Caroline Hewitt

Discuss the Evidence That Implicates a Biological Dysfunction as a Cause for Schizophrenia

Schizophrenia is a mental disorder, which is characterised by a number of both positive and negative symptoms. Positive symptoms are behaviours which are present although should be absent. Examples of these are thought disorders resulting in difficulty in arranging thoughts logically, jumping from one topic of conversation to another and speaking random words. Other positive symptoms of schizophrenia include delu sions whereby the effected person may feel that people are plotting against them and trying to kill them as well as hallucinations whereby the schizophrenic person hears voices in their head telling them to do things. Negative symptoms are also shown by people suffering from schizophrenia and are the absence of behaviours, which are normally present. Examples of these symptoms are a flattened emotional response, a poverty of speech and social withdrawal. It has been suggested that there are different cau ses for the different types of symptoms, for example excess activity in some neural circuits is said to be responsible for the positive symptoms whereas the negative symptoms are said to have developmental causes.

There are many suggestions for the biologi cal causes of schizophrenia, many with varying degrees of supporting evidence. However the five main suggestions are heritability, genes, the 'Neurodevelopmental Hypothesis' (including both prenatal and neonatal abnormalities and brain abnormalities), the dopamine hypothesis and the glutamine hypothesis. Firstly heritability shows how the disease can be inherited from the person's parents. This is illustrated through two main types of methodology, twin studies and adoption studies. Twin studies look at monozygotic and dizygotic twins and measure whether hereditary factors cause schizophrenia by looking at the concordance rate (if both twins have schizophrenia then they are concordant, the percentage of concordant pairs is called the concordance rate.) I f genes were entirely responsible for schizophrenia then monozygotic twins and dizygotic twins would have concordance rate of 100% and 50% respectively.

Kendler conducted a twin study and found that the concordance rate for monozygotic twins was 53% and t he concordance rate for dizygotic twins was only 15%. This shows solid evidence for there being a hereditary component in the cause of schizophrenia. However as the concordance rate is not 100% and 50% for monozygotic and dizygotic twins respectively the n this study also shows that environment does play some role. This theory was criticised as it was suggested that the higher concordance rate was due to monozygotic twins being exposed to a more similar environment than dizygotic twins. However the theor y was proved wrong by a study, which showed that monozygotic twins that were reared apart still showed a 65% concordance rate.

Adoption studies strengthen the evidence to show how there is a hereditary component in the cause of schizophrenia by showing wh ether biological or adoptive relationships explain the familial transmission of the disease. Heston conducted a study whereby a group of children separated from their schizophrenic mothers within three days of birth and raised by adoptive parents were com pared to a group of children separated from non - schizophrenic mothers. Five of the children in the group where the children

were separated from schizophrenic mothers became schizophrenic themselves compared to none in the other group. Also adopted childr en whose biological mothers weren't schizophrenic but their adoptive mothers were, were looked at and it was concluded that this didn't lead to schizophrenia. This again shows evidence for a hereditary aspect to the cause of schizophrenia.

This research has however been criticised as it has been suggested that the environment inside the mothers womb when the baby is growing may have been the cause for schizophrenia. However research conducted by Kety et al disproved this theory by looking at the paternal influence. Adopted paternal half siblings, whereby the biological father was schizophrenic were studied and the results showed that 13% had schizophrenia. Despite the fact that this percentage is rather low, it is however well above the normal average rate of 1% and shows that paternal influence does therefore have some effect.

The second suggested biological cause of schizophrenia is linked to heritability and is the genetic element. There are two types of studies associate with finding the gene respo nsible for schizophrenia and they are linkage studies and association studies. Firstly linkage studies aim to identify chromosomal regions that are the same amongst all schizophrenia but different to normal people. Association studies however focus on ge nes that are possibly associated to schizophrenia due to theory.

Despite the fact that a large amount of research has been carried out investigating whether there is a gene responsible for schizophrenia the evidence has not been conclusive. However there have been some encouraging findings. An example of this is an association study carried out by Murphy, Jones and Owen. They found that 25% of schizophrenics in their sample had a small deletion of chromosome 2211q, suggesting that this may be one of the causes for schizophrenia. Linkage studies have also been conducted, an example of this being the work carried out by Shastry. He concluded that schizophrenia is linked to many genes; examples of this are chromosomes 1, 2, 4, 5, 6, 7, 8, 9, 10, 11, 13, 1 5, 18, 22 and X. Despite there not being a specific gene that has been found to be responsible, this suggests that genes do play a part in the cause of schizophrenia.

Another suggested cause of schizophrenia is the 'Neurodevelopmental Hypothesis.' This hypothesis suggests that schizophrenia is based on abnormalities in the prenatal and neonatal development, which then leads to brain abnormalities in later life. Abnormalities in prenatal and neonatal development are abnormalities brought about by problem s during the pregnancy or shortly after the birth of the child. Dalman et al who concluded that if a person suffered prenatal or neonatal problems such as a premature birth, a low birth rate or poor nutrition during pregnancy then they were more likely to become schizophrenic showed this. Susser et al who studied the children of women in the Netherlands who were pregnant in the winter of 1944-45 further illustrated this point. This winter was the end of the Second World War and during it the Germans had blockaded the Netherlands, this led to poor nutrition for the pregnant mothers. The results from this study supported the theory suggested by Dalman as this poor nutrition then led to a higher percentage of schizophrenia in the participants.

