D1/5 receptor signaling at acquisition required for PV plasticity and long-term fear memory consolidation.

**a.** Analysis of vH D1/5 signaling interference during fear memory acquisition. Note low-PV instead of high-PV shift induction (left), unaffected intermediate-term memory (+6.5h) and absence of long-term fear memory (+24h). **b.** No rescue of PV plasticity or fear memory by D1 agonist delivery at +12h in mice treated with D1/5 antagonist just before acquisition. Note sustainment of low-PV plasticity by D1 agonist at +12h (left), and absence of fear memory (right). Average values from 4-6 mice and 60 PV neurons each; ANOVA, followed by Dunnet’s post-hoc; p < 0.001 (**).
Supplementary Figure 2

Specific suppression of long-term memory consolidation by D1/5 receptor antagonist at +12h.

a. Analysis of average center of mass movement (translocation) velocity in mice subjected to cFC, treated with D1/5 receptor antagonist at different times after acquisition (x-axis), and analyzed at +24h in training context. Note how mice with suppressed fear memory (e.g. +13h) do not move faster than untreated control mice (0h), arguing against loss of freezing due to hyper-locomotion. b. Mice that underwent cFC and were treated with D1/5 antagonist at +12h exhibit robust fear memory at +2d when reconditioned at +24h (orange), indicating that D1/5 antagonist at +12h specifically suppressed long-term consolidation of the fear memory induced 12h before (red). c. NE receptor antagonist delivered at +12h to vH does not interfere with long-term consolidation of fear memory. Average values from 4-6 mice.
Supplementary Figure 3

Specific suppression of individual long-term memories by D1/5 receptor antagonist at +12h.

a. Delivery of D1/5 receptor antagonist to vH at +12h suppresses fear memory consolidation regardless of whether acquisition occurred at 09:00, 15:00 or 21:00. b, c. Memory specific requirement for vH D1/5 receptor signaling at +12h for long-term memory consolidation. b: Specific suppression of TR1 or TR2 fear memory by D1/5 receptor antagonist at +12h after corresponding fear conditioning. c: Enhanced high-PV plasticity upon second cFC protocol, and reversal to high-PV levels comparable to those induced by one cFC protocol upon delivery of D1/5 receptor antagonist at +12h. Average values from 4-6 mice and 60 PV neurons each; Student’s t-test; p<0.001 (**).
Supplementary Figure 4

Pharmacogenetic induction of high-PV plasticity in naïve mice not sufficient to induce freezing.

Mice expressing pharmacogenetic activator virus in vH CA3 PV neurons explored context without foot shocks (noUS), where treated with pharmacogenetic ligand at +3h, and tested for freezing in context at +12h or +24. Average values from 4-6 mice and 60 PV neurons each.
Supplementary Figure 5

No detectable expression of cFos in vH CA3b PV neurons 90min after cFC.

Representative example of c-Fos/PV double-labeling experiment in vH CA3b 90min after cFC. Yellow arrows: PV+ neurons. Bar: 50 μm. Average values from 3 mice and 80 PV neurons each.
Analysis of dye spread and electrode paths.

a. Left: Mouse brain coronal section with location of vH (DG, CA3, CA1). Numbers: antero-posterior coordinates caudal to bregma. Right: Representative image of Bodipy dye targeted at vH CA3 and its spread from the target site 6h after injection. Bar, 300μm. b. 16 channel Neuronexus probes (LFP experiments) inserted in both vH with 16 contacts covering 0.8mm length at the electrode tip. Example of electrode track position revealed with Dye I (right). Bar, 1500μm. c. Representative images of Nissl stained vH section (50 μm) through the cannula track after dye injection (left). Bar: 200 μm. Infusion site (dotted line) and injector track (black line) (right). Bar 100 μm. d. Injection sites from 8 mice into CA3 region of vH.