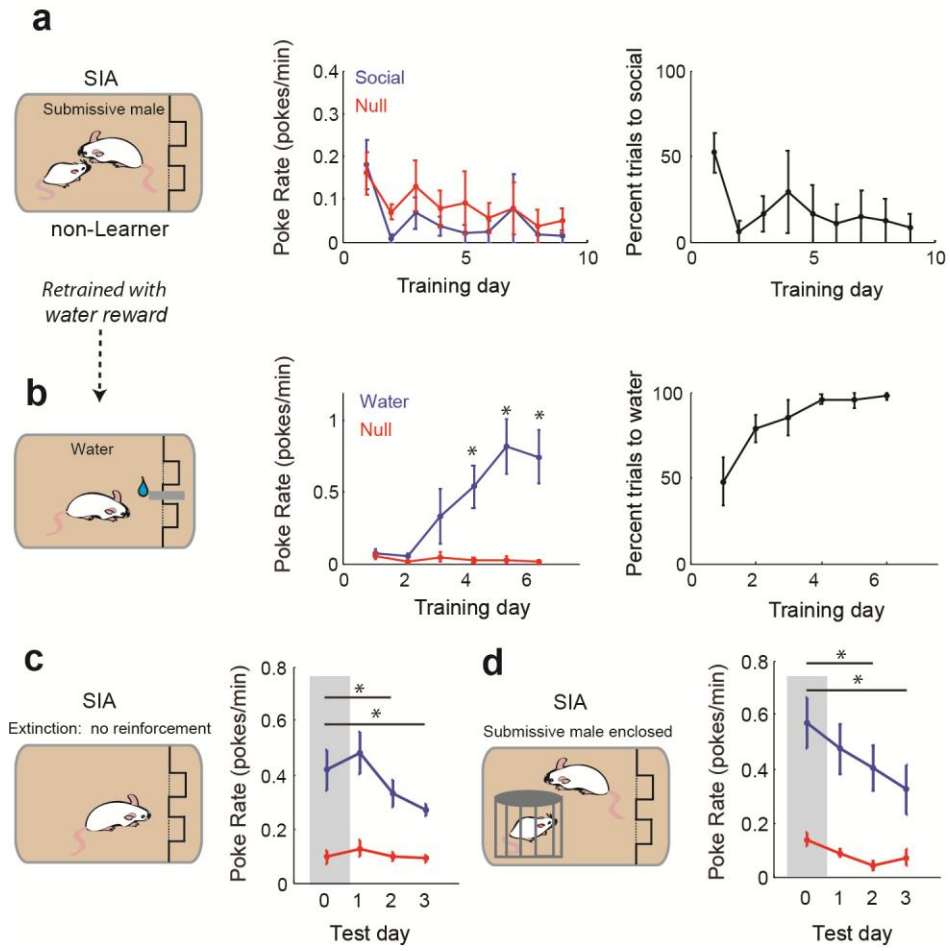
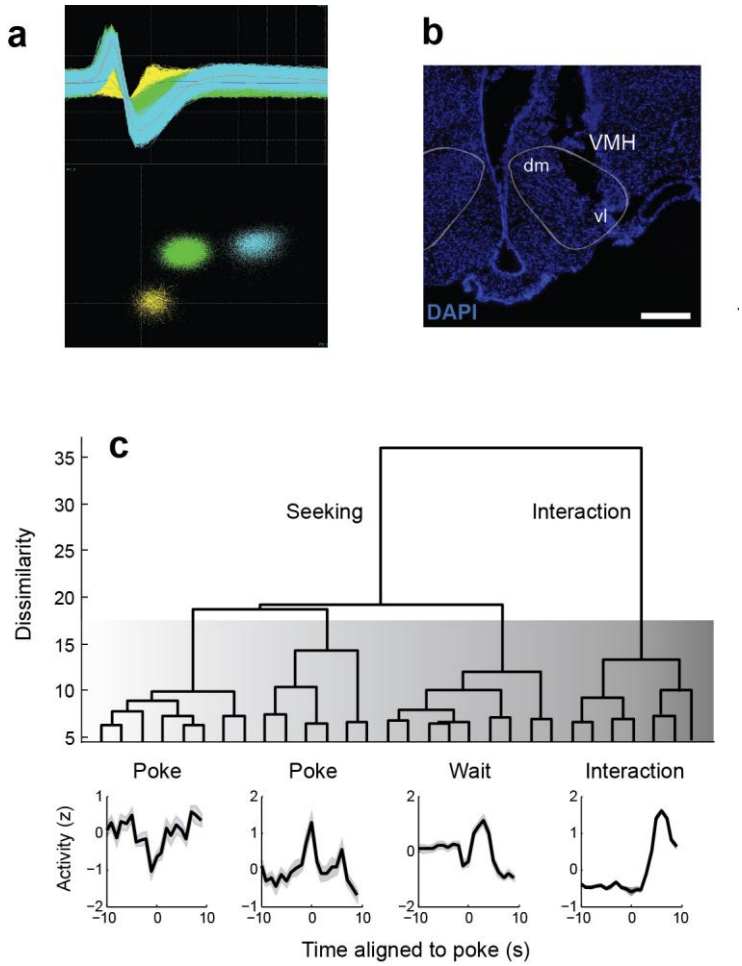


