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A-LEVEL PSYCHOLOGY REVISION NOTES

Eating Behaviour

AQA Psychology 7182 (A-level only)

2025 specification · spec section 4.3.6 · A-level Paper 3

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Contents

2025 spec change: "Explanations for the success and failure of **dieting**" has been **removed**. Restraint theory, disinhibition and the boundary model are retained, but now as psychological explanations for **obesity**. Past-paper questions framed around the success/failure of dieting are now obsolete.

AQA 2025 SPECIFICATION — EATING BEHAVIOUR (4.3.6)

- **Food preferences**: the evolutionary explanation (neophobia, taste aversion); the role of learning (social and cultural influences).
- **Neural and hormonal mechanisms** in the control of eating: the hypothalamus, ghrelin and leptin.
- **Anorexia nervosa**: biological explanations (genetic and neural); psychological explanations (family systems theory; social learning theory; cognitive theory).
- **Obesity**: biological explanations (genetic and neural); psychological explanations (restraint theory, disinhibition and the boundary model).

1 Explanations for Food Preferences: Evolutionary and Learning

The Evolutionary Explanation

Food preferences that aided survival in the **environment of evolutionary adaptedness (EEA)** were naturally selected and persist today:

- **Preference for sweet and energy-dense (fatty) foods** — in an environment of food scarcity, a taste for high-calorie foods promoted survival. Sweetness signalled ripe, energy-rich fruit.
- **Aversion to bitter and sour tastes** — many toxins and unripe or rotten foods are bitter or sour, so avoiding them reduced the risk of poisoning.
- **Neophobia** — a cautious avoidance of *new, unfamiliar* foods. Strongest in early childhood, it protected against eating something dangerous. The matching tendency to accept foods seen others eat safely is sometimes called *neophilia*.
- **Taste aversion learning (the Garcia effect)** — rapidly learning to avoid a food after it is followed by illness, often after a **single pairing** and even with a **long delay** between eating and sickness. This "biologically prepared" learning is highly adaptive.

The Role of Learning

Food preferences are also acquired through experience:

- **Operant conditioning** — foods given as *rewards* become preferred; using food as a reward increases liking for it.
- **Social learning / modelling** — children imitate the food choices of parents, peers and media figures, especially when models are reinforced (appear to enjoy the food).
- **Cultural influences** — cuisine, availability and social norms shape what is considered desirable or disgusting (e.g. spicy food, insects, particular meats vary by culture).

Evaluation

A key strength of the evolutionary explanation is research support for taste aversion. Garcia and Koelling found that rats readily associated a novel taste — but not audio-visual cues — with nausea, demonstrating "biological preparedness" to learn taste–illness links. The implication is that this learning is not arbitrary but specialised for survival, exactly as the evolutionary account predicts. Because the same one-trial, long-delay taste aversion is found across many species including humans, it strongly suggests an evolved, adaptive mechanism rather than ordinary learning.

A further strength is that the approach explains universal, innate-seeming preferences. A preference for sweetness is present in newborn infants and is found in every culture studied, which is difficult to explain through learning alone. This cross-cultural and developmental consistency supports the claim that some food preferences are inherited adaptations, giving the evolutionary explanation genuine predictive power.

However, a major limitation is that the evolutionary account cannot explain cultural and individual variation. Many people acquire tastes for foods that are bitter or initially aversive — chilli, coffee, alcohol — which natural selection would predict we should avoid. This is far better explained by the **role of learning** (exposure, reinforcement, cultural norms). The debate therefore illustrates **nature versus nurture**: rather than competing, the two explanations are complementary, with evolution setting innate biases that learning then modifies.

A counterpoint that further limits the approach is the problem of "evolutionary mismatch". An innate preference for fat and sugar that was adaptive when calories were scarce is now *maladaptive* in an environment where such foods are abundant, contributing to obesity (Section 5). This shows the explanation accounts for the preference but not for why people override satiety in modern conditions. Combined with the criticism that claims about the EEA are speculative "just-so stories" that cannot be falsified, this weakens the scientific status of the explanation.

