

'LUHFW HYLGHQFH RI YLU DO LQIHFWRQ DQG PLWRFKRQGULDO  
VFKLJRSKUHQLD

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**Background:** There is increasing evidence that favors 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.

**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 amniotic fluid cells in women at risk of having a schizophrenic offspring is considered. If being observed the same alterations that those observed previously in the cells of the brain of the studied fetuses, it would intend to these women in risk of having a schizophrenia descendant. From 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.

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