Supplementary Figure 1

Behavior controls for self-initiated aggression task.

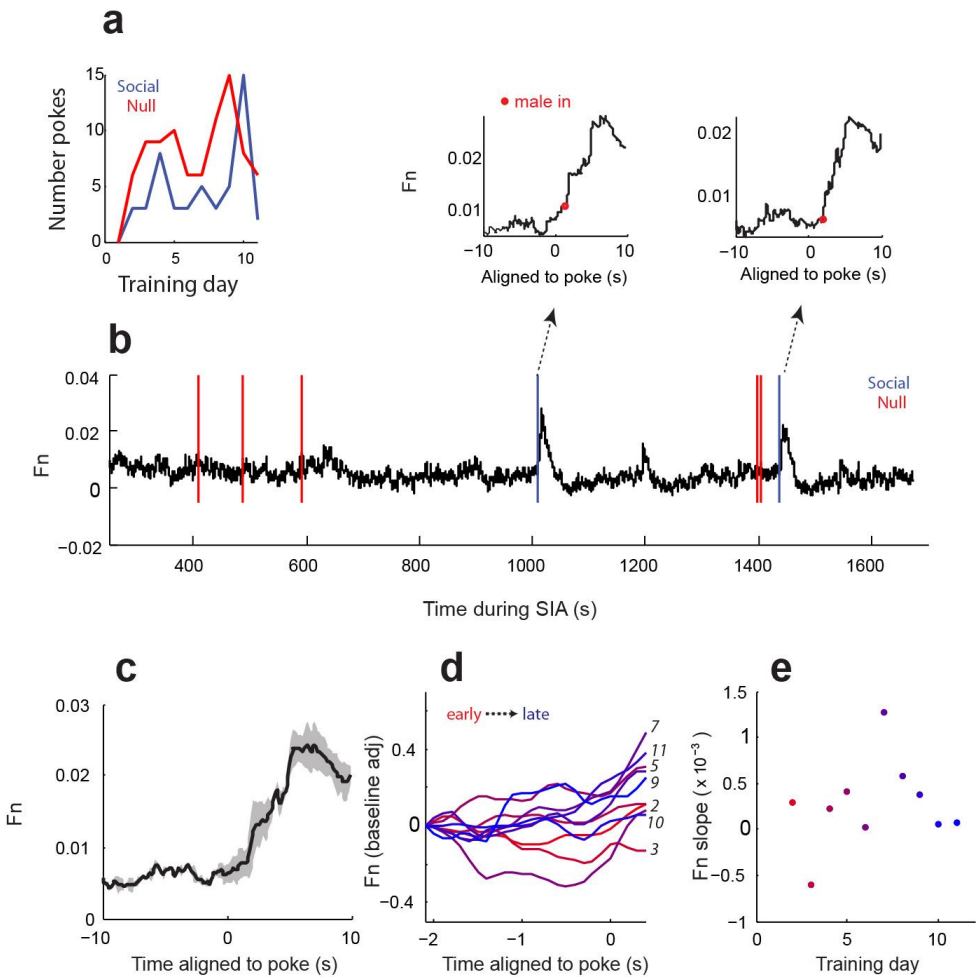
(**a**) Poke rates and percent trials to the social port for "non-Learner" males that did not prefer the social port to the null port. (**b**) These males were retrained on a similar task where they received water rather than a submissive male reinforcement. Poke rates and percent trials to water increased rapidly as a function of training day (Training day 4: t(3) = 3.497, *p = 0.040; Training day 5: t(3) = 3.882, *p = 0.030; Training day 5: t(3) = 3.852, *p = 0.031; N = 4 animals, paired t-test between water and null poke rates). (**c**) Poke rates of trained males that learned the task declined during extinction training compared to previous SIA control day (gray, Extinction day 2 compared with control day: t(9) = 2.981, *p = 0.015,Extinction day 3 compared with control day: t(9) = 2.988, *p = 0.015, t-test, N = 10). (**d**) Poke rates also declined if reinforced by a submissive male in an enclosure (Enclosure day 2 compared with control day: t(6) = 2.467, *p = 0.047,Extinction day 3 compared with control day: t(6) = 2.694, *p = 0.036, t-test, N = 7). All plots show mean ± s.e.m.



