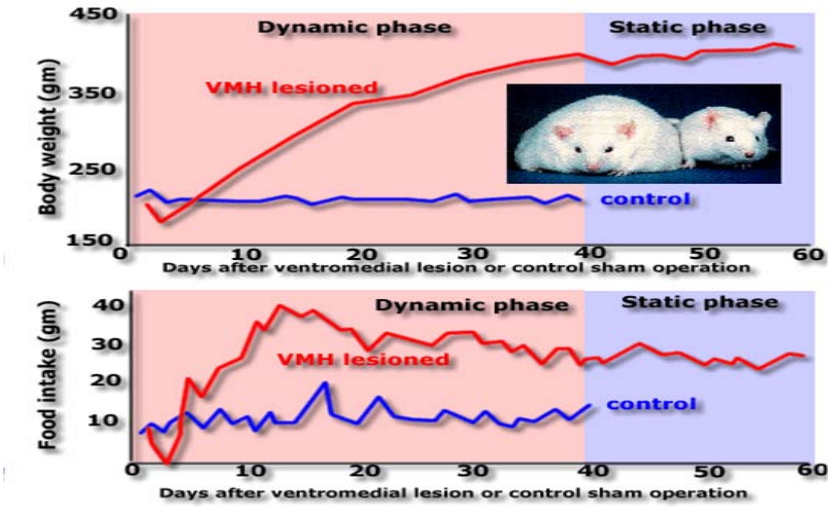
NEURAL MECHANISMS INVOLVED IN CONTROLLING EATING BEHAVIOUR

Very early research showed that although stomach contractions ('hunger pangs') are a strong incentive to start eating, glucose levels *probably* play the most important role in producing feelings of hunger. Hunger levels increase as glucose levels decrease, and researchers found that a decline in blood glucose causes the liver to send a message to the **hypothalamus**, resulting in the consumption of food.

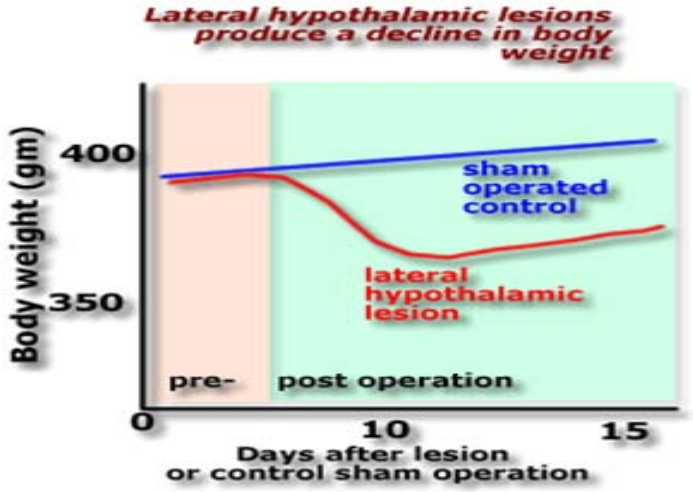
The part of the hypothalamus that receives this message is called the **lateral hypothalamus (LH)** and it was believed that this is our main 'hunger centre', which signals us to 'start feeding'. As our glucose levels increase, another part of the hypothalamus, called the **ventromedial hypothalamus (VMH)** is activated, and this leads to us stopping eating. Thus, the VMH was believed to be our main 'satiety centre', which signals us to 'stop feeding'.

This explanation of how eating behaviour is controlled is called the **'dual centre model of feeding'**. Support for this model came from studies in which the VMH and LH in rats were surgically damaged. For example, **Hetherington & Ranson (1942)** found that if the VMH is damaged, rats will overeat and become *hyperphagic*. If the VMH is electrically stimulated, rats will stop eating.



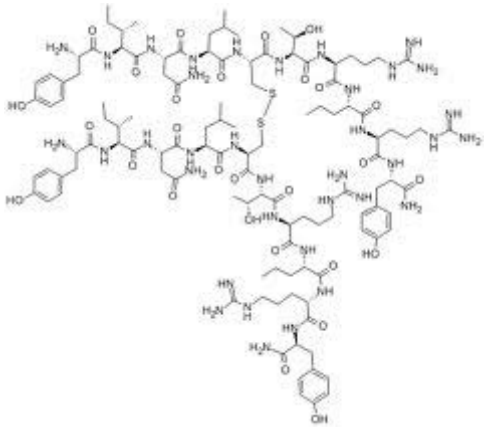
Damage to the VMH causes rats to overeat and become hyperphagic

Likewise, **Anand & Brobeck (1951)** found that damaging a rat's LH led it under-eating and becoming *hypophagic*, whilst electrically stimulating the LH causes rats to start eating even if they have just finished eating.



