

# 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. 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. 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.

There is expanding confirmations that favor the pre-birth start of schizophrenia. These confirmations highlight intra-uterine ecological variables that demonstration explicitly during the second pregnancy trimester creating an immediate harm of the cerebrum of the hatchling. The current accessible innovation doesn't permit seeing what's going on at cell level since the human cerebrum isn't presented to an immediate investigation in that phase of the life in subjects at high danger of creating schizophrenia. Techniques. In 1977 we started an immediate electron tiny examination of the cerebrum of hatchlings at high hazard from schizophrenic moms so as to discovering contrasts at cell level according to controls. Results. In these examinations we include saw inside the cores of neurons the nearness of complete and fragmented viral particles that responded in positive structure with antibodies to herpes simplex hominis type I [HSV1] infection, and mitochondria changes. End. The significance of these discoveries can have pragmatic applications in the avoidance of the sickness remembering its immediate connection to the etiology and physiopathology of schizophrenia. An investigation of amniotic liquid cells in ladies in danger of having a schizophrenic posterity is thought

of. Of being watched similar changes that those watched beforehand in the cells of the cerebrum of the considered hatchlings, it would plan to these ladies in danger of having a schizophrenia relative, past data of the outcomes, the deliberate clinical interference of the pregnancy or an early enemy of HSV1 viral treatment as preventive proportion of the later advancement of the sickness.

Morphological changes of mitochondria might be identified with metabolic and vitality lack in neurons in Alzheimer's illness and other neurodegenerative issue. Mitochondrial brokenness is likewise a sign of beta peptide actuated neuronal poisonousness in Alzheimer's illness. A general change in glucose use, expanded oxidative pressure, and  $Ca^{2+}$  deregulation are extra metabolic deformities in the AD cerebrum that may likewise be related with deficient mitochondrial work the outcome is a pattern of expanded mitochondrial brokenness causing expanded oxidative harm until the cell vitality flexibly falls underneath the edge for cell endurance. In a progression of studies on the morphological and morphometric estimation of mitochondria in Alzheimer's ailment, by electron microscopy we saw generous morphological and morphometric changes in the neurons of the hippocampus, the acoustic cortex, the frontal cortex, the cerebellar cortex, the climbing strands, the thalamus, the globus pallidus, the red core and the locus coeruleus. The morphological adjustments comprised of significant changes of the mitochondrial cristae, collection of osmiophilic material, and reduction of their size, in correlation with the typical controls. Mitochondrial modifications were especially unmistakable in neurons, which indicated loss of dendritic spines and shortened form of the dendritic arborization. The ultrastructural investigation of enormous number of neurons in the thalamus and the red core uncovered that the mitochondrial modifications didn't exist together with cytoskeletal pathology and aggregation of amyloid stores, however they were conspicuous in neurons, which exhibited discontinuity of the cisternae of the Golgi mechanical assembly. Morphometric investigation indicated that mitochondria are essentially decreased in Alzheimer's illness. The connection between the site and degree of mitochondrial variations from the norm and the synaptic adjustments proposes a personal and early relationship between these highlights in Alzheimer's sickness.