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Monocyte chemoattractant protein (MCP)-1 has recently been reported to be up-regulated in many models of neuronal damage including infection, axotomy, alheimer's (mouse model), excitotoxicity, and traumatic brain injury. We investigated this protein and its gene expression 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 for it following neuronal injury with this model. However we did find that mRNA and protein levels of MCP-1 were elevated by TMT treatment. We looked at MCP-1 over a timecourse of 2, 5, 8 hours and 1, 2, 3, 5, 7, and 21 days post TMT (a single injection 8mg/kg i.p.) by RT-PCR and western blot/ELISA, respectively. To follow up this experiment we repeated it using glucocorticoid treatment that is known to negatively regulate MCP-1, and also used low dose (0.5mg/kg) peripheral LPS injection (in a third experiment) as a model for peripheral inflammation. Here we report that MCP-1 is not a reliable biomarker of neural damage. Glucocorticoids suppressed MCP-1 expression in the hippocampus but did not suppress TMT-induced neuronal damage and astroglioisis. Further, peripheral LPS resulted in increases in MCP-1 at 2, 5, 8, and 24 hours that peaked at 8 hours in the hippocampus with no accompanying gliosis in this brain region. Thus MCP-1 increases may be blocked by glucocorticoids in the presence of neuronal damage or be upregulated in the brain by peripheral inflammatory processes without accompanying brain injury.