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Titel des Beitrags:        Differential loss of KIR4.1
                          immunoreactivity in multiple sclerosis
                          lesions.
Abstract:                  Serum antibodies against the glial
                          potassium channel KIR4.1 are found
                          in a subpopulation of multiple sclerosis
                          (MS) patients. Little is known about
                          the expression of KIR4.1 in human
                          normal brain tissue and in MS
                          lesions. We analyzed the expression
                          pattern of KIR4.1 in normal brain
                          tissue and MS lesions of the
                          subcortical white matter by
                          immunohistochemistry. Markers of
                          related glial proteins, myelin, and
                          inflammatory cells were analyzed in
                          parallel. KIR4.1 is expressed in
                          oligodendrocytes and astrocytes in the
                          adult human brain. In
                          oligodendrocytes, KIR4.1 appears as
                          a homotetramer channel, in astrocytes
                          as homo- and heterotetramer
                          channels together with KIR5.1. In
                          acute MS lesions, KIR4.1
                          immunoreactivity (IR) was differentially
                          lost on periplaque oligodendrocytes
                          and perivascular astrocytes. In part of
                          acute lesions, complement activation,
                          apoptotic KIR4.1(+) glial cells, and
                          phagocytes containing KIR4.1(+) fragments
                          accompanied loss of glial
                          KIR4.1 IR. Periplaque reactive
                          astrocytes showed enhanced IR for
                          both KIR4.1 and KIR5.1. In chronic
                          active MS lesions, apart from a
                          general loss of oligodendrocytes in the
                          demyelinated area, we observed a
                          decrease of astroglial KIR4.1 but not
glial fibrillary acidic protein IR. In chronic inactive and remyelinating MS lesions, KIR4.1 IR was restored on astrocytes and found in a subset of presumably new myelinating oligodendrocytes. The expression profile of KIR4.1 in glial cells and stage-dependent alterations of KIR4.1 IR in MS lesions are compatible with an immune response against KIR4.1 at least in a subset of MS patients.