# BIOLOGICAL EXPLANATIONS (NEURAL AND EVOLUTIONARY) OF ANOREXIA NERVOSA

The first case of what is now called anorexia nervosa (AN) was recorded as early as 1694, when it was called *anorexy*, which means 'for want of an appetite'. However, it was not until the early twentieth century that an attempt was made to explain the condition. *Morris Simmonds*, a German pathologist, believed that AN was caused by damage to the pituitary gland. Unfortunately, Simmonds got it wrong. Not surprisingly, therefore, attempts to treat AN using pituitary extracts were unsuccessful. However, there are other neural explanations of AN.

## Brain damage as a neural explanation of anorexia nervosa (AN)

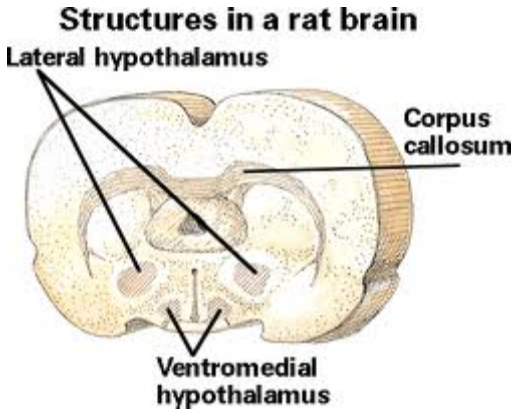
Interestingly, anorectics *sometimes* have illnesses like glandular fever prior to AN developing. This has led to the suggestion that a *virus* may cause AN. This virus would need to damage a brain structure(s) in order for AN to occur, and it has been proposed that the virus damages the **hypothalamus** and interferes with central homeostasis (of which eating is a part).

The hypothalamus is involved in regulating eating behaviour. One structure (the **ventromedial hypothalamus** or **VMH**) acts as a 'stop feeding' centre, and tells us when we have had enough to eat. If this structure is damaged, then rats will overeat and become morbidly obese. This suggests that obesity can be explained in purely neural terms as a result of damage to the VMH.

A hyperphagic rat (left) with a litter-mate

Another structure (the **lateral hypothalamus** or **LH**) acts as a 'start feeding' centre and tells us when we are hungry. Damage to this structure causes rats to significantly lower their food intake. Indeed, some rats

eventually die through starvation. As a result, it has been proposed that AN can also be explained in purely neural terms as a result of damage to the LH. Note, though, that the original research was conducted on rats, so there are likely to be generalisation issues.




Damage to the LH *might* cause AN

Of course, damage to the LH can occur at any time during life. However, there appears to be a *'season of birth'* effect in AN, with anorexics being more likely to be born in late spring. Whether this is merely a statistical quirk or indicative of some underlying causal factor isn't known.


The 'season of birth' effect in AN

Other theories propose that AN is a result of brain damage at or shortly after birth. Swedish research, for example, has shown that significantly more anorectics than would be expected have experienced some kind of birth trauma. British research using scanning devices has shown that there is reduced blood flow in the *anterior temporal lobes* of anorectics as compared with non-anorectics.

The importance of this finding is that these structures are involved in regulating appetite and a sense of fullness - reduced blood flow could

case these functions to be impaired. Also, the lobes are involved in interpreting visual sensations - reduced blood flow might cause visual sensations (such as one's own body image) to be misperceived.

However, although *some* cases of AN might be a result of this kind of brain damage, there are many reasons why brain damage is not a good way of explaining most cases of AN. For example, AN is much more common in females than males. This could only be explained if female brains were more susceptible to this kind of brain damage than male brains. There is absolutely no evidence that this is the case.

Similarly, AN is much more commonly seen in the middle class than the working class, and in some cultures than other cultures. Again, this could only be explained if there were differences between social classes and cultures in terms of vulnerability to this kind of brain damage, and this seems very unlikely.

It is also the case the vast majority of people with AN show no evidence of brain damage, indicating that their AN must have other causes (such as cultural influences). Even if brain damage is seen in AN, the assumption that it was brain damage that caused AN might be wrong. It could, for example, be the case that brain damage is a *consequence* of AN rather than a cause of it.

Finally, this neural explanation is **deterministic** because it says that a specific kind of brain damage inevitably leads to AN. It is also **reductionistic**, and explains a complex behaviour in purely biological terms without having any role for the environment.

**Genetics as an evolutionary explanation of anorexia nervosa (AN)**

Another biological explanation for AN is **evolutionary**. It has been proposed that AN may be the result of **genetic factors**. Family resemblance studies indicate that a person is 4-10 times more likely to develop AN if they have a first degree relative who suffers from it than if they come from an AN free family. **Holland et al's (1984)** study of 16 pairs of MZ twins found a 56% concordance rate, which is also taken as evidence that genes are involved in AN.

However, in both of these kinds of study there is the issue of *shared environments*. The way to control for this is to study MZ twins who have

been separated at or shortly after birth and raised in different environments. No such study has been done in this area because of the issue of a very small sample size. An alternative is to conduct an adoption study. Again, though, no such study has been done.

