AQA Psychology A-level

Option 2: Eating Behaviour

Notes

Part 1 - The evolutionary explanation for food preference

Key Words

Evolutionary explanations - Focuses on the adaptive nature of behaviour. Modern behaviours are thought to have evolved because they solved challenges faced by our ancestors.

Food preferences - Refers to the way in which people choose from among available food based on biological and learned perceptions such as taste, health characteristics and value.

Taste aversion - A learned response to eating spoilt or poisonous food which results in the animal avoiding that food in the future.

Neophobia - An extreme dislike and avoidance of anything that is new or unfamiliar. This reduces the risk of unpleasant or dangerous experiences, which posed an evolutionary threat.

- **Early diets -** Early humans were hunters whose diets consisted of animals and plants from the natural environment.
- Preference for fatty foods would have been adaptive for early humans as in the EEA, energy sources were vital in order to stay alive and find the next meal. Fat contains twice the number of calories of the equivalent carbohydrate or protein, as well as increasing the palatability or 'appeal' of food, making it desirable by increasing the likelihood of the individual surviving and reproducing.
- Preference for meat comes from the fact that meat has lots of nutrients - something you may not have gained from a vegetarian diet e.g. high levels of iron present in red meat.
- Preference for salt may be innate, as evidence shows that babies who had been breast-fed (containing low levels of salt) preferring salted over non-salted cereals (Harris et al, 1990). Since these babies, at age 4 months, had not previously been exposed to high concentrations of salt, this implies that the preference for salt is innate.
- Preference for sweet food as the taste of sweetness is associated with a large amount of sugar and a high concentration of it to give you energy e.g. fruit. Fruit also provides the vitamins and minerals that are necessary for growth and bodily functions e.g potassium to maintain healthy blood pressure and fibre to reduce cholesterol.

- Fructose, which is found in many fruits, is particularly important in evolutionary terms, because it provides an easy and rapid supply of energy. Such a preference has been demonstrated by Steiner (1977), who found that children could even differentiate between different types of sugars. Menella (2014) also found that children who preferred sweet solutions over salty tended to be tall for their age. This may be linked to how in the EEA, those who sought out more calories were more likely to grow and survive.

Taste Aversion - It is a learned response to eating toxic, or spoilt food. When taste aversion takes place the animal avoids eating the food that made it ill, where a bitter or sour taste usually characterises toxic food.

- Taste aversion was proven by Garcia et al (1955) who studied rats who'd been made ill through radiation after eating saccharin, and the rats developed an aversion to the saccharin. This also demonstrates Seligman's theory of learned preparedness, where individuals are more likely to fear 'prepared' stimuli (e.g. fire, deep water etc) which had posed a threat to their evolutionary ancestors, as opposed to 'non-prepared' stimuli. The Garcia et al. study shows that poison is an example of a 'prepared' stimuli and rats can therefore be classically conditioned to avoid it, thus increasing their chances of survival.

Neophobia - Food neophobia is a reluctance to consume new or unusual food. It is a naturally occurring reaction that protects animals from being poisoned. This response is a critical survival strategy for diets that are restricted.

- Ratcliffe et al (2003) suggested that animals with constricted diets, such as koalas, tend not to have neophobia. However, species that have broad diets do display signs of food neophobia. This supports the idea that neophobia is an adaptive behaviour which increases the likelihood of survival by reducing the chance of toxicity or poisoning.

- In humans, neophobia is the reluctance to consume new or unusual foods. Martins et al suggests that neophobia is stronger in animal products than non animal products as animals products pose a greater risk of illness e.g. mass vaccination of animals and disease which can be transmitted from animals to humans (such as Ebola and Salmonellosis).

- Neophobia can also be maladaptive - it can lead to people restricting their diets and they may not get all the nutrients they need or they may not get all the benefits they would do. However, Birch et al. demonstrated that neophobia is strongest between the ages of 2 and 6 years old, where infants are increasingly eating foreign foods without their parents guidance. Neophobia then becomes weaker after this period, to encourage the consumption of other unknown foods which may contain high levels of minerals, vitamins, fats etc.

- Perry et al (2015) found that neophobia is associated with poorer diet quality in children. However, this is not always the case as research has shown that repeated taste exposure increases preference for unfamiliar food (Birch et al). This change may only be temporary and the underlying neophobic tendency is likely to persist.

Evaluation

+ Research supporting the innate predisposition towards high-energy, sugary foods = Babies show a preference for sweet food the first time they have it by having a slight smile, as demonstrated by Jacob Steiner (1977), which is an innate response. This supports the fact that sweet food would have been necessary for survival, due to the high fructose and vitamin content, and there is an evolved preference for it. (Grill et Norgren)

+ Research support for the evolutionary basis of food preference = Research has suggested that there is a link between food preference and stress. Studies reviewed by Torres et al (2008) have found that during periods of stress, most adults have an inclination towards high-fat foods. This may be due to high levels of fat being able to sustain the fight or flight response present during times of stress, and so being more likely to survive due to this prolonged response (compared to those who had chosen a low-fat diet). This link between high-fat foods and an increased chance of survival supports the innate nature and evolutionary basis for food preference.

+ Practical applications of an improved understanding of taste aversion = Taste aversion has helped us understand why food avoidance can occur in chemotherapy. Bernstein and Webster (1980) gave patients a novel tasting ice cream before chemotherapy. When the patients are given a novel food along with a familiar food the patients develop an aversion to the novel food rather than the familiar so it acts as a “scapegoat” to prevent items in the regular diet decreasing in preference and stops aversions to them. This can be explained in classical conditioning terms, where the novel food acts as the neutral stimulus, the radiation is the unconditioned stimulus which together, produces the conditioned response of avoidance to the conditioned stimulus (the novel food).

+ Neophobia is thought to be genetic = Knaapila et al (2007) studied a sample of 468 pairs of adult female twins and measured food neophobia using the Food Neophobia Scale questionnaire The heritability estimate for this sample was 67% suggesting that 2/3 of food neophobia characteristics are genetically determined. This supports the evolutionary explanation that neophobia evolved from early humans because it protected them from eating harmful foods, but still allowed for the discovery of other foreign foods which may’ve offered greater levels of vitamins, minerals and fats.

Part 2 - The role of learning in food preference

Key Words

Learning - The acquisition of knowledge, skills or habits through experience, observation or teaching.

Parental influences (Social explanation)- A child can learn eating behaviour and attitudes to food by observing their parents.

- Brown et Ogden (2004) reported consistent correlations between parents and children in terms of snack food intake, eating motivations and body dissatisfaction.
- Parents may also manipulate the availability of certain foods e.g. “You can have pudding after you've eaten your vegetables”. Birch et al shows that this approach is not successful as the preference for the reward food increases but the preference for the other food decreases.

Peer influence (Social explanation)- Bandura’s social learning theory illustrates how we learn from peers (Attention, retention, reproduction, motivation - the 4 mediational processes). Following the behaviour of peers the same age as you has a great effect. Greenhalgh et al (2009) found that children who sat next to peers with positive modelling were more likely to try an unfamiliar food, whereas negative modelling had a profound effect with children refusing to try the unfamiliar food.

- Birch et al (1980) illustrated how exposure to another child could change food habits. Birch sat children next to others who had a different vegetable preference. After 4 days the children showed a change in their preference of vegetable and this change was still evident weeks later.

Media effects (cultural explanation) - Macintyre et al (1998) found that media has a major impact not only on what people eat but their attitudes to food. People gain knowledge from the media about what to eat however sometimes this information is limited by persona circumstances of age, income.

Adolescents who watch moderate amounts of TV will be exposed to adverts which generally promote unhealthy food, using ‘fun’ themes and promoters to which the children can identify with. Therefore, using the same mechanism as social learning theory, the children identify with and imitate the food choices of these role models. Media restrictions have now been put in place for advertising for children with promotional characters and offers on tv are restricted by governments.

The context of meals (cultural explanation) - In societies like the US and the UK, the need for quick convenient food is increasing with many people grazing rather than eating meals.

