Eating behaviour;

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eating behaviour; * There are a number of physiological processes that influence when and what we eat. * However, as in many aspects of human behaviour, the effects of these physiological processes are mediated by psychological and social ones. * Many different things motivate us in: - what we choose to eat. -when we eat. - how much we eat including convenience, - what it looks like - health concern - ethics. * In other words, our attitudes. EXPECTANCY THEORY; * Expectancy theory explains how decisions may be made in different situations and can be applied to eating behaviour. * The evaluation of any situation or object comprises of two factors: -the perceived likelihood that the object has certain attributes or may lead to certain outcomes. -the value attached has these outcomes (positive or negative). - each attribute/outcome will affect your evaluation of what to eat (e. g. an attribute may be home made/bought or high/low calories). * If this theory is true then when we are given a choice between two foods, we should choose the one with the most desirable attributes or outcomes (Conner & Armitage, 2002). parental attitudes and food preferences; Parents, usually the mother, provide food for the child. Therefore, it is obvious that the mother’s attitude to food will affect the child’s preferences. If the mother is concerned over health aspects of food she will work harder to make sure her child has a balanced diet. If the mother is less aware or less concerned over health issues such as obesity, she will take less care over the child’s diet. As expected, there is a significant correlation between the diets of mothers and children (Ogden, 2007). Parents, especially the mother, provide the key role models for the child. Once the child reaches school, peers become more important. Studies have shown that modelling using admired peers can

increase consumption of fruit and vegetables (Low, Dowey & Horne, 1998). Throughout childhood children are exposed to widespread food advertising television, using peer models, animation etc to make the food more attractive. This can be effective in developing preferences, but unfortunately advertised products tend to be high in fat and carbohydrates, probably contributing to problems such as childhood obesity. A common technique, used by many parents and based on operant conditioning, rewards consumption of a disliked food with a desired food- ‘ you can have some ice cream if you eat your vegetables’. Unfortunately, studies have shown that whilst this technique may work in the short term it increases the desirability of the reward food and decreases the liking for the non-preferred food (Ogden, 2007, Birch, 1999). Similarly, punishing poor eating habits by denying access to a desired food simply increases the preference for the desired food. cultural influences; * Eating concerns more prevalent with western women compared to eastern. * Body dissatisfaction and eating disorders become more prevalent as wealth/class demographically increases. * Food choice takes place within a network of social meanings (Ogden, 2003). Eating behaviour cannot be reduced to the biopsychology of the individual choice of foods & eating behaviours also rely on social communication, individual identity, particularly cultural identity. -for example, the forbidding of foods in certain religions- beef. Fasting is also popular in some cultures. * The consumption of food plays a central role in social interaction: -group identity, in part is created & maintained through food. -types of food are often described as traditional family meal, or the national dish (Ogden, 2003). -cultural differences will determine to an extent

what food people in a certain culture will eat and what is acceptable to eat & how foods are prepared and consumed. -food can also be used for several purposes such as to affirm or develop personal relationships & to demonstrate wealth. * Mealtimes are important for children as they learn food preferences and what is deemed acceptable/unacceptable (Bandura, 1997). * Parental eating behaviour, attitudes and child-feeding practises (determined by culture) strongly influence a child's food acceptance. * Eating or not eating certain types of food s one way in which people seek to change their body shape/size so that it conforms to social ideals. In Western societies (where since world war two, food shortages have been common) thinness in women has been increasingly portrayed as the ideal & studies indicate that over half of the 20th century women have become increasingly dissatisfied with their body image (Feingold & Marzella, 1998). * Comparature studies reveal that discrepancies between actual and ideal body weight are less pronounced in cultures where less emphasis is placed on thinness (Torrolar, 1996). social learning theory; * Three sources to model eating from: -parents. -peers. -media. * Social learning theory explains eating behaviour by suggesting that a child will copy or is influenced by figures that appear important or authoritative to the child. At a young age children become influenced by parents in terms of what they eat & when they eat. A model provided by parents is the only one available. Peers become a next port of call when children want to become more popular & therefore mimic their behaviour. * By the age of 3-4 years children's eating behaviour is no longer driven by biological need but is influenced by responsiveness to environmental cues (e. g. peers/parents attitudes as models) about food

intakes. (Skinner et al, 2002). EXPOSURE; * Repeated exposure to novel food can change a child's preferences to favour food (8-10 exposures) Birch and Marlin, 1982. * Neophobia- fear of trying new food- can change with exposure. MODELLING; * Parents- Duncker (1938) - greater change in children's food preferences if the model is mother, friend or child. * Parents- Olivera et al (2002) - correlation between mothers' and children's' food intake for most nutrients in preschool children. * Peers- Feunekes et al (1998) - 19% of food consumed by adolescents was similar to that consumed by their friends. (Milk used in coffee, alcoholic drinks & several snack foods). FOOD AS A REWARD; * Birch et al (1980) - increased preferences towards food when associated with positive adult attention. MEALTIMES; * Children who eat meals with other family members consume more healthy food and nutrients (Stanek et al, 1990). EATING AT HOME, AT SCHOOL OR AT WORK; * In 1970, only 34% of family's food budged was accounted for by foods consumed outside the home (Kant and Graubard, 2004), by the late 1990's, more than 47%. * 78% of secondary schools have a vending machine. PORTION SIZE; * Lack of knowledge of what an appropriate healthy portion size is & thus many people consumed more ' excess energy'. * Meyer and Gast (2008) ' correlation between parental influence and disordered eating' and Birch and Fisher (2000) ' mother's diet predicts daughter's. the success & failure of dieting; * According to Ogden (2007) these factors include: 1) Media influence- media images have become slimmer over the past 50 years. 2) Family- relationship between mother and daughter- body dissatisfaction and dieting. 3) Ethnicity- mixed evidence, however research does suggest eating disorders are proportional to exposure of Western