Genetic researchers, however, point to the fact that the concordance rate is much higher in MZ twins than in DZ twins (56% versus 7%). So even though both types of twin typically share environments, an MZ twin is 8 times more likely than a DZ twin to suffer AN if their other twin is suffering from it.

However, the concordance rate for the MZs is not particularly large, even though they share the same genes and have shared the same environment. In theory, the concordance rate should be 100%, but in Holland et al's study it is only 56%. The main problem is that, as yet, no *'candidate gene'* has been identified for AN. Critics say that this is because genetic factors don't play a role in AN. Even if a gene was discovered, it would then need to be explained why it would be advantageous to inherit a gene that predisposed people to what is considered to be a serious mental disorder.

However, two evolutionary explanations have been proposed. These are the **'adapted to flee' hypothesis** and the **'reproductive suppression hypothesis'** proposed by **Surbey (1987)**. According to Surbey, AN is 'a reflection of the female ability to alter the rate at which reproductive functions are allowed to mature in response to prevailing environmental conditions'.

Surbey's hypothesis is based on the argument that female non-humans can optimise their lifetime reproductive success by suppressing reproduction when future conditions for the survival of the offspring are likely to be sufficiently better than present ones as to exceed the cost of suppression itself. This argument itself is based on the observation that puberty is delayed in some female non-humans when they are subjected to stress or are in a poor physical condition.

The ability to delay reproduction would have been adaptive in our evolutionary past because it would have enabled a female to avoid giving birth when the conditions were not conducive to her offspring's survival. In the absence of contraceptives, women needed a way of avoiding becoming pregnant when the environmental conditions were poor (e.g.

little food was available). Because menstruation ceases in AN, a woman could therefore avoid becoming pregnant by behaving anorexically. When the environmental conditions improved, the anorectic behaviour would stop and the woman could become pregnant.

Surbey argues that AN is a *'disordered variant'* of a woman's ability to alter the timing of reproduction when she feels unable to cope with the biological, emotional, and social responsibilities of womanhood. For Surbey: *'AN alters a girls developmental trajectory from that of an early maturer to that of a late maturer, and this does not appear to have a negative effect on her reproductive abilities.'*

Usually, the main problem with evolutionary explanations is that they are difficult to falsify. However, this explanation can be falsified. Although it might explain female AN, it cannot possibly explain male AN. Although male AN is rare, an increasing number of men have been diagnosed as AN, and it is now recognised as a disorder affecting both sexes.

Whether or not the explanation from evolutionary psychology has any truth value, it is generally accepted that genetic factors *may* play a role in the development of AN. However, if these genes are to operate, then the individual needs to find themself in the 'right' environment. This is called the **diathesis-stress model**.

The diathesis-stress model says that each of us has some degree of 'biological vulnerability' to AN. If it runs in our family then our biological vulnerability is greater than if it does not. Each of us also experiences different amounts of 'stress' in our lives. If our biological vulnerability is *high*, it won't take too many stressors to 'tip us over the edge'. However, if our biological vulnerability is low, then it will take an awful lot of stress before we are 'tipped over the edge'.

It should also be noted that this evolutionary explanation is also **deterministic** because it says that AN inevitably occurs if a gene is inherited. It is also **reductionistic**, and explains a complex behaviour in purely genetic terms without having any role for the environment.

**FAULTY REGULATION OF BRAIN BIOCHEMISTRY AS AN ALTERNATIVE BIOLOGICAL EXPLANATION**

As we have seen, the hypothalamus is involved in regulating eating. The hypothalamus is influenced by the neurotransmitter ***noradrenaline***, which causes rats to start eating, and to show an increased preference for *carbohydrates*. By contrast, the neurotransmitter ***serotonin*** (which also acts on the hypothalamus) induces satiation and suppresses appetite, especially for carbohydrates. AN may therefore be a result of serotonin acting on the hypothalamus. Interestingly, serotonin has also been implicated in obsessive behaviour, phobia, anxiety and increased vomiting, all of which can co-occur with AN. If an anorectic were to stop eating, especially foods rich in a substance called ***tryptophan***, their serotonin levels would drop which would reduce their anxiety. This would make them feel better, and motivate them to continue not eating (note that this is crossing paths with a ***behavioural*** explanation of AN).

Other biochemical explanations implicate *hormones*. One hormone, called ***CCK-8*** carries messages between nerves in the digestive system. Its job is to *dampen appetite*. Normally, CCK-8 is destroyed by *enzymes*. However, if these enzymes are prevented from doing their job, eating is suppressed. Therefore, AN could be the result of something going wrong with enzyme production.

**NOTE:** These biochemical explanations can be evaluated in exactly the same way as the brain damage explanations were evaluated. For example, changes in brain biochemistry would have to be more likely to occur in *women* than in men because of the sex ratio in AN. Since there is no reason why female brains should experience more changes than male brains, this is a weakness of biochemical explanations. So, in an exam, make exactly the same evaluative points as you made with brain damage explanations.