



Inflammation and Demyelination in the Central Nervous System: International Congress of Neuropathology, Vienna, September 5-10, 1982 (Acta Neuropathologica Supplementa)

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The present report, compares two murine models of virus induced chronic relapsing demyelination. MHV-induced demyelination in the BALB/c mouse results from the direct virus mediated cytolysis of oligodendrocytes. Extensive remyelination by oligodendrocytes is noted. Recurrent demyelination occurs in small areas. Infectious virus persists and 34 Fig. 2: Demyelination in SJL/J mice infected with TMEV. A) Multifocal areas of perivascular demyelination in the spinal cord (110 days post infection). Para phenylene diamine stain. X 250. B) Perivascular inflammatory infiltration within the white matter of the spinal cord (22 days post infection). Paraphenylene diamine stain. X600. C) Localization of TMEV associated antigen in the cytoplasm of oligodendrocytes (45 days post infection). Vibratome section stained with the peroxidase-anti peroxidase technique. X 400. D) Immunoperoxidase staining of viral antigen within inner and outer loops of an oligodendrocyte (45 days post infection) X 60,000. E) Longitudinal section showing viral antigen within Schmidt-Lanterman incisures (80 days post infection). X 49,000. viral antigens are localized within oligodendrocytes and their processes. TMEV-induced demyelination in SJL/J mice is associated with perivascular inflammatory infiltrates and is diminished by immunosuppressive measures. Remyelination by oligodendrocytes is delayed and incomplete. Chronic demyelination is widespread and associated with perivascular inflammatory infiltrates. The virus persists and viral antigen is localized within oligodendrocytes.



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