Media. 4) Social class- eating disorders seem more prevalent among the higher classes (& social groups). * Some women do lose weight and maintain that weight loss. However most women regain the weight loss and some end up eating more. * Eating is often a part of social interaction. So informing friends & family of weight loss goals should help reduce the temptations of food and encourage positive reinforcement (and punishment) from others. * Lots of dieters also join weight loss groups or diet with a friend or partner to maintain motivation. * Diets that often involve STRATEGIES (e. g. calorie counting, CBT) are effective in changing eating behaviour. * Ogden (2000) compared re-gainers, obese and maintainers: the latter endorsed a psychological model of obesity in terms of consequences. -the very fact that some people do maintain the weight loss they have achieved through dieting also suggests that restrained eating does not necessarily lead to problematic outcomes for all people in all situations. PHYSIOLOGICAL REASONS WHY DIETS DO NOT WORK; * Your body has evolved to cope with chronic food shortages by lowering your metabolism and protecting fat stores in terms of starvation- extreme dieting triggers this response. * If you then return to normal eating you end up with more excess calories than before , which are then converted to fat, to overcome the feeling of deprivation during the diet people often also overeat afterwards, which gives you an even bigger weight gain. * You may then start another, ever more restrictive diet to undo the weight gain, but this will then reduce the metabolic rate further and so the pattern of yo-yo dieting continues. PSYCHOLOGICAL REASONS WHY DIETS DO NOT WORK; * There is a lot of evidence to suggest that restrained eating can lead to overeating. * Herman et al (2005) suggest three factors why this

may be the case: -stress triggers overeating in restrained eaters (people who diet). -dieters are more likely to suffer from cravings- this may be the cause of their diet or the result of it. -restrained eaters often have a cognitive boundary for food intake. If they exceed this boundary (called pre-load) eating is disinhibited. Herman & Mack (1975); This was one of the earliest studies using the preload test method. The participants were 45 female students. They were told it was a study on taste experiences. Method; -This was an independent design with 15 participants in each of the conditions. - The first group received no preload. - The second group were given one milkshake as a preload. -The third group were given two milkshakes as a preload. -Groups two & three were asked to rate the taste qualities of the milkshake. - All participants were then given three tubs of ice creams of different flavours and given 10 minutes to rate their taste qualities. -They were told they could eat as much as they wanted. - Finally, all participants were given a questionnaire to assess their degree of dietary restraint. Results; For each group, participants were divided into either high restraint (dieters) or low restraint (non-dieters) sub groups. Herman and Mack found that low restraint participants ate less of the ice cream in the two milkshake preload conditions. This is what you predict, as they would be fuller after two milkshakes compared to one or zero preloads. However, high restraint participants ate significantly more ice cream in the one and two preload conditions than in the zero preload condition. In addition, Herman and Mack found a significant positive correlation across all participants between score on the eating restraint questionnaire and amount eaten after the two preloads, i. e. the higher the restraint score (the more the participants were

attempting to diet) the more they ate after two milkshake preloads. Conclusions; Herman and Mack conclude that the results support a boundary model of dietary restraint. -restrained eaters have a cognitive boundary for food intake. Once this is overcome (by the milkshake preload) the ‘ what the hell’ effect occurs and eating is disinhibited. So they eat more in the preload condition than the zero preload condition, the opposite pattern to normal eaters. Methodological issues; The restraint questionnaire was given after the feeding tests, so the diversion of each group into high and low restraint was what we call post hoc, i. e. after the study had been designed and carried out. Ideally, this should be done before the study, but Herman and Mack felt that giving the questionnaire before the tests would have alerted the participants to the general purpose of the experiment. This might have biased the results. It did mean that the distribution of high and low participants was different across the groups. The correlation between restraint scores and food intake after two preloads is only suggestive. Correlations cannot show cause and effect and some other variables may have produced the association for example- high restraint may be linked to low mood, and it is this that leads to increased eating. A final point is that no account was taken for individual differences. Group sizes were quite small (especially after the division of high and low participants). Some participants may have liked the ice cream more or less than others. Although there were different varieties provided to try to deal with this, it is still possible that dislike of ice cream may have biased the results. boundary model; * The model suggests that our body weight has some sort of set point and our normal food intake does not allow our body to fluctuate much between these

set points. * This is referred to as homeostasis- we eat more if weight reduces & vice versa. * Restrained eaters also set a cognitive limit on food intake (e. g. they limit the amount of calories they can eat per meal). * So restrained eaters have two potential boundaries for food intake, one from the set point and one from the cognitive boundary. WHAT HAPPENS WITH PRE-LOADING; * If a restrained eater is given a high calorie preload that will take them above the cognitive boundary, this disinhibits their feeding behaviour and they will eat until full. * This is referred to as the what the hell effect. * Frequent changing between restrained eating and overeating is thought to widen the gap between your biological boundaries of hunger and satiety, therefore making dieters less sensitive to satiety cues. * Dieters are therefore likely to eat much more than non-dieters before feeling full. EVALUATION; * The boundary model is a good example of combining physiological and psychological factors to explain eating behaviour. * However, much of the experimental work is carried out under controlled conditions in the laboratory. This gives it high levels of reliability but low levels of ecological validity. Dieting in the real world may involve more complicated issues than those studied in the laboratory. * It does not specify the cognitive and emotional (affective) processes that lead to the ‘ what the hell effect’. Other characteristics of restrained eaters have been identified which contribute to their problems maintaining a diet: * Restrained eaters (in common with women diagnosed with anorexia or bulimia) tend to become preoccupied with food and thoughts of food. They may then try to deny that food is important and suppress their thoughts about it. However, studies have shown that when instructed not to think about something we usually

think about it more. It has been demonstrated using thoughts about sex, mood and white bears (Ogden, 2007). Restrained eaters may therefore overeat as a rebound effect from trying to suppress thoughts about food. * Restrained eating is associated with lowered and depressed mood. In addition, depression is linked to low self esteem. So the restrained eaters have increased motivation to eat. However, if they violate their dietary limit, they tend to attribute it to their own useless nature and their inability to stick to a diet. So they continue to overeat following the ‘ what the hell effect’. * Dieting to reduce weight below the body weight set point is also difficult because the body will try to restore the set point in any way it can. Besides increased feelings of hunger, the body will also restore its basal metabolic rate (BMR) as weight is lost. The BMR is the rate at which cells burn energy. A lower BMR reduces energy expenditure, making weight loss difficult even on a diet. Fewer calories are being taken in, but fewer are being used up. Slow weight loss on a diet reduces the motivation to do it. Laboratory studies have shown that restrained eaters often overeat after high calorie preloads. Reviews of controlled studies dieting in the real world similarly conclude that reducing calorie input through dieting is not an effective method of losing weight (Mann et al, 2007). In the long term, between one and two thirds of dieters end up regaining more weight than they lost in the diet. Finally repeated attempts of dieting (yo-yo dieting) can in the long term increase the risks of heart disease. So research into dieting looks rather depressing. However, there is hope. Research suggests that successful weight loss is possible when combined with lifestyle changes (Powell, Calvin and Calvin, 2007). These involve low calorie diets with lifestyle changes such as: -