Supplementary Figure 2

Quantification of VMHvl population response heterogeneity.

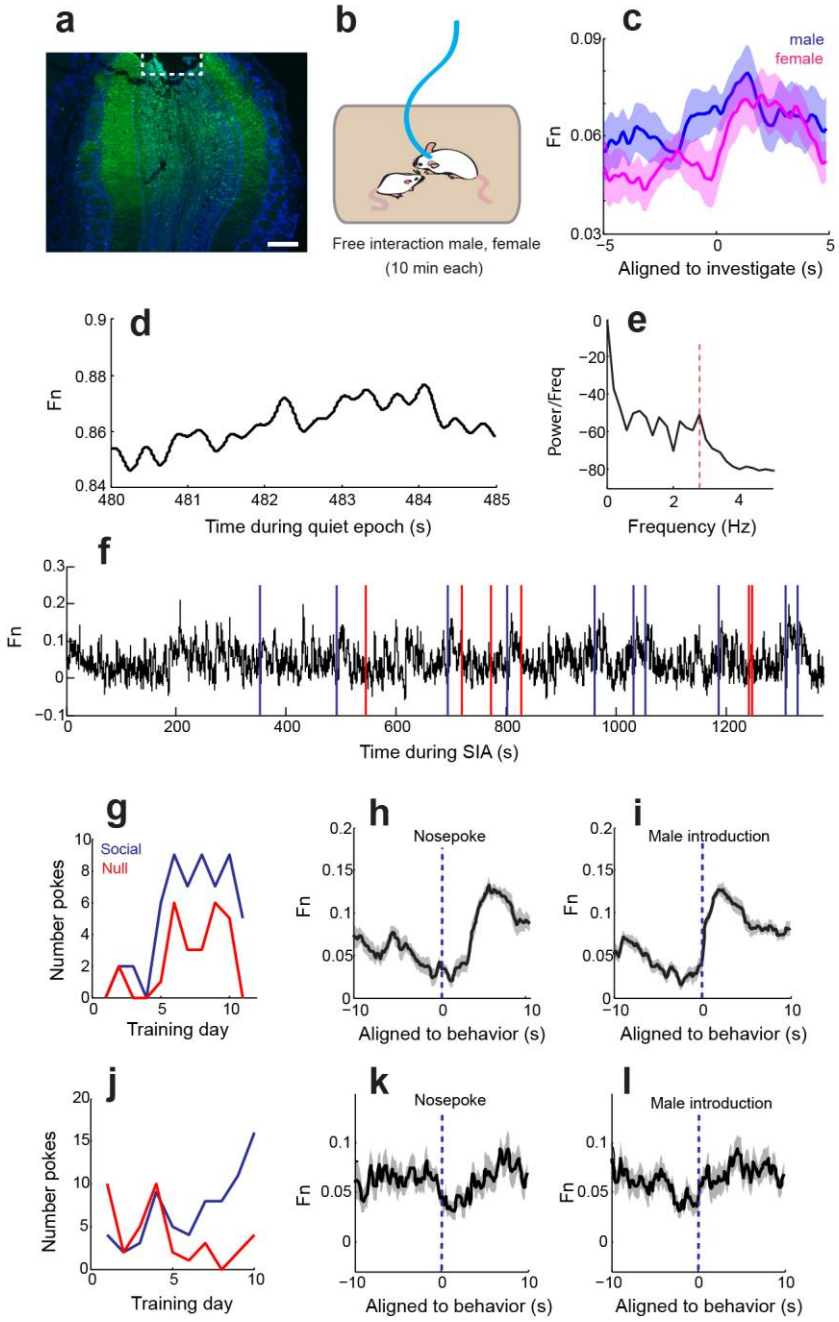
(**a**) Representative waveforms (top) and sorted spikes (bottom) for single units (blue, green) are separable from multi-unit noise (yellow) using PCA analysis of spike waveform shape. Shown are 2 units of 169 total single units. (**b**) Representative histology for electrode implanted in the VMHvl. Blue: DAPI. Scale bar: 250 μm. (**c**) Unbiased clustering of neural activity from population activity matrix (**Fig. 2g**) shows separation between neurons modulated during the poke, wait, and interaction phases. Ward's method, n = 169 neurons. PETH plots show mean normalized activity ± s.e.m. of neurons contained within the four primary clusters.