A final suggestion to illustrate how problems in prenatal development lead to schizophrenia is called 'The Seasonality Effect.' This effect suggests that babies born in late winter and early spring have a higher chance of having schizophrenia. This was shown in a st udy conducted by Kendell and Adams who looked at patients in Scotland. Their findings supported 'The Seasonality Effect' theory as they showed that patients born in February, March, April and May had higher levels of schizophrenia. A possible explanation for this effect is that the mothers of the schizophrenia patients contracted a viral illness during the critical months of development. As more people tend to contract viral illnesses in the winter months this explains how 'The Seasonality Effect' can ex plain how problems in prenatal development can cause schizophrenia.

The second element of the 'Neurodevelopmental Hypothesis' is how brain abnormalities in later life can also cause schizophrenia. Many studies have been conducted to illustrate this and d espite there not being any conclusive evidence there are many suggestions to possible abnormalities in many schizophrenic's brains. One suggestion is ventricular enlargement in schizophrenics, which is thought to be the result of neuronal loss in various regions of the brain. Weinberger investigated this suggestion and concluded that ventricular size varied among schizophrenics and that some were abnormally large. Honer et al also supported the concept that people with schizophrenia had enlarged ventricl es however they also concluded that there were more brain abnormalities. They suggested that schizophrenic people also had high levels of neurotropic factors as well as having abnormalities of the hippocampus, amygdala and other areas of the temporal and frontal lobe.

Further research carried out in this field also showed support for the idea that people with schizophrenia have brain abnormalities. Rioux et al examined post mortem brain tissue and concluded that schizophrenic people had a different pattern of neuronal placement in the anterior section of the parahippocampal gyrus. Therefore despite there not being any conclusive evidence to show that there is a certain abnormality in the brain which causes schizophrenia there is however a substantial amo unt of support for the suggestion that the brains of schizophrenic people are different to those of normal people.

Further evidence for the suggestion that schizophrenia is caused by a biological dysfunction has been shown through pharmacological evidence . This shows how drugs that relieve or provoke the symptoms of schizophrenia can be used to show the causes of the disease. The two main theories involved in pharmacological evidence are the dopamine hypothesis and the glutamine hypothesis.

Firstly the dopamine hypothesis suggests that schizophrenia comes from excess activity at certain dopamine synapses. This evidence comes from drugs that either relieve or aggravate symptoms of schizophrenia. Firstly antipsychotic drugs such as chlorpromazine relie ve the symptoms of schizophrenia. These drugs however also block the dopamine receptors, and so suggest that excess activity at dopamine synapses may be the cause of schizophrenia. This is supported by the fact that other drugs that relieve the symptoms of schizophrenia also block dopamine receptors. Similarly drugs such as amphetamine, which increase the symptoms of schizophrenia, also increase activity at the dopamine synapses. A final theory supporting the

dopamine hypothesis is that stress can also cause schizophrenia. This supports the dopamine theory as when a person is stressed the amount of dopamine released in the pre-frontal increases. It has also been found that the pre-frontal cortex is the area for the cause of schizophrenia; therefore this shows how the increase in dopamine is a possible cause for schizophrenia, thus supporting the dopamine hypothesis.

Further evidence showing a biological dysfunction as a cause for schizophrenia is related to the dopamine hypothesis and is called the glutamate hypothesis. This hypothesis suggests that schizophrenia is caused by deficient activity at certain glutamate synapses. It has been shown that in many areas dopamine inhibits the release of glutamate. According to the dopamine hypothesis in schizophrenic people there is larger amount of dopamine and therefore the glutamate hypothesis suggest that this will lead to a lower amount of glutamate. Therefore antipsychotic drugs that block dopamine synapses lead to an increase in glutamate. Thus it can be concluded that this increase in glutamate relieves the symptoms of schizophrenia and so that the glutamate deficiency is the cause of schizophrenia.

All of the previous evidence discussed above suggests that biology does play an important role in the cause of schizophrenia. However none of the theories are fully conclusive and many lack in supporting evidence. An example of this is the genetic cause of schizophrenia. Despite the fact that many studies have concluded that there is genetic element responsible for schizophrenia no exact gene has been found that is responsible.

Also, as previously discussed, when looking at the hereditary causes of schizophrenia, as monozygotic twins share 100% of their genes if the cause of schizophrenia was hereditary then the concordance rate would be expected to also be 100%. Studies have shown that this is not true and that the concordance rate is much lower at approximately 53% (although this varies in different studies.) Therefore the evidence showing that schizophrenia has a hereditary cause is not completely decisive.

Brain abnormalities as a cause for schizophrenia also highlight the fact that biological theories for the cause of schizophrenia are not completely convincing. The many studies carried out investigating this issue have shown many areas to be responsible for schizophrenia. They haven't however led to an exact region being identified and the specific dysfunction being discovered. This again shows that despite some evidence for the theory there is still a lack of evidence for it to be fully conclusive.

Therefore it can be concluded that there is an implication that biological dysfunction does cause schizophrenia however there is not one single cause for the disease, but a number of causes that result in a person having the disease. It is also possible that biological aspects are not entirely responsible and that other factors such as the environment play a role in the cause of schizophrenia.

Caroline Hewitt

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