

AQA Psychology A-level

Option 2: Eating Behaviour

Summary Notes

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Part 1: Evolutionary Explanations for Food Preference

Evolutionary explanations suggest that some eating behaviours developed because they provided an **adaptive advantage**, increasing our ancestors' chances of **survival and reproduction**. Early humans were **hunter-gatherers**, so food preferences helped them identify foods that provided useful energy and nutrients while avoiding potentially harmful foods.

1. Food Preferences

In the **Environment of Evolutionary Adaptedness (EEA)**, food was not always available. Therefore, preferences for **energy-dense and nutritious foods** would have been beneficial.

Food Preference	Evolutionary Explanation
Fatty food	Fat provides around twice the energy per gram of carbohydrate or protein. It also increases palatability , making energy-dense foods more appealing
Meat	Meat provides important nutrients, including iron , which is needed for normal bodily functioning.
Salt	Salt is essential for normal bodily functioning . Harris et al. (1990) found that 4-month-old breast-fed babies preferred salted cereals despite little previous exposure to high levels of salt, suggesting that salt preference may be innate .
Sweet foods	Sweetness signals sugar and energy . Fruit also provides important vitamins, minerals and fibre , while fructose provides a rapid source of energy. Steiner (1977) found that children could distinguish between different types of sugar. Mennella (2014) found that children who preferred sweet solutions tended to be taller for their age .

These preferences may therefore have helped early humans select foods that provided **energy and essential nutrients**, increasing their chances of survival.



2. Taste Aversion

Taste aversion is a **learned response** in which an individual avoids a food after becoming **ill or poisoned** after eating it.

This would have been adaptive because avoiding the food in the future would reduce the risk of **being poisoned again**.

Garcia et al. (1955) demonstrated taste aversion in rats.

- Rats consumed **saccharin**.
- They were then made ill through **radiation**.
- The rats later developed an **aversion to saccharin** and avoided consuming it.

This shows that animals can **learn to avoid food associated with illness**.

Taste aversion can also be linked to **Seligman's theory of preparedness**. Organisms are more easily conditioned to fear or avoid **prepared stimuli** that present an evolutionary threat, such as **poison, fire and deep water**.

3. Food Neophobia

Food neophobia is the **reluctance to eat new or unfamiliar foods**.

From an evolutionary perspective, this could protect individuals from consuming **poisonous or harmful foods** that they had not encountered before.

Ratcliffe et al. (2003) found that animals with **restricted diets**, such as koalas, showed little food neophobia, whereas species with **broader diets** were more likely to display it. This supports the idea that neophobia may be **adaptive**, as animals that encounter a wider range of unfamiliar foods have greater reason to avoid potentially harmful foods.

In humans, **Birch et al. (1987)** found that food neophobia is strongest between approximately **2–6 years of age**. This is a period when children are increasingly exposed to unfamiliar foods without their parents being present to identify whether they are safe.

However, **repeated exposure** to unfamiliar foods can increase preference for them, suggesting that **learning and experience can influence an evolutionary predisposition**.



AO3 - EVALUATION

One strength of evolutionary explanations of eating behaviour is that there is **research support for the idea that some food preferences are innate rather than entirely learned.**

Steiner (1977) found that newborn babies showed a preference for **sweet tastes** the first time they experienced them, demonstrated through positive facial expressions such as smiling. This supports the evolutionary explanation because sweet foods would have provided early humans with a valuable source of **energy and nutrients**, meaning an inherited preference for sweet tastes would have increased survival chances. Therefore, evolutionary explanations are supported by evidence suggesting that some food preferences may have developed through **natural selection**.

A further strength of evolutionary explanations is that **research supports the idea that food neophobia may be an evolved survival mechanism.** **Knaapila et al. (2007)** studied **468 pairs of adult female twins** using the **Food Neophobia Scale** and found a **heritability estimate of 67%**, suggesting that food neophobia has a strong genetic component. This supports the evolutionary explanation because avoiding unfamiliar foods would have helped early humans avoid potentially **harmful or poisonous foods**, while still allowing them to explore new foods that could provide important nutrients. Therefore, food neophobia may represent an adaptive behaviour that developed because it increased survival.

Another strength of evolutionary explanations is that **research into taste aversion has led to useful real-world applications.** **Bernstein and Webster (1980)** found that chemotherapy patients who consumed a novel-flavoured ice cream before treatment developed an aversion to the new food rather than familiar foods. This supports the idea that food preferences and avoidance behaviours may be influenced by adaptive mechanisms, as the new food acted as a “**scapegoat**”, protecting the patient’s usual diet from becoming associated with illness. Therefore, understanding the mechanisms behind food avoidance has practical value, as it can be applied to improve nutritional outcomes for patients undergoing chemotherapy.

One limitation of evolutionary explanations of eating behaviour is that they may be **reductionist**. These explanations focus heavily on biological adaptations while ignoring other important influences on food preferences. For example, eating behaviour is also shaped by **learning, culture and social experiences**, such as family eating habits and exposure to different foods. This suggests that evolutionary explanations may provide only a partial explanation of eating behaviour because preferences are likely influenced by an interaction between **nature and nurture**. Therefore, evolutionary explanations may oversimplify the complexity of human eating behaviour.

A further limitation of evolutionary explanations is that they may have **limited ability to explain eating behaviours in the modern environment**. Preferences for **high-fat and high-sugar foods** may have been adaptive for early humans because these foods provided energy when food was scarce. However, in modern societies where these foods are widely available, these evolved preferences may contribute to **overeating and obesity**. Therefore, although evolutionary explanations may explain why certain preferences developed, they may not fully explain current patterns of eating behaviour.



Part 2 : The Role of Learning in Food preference

Eating behaviour is **influenced** by **learning and the social and cultural environment**.

Children can learn what, when and how much to eat by observing **parents and peers**, while wider cultural influences such as the **media and meal patterns** can shape food preferences.

