Cell Reports, Volume *26*

Supplemental Information

Plasticity at Thalamo-amygdala Synapses Regulates Cocaine-Cue Memory Formation and Extinction

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SUPPLEMENTAL INFORMATION

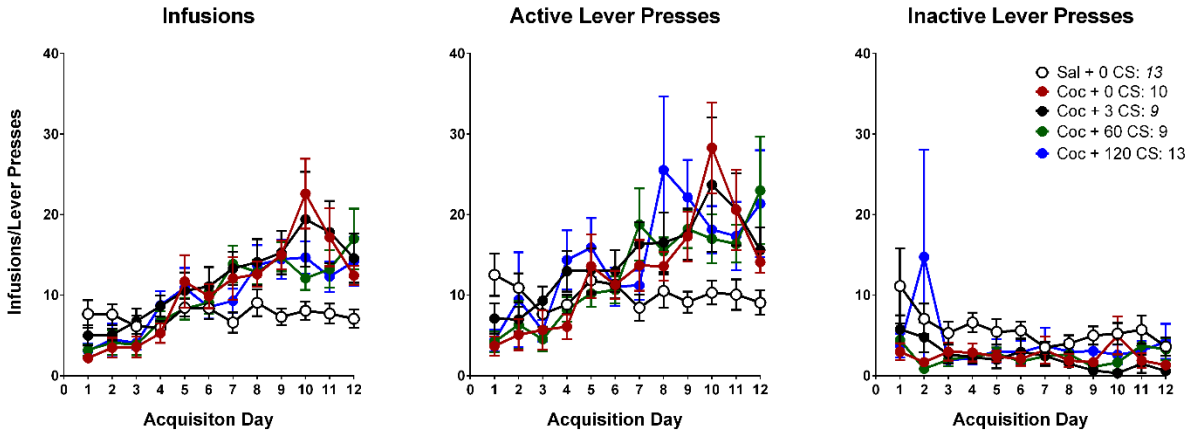

Figure S1. No Group Differences in Acquisition of Self-Administration. Related to Figure 2.

Acquisition data for rats from electrophysiological experiments. There were no differences in infusions earned (*Left*: $F_{(4,49)} = 1.92$, $P > .05$), active lever presses (*Middle*: $F_{(4,49)} = 1.46$, $P > .05$), or inactive lever presses (*Right:* $F_{(4,49)} = 2.19$, $P > .05$) between any Coc SA animals (all two-way ANOVA; n in italics, *number of rats*).

