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Electron microscopic studies of brain tissue in fetuses from schizophrenic mothers

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The neurodevelopmental theory in the aetiology of schizophrenia is considered one of the most consistent at present. Evidence from epidemiological and neuropathological studies indicates that the pathogenic process that culminate in the development of schizophrenia are initiated early in life and has been associated with a variety of prenatal environmental insults to the developing brain, including infection. Although the infectious agents have been proposed as one of the risk factors for schizophrenia the data on the association of a specific infectious agent with prenatal brain evidence is absent. Understanding of the structural abnormalities would allow a better identification of neurodevelopmental processes that contribute to risk for schizophrenia. We have hypothesized that at ultra-high-risk fetuses would have alterations at cellular level that would let us differentiate them to the comparison subjects. A reappraisal of our ultra-structural studies carried out in samples of the left temporal lobe of fetuses at ultra-high risk of developing schizophrenia is presented. The findings obtained are compatible with an active infection of the central nervous system by herpes simplex hominis type I [HSV1] virus. The present results are the first direct evidence that demonstrate the presence of this virus in the central nervous system of fetuses from schizophrenic mothers in the critical period of fetal development. The importance of this finding can have practical applications in the prevention of the illness keeping in mind its direct relation to the aetiology and physiopathology of schizophrenia.