Influence	How it influences food preferences	Research
Parents	Parents are important role models , so children may learn what and how to eat by observing and imitating their parents. Parents can also shape preferences by controlling which foods are available and by using certain foods as rewards . For example, telling a child they can have pudding after eating their vegetables may unintentionally make the pudding more desirable.	Brown & Ogden (2004) found consistent correlations between parents and children in snack food intake, eating motivations and body dissatisfaction , suggesting that parental behaviour can influence children's eating behaviour and attitudes.
Peers	Children can learn food preferences by observing and imitating their peers . This can be explained through Social Learning Theory , as children may pay attention to a peer's behaviour, remember it and reproduce it, particularly when the peer is a similar age or is positively reinforced for eating a particular food.	Birch et al. (1980) found that children's vegetable preferences changed after exposure to peers with different preferences.
Media	The media can influence food preferences by providing information about food and exposing children to food advertising. Advertisements often use fun themes, celebrities and characters that children can identify with. Through social learning , children may imitate the food choices promoted by these role models.	Macintyre et al. (1998) found that the media can influence both what people eat and their attitudes towards food . However, the effect of media information may depend on factors such as age and income , which affect whether people can act on the information.



Culture	Food preferences are influenced by the cultural environment in which a person grows up. Culture teaches people which foods are considered acceptable, desirable or inappropriate , as well as what constitutes a “ proper meal ”. For example, Western cultures may place greater emphasis on meat and vegetables, whereas some East Asian cultures may place greater emphasis on several smaller dishes eaten together.	Different cultures have different attitudes towards foods such as offal and different ideas about a “proper meal”.
Meal context	The context in which food is eaten can shape preferences. Busy lifestyles can increase demand for convenient food, encouraging people to graze rather than eat traditional meals. Eating in front of the TV or in other informal settings may also encourage preferences for food that is quick and easy to prepare.	Maguire et al. (2015) found that the number of takeaway restaurants in the UK increased by 45% over 18 years , with the greatest increase in areas of higher deprivation . Gillman et al. (2000) found that eating more informally, such as in front of the TV, was associated with a greater preference for quickly prepared food .
Flavour-Flavour Learning	Food preferences can also be learned through Pavlovian conditioning . An unfamiliar or neutral flavour can be repeatedly paired with a flavour that is already liked . Over time, the neutral flavour becomes associated with the pleasant flavour, making the unfamiliar food more likely to be preferred.	Havermans & Jansen (2007) found that children developed a greater preference for a vegetable flavour after it had been repeatedly paired with a sweet flavour .



AO3 - EVALUATION

One strength of learning explanations of food preferences is that **research supports the role of social learning in shaping children's eating behaviours**. **Boyland and Halford (2013)** found that food advertising influenced children's **food preferences and intake**, particularly for foods high in **fat, salt and sugar**. The influence was strongest in overweight children, who were more likely to prefer high-energy foods and also watched more television. This supports the role of **vicarious reinforcement**, as children may learn to prefer certain foods by observing and being repeatedly exposed to rewarding messages about them. Therefore, learning explanations are supported by evidence showing that environmental influences can shape food preferences.

A further strength of learning explanations is that **research supports the idea that children learn food preferences from others**. **Wardle et al. (2005)** found that **parental fruit and vegetable consumption was a strong predictor of children's own intake**. This suggests that children may learn eating behaviours by **observing and copying their parents**, who act as important role models. If children regularly see their parents eating fruit and vegetables, they may be more likely to try these foods and develop a preference for them. Therefore, this supports the learning explanation because it shows that **social experience can influence children's food preferences**.

An additional strength of learning explanations is that they have led to **practical methods for improving children's eating behaviours**. Research suggests that **positive modelling and repeated exposure** are more effective than forcing children to eat certain foods. **Savage et al. (2007)** found that parents of children with healthier diets were more likely to use these strategies. This suggests that understanding learning processes can be applied to encourage healthier food preferences, showing the real-world value of learning explanations.

One limitation of learning explanations is that they may **underestimate the importance of individual differences in food preferences**. Although children may learn preferences through observing others, not all children respond to the same social influences in the same way. For example, **Robinson et al. (2001)** found that although parent and child eating behaviours were associated, the influence of parental modelling differed depending on factors such as **gender**, with **girls being more influenced** by parental behaviour than boys. This suggests that learning alone cannot fully explain food preferences because individuals differ in how strongly they are affected by environmental influences.

A further limitation is that some research into learned food preferences may lack **ecological validity**. Studies often measure food choices in **artificial settings** that may not reflect everyday eating behaviour. For example, children's responses to advertisements or experimental food choices **may not represent the complex decisions** they make when choosing food in real life, where factors such as **availability, culture and personal preferences** also influence behaviour. Therefore, learning explanations may have limited ability to explain food preferences outside controlled research settings.



Part 3: Neural and Hormonal Mechanisms

Eating behaviour is regulated by **homeostasis**, which maintains a stable internal environment. The body continuously monitors its energy levels and responds to changes in **glucose**, **ghrelin** and **leptin** to maintain energy balance. The **hypothalamus** is the main brain area responsible for regulating hunger and satiety.

1. Homeostasis

Homeostasis is the process by which the body maintains a stable internal environment. In eating behaviour, it regulates hunger by monitoring the body's energy levels.

When energy levels are low:

- **Glucose levels decrease**
- **Ghrelin levels increase**
- The **lateral hypothalamus (LH)** is activated
- This produces **feelings of hunger** and encourages eating

After eating:

- **Glucose levels increase**
- **Leptin levels increase**
- **Ghrelin levels decrease**
- The **ventromedial hypothalamus (VMH)** is activated
- This produces **feelings of fullness** and reduces eating

As energy levels fall again, glucose and leptin levels decrease and ghrelin levels increase, causing hunger to return. This allows the cycle to **repeat and maintain homeostasis**.



2. The Dual-Centre Model

The **Dual-Centre model** explains how the **hypothalamus regulates eating behaviour** through two opposing centres: the **LH**, which stimulates hunger and eating, and the **VMH**, which produces feelings of satiety and stops eating. The two centres work **antagonistically** to maintain energy balance.

