AO1: Include keywords, facts and explanation about the topic	AO3: State whether supporting/contradicting evidence; Use research evidence – what has been found? What does this suggest? Link to research methodology or issues and debates
Neural Influences **The Limbic System**: area of the brain that helps co-ordinate behaviours that satisfy motivational and emotional urges, such as aggression and fear. **The Amygdala**: responsible for quickly evaluating the emotional importance of sensory information and prompting a response. If stimulated electrically, an animal responds with aggression: snarling and adopting aggressive posture. If the same areas are surgically removed, the animal no longer responds to stimuli that would have previously led to rage. **Kluver and Bucy (1937)** destruction of the amygdala in a monkey who was dominant in a social group caused it to lose its dominant place in the group. **The Hippocampus**: involved in formation of LTM, so allows an animal to compare the conditions of current threats with similar past experiences. For example, an animal previously attacked by another animal would respond with either fear or aggression in their next encounter. Impaired hippocampus function prevents the nervous system from putting things into a relevant and meaningful context, so may cause the amygdala to respond inappropriately to sensory stimuli, resulting in aggressive behaviour. **Boccardi et al (2010)** habitually violent offenders exhibited abnormalities of hippocampal functioning. **Serotonin**: in normal levels, exerts a calming, inhibitory effect on neuronal firing. It typically inhibits the firing of the amygdala, which controls fear, anger and other emotional responses. Low levels remove this inhibitory effect, so individuals are less able to control impulsive and aggressive behaviour – Serotonin Deficiency Hypothesis. When the amygdala is stimulated by external events, it becomes more active, causing the person to act on their impulses, making aggression more likely. Serotonin in thought to reduce aggression by inhibiting responses to emotional stimuli that might otherwise lead to an aggressive response. Low levels of serotonin have been associated with an increased susceptibility to impulsive behaviour, aggression and even violent suicide. **Mann et al (1990)** found that *dexfenfluramine* treatment in males was associated with an increase in hostility and aggression scores.	**Neural Influences** P – Support for the role of the amygdala in aggression in males. E – **Pardini et al** found participants with lower amygdala volumes exhibited higher levels of aggression and violence and remained even after other confounding variables were controlled. E – This supports the role of the amygdala in aggression as a lower volume of the amygdala is associated with less control over the activity in it, making it much easier for a person to engage in aggressive behaviour. L – This increases the validity of the claim that the amygdala has a role in aggressive behaviour as it supports the idea that a reduction in the size of the amygdala would cause aggression. [Supports Kluver and Bucy's study] P – Support for the role of the hippocampus in aggression. E – **Raine et al** found that the hippocampi of the two hemispheres in unsuccessful psychopaths differed in size, an imbalance thought to arise in early brain development. E – This supports the role of the hippocampus in aggression as it suggests that the abnormal development of the hippocampus produces aggressive behaviour. L – Therefore, this explanation is biologically reductionist, as it does not acknowledge the role of aggressive role models in the display of aggression. [Supports Boccardi's study] P – Mixed evidence for the serotonin deficiency hypothesis. E – **Duke et al** found a small inverse relationship between serotonin levels and aggression, anger and hostility. They also found only other-reported aggression was positively correlated to serotonin functioning. E – This supports the serotonin deficiency hypothesis because it shows that it has some relationship with aggression. However, it also suggests that the relationship between serotonin deficiency and aggression is complex because Duke et al found this relationship only in one group. L – Therefore, this explanation is biologically reductionist, as it oversimplifies the relationship between serotonin and aggression.