- Maguire et al (2015) found that in the UK, the number of takeaway restaurants has risen by 45% in the last 18 years, with the areas of highest deprivation seeing the greatest rise.
- Gilemen et al (2000) found that eating more informally in front of the TV led to an increased preference for quickly prepared food.
- Cultural preferences for meat: Britain and France are two of the many countries which favour offal i.e. every part of the animal is eaten. In contrast, despite the USA eating much steak, they are opposed to offal. This shows that cultural differences in what is considered 'edible' are significant.
- Cultural norms of standards for meals: Different cultures will have different ideas of what is a 'proper' meal. In many Western countries, a 'proper' meal will contain meat and vegetables, as opposed to other cultures, such as China and Japan, where there is a greater emphasis on many smaller meals being eaten together.
- The role of culture in food preference: Children identify their parents as role models, and so will imitate their feeding patterns i.e. the frequency and time at which food is served. This is particularly the case if the adults are seen to be vicariously reinforced for obeying these rules, as well as encouraging the child to explore new food through positive flavour-flavour learning ('a type of Pavlovian conditioning where a neutral flavour is paired with an already preferred flavour', and so repeated pairings result in an association between the two).

+ Supporting evidence for the role of the media in influencing food preference = Boyland and Halford (2013) provided evidence that food advertising influences preference and intake in children. However, it affects individuals differently, with adverts for foods high in fat, salt and sugar having a stronger influence on overweight children. Children who had the greatest preference for carbohydrate and high fat foods were also the ones who watched the most television. This supports the idea of the media being a major influence on food preference, through social learning theory and vicarious reinforcement.

+ Support for the role of peer influences in food preference = Jansen and Tenny found that seeing other children model "light" yogurts and drinks led to a preference for other light products in primary school children. This behaviour can be identified as social facilitation i.e. "the tendency for people to perform differently when in the presence of others compared to when they are alone".

— The influence of culture on food preference is limited = Chen and Yang (2014) researched the food preferences made by people over Twitter and found a significant association between healthy food choices and the number of grocery stores around but no association between the number of fast food outlets and healthy or unhealthy choices. This study illustrates that cultural influences do have an effect on learned food

habits but people are able to resist unhealthy habits if a healthier alternative is available. Therefore, the influence of culture on food preference is limited and is determined by individual differences.

— Limited ecological validity in studies of parental influence on food preference = The research into parental influence is limited by being small scale and using selective samples, making the findings difficult to generalise beyond the research setting i.e low ecological validity. Robinson et al (2001) studied 800 children aged 8-9 from a number of different backgrounds and found a complex association of behaviour between parents and children but found that girls were more easily influenced by parental modelling than boys. Wardle et al (2005) found that parental fruit and veg consumption was a strong predictor of children's fruit and veg consumption. This together suggests that there are individual differences in family influences and the extent to which different genders identify with their role models, as well as family influences not entirely determining their children's food preferences.

— Research has found that not all parental influences are effective = Positive modelling and food exposure is effective in promoting healthy eating, whilst restricting access and force consumption is not as effective. A meta-analysis conducted by Savage et al (2007) found the parents of children with healthy food choices used the effective techniques. This supports the claim that children learn food preferences from their parents but not all methods of parental influence are effective.

Part 3 - Neural and Hormonal Mechanisms

Neural Mechanisms in the Control of Eating:

Key Words

Ghrelin- Is a hormone that is released in the stomach and which stimulates the hypothalamus to increase appetite. Its levels increase when a persons bodily resources are low, and triggers the 'on switch' of eating behaviour.

Hypothalamus - An area of the brain which has a number of important functions, including the regulation of body temperature (thermoregulation), hunger and thirst. The lateral hypothalamus is considered to be the 'feeding centre' of the brain, whilst the ventro-medial hypothalamus is the 'satiety centre'. The actions of the LH and VMH are antagonistic.

Leptin - A hormone that plays a crucial role in appetite and weight control. It is normally produced by fat tissue and secreted into the bloodstream where it travels to the brain and decreases appetite, with its main role being as an appetite suppressant.

Homeostasis - It is the mechanism by which an organism maintains a steady internal environment. It detects whether the body has enough nutrients or glucose and returns these levels to normal through positive or negative feedback. In humans, glucose is crucial in producing feelings of hunger - if there is a decrease glucose levels in the lateral hypothalamus, this results in feelings of hunger, due to the secretion of the neurotransmitter NPY (neuropeptide Y), which is a strong hunger stimulant. Once the hunger has been satisfied, through the action of the LH and the suppression of the VMH, this leads to a rise in glucose levels and activates the ventromedial hypothalamus which leads to feeling of satisfaction.

1. The individual begins to feel hungry.
2. Action of the lateral hypothalamus results in increased levels of glucose and leptin, whilst ghrelin levels decrease in the blood.
3. The ventro-medial hypothalamus satiety centre is activated, inhibiting LH activity.
4. The individual feels full, and they stop eating.
5. As a result, glucose and leptin levels decrease, whilst ghrelin levels increase, 'triggering' feelings of hunger.
6. The lateral hypothalamus feeding centre is activated, and the individual begins to feel hungry once again.

The Lateral Hypothalamus — Researchers discovered that damage to the LH in rats caused an absence of eating and so inferred that the stimulation of LH cells elicits feeding behaviour. Researchers also discovered that this was the "on" switch for eating behaviour. The neurotransmitter neuropeptide Y is particularly important, as a powerful appetite stimulant and is released from the LH. Reynolds and Wickens (2000) found that injection of NPY into the hypothalamus caused rats to feed even when satisfied.

The Ventromedial Hypothalamus - Damage to the VMH caused rats to overeat, whilst stimulation of this area inhibited feeding. Damage to the nerve fibres passing through the VMH damages the PVN (the paraventricular nucleus) ,which can result in overeating as the PVN is responsible for many of our cravings. This is because [1]"in energy balance, a key role of the PVN is to convey information from the arcuate nucleus/ARC to other brain areas involved in appetite regulation".

This is part of the 'dual-centre' model of eating behaviour, where both parts of the hypothalamus play different roles in regulating feeding.

Hormonal Mechanisms in the control of eating

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[1] Simpson, A., Martin, N. and Bloom, R. *Hypothalamic Regulation of Appetite,* MedScape, 2008; 3(5):577-592.

Ghrelin- This hormone is released in the stomach and if their levels increase above a set norm, then the arcuate nucleus stimulates the lateral hypothalamus to increase appetite, through the increased secretion of neuropeptide Y. If an individual's bodily resources are low, their gherlin levels increase at an inversely proportional rate i.e. the longer someone has been hungry, the higher their ghrelin levels are. Gherlin has a role in how quickly we feel hungry after we have eaten. The levels almost double before we eat and then rapidly decrease during the 3 hours after we have eaten (Wren et al, 2001).

Leptin - Leptin plays a crucial role in appetite and weight control/loss, as an appetite suppressant. Its normally produced by fat/adipose tissue and secreted into the bloodstream, via the endocrine system, where it travels to the brain and decreases appetite by contributing to the VMH satiety centre mechanism. Leptin has two major functions - it counteracts the effects of neuropeptide Y (a feeding stimulant), and also increases the sympathetic nervous system activity which stimulates fatty tissue to burn energy. This is because leptin levels increase as the number of adipose (fatty) cells increase i.e. a positive correlation.

For example, Teitelbaum (1955) created lesions in the hypothalamuses of rats and discovered that this caused them to overeat. This demonstrates the antagonistic mechanism of the LH and VMH. Lesions in the VMH means that the LH will continually release NPY and so there is a continued feeling of hunger, with no satiety mechanism to curb this.

+ Support for the role of leptin in regulating eating behaviour = Some people develop a resistance to leptin, so it fails to control appetite and weight gain. Leptin resistance is often found in overweight people making it even harder for them to lose weight - but it is not just simply a leptin deficiency. A study by Heymsfield et al (1999) found that obese adults needed doses of leptin 20-30 times above the normal concentration to produce a significant weight reduction. This supports the idea of leptin as an appetite suppressant and its role in regulating eating behaviour.

— Oversimplified explanations = Damage to the LH can cause deficits in other aspects of behaviour such as thirst. Recent research has shown that eating behaviour is controlled by neural circuits and not just the hypothalamus, as previously thought (Sakurai et al 1998). For example, Valassi et al. (2008) highlighted the importance of other hormones, such as CCK (cholecystokinin) which stimulates the enteric nervous system to increase the frequency of impulses sent to the hypothalamus, and other neurotransmitters, such as serotonin and dopamine (which may not exclusively act on the hypothalamus). Therefore, the dual-centre model may be an oversimplified homeostatic control mechanism explanation and with increasingly new research, may only be seen as one of several explanations for regulating eating behaviour.