Centre	Role	Supporting Research
Lateral hypothalamus (LH)	The feeding centre or “ on switch ”. Activation stimulates hunger and eating. The LH is associated with the release of neuropeptide Y (NPY) , a powerful appetite stimulant.	Damage to the LH causes animals to stop eating. Reynolds & Wickens (2000) found that injecting NPY into the hypothalamus caused rats to eat even when they were already satisfied.
Ventromedial hypothalamus (VMH)	The satiety centre or “ off switch ”. Activation inhibits the LH and produces feelings of fullness, reducing food intake.	Damage to the VMH causes overeating. Teitelbaum (1955) found that rats with VMH lesions overate, suggesting that the VMH is involved in controlling satiety.

Hormonal Mechanisms

1. Ghrelin

Ghrelin is a hormone released by the **stomach** when the body's energy stores are low. It stimulates the **arcuate nucleus**, which activates the LH and increases the release of **NPY**, producing feelings of hunger.

Ghrelin also helps regulate **when we feel hungry after eating**:

- Levels **rise before a meal**
- Levels **fall rapidly after eating**
- Levels gradually increase again as the body begins to need food

2. Leptin

Leptin is a hormone produced by **adipose (fat) tissue**. It acts as an **appetite suppressant**, signalling to the brain that sufficient energy has been stored.

- Higher **body fat** → **higher leptin levels**
- Leptin contributes to activation of the **VMH**
- It **inhibits NPY**, reducing hunger
- It increases **energy expenditure** through the sympathetic nervous system



Therefore, leptin helps the body regulate **appetite and long-term energy balance**.

AO3 - EVALUATION

One strength of neural and hormonal explanations of eating behaviour is that **research supports the role of leptin in regulating appetite and body weight**. **Heymsfield et al. (1999)** found that obese adults required doses of leptin **20–30 times higher than normal levels** to produce significant weight loss. This suggests that some individuals may develop **leptin resistance**, meaning leptin is less effective at suppressing appetite despite being present in the body. Therefore, this supports the idea that hormones play an important role in controlling eating behaviour and weight regulation.

A further strength of hormonal explanations is that **research supports the role of ghrelin in increasing hunger and food intake**. **Wren et al. (2001)** conducted a **double-blind study** where participants received either a ghrelin or saline infusion. They found that participants given ghrelin consumed significantly more food, with a **28% increase in food intake** compared with the placebo condition. This supports the role of ghrelin as an **appetite stimulant**, suggesting that hormones can directly influence eating behaviour. Furthermore, the double-blind design reduced the influence of **investigator effects and placebo effects**, increasing the validity of the findings.

One limitation of neural and hormonal explanations is that they **may underestimate the role of environmental factors in eating behaviour**. Although hormones and brain mechanisms influence hunger and satiety, eating is also affected by **social and cultural factors**, such as meal times, food availability and social expectations. For example, **Woods et al. (2004)** argued that social cues can override homeostatic mechanisms that regulate energy balance, as people often eat at set times even when they are not physically hungry. Therefore, biological explanations may be **reductionist** because they cannot fully explain the complexity of human eating behaviour.

A further limitation is that early neural explanations may have **oversimplified the control of eating behaviour by focusing on specific brain areas, such as the hypothalamus**. For example, damage to the **lateral hypothalamus (LH)** was originally thought to only affect eating behaviour, but research has shown it can also influence other behaviours, such as thirst. **Sakurai et al. (1998)** suggested that eating behaviour is controlled by wider **neural circuits** rather than a single brain region, while **Valassi et al. (2008)** highlighted the role of additional hormones such as **CCK** and neurotransmitters including **serotonin and dopamine**. Therefore, neural explanations may provide an incomplete explanation because eating behaviour involves multiple interacting biological systems.



Part 4: Biological Explanations for Anorexia Nervosa

Anorexia nervosa (AN) is an eating disorder characterised by **very low body weight**, an intense **fear of gaining weight** and a **distorted perception of body weight or shape**.

The **biological approach** suggests that AN may be influenced by **genetic and neural factors**, meaning that biological differences may make some individuals more vulnerable to developing the disorder.

1. Genetic Explanations

Research suggests that **AN can run in families**, indicating a possible genetic vulnerability.

- **Strober et al. (2000)** found that first-degree relatives of individuals with AN were around **10 times more likely** to develop the disorder than relatives of unaffected individuals.
- **Holland et al. (1988)** found a **56% concordance rate** for AN in **monozygotic (MZ) twins**, compared with only **5% in dizygotic (DZ) twins**. As MZ twins share 100% of their genes while DZ twins share around 50%, this suggests that **genetic factors may contribute to AN**.
- **Wade et al. (2000)** studied over **2,163 female MZ and DZ twins** and estimated the **heritability of AN at 58%**. They also suggested that genetic factors may contribute to the link between AN and **major depression**.

However, twin studies cannot completely separate **nature and nurture**, because MZ twins share both a greater proportion of their genes and similar environments.

Adoption studies provide a way of separating these influences. **Klump et al. (2009)** studied **123 adopted sibling pairs and 56 biological sibling pairs**, finding heritability estimates of **59–82%** for different aspects of disordered eating. This provides further evidence that genetic factors contribute to vulnerability to AN.



2. Neural Explanations

Neural factor	Role in AN	Supporting research
Serotonin	Serotonin regulates mood, anxiety, appetite and obsessive behaviour . Abnormal serotonin functioning may contribute to appetite suppression, anxiety and obsessive thinking in AN. People with AN have also been found to have low levels of 5-HIAA , suggesting abnormal serotonin functioning.	Bailer et al. (2011) found higher serotonin activity in women recovering from restrictive AN. Those with the highest serotonin activity also showed the highest anxiety levels , suggesting disrupted serotonin may contribute to AN symptoms.
Dopamine	Dopamine is involved in the brain's reward and pleasure system . Abnormal dopamine functioning may explain why food is experienced as less rewarding by people with AN. Restricting food intake may also reduce anxiety caused by increased dopamine activity.	Kaye et al. (1991) found overactivity of dopamine receptors in the basal ganglia of women recovering from AN. Bailer et al. (2012) found that increasing dopamine using amphetamines produced anxiety in women recovering from AN, but euphoria in healthy controls.
Limbic System	The limbic system regulates emotion and emotional processing . Dysfunction may contribute to the pathological thoughts and behaviours associated with AN.	Lipsman et al. (2012) suggested that dysfunction within the limbic system may underlie emotional processing difficulties seen in AN, contributing to disordered eating behaviours.

