

Psychology essays -
schizophrenia
depression neurology



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Schizophrenia Depression Neurology

Discuss the evidence that implicates a biological dysfunction as a cause for either schizophrenia or depression.

There are many psychological theories to explain the causes of schizophrenia, and one of the most influential theories is the biological theory. This includes the structure of the brain, neurology and genetics. “Mental diseases are brain diseases”, this was the view of Wilhelm Griesinger (1845) who based the causes of all mental diseases, such as schizophrenia on biological dysfunction. This essay will aim to explore evidence for and against the view that the cause of schizophrenia is a biological dysfunction, and come to a conclusion as to whether it is the most reliable theory to explain this psychosis.

Firstly the evidence for and against the idea of genetics being a cause of schizophrenia will be discussed. Genetic studies of schizophrenia are rooted in the “common observation” that schizophrenia tends to run in families, that it occurs at a higher rate among relatives of schizophrenic patients than in the general population.

Twin studies are influential for evidences to prove or refute a biological dysfunction being the cause of schizophrenia. The assumption here is that monozygotic twins (two genetically identical individuals) and dizygotic twins (who share 50% of each other’s genes) who are raised in similar environments can show evidence for genetics being the cause of schizophrenia because they are at equal risk of having the disorder.

Kallman famously researched the concordance rates for genetics and Schizophrenia. In *The Genetics of Schizophrenia* (1938) Kallmann stated that there was a concordance rate of 73% between schizophrenic patients and their identical twins. His studies have been criticized for seeming to be inflated. For example a pair of twins in which one showed marked schizophrenic deterioration and the other massive neurotic symptoms would be considered concordant and so there are no strict boundaries to what passes as concordance.

Studies with broader samples have been carried out, for example by Tienari (1968) in Scandinavia. He only found a 6% concordance rate in monozygotic twins, although he did stick to a strict definition of schizophrenia. However Gottesman and Shields (1976) found a higher concordance rate of 35%. Shields pointed out that 70% of the non-psychotic children of schizophrenic parents are “normal”, showing that schizophrenia cannot be completely environmentally determined; otherwise these children would have been affected by the conditions they were subjected to at home.

Shields also gives evidence in favour of the influence of the environment over genetics as he found a slightly higher concordance rate in dizygotic twins than in other siblings. However these results were not statistically significant. He also found a higher rate in same-sex siblings than opposite sex siblings which is important because schizophrenia is shown not be caused by a sex-linked gene, so these findings must be a result of how the siblings were reared.

Rosenthal (1974) also criticizes twin studies because they can also be explained by family studies. Schizophrenia is likely to affect a whole family, but especially if the siblings are similar, because they are generally given the same parental treatment. Therefore it is possible that they contract the same disorder, not due to a genetic link but because of family treatment.

Rosenthal looked at studies which separated genetic and family factors. For example Heston (1966) investigated children of schizophrenic parents who had been adopted away, and found that 8 children out of 47 ended up being hospitalized for schizophrenia, compared to none of the controls. This therefore supports the cause of schizophrenia being a biological dysfunction. Karlsson (1966) also found a greater incidence of schizophrenia among biological siblings of adopted schizophrenics than among their foster siblings.

Rosenthal (1974) studied the rate of schizophrenia in adult adoptees whose biological parents were schizophrenic. Using a psychiatric mental examination, he found an insignificant rate of schizophrenia when a strict definition of schizophrenia was used and a significant rate when a broad spectrum definition was used. Therefore Rosenthal concluded that children were benefiting from being reared away from their schizophrenic parents as this could protect them from developing the disorder.

It seems here that the studies trying to find a genetic cause for schizophrenia have been somewhat inconsistent. However with all the findings accumulated, there is definite evidence for the cause of schizophrenia being genetic.

Biochemical theories are also another important part of the idea that schizophrenia is caused by a biological dysfunction. There are two main theories, the transmethylation theory and the dopamine hypothesis. Hoffer, Osmond and Smythies (1954) began the transmethylation theory. They noted that there was a structural similarity between the catecholamine, dopamine and mescaline. They hypothesised that abnormal methylation of dopamine would produce a mescaline-like substance, an endogenous hallucinogen that they called DMPEA.

There is in fact an N-methylating enzyme that is involved in the normal synthesis of epinephrine, and this or a similar enzyme might create DMPEA. Friedhoff and Van Winkle (1962) found an abnormal amine in the urine of fifteen schizophrenics but in none of the normal controls, and this was identified as DMPEA. There have been contradictory findings however. DMPEA has been found in the urine of normal people as well as schizophrenics. Pollin, Cardon, and Kety (1961) carried out an influential experiment where the methyl-rich amino acid methionine was administered to a group of schizophrenic patients.

This resulted in a significant amount of the patients having a brief intensification of their psychotic symptoms. Wyatt, Termini, & Davis (1971) found that control groups reacted very differently to the methionine, showing fatigue and anxiety. Rosengarten and Friedhoff (1976) stated that even though there are higher levels of hallucinogens in schizophrenics than in controls, we do not know what role these substances play. They suggest that these substances are related to symptom formation, so are secondary, not

central to the etiology of schizophrenia. There is no conclusive evidence to support transmethylation; however it has not been disproved.