Damage to the LH causes rats to undereat and become hypophagic

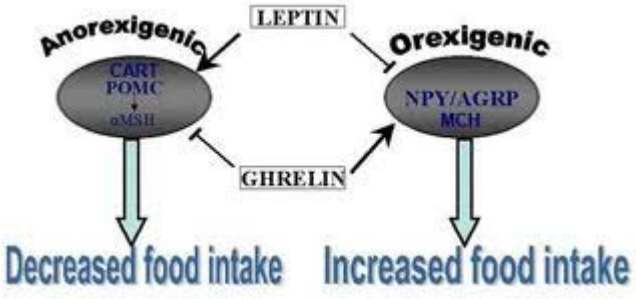
Exactly what causes the LH to be activated is hotly debated. Some research suggests that a very small protein in the arcuate hypothalamus called **neuropeptide Y (NPY)** is responsible for initiating eating. NPY is an *orexigen* (it stimulates appetite). When NPY is injected into the LH, rats immediately begin eating, even if they are satiated. Furthermore, repeated injections of NPY produce obesity in rats in just a few days.



Neuropeptide Y

As far as the VMH is concerned, a feeling of fullness is signalled by stretch receptors in the stomach and a stomach hormone called **cholecystokinin (CCK)**. CCK sends a message to the VMH which activates *anorexigens* (peptides which cause a loss of appetite). Thus, when CCK is injected into rats, there is a reduction in appetite and weight gain is suppressed.

NOTE: At this point, things *could* get very complicated. For example, research suggests that the LH is also activated by a protein called **leptin** and a hormone called **ghrelin**. This hormone is secreted from an empty stomach. Its concentration in the blood falls after each meal, and then rises again signalling it is time for the next meal. Research also suggests that neurons in the hypothalamus are also directly sensitive to **glucose** and **insulin** concentrations. Moreover, CCK is not the only chemical that decreases food intake. Others include **bombesin**, **serotonin**, and **corticotrophin-releasing hormone**. Drugs that affect eating behaviour include **nicotine**, **amphetamine**, and certain **analgesics** (decrease food intake) and **marijuana**, **anti-psychotic drugs**, and **SSRI anti-depressants** (increase food intake).



You can safely ignore the above and concentrate on evaluating the role of the LH and VMH as brain structures involved in eating behaviour.

The view that the LH is the 'on switch' for eating has, however, been criticised. For example, damage to the LH also causes deficits in thirst as well as hunger. This would suggest that the LH may be involved in other behaviours and not just eating. It has also been found that damage to the LH does not cause eating to stop completely. Instead, rats will stop eating and lose weight for a period of time (the 'dynamic phase'), but then eat a sufficient amount to maintain a new, but lower, body weight (the 'static phase').

Recent research suggests that eating behaviour is also controlled by neural circuits that run throughout the brain, and not just in the hypothalamus. This would possibly explain why damage to the LH does not cause eating to stop completely. Therefore, although the LH plays a role in initiating eating behaviour, it does not actually seem to be the brain's 'on switch' for eating behaviour.

A similar issue concerns the VMH. **Gold (1973)** showed that damage to nerve fibres passing through the VMH also tend to damage another area of the hypothalamus called the *paraventricular nucleus (PVN).* In fact, if the PVN on its own is damaged, then hyperphagia occurs, suggesting that

it rather than the VMH is important in eating. Research has also found that the PVN detects the specific foods our body needs, and that it may also be the part of the brain responsible for 'cravings' for particular types of food.

As with the LH, it has also been found that damage to the VMH does not cause rats to continue to overeat and gain weight *indefinitely*. Instead, they will over-eat for a period of time (the 'dynamic phase'), but then eat a sufficient amount to maintain a new, and higher, body weight (the 'static phase').

The view that eating and satiation are controlled by brain structures suggests that **nature** rather than nurture plays the major role in these behaviours. However, although the LH and VMH are important in controlling eating behaviour, they don't explain everything. The precise mechanisms involved are extremely complicated. Moreover, this approach is **reductionistic** and focuses on biological mechanisms without taking into account the influential role played by social, cognitive, and developmental factors in eating behaviour.

There are many learned and cognitive aspects of eating behaviour. These include attractive sights and smells which can initiate hunger even if we have just eaten. Our eating behaviour is also related to our habits (when and how much to eat), how stressed we are ('comfort eating'), and our culture's attitudes to foods. All these external stimuli provide signals to the hypothalamus to make us feel hungry, even though they are not physiological signals.

The dual centre model of feeding also seems to deny us **free-will**, because it assumes that we will respond to a signal that we are hungry by eating, and respond to a signal that we are full by stopping eating. However, we can sometimes override our physiological drive to eat because, for example, we don't like the food that has been given to us. Likewise, we can also override our physiological drive to stop eating because, for example, more food is available than usual.

As well as this, it should be remembered that a lot of experimental research has been conducted on rats, which raises serious **ethical issues** about harming non-humans. It also raises issues about **generalising** research findings from non-humans to humans.