+ Supporting evidence for the role of ghrelin as an appetite stimulant = Wren et al (2001) provided research supporting the role of grehlin in appetite control. A double blind study was carried out and participants were either given grehlin or a saline infusion intravenously. One week later participants experienced the second condition and appetite was measured in terms of the volume of food they consumed. The results showed a significant increase in food consumption in the grehlin sample with a mean difference of 28%. This supports the role of ghrelin as an appetite stimulant and its precursor effects on the release of NPY from the LH, as signaled by the arcuate nucleus. A double-blind study removes the confounding influence of investigator effects and psychological placebo effects, thus increasing the internal validity of the findings.

— Ignoring the role of social and cultural factors = For a hunger mechanism to be adaptive it must anticipate and prevent energy deficits as well as promote levels of consumption so that bodily resource levels, such as glucose, are well above the optimal level. Food intake based solely on homeostasis is limited and far more influenced by social and cultural norms e.g. set meal times. These social factors, as suggested by Woods et al (2004), can override the homeostatic glucose control mechanisms, as these mechanisms are only used in 'emergency' scenarios. Therefore, the focus on glucostatic mechanisms is an example of biological reductionism and ignores the role of social and cultural factors.

Part 4 - Biological Explanations for Anorexia Nervosa

Key terms

Anorexia Nervosa — A type of eating disorder in which an individual, despite being seriously underweight, fears that she or he might become obese and therefore engages in self starvation to prevent this happening. The DSM-IV criteria for Anorexia Nervosa are A. Weight loss leading to a maintenance of body weight less than 85% of that expected, B. Intense far of gaining weight or becoming fat and C. Disturbance in the way in which one's body weight or shape is experienced, alongside denial of the seriousness of the current low body weight.

Biological explanations — An approach to explaining behaviour which suggests that a full understanding of thoughts, emotions and behaviour must include an understanding of their biological basis, i.e. the role of genetics, neural correlates and hormones.

Genetic explanations — These explanations suggest that the likelihood of behaving in a particular way is determined by a persons's genetic makeup, i.e. it is inherited from parents. In the case of AN, this includes the roles of candidate genes, such as Ephx2.

Neural explanations — Explanations which involves areas of the brain and nervous system and the action of chemical messengers in the brain known as neurotransmitters as a means of controlling and predicting behaviour.

Genetic explanations

- **Family studies** have shown that eating disorders such as AN run in families. First degree relatives of individuals with AN have a 10 times greater chance of developing AN than relatives of unaffected individuals (Strober et al, 2000). It therefore follows that people may inherit a vulnerability towards developing eating disorders (Tozzi et al, 2005).
- Holland et al. found concordance rates of 56% for monozygotic twins, compared to only 5% of dizygotic twins, out of a sample of 45 pairs of female twins. Since MZ twins share 100% of their genes with each other, whilst DZ twins share only 50%, these concordance rates are highly suggestive of a genetic basis to AN.
- **Twin studies** suggest a high heritability for AN, varying from 28% to 74%. For example, Wade et al (2000) interviewed over 2163 female MZ and DZ twins, evaluating them on the DSM-IV criteria for AN. The researchers found a heritability estimate of 58%, alongside reaching the conclusion that "the comorbidity between anorexia nervosa and major depression is likely to be due to genetic factors that influence the risk for both disorders". Since these concordance rates were over 50%, such twin studies suggests that there may be candidate genes which lead to a genetic predisposition of some individuals towards AN.
- Conclusions drawn from twin studies are difficult to make reliably as MZ twins share the same genes and environment, making it hard to separate the influences of nature and nurture. **Adoption studies** avoid this because each pair of twins do not share the same environment. For example, Klump et al (2009) studied 123 adopted sibling pairs and 56

biological sibling pairs, finding heritability estimates ranging from 59% - 82% for the different aspects of disordered eating.

Evaluation of Genetic explanations:

– Problems with the genetic explanation = Fairburn et al (1999) found that although many studies have been conducted, the actual heritability estimate is still unknown. The researchers point out that results have been inconsistent and the studies not always replicated, making it difficult to draw reliable conclusions. Many studies violate the equal environments assumption ([1]'that identical and fraternal twin pairs experience equivalent trait-relevant environmental exposures'). Twin researchers also assume than MZ and DZ twins raised together have equal environments. However, this may not be the case as it has been shown that MZ twins tend to be treated more similarly than DZ twins (Loehlin and Nicols, 1976), and so their similar environmental factors may have a greater influence on the development of AN compared to DZ twins.

– The genetic explanation ignores the role of media in AN = The media's portrayal of ultra-thin models is an important risk factor for eating disorders. However, Builk (2004) suggests that genetically vulnerable individuals might seek out images themselves of thin role models. Vaughn et Fouts conducted a longitudinal study which found that adolescent girls anorexia symptom severity increased over a period of 16 months when they read more fashion magazines during this time. This suggests that social learning theory, through the use of mediational processes and vicarious reinforcement, may be an explanation for why adolescent girls identify with ultra-thin models role models, and hence progress the development of AN.

–In the US, treatment for AN is restricted under many insurance plans as it is not considered to have a biological basis = In 2014, the US tried unsuccessfully to get eating disorders included in essential health benefits through legislation. However, research creates a case allowing AN to be considered in the same way as schizophrenia as they are both considered to be biologically based e.g. [2]AN seems to affect between 1 and 4% of schizophrenia patients, and so there is a high prevalence of comorbidity between schizophrenia and eating disorders.

Neural explanations

- Serotonin: Disturbances in the levels of the neurotransmitter serotonin may be responsible for producing characteristic symptoms of AN, such as appetite suppression and obsessiveness. For example, Bailer et al (2011) measured serotonin activity in women recovering from either restrictive type anorexia or binge eating and found higher levels of serotonin activity. In addition, the researchers found that the women with the highest levels of serotonin had the highest level of anxiety, suggesting that persistent disruption of serotonin levels may lead to increased anxiety which could trigger AN. Those suffering from

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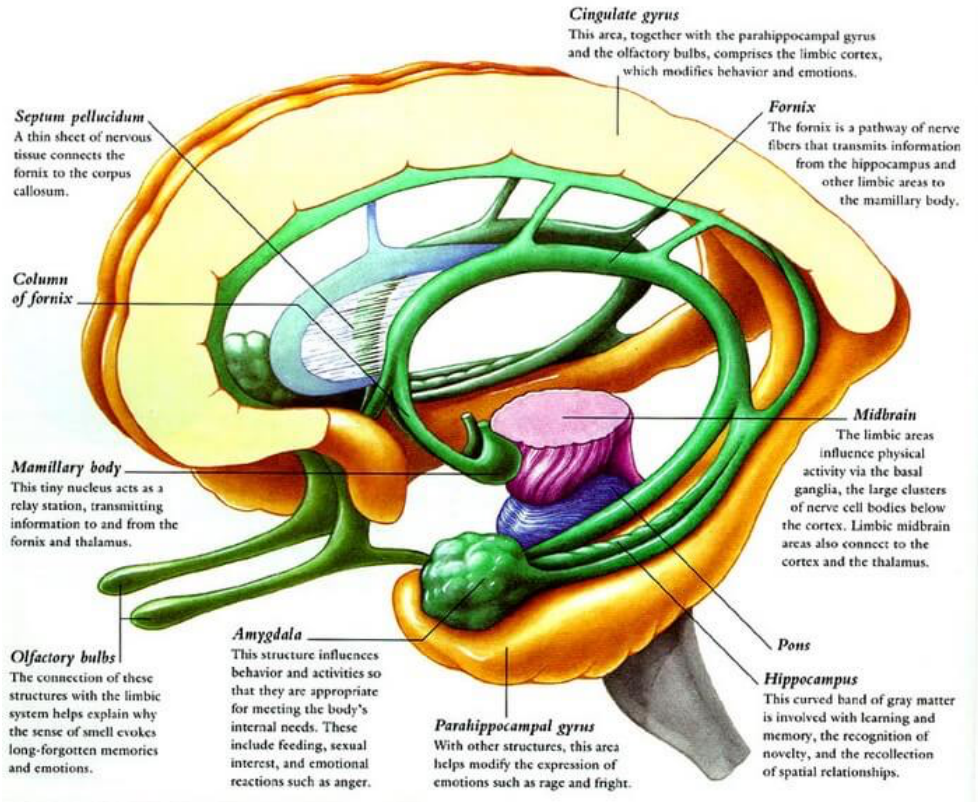
[1] Fosse R, Joseph J, Richardson K. A Critical Assessment of the Equal-Environment Assumption of the Twin Method for Schizophrenia. *Frontiers in Psychiatry*. 2015;6:62. doi:10.3389/fpsyt.2015.00062.