physical exercise. -group and individual support. -self monitoring- the person is encouraged to keep a diary and records to monitor their progress. This encourages their sense of being in control. It is also important to set realistic goals. Although on average weight loss was only 7lb on the programmes reviewed by Powell et al it was sustained over two years, and this level of weight loss has significant health benefits for the overweight. weight loss; pharmacological & surgical techniques; It is so hard to lose weight by dieting that many people turn to the use of drugs and surgery instead. Given the widespread desire to lose weight, especially amongst women, this is an extremely lucrative market for drug companies and there are several compounds on the market. * Orlistat: this drug prevents the absorption of fat from the intestine so that it is excreted rather than processed into fatty tissue. It can produce sustained weight loss (Powell et al, 2007), but it is associated with unpleasant side effects such as intestinal discomfort and oily fasces. * Sibutramine: this acts on serotonin pathways in the brain that are involved in brain regulation of food intake. Again it can lead to significant weight loss, but unpleasant side effects include increased blood pressure. Drugs are never recommended for long term use, and as they are not tackling the psychological aspects of dieting, weight gain is likely when drugs are stopped. Along with side effects their use would only be recommended when obesity becomes a serious threat to health. The weight loss drug Rimonabant has been withdrawn in Europe over fears that it leads to psychological disorders such as anxiety and depression. Surgery is recommended only for the serious or morbidly obese. The two basic types are: * Gastric band: this is a band that is placed around the stomach and

reduces its effective size. Therefore, less food needs to be consumed before the person feels full. * Gastric bypass: a tube is inserted between the top of the stomach, where the food goes in, and the duodenum. This significantly reduces the effective size of the stomach and only small meals can be eaten. These techniques mean that stomach factors in feeding are avoided and that appetite is reduced. These surgical procedures can be very effective. However, apart from the dangers of stomach surgery, they are associated with side effects such as abscesses, pneumonia and other lung infections, and have a significant mortality rate of 2-4per cent. They would only be used when obesity has become a life-threatening condition. neural mechanisms; Humans, along with all mammals are homeostatic animals. The term homeostasis is technically defined as ‘ the maintenance of a constant internal environment’. This means that we try to keep our body’s physiological consistent within narrow limits. The best example is body temperature which is regulated at 98. 6degrees. if it falls we generate heat through activity or wearing warmer clothes, and if it rises we lose heat through perspiration or wearing less. Our body is designed to operate at 98. 6degrees and it is therefore crucial that we maintain that body temperature. Our diet is essential to homeostasis as it provides the nutrients that allow physiological processes to be regulated between narrow limits. One of the most obvious signs of homeostasis is that outside of the growth phases of childhood and in conditions such as pregnancy humans regulate their body weight within reasonably narrow limits. To maintain a consistent body weight people therefore have to regulate their eating behaviour and food intake. To study this we look at the patterns in eating behaviour, e. g. do we wait until

we are hungry to start eating? How is the frequency and size of meals regulated so that long term body weight stays fairly consistent? We can find some clues in everyday behaviour. * We do not usually wait until we feel hungry before we eat. Meals, at least in prosperous countries follow a regular pattern so that hunger is anticipated and we rarely experience uncomfortable hunger. * We usually feel full or satiated at the end of a meal, which is long before the nutrients have been fully absorbed into our bodies. What signals tell the brain that enough food has passed in a given meal? * If the brain mechanisms controlling eating behaviour are so sensitive to body weight, what signals alert them to changes in body weight? THE DIGESTIVE TRACT; The aim of digestion is to break down food into its essential components such as sugar and amino acids. -the process begins in the mouth where food mixes with saliva. -saliva contains enzymes, chemicals that are vital in breaking down carbohydrates into sugars and converting proteins into amino acids. -following swallowing, food passes down through the oesophagus into the stomach where the process of digestion continues. -in the stomach food passes to the duodenum which is the first part of the small intestine. The duodenum and small intestine contain a cocktail of enzymes that complete the process of digestion. -the products of digestion, such as glucose and amino acids are absorbed into the bloodstream through the walls of the small intestine. -the waste products of digestion pass into the large intestine and are externally excreted. INSULIN AND GLUCAGON; Insulin and glucagon are two hormones released from the pancreas gland and they play a vital role in eating behaviour and body weight regulation. -Insulin controls blood glucose levels by allowing glucose in the bloodstream

to enter the cells of the body. -Glucose is the main energy source for cells and it is vital to their function. -Glucagon is stored in the liver and in muscles and along with fatty tissue; it makes up the main reserve of energy for the body. -Insulin is also crucial in allowing fats in the bloodstream to be stored as fat [or adipose]. -Adipose cells make up the fatty tissue of the body & are another key energy reserve. They are also important in determining body weight. In the condition known as diabetes, insulin levels are low. This can be due to damage to the cells of the pancreas gland that secretes insulin (type one diabetes) which usually occurs early in life. Type two diabetes occurs usually in later life when the pancreas gradually fails to secrete enough insulin. This is often associated with obesity & is far more common than type one diabetes. In the absence of insulin, levels of blood glucose rise (hyperglycaemia) and this can have potentially disastrous consequences including confusion, delirium, loss of consciousness, and, in the long term, heart attacks and blindness. Diabetes requires regular treatment with insulin, although this had to be carefully monitored. Too much insulin means that more glucose is stored within cells & therefore blood levels of glucose fall dramatically. This can also have severe consequences. dual centre model; * Each individual has a set point and their weight is regulated around that set point. * Homeostatic mechanisms ensure we alter our appetite and metabolism to keep us to this set point. * The main area involved in regulating appetite is the hypothalamus. HYPOTHALAMUS; * Patients with tumours in the hypothalamus tend to become obese. * Methods used to research the hypothalamus: -lesioning in animals. -investigation of feeding patterns after brain damage. -effects of neurotransmitters introduced into