Supplementary Figure 3

GCaMP6 signal in a non-learner.

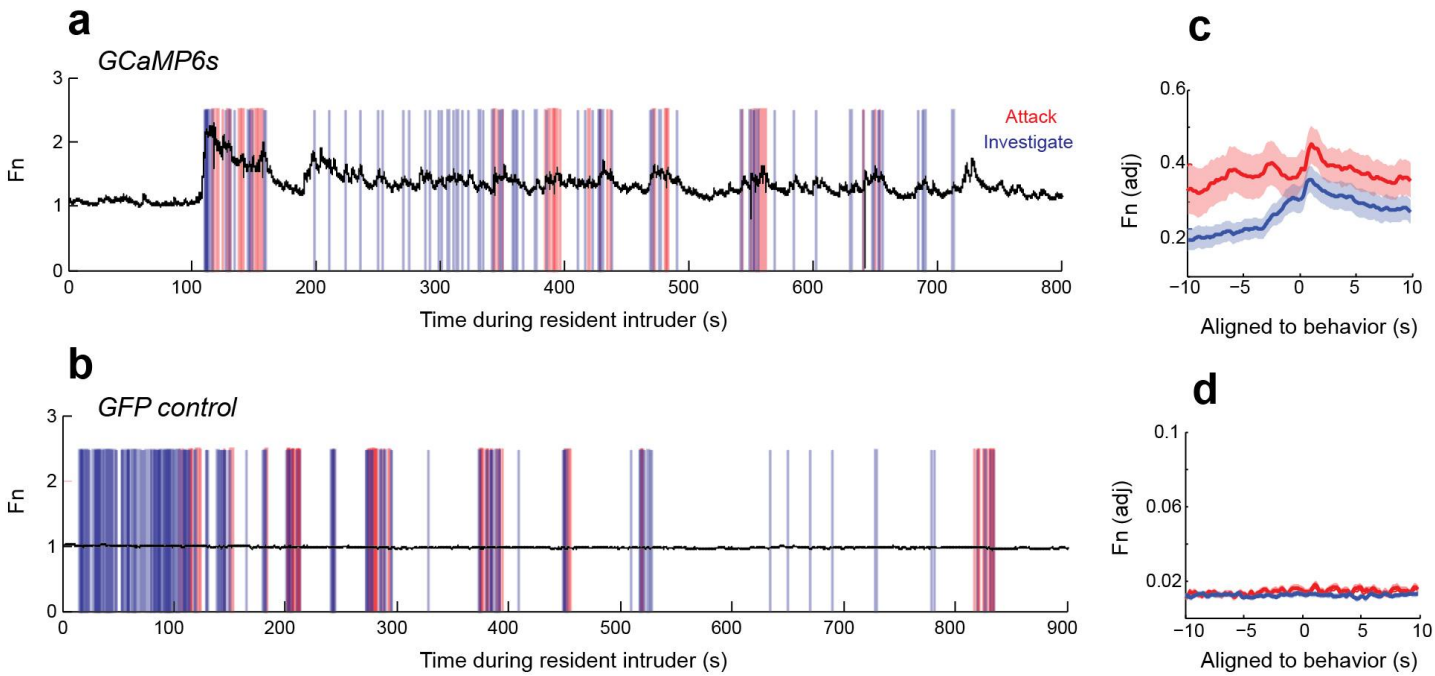
(**a**) Learning curve for an animal that did not meet learning criteria for task learning. (**b**) GCaMP6 signal for the final training session of the non-learner. Poke times for social (blue) and null (red) are indicated by vertical lines. Insets show responses aligned to nosepokes (blue vertical), with red dots indicating the introduction of the male. (**c**) Mean social poke-aligned GCaMP6 response ± s.e.m. for final session. (**d**) Poke aligned activity for all sessions for the non-learner. Shading shows transition from early (red) to late (blue) training. (**e**) Slopes of activity shown in (**d**) as a function of training day show no consistent effect.