[2] Youssef Kouidrat, Ali Amad, Jean-Daniel Lalau, and Gwenole Loas, "Eating Disorders in Schizophrenia: Implications for Research and Management," Schizophrenia Research and Treatment, vol. 2014, Article ID 791573, 7 pages, 2014. doi:10.1155/2014/791573

AN were found to have low levels of metabolites, such as 5-HIAA, which are produced by the breakdown of serotonin. These low levels suggest that AN sufferers have abnormally functioning serotonin systems, where the neurotransmitter is now always broken down.

- **Dopamine:** Kaye et al (1991) used a PET scan to compare dopamine activity in the brains of 10 women recovering from AN and 12 healthy women. They found overactivity in the dopamine receptors of the women with AN in the basal ganglia, where dopamine plays a part in the interpretation of harm and pleasure. Increased dopamine activity in the basal ganglia leads people to interpret rewards differently. This matches the fact that individuals with AN find it difficult to associate good feelings with things, such as food, which people find pleasurable.



- Secondly, Bailer et al (2012) found that AN sufferers injected with amphetamines, causing an increase in dopamine levels, experienced anxiety whilst healthy control patients experienced euphoria. Therefore, restricting food intake may be a way of controlling anxiety through reducing bodily dopamine levels.

- **Limbic system dysfunction:** Lipsman et al (2012) suggests that the neural roots of AN appear to be related to a dysfunction in the limbic system. Researchers claim that dysfunction in these areas whose normal functions is to regulate emotion, can lead to deficits in emotional processing, which may lead to pathological thoughts and behaviours that are typical of AN.

Evaluation of Neural explanations:

— Problems with the serotonin explanation = SSRIs, which increase the levels of serotonin in the brain by reducing the breakdown at the presynaptic cleft, are ineffective when used with AN patients. Ferguson et al found no difference in symptom outcomes between patients taking SSRIs and patients of a similar age who were not taking an SSRI. A second problem with serotonin explanations is the effort to isolate serotonin as the single neurotransmitter responsible for AN. In reality, serotonin interacts with dopamine, where both are precurosors

to noradrenaline, which may also interact with the neurotransmitter GABA. Therefore, the focus on serotonin is a very simplified explanation for AN.

+ Research has supported the role of dopamine = Food aversions, weight loss, and distorted body image cognition have been found to be related to an increased activity in dopamine pathways (Kaye 2008). Barbeto et al (2006) found that increased blink activity is indicative of higher levels of dopamine activity, with a correlation found between blink rate and the duration of AN, suggesting that [3]there is a significant positive correlation between blink rate and the duration of illness. Kaye et al (1999) found that recovering AN patients displayed decreasing HVA/homovanillic acid levels (a metabolite of dopamine). This study is particularly useful because it removes the confounding variable of HVA levels decreasing during AN due to malnutrition or a lack of exercise.

+ Biological Explanations of AN reduce stigma = Such explanations also prevent the belief that it's "their fault" , alongside offering the possibility of treatment. For example, deep-brain stimulation/DBS , particularly of the nucleus accumbens in acute chronic cases, may therefore be a potential treatment for AN (counteracting the abnormalities in the limbic system), where [4]Lipsman et al. found an average BMI of 3.5 points to 17.3 within the AN group, with 6 of the 16 patients achieving a normal BMI.

Part 5 - Family Systems Theory and Anorexia Nervosa

Key words

Autonomy — The freedom to make decisions and determine actions without the constraints imposed by others, and the extent to which we are dependent from others.

Control — Means to direct or to exercise authoritative influence over events of behaviours. In the case of AN, sufferers exert control in an effort to reduce family dependence.

Family systems theory — A psychodynamic explanation which argues that in order to understand a family system, we must look at the family as a whole and not just at its individuals members.

Minuchin et al (1978) developed the psychodynamic family theory, which suggests that a dysfunctional family was the prerequisite for the development of AN. The psychodynamic family was a necessary context for the development for AN, so treatment must aim to change the way in which the family functions.

Characteristics of the "psychosomatic" family

1. Enmeshment — Describes a family where the parents are excessively emotionally- involved with their children but may be dismissive of their emotional needs. This can make it hard for

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[3] Increased dopaminergic activity in restricting-type anorexia nervosa, Barbato, Giuseppe et al. *Psychiatry Research*, Volume 142 , Issue 2 , 253 - 255

[4] Nir Lipsman, Eileen Lam, Matthew Volpini, Kalam Sutandar, Richelle Twose, Peter Giacobbe, Devin J Sodums, Gwenn S Smith, D Blake Woodside, Andres M Lozano. **Deep brain stimulation of the subcallosal cingulate for treatment-refractory anorexia nervosa: 1 year follow-up of an open-label trial**. *The Lancet Psychiatry*, 2017; DOI: 10.1016/S2215-0366(17)30076-7

the child to develop an independent self concept. The self-identity of each family member overlaps with other members, due to a lack of boundaries and respect for each other's privacy. Enmeshed families are over involved with each other and there is a lack of boundaries. Barber and Beuhler contended that enmeshment stifles the development of children skills to deal with common social stressors and make the development of AN more likely. The assumption is made that each member understands and can speak for the other. This leads to the adolescent AN sufferer having the desire to distinguish themselves from other family members, and particularly the mother.

2. Control/Overprotectiveness— The psychosomatic family is characterised by overprotective control, where there is a high degree of concern for each other's welfare. This can lead the individual to try and gain back some control by refusing to eat. Particularly important, according to Palazzoli (1974), is the role of the mother: she sees her actions as an example of self-sacrifice for her daughter, and so will automatically blame the daughter whenever any of her 'decisions' are bad. This in turn reduces the independence of the anorexic daughter. The mother plays a particularly important role by being intrusive and domineering.

3. Rigidity — Rigid families show an inflexibility in their adaptation to new situations. In the face of stress, they increase the rigidity of their patterns of behaviour and resist any exploration of alternatives. Any attempts at 'self-differentiation', such as the daughter trying to assert more independence, is quickly extinguished by the family, who cannot and will not adapt to internal or external threats to their circumstances. This may be reflected in the child starving themselves, in an attempt to 'control their own destiny' and achieving autonomy by acting against the wishes of her parents (Bruch, 1978).

4. Lack of conflict resolution/ Conflict Avoidance— Munichin believes that AN families were in a state of constant but unresolved conflict, with conflicts being abandoned rather than solved. In families with AN, patients typically present a strong façade of togetherness in order to avoid conflict (Latzer et al, 2002). An excerpt from their (Latzer et al) report reads: [5]"The families of eating disorder patients were found to be less cohesive, expressive, and encouraging of personal growth than were controls … this may reflect family difficulties in supporting the child during the process of separation individuation, and exploration of the outside world".

Within the family where child is involved in conflict, Munichin identified 3 characteristics of patterns of conflict-related behaviour:

A. Triangulation — Involves a pair of family members either incorporating or rejecting a third family member.

B. Parent-child coalition — The child now moves into a more stable coalition with one parent against the other. The coalition parent will respond to the child with excessive concern (enmeshment) whilst the other parent withdraws.

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[5] Yael Latzer, Zipora Hochdorf, Eitan Bachar, Attachment Style and Family Functioning as Discriminating Factors in Eating Disorders, *Contemporary Family Therapy*, 2002, Volume 24, Number 4, Page 581.

C. Detouring — When parents are unable to solve conflicts by themselves they direct focus away from themselves and onto the child. The child consequently becomes the scapegoat because their wellbeing is sacrificed for avoidance of marital conflict.

Evaluation of Family Systems Theory:

+ Research support for enmeshment = Manzi et al (2009) found that cohesion between family members was linked to positive outcomes and psychological wellbeing amongst adolescents, whereas enmeshment had the opposite effect. Family cohesion was indicative of supportive family interactions, whereas enmeshment was rooted in manipulation and control. This supports the idea that enmeshment may be a trigger for AN because starvation may be an attempt of the child to differentiate themselves from other family members and maintain a degree of control/autonomy, which increases the thinner they become.

— Problems with the psychosomatic family model = Research has tried to establish characteristics that are specific to families in which a member has AN. However, this has produced inconsistent findings. For example, Kog et Vandereycken failed to find the characteristics provided by the psychosomatic family model and instead proposed that this model can be simplified to [6]3 components: the intensity of intrafamilial boundaries, the degree of the family's adaptability, and the family's ways of handling conflicts. There is also growing evidence that families in which someone has an eating disorder are a diverse group in terms of the nature of family relations. Therefore, this suggests that the psychosomatic model is an over-simplified explanation for AN, and can only be applied to some families, hence not being a universal explanation.