AO1: Include keywords, facts and explanation about the topic	AO3: State whether supporting/contradicting evidence; Use research evidence – what has been found? What does this suggest? Link to research methodology or issues and debates
	Supports: P – A support for the frustration-aggression hypothesis is that it can be used to explain mass killings in the real world. E – **Staub (1996):** mass killings are often rooted in the frustration caused by social and economic difficulties within society. These frustrations lead to scapegoating and then discrimination and aggression against that group. E – This supports the frustration-aggression hypothesis because it shows that the central concepts can be generalised to explain aggressive behaviour in the real world. L – Therefore, it is **valid** because it shows that the frustration-aggression hypothesis is a relevant explanation for aggression in modern times, **increasing its temporal validity.** P – Another support is that it can be used to explain sports violence in the real world. E – **Priks (2010):** supporters were more likely to fight with opposition supporters when the team performed worse than expected. A one-position drop in the league also led to a 5% increase in unruly behaviour. E – This supports the unjustified frustration explanation, as it shows that individuals who feel that the outcome of the situation is unjustified will express much higher levels of aggression. L – Therefore, the explanation is valid, as it can be used to develop safety measures at football events to reduce the levels of aggression directed at football players, which will have positive effects on the economy, as fewer individuals will get into trouble with the police for their aggressive behaviour.

AO1: Include keywords, facts and explanation about the topic	A03: State whether supporting/criticising evidence; Use research evidence – What has been found? What does this suggest? Link to research methodology or issues and debates
Food Preference: refers to the way in which people choose from among available foods on the basis of biological and learned perceptions such as taste, health characteristics, value, habit, etc. **Learning:** is the acquisition of knowledge, skills or habits through experience, observation or teaching.	In contrast to evolutionary explanations, learning explanations focus on cultural and environmental influences on food preferences.
Social Influences: Parental Influences: observation of eating behaviour and attitudes of parents can lead to children acquiring these behaviours. **Brown and Ogden (2004)** reported consistent correlations between parents and their children in terms of snack food intake, eating motivations and body dissatisfaction. Parents may also manipulate the availability of certain foods, either as reward or because of perceived health gain. One food may be an offer of reward to another: 'You can't have any ice cream unless you eat some fruit'. **Birch et al. (1984)** has generally shown that although the preference for the food used as the reward increases, there tends to be a decrease in the preference for the distasteful food.	1. Criticising evidence for parental influences is provided by **Russell et al. (2015)** They found that not all methods used for influencing food preferences in children, aged 2-5 years, were effective. Parents with healthy eating preferences were likely to influence their children to eat healthily and vice versa. This supports the parental influences explanation for food preferences, however, it shows that some approaches used to influence children are more effective than others. Therefore, this explanation is environmentally deterministic because it only acknowledges the learning aspect of acquiring certain food preferences; it does not account for the cognitive factors that play a role in which foods a person wants to eat.
Peers: Social learning theory emphasises the impact of observing other people on our own attitudes and behaviours. Behaviour of same-age peers has had a powerful influence on the food preferences of children. **Greenhalgh et al. (2009)** found that the observation of peers had both positive + negative effects on food preferences. Children exposed to positive modelling were more likely to try these foods. Negative modelling inhibited novel food consumption. **Birch (1980)** showed how exposure to another child could change food preferences. For 4 consecutive lunchtimes, children were seated next to children who have different vegetable preferences. These children showed a change in their vegetable preference that was still evident at a follow-up several weeks later.	2. Supporting evidence for the role of peers in the influence of food preferences is provided by **Wardle et al. (2005)** They found that parental fruit and vegetable consumption was a strong predictor of children's fruit and vegetable consumption. The increase of this type of behaviour in social facilitation serves as a way of ensuring we are consuming foods that are demonstrated to be safe by others. This supports the role of peers explanation for food preferences because it shows that observing other people has an impact on our own food preferences in the presence of peers. Therefore, this explanation supports the nurture side of the nature-nurture debate because this explanation relies heavily on the social learning theory which suggests behaviours are learnt through observation, proposed by Bandura.
