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Regional profile and time course of neuropathology following kainic acid intoxication in FVB mice

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We have evaluated the regional pattern of neuropathology and the extent of reactive gliosis following kainic acid intoxication of FVB mice. Male mice were injected intraperitoneally with saline or 20 mg/kg kainic acid, and scored for seizure severity according to the Racine scale. At 6h, 12h, 24h, 3d, and 7d following treatment, mice were perfused and processed for histology. Sequential sections were stained for Nissl, cupric-silver, and GFAP immunohistochemistry. To evaluate the time course and magnitude of reactive gliosis, additional animals were treated and allowed to survive 1, 3, 7, or 21 days, then processed for biochemical analysis of GFAP by ELISA. Within 15 minutes of treatment, kainic acid caused seizures in all mice which ranged from stage 2 to stage 5, with a modal activity of stage 3. By six hours post-treatment, kainic acid caused the appearance of argyrophilic neurons and processes in hippocampus and entorhinal cortex. By 12 hours, argyrophilia was observed in cortical layers III - VI, thalamus, subiculum, amygdala, hippocampus, and striatum. The magnitude and intensity of argyrophilia continued to increase through 24 hours. By three and seven days post-treatment, argyrophilia was attenuated; however, silver-stained neurons were still observed in hippocampus and septum. Nissl staining failed to reveal neuronal damage. GFAP immunohistochemistry revealed a robust astrogliosis in brain regions that correlated with cupric-silver staining. Quantification of GFAP levels by ELISA revealed increased protein levels by 1d following treatment, which continued to elevate, and reached a significant nine-fold increase by seven days post-treatment. By 21 days, levels were attenuated, but remained significantly elevated compared to saline controls. Our data reveal persistent damage signals in FVB mice following moderate seizure activity at the latest time points examined, compared to previously reported resolution of argyrophilia and gliosis in C57BL/6J mice.

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