— Inconclusive support from family-based therapy = The success of family focused therapy is a key part of recovery. Carr et al (2000) concluded that there is compelling evidence for the effectiveness of family intervention, citing Shadish and Baldwin (2003), who found that over 71% of families experienced improved family dynamics, compared to a control group, after familial interventions. However, Grange et Eisler (1987) pointed out that whilst there is some evidence that family therapy is accompanied by changes these may not happen in all families, and therefore has limited practical applications, further questioning the validity of the psychosomatic family model explanations.

— Gender bias in family systems = Gremillion claims that there is a gender bias in family systems because they tend to focus only on mother-daughter relationships, with enmeshment is nearly always being seen as maternal. Focus on therapy tends to focus on reforming mothers and that there is a lack of awareness that fathers too contribute to the enmeshment and that the father's overly controlling tendency is often overlooked. Therefore, this may lead to stereotyping of an 'excessively overprotective and rigid' mother and parent-blaming, whereas both parents in reality have a role to play.

+ Research support for lack of conflict resolution = Latzer and Gaber (1998) carried out an observational study of conflict resolution in 40 families with AN and 40 matched families without AN. Parents and daughters were told to choose two areas of disagreement and were asked to keep off the topic of food. The families with AN had greater difficulty choosing

[6] Kog, E., Vandereycken, W. and Vertommen, H. (1985), The psychosomatic family model. A critical analysis of family interaction concepts. Journal of Family Therapy, 7: 31–44. doi:10.1046/j..1985.00663.x

topics and remaining focused on them whilst moving towards resolution. In addition, [7]"the pattern of conflict avoidance is also reflected in a low level of measured criticism and hostility in relatives of AN patients". This supports Munichin's claim that there is an avoidance of conflict where one family member has AN, and the role of conflict resolution in the development and maintenance of AN.

Part 6 - Social Learning Theory and Anorexia Nervosa

Key words

Media — The various means of communication such as radio, television, newspapers and the internet that reach or influence people widely.

Modelling — A form of social learning where individuals learn a particular behaviour by observing and imitating a role model performing that specific behaviour. This effect is greater if the role model is seen to be attractive, have high social status, being the same gender as the observer and of a similar age. The role model does not need to be physically present in the environment to exert influence.

Reinforcement — A term used in psychology to refer to anything that strengthens a response and increases the likelihood that it will occur again in the future. Vicarious reinforcement, according to social learning theory, increases the likelihood that an observed behaviour will be repeated if the model is seen to be rewarded.

Social learning — Learning through the observation and imitation of behaviours modeled by the role model, who has been identified by the observer. The modeled behaviors that are seen to be rewarded are more likely to be imitated by the observer, through the process of vicarious reinforcement. Learning occurs both directly (operant and classical conditioning) and indirectly (vicarious reinforcement).

Bandura believed that people learn by observing the behaviour of others as well as observing the outcomes of these behaviours. Social learning theory suggests that children pay attention to the role models in their lives and if they observe behaviour that is likely to produce a positive response from others, as judged by the role model being vicariously reinforced for displaying this behaviour, then they will imitate them in the belief that they will also receive the same positive feedback. Such learning depends upon the 4 mediational processes (cognitive processes which come between stimulus and response): Attention, retention, motivation and motor reproduction.

Maternal role models — Studies have found similarities between mother-and-daughter restrained eating and dieting behaviours amongst children as young as 10 years old, with such children also tending to overeat in a behavioural test of restraint (Hill et al, 1989). Smolak et al suggested that mothers who complain about their weight are more likely to have children who have their own weight concerns, with these influences being greater for daughters rather than sons.

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[7] Yael Latzer, Lee B. Gaber, Pathological Conflict Avoidance in Anorexia Nervosa: Family Perspectives, *Contemporary Family Therapy*, 1998, Volume 20, Number 4, Page 539

Peer influence — Peer reinforcement is particularly important during adolescence, so they may be susceptible to peer influence over eating patterns. A US study found that dieting amongst friends was linked to unhealthy weight control behaviours, such as taking diet pills or purging (Eisenburg et al). Jones and Crawford (2006) found that overweight girls and underweight boys were more likely to be teased, suggesting that teasing serves to enforce gender based ideals. Their [8]results show that despite girls reporting more appearance conversations, boys perceived more appearance pressure and teasing. In addition, Keel et al (2012) investigated the influence of college peers, both male and female, on disorganised eating. The researchers found that [9]'for women, college roommate dieting significantly predicted Drive for Thinness, Bulimia scores, and purging at a 10-year follow-up'. This further demonstrates the strong impact of peer influences on disorganized eating, through the mechanism of social learning, particularly due to some college peers being identified as role models.

Media influence — The portrayal of thin models on TV and in fashion magazines is a contributory factor for body image concerns. The media does not influence everyone in the same way as individuals with low self esteem are more likely to compare themselves to idealised images. Low self esteem plays a crucial role in the development of eating disorders. Button et al (2005) found that girls with low self esteem aged 11-12 years old were at a significantly greater risk of developing an eating disorder aged 15-17. A report published by the BMA expressed concern for the use of thin models and reported that the degree of thinness exhibited was not only unachievable but also biologically inappropriate. These ideas were further supported by Dittmar et al (2006), who found that a significant proportion of the 162 British girls, aged 5-8 years old, involved in the study reported low self-esteem and increased body dissatisfaction when viewing the Barbie dolls, compared to a control group who'd viewed the Emme dolls (who have a more realistic body shape). This deep-rooted body dissatisfaction at a young age may manifest itself as AN in later life, particularly due to Barbie being such an attractive role model and possessing many characteristics (such as glamour, popularity and attractiveness) which girls in particular aspire to.

Evaluation of Social Learning Theory and Anorexia Nervosa:

— Maternal influence is more complex than social learning = Research on the role of mothers has not always produced consistent results. For example, Pike et Rodin (1991) found that there was no evidence for daughters imitating the weight concerns of their parents. Ogden and Steward (2000) found that although mothers and daughters were of a similar weight, there

[8] Jones, D.C. & Crawford, J.K. J Youth Adolescence (2006) 35: 243. https://doi.org/10.1007/s10964-005-9006-5

[9] Keel PK, Forney KJ, Brown TA, Heatherton TF. Influence of College Peers on Disordered Eating in Women and Men at 10-Year Follow-up. *Journal of Abnormal Psychology*. 2013;122(1): 10.1037/a0030081. doi:10.1037/a0030081.

was no association for restrained eating. This suggests that although most daughters would identify with and view their mothers as role models, and so be expected to adopt similar eating habits through social learning theory, this is not always the case. This therefore casts doubts on the idea that SLT is a major explanation for AN.

+ Research support for peer influence = Costa-Fonta and Jofre Bonet (2009) investigated the effects of peer weight and developing an eating disorder. The researchers found that peers with a larger body mass had a smaller likelihood of developing AN, suggesting that having peers with an average or higher BMI protects individuals from eating disorders, through the mechanisms of social learning theory. Secondly, [10]several opinions from peers of an individual's own body mass were found to decrease the likelihood of women being thin or extremely thin. These pieces of evidence further support the role of peer influence as an explanation for anorexia nervosa.

+ Research support for media influence = In a study conducted by Becker et al (2011), eating attitudes amongst Fijian children were studied following the introduction of TV in 1995. After exposure to TV, it was found that the children expressed a desire to lose weight, and this desire increases with the more friends the individual has who had access to TV, videos or DVDs. This increase in the will to lose weight may be due to the discussions made amongst friends about the ultra-thin models they have seen on TV, creating a mutual goal. However, other research has shown that intervention prior to media exposure can prevent the adverse effects. This suggests that media can be a powerful influence but can be prevented through education.

+ Explains gender differences = Some studies have found that reading magazines is a more consistent predictor of the development of eating disorders than watching TV. Harrison and Cantor (1997) found no correlation between watching TV and eating disorders but found a correlation between reading fitness magazine and attitudes towards food and dieting. As the researchers found, [11]'For women, media use predicted disordered-eating symptomatology, drive for thinness, body dissatisfaction, and ineffectiveness. For men, men, media use predicted endorsement of personal thinness and dieting'. This shows that media influences, through the mechanism of SLT, can explain gender differences in bodily attainment goals. Men suffer predominantly from bigorexia (Jones and Morgan, 2010) due to increased exposure to athletic and muscular role models, whereas females suffer increasingly from anorexia, due to increased exposure to Size Zero models in socially-desirable contexts.

