



Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

Elsevier hereby grants permission to make all its COVID-19-related research that is available on the COVID-19 resource centre - including this research content - immediately available in PubMed Central and other publicly funded repositories, such as the WHO COVID database with rights for unrestricted research re-use and analyses in any form or by any means with acknowledgement of the original source. These permissions are granted for free by Elsevier for as long as the COVID-19 resource centre remains active.

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 AntiAChR 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