AO3 - EVALUATION

One strength of genetic explanations of anorexia nervosa is that **research suggests there is a genetic vulnerability involved in the development of AN**. Twin studies have found higher concordance rates for anorexia nervosa in **monozygotic (MZ) twins** compared with dizygotic (DZ) twins, suggesting that shared genes contribute to the risk of developing the disorder. This supports the genetic explanation because MZ twins share **100% of their genes**, meaning the greater similarity between MZ twins suggests genetic factors influence the development of AN. Therefore, research provides support for the idea that some individuals may inherit a vulnerability towards anorexia nervosa.



Another strength of neural explanations is that **research supports the role of dopamine abnormalities in anorexia nervosa**. **Kaye et al. (1999)** found that recovering AN patients showed reduced levels of **homovanillic acid (HVA)**, a dopamine metabolite, suggesting that abnormal dopamine functioning is associated with the disorder. This study is particularly useful because recovering patients were less affected by **malnutrition**, meaning the dopamine abnormalities were less likely to simply be a consequence of weight loss. Therefore, research supports the idea that neural factors may contribute to the development of AN.

A further strength of biological explanations is that they may have **important real-world applications by reducing stigma and improving treatments for anorexia nervosa**.

Biological explanations suggest that AN is not simply a choice or the individual's fault, which may reduce blame towards sufferers. Furthermore, research into neural abnormalities has contributed to possible treatments such as **deep brain stimulation (DBS)**. **Lipsman et al. (2013)** found that DBS targeting the **nucleus accumbens** resulted in improvements in BMI, with **6 out of 16 patients achieving a normal BMI**. Therefore, biological explanations have practical value because they can improve understanding of AN and contribute to new treatment approaches.

One limitation of genetic explanations is that the **heritability of anorexia nervosa is difficult to establish**. **Fairburn et al. (1999)** reviewed research into genetic influences on AN and concluded that estimates of heritability were inconsistent and not always replicated. Furthermore, many twin studies may violate the **equal environments assumption**, which assumes MZ and DZ twins experience similar environmental influences. However, **Loehlin and Nichols (1976)** found that MZ twins tend to be treated more similarly than DZ twins, meaning environmental factors may contribute to the higher concordance rates seen in MZ twins. Therefore, genetic explanations may overestimate the influence of genes because they cannot fully separate genetic and environmental effects.

A further limitation of genetic explanations is that they may **underestimate the role of social and cultural influences in anorexia nervosa**. For example, the media often promotes an **ultra-thin body ideal**, which may increase body dissatisfaction and encourage restrictive eating behaviours. **Vaughn and Fouts** found that adolescent girls who read more fashion magazines showed increased anorexia symptoms over a **16-month period**. This suggests that social learning processes, such as **identification and vicarious reinforcement**, may contribute to AN development. Therefore, genetic explanations alone may be incomplete because anorexia nervosa is likely caused by an interaction between **biological vulnerability and environmental factors**.

Another limitation of neural explanations is that they may be **too simplistic by focusing on individual neurotransmitters, such as serotonin, rather than considering the interaction between different systems**. For example, **Ferguson et al. (1999)** found that **SSRIs**, which increase serotonin activity, were ineffective in improving anorexia nervosa symptoms compared with patients who did not receive SSRIs. This suggests that serotonin abnormalities alone cannot explain AN. Furthermore, serotonin interacts with other neurotransmitters, including **dopamine, noradrenaline and GABA**, meaning anorexia nervosa is unlikely to result from a single neurotransmitter imbalance. Therefore, neural explanations may provide only a partial explanation of anorexia nervosa.



Part 5: Psychological Explanations of Anorexia; Family Systems Theory

Family systems theory is a **psychodynamic explanation** which argues that anorexia nervosa (AN) should be understood in the context of the **family as a whole**, rather than focusing only on the individual.

Minuchin et al. (1978) suggested that a particular type of dysfunctional family, known as a “**psychosomatic family**”, may contribute to the development of AN. These families are characterised by **enmeshment, overprotectiveness/control, rigidity and avoidance of conflict**. The individual's eating behaviour may therefore become a way of gaining **autonomy and control** within the family.

Characteristic	Description
Enmeshment	Family members are overly involved in each other's lives, with few clear boundaries or little privacy. Parents may be highly involved in their child's decisions and emotions, making it difficult for the child to develop an independent identity . The individual may therefore use food restriction to establish a sense of separation from the family.
Overprotectiveness / Control	Parents show a high level of concern and control over their child's welfare and decisions. This can limit the child's independence and autonomy . Refusing to eat may become a way for the individual to regain control over their own life. Palazzoli (1974) particularly highlighted the controlling and intrusive role of the mother.
Rigidity	The family is inflexible and struggles to adapt to changes or stressful situations. Attempts by the child to become more independent may be discouraged or rejected. Bruch (1978) suggested that food restriction can therefore become a way for the individual to assert independence and control .
Conflict avoidance	Conflict exists within the family but is not openly resolved . Instead, family members may maintain a façade of togetherness and avoid addressing problems. Latzer et al. (2002) found that families of patients with eating disorders were less cohesive, expressive and supportive of personal growth than control families.

Conflict-related Patterns

Minuchin (1978) also identified three patterns that may occur when conflict exists within the family:

- **Triangulation** – two family members involve a **third person** in their conflict, either by bringing them into the disagreement or rejecting them.
- **Parent-child coalition** – the child forms a **stronger relationship with one parent**



against the other. One parent may become excessively concerned with the child while the other withdraws.

- **Detouring** – parents redirect attention away from their own conflict and towards the **child**, making the child's problems the focus of the family and effectively making them a **scapegoat**.

Overall, Family Systems Theory suggests that AN can develop within a family where **enmeshment, control, rigidity and unresolved conflict** prevent the individual from developing sufficient **autonomy and independence**.