Part 7 - Cognitive Theory and Anorexia Nervosa

Key words

Cognitive theory — When applied to disorders, this is any explanation about the way in which a person processes information that affects their feelings and their behaviour. The focus, according to the cognitive approach, is on how our mental processes (e.g. attention and perception) can affect our behaviour.

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[10] Costa-Fonta and Jofre-Bonet., Body Image, Peer Effects and Food Disorders: Evidence from a Sample of European Women, *LSE Health*, November 2009, Working Paper No: 15/2009.

[11] Harrison, K. and Cantor, J. (1997), The relationship between media consumption and eating disorders. Journal of Communication, 47: 40–67. doi:10.1111/j.1460-2466.1997.tb02692.x

Distortions — Thinking that has a bias such that what is perceived by a person does not match reality. These may be a result from comparison with others, (i.e. thin models) leading to a misperception that the individual must be overweight, which in turn leads to feelings of self disgust and an attempt to lose weight. For example, Murphy et al (2010) believes that cognitive distortions are at the root of AN symptoms, particularly the tendency to overestimate personal weight and body size. Using the Body Image Assessment, Williamson et al (1993) supported this idea by showing how the (37) AN patients were significantly more likely to overestimate the size of their silhouette compared to the (95) healthy controls.

Irrational beliefs — Beliefs that are unhelpful, illogical and inconsistent with our social reality. In contrast, rational beliefs are helpful, logical and consistent with our social reality. Characteristic irrational beliefs of AN include absolutist thinking ("I have eaten an apple and so I have no self-control") and catastrophising ("I have gained half a kilogram and so I will be obese"). These irrational beliefs may stem from the anorexic's tendency towards 'never-ending' perfectionism - once they have reached their goals, the bar is set even higher, leading to a constant feeling of failure. For example, Hewitt et al (2003) argues that this façade of perfectionism is made up of [12]'perfectionistic self-promotion, non-display of imperfection and non-disclosure of imperfection'. Individuals may also develop self-harming habits due to faulty beliefs about themselves and the world around them. For example, a typical irrational belief of AN patients is that they must be thin for others to like them.

The Cognitive Behaviour Model — Garner and Bemis (1982) found that anorexia patients have many characteristics in common e.g. they are typically perfectionists and often full of self-doubt. These characteristics, coupled with an individual's exposure to cultural ideals of thinness, leads them to form extreme ideas about the importance of body weight and the irrational belief that losing weight will reduce their distress and make them more attractive to others.

Losing weight becomes self-reinforcing for the individual because of the positive comments from others. This can be explained by positive reinforcement according to behaviourism. Once the importance of thinness is established, anxiety about eating increases and this develops into a fear of food and weight gain, meaning that food avoidance becomes the norm. As the individual becomes more and more socially isolated, this reduces the chance of viewing their style of thinking as irrational.

The Trans-diagnostic Model — Fairburn et al (2003) suggested that we should see AN symptoms as manifestations of a more broadly defined eating disorder. According to this model, the underlying cause of all eating disorders is the same set of cognitive distortions. This involves the overestimation of body weight, appearance, and emphasis on self-control which Fairburn believes to be the central factor in AN. This, paired with a reduced capacity for 'set-shifting' , according to Treasure and Schmidt's interpersonal maintenance model of AN (2013), may lead to the development of AN. For those with AN ,their sense of self-esteem is determined by their weight and appearance, whereas for a healthy person it would be their achievements. The restriction of food intake that is a characteristic of AN is maintained by the mechanism below:

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[12] Hewitt, P. L., Flett, G. L., Sherry, S. B., Habke, M., Parkin, M., Lam, R. W., . . . Stein, M. B. (2003). The interpersonal expression of perfection: Perfectionistic self-presentation and psychological distress. *Journal of Personality and Social Psychology, 84*(6), 1303-1325.

1) An enhanced sense of self control leads to increased self esteem. When an individual with AN decides not to eat or to eat very little this provides positive reinforcement in the form of self esteem (a behaviourist mechanism).

2) The physiological and psychological changes they receive, such as starvation, are perceived as being the result of failure of self control, which in turn leads to a greater reliance on food restriction.

3) Due to their focus on weight, the individual engages in increased self monitoring of their weight, where any weight gain or loss perceived as being too slow leading to an increased effort to restrict intake.

Evaluation of cognitive theory of Anorexia Nervosa

+ Research support for role of cognitive factors in AN = Lang et al (2015) compared the performance of 41 children with AN with 43 control healthy participants and found no difference in IQ. However, the AN group showed more inflexibility and an inefficient cognitive processing style, thus potentially supporting Treasure and Schmidt's interpersonal maintenance model of AN, which is characterised by 'cognitive inflexibility'. AN patients were also unable to overcome previously held beliefs or habits in the face of new information, suggesting that this inflexibility may be an underlying factor in AN.

+ Support from Stroop Test studies = Ben Tovim et al used a version of the Stroop test called the Food Stroop test and found that compared to normal patients, AN patients found it harder to name the colours of highlighted words that were relevant to their weight concerns. This suggests a preoccupation with those stimuli and words related to their anorexia, and so an selective attentional bias. This may in turn explain why sufferers of AN are over-critical of themselves and suffer from obsessive thoughts.

+ Support from CBT-E therapy = This form of therapy is designed to address the underlying cognitive factors in AN. Fairburn et al compared this to the next leading non- cognitive alternative (IPT). 130 patients with an eating disorder were randomly assigned to one of the two therapies. The researchers found that, at the end of 20 weeks of treatment, 2/3 of the CBTE group met the criteria for remission whereas only 1/3 of the IPT group did, further indicating that CBTE is an effective treatment and cognitive issues are a root cause of AN. Grave et al. (2013) provides further evidence for the effectiveness of CBT-E, showing that when given to 99 adult AN patients over 40 weeks, [13]"64% of the patients completed the outpatient treatment and experienced a significant increase in weight; on average, a 7.47 kg increase".

— Methodological limitations = Viken claims there is a limitation of cognitive theories and that Garner and Bemis have an over reliance of self reports. For example, there is an assumption that thoughts of weight and thinness are accessible through verbal self- report measures. This ignores the issue of social desirability: many patients with AN would not want to disclose such

[13] Fairburn CG, Cooper Z, Doll HA, O'Connor ME, Palmer RL, Dalle Grave R. Enhanced cognitive behaviour therapy for adults with anorexia nervosa: A UK–Italy study. *Behaviour Research and Therapy*. 2013;51(1):R2-R8. doi:10.1016/j.brat.2012.09.010.

thoughts in fear of being diagnosed and then treated for an eating disorder. This is also particularly difficult considering that a characteristic symptom of AN is a denial of any type of illness or irrationality. Therefore, these methodological limitations reduce the validity of cognitive explanations for AN.

Part 8 - Biological Explanations for Obesity

Key Words

Biological explanations — A belief that a full understanding of thoughts, emotions and behaviour must include an understanding of their biological basis i.e. the role of genetics, neural correlates and hormones. This is based on the assumption of the biological approach, that everything psychological has a biological origin.

Genetic explanations — The likelihood of behaving in a particular way is determined by a person's genetic makeup i.e. the (candidate) genes inherited from the parents. Genes code for proteins and are short sections of DNA, found on the same area on a pair of homologous chromosomes.

Neural explanations — These explanations involve areas of the brain and nervous system, alongside the action of chemical messengers in the brain known as neurotransmitters. Such factors are thought to control behaviour. In obesity, the activity of the hypothalamus, as well as levels of serotonin and dopamine are considered to be particularly important.