Supplementary Figure 4

Responses in the main olfactory bulb increase during social interactions but not during nosepoke.

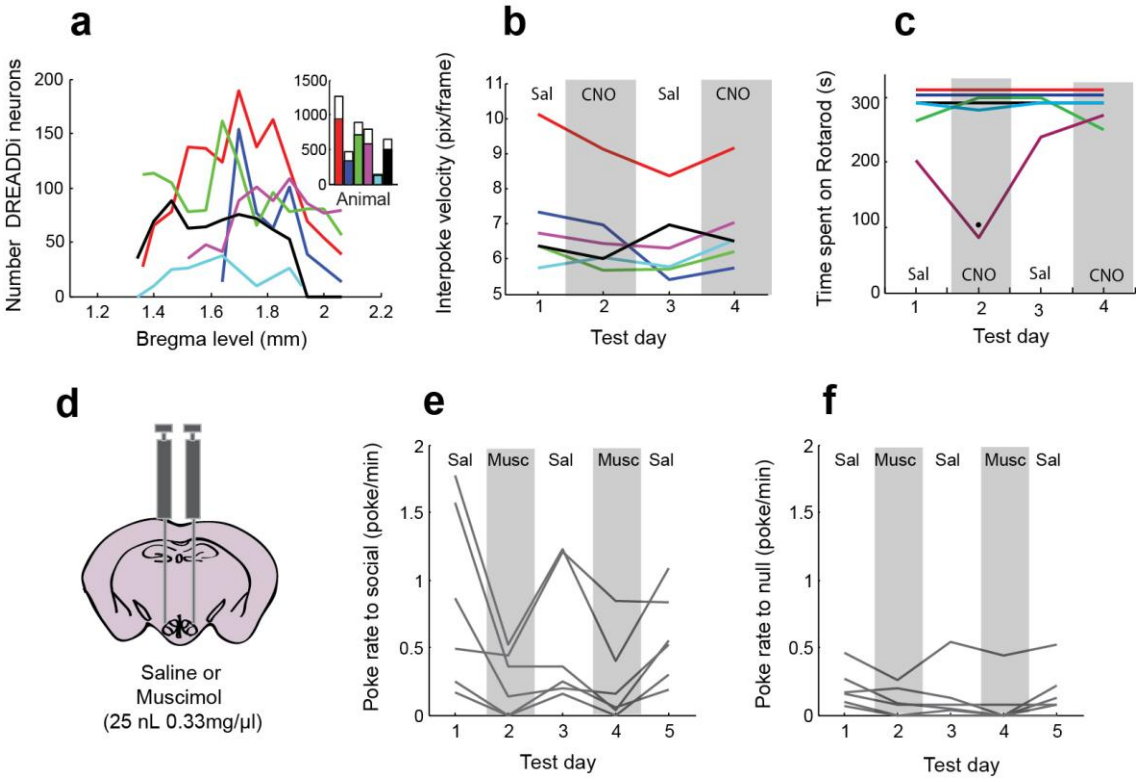
(**a**) Representative image showing histology, fiber placement, and GCaMP6s expression in the olfactory bulb. Scale bar indicates 250 μm. (**b**) MOB activity was recorded during free social interactions with a male or a female. (**c**) MOB activity increases during investigative episodes of either males or females. (**d-e**) Activity during quiet non-investigating epochs (**d**) has increased power at a frequency matching resting mouse respiratory rate (**e**, ~2.7 per s, red vertical). (**f**) Example MOB activity during SIA task, with social and null pokes indicated by blue and red vertical lines respectively. (**g-l**) Learning curves (**g, j**), and population GCaMP6 responses aligned to nosepoke (**h, k**) and male introduction (**i, l**) for two individuals. **c, h-i, k-l** show mean ± s.e.m.



