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Erratum

2017-04-01

The Effects of Adrenergic Receptor Antagonists on Avian Memory

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ABSTRACT

In mammals, fear conditioning is influenced by both the adrenergic system as it contributes to consolidation and reconsolidation of memories and the cerebellum as it relates to the consolidation of fear based memories. The arcopallium, posterior pallial amygdala, and cerebellum are thought to be homologs to the mammalian amygdala and cerebellum respectively. The adrenergic system appears to have a conserved distribution, but species specializations for cued memory have been found. We have previously shown that several functions of the cerebellum are conserved between mammalian species and the zebra finch. Lesions of the cerebellum result in deficits in spatial learning, postural adjustments, and timing of learned vocalizations. In contrast, we have tested for a conserved role of the adrenergic system in spatial and cued fear conditioning memory and have found no evidence that different doses of adrenergic antagonists, either given at several time points or chronically during learning, affect learning or retention of memory in spatial & cued fear conditioning tasks as they do in rodents. The neural circuitry underlying fear conditioning is well known and, because the β -adrenergic receptor system and cerebellum are known to be involved in fear conditioning, we tested whether the β -adrenergic antagonist, propranolol, would interfere with retention of fear memories. We did not see any behavioral deficits in learning or retention under these conditions. Thus, it appears that the role of the adrenergic system in fear conditioning is not conserved across species.

INTRODUCTION & BACKGROUND

Short-term memories (STM) must be converted to long-term memories (LTM) through consolidation^{1, 2}. Reactivation of these memories to an active state allows them to be modified before becoming reconsolidated^{3,4}. The adrenergic (AR) system is involved in the consolidation and reconsolidation of limbic system-dependent memories⁵. In mammals, AR receptor antagonists, such as the non-selective β -AR antagonist, propranolol, impair spatial and emotional memory if administered after reactivation^{4,6,7,8,9}. In

chick models, the AR system is involved in classical conditioning of taste aversion and contextual learning⁶. In previous experiments, our lab has investigated the effects of propranolol and an α -AR antagonist, phentolamine, on zebra finch learning and consolidation during a spatial maze protocol. Memory was not impaired when given 20-mg/kg dose of phentolamine or 20 mg/kg and 40 mg/kg doses and administered 0 or 25 minutes before or after reactivation or administered chronically for propranolol. In the present study, we examined the zebra finch model to confirm a conserved role of AR system in

reconsolidation of memory after a fear-conditioning protocol.

MATERIAL & METHODS

Fear Chamber: We used an adapted fear conditioning chamber with a grid floor. A routed PVC sheet was fitted under the floor to ensure the bird's feet connected with a minimum of 2 different bars at a time. Electrode gel was applied to the feet to increase conductivity. Speakers emitted a pure tone stimulus of 800 Hz. **Pairing:** low-frequency tone (conditioned stimulus; CS) with 3.5mA foot shock (US). **Males (n=18)** received 5 trials/day for 7

days, inter-trial intervals varied among 4 durations (60, 80, 100 or 120s) to provide a control against prediction. Flight Response duration and latency to response following CS onset were recorded using an image analyzer (Ethovision). 24 hr post-training, birds were assigned to 4 treatments: propranolol or saline 5m before reactivation, propranolol or saline 5m after reactivation. A single extinction trial (CS only) was used as a reactivation trial that should impair memory. A recall trail (CS-US paired) was given 48hr post-injection and reactivation.

RESULTS

No differences existed between groups prior to treatment on either the training trials or the reactivation trail (not shown) and there were no differences in recall between subjects in the post-injection recall trail (**Figure 1**).

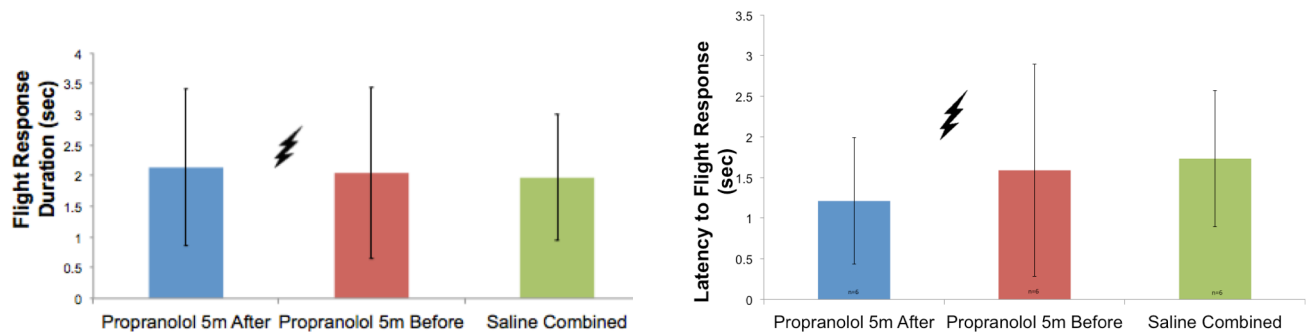


FIGURE 1. Post-Injection Recall Trial. No significant differences between the groups in Flight Response duration or latency to response. The birds that learned the fear-conditioning protocol should show longer flight duration and shorter latency to flight. There were no differences in learning shown between the groups.

IV. CONCLUSION

AR antagonists do not alter fear memory reconsolidation in zebra finches. The role of the AR system in reconsolidation of this memory type may not be conserved. The effective injection times and doses vary among tasks, species⁹, and systemic versus local administration complicating experimental identification of effects. To avoid repeated testing of animals at a large number of post learning injection times, we will next examine the location of immediate early gene activation that occurs during fear conditioning with and without an adrenergic antagonist. While we have yet to see behavioral differences caused by adrenergic antagonists due to timing precision for the administration of the treatments, we are hoping to see possible IEG expression differences in the brain of the zebra finch. The length of clearing time for antagonists will allow us to see decreases in IEG activation that antagonists caused over the 30 minutes between consolidation learning and sacrifice. These experiments should clearly show the

parts of the brain involved in fear conditioning.

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