parts of the brain. -impact of drugs on eating. * Two areas of the hypothalamus involved in eating behaviour: -ventro-media hypothalamus (VMH) as a satiety centre. -lateral hypothalamus (LH) known as the hunger centre. [VMH & LH are also known as VMN & LN, N stands for nucleus.] THE LATERAL HYPOTHALAMUS; * Contains the feeding centre. * This initiates eating. * It responds to decreased blood glucose & increase in ghrelin hormone when the stomach is empty. THE VENTRO-MEDIAL HYPOTHALAMUS; * Contains the satiety centre. * This inhibits eating behaviour when we are full. * Responds to: -an increase in blood glucose. -a decrease in ghrelin and to CCK a hormone released when food is detected in the duodenum. -leptin a long term satiety signal released by fat cells. Increase in blood glucose, decrease in ghrelin release. Increase in blood glucose, decrease in ghrelin release. Satiety-feeding stops, feelings of declining nourishment levels. Satiety-feeding stops, feelings of declining nourishment levels. VMH Satiety Centre activated. VMH Satiety Centre activated. LH feeding centre activated. LH feeding centre activated. Hunger -> eating starts. Hunger -> eating starts. ROLE OF HYPOTHALAMUS-EVIDENCE; * Aphagia (failure to eat when hungry) can be caused by damage to the LH. * Anand & Brobeck (1951)-lesions in the LH of rats leads to loss of feedings and aphagia. * Research shows that stimulation of LH of rats produces feeding. -rats who’s VMH had been lesioned developed overeating and obesity. -however, Gold (1973) found that lesions restricted to the VMH alone did not result in hyperphagia and only produced overeating when they included other areas such as the parvoventicular nucleus. -subsequent research did not support Gold’s findings. evaluation of dual process model; * Much of the research done in

support of DOM has been done on animals. This means that it cannot be extrapolated onto humans. Together with this, using animals in research is unethical as animals are unable to give consent. * The theory focuses only on biological factors, this makes it reductionist as it fails to consider other psychological approaches. For example behaviourists may suggest that life events play a large role in eating behaviour (family grievance leads to reduce in eating). * The theory only consists of the nature approach and doesn’t look at how nurture can affect eating behaviour. * Case studies used in research help to avoid ethical issues, they are not inducing disorders, only making observations. the role of the stomach; * Does the presence or absence of food in the stomach cause signals to the brain’s mechanisms controlling eating? * Cannon and Washburn (1912) found that stomach contractions correlated with hunger pangs. They inserted a tube into the throat of a participant (and into the stomach). Air was pumped into the pipe to inflate a balloon attached to the other end. Stomach contractions could then be measured by change in air pressure in the balloon. It was found that the participant was not hungry there were no contractions suggesting that stomach contractions caused hunger. (determinism). * However, subsequent research suggests stomach contractions do not necessarily have to occur for hunger to start: - people still feel hungry (or full) even if the nerves from the stomach to the brain are cut. Or even if a patient has no stomach at all! the role of ghrelin; * Ghrelin is secreted from the walls of the stomach and the amount that is produced is directly proportionate to the emptiness of the stomach. * That is- the emptier the stomach, the more ghrelin is secreted. * Recent research suggests that the hormone ghrelin plays a large part in

hunger feelings and satiety. * Evidence suggest that ghrelin acts directly on the brain’s mechanisms of feeding behaviour, including the hypothalamus. CUMMINGS ET AL (2004); Cummings et al (2004) investigated changes in blood ghrelin levels over time between meals. -Six participants were allowed to eat lunch, then ghrelin levels were monitored from blood samples taken every five minutes (from a tube or catheter inserted into the vein) until participants requested their evening meal. -Participants assessed their degree of hunger every thirty minutes. -Findings were that ghrelin levels fell immediately after lunch, reaching their lowest level at about 70 minutes. Then they slowly began to rise, peeking as participants requested their evening meal. Importantly, in five out of the six participants, ghrelin levels were closely correlated with the degree of hunger reported by the participants. The authors concluded that ghrelin levels directly reflect stomach emptiness and are closely related to subjective feelings of hunger. This supports a role given for ghrelin as a key appetite signal in humans. Methodological Issues; Participants were isolated from time and context cues so that any changes in ghrelin would be due to hunger, rather than reflecting an automatic response to meal time. There were only six participants and all were male. Therefore, there could be problems of generalising results to wider population and females in particular. Data was correlational, so we cannot say that ghrelin causes feelings of hunger. The results support previous research. Ethical Issues; The study involved inserting a catheter into the participants vein so that blood samples could be collected. The researchers obtained approval for this from their local ethical committee. Participants were volunteers who gave informed consent and who were fully

debriefed afterwards. glucostatic theory; * Glucose is a simple sugar that we get from food that gives us energy. * The glucose that is not used immediately is stored in the liver & fat cells. * Blood glucose levels are constantly monitored by the sensors in the liver and the hypothalamus- if levels are low stored glucose is released. * Changes in supply of glucose available generates signals to the brain that regulate hunger and appetite. evaluation of glucostatic theory; * Levels of energy source do not usually decrease to anywhere near the threshold needed for trigger eating. Certainly a small but reliable decline in blood glucose occurs a few minutes before the initiation of spontaneous eating (Campfield et al, 1996). This is determinable. Neural mechanisms; controlling satiation; cck; * Cholecystokinin (CCK) seems to be the hormone associated with satiety. * Does the opposite of ghrelin. * As food passes from the stomach to the small intestine through the duodenum CCK is released. * Sends messages to the brain to inhibit eating. * Smith et al (1982) injected CCK into animals and humans reduce meal size. -animals with a genetic mutation that stops CCK being released become obese. -leptin; * Body weight is determined by muscle mass & body weight. * Fat is stored in adophocytes are controlled by diet and then the number is fixed. * So fat levels controlled by the amount of fat held in the adophocytes. * Leptin is a hormone released from the adophocytes. * The more fat stored- the more leptin to be released. * Leptin travels back to the hypothalamus and signals to us to reduce food intake (feelings of satiety) * Mice missing gene for leptin production overeat and become obese (the ob gene). * Injections of leptin into ob mice reduces food intake. -However, obese humans have normal or high levels of leptin, so this cannot be the complete