Supplementary Figure 5

GFP control for movement-induced artifact.

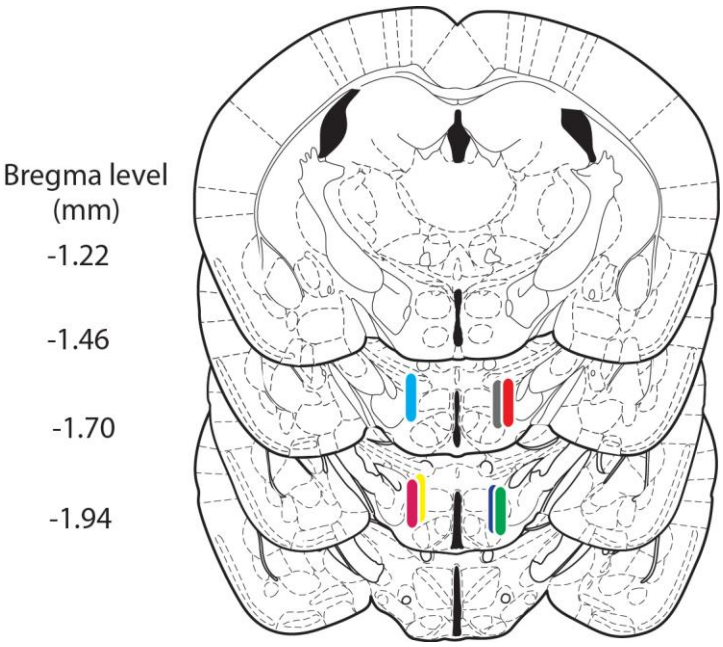
(**a**) Optical recording of GCaMP6 signal in VMHvl neurons during 15 minute resident intruder test shows responses during social interactions. (**b**) Optical recording of VMHvl neurons expressing GFP. (**c-d**) Behavior aligned fluorescence for attack (red) and investigation (blue) for GCaMP6s (**c**) and GFP (**d**) expressing animals. Activity shown is mean ± s.e.m. using 100 ms bins.



Supplementary Figure 6

Control data for pharmacogenetic inactivation experiments.

