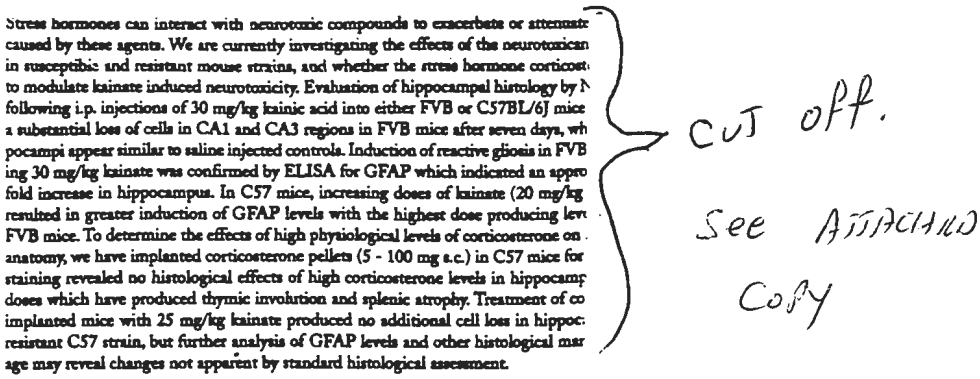
Benkovic, S. A., O'Callaghan, J. P. & Miller, D. B. Impact of the stress hormone corticosterone on hippocampal response to neurotoxic injury in susceptible and resistant mouse strains. Soc. Neurosci. Abst. 26: 2280, 2000.



Stress hormones can interact with neurotoxic compounds to exacerbate or attenuate caused by these agents. We are currently investigating the effects of the neurotoxican in susceptible and resistant mouse strains, and whether the stress hormone corticost to modulate kainate induced neurotoxicity. Evaluation of hippocampal histology by N following i.p. injections of 30 mg/kg kainic acid into either FVB or C57BL/6J mice a substantial loss of cells in CA1 and CA3 regions in FVB mice after seven days, wh pocampi appear similar to saline injected controls. Induction of reactive gliosis in FVB ing 30 mg/kg kainate was confirmed by ELISA for GFAP which indicated an appro fold increase in hippocampus. In C57 mice, increasing doses of kainate (20 mg/kg resulted in greater induction of GFAP levels with the highest dose producing lev FVB mice. To determine the effects of high physiological levels of corticosterone on anatomy, we have implanted corticosterone pellets (5 - 100 mg s.c.) in C57 mice for staining revealed no histological effects of high corticosterone levels in hippocamp doses which have produced thymic involution and splenic atrophy. Treatment of co implanted mice with 25 mg/kg kainate produced no additional cell loss in hippoc resistant C57 strain, but further analysis of GFAP levels and other histological mar age may reveal changes not apparent by standard histological assessment.

cut off.

See Attached Copy

Abstract View

IMPACT OF THE STRESS HORMONE CORTICOSTERONE ON HIPPOCAMPAL RESPONSE TO NEUROTOXIC INJURY IN SUSCEPTIBLE AND RESISTANT MOUSE STRAINS.

S.A. Benkovic*; J.P. O'Callaghan; D.B. Miller

Dept Toxicol & Molec Biol, Nat'l Inst Occupational Saftey & Hlth, Morgantown, WV, USA

Stress hormones can interact with neurotoxic compounds to exacerbate or attenuate the toxicity caused by these agents. We are currently investigating the effects of the neurotoxicant kainic acid in susceptible and resistant mouse strains, and whether the stress hormone corticosterone is able to modulate kainate induced neurotoxicity. Evaluation of hippocampal histology by Nissl staining following i.p. injections of 30 mg/kg kainic acid into either FVB or C57BL/6J mice has revealed a substantial loss of cells in CA1 and CA3 regions in FVB mice after seven days, while C57 hippocampi appear similar to saline injected controls. Induction of reactive gliosis in FVB mice receiving 30 mg/kg kainate was confirmed by ELISA for GFAP which indicated an approximate four-fold increase in hippocampus. In C57 mice, increasing doses of kainate (20 mg/kg - 35 mg/kg) resulted in greater induction of GFAP levels with the highest dose producing levels similar to FVB mice. To determine the effects of high physiological levels of corticosterone on hippocampal anatomy, we have implanted corticosterone pellets (5 - 100 mg s.c.) in C57 mice for 7 days. Nissl staining revealed no histological effects of high corticosterone levels in hippocampus following doses which have produced thymic involution and splenic atrophy. Treatment of corticosterone-implanted mice with 25 mg/kg kainate produced no additional cell loss in hippocampus in the resistant C57 strain, but further analysis of GFAP levels and other histological markers of damage may reveal changes not apparent by standard histological assessment.