answer. * Could be the brain of some obese people insensitive to leptin levels. Issues- -Ethical issues with using non human animals. Debates- is the biological approach reductionist. Free will Vs determinism (can biological drives be overridden?) Approaches- -Compare and contrast explanations for eating behaviour. neural mechanisms; evaluation; * Reflection on people's hunger/satiety. * Insight into brain chemicals- could be used to develop medical interventions to help change what we eat. * Provides explanations of some differences in eating behaviour. * Studies such as these involving legions in the LH and VMH in rats have supported the role of the hypothalamus in eating behaviour. * Studies involving electrical stimulation of these centres have confirmed their role in feeding & satiety. * Such studies provide sound scientific evidence but there is the issue of extrapolating data findings from animals to humans. * Reductionism- focus exclusively on biological factors of hunger and satiety. * Biological determinism- focuses exclusively on the role of nature and no space left to choice & social and cultural influences. * There is substantial and convincing evidence that social, cultural & psychological factors affect our eating behaviour as in evident from psychological explanations of eating disorders. * Use of animals in research implies lack of generalisability. * Highly controlled lab experiments may lack ecological validity. * Physiological drives can be overridden (e. g. desire to lose weight, dislike of certain foods, fear of losing control, social cues to continue eating, and food availability). evolutionary explanations; * The human line rapidly became omnivores. It is significant that even in modern hunter gatherer societies meat still makes up a large fraction of the diet, 20-90% depending on the season. In contrast,

chimpanzees will have a maximum of 4% in their diet. (Buss, 2008). * Evidence to suggest we have evolved to become omnivores can be noted when looking at the digestive system: -Humans have a relatively long duodenum and small intestine specialised for the digestion & absorption of protein. * Chimpanzees, gorillas and monkeys have a relatively long large intestine, specialised for the digestion of plant materials. * There are several advantages to having an omnivorous diet: -meat gives humans a lot of proteins. -it is a more efficient means of gaining protein than scrounging for plants & leaves. -hunting would require special social skills (and development of these)- tool making and tool use, weapon making and use, skills of navigation and social co-operation (this puts pressure on the evolution of language & other social skills). * These skills would provide the selection pressure for a larger brain, i. e. individuals who are capable of these complex actions would have been more likely to survive & breed. The evolution of a hunter gatherer society would have other implications: -division of labour, the physical demands of hunting would be more suited to males, while females would be more concerned with child-care & gathering berries, leaves etc. -skilled hunters would acquire prestige, in modern hunter gatherer societies the successful hunter shares his kill with other members of his group alongside his family. This gives him esteem and power in the group as-well as sexual favours and greater opportunities to spread his genes. In fact, in some modern tribes, women can divorce men who do not provide food (Buss, 2008). * However, these implications are not necessarily applicable. As food is more readily available in supermarkets & so less hunting is needed. Women are also less likely to be impressed by a man

doing the supermarket shopping than if they hunted animals themselves. * Omnivores have a wide ranging diet & can therefore be exploited to many different food sources. This leaves them open to a variety of plant toxins and food that has gone off. Meat is a major source of food poisoning even today. During evolution various methods have evolved to cope with this problem: -cooking is one solution for killing bacteria in meat (Wrangham et al, 1999). This provides an advantage to humans over other species. Cooking also makes food easier to chew, and it is noticeable that our chewing teeth (molars) have decreased in size (Lucas et al, 2006). -spices have always been a part of the human diet. Spices such as onion and garlic are especially effective in killing bacteria. We would not have a genetic tendency to use spices, but cultural transmission would quickly spread the word that they help prevent food poisoning. Today, more spices are used in hot countries and meat (Sherman and Hash, 2001) which is more dangerous if spoiled. -taste receptors help to indicate food that has gone off. These tastes lead us to pull facial expressions of disgust that is similar across human infants and other young primates, such as chimpanzees (Steiner et al, 2001) that feeling leads us to avoid those foods in the future. -food neophobia is being afraid of the new. This means that animals have a powerful tendency to avoid food that they have not yet encountered. An aspect of neophobia is that we tend to show a greater liking of foods once they become familiar (Frost, 2006), we do show a variety of choice in foods that we know are safe. -taste aversion learning, if we eat a food that makes us sick, it would be an evolutionary advantage to avoid it in the future. This can be shown in humans, but has been more dramatically shown in non-humans (Garcia, Rusiniak and Brett,

1977) made wolves sick with lamb’s meat contaminated with poison wrapped in sheepskin. When allowed to approach the wolves would sniff and leave the sheep alone. They had learnt that to associate the poison with the sheep. Taste aversion learning is highly specific and extremely intense. Just one experience will affect the animal’s behaviour. * Babies and young children can show a range of taste preferences. In some cases this is very narrow such as surviving largely on peanut butter sandwiches. Some of their dislikes are sensible, some vegetables such as broccoli and Brussels sprouts contain chemicals which can be harmful for the young (Nesse & Williams, 1994). PREGNANCY AS THE EXCEPTION; * Most females settle their diet down, but the one exception to this is females in the early stages of pregnancy. Pregnancy leads to be associated with a change in dietary needs, and in particular the avoidance of certain foods, so much so, that disgust & vomiting can be a reaction. * Morning sickness is found in at least 75% of women. An exploration of this is found in the embryo protection hypothesis (Profet, 1992). * Surveys show that foods that cause a reaction consistently are: -coffee. -meat. -alcohol. -eggs. -vegetables. (Buss, 2008). * Morning sickness is most severe in the early weeks of pregnancy when the baby’s major organs are developing and the baby is most vulnerable. * Usually it eases off after and disappears as the baby becomes fully formed. * This is because alcohol, coffee & tea all contain caffeine which can damage the baby’s major organs. * Meat, eggs & vegetables all contain bacteria (meat & eggs) toxins (vegetables) which are harmful to a developing foetus. * The sickness reaction therefore helps the mother to avoid foods that may be harmful, while vomiting helps prevent any toxins from entering the blood