2 Neural and Hormonal Control of Eating: Hypothalamus, Ghrelin and Leptin

> **HOMEOSTASIS**
> Eating is regulated by **homeostasis** — the body's tendency to maintain a stable internal state (here, energy balance). The hypothalamus monitors signals of energy stores and triggers hunger or satiety to keep weight stable.

The Hypothalamus — Dual-Centre Model

Region	Role
Lateral hypothalamus (LH)	The "hunger / feeding centre". Activated by falling glucose and by **ghrelin** (and neuropeptide Y). Stimulation triggers eating; damage causes **aphagia** (failure to eat).
Ventromedial hypothalamus (VMH)	The "satiety centre". Signals fullness and inhibits eating. Damage causes **hyperphagia** (overeating) and obesity.

Hormones

- **Ghrelin** — the "hunger hormone", secreted by the **stomach** when empty. Levels rise before meals and fall after eating; ghrelin acts on the hypothalamus to stimulate appetite.
- **Leptin** — secreted by **adipose (fat) cells**. It signals long-term energy stores and **satiety**: high leptin suppresses appetite. *Leptin resistance* (the brain ceasing to respond to leptin) is linked to obesity.

Evaluation

A strength of the neural and hormonal account is strong, objective supporting evidence. Animal lesion studies show that damaging the LH and VMH produces exactly the predicted aphagia and hyperphagia, and people with rare leptin deficiency overeat and become severely obese until given leptin. Because these findings come from controlled, physiological measurement rather than self-report, they have high internal validity, providing convincing evidence that the hypothalamus and these hormones really do regulate eating.

This account also has valuable real-world and economic application. Understanding ghrelin and leptin has informed anti-obesity drugs and helps explain why appetite increases after weight loss (rising ghrelin), which is one reason diets fail. Given the enormous cost of obesity to the NHS, interventions grounded in this biology have clear economic value, strengthening the case for the approach.

However, a limitation is that the simple dual-centre model is oversimplified. Eating involves far more than two "on/off" switches: other hypothalamic regions (e.g. the paraventricular nucleus), the brainstem and the cortex are all involved, and damage to areas beyond the LH/VMH also affects eating. This means the classic model, while a useful starting point, does not capture the true complexity of the neural control of eating.

A counterpoint is that homeostatic explanations are biologically reductionist. They explain eating as a response to internal energy needs, yet humans frequently eat when *not* hungry — when stressed, bored, socialising or simply faced with palatable food. By ignoring these powerful cognitive, emotional and social influences, a purely neural/hormonal account cannot fully explain human eating behaviour, and is best combined with the psychological explanations in Sections 4 and 6.

3 Anorexia Nervosa: Biological Explanations

Anorexia nervosa (AN) is a serious eating disorder involving restriction of food intake, significantly low body weight, an intense fear of gaining weight, and a distorted body image. It affects around 1% of people, predominantly females, and has one of the highest mortality rates of any mental disorder.

Genetic Explanations

- AN **runs in families**: first-degree relatives of sufferers are at substantially higher risk.
- **Twin studies** show higher concordance in MZ than DZ twins — **Holland et al. (1988)** reported around 56% concordance for MZ vs 5% for DZ twins.
- AN is **polygenic**; candidate genes affect the **serotonin** system.

Neural Explanations

- **Serotonin** — disturbed serotonin activity is implicated, affecting anxiety, mood and appetite regulation.
- **Dopamine** — abnormalities in the dopamine reward system may make food restriction feel rewarding/reinforcing.
- **Structural/functional differences** — e.g. in the **insula** (which integrates taste and body-state signals) and reduced grey matter.

Evaluation

A strength of the biological explanation is twin-study evidence of a genetic basis. Holland et al. (1988) found around 56% concordance for AN in MZ twins versus just 5% in DZ twins. Since MZ twins share 100% of their genes and DZ twins only 50%, this large gap points to a substantial genetic contribution, supporting the claim that AN is at least partly inherited rather than purely socially caused.

However, a limitation is that concordance is well below 100%, which the model cannot explain on its own. If AN were entirely genetic, MZ twins would always be concordant, yet around 44% are not. This shows genes create a *vulnerability* rather than a certainty, and that environmental triggers — dieting, media pressure, family dynamics — must also play a role. This is a strong argument for a **diathesis-stress / interactionist** account rather than a purely biological one, illustrating the **nature–nurture** debate.

