well detailed. Evaluation includes at least one strength and one limitation, both effectively explained. Clear, coherent, focused; specialist terminology used effectively.
3	5–6	Knowledge generally accurate; evaluation mostly effective but limited in places. Reasonable structure.
2	3–4	Some accurate knowledge. Evaluation limited; mainly descriptive.
1	1–2	Knowledge limited or muddled. Little or no evaluation.
0	0	No relevant content.

Indicative AO1 content:

- **Preference for sweet / energy-dense foods:** adaptive in the ancestral environment (EEA), where calories were scarce — a taste for sweet (ripe fruit) and fatty foods aided survival and was naturally selected.
- **Bitter aversion:** many toxins taste bitter, so an innate dislike of bitterness protected against poisoning.
- **Neophobia:** a cautious avoidance of novel foods (strongest in early childhood) reduced the risk of eating something dangerous.
- **Taste aversion learning** (the Garcia effect): rapidly learning to avoid a food after a single episode of illness — even with a long delay — is a highly adaptive, evolved survival mechanism.

Indicative AO3 content (any combination):

- **Strength — supporting evidence for taste aversion:** **Garcia & Koelling** showed rats readily associate taste (not audio-visual cues) with nausea, demonstrating biological preparedness consistent with the evolutionary account.
- **Strength — explains universal preferences:** a preference for sweetness is present in newborns and across all cultures, supporting an innate, evolved basis rather than purely learned tastes.
- **Limitation — cannot explain variation:** the approach struggles to account for cultural and individual differences (e.g. acquired tastes for chilli, bitter coffee or alcohol), which learning explanations handle better.
- **Limitation — evolutionary mismatch:** a once-adaptive preference for fat and sugar is now maladaptive in calorie-rich environments, contributing to obesity — the theory explains the preference but not why we override satiety today.

- **Limitation — difficult to test / "just-so stories"**: claims about selection pressures in the EEA are speculative and hard to falsify, weakening the scientific status of the explanation.
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Discuss psychological explanations for anorexia nervosa. Refer to the case of Maya as part of your discussion.

Marks for this question: AO1 = 6 marks, AO2 = 4 marks, AO3 = 6 marks

Level	Marks	Descriptor
4	13–16	Knowledge of psychological explanations is accurate and generally well detailed. Application to Maya is effective and integrated across the stem. Discussion is thorough and effective. Clear, coherent and focused; specialist terminology used effectively.
3	9–12	Knowledge evident with some accuracy. Application mostly effective. Discussion mostly effective but limited in places.
2	5–8	Some accurate knowledge of one or more explanations. Application limited. Discussion superficial / mainly descriptive.
1	1–4	Knowledge limited; little or no application or discussion.
0	0	No relevant content.

Indicative AO1 content — credit any of the three psychological explanations (a good answer covers two or more):

- **Family systems theory (Minuchin):** AN develops within a dysfunctional family characterised by **enmeshment** (over-close relationships with weak boundaries), denial of **autonomy** (the child is not allowed independence) and over-**control**. Refusing food becomes the child's way of asserting control and individuality.
- **Social learning theory:** disordered eating is learned through **modelling** (imitating thin celebrities/models), **reinforcement** (praise/attention for thinness or weight loss) and **media** promotion of the thin ideal.
- **Cognitive theory:** AN involves **distortions** (notably body-image distortion — perceiving oneself as fat) and **irrational beliefs** / faulty schemas about weight, food and self-worth.

Indicative AO2 content — engagement with Maya:

- **Family systems:** "extremely close to her mother, who plans almost every part of Maya's daily life and rarely allows her to make her own decisions" = enmeshment and a denial of autonomy/over-control; restricting food may be Maya's bid for control.
- **SLT:** spending "hours each day following... very thin models and influencers" and wanting to "look just like them" = modelling and media influence / thin-ideal internalisation.
- **Cognitive:** being "convinced that she is fat" while "severely underweight" = body-image distortion; "only be happy once she has lost more weight" = an irrational, distorted belief.

Indicative AO3 content:

- **Strength — research support for media/SLT (Becker et al. 2002):** after television was introduced to Fiji, disordered-eating attitudes among girls rose sharply, supporting the role of media modelling in AN.
- **Strength — cognitive distortions are well evidenced:** studies consistently find body-image distortion in AN patients, and this underpins the success of **CBT** as a treatment — giving the cognitive account practical/applied value.
- **Limitation — direction of causality:** distorted cognitions and family tension may be a *consequence* of starvation/AN rather than its cause (semi-starvation itself impairs thinking), so cause and effect are hard to separate.
- **Limitation — ignores strong biological evidence:** purely psychological accounts neglect the substantial genetic/neural evidence (MZ > DZ concordance); an **interactionist / diathesis-stress** model is arguably more complete.
- **Limitation — socially sensitive (family systems):** blaming family dynamics risks stigmatising parents (echoing discredited "blame-the-mother" accounts), so these explanations must be used with ethical care.
- **Limitation — lack of specificity:** huge numbers of girls are exposed to thin-ideal media and controlling families, yet very few develop AN — the explanations cannot easily say why particular individuals are affected.

Top-band answers will (1) describe at least two psychological explanations (family systems, SLT, cognitive) accurately; (2) map Maya's mother/enmeshment onto family systems, her social-media use onto SLT/media, and her "convinced she is fat" belief onto cognitive distortion; (3) evaluate with named evidence (e.g. Becker, body-image research) and at least one limitation; and (4) reach a clear conclusion — typically that psychological explanations (especially cognitive and media) are well supported and clinically useful, but are best combined with biological factors and limited by problems of causal direction.