stream and affecting her baby. However this may be hard to generalise to the present as standards of hygiene are higher. evolutionary explanation; evaluation; * Our biological systems for controlling food intake have a long evolutionary history, and there are many similarities between humans and animals. * Fossil evidence on changes to our digestive system (or teeth, digestive system) support hypotheses on the evolution of meat eating and the shift to a hunter gatherer society. However there is a limited amount of evidence and evolutionary explanations are speculative. * The shift to meat eating helps to explain the selective pressure on brain evolution in order to cope with new technical and social skills. * An evolutionary approach can explain many features of our digestive systems e. g. our range of taste qualities. * The evolutionary approach can also explain unusual aspects of food preferences such as morning sickness. * As part of the biological approach, evolutionary explanations are reductionist. They do not take into account cultural transmission of behaviours related to food & feeding, and they do not place enough emphasis on social and cultural changes in, for example, the widespread availability of food. In western societies we no longer behave as hunter gatherers and the evolutionary approach is therefore limited. * Very nature dominated, no appreciation of nurture. mood and eating behaviour; * Studies seeking to investigate the effect of mood and distress on eating look for the existence of either hyperphagia (excessive over eating) or hypophagia (excessive under eating). * A sustained increase or decrease in appetite can be an important symptom of depression or other mood disorders (Davey, 2008). * Ogden (2007) notes that dieters who overeat in response to low mood may be seeking to mask

their negative mood (dysphoria) with a temporary heightened mood induced by eating- a phenomenon she refers to as the “ masking hypothesis". Determinism. * Studies indicate that dieters eat more than non dieters when anxious, regardless of how palatable their food is. * There is also a considerable amount of research to suggest that certain people experience an irresistible desire to consume sweet or starchy food in response to a low mood state. This has been labelled as carbohydrate craving syndrome (can be deterministic) * It has been suggested that the eating of carbohydrates specifically triggers an improvement in mood. * One explanation for this is that carbohydrates are an important source of amino acids (tryptophan), an essential building block for serotonin. It has been suggested that people who crave carbohydrates prefer to each foods rich in carbohydrates, as a means of self medicating their low mood (reductionist) -support for this existence of carbohydrate craving is shown in a recent experimental trial by Corsica and Spring (2008). * Stress is commonly thought to occur when the perceived demands made of an individual exceed or threaten to exceed the perceived resources available to that individual to cope with such demands (Lazarus & Folkman, 1984). In this transactional model of stress developed by Lazarus & colleagues, cognitive appraisal is considered the first step in the stress response. This means the response to stress will be idiosyncratic, dependent on the primary appraisal made by the individual and their secondary appraisal of the resources available to that particular event or situation. Thus stress, like beauty is in the eye of the beholder. * Conner & Armitage (2002) observe that two general hypotheses have been investigated in relation to stress and eating behaviour. The first- the general effect hypothesis

proposes that stress changes the consumption patterns of food in general; the second- the individual difference hypothesis- emphasises that stress leads to changes in eating behaviour only in certain groups. THE GENERAL EFFECT HYPOTHESIS; * The general effect hypothesis suggests that stress produces physiological changes that explain changes in eating behaviour. * Studies to examine this hypothesis have usually entailed exposing animals; particularly rats (ethics), to physical stressors (such as tail pinching and electric shocks) and observing changes in eating behaviour. * In general such research has produced results that provide some support for this hypothesis, although not consistently so (Conner & Armitage, 2002). Isolation, a chronic stressor, does appear to increase both consumption of food and weight in rats, but studies with human participants have mixed results. THE INDIVIDUAL DIFFERENCE HYPOTHESIS; * The individual difference hypothesis suggests that differences in biology, attitudes to eating, and learning history provide variations in vulnerability to the effects of stress. * Those with a high vulnerability respond to stress by effecting an environmental or psychological change that encourages eating. * Whereas low vulnerability individuals make psychological or environmental changes that do not encourage eating (Conner & Armitage, 2002). * A number of high/low vulnerability groups have been suggested including: -external & internal eaters. -restrained & non restrained eaters. -emotional & non emotional eaters. MOOD AND EATING BEHAVIOUR; * According to externality theory (Schacter et al, 1968) people who are external eaters eat in response to food related stimuli, irrespective of whether they are hungry or full. By contrast, internal eaters respond more to hunger when deciding when &

what to eat. It is assumed that when we are stressed, our internal cues to hunger are reduced and our external cues are heightened. * If this assumption is correct, increased levels of stress should reduce eating in internal eaters, but increase eating in external eaters. Research by Conner et al (1999) provides some support for this theory. * People who use self control processes to try to suppress their intake of food are referred to by some psychologists as restrained eaters. According to restraint theory, if these self control processes are undermined by stressful events, disinhibitation of eating is more likely to occur, leading to an excessive intake of food. Certainly, the findings of a number of studies appear to support the hypothesis that stress produces a greater increase in eating in restrained eaters (Herman et al, 2005). However, as Conner & Armitage (2002) note, these findings are exclusively in relation to young women (gender bias). * Emotional eaters tend to increase their intake of food when they are anxious or emotionally aroused. By contrast, non emotional eaters do not show a response to emotion. The way in which stress is thought to lead to increased food consumption in emotional eaters is through a failure to distinguish between anxiety and hunger (Conner and Armitage, 2002). * There is also a limited number of studies investigating the impact of emotional eating on the relationships between eating and overeating: the findings of a few studies offer some support for this hypothesis (e. g. Oliver et al, 2000), whereas the findings of others do not (e. g. Conner at al, 1999). A recent study by Van Stirien and Ouwens (2009) suggests that alexithymia (that is, difficulty identifying and describing feelings), may be important in moderating factor in the link between distress and overeating. emotion and

eating behaviour; Hunger is associated with increased arousal, vigilance and irritability, while after a meal we feel calm and sleepy and have general pleasuring feelings. More strikingly studies have shown that people who are depressed or stressed increase their carbohydrate [especially sugar] and fat content of their meals, Gibson (2006). The change is associated with better mood and more energy, Macht, Gerer & Ellgring (2003). People also find that sweet tastes [as in carbohydrates like sugar] pleasurable. The effect is so widespread that two mechanisms have been proposed to account for it. THE SEROTONIN HYPOTHESIS; The serotonin hypothesis: carbohydrates such as chocolate contain the amino acid tryptophan. This is used by the brain in the manufacture of the neurotransmitter serotonin. Low levels of serotonin are associated with depression, and it has been proposed that people with stress or depression take in more carbohydrates because it leads to increased levels of serotonin in the brain. This reduces their depression [Gibson, 2006]. Unfortunately, this increase in serotonin levels only occurs when we take in pure carbohydrates, which is extremely rare. The presence of even a small amount of protein, as in chocolate, prevents the tryptophan from entering the brain, and so serotonin levels will not change (Benton, 2002). The serotonin hypothesis is unlikely to explain the antidepressant effects of high carbohydrate diets. THE OPIATE HYPOTHESIS; In the brain we also have opiate neurotransmitters. Two examples are enkephalin and beta endorphin. They are released from neurons and act as synapses with opiate receptors. Opiates are chemically very similar to the opiate drug in heroin , and heroin acts on these opiate pathways. Heroin is a highly addictive drug which can also produce pleasurable feelings and euphoria. Therefore, it seems likely

