

Outline and evaluate the role of neural mechanisms in controlling eating and satiation. (8 and 16 marks)



The hypothalamus is a gland in the brain that is responsible for homeostasis. Through homeostasis, our body tries to maintain our weight to its “set-point”. The dual hypothalamic process states that we have an ‘on switch’, the lateral hypothalamus (LH), and an ‘off switch’, the ventromedial hypothalamus (VMH), which controls eating behaviour.

The LH is known as the “feeding centre” of the hypothalamus as it stimulates feeding in response to signals from the body such as high levels of ghrelin or low levels of either glucose or leptin. Early research has shown that damage to this region causes aphasia.

The VMH is the “satiety centre” and inhibits feeding as it is stimulated when our body produces high levels of glucose or leptin and produces CCK. Damage to this region causes hyperphagia.

As glucose levels decrease the LH is activated, resulting in hunger, causing the person to eat and so glucose levels rise which, in turn, activates the VMH, leading to feelings of satiation.

There are many neural signals involved in messaging with the hypothalamic centres. One of them is the neurotransmitter neuropeptide Y which is produced by fat cells and stimulates the LH, thus increasing eating behaviour.

A study by Ranson found supporting evidence. He showed that lesions in the LH in rats lead to the loss of feeding behaviour (aphagia) and lesions in the VMH lead to an increase in feeding behaviour and body weight. This clearly shows the effects of the different centres. However, it must be noted that the study lacks external validity as it was carried out on rats who have a different psychological makeup. Animals also have different eating motives and so it is unclear whether the influences are the same for humans as our eating is not just geared (like many mammals) as an automatic stimulus-response drive but mood and cognition, for example, also play a part. This may then affect the extent to which the study can support the dual process model. Nevertheless, the effect has been seen in humans where tumours in the VMH have led to excessive binge eating, showing that Ranson’s study is quite reliable.

There are also practical real-life applications. This is because neuropeptide Y is produced by fat cells and so it can lead to a vicious cycle where the more fat on the body, the more NPY there is and so the more the person eats which in turn leads to more fat cells and so on. Therefore by targeting those with an increased level of NPY it should be possible to treat obesity by giving them a drug that turns off NPY. In addition, people with obesity could have a gastric band fitted as they have shown to reduce ghrelin levels.

Nevertheless, it is not that simple as eating is controlled by many more factors than just neural mechanisms. This highlights the reductionist nature of the dual process model as the hypothalamus is not the only influencing factor as we know people eat more of tastier foods and in the company of other people and that mood and culture evidently influence feeding behaviour so we cannot conclude a wholly biological effect. Additionally, Sakurai found that the LH does not only act as an “eating centre” but it also controls other behaviour and therefore ideas about the LH may be too simplistic; perhaps the LH controls other behaviour such as mood and we know that, in turn, mood may then affect eating behaviour.

A further problem is the fact that the model only focuses on the role of nature and is deterministic. It argues that our set-point is determined by our biology and thus it cannot account for free-will or how this can shape attitudes towards food and override biological urges. For example, people who diet or who develop anorexia evidently use their free-will to override their biological hunger signals or people may use their free-will in social situations when they continue eating even when they’ve reached satiety.

Additional problems with homeostatic explanations is that hunger mechanisms should, in theory, be adaptive to prevent deficits in energy rather than simply reacting to them. If hunger is only triggered when energy levels fall below their optimum, then this does not fit with the evolutionary perspective in which our biology has evolved as we should be able to promote levels of consumption which keep us above our optimum level to buffer against future lack of food. Therefore the evolutionary theory is an incomplete explanation on its own. However, in defence of the homeostatic

explanation, it could have evolved to stop us from storing fat and so keep us agile to be able to continue gathering food and resources.

The differences between males and females are also being overlooked (beta bias) and although some may argue that the differences don't apply here, researchers have found evidence that neural pathways associated with pain differ in males and females so there is a possibility that neural pathways associated with eating behaviour may also differ in men and women.