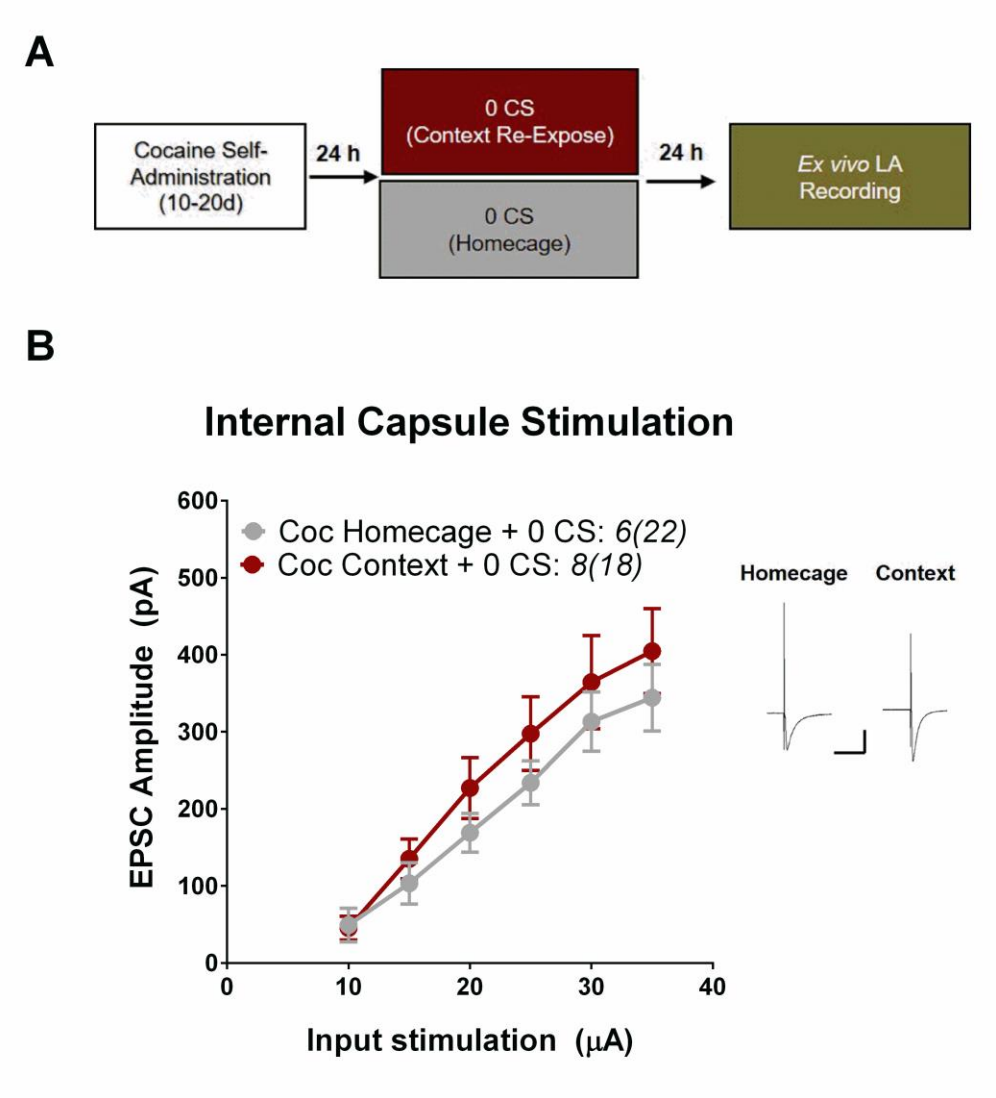

Figure S2. Potentiation of Thalamo-Amygdala Synapses following Drug-Cue Learning is not Context-Dependent. Related to Figure 2.

(A) Experimental timeline. Rats self-administered Coc for 10-20 d. 24 h following the last day of acquisition, one group of rats was returned to the operant chamber, but received 0 CS presentations, while a second group of rats remained undisturbed in their home cage. The following day, rats were euthanized and slices were prepared for electrophysiological recordings. T-LA EPSCs were elicited by stimulating fibers from the internal capsule.

(B) No difference in EPSC amplitude between the two groups. Average EPSC amplitude for the two groups at various stimulation intensities. Context re-exposed animals show slightly higher, yet nonsignificant EPSC amplitude than home cage controls (Two-way ANOVA, $F_{(1,12)} = 0.84$, $P > .05$; n in italics, *number of rats (number of neurons).* Scale bars: 50 ms, 200 pA.