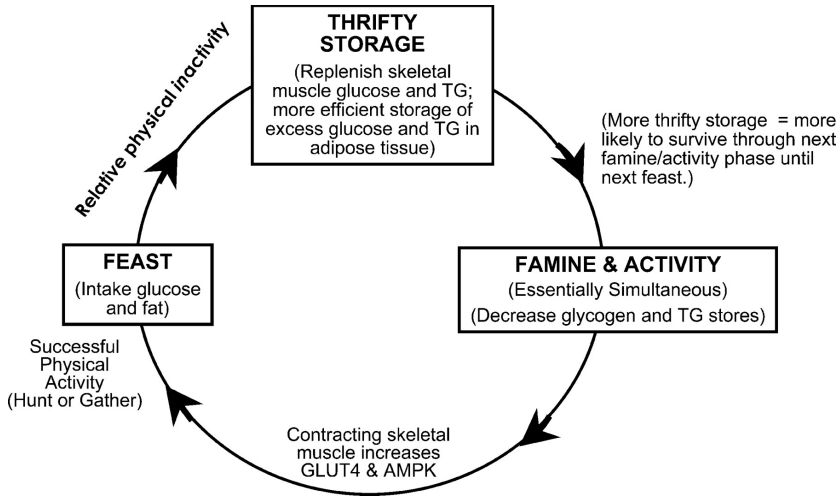
Twin and adoption studies — Maes et al (1997) conducted a meta-analysis of 75,000 individuals and found that the heritability estimates for BMI was 74% and 32% in MZ and DZ twins respectively. Even when MZ twins are reared apart they are more alike in terms of BMI than DZ twins. This reduces the impact of the environment on BMI.
Stunkard et al gathered information from 540 adult adoptees, their biological parents and adoptive parents. The researchers found a strong relationship between weight category for adopted individuals and their biological parent but no significant relationship between adoptee and adoptive parent, illustrating that there is a genetic link. This is further supported by concordance rates for first-degree relatives being between 20% and 50% (Chaput et al, 2014), where parent and child share 50% of their genes with each other.

Neural Explanations (Hypothalamus) — The arcuate nucleus is a collection of neurons in the hypothalamus which monitors sugar levels in the blood and signals the lateral hypothalamus to release NDP-Y when energy levels are low. Therefore, when the arcuate nucleus is activated, it sends messages to other parts of the body producing the desire to eat. However a malfunction may lead to overeating and obesity. For example, Licino et al (2004) shows that an inability to produce leptin, an appetite suppressant, is strongly linked with extreme obesity, where replacement of leptin in such individuals can cause weight loss rates exceeding 40%.

Leptin is a hormone secreted by fat cells that inhibits food intake and controls appetite, so a lack of Leptin could lead to overeating. This has been illustrated by Bates and Myers (2009) which showed that a disruption of Leptin signalling in the hypothalamus leads to obesity. Gibson et al reported that children in Pakistan who suffered from severe obesity experienced

dramatic effects on appetite metabolism and weight loss after 4 years of treatment with Leptin injections, supporting the role that Leptin plays in regulating appetite.

The Thrifty Gene Hypothesis also puts forward the idea that these genes allow those to gorge when food is abundant. This may have an evolutionary basis because in the times of early humanity, those who ate when food was abundant were able to store reserves of body fat for when food was scarce. However, now these genes do not provide the same evolutionary advantage as they promote fat deposition in preparation for a famine that almost never comes. However, Speakman (2008) argues that if the thrifty gene promotes obesity then the majority of people should be obese, whereas obesity is only linked to a few cultures and countries. This is especially the case due to all humans sharing common ancestors and all had originally experienced similar environmental/selective pressures.



Serotonin: Obesity is commonly associated with abnormally low levels of the monoamine neurotransmitter serotonin and its metabolite (5-HIAA), suggesting low levels of serotonin breakdown. Low serotonin levels means that fewer nervous impulses are transmitted to the ventro-medial hypothalamus, resulting in a lower rate of activation of the satiety centre, leading the individual to constantly believe that they are still hungry. Therefore, this leads to over-eating, particularly calorie-dense foods such as sweets and fatty items.

Dopamine: Obesity is also associated with low levels the neurotransmitter dopamine and low levels of D2 receptors (Weng et al,2001) particularly in the striatum. This results in less activation of the 'pleasure' centres of the brain, such as the amygdala and hypothalamus, and so the individual does not always feel content/happy after eating. Therefore, over-eating may be an attempt to increase this rate of activation and increase pleasure for the individual.

Evaluation of Biological Explanations for Obesity:

– The expression of genetic influences varies with age = Research suggests that the genetic contribution towards BMI is not stable across a person's lifetime. For example, Elks et al (2011) conducted a meta analysis of 88 studies and found that heritability estimates varied according to age. The researchers found that the heritability estimate was highest during childhood and then decreased during adulthood due to greater gene expression in childhood. On the other hand, in adulthood there is a greater emphasis on dietary and exercise habits which decreases the genetic contribution to the individual's BMI. This suggests that genetic explanations of obesity are not only limited towards individuals, but also towards their age, suggesting that such explanations may have little practical value in terms of reducing the incidence of obesity.

– The problem of time and geography = In 2015, the government report stated that there has been a sharp rise in obesity rates over the last 20 years. In 1993,13% of UK males were

classified as obese, compared to 26% in 2013. However, the composition of the gene pool has remained the same in this time, suggesting that an explanation based on genetics alone cannot explain the sudden increase in obesity rates. Similarly, within the same culture obesity rates are different with a 5% decrease in obesity rates in the countryside of China but more than 20% are obese in major cities, suggesting that there is likely to be other contributory factors apart from genetics, such as the influence of media, the accessibility of fast foods and the lack of physical exercise of those living in cities.

+ Research support for leptin = Montague et al (1997) reported the case of two severely obese cousins in Pakistan and found that they both had very low levels of leptin. Gibson et al (2007) also found similar results, where after 4 years of treatment with leptin injections it had beneficial effects on appetite, metabolism and weight. For example, [14]"leptin replacement reduced brain activation in regions linked to hunger (insular, parietal and temporal cortex) while enhancing activation in regions linked to inhibition and satiety (prefrontal cortex)." Therefore, this supports the role of leptin in regulating appetite and its deficiency as a risk factor for obesity.

+ Advantages of biological explanations = The biological explanations offered are out of a person's control and are therefore less stigmatising, leading to less stereotyping and prejudice against obese individuals. An increased scientific understanding of the biological mechanisms of obesity can lead to the development of treatments to deal with the disorder e.g. the use of sibutramine and orlistat produce, on average, 3-5 kg of weight loss (Jain et al, 2005).

– Problems with the Thrifty Gene Hypothesis = Speakman (2008) argues that if the thrifty gene hypothesis was correct, then the majority of the population would be obese because most people should have inherited these "thrifty genes". For example, a study by Ng et al (2014) found that more than half of the world's obese population live in the same 10 countries, with a 23.8% increase in BMI for boys and a 22.6% increase in BMI for girls in developing countries. This suggests that cultural factors (such as reduced physical activity and easier access to 'fast food') may be a better explanation for obesity than evolution.

Part 9 - Psychological Explanations for Obesity

Key words

Restraint theory (Herman and Polivy, 1975) = Restraint is the conscious restriction of food intake to prevent weight gain or to promote weight loss. This theory proposes that attempting to restraint eating actually increases the probability of overeating. It is characterised by the ideas of cognitive control and paradoxical outcomes.

Cognitive control is the concept that we consciously think about and control what we eat, by making clear distinctions between 'good' and 'bad' foods, alongside making judgements about the quantities of each food we can eat. However, this pre-occupation with food means that we ignore the physiological messages that our body sends us when we are hungry and when we are full, hence resulting in paradoxical outcomes through disinhibition.

There are different types of restraint. For example, "rigid " restraint represents 'all or nothing' dieting, whilst "flexible " restraint is a less strict approach to dieting. Provencher et

[14] Baicy K, London ED, Monterosso J, et al. Leptin replacement alters brain response to food cues in genetically leptin-deficient adults. *Proceedings of the National Academy of Sciences of the United States of America*. 2007;104(46):18276-18279. doi:10.1073/pnas.0706481104.

al (2003) reported that rigid restraint tends to be positively correlated with the amount of body fat, waist circumference and BMI, whereas flexible restraint tends to be negatively correlated. This suggests that, on average, flexible restraint eating is more effective than the rigid alternative. Herman and Mack (1975) conducted a study where participants who had eaten nothing were split into 3 groups where they either had no milkshake, one milkshake or two. This was then followed by ice-cream. The researchers found that the unrestrained eaters ate less of the ice cream if they had had the milkshake, the restrained eaters who had not had the milkshake had a limited amount of ice cream, whereas those who had the milkshake had more ice cream. The study showed that [15]"the expectation that high restraint subjects' intake would vary directly with preload size, while low restraint subjects would eat in inverse proportion to preload size, was confirmed".