that the brain’s opiate pathways are part of our rewards system, a network of pathways that control our feelings of pleasure and reward. Our reward system is activated by natural rewards such as food and drink. If the rewarding systems of food depend on the opiate/endorphin system, then we would expect some interaction between opiates and feeding behaviour, and this is what we would find (Grigson, 2002) (Gibson, 2006): -Opiate drugs increase food intake and increase the perceived tastiness of food. -Blocking the endorphin system with the drug naxolene reduces food intake, especially sweet foods, and suppresses thoughts about food. This shows that the system is involved in feeding regulation. -Sweet foods increase the release of endorphins in the brain. So we feel better after eating sweet carbohydrates as these foods in particular activate our natural reward pathways. This effect would be more obvious in people with depression or those highly stressed, but even in normal circumstances sweet foods can improve mood. Because food is so vital we are very efficient at learning associations between taste and consequences. This applies to positive effects as well- we learn to associate the mood improving effects of carbohydrates, especially sugars with the sweet taste. So when we taste food, we have expectations about the consequences, and this applies to physiological systems as well: -Glucose reliability improves performance in cognitive tasks. However, if people are given a glucose drink but are told it is a placebo, then the effect disappears. Our expectations override the actual intake of glucose. -The sweet taste of a glucose solution immediately produces a release of insulin from the pancreas gland, anticipating a rise in blood glucose levels. This happens even when drinks are sweetened with saccharine, a compound that

is not processed by the body. However, we have learnt that sweet tastes usually mean glucose, so our body prepares itself. Anticipation and expectation on the basis of learning and experience are vital parts of feeding behaviour (Gibson, 2006). anorexia; biological explanations; GENETIC; * Anorexia may have a genetic component which makes the individual more vulnerable to developing the disorder. This is supported by observations that anorexia tendencies seem to run in families. * Family studies have shown that levels of anorexia are higher in individuals who have relatives with the disease. -this may not just be because families share the same genes but also because they share the same environments. -they are rare diseases and so studies are only done on select participants & so lack reliability. -virtually all studies have been done in western populations. Therefore lacks population validity. -nobody has suggested that genes will be the complete answer as a 100% concordance rate for MZ twins has been found (IDA). * Holland et al (1984) aimed to find the genetic contribution to AN using MZ and DZ twin pairs where one of each pair has been diagnosed with anorexia. -justification- MZ twins are genetically the same. If a characteristic is determined fully by genetics then both twins will have it. DZ twins are only as similar as brother and sister. However they do share the same environment so they act as a control for the fact that M| twins also share the same environments. -participants- 16 MZ female twin pairs and 14 DZ female twin pairs. One of each pair was diagnosed with AN. Blood analysis and ‘ physical similarity’ questionnaire were used to determine MZ twins. -findings- if one MZ twin had anorexia the other twin would have a likelihood of 55% concordance rate, for DZ twins it was 7%. -conclusions- concordance

rate for MZ twins was significantly greater for MZ twins. Findings suggest genetic involvement. Commentary -allocation was based on physical similarities of pairs, this is not a reliable method. -MZ/DZ studies assume environmental influences are the same for both twin. However MZ twins are probably treated similarly than DZ twins whilst growing up and this extra closeness may affect concordance rate. -concordance rate was only 55%, so non-genetic factors are almost as important as genetic factors. -although only few, studies do support genetic factors. -studies should be careful in fully informed consent and right to withdraw. They should make help available (after care) if it is needed. EVOLUTION; * If there is a gene that makes an individual vulnerable to a disorder then evolutionary psychologists would suggest that the behaviour associated with that disorder may carry an adaptive advantage. If this wasn't the case then individuals displaying the behaviour would fail to survive and reproduce. The Adapted to Flee from Famine Hypothesis (AFFH) suggests that anorexics tendency to be highly active and restless when starving could have been useful behaviour in the EEA response to famine. It would mean that, rather than becoming inactive as most of us would have starved, they would become active and migrate to other areas. * Guisinger (2008) suggested that anorexia nervosa is a reflection of behaviours that were adaptive in the EEA response. -there is no direct evidence for this model. It does not explain why anorexia nervosa would have seemed sensible for it to affect both men and women in the EEA. -it is also impossible to test scientifically & has to rely on a great deal of speculation. NEUROTRANSMITTERS; * An excess of the neurotransmitter serotonin may underlie anorexia. Some anorexic behaviour is very similar to

the obsessional and anxious behaviour seen in OCD sufferers. It is known that high levels of serotonin probably causes OCD behaviour, and it therefore suggested that it may be involved in anorexia. * Early studies (Kaye et al, 2005) found a reduction in levels of serotonin metabolite (inhibits serotonin) in people with eating disorder. This would suggest serotonin pathways are underactive. * The introduction of brain scanning techniques has transformed the area. In PET scans, a drug is injected that combines with serotonin receptors. It travels to the brain and binds to serotonin receptors. A brain scan is taken and the drug shows up brightly lit areas. These can be measured, and this gives us an estimate of the number of serotonin receptors in different parts of the brain. -this does not show conclusively that changes in the serotonin system CAUSE eating disorders. The loss of body weight in anorexia for example could produce alterations in our body's physiological systems. These alterations may be so profound that they persist even after the body has recovered. They are still secondary to the disease rather than cause it. * Using PET scans it has been show that there are fewer serotonin receptors in the brains of people with eating disorders (Frank et al, 2002, Kaye et al, 2005). They also show these changes are found in people who have recovered from eating disorders , i. e. they are not due to loss of body weight or other physiological symptoms associated with anorexia. (Kaye et al, 2005). NEUROANATOMY; * The hypothalamus is the part of the brain that appears to be involved in our motivation to eat and stop eating. The lateral hypothalamus is the area of the brain that is involved in feelings of hunger and motivation to eat & it is suggested that it may be damaged in anorexics. * Research has shown that damage to the lateral

