Medial Caudate Putamen Lesions and Rat Serial Pattern Learning

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Introduction

In prior research in our lab, MK-801, an NMDA receptor antagonist, disrupted serial pattern learning in rats when the serial pattern was a sequence of 24 response elements arranged in eight 3-element chunks. The final element of the sequence violated the overall pattern structure. MK-801 rats learned within-chunk elements as fast as controls, but showed permanent inability to learn the violation response and, to a lesser degree, chunk boundary responses. Because NMDA receptors have been localized in the basal ganglia (among other structures) and because the basal ganglia have been implicated in sequential learning and memory, we examined the effects of lesions of the medial caudate putamen (MCPu) of the basal ganglia on rat sequential learning compared to those of MK-801. In the present study, 7 rats received medial caudate putamen excitotoxic lesions later confirmed by histological analysis. Rats were then trained on the same pattern as in previous studies.

Methods

Subjects: 7 naïve male Long Evans rats, 90 days old at the time of surgery, received bilateral medial caudate putamen (MCPu) lesions. The excitotxic lesions were produced with quinolinic acid injections to the MCPu.

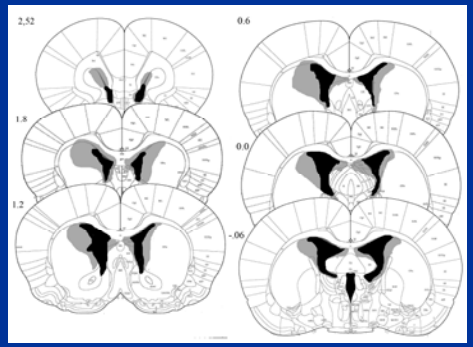
Apparatus

In a Plexiglas octagonal chamber equipped with a nose poke receptacle on each wall, rats learned to produce a highly structured serial pattern of responses for water reinforcement:

123-234-345-456-567-678-781-818

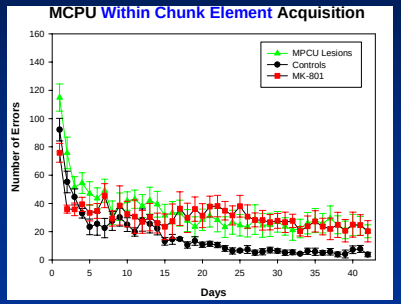
Integers indicate the clockwise position of correct responses on successive trials. Trials were separated by 1 s except where dashes indicate 3-s phrasing cues. The final element, 8, was the violation element.

Histological Analysis

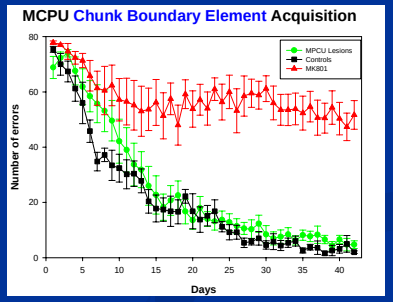


The largest and smallest lesions of MCPu. The gray shading represents the largest lesion and the black shading, the smallest lesion (from Paxinos and Watson 2005).

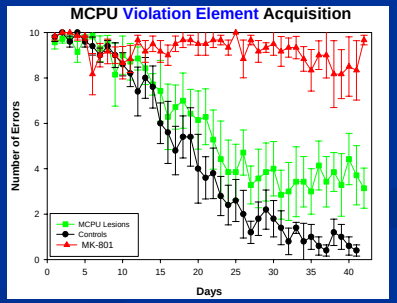
Results



MCPu lesions caused a deficit for learning within-chunk responses similar to MK-801 effects. Approximately 70-100% of errors were perseverations.



MCPu lesions had no effect on learning chunk boundary elements. Errors were approximately 50% perseverations and 50% overextensions of the rule.



MCPu lesions caused a significant deficit in learning the violation element though not as severe as the MK-801 treatment effect. Errors were approximately 54% perseverations and 42% overextensions.

Discussion

Medial caudate putamen lesions caused learning deficits for within-chunk elements and the violation element, but not for chunk boundary elements. The observed deficits were generally less severe than those caused by MK-801. Prior research demonstrated that dorsal hippocampal lesions as well as medial frontal lesions had little effect on learning chunk boundary elements and no effect on within-chunk or violation elements.

These results support the claim that serial pattern learning is subserved by multiple dissociable brain and cognitive systems.

Conclusions

- MCPu lesions caused differential, dissociable effects on serial pattern learning.
- Like MK-801, MCPu lesions produced a deficit in serial pattern learning. However, the effects caused by MCPu tend to be less severe than those produced by MK-801.
- MCPu lesions caused a deficit on within-chunk element learning as well as violation element learning, but had no effect on chunk boundary element learning.
- Responding to the different elements of serial patterns appears to be supported by different psychological processes that are subserved by different brain substrates.