AO3 - EVALUATION

One strength of family systems theory is that there is **research support for the role of enmeshment in anorexia nervosa**. **Manzi et al. (2009)** found that family cohesion was linked to positive psychological outcomes, whereas **enmeshment** was associated with poorer wellbeing. This supports the idea that blurred family boundaries and excessive control may contribute to AN, as starvation may provide an individual with a sense of **control and autonomy** when they struggle to differentiate themselves from family members. Further support comes from **Latzer and Gaber (1998)**, who found that families with a member with AN experienced greater difficulty resolving conflicts compared with control families. Therefore, dysfunctional family patterns may contribute to the development and maintenance of anorexia nervosa.

A further strength of family systems theory is that it has led to **effective real-world treatments, particularly family-based therapy**. Family interventions aim to reduce harmful family patterns by improving communication, reducing conflict and increasing support for the individual with AN. **Shadish and Baldwin (2003)** found that over **71% of families** showed improved family functioning following family interventions. This suggests that addressing family relationships can improve outcomes for individuals with AN. Therefore, family systems theory has practical value because it has contributed to treatments that can support recovery.

One limitation of family systems theory is that the **psychosomatic family model may be an oversimplified explanation of anorexia nervosa**. Research attempting to identify specific family characteristics linked to AN has produced inconsistent findings. For example, **Kog and Vandereycken (1989)** failed to find support for the specific characteristics proposed by the psychosomatic model and instead suggested that family difficulties are better explained through factors such as **family boundaries, adaptability and conflict management**. This suggests that families affected by AN are a diverse group and may not all share the same dysfunctional patterns. Therefore, family systems theory may only explain AN in some families rather than providing a universal explanation.

A further limitation is that family systems theory may create **gender bias and parent-blaming**. **Gremillion (2003)** argued that family systems explanations often focus heavily on **mother-daughter relationships**, with enmeshment frequently being viewed as a maternal issue. This may lead to **stereotypes of the “overprotective” or “controlling mother”**, while overlooking the contribution of fathers and wider family dynamics. Therefore, family systems theory may unfairly place responsibility on parents and fail to consider the complex range of factors involved in the development of anorexia nervosa.



Part 6: Psychological Explanations of Anorexia; Social Learning Theory

Social Learning Theory (SLT) suggests that anorexia nervosa can develop through **observing and learning from other people**. Individuals may learn attitudes and behaviours relating to **food, dieting, weight and body image** by watching others and then **imitating** them.

Modelling occurs when an individual **observes and imitates a role model's behaviour**. People are more likely to imitate a role model when they:

- **Identify** with them.
- See them as **attractive or successful**.
- See their behaviour being **rewarded**.

In Anorexia Nervosa, important role models can include **family, friends and media figures**.

Role model	What may be observed	What may be learned	Supporting evidence
Family	Parents dieting , restricting food, talking negatively about their weight/appearance , or placing importance on being thin .	The child may learn that thinness is desirable and that restricting food is an appropriate way to control weight.	Hill et al. (1989) found similarities between mothers' and daughters' restrained eating and dieting behaviours , supporting parental modelling.
Peers	Friends dieting , discussing their weight/appearance , or restricting their food intake.	Young people may copy these behaviours, particularly when dieting is viewed positively. This may create pressure to conform to the group's body ideals to fit in and gain social acceptance .	Keel et al. (2012) found that female college students whose roommates dieted were more likely to develop a drive for thinness and disordered eating behaviours .



Media	TV, films, magazines and social media may present thin bodies as attractive, successful, popular and desirable .	Individuals may compare themselves with idealised body images and begin dieting or restricting food to try to achieve the ideal. Those with low self-esteem may be particularly vulnerable to negative comparisons.	Dittmar et al. (2006) found that young girls who viewed Barbie dolls experienced greater body dissatisfaction than girls who viewed dolls with a more realistic body shape.
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Once a behaviour has been learned, **reinforcement** can make it more likely to continue.

- **Vicarious reinforcement** - Seeing another person being **praised or admired for being thin** can make dieting and weight loss appear **rewarding**, encouraging the individual to imitate the behaviour.
- **Direct reinforcement** - The individual may receive **compliments after losing weight** or experience a sense of **achievement and control** from restricting food. These positive consequences can encourage them to **continue restricting their intake**.

Overall: According to **Social Learning Theory**, observing role models who promote thinness may lead individuals to **imitate dieting and restrictive eating behaviours**, particularly when these behaviours appear to be **rewarded or socially valued**.

AO3 - EVALUATION

One strength of SLT explanations is that **research supports the role of media influences in the development of anorexia nervosa**. **Becker et al. (2011)** investigated eating attitudes in Fijian children following the introduction of television in 1995. They found that after exposure to TV, children reported an increased desire to **lose weight**, with stronger effects among those who had more friends exposed to TV, videos or DVDs. This suggests that media exposure may influence eating attitudes through **modelling** and **social reinforcement**, as individuals learn and discuss unrealistic body ideals. Therefore, SLT provides a useful explanation for how cultural influences may contribute to AN.

A further strength of SLT is that research suggests **peer influence can affect body ideals and eating behaviours**. **Costa-Font and Jofre-Bonet (2009)** found that women with peers who had a **higher or average BMI** were less likely to develop anorexia nervosa, suggesting that peer groups can influence attitudes towards body size. They also found that **positive comments from peers** about an individual's body reduced the likelihood of extreme thinness. This supports SLT because individuals may learn and adapt their eating behaviours through **social feedback and approval**. Therefore, peer interactions may play an important role in the development of AN.



One strength of SLT explanations is that they can **explain gender differences in eating disorders**. **Harrison and Cantor (1997)** found that **media use predicted drive for thinness, body dissatisfaction and disordered eating in women**, whereas in men it was more strongly associated with **dieting and muscular ideals**. This suggests that individuals learn different body standards depending on the **role models** they are exposed to. Therefore, SLT can help explain why females are more likely to develop disorders involving **thinness**, whereas males may be more vulnerable to disorders involving **muscularity**, such as **bigorexia**.