hypothalamus causes loss of appetite resulting in a self starvation syndrome. (Hobel & Teitelbaum, 1968). * Davey (2008) indicates that it is unlikely that problems associated with the lateral hypothalamus are a central causal factor in anorexia. * Animal studies indicate that damage to this part of the hypothalamus result in lack of hunger. By contrast, those who suffer anorexia usually report experiencing intense feelings of hunger. -ethical problems in using animals. -hard to extrapolate data from animals onto humans. anorexia; psychological explanations; the diathesis stress model; * The diathesis stress model is commonly used to explain the development of psychological disorders (Bennett, 2005). * The word diathesis refers to an underlying vulnerability to develop a certain disorder. While the vulnerability may be biological (such as genetic predisposition), it may also refer to predispotioning factors that are largely psychosocial in origin- for example- early traumatic experience, personality traits or particular cognitive schemata. * For a vulnerable individual to develop a particular disorder, some form of precipitating stress is required. * Once a disorder has developed, any psychological explanation for that disorder needs to account for the factors that help to maintain it. * The diathesis stress model can help us to understand the development of eating disorders from a psychological perspective- it provides a framework for examining the psychosocial factors implicated in the development of these distressing (and sometimes life threatening) disorders. psychodynamic; * Psychological disorders are a manifestation of repressed emotional problems. The symptoms of eating disorders symbolise repressed conflicts & motives in the unconscious mind. Conscious: Refusal to eat. Desire to be thin. A prepuburescent body. Lack of

menstruation. Unconscious: Desire to remain a child. Traumatic sexual experiences in childhood. Fear of adult sexuality. * Bruch (1973)- Anorexia seen as an attempt by the individual to exert some sort of control and be autonomous. * Crisp (1980)- Anorexia an attempt by girls to remain as children and postpone adulthood. * Minuchin et al (1998)- girls develop anorexia as a means of diverting the attention from other family patterns. They suggest that families of anorexics have one of the following problems: -enmeshment (parents over involved in children's affairs). -overprotection. -rigidity- need to maintain status quo in family. -conflict avoidance/lack of resolution. * Wonderlich et al (1996). -Survey of 1099 American women. -Sexual experiences during childhood. -Women with a history of sexual abuse had elevated risk of eating disorders. -Some issues surrounding retrospective data, but has been confirmed in other studies. GENERAL CRITICISMS; * Some support, mainly from women abused in childhood. * However, not all sufferers develop eating disorders and not all eating disorder sufferers have been abused. * Many aspects difficult to test e. g. unconscious motives, repressed memories. * Difficult to determine if the problems within families are a cause, or an effect of anorexia. * Over-reliance on case studies which cannot be generalised. * However- family systems therapy has been effective as part of the treatment for anorexia. behavioural; * Psychological disorders are the result of abnormal learning experiences. * Eating disorders are a set of learned emotional and behavioural responses to food, eating and behaviour. CLASSICAL CONDITIONING; * A learned association between eating and anxiety. OPERANT CONDITIONING; * Reinforcement of dieting/weight loss behaviours. -compliments from other (positive

reinforcement) -avoidance of e. g. bullying (negative reinforcement). SOCIAL LEARNING THEORY; * Observation of slim role models being admired/successful (vicarious reinforcement). * Observation of overweight people being punished. * Tendency to imitate in order to obtain some reinforces and/or avoid some punishments. EVIDENCE; * Stregal, Moore & Bulik (2007) argue: -girls internalise culturally defined standards of beauty. -in some girls this creates tension between the real self and the idealised self. -this leads to dissatisfaction and dieting. -in some vulnerable girls this leads to an eating disorder. -the process may be helped by social learning and reinforcement. * Anorexia is identified within all cultures but it is more prevalent in cultures where the western lifestyle is portrayed through media images. * Groesz et al (2002)- meta analysis of 25 studies- concluded that body dissatisfaction increased with media images of thin women. * Becker et al (2002)- the introduction of western tv into Fiji lead to a significant increase of eating disorders over five years. cognitive; * Several cognitive factors are associated with eating disorders including obsessive thoughts, inactive judgements & rigid thought patterns. * Cognitive models of eating disorders suggest that the sociocultural influences are translated into behaviour through cognitive processes. * Central to such models is the concept of weight based schemas, this is, organized patterns of thinking about weight in which self worth is judged on the basis of body weight & shape. Once such schemas are established they distort an individual’s subsequent perception and interpretation of experience. * As a consequence, the maintenance of self worth becomes increasingly dependent upon weight control (Bennet, 2005). Studies have certainly found that negative self beliefs are

characteristic of individuals with anorexia (Cooper, 2005). * Individuals with eating disorders often report experiencing obsessive thoughts about food and eating, body weight & shape. Although many people try to suppress these thoughts, many report that they are not successful in doing so and have little control over their obsessions. * Some individuals, especially those with anorexia report deriving comfort from such preoccupations (Polivy & Herman, 2002). * Cooper (2005) found people with eating disorders experience disturbances in informational processing with regard to food and eating, weight and body shape. The use of a modified stroop test has revealed attentional biases in individuals with anorexia , with greater attention given to words related to food, weight & body shape. * Individuals with anorexia nervosa exhibit a bias for food words (Polivy & Herman, 2002). Studies have also found evidence of memory biases in individuals with eating disorders- words related to food, weight and body shape are more likely to be recalled (Cooper, 2005). * The need for control is expressed by individuals with anorexia and bulimia. However, individuals with anorexia are more capable of sustaining long term control over their eating than those with bulimia, who tend to be more impulsive. (Fairbaum, 1997). * Fairbaum and colleagues suggest that in anorexia nervosa, once attempts to restrict eating begin, they are reinforced by three main feedback loops which helps the eating disorder to become self perpetuating (Fairbaum et al, 1998). -the first feedback loop is an enhanced sense of being in control derived from dietary restrictions (reductionist). This is a positive reinforce of restrained eating behaviour, and control over eating gradually becomes an indicator of self control in general and self worth. -the second feedback loop comprises

the various physiological and psychological changes associated with self starvation that encourage further dietary restriction. For example, intense hunger may be interpreted as a perceived threat to control over eating thereby motivating an individual to restrict their food intake still further. In effect these changes act as negative reinforcers of further restrained eating.

-the third and most culturally specific mechanism is concerns about body shape & weight that derive from the social value attached to thinness (and the resultant imperative to avoid fatness) in western societies.