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Elevated titres of interferon (IFN) were found in serum from 24.4% of 82 patients with psychosis compared to only 3.1% of 64 controls (p= .0003). Positive patients were more likely to have recent onset/exacerbation of their illness and to be on low dose or no medication. No interferon was detected in the cerebrospinal fluid of 65 patients or 20 controls. These findings suggest immunological abnormalities or viral infections in some patients with psychosis.

Abstract 30. Interferon production in blood cell cultures of schizophrenic patients
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Complementary to the virus hypothesis of schizophrenia, immunological factors may play a role in the genetic determined vulnerability to the disease. The individual's ability to produce interferon in response to a virus infection will probably have a significant influence on the course of infections. For this reason we investigated possible differences between schizophrenic patients and healthy individuals in their ability to produce interferon. Using a whole-blood technique, lymphocyte proliferation responses (LP) and interferon titres, with and without stimulation by PHA, Con A, C. parvum or NDV were determined in 30 schizophrenic patients and 30 controls. The schizophrenic patients produced significantly less interferon than controls after stimulation by the alpha interferon inducers C. parvum or NDV and by the gamma interferon inducers PHA and Con A. Differences in the LP test were also evident. These results indicate a defect in interferon production in leucocyte cultures of schizophrenic patients. They furthermore are the first evidence that a disturbance in the interferon system may exist in schizophrenia. However, as yet is has not been vigorously excluded that drugs taken by the patients have contributed to the data observed.

WORKSHOP 1: 10 Abstracts

Animal Models in the Study of the Role of Viruses in Etiology of Mental Diseases

Abstract 31. Animal models in behavioral neurovirology
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The nature of viral-induced behavioral abnormalities produced by scrapie, murine cytomegalovirus and herpes simplex will be considered. The data show that each of these pathogens are capable of producing more than one profile of behavioral effects. Both scrapie and herpes simplex produce different behavioral syndromes in mice of different genetic backgrounds. Likewise, age at infection is a determinant of the behavioral effects of cytomegalovirus and herpes simplex. Localization of herpes simplex infection within the CNS will also be considered. Both the nature of the behavioral syndrome as well as that of the underlying neuropathology depend upon the specific locus of infection. Such results show that host and viral characteristics interact to produce the particular behavioral abnormalities observed.

Abstract 32. Borna virus encephalitis in rats: behavioral changes of aggression leading to apathy
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Borna disease virus is an unclassified agent which causes sporadic fatal encephalitis in horses and sheep in Germany. Pathogenesis of the infection in rats showed that the agent replicated exclusively in the nervous system with a predilection for neuronal groups in the
cerebral hemispheres, including the olfactory bulbs, septal nuclei, cingulate gyrus, hippocampus and hypothalamus. Infection of 4-week-old rats resulted in a persistent infection in the CNS. Animals developed a transient immunologically specific encephalitic response which caused bizarre, hyperactive and aggressive behavior. Rats recovered from the encephalitis with hydrocephalic pathological deficits resulting from lysis of some infected neurons. Behavior hence was characterized by inactivity and passiveness. Infection in the brain persisted. Immunosuppression of the mice resulted in very similar virus-cell interaction but no encephalitis occurred and animals did not become ill. The behavioral disease was therefore due to responses of infected neurons triggered by specifically sensitized inflammatory cells.

Abstract 33. Transmission studies of neurological and psychiatric disease in the marmoset
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The behaviour of 6 marmosets (Callithrix jacchus) inoculated intracerebrally with CSF from schizophrenic patients was compared over a 2½ year period with the behaviour of marmosets inoculated with CSF from patients with neurodegenerative disease or from patients undergoing spinal anaesthesia who suffered from no neurological or psychiatric disorders. Animals inoculated with CSF from psychiatric or neurological patients became progressively and significantly less active than animals inoculated with CSF from other patients although, post-mortem, no consistent differences between groups of animals were found in light- or electron-microscopic brain examination, brain biochemical analysis or viral isolation.

In another experiment all 4 marmosets inoculated intracerebrally with brain material from a patient with the Gerstmann-Sträussler syndrome (G.S.S.) developed a spongiform encephalopathy which was indistinguishable from that shown by another 4 marmosets inoculated with brain material from a typical case of Creutzfeldt-Jakob disease in a comparable time course of 20-32 months. The familial occurrence of a viral disease in G.S.S. will be discussed.

Abstract 34. Inoculation of schizophrenic brains to rodents and primates: behavioral and neuropathological observations
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J. Langloss and C.J. Gibbs, Jr.
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To test the possibility that schizophrenia might result from a transmissible agent analogous to the CJD agent, homogenized brain from various cortical and subcortical regions of 10 patients with chronic schizophrenia was inoculated intracerebrally into 37 primates and 22 rodents. No gross behavioral peculiarities were noted in routine observations by veterinarians and animal technicians over a five year follow-up period. One squirrel monkey and one guinea pig developed ataxia and tremor at 44 and 20 months respectively. Reinoculation with original inoculum has been negative at 40 months. Nineteen rodents and 11 primates died during the follow-up period. Histopathological examination of rodent brains showed cerebellar and hippocampal gliotic changes in 5/9 experimental cases and 3/9 controls. Similar findings were not observed in primate brains which could not be differentiated from controls. While these results do not support a transmissible agent model for schizophrenia analogous to CJD, it is not conclusive evidence against a virus as certain known viral illnesses also cannot be transmitted (e.g. SSPE).

Abstract 35. Coronavirus infection in rats: induction of an autoimmune response against brain antigen
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