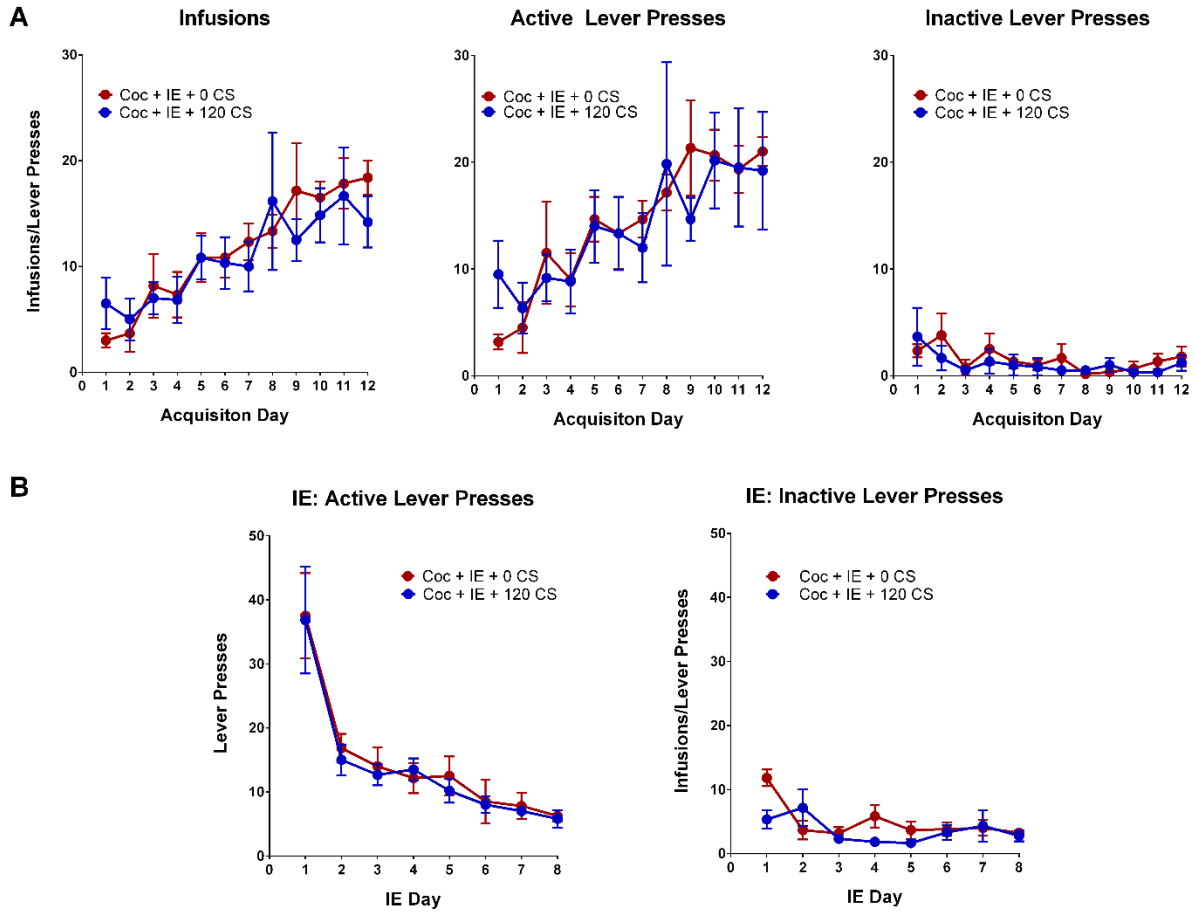

Figure S3. No Group Differences in Acquisition of Self-Administration or Instrumental Extinction. Related to Figure 2.

(A) Comparison of acquisition data for rats that underwent instrumental extinction (IE) prior to drug-cue re-exposure sessions. Following IE, rats received either no cue re-exposure (0 CS) or extended cue extinction (120 CS). During SA, there were no differences in infusions earned (*Left:* $F_{(1,11)} = 0.02$, $P > .05$), active lever presses (*Middle:* $F_{(1,11)} = 0.10$, $P > .05$), or inactive lever presses (*Right*: $F_{(1,11)} = 0.45$, $P > .05$) between the two groups (all two-way ANOVA; n = 6 rats/group).

(B) Comparison of IE data. There were no differences in active lever presses (Middle: $F_{(1,11)} = 0.07$, $P > .05$), or inactive lever presses (Right: $F_{(1,11)} = 1.99$, $P > .05$) between the two groups (all two-way ANOVA).

