Outline and evaluate the role of neural mechanisms in eating

The hypothalamus is a structure in the forebrain which has centres for control of eating behaviour. The Lateral hypothalamus (LH) is the part of the hypothalamus that functions as a feeding centre, stimulating feeding in response to signals from the body. This was discovered after the ventromedial hypothalamus (VMH) which is the part of the hypothalamus which functions as a satiety centre to inhibit feeding in response to signals from the body.

Heatherington and Ranson published one of the most famous studies on the control of eating behaviour. They demonstrated that lesions in the ventromedial hypothalamus of the brain caused rats to overeat and become dramatically obese. These findings suggest that lesioning the ventromedial hypothalamus destroys a centre vital for the control of feeding behaviour. Due to this it was assumed that the ventromedial hypothalamus is acting as a satiety centre due to the fact that when lesioning does occur in this part of the brain the rat overeats and becomes obese.

Further research into lesioning of the brain was carried out by Anand and Brobeck, who found that a lesion in the lateral hypothalamus of rats led to a loss of feeding behaviour (aphagia). Due to the rats stopping eating when the lateral hypothalamus is removed it was suggested that this area of the brain acts as a feeding centre, the opposite of the ventromedial hypothalamus. Later studies confirmed these findings and further studies using electrical stimulation of the VMH lead to inhibited feeding, while stimulation of the LH produced feeding, so confirming these functions. These studies both of the lesioning and the electrical stimulation confirm the role of the brain, and the hypothalamus in eating behaviour, with strong agreement with the results found.

However all of the research conducted was on rats. Due to this the role of animal rights arises.

The apparent discovery of a satiety centre and a feeding centre led to a fairly straightforward model of the neural control of feeding behaviour. When blood glucose levels fall this is sensed by the hypothalamus, hunger is experienced and the lateral hypothalamus is activated, leading to some behaviour. Food intake leads to an increase in blood glucose. This is picked up by the hypothalamus and the VMH is activated and stops feeding, and the cycle continues.

Although much of the work is now over 50 years old contemporary research has confirmed the central role of the hypothalamus in feeding behaviour. However, it isn’t that simple and the pathways are involved are very complex as the hypothalamus receives a variety of signals indicating when feeding should start and stop. Therefore using the biological approach to explain the complex actions of eating behaviour is reductionist, focusing only on the biological systems regulating food intake and body weight. It ignores the psychological, social and cultural factors that influence our eating behaviour. In order to be able to fully explain eating behaviour requires research into all of these areas as well as biology is needed and therefore an internationalist approach is more suitable in order for us to have the best explanation to feeding and eating behaviour.

An important role is played by ghrelin, a hormone which is secreted from the stomach. The amount of ghrelin released is directly proportional to the emptiness of the stomach, as the time from the last meal increases and ghrelin is secretion is increased. Ghrelin activates the VMH- satiety centre in the brain as well as the LH-which in turn provides us with the signal of feeling full. This signal is then decreased as blood glucose levels decrease. So the level of ghrelin in the blood is directly proportional to signalling the hypothalamus in the brain and feelings of hunger.

Cummings found that injections of ghrelin increase food intake and body weight in animals, and humans. Ghrelin levels were measured by blood samples taken every five minutes from a catheter inserted into a vein. The results showed that ghrelin levels fell immediately after eating lunch, reaching their lowest level at about 70 minutes then they slowly began to rise, peaking as participants requested their evening meals. In five out of the six participants used, ghrelin levels were closely correlated with the degree of hunger reported. It was concluded that ghrelin levels directly reflect stomach emptiness and are closely related to subjective feelings of hunger. This supports a role for