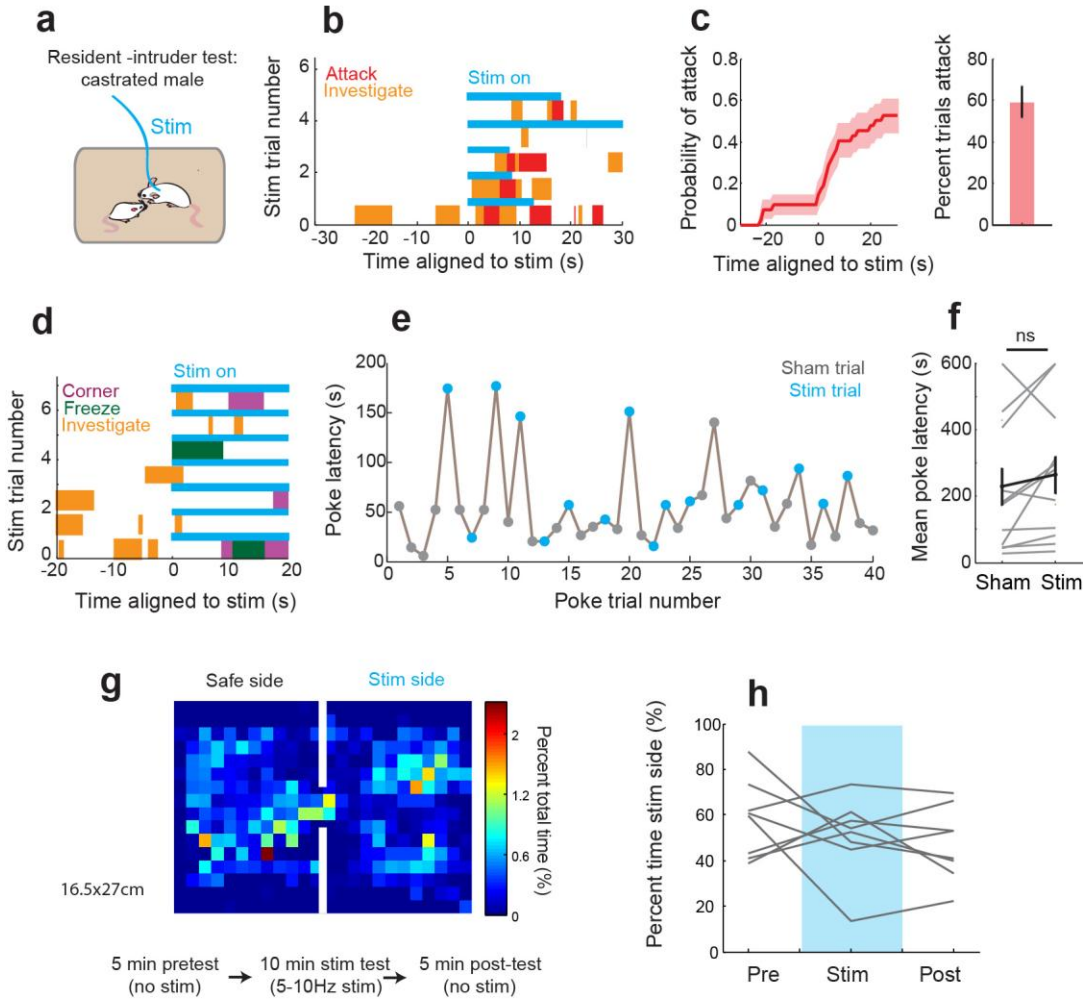
(**a**) Total number of DREADDi infected neurons shown for each animal, with colors coded as in **Fig. 5d and f-g**. Inset shows the number of neurons inside the VMHvl (color) compared with the number of neurons outside the VMHvl border (white) for each animal. (**b-c**) Inactivation of the VMHvl does not cause changes in the interpoke interval movement velocity (**b**, $F_{3,15} = 1.75$, $p = 0.200$) or on ability to run on a rotarod (**c**, $F_{3,15} = 0.74$, $p = 0.546$, one way repeated measures ANOVA). Dot represents an animal that was bitten on the foot by the intruder during CNO inactivation, and rotarod trial was aborted early. Lines were spaced for clarity. (**d-f**) Inactivation using muscimol also decreased poke rate for the social port (**e**, $F_{4,20} = 4.10$, $^*p = 0.014$) but not the null port (**f**, $F_{4,20} = 2.22$, $p = 0.104$, one way repeated measures ANOVA) on test days relative to saline-injected control days (N = 6 animals).



Supplementary Figure 7

Cannula placement for light delivery to functional ChR2 sites during the SIA task.

