Little, A.R. and O'Callaghan, J.P.: Corticosterone suppresses the early increase in MCP-1 associated with chemically-induced brain damage but does not suprees accompanying gliosis. Proceeding of the Neuroinflammation in Alzheimers Disease Satellite Meeting, World Alzheimer's Disease Conference, Washington, D.C. 2000.

We have investigated the role of inflammation in glial activation using a well characterized model of hippocampal neuronal damage (trimethyl tin-TMT) in the rat. A very limited involvement for cytokines was found with a large and early increase limited to the chemokine monocyte chemoattractant protein (MCP)-1 mRNA based on semi-quantitative RT-PCR. The time-course was 2, 5, 8 hours and 1, 3, 5, 7, and 21 days post TMT (a single injection 8mg/kg i.p.). MCP-1 was increased as early as 1 day and peaked by day 5. 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. Cytokines are implicated in glial activation. We hypothesized that if MCP-1 was an important component of this cellular response, the increases we had observed previously in MCP-1 would occur in spite of cort treatment. Our results show that TMT-induced increases in MCP-1 were blocked by cort treatment however glial activation occurred normally, based on increases in GFAP mRNA and protein. These results, based on quantitative PCR using Taqman technology, extend and corroborate our earlier findings by RT-PCR to show that increases in MCP-1 (or other inflammatory mediators) are not necessary for glial activation. Chemokines may play a role in neuronal injury, but not in damage-induced gliosis.