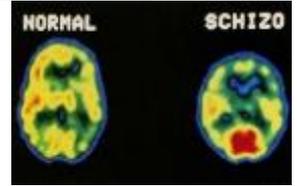


OUTLINE AND EVALUATE BIOLOGICAL EXPLANATIONS OF SCHIZOPHRENIA (8 and 16 marks)



One biological explanation focuses on genetics. Gene's code for a person's characteristics and neuroanatomy so bio-psychologists believe that genes are the cause of schizophrenia and that rates of schizophrenia will be higher between relatives as the offspring of a schizophrenic will inherit this gene. This gene could then either code for faulty dopamine neurones which could influence the course of the disorder or it could impact the development of neuroanatomy which could also be a contributing factor. The impact of genetics is supported by the prevalent statistic of 1% of the population around the world being schizophrenic as this shows that the prevalence does not vary with environment and so the cause must be genetic.

Twin studies have been used to establish the link between genes and the development of schizophrenia. The concordance rate for MZ twins is 40% whereas with DZ twins it is 6%. This clearly shows a genetic vulnerability for the illness because if there was no genetic influence then there wouldn't be a difference in concordance rate. However, because the rate was not 100% either, other factors must also need to be considered. Perhaps a diathesis-stress model would be more appropriate.

The problem with using MZ twins is that an assumption is made that they are born in the same way. This may not be the case as often one twin has taken more nutrients than the other so is born bigger or more developed and this could affect the development of the central nervous system. Twins studies are also low in internal validity as sample sizes are usually small and the fact that schizophrenia appears to run in families may be just due to the common rearing patterns (such as high Expressed Emotion) and so studies may not be measuring what they claim to be.

Adoption studies may be a more effective method. Kety found supporting evidence as she concluded that rates of schizophrenia were higher in adoptees whose biological parents had also been diagnosed even if they were raised by non-schizophrenic adults, showing that the genetic influence is more significant than environmental factors.

The problem with adoption studies, however, is that the time spent with their schizophrenic parent before adoption could have influenced the way they behaved as they may imitate schizophrenic behaviours which could increase the likelihood of them developing the disorder themselves.

Nevertheless, all research done using adoptees show the same results, suggesting research into this area is reliable. For example, Tienari supports Kety's conclusion as he found that 10% of adopted children whose biological mothers had schizophrenia developed the illness themselves. However he did find that the illness only revealed itself if the adoptive family was psychologically disturbed and so this could criticise the genetic explanation by highlighting how some individuals may have a vulnerability to schizophrenia but that they could be protected by a healthy family environment.

Even though there is a lot of research on the genetic influence, no gene has yet been discovered which is responsible for schizophrenia. This makes it hard to rely on genetic factors as being the main cause. However the biological approach does recognise that genes can be turned on and off by environmental factors so it is not completely deterministic.

A new way of trying to explain schizophrenia, as highlighted in the January 2016 edition of "the pharmaceutical journal", is that the illness may be caused by an overactive immune system

which accelerates the symptoms and this impaired immune system may be innate and genetic in nature, further highlighting the effects of genetics on schizophrenia.

Another explanation is the dopamine hypothesis which believes that schizophrenia results from an excess of dopamine activity resulting in dopamine firing too easily or too often. This is significant as dopamine plays a key role in guiding attention, so disturbances in this process may lead to the problems related to attention, perception and thought.

Evidence supporting this hypothesis comes from research into antipsychotics which reduce schizophrenic symptoms by blocking dopamine receptors. This would suggest that it is overactive dopamine receptors that causes the symptoms. L-dopa is used to treat Parkinson's disease and works by increasing dopamine. Research has found that L-dopa can produce schizophrenic symptoms which indicate that dopamine is related to the symptoms.

Additionally, high levels of dopamine are not found in all schizophrenics and it may be an incomplete explanation as antipsychotics take several weeks to reduce positive symptoms even though they begin blocking D2 receptors very quickly. This suggests that their therapeutic effect may be due to the effect this blockade has on other brain areas. For example serotonin neurons regulate dopaminergic neurons so dopamine may just be one piece in a much wider picture.

A weakness, however, is that it is unclear whether high dopamine levels causes the disorder or whether it is caused by the disorder. Much of the supporting research comes from post-mortems but the people have usually taken antipsychotics for a long time so this uncertainty as to whether high dopamine is a cause or effect makes it difficult to establish a cause and effect relationship. Perhaps the higher levels of dopamine found in much