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We have investigated the role of inflammation in toxicant-induced glial activation using a well-characterized model of hippocampal neuronal damage (trimethyl tin-TMT) in the rat. Despite the potential role of inflammation in glial reactions associated with neurological diseases, such as Alzheimer's disease, we find very limited involvement of pro-inflammatory cytokines following neuronal injury of the hippocampus due to exposure to TMT. We evaluated the mRNA levels of a variety of cytokines/chemokines and trophic factors at 2, 5, 8 hours and 1,2, 3, 5, 7, and 21 days post TMT (a single injection 8mg/kg i.p.). TNF-alpha and IL-1beta, the most well-known components of the pro-inflammatory response, were not increased hours to weeks post exposure. TGF-beta and CNTF mRNA were elevated but not in a temporal pattern consistent with inflammation. In contrast, the chemokine, MCP-1 was increased as early as 3 days and peaked by day 5. MCP-1 protein also was increased supporting a functionally important change in the levels of this gene product. To determine if this chemokine or other inflammation-related signals were necessary for glial activation we repeated the TMT-exposure while giving 200μg/ml of the anti-inflammatory steroid corticosterone (CORT) in the drinking water 5 days prior to TMT administration. We hypothesized that if MCP-1 was an important component of this cellular response, the MCP-1 increases we had observed previously would occur in spite of CORT treatment. Our results show that TMT-induced increases in MCP-1 were blocked by CORT treatment without affecting the increase in the astroglial activation marker GFAP (mRNA and protein). These results, based on quantitative PCR using Taqman technology, extend and corroborate our earlier findings by RT-PCR, Atlas Array, and RPA and show that increases in MCP-1 and other inflammatory mediators that are inhibited by CORT, are not necessary for glial activation. Inflammatory mediators may play a role in neuronal injury, but not in damage-induced glial activation.