SHORT COMMUNICATION

Concrete evidence of viral infection and mitochondrial alterations in fetuses at significant risk for schizophrenia in the brain

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ABSTRACT

There is a substantial body of science that suggests schizophrenia begins in the womb. These findings point to intrauterine environmental variables acting especially during the second pregnancy trimester, causing direct fetal brain injury. Because the human brain is not exposed to direct analysis in that period of development in persons at high risk of developing schizophrenia, existing technology does not allow for observation at the cellular level. In 1977, a direct electron microscopic study of the brains of fetuses at high risk from schizophrenic mothers to see if there were any cellular changes between them and controls.

Keywords: *Pregnancy; Womb; Schizophrenia; Influenza*

INTRODUCTION

There is a research that suggests schizophrenia starts in the mother's womb only. These findings hint to intrauterine environmental influences acting especially during the second pregnancy trimester, causing direct harm to the fetus's brain [1]. Because the human brain is not subjected to a direct analysis in that period of life in persons at high risk of developing schizophrenia, existing technology does not allow for observation of what is happening at the cellular level. Within the nuclei of neurons, these analyses discovered the presence of full and partial viral particles that reacted positively with antibodies to the Herpes Simplex Virus Type I (HSV1) virus, as well as mitochondrial changes [2]. Given its direct link to the etiology and physiopathology of schizophrenia, the significance of these results can have practical applications in the prevention of the illness.

A study of amniotic fluid cells in women who are at risk of miscarriage is considered. It is regarded having a schizophrenic offspring [3]. If the same alterations in the cells of the brain of the studied fetuses were observed, it would suggest to these women at risk of having a schizophrenia descendant, prior knowledge of the results, a voluntary medical interruption of the pregnancy, or an early anti HSV1 viral treatment as a preventive measure against the illness's later development. Schizophrenia is a severe neurodevelopmental illness that can be caused by both hereditary and environmental factors. Inflammation and prenatal viral/bacterial infections have important roles in the development of schizophrenia.

The offspring of mice prenatally infected with influenza at E7, E9, E16, and E18 show severe gene, protein, and brain structural abnormalities post-natally, according to a viral model of schizophrenia evaluated in mice described in this paper [4]. Similarly, we provide findings from animals that were infected with bacteria or injected with a synthetic viral mimic which showed brain anatomical and behavioral abnormalities. Human serologic evidence has also proven critical in bolstering the viral explanation of schizophrenia. People who are born seropositive for bacterial and viral agents have a much higher chance of developing schizophrenia. While the exact processes of prenatal viral/bacterial infections and brain disease remain unknown, new research suggests that the maternal inflammatory response may be linked to fetal brain damage [5]. There are additional therapy alternatives that are both preventative and therapeutic.

Data from epidemiology, human serology, and experimental animal models are presented in this article to support the viral hypothesis of schizophrenia. Prenatal exposure to a wide range of viral and bacterial

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infections-or even inflammation-may gradually change fetal brain development, leading to neuropsychiatric effects for the kid later in life, according to mounting evidence [6]. More than 30 years ago, a relationship was discovered between influenza illnesses in pregnant women and an elevated risk of schizophrenia in their offspring. Since then, evidence has accumulated that a variety of illnesses contracted during pregnancy may raise the child's risk of autistic spectrum disorder and depression [7].

CONCLUSION

Following that, investigations in animal models revealed that both pregnancy infections and inflammation may cause direct harm to neurons and neural progenitor cells, as well as indirect injury via microglia and astrocyte activation, which can cause cytokine production and oxidative stress. Infectious exposures can disrupt neurotransmitter transmission in the developing brain by altering placental serotonin synthesis.

Schizophrenia is a long-term mental condition that affects about 1% of the population. Schizophrenia often produces a substantial, permanent impairment in social and vocational functioning beginning in early adulthood. The expenses of therapy and lost productivity make this condition one of the most expensive disorders in medicine from a public health aspect. Despite the high financial and emotional consequences, schizophrenia research lags considerably behind those of other major medical conditions. The lack of knowledge about the etiology and neurology of this condition is a major roadblock to creating more effective treatments. New technologies, including as neuroimaging and molecular genetics, are reducing the barriers that previously stymied considerable advancement in the field [8].

Genetics is one of the most quickly evolving areas. Genes play a significant effect in the development of schizophrenia, according to family, twin, and adoption studies. Heritability estimates generally vary from 50%-85%. Linkage studies were initially ineffective in isolating important genes, but more recent techniques employing increasingly sophisticated methods have revealed multiple chromosomal areas that may hold genes of little influence. Schizophrenia appears to be the outcome of a complex combination of genes, some of which interact with environmental variables. Stress, infections, obstetrical problems, and in utero insults have all been investigated as potential environmental influences. None of these have been proven to be causal in any way.

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