One limitation of SLT is that children **do not always simply imitate the behaviours and attitudes of role models**. **Pike and Rodin (1991)** found **no evidence** that daughters adopted their mothers' weight concerns, while **Ogden and Steward (2000)** found that although mothers and daughters had similar body weights, there was **no association between restrained eating**. This suggests that exposure to a role model does not automatically lead to the same eating behaviours. Therefore, SLT may overestimate the role of **modelling** and cannot fully explain why some individuals develop AN while others exposed to similar influences do not.

A further limitation of SLT is that it may **underestimate the role of biological factors in anorexia nervosa**. Research suggests that AN has a **genetic and neural component**, meaning some individuals may have a **biological vulnerability** that increases their risk of developing the disorder. For example, **Kaye et al. (1999)** found **dopamine abnormalities** in individuals recovering from AN, suggesting biological factors contribute to the disorder. Therefore, SLT may provide an incomplete explanation because **learning experiences alone are unlikely to cause AN**.



Part 7: Psychological Explanations of Anorexia; Cognitive Theory

The **cognitive approach** suggests that Anorexia Nervosa is influenced by the way individuals **process information and interpret themselves, their bodies and food**. This can lead to **cognitive distortions** and **irrational beliefs** that maintain restrictive eating behaviour.

1. Cognitive Distortions

Cognitive distortions are biased patterns of thinking where a person's perception does not match **reality**. In AN, individuals may **overestimate their body size or weight**, despite being underweight.

For example, an individual may compare themselves with **thin models** and develop the belief that they are overweight. This can lead to **anxiety, self-disgust and further attempts to lose weight**.

Williamson et al. (1993) used the **Body Image Assessment** and found that **37 women with AN** were significantly more likely to **overestimate their body size** than **95 controls without AN**. This supports the role of distorted body perception in AN.

Individuals with AN may also develop **irrational beliefs** about food, weight and self-control.

These can include:

- **Absolutist thinking** – "I ate an apple, so I have no self-control."
- **Catastrophising** – "I gained 0.5 kg, so I will become obese."
- **Conditional beliefs** – "I must be thin for other people to like me."

These beliefs may be linked to **perfectionism**, where individuals set extremely high standards for themselves. Even after reaching a weight goal, they may set an even lower target, creating a **never-ending cycle of perceived failure**.

2. Cognitive Behaviour Model

Garner and Bemis (1982) suggested that people who develop AN may have **perfectionistic and self-doubting personalities**. These characteristics can make individuals particularly vulnerable to **cultural pressures to be thin**.

They may develop beliefs that being thin makes them **more attractive, successful or worthy**, leading to **dieting and food restriction**.



Restriction can then be reinforced because the individual may experience:

- **Compliments from others**
- A sense of **achievement**
- A sense of **control**

At the same time, **eating and gaining weight become associated with anxiety**, increasing fear of food and weight gain.

Continued restriction may also lead to **social withdrawal**, reducing opportunities for others to **challenge distorted beliefs**.

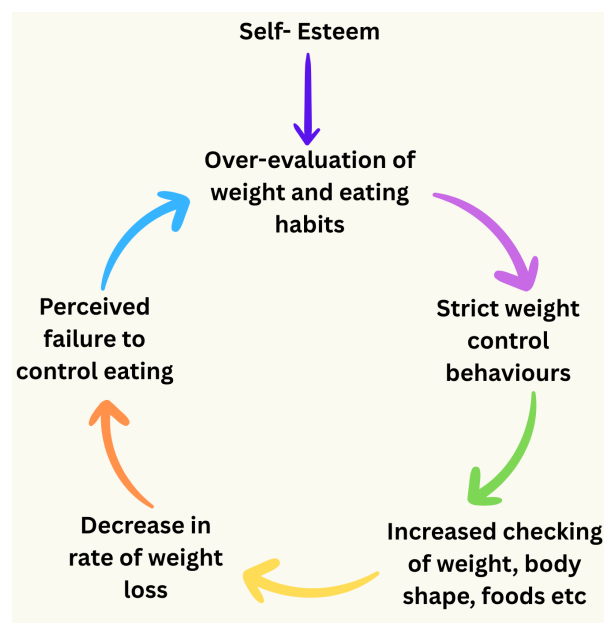
3. Fairburn's Transdiagnostic Model

Fairburn et al. (2003) suggested that eating disorders share a **common underlying cognitive process**, rather than each disorder having completely separate causes.

The central feature is an **overvaluation of weight and appearance**. In AN, an individual's **self-esteem becomes strongly dependent on their weight, shape and ability to control their eating**, rather than other achievements or aspects of their identity.

This can create a cycle:

1. **Restriction increases self-control** → the individual feels successful and their **self-esteem increases**.
2. **Starvation produces physical and psychological changes** → these may be interpreted as evidence that they are **losing control**, increasing the need to restrict further.
3. The individual becomes highly focused on **monitoring their weight and appearance** → perceived lack of progress leads to **even greater food restriction**.



Treasure and Schmidt (2013) also highlighted **reduced set-shifting** as a factor that may help maintain AN. This refers to difficulty **changing one's thoughts or behaviour when circumstances change**, which may make it harder for someone with AN to break away from rigid patterns of eating and thinking.

Cognitive explanations therefore suggest that AN is maintained by **distorted body perception, irrational beliefs, perfectionism and an overvaluation of weight and self-control**. These thought patterns can create a cycle in which **food restriction temporarily increases self-esteem**, reinforcing the behaviour and making the disorder difficult to break.

AO3 - EVALUATION

One strength of cognitive explanations is that **research supports the role of cognitive factors in anorexia nervosa**. **Lang et al. (2015)** compared 41 children with AN to 43 healthy controls and found no difference in IQ. However, the AN group showed greater **cognitive inflexibility** and inefficient processing styles, meaning they struggled to adapt their thinking when presented with new information. This supports **Treasure and Schmidt's interpersonal maintenance model**, which suggests that cognitive inflexibility contributes to the maintenance of AN because individuals may struggle to overcome rigid beliefs about food, weight and body image. Therefore, research supports the idea that dysfunctional thinking patterns contribute to anorexia nervosa.

A further strength of cognitive explanations is that they have **led to effective treatments**.

