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| **Neural and Hormonal Mechanisms in the control of Eating Behaviour** | | | |
| **Neural and Hormonal Mechanisms AO1** | | | |
| **The role of the hypothalamus**  The hypothalamus, a pea sized structure of the brain, has a crucial role in integrating the nervous and endocrine systems. It is also involved in maintain homeostasis, the balancing of bodily processes within certain limits. This biological mechanism regulates the level of glucose in the blood. Glucose is the most basic of sugars and the body’s main source of energy. Fluctuations in blood glucose concentration are first of all detected by glucose sensing neurons in the hypothalamus. The hypothalamus can then regulate glucoses levels by influencing the output of insulin and anti-insulin hormones, e.g. glucagon. Both insulin and glucagon are secreted from the pancreas, and play a key role in maintaining blood glucose homeostasis within a very narrow range of values. | | | |
| **The dual-centre model of eating**  According to the dual centre model of eating behaviour, two structures of the hypothalamus provide the homeostatic control.  Lateral hypothalamus (LH): Often described as the ‘feeding centre’ or ‘on switch’ of the hypothalamus, the LH contains cells that detect levels of glucose in the liver. The LH is activated when glucose levels fall below a certain level. This causes the individual to become hungry, and triggers the motivation to eat, along with all its accompanying behaviours such as searching for and preparing food.  A further neural mechanism associated with LH activity is the secretion of a hypothalamic neurotransmitter called neuropeptide Y (NPY), which is closely associated with hunger and a reduction in physical activity. NPY is a powerful stimulant of hunger. Rats injected with NPY directly into the hypothalamus will eat excessively, and eventually become obese as the injections continue.  Ventro-medial hypothalamus (VMH): This part of the hypothalamus is the ‘satiety’ centre’, the ‘off switch’ of eating behaviour. Eating food provides the body with glucose, so the levels of glucose circulating in the bloodstream and stored in the liver (as glycogen) rise once again. These levels are detected by cells in the VMH. Activity in the VHM is then triggered once they increase past a set point. LH activity is inhibited at the same time. The individual becomes satiated; they feel full and stop eating.  Damage to the VMH is linked with continued eating past the point of satiety. Alexander Reeves and Fred Plum (1969) reported the case of a woman whose weight more than doubled in a two-year period. A post-mortem investigation revealed that she had a tumour on her VMH, which caused its normal ‘stop eating’ function to fail. | | | |
| **The role of ghrelin**  Ghrelin is a hormone secreted by the stomach. It is a hormonal marker of how long since we have last eaten because the amount produced is closely related to how empty our stomach is – more ghrelin is released the longer we go without food. Ghrelin levels are detected by receptors in a part of the hypothalamus called the arcuate nucleus. When levels rise above a set point, the arcuate nucleus sends signals to the lateral hypothalamus to secrete neuropeptide Y.  Ghrelin is now known to be an appetite stimulant in humans. Alison Wren et al. (2001) found that given intravenously, ghrelin caused a short term increase in the amount of food eaten. The amount of ghrelin circulating in the bloodstream doubles just before a meal, decreasing very quickly afterwards, and is closely correlated with subjective feelings of hunger. | | | |
| **The role of leptin**  Leptin is a hormone produced by adipose (fat) cells. Levels of leptin in the blood increase along with fat levels, and these are detected in the brain by the VMH. As leptin is an appetite suppressant, it contributes to the VMH satiety mechanism outlined above. Once levels increase beyond a certain point, the individual feels full and stops eating. Julio Licinio et al. (2004) studied an extremely rare genetic condition in which individuals are unable to produce leptin naturally. This condition is associated with severe obesity. Treatment involves leptin replacement therapy, and over an 18-month period the researchers found that this led to an average weight loss of more than 40% and a reduction in food intake initially of 49%. | | | |
| **Neural and Hormonal Mechanisms AO3** | | | |
| **Research support**  P: A strength of neural mechanisms is that there is research support. For the dual-centre model, this comes from lesion studies in rats.  E: For example, Hetherington and Ranson (1942) showed that lesioning the VMH of rats caused these animals to over eat and eventually become severely obese.  E: This shows that creating surgical wounds in various strategic brain areas confirms their roles in controlling eating behaviours.  L: As a result this strengthens the credibility of neural mechanisms, as the research directly shows that the VMH is in control of satiety. | **An oversimplified explanation**  P: It can be argued that this explanation is oversimplified as it ignores other biological factors.  E: For example, a hormone called cholecystokinin (CCK) is produced in the upper intestine. It activates the nerve that sends signals from the gastro-intestinal tract to the hypothalamus. These signals indicate satiety and contribute to the ‘stop eating’ mechanism.  E: It might even be that CCK is an even more powerful appetite suppressant than leptin. Several other neurotransmitters such as serotonin and dopamine also appear to be involved in eating behaviour.  L: This suggests that eating behaviour is more complex then this explanation perceives it to be. | **Roles of social and cultural factors**  P: A limitation of this explanation is that it ignores the role of social and cultural factors.  E: For example normal meal time is much less under neurochemical control and is usually initiated by social and culturally factors that are related to lifestyle.  E: Therefore Woods (2004) suggests that the view that the LH feeding centre always detects falls in blood glucose levels and stimulates hunger is outdated. In fact, this only occurs in ‘emergency’ conditions of severe energy deprivation.  L: This suggests that a purely biological approach to understanding eating behaviour tends to ignore potentially important non-biological factors that may be more influential. | **Non-human animal research**  P: Another limitation for the neural and hormonal explanation is that much of what we understand about it comes from research with non-human animals, especially rats.  E: For example research into the neurotransmitter neuropeptide Y (NPY) which is associated with the LH was done on rats. It was found that when NPY was injected to the hypothalamus of rats, they would eat excessively and eventually become obese as the injections continued.  E: However, it is important to be cautious about extrapolating such findings to humans without considering the differences between species that may make such generalisations invalid.  L: As a result, we are unable to apply research findings on neural and hormonal mechanisms to humans. It may be that human eating behaviours may differ to those of rats. |