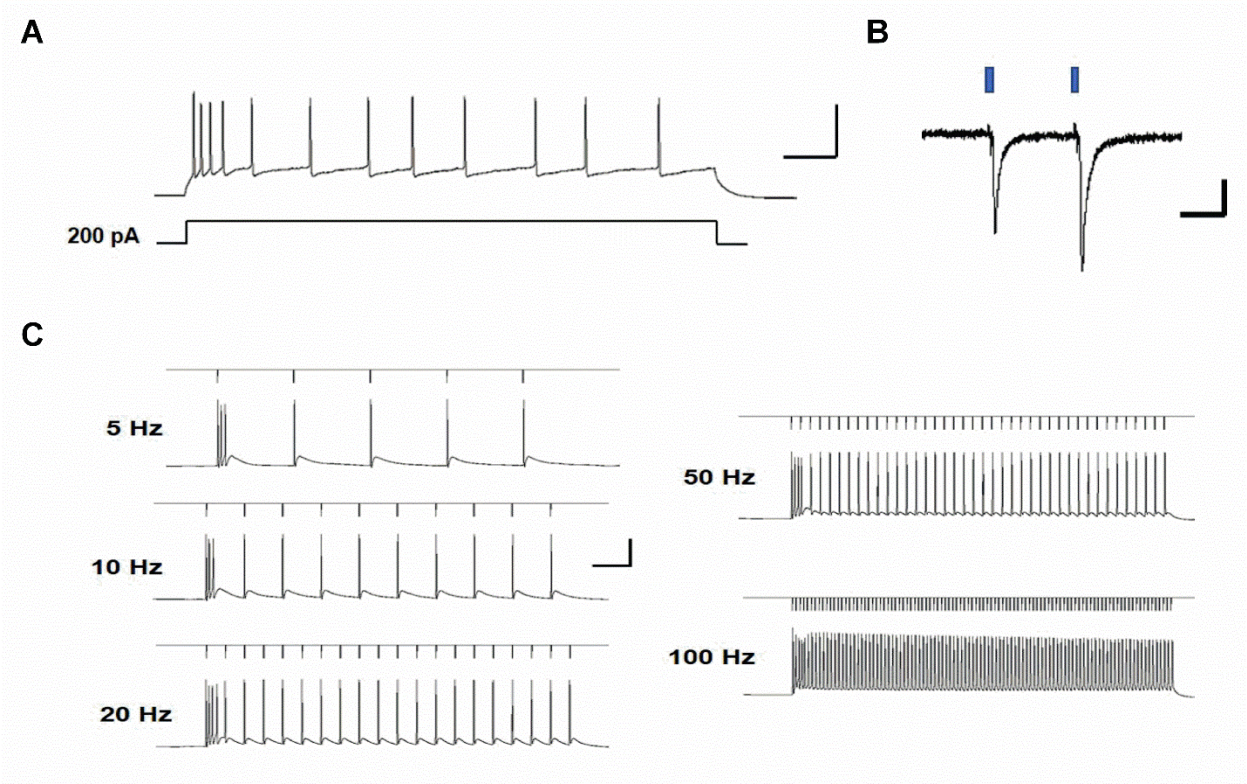

Figure S4. Sample Electrophysiological Properties of LA and MGN Neurons. Related to Figures 2 – 4.

(A) Sample current clamp recording from LA neuron. Injection of a prolonged depolarizing current (0.2 nA, 1 s) demonstrating action potential firing that shows spike frequency adaptation typical of principal neurons (*See Kim et al., 2007a*). Scale bars: 100 ms, 40 mV.

(B) Sample voltage clamp recording from LA neuron receiving projections from AAV-oChIEF-infected MGN neurons. EPSCs were elicited by two brief (1 ms) pulses of blue light (473-nm) separated by 50 ms. Scale bars: 25 ms, 50 pA.

(C) Sample current clamp recordings from AAV-oChIEF-infected MGN neurons. Action potentials were elicited by blue light stimulation (5-100 Hz), demonstrating the capacity for MGN-infected neurons to respond to both low and high frequency stimulation. Scale bars: 100 ms, 40 mV.

