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FUNCTIONAL IMPLICATIONS OF CLASS I MHC MODULATION IN NEURAL TISSUE
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Although most normal neural cells lack MHC products, greater expression can be seen in pathological or experimental situations (reviewed in Lampson, Trends in NeuroSci, May, 1987). Whether these molecules serve an immunological role, or a non-immunological role in cell differentiation or homeostasis is not known. We have focused on class I molecules. We confirm that class I proteins with the appropriate mRNA, structure (1 and 2-D gels), and polymorphic specificity can be produced by human neural cell lines. Yet, in vivo, class I expression is not detected on neurons or glial cells in the adult, in any developing neural tissue in the mouse embryo, nor in the regenerating olfactory epithelium. Nor is class I detected on neural cells normally exposed to blood-borne elements (barrier-free areas of normal brain, tumor metastases) or the external environment (olfactory nerve endings), or following trauma (stab wound). Thus, the accumulated evidence argues against a role for class I molecules in normal growth, differentiation, maintenance or repair of neural cells, and against class I modulation as a non-specific response to injury. These studies provide a background for interpreting the class I modulation that is seen in specific clinical situations.

EXPRESSION OF VOLTAGE-GATED CALCIUM CHANNELS IN TUMOUR CELL LINES OF NEUROECTODERMAL OR OTHER ORIGIN
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The Lambert-Eaton Myasthenic Syndrome (LEMS) associates in 50% of cases with carcinoma, almost exclusively small cell lung cancer (SCLC), a tumour believed to be of neuroectodermal origin. The defect in neurotransmitter release at the motor nerve terminal appears to be due to interference with the function of voltage-gated Ca$^{2+}$ channels (VGCC) caused by binding of
IgG. We have shown that cells of a human SCLC line express VGCC, and that voltage-gated (K$^+$ stimulated) Ca$^{2+}$ flux is inhibited by LEMS IgG (Roberts et al, Nature 1985). Here we have investigated the expression of VGCC, and their inhibition by LEMS IgG, in 8 SCLC lines, 3 non-SCLC human lung cancer lines, and 7 other non-lung cancer cell lines (human and rodent), 3 of neuroectodermal origin.

$^{45}$Ca$^{2+}$ flux was measured at two K$^+$ concentrations (2.9 and 96mM) as previously described. Line cells were tested for labelling with the monoclonal antibody UJ13A (human neuroectodermal-specific) and for Bombesin-like activity. All SCLC lines showed K$^+$ stimulated Ca$^{2+}$ flux, which was inhibited by LEMS IgG in the four lines tested; seven were >80% UJ13A$. Ca^{2+}$ flux could not be demonstrated in the three non-SCLC lines, which were $\leq$10% UJ13A$. K$^+$ stimulated Ca$^{2+}$ flux was found in three rodent cell lines of neuroectodermal origin, but the profile of inhibition by LEMS IgGs differed from that in human SCLC lines. VGCC may be a functional marker for SCLC and for certain other neurosecretory/neuroectodermal cell types.

Tumor necrosis factor induces MHC class I antigen expression on mouse astrocytes.

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The brain is immunologically privileged because of the blood-brain-barrier, the lack of lymphatic drainage and the lack of expression of major histocompatibility complex (MHC) antigens on neural cells. To understand the pathogenesis of immunne mediated MHC-restricted reactions in the brain it is necessary to identify factors that induce MHC antigen expression on neural cells. $\alpha/\beta$ as well as $\alpha$-interferons (IFN) and the coronavirus-induced factor have been the only identified factors that induce MHC expression on neural cells. Tumor necrosis factor (TNF) has been reported to increase MHC class I antigen expression on endothelial cells and dermal fibroblasts (Collins 1985). Thus we examined the effect of TNF on neural cells. Glial cell cultures prepared from newborn mouse brain were incubated with graded amounts of mouse TNF and tested for expression of MHC class I and class II surface antigens by double immunofluorescence using anti-GFAP antibodies as astrocyte marker, and anti-GalC as oligodendrocyte marker. TNF induced expression of MHC class I but not la antigens on mouse astrocytes, but not on oligodendrocytes, in vitro. Blocking experiments with anti-TNF and anti-IFN revealed that the reaction was TNF specific. Thus TNF may play a role in the immunopathogenesis of neurologic diseases that involve MHC