Cultural Influences: Media Effects: The role of social learning is also evident in the impact of TV and other media on food preferences. **MacIntyre et al. (1998)** found that the media have a major impact both on what people eat, and also their attitudes to certain foods. However, many eating behaviours are limited by personal circumstances such as age, income and family circumstances. People thus appear to learn from the media about healthy eating but must place this information within the broader contexts of our lives. This led chef Jamie Oliver to set up his BBC 'Sugar Rush' campaign in 2015 to inform the British public of the dangers of too much sugar in out diet.	3. Another criticism of the parental influence explanation for food preferences is that they are ethnocentric. Criticising evidence is provided by **Robinson et al. (2001**) who studied 800 8-9 year old children from a number of different backgrounds. They found a complex association between behaviour of parents and food preferences of children, with girls being more influenced by parental modelling than boys. This criticises the parental influence explanation for food preferences because it assumes that all cultures are the same, meaning that this view is seen as universal, whereas this is not the case.
The Context of Meals: 'grazing' rather than eating meals and the desire for convenience foods are increasingly common. As a result, people learn to rely on takeaway meals as a way of feeding themselves. **Maguire et al. (2015)** found in the UK, the number of takeaway restaurants had risen by 45% in the last 18 years, with areas of high deprivation seeing the highest rise. **Gillman et al. (2000)** commented on the decline of the family meal in Western cultures, with more young people choosing to eat while watching TV. Eating meals in front of the TV was associated with greater consumption of pizza and salty snacks and less consumption of fruit and vegetables. Suggested that eating 'informally' leads to preference of quickly prepared snack foods, rather than meals. Parents whose children watched more TV tended to choose foods that were easy to prepare, as children ate them without	4. There is a real-life application to media effects on the influence of food preferences in children. Supporting evidence is provided by **Cairns et al. (2013)** who observed countries developed regulations concerning unhealthy food advertising on TV. They typically focus on limiting the quantity of the advertising of unhealthy foods, so that it reduces the effect of such advertising on children's food preferences. This supports the view that the media has an effect on the food preferences of children because it can easily change their attitudes and behaviours towards healthy food. This can be linked to **psychology and the economy** – if more people develop unhealthy eating preferences, it would cause them to have health problems. This influx of health problems, such as obesity and diabetes, could increase the amount of money the NHS would need to diagnose and treat them.

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Modelling and Reinforcement: **Modelling** – A form of learning where individuals learn a particular behaviour by observing another individual performing that behaviour. Models may be parents or peers, or 'symbolic' models, such as someone portrayed in the media. Models provide examples of attitudes to food or dieting behaviour that can be observed by the individual and imitated by them. **Reinforcement** – Term used to refer to anything that strengthens a response and increases the likelihood that it will occur again in the future. Positive response: makes individual feel better about themselves and makes them want to continue. (losing weight) Vicarious reinforcement: expect the same response from others. (so continue to lose weight).	1. On one hand, there is research support for the peer influence explanation of the development of AN through social learning. **Costa-Font and Jofre-Bonet** found individuals who had peers with a larger BMI had a lower likelihood of subsequently developing an eating disorder such as AN. This association between BMI and the likelihood of becoming anorexic was even greater in younger women. Therefore, this supports the role of social learning in the development of AN because it shows that adolescents/ younger individuals are much more susceptible to peer influences, especially their BMI, which increases the validity of this explanation. Thus, this explanation is deterministic, because it suggests that AN will always be caused by peer influences. 2. A criticism of the maternal role models explanation for the development of AN is that results from studies have not always been consistent. **Ogden and Steward** found that, although mother and daughters were similar in their weight and BMI, there was no associations for their restrained eating or body dissatisfaction. Therefore, this criticises the maternal role models explanation of AN, as the explanation suggests the transmission of eating disorders would be consistent between mothers and daughters, which decreases the validity of the explanation. Thus, this explanation is reductionist, because it does not acknowledge that there may be other risk factors for the development of AN. 3. On the other hand, a criticism of the peer influence explanation is that research does not always show a significant relationship between peer influence and the development of AN. **Shroff and Thompson** found no correlation among friends on measures of disordered eating in an adolescent sample. Although Jones and Crawford found overweight girls and underweight boys were more likely to be teased, these gender differences did not emerge until adolescence. Therefore, this criticises the role of peers as an explanation of AN because it shows that gender-based ideals have a mush larger impact on adolescents than in children, which decreases the validity of this explanation. Thus, this explanation is deterministic, because it suggests that AN will always be caused by teasing, whereas research has not continually supported this claim. 4. In addition, a support for the social learning explanation for AN is that research supports the role of media influences. **Becker et al.** found that, after exposure to TV, girls stated a desire to lose weight in order to become more like Western television characters. However, **Yamamiya et al**. shows that instructional intervention prior to media exposure to idealised female images prevents the adverse effects of exposure. Therefore, this supports media influences because it shows that media can be a powerful influence on the way individuals perceive their body image, and their likelihood of developing eating disorders, which increases the validity of this explanation. Thus, this explanation is gender-biased, because these studies were mainly conducted on girls, not boys, which suggests that these results must be true for males as well – this research has a beta bias.