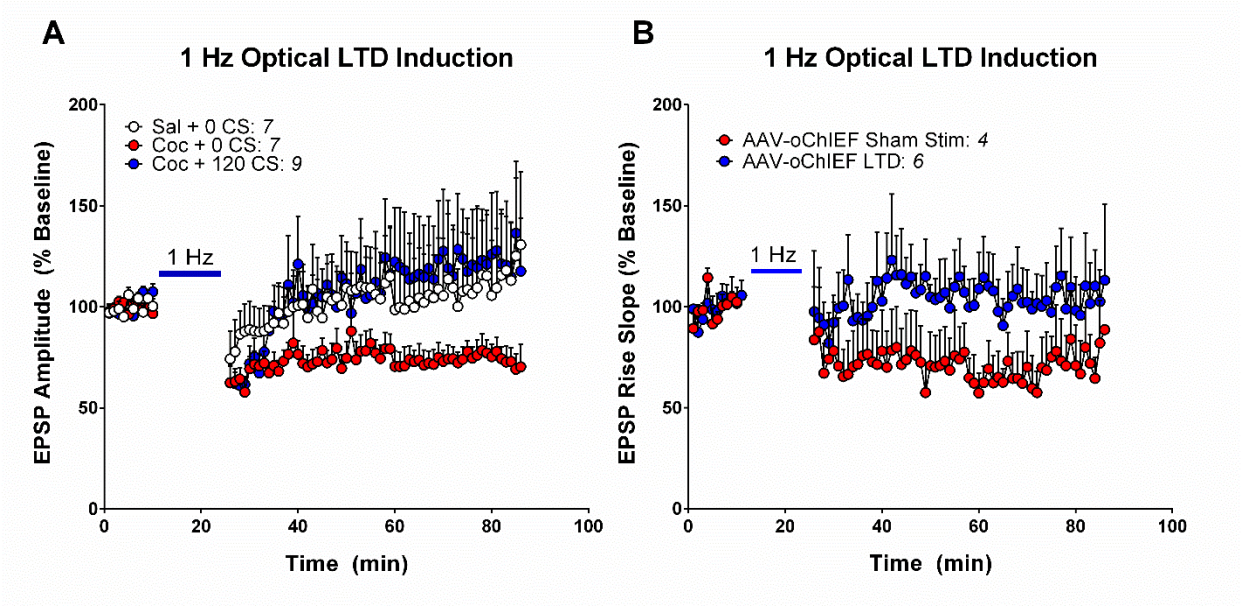

Figure S5. Further Characterization and Behavioral Effects of MGN-LA Optical LTD. Related to Figures 5 – 6.

(A) Scatter plots demonstrating effect of *ex vivo* optical LTD induction on EPSP peak amplitude at MGN-LA terminals. 15 min. of 1 Hz blue light stimulation induced a sustained reduction in EPSP amplitude only in Coc-trained non-CS re-exposed animals, with no effect on Sal-trained or Coc-trained 120-CS re-exposed animals; *n in italics, number of neurons.*

(B) *In vivo* optical LTD induction occludes *ex vivo* LTD. Rats received either 15 min. of 1 Hz blue light stimulation or SHAM stimulation. 24 h later, amygdala slices were prepared and the same stimulation protocol was used to measure the effect of *ex vivo* LTD. EPSP rise slope at MGN-LA terminals was reduced by *ex vivo* optical stimulation in neurons from animals that had received *in vivo* SHAM stimulation, but not in neurons from animals that had received *in vivo* optical LTD. *n in italics, number of neurons.*

