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Cell death of ED1$^+$ cells in the central nervous system of Dark Agouti rats at the peak of experimental autoimmune encephalitis

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Infiltration of macrophages into the central nervous system (CNS), as well as activation of microglia is a hallmark of multiple sclerosis and its animal model - experimental autoimmune encephalomyelitis (EAE). Cell death in EAE has been demonstrated as an essential mechanism in the local regulation of the inflammatory reaction, but also as one of the major factors contributing to the destruction of the CNS tissue. Here, cell death of ED1$^+$ cells (macrophages/microglia) in the spinal cord of EAE rats was investigated. Cell death in general was assessed using the TUNEL assay, while cleaved caspase-3 immunostaining was employed as the marker of "classical" apoptosis. Dark Agouti (DA) rats were immunized with spinal cord homogenate emulsified in complete Freund's adjuvant. Infiltrates of immune cells were detected both in white matter (WM) and grey matter (GM) of spinal cords in DA rats at the peak of EAE. ED1$^+$, TUNEL$^+$ and caspase-3$^+$ cells were detected within, but also outside the infiltrates. While there were no differences in the proportion of TUNEL$^+$ ED1$^+$ cells between infiltrates and non-infiltrated areas in WM, there were more ED1$^+$TUNEL$^+$ cells in GM in infiltrates than in non–infiltrated areas. A similar distribution was observed for ED1$^+$caspase-3$^+$ cells. The observed discrepancy in distribution of dead ED1$^+$ cells in infiltrates and non-infiltrated areas in GM and WM of spinal cord indicated that differential spatial regulation of macrophage/microglia cell death occurred in DA rats. These findings contribute to the understanding of pathogenesis of EAE in DA rats. It also opens new perspectives for a research aiming at more efficient treatment of multiple sclerosis.