Maternal Role Models: Research has spotlighted the mother-daughter relationship, with some researchers suggesting that mothers 'model' weight concerns for their daughters. **Hill et al**. found similarities between mothers' and daughters' restraint and dieting behaviours among children as young as 10 years old. **Smolak et al**. mothers who complain about their own weight are more likely to have children who have their own weight concerns. These influences tend to be greater for daughters than they are for sons, and mothers are more influential than fathers in this form of social learning.	
Peer Influences: Particularly influential in adolescence, so adolescents may be especially susceptible to peer influence on patterns of disordered eating. **Eisenberg et al.** US study – found dieting among friends was significantly related to unhealthy weight control behaviours, such as the use of diet pills or purging. **Jones and Crawford**: found overweight girls and underweight boys were most likely to be teased by their peers, suggesting that through teasing, peers serve to enforce gender-biased ideals. **Gravener et al.** examined association between peer dieting and thrive for thinness in over 2000 men and women of 3 age groups. Found significant associations between perceived peer dieting and a drive for thinness in both men and women. Women: association was strongest in late adolescence ad for same-sex peers. Men: strength of association did not differ by age group or the sex of dieting peers.	
Media Influences: **Jones and Buckingham**: show that individuals with low self-esteem are more likely to compare themselves to idealised images portrayed in the media. Low self-esteem plays a part in the development of eating disorders, such as AN, and the portrayal of abnormally thin women in the media can contribute to this**. Button et al**. found girls with low self-esteem at age 11-12 were at significantly greater risk of developing an eating disorder at age 15-17. **A report by the BMA** expressed concern about the use of very thin models to advertise products and model clothes for the fashion industry. Found image of very slim models in the media were in stark contrast to he body size and shape of most children and young women who are becoming increasingly heavier. Concluded the degree of thinness exhibited by these models is both unachievable and also biologically inappropriate and provides 'unhelpful role models for young women.' Study by *Health* magazine: concluded, in the USA, 32% of female characters on TV are underweight, compared to just 5% of the female audience.	

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The Nature of Sexual Selection: In 1871, Charles Darwin developed the theory of sexual selection. This theory explains the evolution of characteristics that confer a reproductive advantage as supposed to a survival advantage. In sexual selection, an individual's survival is not at stake, but rather it is their ability to leave more offspring.	**Definitions**: **Sexual selection**: A key part of Darwin's theory explaining how evolution is driven by competition for mates, and the development of characteristics hat ensure reproductive success.
Intra-sexual Selection: Individuals of one sex must outcompete other members of their sex in order to gain access to the members of the other sex. Successful individuals are able to mate and so are able to pass on their genes. Whatever characteristic leads to success in these same-sex contests becomes more widespread in the gene pool by virtue of the reproductive advantage this gives to the winners.	1. Criticising evidence for Buss' study has been provided by **Buller. (2005)** He argues that the majority of studies attempting to determine female mate preferences have been carried out on female undergraduates, who expect to achieve high education and expect a high income. This is a criticism because it reinforces a gender bias between what females and males prefer in a potential partner. This suggests that all women prefer the same characteristics in a partner, whereas that is not the case in real-life.