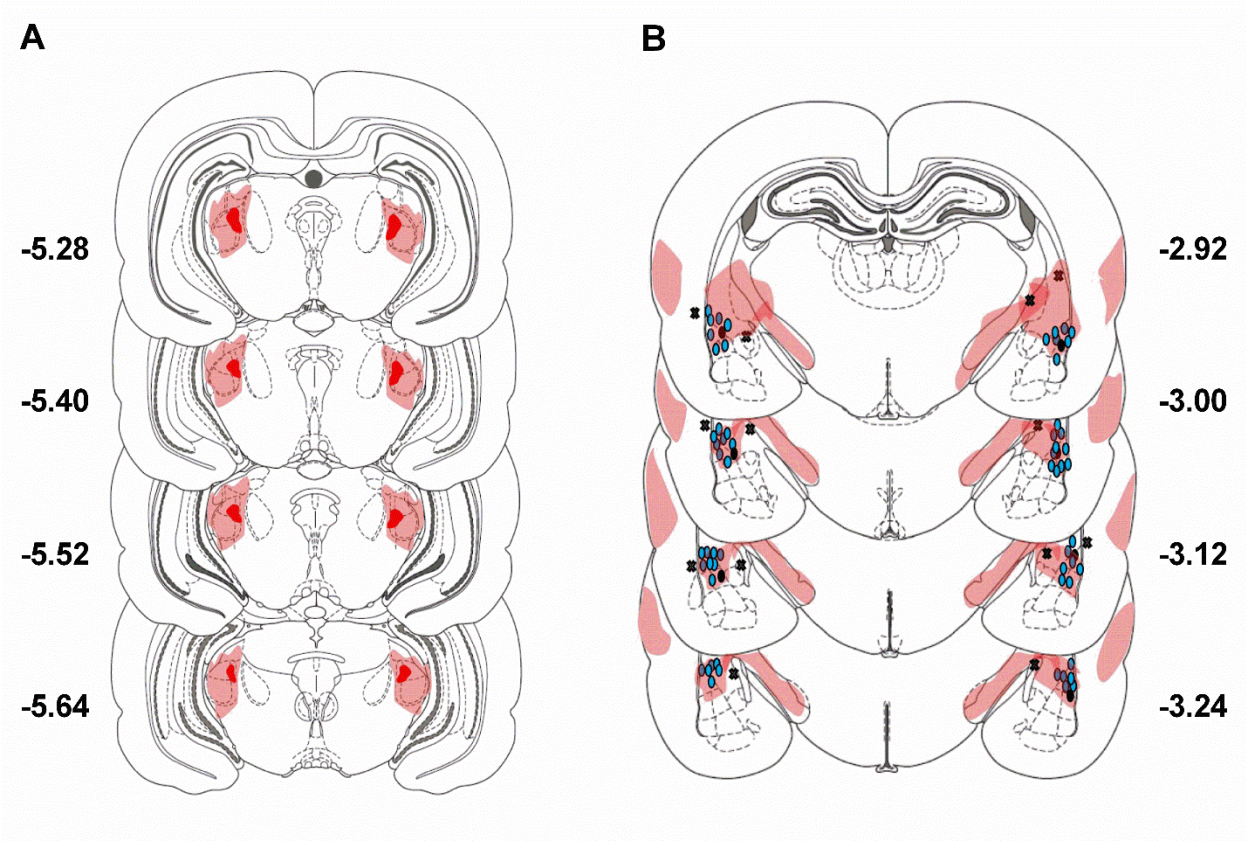

Figure S6. Histological Verification of AAV Injection and Optic Fiber Placements. Related to Figures 4 – 6.

(A) Schematic showing injection of AAV-oChIEF-tdTomato throughout the anterior-posterior extent of the MGN (For Figures 4 and 5). Dark red shading shows representation of smallest acceptable virus spread, and light pink shading shows representation of largest acceptable spread. Inclusion required dual hemisphere viral expression.

(B) Schematic showing spread of AAV-oChIEF-tdTomato (corresponding to Figures 4-6) and optic fiber placements (corresponding to Figure 6) throughout the anterior-posterior extent of the LA. Light pink shading shows representation of AAV-infected MGN-projecting neurons. There is robust expression through the internal capsule targeting the LA. Notably, there is also expression in auditory temporal cortex, which receives dense projections from the MGN. Blue circles correspond to successful optic fiber placement in both hemispheres. Black circles correspond to successful optic fiber placement in only one hemisphere. Black "X" corresponds to unsuccessful fiber placement. To be included in final analysis, rats required viral expression in the LA as well as successful placement of fibers in both hemispheres. Coordinates are in mm, posterior from bregma.