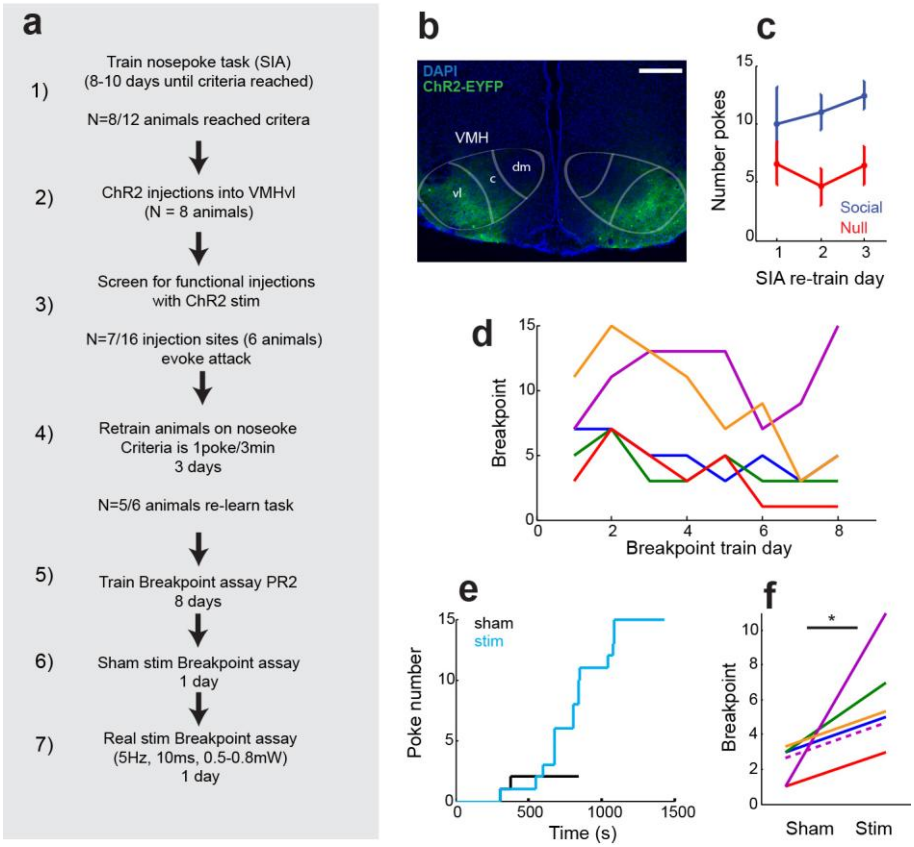
Locations of cannulas (N =7 sites from 6 animals, see Figure 6). Colors correspond to colors used in Fig 6e and g.



Supplementary Figure 8

Control data for optogenetic stimulation of VMHvl neurons.

(**a**) Animals were screened for functional injection sites by testing for stimulation-evoked attack of a castrated male. (**b**) An example session showing stimulation-induced behavioral changes from a functional site. (**c**) Accumulated probability of attack after stimulation onset for functional sites (left). Percent of "successful" trials where stimulation evoked attack (right). N = 7 sites. (**d**) Example of stimulation-evoked behavior from a non-functional injection site. (**e**) Stimulation during the SIA task of the same non-functional site as in (**d**). (**f**) No significant difference in mean poke latency between sham and real stimulation in non-functional sites (t(10) = -1.764, p = 0.102, paired t-test, N = 11 sites in 9 animals). (**g**) Heat map showing behavior during real time place preference (RTPP) of a representative animal. Light was delivered whenever the animal entered the right side of the chamber. (**h**) Stimulation did not significantly bias the animal towards or away from the stimulation paired chamber ($F_{2,7}$=1.05, p = 0.3752, single factor, repeated measures ANOVA, N = 8 animals). **c, f** show mean ± s.e.m.



Supplementary Figure 9

Optogenetic stimulation of VMHvl increases breakpoint using a progressive-ratio reinforcement schedule.

(**a**) Testing and training steps for breakpoint stimulation experiments. Animals were trained and tested using a PR2 schedule. (**b**) Example histology showing ChR2-EYFP bilateral expression in the VMHvl that was sufficient to evoke attack on either side (step 3, **a**). Scale bar indicates 250 μm. (**c**) Animals were retrained on the SIA task (step 4) to insure sufficient poke rates for PR testing (N = 6 animals). Learning curves show mean ± s.e.m. for social (blue) and null (red) ports. (**d**) Behavior during breakpoint training (8 days, N = 5 animals). (**e**) Example behavior showing cumulative nosepoke number during sham (black) and stim (blue) testing of the PR2 breakpoint assay. (**f**) Optogenetic stimulation of VMHvl increased breakpoint across animals ($t(5) = -2.803$, $^{*}p = 0.038$, N = 6 sites in 5 animals with functional injection sites, paired *t*-test). Lines have been spaced for clarity. Dotted and solid lines of the same color represent sites from the same animal.