Fairburn et al. (2003) compared **CBT-E** with interpersonal therapy (IPT) in 130 patients with eating disorders. After 20 weeks, around **two-thirds of the CBT-E group** met criteria for remission compared with around **one-third of the IPT group**. Further support comes from **Grave et al. (2013)**, who found that after 40 weeks of CBT-E, 99 adult AN patients showed a significant increase in weight, with an average increase of **7.47kg**. This suggests that targeting dysfunctional thoughts and beliefs can improve symptoms. Therefore, cognitive explanations have practical value because they have contributed to effective treatments for AN.

One limitation of cognitive explanations is that they may **rely too heavily on self-report measures**. **Vandereycken and Vanderlinden (1983)** found that individuals with **anorexia nervosa** may **deny or underestimate their symptoms**, suggesting that self-report measures may not always provide an accurate representation of their thoughts and beliefs. This is a problem because cognitive explanations assume that thoughts about weight, food and body image can be **accessed and reported accurately**. Therefore, cognitive explanations may have **reduced validity** because researchers may not gain a true understanding of the cognitive processes involved in AN.

A further limitation of cognitive explanations is that they may **underestimate the role of biological factors in anorexia nervosa**. Research suggests that AN has a biological basis, including **genetic vulnerability and neurotransmitter abnormalities**, meaning some individuals may be more predisposed to developing the disorder. For example, **Kaye et al. (1999)** found **abnormalities in dopamine functioning** in individuals recovering from AN, suggesting that biological factors contribute to symptoms. Therefore, cognitive explanations may provide an incomplete explanation because they do not fully account for the interaction between biological vulnerability and cognitive processes.



Part 8: Biological Explanations for Obesity

The **biological approach** suggests that obesity is influenced by **genetic, neural and hormonal factors**. These factors can affect appetite, hunger, satiety and the way the brain responds to food.

Genetic explanations suggest that some people may inherit a **greater vulnerability to obesity** because genes can influence factors such as **appetite, metabolism and fat storage**.

1. Twin and Adoption Studies

Research comparing relatives with different levels of genetic similarity suggests that **body weight and BMI have a genetic component**.

- **Maes et al. (1997)** conducted a meta-analysis of **75,000 individuals** and found BMI heritability estimates of **74% for MZ twins** compared with **32% for DZ twins**.
- MZ twins who were **raised apart** were still more similar in BMI than DZ twins, suggesting that this similarity cannot be explained entirely by their shared environment.
- **Stunkard et al. (1986)** studied **540 adult adoptees**, their biological parents and adoptive parents. The adoptees' weight was more strongly related to their **biological parents' weight** than their adoptive parents' weight, supporting a genetic influence.

These findings suggest that **genes contribute to individual differences in body weight**, although they do not mean that obesity is determined entirely by genetics.

2. The Thrifty Gene Hypothesis

Neel (1962) proposed the **Thrifty Gene Hypothesis** as an evolutionary explanation for why some people may be genetically more vulnerable to **obesity**.

The hypothesis suggests that genes which promote **efficient energy storage** may have been advantageous when food supplies were **unpredictable**. Being able to store energy as fat during plentiful periods meant these energy reserves could be used when food became scarce, increasing **survival and reproductive success**.

Environment of Evolutionary Adaptedness (EEA)	Modern environment
Food supply was often unpredictable	High-calorie food is widely available
Periods of food shortage were common.	Famine is relatively rare.
Storing excess energy as fat could improve survival during shortages.	Efficient energy storage can lead to excess fat accumulation .
Thrifty genes = advantageous	Thrifty genes = potentially maladaptive, increasing obesity risk.



Speakman (2008) challenged this explanation. He argued that if thrifty genes were a major cause of obesity, **most people should be obese**, because humans share common ancestors who experienced similar periods of food shortage. However, obesity rates vary considerably between **populations and cultures**, suggesting that **environmental factors must also contribute**.

3. Neural and Hormonal Explanations

The **hypothalamus** plays an important role in regulating **hunger and satiety**. The **arcuate nucleus**, within the hypothalamus, monitors energy levels and helps regulate appetite. When energy levels are low, it can stimulate the **lateral hypothalamus** and increase the release of **neuropeptide Y (NPY)**, which stimulates hunger.

Mechanism	Role in Obesity	Supporting research
Leptin	Leptin is produced by fat cells and acts as an appetite suppressant . It signals to the brain that sufficient energy has been stored and helps reduce food intake. A lack of leptin or disruption of leptin signalling can therefore lead to overeating and obesity.	Licinio et al. (2004) found that individuals unable to produce leptin experienced extreme obesity , while leptin replacement produced substantial weight loss. Bates & Myers (2009) also linked disrupted leptin signalling in the hypothalamus to obesity.
Serotonin	Serotonin is involved in appetite and satiety . Low serotonin activity may reduce activation of the VMH , reducing feelings of fullness and increasing food intake, particularly of calorie-dense foods.	Low levels of serotonin and its metabolite 5-HIAA have been associated with obesity, suggesting abnormal serotonin functioning may contribute to appetite dysregulation.
Dopamine	Dopamine is involved in the brain's reward and pleasure system . Lower dopamine activity or fewer D2 receptors may mean that eating produces less pleasure, potentially encouraging overeating to increase the reward response.	Wang et al. (2001) found lower D2 receptor availability in the striatum of people with obesity, supporting a link between dopamine functioning and overeating



AO3 - EVALUATION

One strength of biological explanations is that **research supports the role of leptin in regulating appetite and body weight**. **Montague et al. (1997)** reported the case of two severely obese cousins who had very low levels of leptin, suggesting that leptin deficiency may contribute to obesity. Further support comes from **Gibson et al. (2007)**, who found that **leptin replacement treatment improved appetite regulation, metabolism and weight loss** after four years. Therefore, research supports the idea that **biological abnormalities in appetite regulation can contribute to obesity**.

