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Journal of Mental Health and Psychiatry

Perspective

Evidence of Prenatal Viral Impact and Mitochondrial Disruption in Schizophrenia-Susceptible Fetuses

Segundo Mesa Castillo*

Psychiatric Hospital of Havana, CA 10800, Cuba

DESCRIPTION

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. 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 a etiology 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 fetuses, 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.

CONCLUSION

The findings presented in this study offer compelling support for the hypothesis that schizophrenia may have a prenatal origin, particularly linked to intrauterine environmental factors during the second trimester of pregnancy. By employing direct electron microscopic analysis of fetal brain tissue from pregnancies at high risk due to maternal schizophrenia, the research has provided rare cellular-level evidence that is otherwise inaccessible during this critical developmental period. The detection of complete and incomplete viral particles within neuronal nuclei specifically those reacting positively to herpes simplex virus type 1 (HSV1) along with notable mitochondrial abnormalities, suggests a possible viral etiology contributing to the neuropathology of schizophrenia. These observations have profound implications for the future of schizophrenia research, prevention, and early intervention strategies. If similar alterations can be identified in gametes or amniotic fluid cells from women at high risk of giving birth to offspring with schizophrenia, this could open new avenues for early detection of vulnerability to the disorder. Such findings would enable the consideration of preventive interventions, such as early antiviral treatments targeting HSV1 or, in extreme cases, the ethical and voluntary decision regarding medical interruption of pregnancy based on informed risk. While these applications remain theoretical and would require extensive validation and ethical scrutiny, they mark a significant step forward in understanding schizophrenia as a neurodevelopmental disorder with potential infectious or environmental triggers during gestation. Further research is essential to confirm these preliminary findings, understand the mechanisms by which HSV1 and mitochondrial dysfunction may contribute to schizophrenia onset, and explore safe, effective preventative measures. Ultimately, this study lays the groundwork for an innovative shift in focus from post-onset management of schizophrenia to prenatal prevention and risk reduction in vulnerable populations.

***Correspondence to:** Castillo SM, Psychiatric Hospital of Havana, CA 10800, Cuba, E-Mail: segundo@infomed.sld.cu

Received: April 04, 2025; **Manuscript No:** JMPH-25-7974; **Editor Assigned:** April 07, 2025; **PreQc No:** JMHP-25-7974(PQ); **Reviewed:** April 22, 2025; **Revised:** April 29, 2025; **Manuscript No:** JMHP-25-7974(R); **Published:** June 02, 2025

Citation: Castillo SM (2025). Evidence of Prenatal Viral Impact and Mitochondrial Disruption in Schizophrenia-Susceptible Fetuses. J Ment Health Psychiatry, Vol.1 Iss.1, June (2025), pp:7.