What Function Is Served By MHC Upregulation in Neural Cells?

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The possibility of immune rejection, and the role of the major histocompatibility complex (MHC) proteins, in neural transplantation is increasingly appreciated. Although MHC proteins are not detected in normal neural cells *in situ*, greater expression has been reported following transplantation, trauma, or growth in culture. The function served by this upregulation is not known; a non-immunological role in development or tissue repair has been proposed. We asked whether similar upregulation occurs during normal development or regeneration, or after exposure to immunoactive cytokines.

Immunostaining was used to reveal class I and II MHC antigens in neural tissues *in situ*, as

listed in Table I. Among these tissues, strong MHC expression by microglia, endothelial cells, ependymal cells, connective tissue cells, and inflammatory cells was seen. Yet, MHC expression was not detected in neurons, astrocytes, or oligodendrocytes in any case. Nor were growth or cell contacts of neuroblastoma cell lines dependent upon MHC expression /1/.

Taken together, our findings do not reveal a non-immunological role for MHC upregulation in neural development, regeneration or homeostasis. Thus, it may be possible to prevent the MHC upregulation that has been reported in other contexts, without compromising normal neural function.

TABLE I
CNS TISSUES IN WHICH NEITHER MHC CLASS I NOR CLASS II WAS
DETECTED IN NEURONS OR GLIA

KIND OF TISSUE	SPECIFIC TISSUES EXAMINED*
Normal adult CNS.	Human brain; spinal cord. Rat brain. Mouse brain.
Barrier-free CNS.	Mouse, area postrema; stab wound. Rat stab wound.
IFN-g-treated CNS.	Mouse brain. Rat brain.
Regenerating/developing.	Mouse, olfactory epithelium; developing embryo. Rat, developing embryo.
Pathological.	Human brain, demyelinating disease. Human spinal cord, ALS. Human brain, tumor-associated. Rat brain, tumor-associated.

*For original reports, see refs. 2-8, and Grabowska and Lampson, this volume (11.P4).

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