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

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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-1 [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.

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Combat cancer with IgE: Molecular and comparative aspects

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Immunotherapy of cancer is becoming more and more important in clinical oncology. Especially monoclonal antibodies of the IgG class are commonly used, due to their tumoricidal effects, which have been proven for many indications, together with their good tolerability and safety profile. Recently, also novel antibodies have entered clinical oncology: this engineered IgGs, designed to enhance ADCC are highly successful in clinical trials. The next logical step would be the implementation of other Immunoglobulin classes than IgG. We demonstrated in several studies, that IgE antibodies, specific against tumor-associated antigens are capable to induce apoptosis of tumor cells and can mediate high levels of ADCC. In addition, they are also well tolerated *in vivo*. Thus, monoclonal IgE antibodies could be valuable options for combating cancer. As the IgE biology of man and mice are highly distinct, novel approaches for further investigations of these antibodies are needed. For this reason, we pursue comparative oncology studies by generating dog monoclonal antibodies in order to establish immunotherapy options also for dog cancer patients. Finally, data from these clinical trials will add valuable information on the full potential of IgE antibodies in clinical oncology.

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