A further limitation is the problem of cause and effect. Neural differences such as altered serotonin activity and reduced grey matter could be a *consequence* of self-starvation rather than its cause, because prolonged under-eating itself changes brain chemistry and structure. Without longitudinal data showing these differences *precede* the disorder, the causal claim of the neural explanation remains uncertain.

Finally, a purely biological account is reductionist and risks being deterministic. Reducing AN to genes and neurotransmitters overlooks the well-evidenced roles of family systems, social learning and cognition (Section 4). It can also imply that sufferers are simply "wired" to develop AN, which is both an oversimplification and potentially unhelpful for treatment, since it downplays the psychological factors that therapies such as CBT successfully target.

4 Anorexia Nervosa: Psychological Explanations

Family Systems Theory (Minuchin)

AN develops within a dysfunctional family characterised by:

- **Enmeshment** — over-close relationships with weak personal boundaries; family members are over-involved in each other's lives.
- **Denial of autonomy** — the child is not allowed to become independent.
- **Over-control** and conflict-avoidance. Refusing food becomes the child's way of asserting *control* and individuality.

Social Learning Theory

- **Modelling** — imitating thin models, celebrities and influencers (the "thin ideal").
- **Reinforcement** — receiving praise or attention for thinness or weight loss.
- **Media** — repeated exposure to idealised thin bodies promotes internalisation of the thin ideal.

Cognitive Theory

- **Distortions** — particularly **body-image distortion**: perceiving oneself as fat despite being underweight.
- **Irrational beliefs** and faulty **schemas** about weight, food, control and self-worth.

Evaluation

A strength of the social learning / media explanation is research support from a natural experiment. Becker et al. (2002) found that after television was introduced to Fiji, disordered-eating attitudes among adolescent girls rose sharply, with many expressing a wish to lose weight to resemble Western characters. Because this tracked a real change in media exposure over time, it provides convincing evidence that modelling of the thin ideal contributes to disordered eating, supporting the SLT account.

The cognitive explanation is also well supported and clinically useful. Body-image distortion is reliably found in AN patients, and the cognitive account directly underpins **CBT**, which challenges distorted beliefs about weight and food. The effectiveness of a therapy derived from the theory provides indirect support for it, and has economic value: helping sufferers recover reduces the very high costs of long-term inpatient treatment and lost productivity associated with this disorder.

However, a limitation common to these explanations is the problem of cause and effect. The distorted cognitions and family tensions seen in AN may be a *consequence* of the disorder (and of starvation, which impairs thinking) rather than its cause. Because much of the evidence is correlational and gathered after onset,

it cannot establish that faulty thinking or family dysfunction came first, weakening the causal claims.

A further limitation is that family systems theory is socially sensitive, and the explanations lack specificity. Blaming family dynamics risks stigmatising parents, echoing the discredited "blame-the-mother" accounts once applied to other disorders, so it must be used with ethical care. Moreover, the vast majority of girls are exposed to thin-ideal media and many to controlling families, yet only around 1% develop AN. This inability to explain *why specific individuals* are affected strongly implies a biological vulnerability is also required — pointing again to an interactionist account.

5 Obesity: Biological Explanations

Obesity is usually defined as a body mass index (BMI) of 30 or above. It develops when energy intake chronically exceeds energy expenditure and is associated with serious health risks.

Genetic Explanations

- **BMI is highly heritable.** Twin and adoption studies — e.g. **Stunkard et al. (1986)** — found adoptees' weights resembled their **biological** parents far more than their adoptive parents.
- **Candidate genes** such as the **FTO gene** are associated with raised obesity risk.
- The **thrifty gene hypothesis**: genes that promoted efficient fat storage during ancestral famines now predispose to obesity in food-rich environments.

Neural Explanations

- **Leptin resistance** — the brain stops responding to leptin's satiety signal, so eating continues despite ample fat stores.
- **Hypothalamic dysfunction** — e.g. impaired VMH (satiety) function.
- **Set-point theory** — the body defends a particular (sometimes raised) weight, resisting weight loss.
- **Reward circuitry** — differences in dopamine reward responses to food.