A further strength of biological explanations is that they have led to **useful treatments for obesity**. Understanding the role of **biological mechanisms** means obesity can be viewed as a disorder influenced by factors **outside an individual's control**, which may reduce **stigma and blame** towards obese individuals. This research has also contributed to the development of **drug treatments** such as **orlistat and sibutramine**, which target biological processes involved in weight regulation. **Jain et al. (2005)** found that these treatments produced an **average weight loss of 3–5kg**, suggesting that targeting biological mechanisms can help individuals manage obesity. Therefore, biological explanations have **real-world value** because they can improve attitudes towards obesity and contribute to the development of **effective interventions**.

One limitation of biological explanations is that **genetic factors cannot explain the rapid increase in obesity rates**. **HSE / NHS Digital Report (2015)** found that **obesity rates in UK males increased from 13% in 1993 to 26% in 2013**, despite the **human gene pool remaining relatively unchanged**. This suggests that **environmental factors**, such as increased availability of high-calorie foods and reduced physical activity, must also contribute to obesity. Therefore, **biological explanations alone provide an incomplete explanation** because they fail to account for changes in lifestyle.

A further limitation of evolutionary explanations of obesity is that the **thrifty gene hypothesis may not be supported**. **Speakman (2008)** argued that if **thrifty genes** were responsible for obesity, a much larger proportion of the population should be obese because these genes would be widespread. Further evidence comes from **Ng et al. (2014)**, who found that **obesity rates increased most dramatically in developing countries**, where there have been rapid changes in **diet and lifestyle**. Therefore, this suggests that **environmental factors may be a stronger explanation for obesity than evolutionary adaptations**.



Part 9: Psychological Explanations for Obesity

Psychological explanations suggest that obesity can be influenced by **thoughts, attitudes and behaviours surrounding food**, rather than purely biological factors.

1. Restraint Theory

Herman and Polivy (1975) proposed **Restraint Theory**, which suggests that **consciously restricting food intake can actually increase the likelihood of overeating**.

Restrained eaters use **cognitive rules** to control their eating rather than relying on natural hunger and fullness signals.

They may:

- Label foods as “**good**” or “**bad**”
- Set rules about **what and how much** they can eat
- Try to ignore **physiological hunger**

This means that eating becomes controlled by **thoughts and rules**, rather than the body's natural signals.

There are two main forms of restraint:

Rigid Restraint	Flexible Restraint
All-or-nothing dieting with strict food rules.	A less strict approach that allows some flexibility in food choices.
More likely to lead to disinhibition when the diet is broken.	Less likely to result in extreme overeating.

Provencher et al. (2003) found that rigid restraint was associated with **higher body fat, waist circumference and BMI**, whereas flexible restraint was associated with lower levels.



2. Disinhibition

This can happen when a restrained eater believes they have **broken their diet**. Instead of returning to their normal eating rules, they may think that they have already failed and continue eating.

This can lead to **overeating or binge eating**.

Bond et al. (1998) identified three types of disinhibition:

- **Habitual** → overeating as part of everyday habits.
- **Emotional** → overeating in response to emotions such as stress.
- **Situational** → overeating because of environmental or social situations.

3. The Boundary Model

Herman and Polivy (1984) developed the **Boundary Model** to explain how restraint can lead to overeating.

For an unrestrained eater, eating is mainly controlled by **physiological signals**:

Hunger → eating → fullness → stop eating

For a restrained eater, eating is controlled more by **cognitive rules** and a self-imposed limit on how much they should eat.

Hunger → eating → self-imposed diet boundary → fullness

The **Zone of Biological Indifference (ZBI)** is the point where a person is neither hungry nor full. Restrained eaters have a **wider ZBI**, meaning their eating is less closely controlled by physiological hunger and fullness.

If they cross their **self-imposed dietary boundary**, the “**what the hell effect**” may occur. They believe they have already failed their diet, so they give up their restraint and continue eating **even after they are physically full**.



AO3 - EVALUATION

One strength of restraint theory is that **research supports the idea that attempts to restrict eating can paradoxically lead to overeating**. **Wardle and Beales (1988)** randomly allocated **27 obese women** to a **diet group, exercise group or no-treatment group** for seven weeks. They found that women in the diet condition **consumed more food** than those in the other groups, supporting the idea that strict dietary restraint can lead to a **loss of cognitive control**. Therefore, this provides support for the role of **dysfunctional eating patterns** in the development of obesity.

One practical application of psychological explanations is the use of **weight-management programmes to reduce disinhibition and rigid restraint**. These programmes can help individuals identify triggers for overeating, such as emotional or situational cues, and encourage **flexible restraint** rather than strict “all-or-nothing” dieting. **Provencher et al. (2003)** found that rigid restraint was associated with higher BMI and body fat, whereas flexible restraint was associated with lower levels. This suggests that applying psychological explanations to weight-management programmes could help individuals develop healthier eating behaviours and **reduce obesity**.

A further strength of psychological explanations is that **research supports the role of disinhibition in overeating**. **Ogden and Wardle (1990)** found that restrained eaters experienced increased feelings of **rebelliousness and defiance** after eating high-calorie foods, suggesting that overeating may occur as a response to breaking self-imposed dietary rules. Further support comes from **Loro and Orleans (1981)**, who found that obese binge eaters often reported binge eating as a way of expressing **resentment**. Therefore, psychological explanations provide a useful explanation for why some individuals **lose control over eating despite attempting to restrict their intake**.

One limitation of psychological explanations is that **disinhibition may not be a universal explanation for obesity**. **Atlas et al. (2000)** found that **restraint and disinhibition scores were lower in African-American students compared with white college students**. Similar research has also suggested that **men show lower levels of disinhibition than women**. This suggests that the influence of **cognitive control and restrained eating varies between individuals and cultures**. Therefore, psychological explanations may only explain obesity in certain groups rather than providing a complete explanation.

A further limitation of psychological explanations is that **they may underestimate the role of biological factors in obesity**. Research suggests that appetite and weight regulation are influenced by biological mechanisms, such as **leptin**, which controls hunger and satiety. For example, **Montague et al. (1997)** found that **two severely obese individuals had very low levels of leptin**, suggesting biological vulnerability can contribute to obesity. Therefore, psychological explanations may provide an incomplete explanation because **eating behaviour is influenced by both psychological and biological factors**.

