density). Preliminary results indicate that the expression of these GAA is preferentially increased during the G2/M phase of cell cycle (FACS analysis). GAA positivity was observed in every passage of the cell lines examined, whereas GFAP was indetectable after a few passages and neurofilaments (all lines positive) were drastically reduced in only one line. The Ags detected by GE2 and CG12 MoAbs are 50KD and 120KD surface structures respectively. Experiments are underway to further investigate the structure and biological significance of GE2 and CG12 GAA.

## A Study of Persistent Viral Infections Using Nude Mice and a Temperature-Sensitive Mutant of Vesicular Stomatitis Virus

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Although a host-parasite relationship between persistent viruses and immunocompetent mammals has been recognized for many years, how this symbiotic relationship is maintained is not known. We have developed a model system to study the roles of both the host and the parasite by using nude (nu/nu) mice and a temperature-sensitive mutant of vesicular stomatitis virus (tsG31 VSV). When the nude mice were intracerebrally infected with the temperature-sensitive VSV, all animals died after 12 days of infection. If, however, the mice were reconstituted with syngeneic T-lymphocytes, 70% of the animals survived for at least 21 days. The amount of antibody against the virus produced in the mice was dependent upon how many T-lymphocytes were used to reconstitute the animals. Infectious VSV was isolated from the brain of all the infected animals that did not have high titers of antibody in their sera. When the brain isolated VSV was inoculated into Swiss outbred mice, most of the animals died, in contrast to tsG31 VSV where less than 5% of the animals succumbed. To further characterize the brain isolated virus, the virus was cloned and analyzed for temperature-sensitivity. All the clones were less temperature-sensitive than the original tsG31 VSV, and most of the clones caused a more aggressive disease when infected into Swiss outbred mice. Unlike the tsG31 VSV, most of the clonal isolates had a temperature-sensitive defect in RNA synthesis. Studies are currently under way to learn how T-lymphocyte reconstituted nude mice, without detectable antibody against VSV, survive when VSV is replicating in their central nervous system.

## CORONAVIRUS JHM INDUCED DEMYELINATING ENCEPHALOMYELITIS IN RATS: ANALYSIS OF THE INTRATHECAL IMMUNE RESPONSE.

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Intracerebral infection of Lewis rats with the murine coronavirus JHM may result in clinically relevant encephalomyelitides accompanied by significant primary demyelination in the CNS. To date, there is growing evidence that beside the genetic background of the host and the virus, the immune system determines largely the course of this disease process.

Analysis of the intrathecal humoral immune response in diseased Lewis rats revealed low-titered JHM-specific antibody responses of oligoclonal

nature. However, resistant BN rats showed vigorous intra-blood-brain barrier synthesis of a broad isoelectric pattern of JHM specific antibodies, indicating a protective character of brain resident, JHM-specific plasma cells. To evaluate topographical relationships between cells of the lymphoid system and virus infected target cells in situ, serial cryostate sections of CNS material were immunostained by a comprehensive panel of monoclonal antibodies, detecting astrocytes, oligodendroglia cells, nerve cells, B lymphocytes, macrophages, T helper and non-helper cells, class II proteins and virus specific proteins. Stained lesions were analyzed by computer-aided cytophotometry and digitized pictures demonstrated complex arrangements of the involved cell types.

The data suggest, that in the absence of a significant humoral immune response to the virus, macrophages and T cells play an important role in the demyelination process.

## Suppression of Passive Transfer Acute Experimental Myasthenia by $F(ab')_2$ Fragments.

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Passive transfer of anti-acetylcholine receptor monoclonal antibody (mAb) results in acute experimental myasthenia gravis (EAMG), characterized by an acute transient weakness of skeletal muscles, inflammatory cells (mostly macrophages) at the muscle endplate, and decremental electromyographic (EMG) responses. Recently, we have shown a correlation between the ability of an individual mAb to activate complement by the classical pathway *in vitro* and the EAMG-evoking potential of that antibody *in vivo*. In the present study, $F(ab')_2$ fragments of a potent EAMG-inducing mAb (132A) were used to suppress the induction by intact mAb of passive transfer EAMG. Female Lewis rats were injected by cardiac puncture with either intact mAb (5 mg/kg) or with intact mAb (5 mg/kg) plus $F(ab')_2$ fragments (10 mg/kg). At 24 h after injection rats receiving intact mAb alone were moribund or unable to stand, showed massive inflammation at muscle endplates and had decremental EMG responses of up to 60%. Rats receiving $F(ab')_2$ as well as intact mAb were able to ambulate, had minimal inflammation at muscle endplates and had no or, in the case of one animal, moderate decrement. All rats had circulating mAb as determined by ELISA. These findings suggest that $F(ab')_2$ fragments protected muscle endplates by competing for binding sites with intact mAb, thus decreasing the surface density of mAb Fc below that required to fix complement and trigger an inflammatory episode.

## LEUKOCYTE DIFFERENTIATION ANTIGENS IN THE THYMUS AND PERIPHERAL BLOOD OF MYASTHENIC PATIENTS BEFORE AND AFTER THYMECTOMY.

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An extensive panel of different monoclonal antibodies was used to identify (with single or dual simultaneous standard immunofluorescence technique) 15 leukocyte differentiation antigens (CD1,CD3,CD4, CD5,CD7,