Neural mechanisms in eating behaviour

AO1

Homeostasis - detecting internal environment and then sustaining a stable state. In humans, glucose levels determine hunger. Low levels - high hunger.

The lateral hypothalamus - the ‘on’ switch for hunger. Removing LH in rats caused an absence of eating. Neuropeptide Y, when injected into caused instant eating. After a few days it caused obesity.

Leptin and obesity - some rats are born with both obese genes ob/ob which is defective in producing Leptin. Leptin is in fat and secretes to decrease your appetite. This proves a genetic predisposition for obesity.

The ventromedial hypothalamus - damage to the VH causes overeating and therefore obesity. The paraventricular nucleus detects our need for specific foods and therefore is responsible for cravings.

Neural control of cognitive factors - getting hungry by the thought, smell or sight of a particular food.

The amygdala - selection of food based on previous experience. Rolls found that rats who had the amygdala removed ate familiar and unfamiliar foods equally whereas the rats that had the amygdala intact would go for familiar.

The inferior frontal cortex - receives messages from the olfactory bulb (part of the brain that processes smell). Because smells influence the taste of food, removing IFC causes decrease in eating. (Kolb and Whishaw)

Limitations of a homeostatic explanation - for this theory to work, it has to be able to adapt and prevent hunger issues. It must promote the ability to eat small amounts rather than wait till you are ‘staving’. Carrying some reserve in case food is not available.

The role of the lateral hypothalamus - although the LH is important it is not the solely responsible for eating. LH also impacts behaviour and sex. Neural circuits in the brain also control eating behaviour.

NPY - Marie and al (2005) genetically altered mice to not produce NPY and found no change in eating behaviour and said that the injection of NPY was a flood of the gene and therefore produced a lab - reaction. This isn’t. The role of the ventromedial hypothalamus - Gold (1973) lesions produced VMH didn’t effect eating on its own but when in conjunction with other parts of the brain.

Kluver - Bucy syndrome - damage to A and IPC caused abnormal feeding. Patients with this syndrome showed increased appetite and appetite for non-food items. Food cues no longer represent an award to the individual.

Research support - Zald and Pardo (1997) good smells showed little blood flow to A using pet scans on healthy adults however bad smells showed high blood flow.

Evolutionary approach - people actually eat because of an evolutionary adaptation to sat what would be good for survival.

RWA - NPY plays an important part in eating behaviour. Yang et al (2008) NPY is produced by abdominal fat which makes people eat more and therefore get more fat. Yang said you could take drugs that prevent the production of NPY.

Stress and hunger - the body produces high quantities of GRELIN in response to stress. GRELIN also produces a larger appetite and therefore results in comfort eating when stressed.