

# What is the likely role of genetic transmission in schizophrenia essay sample

[Environment](#)



Schizophrenia has been defined as a “ significant loss of contact with reality, often referred to as psychosis” (Butcher, Mineka, Hooley & Carson 2004 p. 458). Although schizophrenia is termed as one illness, it is more likely that it is a combination of disorders with “ a variety of etiologies, courses and outcomes’ (American Psychiatric Association 1997 p. 49). The symptoms of the illness include hearing voices and a conviction that external forces are interacting on the person. An overall disillusionment with life results from these symptoms and tends to lead to detrimental effects on everyday functioning. The causes of this disorder are unclear, however it has been shown to be a combination of environmental and genetic factors, the two interacting to cause the onset of schizophrenia.

The discussion of nature and nurture in any disorder is an important one, and just as relevant with schizophrenia. A meta analysis collaborated by Gottesman (1991) showed that there was over a 45% risk of identical twins both suffering schizophrenia if one already suffers from the disease (sharing 100% of the same genes). Fraternal twins (sharing 50% of the same genes) had a 20% chance of both suffering from the disease, this steadily decreases as the percentage of genes shared reduces. This is good evidence for a genetic basis to schizophrenia, but does not explain exactly why schizophrenia occurs, as if schizophrenia was completely genetically based, there would be 100% concordance rates in identical twins. We can assume that if a certain percentage is accounted for through genes, there will be other factors that influence the onset of schizophrenia.

Other factors that may influence the onset of schizophrenia may be the environment that a person lives in. It could be said that due to the genetic

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factor involved in genes, different environments could speed up or slow down the onset of schizophrenia dependant on the stimuli that a person comes into contact with. Another factor may be problems in birth and childhood, which could lead to abnormalities in adulthood, resulting in more predispositions to the disorder than someone without these disadvantages.

Schizophrenia may be detectable from a very early age, giving support for a genetic basis for the illness, as long-term environmental effects may not have had enough time to seriously affect the sufferer. Research by Walker in 1993 examined family home movies of 32 children who later developed schizophrenia. The examiners watching the videos were unaware that the children later developed an illness, and were asked to comment on the children's behavior. It was found that there were significant abnormalities in emotional and motor activities in the schizophrenia sufferers than their relative siblings. This may be some evidence for genetic basis for schizophrenia, due to there being signs of the disease from an early age. It may be overstating the evidence to suggest that schizophrenia may be detectable just from behavior, but a general sense that a person may develop a disorder in adulthood could be concluded from any unusual childhood activity. The reasons for this unusual behavior may result from earlier problems within the family or a genetic predisposition to illness.

Heston (1966) studied children whose mothers had been hospitalised due to schizophrenia. These children had been looked after by other family members or put into foster homes. It was found that these children had a 16% chance of becoming schizophrenic, compared to a 1% chance of a

random sample of similar children in the same area. It was also found that these children were significantly more likely to become mentally retarded, neurotic and psychopathic. This is evidence for the claim that schizophrenia comes from a multitude of other abnormalities, as well as that there may be a genetic cause that carries over generations. The reasons for the children of the mothers developing the disorder may be due to two factors, one is that the similar genes of the child to the mother lead to the predisposition of schizophrenia. Another is that the unrest of the child's development, due to the hospitalization of the mother and living with a new or altered family could lead to a difficult development which in turn could lead to disorders in adult life.

Tienari (1994) conducted a follow up study to Heston's 1966 research, examining the same children of mothers in Finland. As these children grew up, several factors were examined. One was communication deviance. This was the quality how easy speech and conversations were to follow. High communication deviance indicated low quality of speech. It was found that the children (who had now grown up) of the mothers who had been hospitalized due to schizophrenia were more likely to develop schizophrenia in high deviance conditions. The children in the control group (none with parents with mental disorders) were not affected by high or low deviance conditions. Evidence for the role of the gene-environment relation is shown in children in the experimental condition with low deviance families. These children had less signs of schizophrenia than the control group and the high deviance experimental group. This suggests that there may be a direct link between genetics and environment in the development of schizophrenia.

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From this research it could be said that although genes play a role in a person's susceptibility to developing the disease, it is the environment that produces the triggers that may act as a catalyst (or a deceleration) for the development of schizophrenia.

This is shown in the case of four quadruplets who all developed schizophrenia (Rosenthal 1963). They were named the 'Genain quads', Greek for 'dreadful gene'. All four of the quadruplets' developed the disease at separate points in their life, all with different levels of severity.

Environmental factors such as birth complications have been noted to be a catalyst for the onset of schizophrenia previously, and holds true in this case.

The last born, who suffered complications, was the first to develop schizophrenia and had the most severe case of the disease. One of the quadruplets', who had the least severe case, was able to marry and lower medication until not needed anymore, but was reported to be far from mentally healthy. This example may suggest that schizophrenia may indeed have a genetic basis, but different experiences with the environment may trigger the disease or accelerate its onset. It was said that the probability by chance alone of all four quadruplets developing schizophrenia was 1 in 1.5 billion births. There are however some weaknesses involved with twin studies. Results may be slightly overstated due to the similar environments that twins live in. Because of this, a genetic basis for disorders may be inflated where it is quite possible that the environment may have played an at least equal part in the onset of the disorder.

Twin and adoption studies are able to present relations between the genetics of a set of people and their susceptibility of developing the disease. A new method of looking at individual genes has become important more recently, that of molecular genetics. This looks at which gene (or genes) influence the onset of schizophrenia using a technique called segregation analysis and aims to locate which genes are influential in schizophrenia. Current research has singled out a few possible chromosomes which may be involved in schizophrenia. 22, 6, 7 and 1 among others have been investigated as to possible key areas where schizophrenia genes may lay (Brzustowicz et al. 2000). Molecular genetics may be able to detect a susceptibility to schizophrenia even before birth, if the genes involved in the disorder can be singled out and detected. However as schizophrenia involves a multitude of elements and abnormalities, this technique may take some time until a definitive set of genes is found.

From the research discussed, it is likely that a combination of genes and environmental conditions act as a base for the onset of schizophrenia. A predisposition to the illness through certain genes results in environmental exposures having effects that may either accelerate or slow down the onset of the disease. If certain genes are never 'turned on' at all by the environment, it could be said that the onset of schizophrenia may never occur at all. It is also possible that childhood and birth complications may have an effect on the speed of onset of the illness. Cannon et al (1993) found that it was only those who had a parent with schizophrenia as well as birth complications that later were found to have brain abnormalities, including enlarged ventricles within the brain.

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This could suggest that life events may have an exacerbated effect if a person has family members with the disease, giving evidence towards a combination of gene and environmental effects. Evidence so far has been inconsistent as to an exact proportion of genes and environment relating to the onset of schizophrenia. Because of this, it could only be concluded from the research discussed that just under half of all factors involved may be due to genes, with environmental conditions and any birth complications accounting for the rest of the possible onset of the disorder. In the future, molecular genetics may be able to better define the proportion of genes and the environment, which could lead to a more advanced treatment of the disorder. This may also lead to more advanced detection methods, as single genes influential in the onset of schizophrenia could be pinpointed and if possessed, the sufferer could be treated before the symptoms of schizophrenia become apparent.

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