



## Direct evidence of viral infection and mitochondrial alterations in the brain of fetuses at high risk for schizophrenia

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### Abstract:

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 . 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. 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. 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 indispensable in supporting the viral theory of schizophrenia. Individuals born seropositive for bacterial and viral agents are at a significantly elevated risk of developing schizophrenia.

### Biography:

Segundo Mesa Castillo. As Specialist in Neurology, he worked for 10 years in the Institute of Neurology of Havana, Cuba. He has worked in Electron Microscopic Studies on Schizophrenia for 32 years. He was awarded with the International Price of the Stanley Foundation Award Program and for the Professional Committee to work as a fellowship position in the Laboratory of the Central Nervous System Studies, National Institute of Neurological Diseases and Stroke under Dr. Joseph Gibbs for a period of 6 months, National Institute of Health, Bethesda, Maryland, Washington D.C. USA, June 5, 1990. At present he is member of the Scientific Board of the Psychiatric Hospital of Havana and give lectures to residents in psychiatry.

### Publication of speakers:

1. Yolken RH, Torrey EF. Viruses, schizophrenia and bipolar disorders. *Clin Microbiol Rev* 1995; 8: 131-145.
2. Mesa CS. An ultrastructural study of the temporal lobe and peripheral blood in schizophrenic patients. *Rev Neurol* 2001; 33: 619-623.

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