Inter-sexual Selection: Members of one sex evolve preferences for desirable qualities in potential mates. Members of the opposite sex who possess these characteristics will then gain a mating advantage over those who do not. The preferences of one sex therefore determine the areas in which the opposite sex must compete.	2. Supporting evidence for Buss' study is that it took place across 37 different cultures. This is because it takes into account all cultural factors that could affect the study. This means that Buss' study has cultural relativism, as looking at different cultures together can ultimately find universal criteria for what each sex looks for in a long-term partner across the world. (which he did find in the study) This increases the validity of the study as it allows psychologists to take a nomothetic approach to sexual selection.
Sexual Selection and Long-term Mate Preferences: The rationale behind sexual selection is that random mating is essentially unbeneficial for future generations. It is beneficial to be 'choosy' as the genetic quality of a mate will determine half of the genetic quality of any offspring: LOW-QUALITY MATES → UNATTRACTIVE, UNHEALTHY OFFSPRING HIGH-QUALITY MATES → ATTRACTIVE, HEALTHY OFFSPRING Buss' research explored sex differences in long-term mate choice and found universal trends in male and female preferences.	3. Criticising evidence for sexual selection has been provided by **Nettle and Clegg. (2006)** They compared a sample of British poets and artists and a control group of males in non-creative professions. They found that males in the creative professions tended to have more sexual partners and their creative output positively correlated with their number. This criticises the evolutionary explanation for partner preferences as it proves that there are some human traits that serve no survival purpose that have evolved as a result of sexual selection. This suggests the evolutionary explanation can be interactionist, as biological factors and personality-type determine which members reproduce.
Buss (1989) – KEY STUDY **AIM**: To investigate the characteristics males and females look for in a long-term partner. **PROCEDURE**: Involved 10 000 people from 37 different cultures. Participants were asked to rank 18 characteristics on how important they would be in choosing a mate. A 4-point scale was used, ranging from '3' (indispensable) to '0' (irrelevant). **FINDINGS**: Among the main results were: 1. **Resources**: women more than men desire 'good financial prospects'. A desire for men with resources or, qualities such as ambition or industriousness. 2. **Physical attractiveness**: men placed more importance on attractiveness – cue to health and fertility in potential mates. 3. **Youth**: men universally wanted mate younger than them – value of increased fertility.	4. Criticising evidence for the evolutionary explanation for partner preferences has been provided by **Bernstein. (2015)** He pointed out that gender differences in mate preference patterns may be due to cultural traditions. The fact that women have been denied political and economic power in many cultures might cause the tendency for them to rely on men for security and economic stability. This is a criticism of the evolutionary explanation for partner preferences as there may be other factors, such as socio-economic factors that may influence partner preferences in different cultures. This makes the evolutionary explanation deterministic, as it assumes only biological factors influence partner preferences.

AO1 : Include key words, facts and explanation about the topic

CASE STUDY:
Evidence for a 'free-running' circadian rhythm comes from a series of studies conducted by the French cave explorer, Michel Siffre. He has subjected himself to long periods of time living underground in order to study his own circadian rhythms. Living underground: no external cues to guide circadian rhythms – no daylight, no clocks or radio. He woke, ate and slept when he felt it was appropriate to do so. Only thing influencing his behaviour was his internal 'free-running' circadian rhythms. First underground stay of 61 days in southern alps in 1962 – resurfaced on 17 September believing date was 20 August. Second: 6 months in cave in Texas, - natural circadian rhythm settled to just over 24 hours, with some dramatic variations. Then in 1999: interested in effects of ageing on circadian rhythms – body clock ticked more slowly compared to when he was a young man – sometimes stretching his circadian rhythms to 48 hours.

The Sleep-Wake Cycle:
Circadian rhythms not only dictate when we sleep, but when we are awake. Light/darkness: external signals determine when we feel need to sleep and when to wake up. Also dips and rises at different times in day, so strongest sleep drive occurs in two 'dips,' between 2-4am and between 1-3pm. Sleepiness experienced during circadian dips less intense when we have sufficient sleep and more intense when we are sleep deprived. Sleep and wakefulness are also under homeostatic control. Awake for long periods: need for sleep is increasing – amount of energy used up during wakefulness. Drive for sleep increases gradually throughout day – maximum in late evening – most people fall asleep. Keeps us awake as long as there is daylight, prompting us to sleep as it becomes dark. Homeostatic system: makes us sleepier as time goes on throughout waking period, regardless of night and day. Internal circadian 'clock' described as 'free-running' – a cycle of 24-25 hours is maintained, even in absence of external cues. Intolerant of major alterations in sleep and wake schedules – causes biological clock to become completely out of balance.

