P 119 EXPRESSION OF CU/ZN SUPEROXIDE DISMUTASE IN IMMATURE RAT BRAIN AFTER EXCITOTOXIC DAMAGE

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Cu/Zn superoxide dismutase (SOD) is a key antioxidant enzyme involved in superoxide detoxification in normal cellular metabolism and after cell injury. In adult animals, Cu/Zn SOD is highly expressed in astrocytes and neurones, being undetectable in microglial cells. When overexpressed, Cu/Zn SOD is neuroprotective in a wide number of experimental injury models including excitotoxicity and ischemia. However, the overexpression of Cu/Zn SOD in postnatal animals has been reported to exacerbate hypoxic-ischemic injury, suggesting a differential redox balance in the adult and the immature brain. In this study, we analysed the expression of Cu/Zn SOD at different postnatal times (from P9 to P16) by immunohistochemistry and western blot. Moreover, we have analysed the Cu/Zn SOD temporal expression pattern after an intracortical injection of NMDA to postnatal animals (P9). Our results show that, in contrast to adult animals, in the postnatal brain Cu/Zn SOD was expressed in astrocytes but also at high levels in different neurones as those found in cortical layers 3 and 5, hippocampus, septum and thalamus. No expression was detected in microglial cells. The total enzyme level increased slightly with development from P9 to P16. Following the excitotoxic damage, Cu/Zn SOD was upregulated in neurones and reactive astrocytes in the lesion border at 3 days. The further sustained increase corresponded with an extensive induction of Cu/Zn SOD in the glial scar at 5 and 7 days. This paralleled the total enzyme level that increased by 3 days and remained upregulated at 7 days post-lesion, the last survival time analysed. In conclusion, the Cu/Zn SOD expression pattern observed after an excitotoxic lesion to the postnatal brain does not differ from the reported in the adult brain, and thus can not account for the reported differences in the lesion outcome.

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