Disinhibition — The removal of normal inhibitions to over-eating, resulting in the tendency to over eat in response to a range of different stimuli. Obesity may be explained as a cycle of restraint and disinhibition. The individual undergoes restrained eating and then, due to the feeling of 'missing out', proceeds to 'over-eat' which may lead to a binge. This is particularly likely if they are exposed to stimuli which reminds them of their favourite food e.g. eating chocolate when they are stressed (internal factors) or the smell of chocolate (external factors). Disinhibition is made worse through absolutist thinking - the individual does not stop excessively eating because they see that restraint has failed.

Bond et al (1998) identified 3 types of disinhibition, which are positively correlated with weight gain. These can be further simplified into internal and external factors:

1) Habitual - the tendency to overeat in response to daily life

2) Emotional - the tendency to overeat in response to emotional states

3) Situational - the tendency to overeat in response to environmental cues i.e. social events

The Boundary Model — In an attempt to explain why dieting leads to overeating, Herman and Polivy (1984) developed the boundary model. According the model, food intake is along a continuum with hunger at one end and fullness at the other. The researchers propose that there is a zone of biological indifference where the individual is neither hungry or full. Restrained eaters have a larger ZBD, so they have a lower threshold for hunger but need more to feel full. Herman and Polivy claim that dieters set a self-imposed boundary but if they go over this boundary, the "what the hell effect" (pasivity and resignation) kicks in and they continue to eat past the point of satiation.

It is important to make the distinction that for restrained eaters, more of their eating behaviour is controlled by cognitive factors, rather than physiological, and the opposite is true for a normal eater.

Evaluation of Psychological Explanations for Obesity:

+ Support for restraint theory = Wardle and Beales (1988) randomly allocated 27 obese women to a diet group, an exercise group or a non treatment group for 7 weeks. At weeks 4 and 6, participants were assessed and results showed that in the diet condition. Women in the diet group ate more than in the other two groups, as predicted by restraint theory and so

[15] Herman, C. P. and Mack, D. (1975), Restrained and unrestrained eating1. Journal of Personality, 43: 647–660. doi:10.1111/j.1467-6494.1975.tb00727.x

supporting the ideas of a lack of cognitive control leading to paradoxical outcomes. However, the highly-controlled nature of laboratory settings may have been unfamiliar and potentially stressful for the participants, leading them to change their eating patterns as a result. This results in reduced ecological validity of the findings.

+ Supporting evidence for the "what the hell effect" = This may be a characteristic of the breakdown in the dieter's self control. Ogden and Wardle (1990) argue that rather than a desire to eat, the individual may overeat as a form of rebellion against their self-imposed food restrictions. This was supported by Ogden and Wardle's study which showed that [16]"the restrained eaters experiences increased feelings of rebelliousness and defiance after both eating situations, with the increase being particularly pronounced after the high-calorie food". Further support was given by Loro and Orleans (1981), who found that obese binge eaters frequently report binging as a way of unleashing resentment. Therefore, there is supporting evidence for the role of disinhibition, passivity and resignation in the development of obesity.

— Disinhibition may not be important in all groups = Atlas et al (2000) reported that restraint and disinhibition scores were lower in African-American students compared to white college students. Similar restraint studies have shown that disinhibition is lower in men than women, so women are more easily influenced to gain weight. Nevertheless, this suggests that the disinhibition is affected by individual differences and so may not be a universal cognitive explanation for all cases of obesity.

+ Disinhibited eating may be related to attachment style = Research (Wilkinson et al, 2010) suggests that disinhibition may be linked to an insecure-avoident attachment style. For example, the researchers concluded that [17]"attachment anxiety is a good predictor of disinhibited eating… and it is through this relationship that attachment anxiety also predicts BMI. Such findings are consistent with other studies, showing an association between attachment orientation and other disinhibited behaviours, including alcohol and substance abuse". Therefore, such disinhibitions may be a method of scapegoating or transferring the anxiety and negative emotions associated with poorly-formed, insecure childhood attachments.

Part 10 — Explanations for the Success and Failure of Dieting

Key words

Dieting — A deliberate reduction of food intake in an attempt to lose weight.

Hedonic eating — Stroebe suggested that those who had problems maintaining their diets may be more sensitive to the hedonic (pleasurable) properties of food. This may be due to the increase in sensitivity towards the perception of attractive food triggering a desire to eat and as a result, any conflicting goals such as a diet will be inhibited.

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[16] Ogden, J. and Wardle, J., Cognitive and Emotional Responses to Food, International Journal of Eating Disorders, Wiley Publications, 1991.

[17] Wilkinson, L., Rowe. A.C., Bishop, R.J. and Brunstrom, J.M. (2010), Attachment Anxiety, Disinhibited Eating and Body Mass Index in Adulthood, International Journal of Obesity, 34: 1442—1445.

Attention allocation — Mischel and Ayduk suggested that the difficulty in resisting tempting foods may be because once the temptation has triggered pleasurable thoughts, restrained eaters find it hard to withdraw their attention from the attractive food items.

Role of denial — Research has shown that attempts to suppress or deny a thought has frequently the opposite effect, i.e. Wegner et al (1987) asked participants not to think about a white bear and others to think about a white bear, then ringing a bell if they did. He found that the participants who were told not to think about the bear rang the bell more often. This therefore implies that as soon as a food is denied it becomes more attractive, due to suppressing thoughts about it.

Redden suggests that people usually like things less as they repeat them, such as sticking to a salad diet, and that we should focus on details because we become bored less easily and are able to stick to it. For example, the jelly bean task involved participants who were either given a number of the jelly beans or provided with great detail (e.g. flavour and shape). The researchers found that the participants who'd been given greater detail actually ate more jelly beans i.e. the results demonstrate [18]"the 'specificity effect'… subcategorization focuses people's attention on differentiating aspects, making the episodes seem less repetitive and consequently less satiating".

The Spiral Model (Heatherton and Polivy) — Body dissatisfaction amongst adolescents increases the likelihood of them to attempt to lose weight. Although this initial attempt is successful, prolonged weight loss is difficult, leading to frustration and anxiety. Nonetheless, the individual sticks to their original plan and further restricts their eating, regardless of the fact that such restraint and disinhibition simply makes it even harder to lose weight, due to increasing ghrelin levels and decreasing leptin levels. This may result in depression.

Ironic Process Theory — Link to Wegner's white bear experiment, which increases the likelihood of disinhibited eating due to an increased (rather than decreased) preoccupation with food and a reduced capacity for cognitive processing.

Evaluation of Explanations for Failure and Success of Dieting:

+ Research support for the Ironic Process Theory = Soetens et al (2006) divided participants into restrained and unrestrained eaters, where the restrained group was further subdivided into high or low disinhibition. The disinhibition restrained eating group used more suppression than the other groups and also illustrated a 'rebound' effect where they thought more about their food after consumption. Wegner admits that the "ironic" effect observed is not huge but detectable.

+ Support for the Hedonic Theory = The hedonic theory claims that restrained eaters are more likely than unrestrained eaters to focus on the pleasure of food. Brunstrom et al (2004) tested salivary reactivity in 40 participants when they were close in proximity to hot pizza. Restrained eaters showed a greater salivary response than unrestrained eaters, suggesting that there is a difference in perception of food for both groups.

+ Improved understanding of the effectiveness of anti-dieting programmes = Concerns about the damaging effects of many diet programmes has led to the development of programmes aimed at healthy eating instead. These programmes emphasise regulation and prevention of inappropriate attitudes to food. Higgins and Gray conducted a meta analysis of anti-dieting programmes and found that it was associated with improvements in eating behaviour and

[18] Redden, J.P. (2008) Reducing Satiation: The Role of Categorization Level, Journal of Consumer Research, 34(5), pp. 624-634.

weight stability, rather than weight change. This suggests that eating when hungry and stopping when satiated is better than restricted or unrestricted eating.

— Limitations of anecdotal evidence = Many studies of dieting come from individual studies. The main limitation is that memory is not 100% accurate nor is the evaluation of success or failure. This creates unreliability, and so casual connections are made without accounting for the effects of extraneous variables.

+ Free will or determinism? = Lipoprotein lipase is an enzyme produced by adipose fat cells to help restore calories as fat. LPL makes it easier to regain lost weight. For example, Kern et al (1995) studied 9 people who lost an average of 90 pounds and had their LPL levels measured both before and after dieting. The researchers found that levels of LPL rose after weight loss and the heavier the person was to start with the higher the LPL levels were. This suggests that the body was almost fighting to regain the weight (Kern et al). This implies that some people will struggle to lose weight regardless of their intentions because of genetics, as the weight loss activated the gene producing the LPL.