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infusion, indicating that these nodes have a significant role in systemic immunization. (Supported by USPHS Grant NS-11050.)

Role of Cellular Immune Factors in Coronavirus A59 Induced Demyelination. M.J.M. Koolen*, M.J. Buchmeier and C.J. Lucas*. (Dept. of Immunology, Scripps Clinic and Research Foundation, LaJolla, CA, and *Central Lab Netherlands Red Cross Blood Transfusion Service, incorporating the Lab of Exp and Clin Immunology of the University of Amsterdam, Amsterdam, The Netherlands)

Cellular immune factors involved in mouse hepatitis virus (MHV) strain A59 and a temperature-sensitive mutant (ts-342)-induced demyelination were studied in normal and athymic nu/nu BALB/c mice as well as in mice depleted of a specific subset of T lymphocytes in vivo.

Intracerebral inoculation of normal BALB/c mice with 10^5 PFU of ts-342 resulted in prolonged infection of the central nervous system, whereas 100 PFU of the wild type virus were lethal.

In athymic nu/nu mice, both wild type virus and ts-342 caused a fatal hepatitis suggesting that cellular immune factors are involved in the protection of mice against lethal MHV-infection.

Furthermore, significant levels of proliferation, measured as 3H-thymidine incorporation, were observed when splenocytes isolated from ts-342 infected normal mice were cultured in the presence of either viral antigen or myelin basic protein (MBP). The responder cells were shown to be T lymphocytes, and in vivo depletion of the L3T4 population reduced the proliferative response to MBP to baseline levels.

Immunoblot Analysis of Anti-AChR Antibodies in Myasthenia Gravis. R. Mantegazza, P. Romagnoli, F. Baggi, O. Simoncini, D. Neumann*, F. Cornelio and S. Fuchs*. (Dept. of neuromuscular Disease, Milan, Italy and *Dept. of Chemical Immunology, The Weizmann Institute of Science, Rehovot, Israel)

Fine specificity of anti acetylcholine receptor antibodies (a-AChR-Abs) in Myasthenia Gravis (MG) and their relationship to the pathogenesis are not completely defined. By the mean of immunoblotting techniques we tried to achieve more insight in the composition of the different Ab-subpopulations. AChR from Torpedo California (T-AChR) was purified, blotted onto nitrocellulose paper, probed with sera from patients in different clinical conditions and revealed on autoradiography by means of Prot A 125I. 45 patients were analyzed with this method and the double immunoprecipitation conventional method. Sera from differently affected patients exhibited different binding patterns to the subunit of T-AChR.

Higher sensitivity was displayed by blot technique. Qualitative analysis showed that higher immunogenic epitopes are shared by α, β and δ subunits; γ-subunit showed only a mild binding capacity. Sera from patients affected by ocular forms of MG displayed a greater binding to α-subunit while in sera from generalized MG such a preponderance was not found. Stronger positivity for α and δ subunit was found among younger patients (i.e., onset of MG before 40 yrs).

IgG Subclasses of Antibodies Reacting With HIV and Myelin Basic
Protein in CSF from HIV Infected Patients. T. Mathiesen, A. Sonnerborg and B. Wahren. (Dept. of Neurosurgery, Karolinska Hospital, Stockholm, Dept. of Virology, National Bacteriological Laboratory, Stockholm, Dept. of Infectious Diseases, Roslagstuuls Hospital, 11489 Stockholm, and The Central Microbiological Laboratory of Microbiology of Stockholm County Council, 10122 Stockholm, Sweden)

Serum and cerebrospinal fluid (CSF) from 15 patients infected with Human Immunodeficiency Virus (HIV) were analyzed by IgG subclass enzyme linked immunosorbent assays to detect antibodies reactive with HIV and myelin basic protein (MBP). All samples were pretreated to solubilize immune complexes. Three patients had encephalitis or dementia (group I), five had polyneuropathies (group II) and three had cognitive impairment (group III). The remaining four patients were neurologically asymptomatic (group IV). All HIV infected patients had HIV IgG1 in their CSF. HIV IgG2 was found in CSF from 2 patients, HIV IgG3 in 2 and HIV IgG4 in 3 patients. HIV IgG subclass expression in CSF became restricted to IgG1 in AIDS patients. Anti-MBP IgG1 was found in 6 CSF samples, IgG2 in one CSF sample and IgG3 in 6 CSF samples. All patients in group I had evidence of intrathecally synthesized anti-MBP IgG2 or 3. Intrathecally synthesized IgG was also found in three group II, one group III and one group IV patient. This is the first report of CSF anti-MBP IgG in HIV infected patients. It could only be detected after solubilization of immune complexes. In demyelinating diseases anti-MBP antibodies may correlate with disease activity but have not been shown to be pathogenic. Our findings add HIV infection to a group of diseases in which the central nervous system is attacked and anti-MBP antibodies are synthesized.

Neuronal Migration Defects in Mutant Mice with Immune System Dysfunctions. R.S. Nowakowski, K.K. Varaklis and P.R. Patrylo. (Dept. of Anatomy, UMDNJ-Robert Wood Johnson Medical School, Piscataway, NJ)

We have observed that four single autosomal mutations that are known to produce immune system dysfunctions also have pleiotropic effects on neuronal migration during the development of the central nervous system.

Two of these mutants, motheaten (meY/meY) and beige (bg/bg) mice have similar disorders in the hippocampal formation and the cerebellar cortex. In the hippocampal formation of both motheaten and beige there is a disruption of the migration of granule cells resulting in a trail of granule cells along the migratory route which extends from the ventricular zone to the dentate gyrus. Additional ectopic granule cells are located in the molecular layer of the dentate gyrus, and there are also ectopic pyramidal cells in the stratum oriens or area CA3. In the cerebellar cortex, islands of ectopic granule cells are located subpially, in the former position of the external granule cell layer.

The other two mutants, lipopolysaccharide response defect (LPSd/LPSd) and balding (bal/bal), also have evidence of a disruption in the migration of granule cells in the hippocampal formation. The distribution of granule cells along the migratory pathway to the dentate gyrus is similar to that in the motheaten and beige mutations. However, there are no ectopic granule cells in the molecular layer nor does the migration of granule cells in the cerebellar cortex appear to be adversely affected by these mutations. The ectopic pyramidal cells in area CA3 found in motheaten and beige are present in LPSd but not in balding.

Thus, four independent and distinct single autosomal mutations, three (meY, bg, bal) of which arose spontaneously in the C57BL/6J inbred strain, produce phenotypically