Other Circadian Rhythms:
Core body temperature: lowest – 36 degrees Celsius – 4:30am – highest – 38 degrees Celsius – 6pm. Normal circadian rhythm: sleep – core body temperature begins to drop – body temperature starts to rise during last hours of sleep – feeling of alertness in morning. Small drop in body temperature – in people between 2pm and 4pm – many people feel sleepy in early afternoon.
Hormone production: Production and release of melatonin from pineal gland – peak levels occurring during hours of darkness – activating chemical receptors in brain – melatonin encourages feelings of sleep – dark: more melatonin is produced – light: production of melatonin drops and person wakes.

AO3: State whether supporting/contradicting evidence; Use research evidence – What has been found? What does this suggest? Link to research methodology or issues and debates

1. Firstly, there is support for the importance of light in the sleep-wake cycle. **Hughes** tested the circadian hormone release in four participants stationed at the British Antarctic Station. He found, at the end of Antarctic summer, cortisol levels followed a familiar pattern, reaching the highest point as the participants awoke, and their lowest as the participants as they returned to bed. After three moths of darkness, the peak levels of cortisol were at noon rather than as the men awoke. Therefore, this supports the importance of light in the sleep-wake cycle because the variations in light available in polar regions and around the world can cause variations in hormone production. Thus, this explanation is androcentric, because the study was conducted on four male participants, which does not acknowledge that there are gender differences in the sleep-wake cycle.
2. On one hand, a criticism of the sleep-wake cycle is that there are individual differences. **Duffy et al** found that 'morning-people' prefer to rise early and go to bed early - 6am and 10pm – whereas 'evening-people' prefer to wake and go to bed later – 10am and 1am. Therefore, this criticises the sleep-wake cycle because it suggests that individuals are innately different when it comes to when their circadian rhythms reach their peak. Thus, this explanation is reductionist, as it oversimplifies circadian rhythms to be the same in all people.
3. On the other hand, a support for circadian rhythms comes from a real-life application – chronotherapeutics. The risk of heart attack is greatest during early morning hours after awakening. Chronotherapeutic medications have been developed with a novel drug delivery system. **Evans and Marian** found these medications can be administered before the person goes to sleep, but the actual drug is not released until the vulnerable period. Therefore, this supports because circadian rhythms can be used to effectively prevent heart attacks and other medical conditions. Thus, this explanation can be linked to **psychology and the economy** – using chronotherapeutic medications would increase the average life expectancy for individuals in the UK. This increases the validity of circadian rhythms.
4. Finally, a criticism for circadian rhythms is that the research methodology is flawed. In most studies, participants are isolated from variables that might affect their circadian rhythms, such as clocks, radios, and daylight. However, they were not isolated from artificial light because It was generally thought that dim artificial light would not affect circadian rhythms. **Czeisler et al** altered participants' circadian rhythms down to 22 hours, and up to 28 hours, by using dim artificial lighting alone. Therefore, this criticises because artificial lighting acted as an confounding variable, which means the results are unreliable.