Some researchers try to understand the action of neuroleptic drugs, to understand the cause of schizophrenia. Neuroleptic drugs such as Thorazine alleviate schizophrenic symptoms and understanding their chemical structure and activity in the brain could help to see the biochemistry of schizophrenia. It was found that the drug influenced dopamine transmission in the brain, the dopamine receptors were blocked. This is where the dopamine hypothesis was developed from. Sachar et al (1978) and Snyder (1978) investigated this idea and confirmed that the clinical effectiveness of a neuroleptic drug is correlated with its potency as a dopamine blocker.

Another route of the dopamine hypothesis is that long term use of amphetamines can produce a state clinically indistinguishable from schizophrenia. Both amphetamines, which cause the release of catecholamine (dopamine and norepinephrine), and L-dopa, a precursor of dopamine, intensify pre-existing schizophrenic symptoms. Snyder therefore concluded that "one can titrate symptoms by manipulating brain dopamine".

A weaker route for the dopamine hypothesis is based on findings which have shown lowered plasma levels of monoamine oxidase (MAO). MAO is an enzyme which breaks down neurotransmitters including dopamine. Wyatt and Murphy (1976) found that identical twins discordant for schizophrenia both had low levels of MAO, which strongly suggests it is genetic. Davis (1978)

added that schizophrenia which is a disease of young adulthood, occurs at a time in the life cycle when MAO levels are at their lowest.

There is also a clinical finding that some borderline patients can become schizophrenic if they are given MAO inhibitors, giving evidence for the balance of dopamine and MAO being critical for mental life. But it must be remembered that most people do not become schizophrenic after being given MAO inhibitors.

There is a great deal of evidence to show that antipsychotic drugs block dopamine, and that this is beneficial to schizophrenic patients, however it cannot be concluded that the dopamine level plays a major causal role in schizophrenia. What is known of neuroleptic drugs is limited because dopamine does not only have a critical role in therapeutic pathways but it also affects other pathways, which can for example cause side effects as seen in Parkinson's disease.

There are other debates, for example Friedhoff and Alpert (1978) proposed that because it takes up to six weeks before the therapeutic action of neuroleptic drugs takes place, it may actually be due to a compensatory increase in the sensitivity of postsynaptic receptors to the available dopamine and not because of the blockade of dopamine receptors. Therefore the dopamine hypothesis is very influential, but it cannot be confirmed as a primary biological dysfunction which causes schizophrenia as the knowledge which is held on its impact on the brain is not developed enough yet.

There are also neurophysiological theories for the cause of schizophrenia, which deal directly with the study of the brain functioning through electrical
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stimulation. Many researchers have attempted to isolate the portion of the brain responsible for schizophrenia. Sem-Jacobsen (reported in Shagass, 1969) found that stimulating the parietal lobe could produce psychosis-like symptoms. Mednick (1970) in his work on high-risk children, found more perinatal complications and anorexia in children who became schizophrenic.

He hypothesized that the locus of damage from perinatal complications was the hippocampus. He subsequently discovered that rats with hippocampus damage showed some of the same learning difficulties as did pre-schizophrenic children. Hippocampal damage may permit oversecretion of the adrenocorticotrophic hormone (ACTH) during periods of stress and could explain schizophrenic's excessive arousal. Also the hippocampus could explain overarousal in schizophrenia, as it may have a defective capacity to inhibit the reticular information, leading to a constant state of alertness.

These are good explanations for a biological dysfunction being the cause of schizophrenia; however each biological dysfunction discussed should not necessarily be looked at individually as the cause of the disorder.

The cerebral blood flow has also been investigated in schizophrenics. Both schizophrenics and normals have the same cerebral blood flow overall, however in schizophrenics, a lower flow has been found in the frontal lobes and higher postural flows. (Kety et al. 1948). This finding has caused Franzen and Ingvar (1975a, 1975b) to speculate that these flows are associated with a syndrome of unusual elaboration of sensory messages and perceptual disturbances. These are also important findings for the cause of schizophrenia being a biological dysfunction.

It seems too simple to assume that schizophrenia could be caused by one single defect in the brain. A lot of researchers have been more concerned with the involvement of the whole central nervous system (CNS) instead of trying to locate the site of brain dysfunction. Hertzog and Birch (1966) and Gittelman and Birch (1967) found a high incidence of mild neurological impairment indicated by intellectual assessment, adventitious motor overflow, auditory visual intersensory integration, and clinical neurological exams in children and adolescents diagnosed schizophrenic.

Gittelman and Birch concluded that 80% of these children had some CNS pathology and suggested a connection with the high incidence of perinatal complications in these children's histories. Hertzog and Birch found that the degree of CNS disturbance in adolescents correlated with severity of their psychotic illness.

However these studies must be looked at with caution. It seems probable that the earlier a disorder manifests itself, the more likely it is to be caused by organic factors. Also it is not clear that "childhood schizophrenia" is related to any form of adult schizophrenia, but these are still influential investigations.

In conclusion it would seem that the evidence for schizophrenia being caused by a biological dysfunction is quite solid. The experiments carried out are mostly in very controlled settings, very scientific and not at all subjective, which is advantageous to this explanation.

However other explanations for the cause of schizophrenia must not be ruled out, for example it would be naive to say that the environment is not

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influential on this disorder. Therefore it is a good theory on its own, but is an even better explanation when used with other theories of schizophrenia as an addition.

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