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

Dr. Segundo Mesa Castillo
Psychiatric Hospital of Havana, CA 10800, Cuba.
Email Id-segundo@infomed.sld.cu

Abstract

Introduction: 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.

Background: Schizophrenia is often accompanied by systemic inflammation and cell-mediated immune (CMI) activation as shown by increased levels of cytokines, interleukin 2 receptors (IL-2Rs), interleukin 1 receptor agonist (IL-1RA) (Lin et al., 1998; Maes et al., 2000; Miller et al., 2011; Zhang et al., 2004), acute phase reactants such as IL-1β, IL-6, and transforming growth factor (TGF)-β in plasma of subjects with schizophrenia (Meyer, 2011, 2013–this issue; Miller et al., 2011). Cytokines may be divided into various categories based on their role in inflammation and their derivation. Pro-inflammatory cytokines, such as interleukin (IL)-1β, IL-6, and tumor necrosis factor (TNF)-α, are synthesized upon activation of the innate immune system and are responsible for febrile reactions, activation of phagocytes, vascular permeability, and release of inflammatory mediators, all of which are essential to the inflammatory response (Meyer, 2011, 2013–this issue). Many anti-inflammatory cytokines, such as IL-10 and TGF-β1, act to inhibit pro-inflammatory cytokine production, and these two factors in particular have been shown to exert an anti-hypoxic effect.

Method:- rain structural abnormalities in PolyI:C exposed offspring have also been noted. Abnormal proliferation of cortical progenitor cells and impaired expression of Pak6, a regulator of gene transcription, have been found in the cerebral cortex in the offspring of mice exposed to PolyI:C injection (Soumiya et al., 2011). Additionally, altered development of the cerebellum (Shi et al., 2009), has been observed. Other groups have demonstrated concordant neuroanatomical abnormalities, such as ventriculomegaly, between offspring of rodents exposed to PolyI:C during pregnancy and patients with schizophrenia. Prenatal PolyI:C exposure has also been shown to reduce the number of Reelin and Parvalbumin positive cells in the medial prefrontal cortex following exposure on E9 and E17 and in the hippocampal formation and dentate gyrus following exposure on E9

Results: 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.

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.

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