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# Direct Evidence of Viral Infection and Mitochondrial Alterations in the Brain of Fetuses at High Risk for Schizophrenia

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There is increasing evidences that favor the prenatal beginning of schizophrenia. These evidences point toward intra-uterine environmental factors that act specifically during the second pregnancy trimester producing a direct damage of the brain of the fetus [1]. The current available technology doesn't allow observing what is happening at cellular level since the human brain is not exposed to a direct analysis in that stage of the life in subjects at high risk of developing schizophrenia. Methods. In 1977 we began a direct electron microscopic research of the brain of fetuses at high risk from schizophrenic mothers in order to finding differences at cellular level in relation to controls. Results [Figure 1].

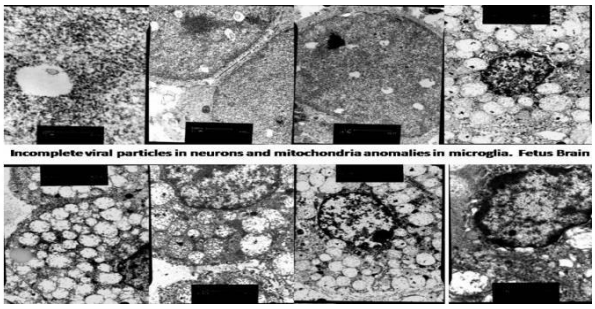


**Figure1:** Electron microscopic findings in the brain samples of the foetuses studied

In these studies we have observed within the nuclei of neurons the presence of complete and incomplete viral particles that reacted in positive form with antibodies to herpes simplex hominis type I [HSV1] virus, and mitochondria alterations [2]. Conclusion. The importance of these findings can have practical applications in the prevention of the illness keeping in mind its direct relation to the aetiology and physiopathology of schizophrenia.

A study of the gametes or the amniotic fluid cells in women at risk of having a schizophrenic offspring is considered. Of being observed the same alterations that those observed previously in the cells of the brain of the studied foetuses, it would intend to these women in risk of having a schizophrenia descendant, previous information of the results, the voluntary medical interruption of the pregnancy or an early anti HSV1 viral treatment as preventive measure of the later development of the illness. Schizophrenia is a severe neurodevelopmental disorder with genetic and environmental etiologies. Prenatal viral/bacterial infections and inflammation play major roles in the genesis of schizophrenia.

In this review, we describe a viral model of schizophrenia tested in mice whereby the offspring of mice prenatally infected with influenza at E7, E9, E16, and E18 show significant gene, protein, and brain structural abnormalities postnatally. Similarly, we describe data on rodents exposed to bacterial infection or injected with a synthetic viral mimic (PolyI:C) also demonstrating brain structural and behavioral abnormalities. Moreover, human serologic data has been indispensible in supporting the viral theory of schizophrenia. Individuals born seropositive for bacterial and viral agents are at a significantly elevated risk of developing schizophrenia. While the specific mechanisms of prenatal viral/bacterial infections and brain disorder are unclear, recent findings suggest that the maternal inflammatory response may be associated with fetal brain injury. Preventive and therapeutic treatment options are also proposed.

This review presents data related to epidemiology, human serology, and experimental animal models which support the viral model of schizophrenia. We present the accumulating evidence that prenatal exposure to a wide variety of viral and bacterial infections—or simply inflammation—may subtly alter fetal brain development, leading to neuropsychiatric consequences for the child later in life. The link between influenza infections in pregnant women and an increased risk for development of schizophrenia in their children was first described more than 30 years ago. Since then, evidence suggests that a range of infections during pregnancy may also increase risk for autism spectrum

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disorder and depression in the child.

Subsequent studies in animal models demonstrated that both pregnancy infections and inflammation can result in direct injury to neurons and neural progenitor cells or indirect injury through activation of microglia and astrocytes, which can trigger cytokine production and oxidative stress. Infectious exposures can also alter placental serotonin production, which can perturb neurotransmitter signaling in the developing brain.

Schizophrenia is a chronic mental illness affecting approximately 1 percent of the population. Beginning in early adulthood, schizophrenia typically causes a dramatic, lifelong impairment in social and occupational functioning. From a public health standpoint, the costs of treatment and lost productivity make this illness one of the most expensive disorders in medicine. Despite the tremendous economic and emotional costs, research on schizophrenia lags far behind that on other major medical disorders. A primary impediment to developing more effective treatment is the limited understanding of the etiology and neurobiology of this disorder. New technologies, such as neuroimaging and molecular genetics, are removing the obstacles that once blocked major progress in the field. Although the stigma associated with the illness has not yet been eliminated, these new techniques have markedly altered the conception of the nature of schizophrenia.

One of the most rapidly changing fields is genetics. Family, twin, and adoption studies have clearly shown that genes play a prominent role in the development of schizophrenia. Estimates of heritability typically range from 50 to 85 percent. Initial attempts to isolate major genes using linkage studies were unsuccessful, but more recent approaches using increasingly sophisticated methods have uncovered several chromosomal regions that may harbor genes of minor effect. It seems likely that schizophrenia is the result of the interaction of many genes, some of which also interact with environmental factors. Investigations of environmental factors have looked at the role of stress, viruses, obstetrical complications, and in utero insults, among others. None of these have been definitively shown to be causative. It is possible that different combinations of genetic and environmental factors affect specific neurobiological systems, leading to a final common pathway of neural dysfunction.

References

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