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Endogenous Pacemakers - The Superchiasmatic Nucleus: The SCN lies in the hypothalamus. It plays an important role in generating the body's circadian rhythm. It acts as the 'master clock' with links to other brain regions that control sleep and arousal, and has controls other biological clocks throughout the body. Neurons within the SCN spontaneously synchronise with each other, so that their target neurons in sites elsewhere in the body receive correctly time-coordinated signals. These peripheral clocks can maintain a circadian rhythm, but not for very long, which is why they must be controlled by the SCN. Possible because of SCN's built-in circadian rhythm, which only needs resetting when external light levels change. SCN receives information about light levels via the optic nerve. Happens even when our eyes are shut, because light penetrates the eyelids. Slow biological clock - morning light automatically adjusts clock, putting rhythm in step with outside. SCN regulates manufacture and secretion of melatonin in pineal gland via interconnecting neural pathway. **The Pineal Gland**: SCN sends signals to pineal gland – increase production and secretion of melatonin at night; decrease as light levels increase in the morning. Melatonin - induces sleep by inhibiting brain mechanisms promoting wakefulness. Pineal and SCN function jointly as endogenous pacemakers in the brain. Sensitivity of pineal gland and SCN to light, and role of melatonin in controlling sleep and activity, mean, despite endogenous nature of these clocks, their activity must be synchronised with the light-dark rhythm of world outside.	1. A support for endogenous pacemakers shows that the SCN is important in controlling circadian rhythms. **Morgan** found hamsters who were transplanted with abnormal SCN neurons displayed the abnormal circadian rhythm of 20 hours. In the reverse experiment, the transplant of normal SCN neurons into the brain of abnormal hamsters caused their circadian rhythm to change to a pattern of 24 hours. This suggests that the SCN is important in maintaining the circadian rhythms of humans and supports the idea that it has control over the other biological clocks in the body. This provides an evolutionary explanation to biological rhythms, as this study was conducted on hamsters. This suggests that this characteristic promoted the survival and reproduction of humans, and thus was naturally selected. However, a more accurate explanation would be the diathesis-stress model. There is a biological vulnerability, such as being born with genes for the abnormal development of the SCN. However, not everyone who possesses these genes displays an abnormal circadian rhythm, which suggests the expression of these genes depends on the environment, which is a form of stressor that triggers the condition. Therefore, this explanation emphasises the role of nature, but ignores the role of nurture.
Exogenous Zeitgebers – Light: Receptors in SCN are sensitive to changes in light levels during day and use this information to synchronise activity of the body's organs and glands. Light – resets internal biological clock each day, keeping it on 24-hour cycle. Rods and cones in the retina of the eyes detect light to form visual images. 3rd: a protein called melanopsin, which is sensitive to natural light, is critical in this system. A small number of retinal cells contain melanopsin and carry signals to the SCN to set the daily body cycle. **Social Cues: Aschoff et al (1971)** showed individuals are able to compensate for the absence of zeitgebers such as natural light by responding to social zeitgebers instead. **Klein and Wegmann (1974)** studied jet lag and found the circadian rhythms of air travellers adjusted more quickly if they went outside more at their destination. Thought to be because they were exposed to social cues of new time zone, which acted as a zeitgeber. Circadian rhythms of blind people were thought to be no different to sighted people as both groups were exposed to the same social cues. Both examples can now be better explained in terms of light exposure acting as a zeitgeber. Sleep-wake cycle: blind people - still influenced by light during the day, even though they have no visual perception. Connections exist between the eye and the SCN that do not involve those parts of the visual system on which the perception of light depends.	2. A support for exogenous zeitgebers in circadian rhythms is that light can also set the circadian rhythms in blind people. **Skene and Arendt** estimate the vast majority of blind people who still have some light perception have normally entrained circadian rhythms. This suggests that the pathway from retinal cells containing melanopsin are still intact. Further evidence shows that people without light perception have abnormal circadian patterns. This supports the role of exogenous zeitgebers in the maintenance of circadian rhythms, because it shows that light and melanopsin play an important role in relaying information to the SCN. This makes this explanation biologically deterministic, because it suggests only the inability to perceive light will cause abnormal circadian rhythms. Thus, this explanation ignores the fact that there are social zeitgebers that can compensate for the absence of other zeitgebers, such as natural light. In addition, there is never 100% concordance rate between MZ and DZ twins, which means that genes do not entirely determine our behaviour – there are environmental zeitgebers which can also reset circadian rhythms. Therefore, this explanation emphasises the role of biology, but ignores the role of social cues in resetting circadian rhythms.