**Schizophrenia Nature vs. Nurture**

Schizophrenia is a chronic psychiatric disorder that influences brain development and expresses a combination of cognitive dysfunctions and psychotic symptoms. Several kinds of research have been done on the pathogenesis and etiology of schizophrenia since the term was first coined in 1911 (Guo et al., 2019). Both nurture and nature prominently fit in understanding the onset of schizophrenia. Nature is the genetic and biological contributions, while nurture is the environmental and psychological contributions (St Clair & Lang, 2021). In the context of its pathophysiology, the interpretation of schizophrenia is generally supported by the fact that middle mental health problems frequently occur before the genesis of psychosis schizophrenia. Comprehensive research on its genetic architecture and risk environmental factors has also been delineated and identified (Stilo, Simona, & Murray, 2019). However, it is still challenging to explain the heritability of schizophrenia, and perceived environmental factors also cannot fully explain the variance unrelated to genetic factors. There is still much research to be done in this area, but there is evidence suggesting that both nature and nurture play a role in the development of schizophrenia. Since cerebral hypoxia and paternal age are nurture risk factors and the disease develops due to changes in the levels of serotine and dopamine, nature and nurture cause schizophrenia.

**Schizophrenia Nurture**

The broad definition of the nurture aspect of schizophrenia includes everything except genes. Scientists have defined environmental factors as the nutritional, social, and chemical environment in the womb during the expectancy period, stress, drug use, etc. The landscape of environmental and psychological risk factors is snowballing, suggesting myriad risk factors during earlier disease pathophysiology (Stilo, Simona, & Murray, 2019). Current epidemiological evidence shows that specific circumstances such as obstetric complications, migrant status, famine, and prenatal infections could increase the risk of developing schizophrenia. Other psychological evidence has strongly suggested cerebral hypoxia, severe pregnancy, and paternal age could be other nurture risk factors (St Clair & Lang, 2021). As much as the pathophysiological connection between nurture and risk factors remains tentative, environmental epidemiological evidence potentially forms a bridge to molecular genetic experiments. For example, screening genes in prenatal nutrition and performing serological tests from epidemiological samples.

Moreover, a scientific line of evidence suggests that the nurture aspect of schizophrenia consists of widely stochastic and epigenetic phenomena that cannot be detected with epidemiological models. However, various social factors, such as stressors, and physical factors, such as cannabis smoking, are implicated in leading to liability to schizophrenia (Stilo, Simona, & Murray, 2019). Some studies have also demonstrated that the replicable and solid literature evidence resulting from the disease is less substantial than postnatal factors (Guo et al., 2019)). However, this does not imply that prenatal factors are less important. Broad attention has been given to four prenatal environmental exposure; seasons of birth, prenatal exposure to infections, the time of conception, and effects of prenatal malnutrition. It is established that this prenatal factor causes causal molecular and biochemical mechanisms that translate environmental and genetic risk into schizophrenia phenotype.

**Schizophrenia Nature**

Studies on the modern twin and adoption research became instrumental in accepting the genetic hypothesis causation of schizophrenia, which later laid a search for molecular genetic risk factors. Classical genetics suggest that schizophrenia is likely to be genetically heterogenous, with multiple alleles and loci contributing to overall genetic risk. Genome-wide studies have revealed that the oligogenic model of the disease involves 10- 20 genes. A robust genetic context of hereditary posits that no doubt a relative with a history of psychiatric disease has been at risk of developing schizophrenia compared to the general population (Gejman et al., 2010). It should be noted that inheriting schizophrenia is not an estimation of the disease etiology but rather the cause of variation in population genetics. For instance, in monozygotic twins, the risk of schizophrenia and other related disorders is similar for the offspring of both affected and unaffected monozygotic twins. It implies that the unaffected one carries a heritable genetic factor for the disease without expressing it. Change in neurotransmitter mechanism is another biological evidence that explains the nature aspect of schizophrenia. Neurotransmitters are brain message carrier chemicals. No evidence links neurotransmitters and schizophrenia since the drugs perceived to later neurotransmitter levels relieve some schizophrenia symptoms (Gejman et al., 2010). However, the disease may develop due to changes in the levels of two neurotransmitters; serotine and dopamine. A widely accepted theory is if dopamine levels increase in certain brain parts, i.e., the mesolimbic pathway, mesolimbic functioning will increase. This results in hallucinations and delusions, which are early symptoms of schizophrenia (Gejman et al., 2010). A revised dopamine hypothesis argues that dopamine abnormal dopamine levels in the prefrontal brain and mesolimbic region are present in schizophrenia.

**Conclusion**

A modest amount has been learned about the environmental and genetic architecture of schizophrenia. The debate on the nature vs nurture aspects of schizophrenia is still on. However, there is a unified understanding that the root cause of schizophrenia is likely to be genetic and biological (nature). However, the contributing and trigger factors (nurture) are likely environmentally related. Scientists widely accept both theories. However, how nurture and nature interact to cause schizophrenia is still open to further research.

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