After exposure, soman, an organophosphorus nerve agent, irreversibly inhibits acetylcholinesterase which causes excess acetylcholine concentration in the central and peripheral nervous systems. Soman exposure may induce a cholineretic crisis and the development of status epilepticus (SE), intense grand mal seizures, respiratory paralysis and possibly death (McDonough & Shih, 1997). After exposure, soman encourages extensive neuron loss in the piriform cortex, hippocampus, amygdala, and thalamus (Baille et al., 2005; McLeod, Singer, & Harrington, 1984). In severe brain injuries, respiratory paralysis and possibly death (McDonough & Shih, 1997).

The purpose of this study was to investigate the extent and maturation of the neuroinflammatory response in rat amygdala and cerebellum by examining inflammatory protein increases following soman exposure up to 24 hours after SE onset. It is predicted that the inflammatory protein concentrations will be higher in the amygdala than in the cerebellum due to the amygdala’s particular vulnerability to initial injury, and brain areas handle the damage differently. In order to show significant inflammatory protein expression. This shows that neuroinflammation after soman exposure in rats.

The amygdala showed more inflammatory protein expression compared to the cerebellum, as predicted, yet the cerebellum still showed significant inflammatory protein expression. This shows that neuroinflammation can affect brain regions not obviously damaged by initial injury, and brain areas handle the damage differently. In order to gain further comprehension of the neuroinflammatory process, the cell types that emit these proteins at the studied time points can be further investigated using immunohistochemistry. Another way to further this research is to test the effectiveness of thalidomide in reducing neuroinflammation after soman exposure in rats.

**Materials and Methods**

Adult male Sprague-Dawley rats were treated with axosmine chloride 30 minutes prior to soman administration and with atropine methyl nitrate 1 minute after soman administration. Control animals received axosmine chloride, atropine methyl nitrate and saline, while naïve animals received no injections. Ten, in hours, was measured after seizure onset.

Amygdala and cerebellum brain samples were taken from experimental and control rats at 0.5, 1, 3, 6, 12, or 24 hours after onset of convulsions. This brain tissue was then cryopulverized and experimental and control rats at 0.5, 1, 3, 6, 12, or 24 hours after onset.

**Results**

Of the eleven inflammatory cytokines studied, concentrations of eight significantly increased: IL-1α, IL-1β, IL-6, GRO-KC, MCP-1, MIP-1α, MIP-2, and VEGF as shown in Graphs 1, 2, and 3. Data were analyzed using a one-way ANOVA with a post-hoc Dunnett’s analysis comparing to vehicle control. A significant change was observed for MIP-1α in the cerebellum during the time course, and no significant changes were observed for IL-18 and TNF-α in the amygdala (these data not shown). Naïve and controls were not significantly different from each other in any one time point or region and were pooled.

**Conclusion**

The Ingenuity® Pathway Analysis software suggested one drug, thalidomide, that inhibits several inflammatory pathways.

**References**


