

Genetic Explanation of Schizophrenia

Background

The genetic explanation assumes that behaviour is the result of inherited characteristics determined by our DNA. In order to measure the likelihood of genetic vulnerability psychologists use a 'concordance rate' which indicates whether people are more or less predisposed to developing Schizophrenia. For example a concordance rate of 100% would mean that there is a 100% chance that the disorder is genetically predisposed.

Many studies have shown that disorders such as Schizophrenia tend to run in families and the severity of a parent's disorder can influence the likelihood of the child developing the same disorder. However, we have to be cautious as this does not take into account factors within the environment. Evidence from twin studies gives us a good idea of the genetic relatedness, as identical twins (MZ) share 100% of their DNA; and studies argue they predominately show higher concordance rates.

Evidence

Twin and adoption studies have clearly indicated that genes play a major role in the etiology of Schizophrenia (**Gottesman 1991**).

Gottesman and Shields (1966) studied 57 pairs of twins of whom at least one had a diagnosis of Schizophrenia. They found that an identical twin was at least 42 times more likely to be schizophrenic than a person from the general population and a fraternal/ non-identical twin of the same sex was at least 9 times as likely.

Gottesman and Shields (1976) reviewed data from 711 participants in a longitudinal study, of which 210 were identical (MZ) twins and 319 were non-identical (DZ) twins. They suggest that concordance rates were as high as 58% for identical twins and 12% for non-identical twins. This offers a strong genetic basis for the development of Schizophrenia.

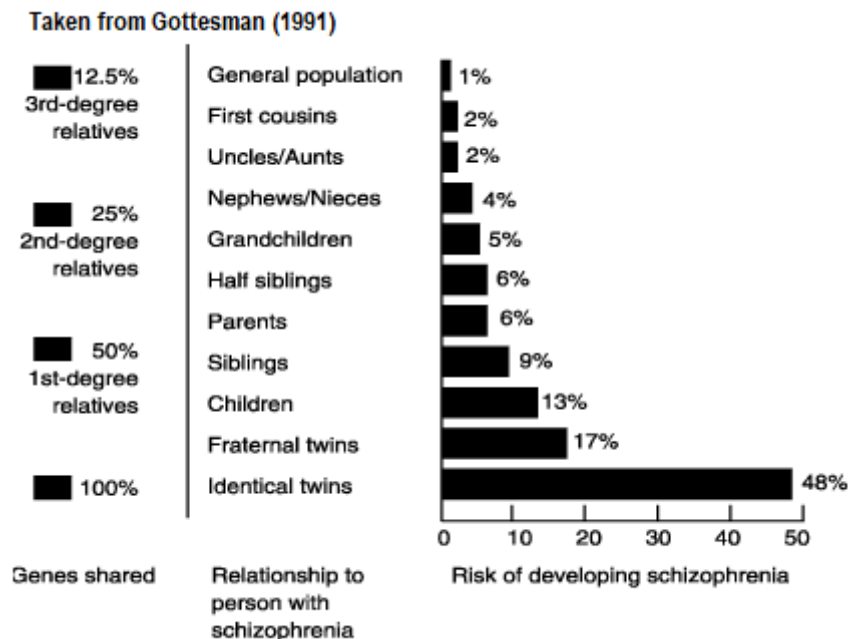
In a more recent study by **Gottesman et al (2010)** when investigating disorders in offspring with two psychiatrically ill parents, agreement was 27% for a diagnosis of Schizophrenia. This decreased to 7% with only one parent with a diagnosis and 0.86% in offspring with neither parent diagnosed.

Tienari et al (1969) identified 112 adopted children whose biological mothers had been clinically diagnosed with Schizophrenia and compared them to a control group (whose mothers had no history of mental illness). The children had all been adopted by the age of 4 years and were subsequently followed up eighteen years later for clinical diagnoses of Schizophrenia. The figures showed that 7% of the biological children of mothers who had been previously diagnosed with Schizophrenia had been diagnosed themselves compared with 1.5% of the control group. This supports the genetic explanation of Schizophrenia.



Application

If we can show the genetic vulnerability of Schizophrenia we could explore the likelihood of people developing the disorder. Statistics from studies show us the concordance rates in those with increased risk of a predisposition to Schizophrenia compared to others in the general population. This would mean we could assess the genetic vulnerability of first degree relatives to others.



Evaluation



One supporting argument is that if we can identify those more at risk of Schizophrenia by exploring their genetic vulnerability or the increased risk due to inherited characteristics; we may be able to assess their likelihood of developing the disorder earlier on in life, possibly preventing or reducing the risk of diagnosis. This has many useful applications in real life.



Also if family history is a positive indicator of Schizophrenia it may mean that patients could get access to biological interventions such as drug therapy (e.g. antipsychotic drugs). This increases the predictive validity.



This argument supports the nature debate in Psychology, which helps our understanding of the biological influences on behaviour.



However, if genes were the only explanation for Schizophrenia we could expect to see concordance rates of 100%, which given all the evidence to date is not the case. More so, many studies offer different rates, which highlights issues with the reliability of such findings.



Also twin studies are often used as supporting evidence on the basis of their strong genetic relatedness; however, this raises issues with generalisation, as not everyone is a twin.



Further studies have shown support for the nurture debate indicating that environmental influence may also play a part (e.g. the diathesis-stress model).

