The goal of the current study was to begin to investigate the role of dopamine signaling in the DMS and DLS in fear extinction and renewal.

**Methods**

- **Experiments 1 and 2:** A GABA	extsubscript{A}/GABA	extsubscript{B} agonist drug cocktail (0.03/0.3 nmol/μl Muscimol/Baclofen) was injected bilaterally into the DMS or DLS.
- **Experiment 3:** A D1 antagonist, SCH-233965 (1.0 μg/μl), was injected bilaterally into the DMS.

**Hypothesis**

- Inhibition of the DMS will increase the reliance on the habit learning strategy involving the DLS, thereby rendering fear extinction resistant to fear renewal.

**Goal**

To investigate the role of the DMS in fear extinction.

**Experiment 1**

**Goal**

To investigate the role of the DLS in fear extinction.

**Hypothesis**

Inhibition of the DLS will increase reliance on the goal-directed learning strategy involving the DMS, thereby enhancing fear extinction in a context-dependent manner.

**Experiment 2**

**Goal**

To investigate the role of D1 signaling in the DMS and DLS in fear extinction.

**Hypothesis**

Based on our prior work, blocking D1 signaling in the DMS will impair fear extinction memory.

**Experiment 3**

**Goal**

D1 receptor signaling in the DMS, but not DLS (not shown) contributes to fear extinction learning.

**Results**

Fear extinction supported by the DLS (DMS inactivation) is resistant to renewal

**Results**

Fear extinction supported by the DMS (DLS inactivation) is strengthened but remains context-dependent

**Results**

D1 receptor signaling in the DMS, but not DLS (not shown) contributes to fear extinction learning

**Conclusions**

- Fear extinction learning supported by DLS, habit strategies (DMS inactivation) is resistant to fear renewal, whereas fear extinction learning supported by DMS, goal-directed strategies (DLS inactivation) improves extinction retention, but the extinction memory remains context-dependent.
- D1 receptor signaling in the DMS contributes to the role of the DMS in supporting fear extinction, but mechanisms other than D1 receptor signaling contribute to the role of the DLS.
- Results suggest that substantia nigra activation enhances extinction memory through D1 signaling in the DMS and renders the extinction memory resistant to renewal through a D1-receptor independent mechanism in the DLS.