

## 2005 IBNS Abstracts:

**THE EFFECTS OF KETAMINE ON THE EXPRESSION OF NMDA NR2B RECEPTOR SUBUNITS.** Hoxha N.; Mickley G. A. Department of Biology and The Neuroscience Program. Baldwin-Wallace College, Berea, OH 44107 USA.

Ketamine, a potent noncompetitive NMDA receptor antagonist has shown to impair learning and memory in a conditioned taste aversion (CTA) paradigm in adults and neonatal rats. However, in fetal rats ketamine was reported to produce paradoxical effects: ketamine given on embryonic day 18 (E18) enhances memory, whereas, administration of the antagonist on E19 blocks memory of the CTA. Objective of the present study was to characterize the expression of NMDA NR2B receptor subunits in the hippocampus and gustatory neocortex (GNC) in E18 and E19 fetal rats since, over-expression of NR2B subunits has shown to correlate with enhanced learning and memory. Pregnant dams at either E18 or E19 were injected with ketamine (100mg/kg, i.p.) or saline, and after 2, 4, or 24 hours, the hippocampus collected was homogenized and examined for NR2B subunit expression by western blot analysis. Expression of NR2B subunits in GNC were assessed 24 hours after ketamine injection. Two hours after ketamine, levels of NR2B subunits in hippocampus of E18 and E19 fetal rats were unchanged compared to the saline controls. However, after 4 and 24 hours of ketamine treatment the E19 group showed reduced levels of NR2B subunits; but, in E18 group the levels of NR2B subunits were unchanged. Ketamine after 24 hours produced increased levels of NMDA NR2B subunits in GNC in E18 pups; however, no change was observed in E19 group. In conclusion, these age-dependent changes in how ketamine influences NMDA receptor populations should encourage future studies aimed at confirming the role of NR2B subunits in the production of "ketamine paradox". *Supported by: Edith Robinson Grant and Thomas Surrarrer Fund.*

**MEMORY DEFICITS IN NEONATAL AND JUVENILE RATS FOLLOWING CHRONIC STRESS** Hoxha, Z., Mickley, G.A. Department of Psychology and The Neuroscience Program, Baldwin-Wallace College, Berea OH 44017 USA

Exposure to chronic stressors produces negative physiological and psychological changes in organisms. Previous studies from this laboratory have measured memory deficits in children and adolescents exposed to traumatic events during the war in Kosovo. Results indicated that exposure to traumatic war experiences caused short term memory impairments when tested four years after the war (Hoxha, 2004). The current experiment attempts to further explore the relationship between chronic stress exposure in young animals and subsequent memory problems. Fifty-two male and female rats were divided in four groups that received the following treatments: (1) stressed as fetuses on E15-E18; (2) not stressed as fetuses on E15-E18 (fetal controls); (3) stressed as juveniles on P16-P19; (4) not stressed as juveniles on P16-P19 (juvenile controls). Stressed animals received two days of foot shock (0.05mV; three times per trial/ three trials a day) and two days of restraint stress (three trials a day for 20 min). Controls remained in their home cage during these periods. During P25-P27 all animals were tested in a water maze (five trials a day over 3 days) for spatial memory. All rats were sacrificed on P28 for histological analysis. Group differences in learning and memory were most prominent during the last 2 days of behavioral testing. On the second day of testing, rats stressed as fetuses took longer to find the hidden platform than did non-stressed controls. However, rats stressed as juveniles did not show this effect. On the final day of testing, both rats stressed as fetuses or juveniles performed more poorly than did their respective controls. Future analyses will consider the physiological substrates of these stress-induced behavioral deficits.