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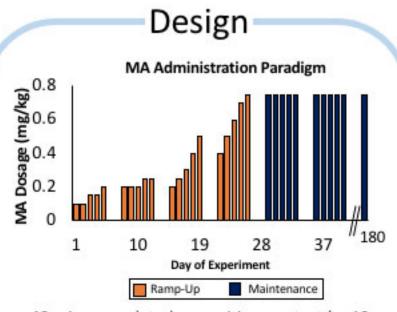
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Does Methamphetamine (MA) Cause Cognitive and Neurological Deficits? An Ecologically Valid Approach

Claire Stover

Methamphetamine (MA) Use Disorder is a growing public health concern in the United States. MA is widely believed to cause cognitive and neurological deficits. However, current animal constructs of MA abuse do not model human use patterns. MA users start at a low dose and ramp-up to ~0.75 mg/kg. Current models employ higher MA doses (~3-15 mg/kg), often lack a ramp-up period, and are rarely longitudinal. This proposal will investigate whether an ecologically valid model of MA abuse will demonstrate poor cognitive and neurological outcomes seen in current models.



- 48 mice completed a cognitive pre-test (n=12 per task) prior to MA administration
- MA was administered via injection to n=40 mice 2x/day, 5 days/week, for 180 days
- Days 1-30 = "ramp-up" period
- Days 31-180 = "maintenance" period
- Mice completed cognitive post-test on day 184 and were sacrificed for immediate early gene (IEG) analysis
- * = expected p < 0.05; ** = expected p < 0.01 for t-tests performed

Conclusions

- This study will clarify whether MA abuse causes cognitive and neurological deficits
- We predict that MA will cause minor yet statistically significant deficits in IEG expression in caudate nucleus and nucleus accumbens, delay-discounting, and perseverance errors in cognitive flexibility
- These findings will inform MA Use Disorder treatments, as some clinicians feel that patients must be too cognitively impaired to respond to cognitive-behavioral treatments



Neural Histology

Caudate Nucleus: memory, reward, motivation, and learning
Nucleus Accumbens: motivation, reward, operant conditioning
Immediate Early Gene: arc. Proxy for neuronal activity.
Measured via brain dissection and qPCR.