Evaluation

A strength of the genetic explanation is powerful adoption-study evidence. Stunkard et al. (1986) found that adoptees' body weights closely resembled those of their **biological** parents but not their adoptive parents. Because adoptees are raised in a different food environment from their biological parents, this design separates genes from upbringing, providing strong evidence that genetic factors influence body weight independently of the home environment.

Converging biological evidence further supports the approach. Specific obesity-related genes such as FTO have been identified, and rare cases of leptin deficiency cause severe early obesity that is reversed by leptin treatment. This biological evidence has practical value, helping to de-stigmatise obesity by showing it is not simply a matter of willpower, and informing medical interventions.

However, a major limitation is that genes alone cannot explain the rapid rise in obesity. Obesity rates have soared within just a few decades — far too quickly for the gene pool to have changed. This points firmly to the **obesogenic environment** (cheap, calorie-dense food and sedentary lifestyles) interacting with genetic predisposition. The most convincing account is therefore **interactionist**: genes load the gun, but the environment pulls the trigger, illustrating the nature–nurture debate.

A further limitation is that a purely biological explanation is reductionist and risks determinism. Explaining obesity solely through genes and brain mechanisms neglects the psychological factors — restraint, disinhibition and emotional eating (Section 6) — that strongly affect intake. Framing obesity as biologically

determined could also discourage people from attempting behaviour change, when evidence shows psychological and environmental interventions can be effective.

6 Obesity: Psychological Explanations

Restraint Theory

Restraint theory proposes that deliberately attempting to eat less (cognitive **restraint**) paradoxically makes **overeating** more likely. Trying not to think about food increases its salience, and the restriction is hard to sustain — so restrained eaters are prone to bouts of overeating.

The Boundary Model (Herman & Polivy)

> **THE BOUNDARY MODEL**
> Eating is bounded by hunger (a lower, physiological boundary) and satiety (an upper, physiological boundary). **Restrained eaters** impose an additional **cognitive "diet boundary"** below their natural satiety point. Once they cross this self-imposed boundary, restraint collapses and they eat freely up to (or beyond) satiety.

- **Disinhibition** — the breakdown of restraint, often triggered by one "forbidden" food.
- The **"what-the-hell" effect** — the thought "I've broken my diet, so I might as well carry on" releases the restraint and produces overeating.

Evaluation

A strength of the boundary model is experimental support for disinhibition. In "preload" studies (e.g. Herman & Mack), restrained eaters who were given a high-calorie milkshake went on to eat *more* ice cream than unrestrained eaters — the opposite of what dieting intends. Because this counter-regulation was produced under controlled conditions, it provides convincing evidence that crossing the cognitive diet boundary triggers disinhibited overeating, supporting the theory.

The theory also has valuable practical and economic application. It explains why restrictive dieting so often backfires, and supports non-restrictive, "intuitive eating" approaches that may be more sustainable. Given the very high cost of obesity-related illness to the NHS, interventions informed by restraint theory — which discourage rigid dieting — have clear economic as well as health value.

However, a limitation is that the theory cannot explain all obesity. Many obese people are not dieters or restrained eaters, and conversely not all restrained eaters overeat or become obese. This shows that restraint and disinhibition describe *some* patterns of overeating rather than the whole phenomenon, so the theory is better seen as an explanation of disinhibited eating than a complete account of obesity.

A counterpoint is that the psychological account, taken alone, ignores biology and is therefore incomplete. By focusing on cognitive restraint, the theory neglects the genetic and neural factors (Section 5) — leptin resistance, set-point, reward circuitry — that also drive overeating. As with anorexia, the fullest explanation of obesity is **interactionist**, combining a biological predisposition with psychological and environmental triggers.

These notes were prepared for Simply Psychology and cover spec section 4.3.6 of the AQA Psychology 2025 specification (A-level only, Paper 3). "Explanations for the success and failure of dieting" was **removed** in 2025; restraint theory and the boundary model are retained as explanations for obesity. For deeper coverage, see simplypsychology.org/eating-behavior.html.