

## Supplemental Methods (Anderson and Strowbridge 2014)

***In vivo recordings.*** Wild-type C57BL/6 mice (P21-30, either sex; Harlan Laboratories) initially underwent a surgical procedure (ketamine 100 mg/kg, xylazine 10 mg/kg i.p. anesthesia) to mount a small titanium plate (9 x 25 mm) with two bolt holes on the dorsal surface of the skull using Metabond adhesive (Parkell). One day following surgery, mice were water restricted and maintained at 85% of their presurgery body weight throughout the study. Mice were positioned near the apex of a levitated 20.3 cm diameter Styrofoam ball using two 4-40 metal bolts that mounted the head plate to the steel superstructure surrounding the treadmill during conditioning and recording sessions. Mice could sit or walk on the treadmill, or groom themselves while head-restrained. Motion of the treadmill was detected using an optical mouse (MX-518, Logitech) connected to a portable netbook computer. Custom VB.NET (Microsoft) programs converted treadmill velocity into two analog signals (rotation along the treadmill midline induced by forward walking or running and rotation along the equator) using a USB multifunction device (U3, Labjack) every 100 ms. The rapid transitions in plots of treadmill velocity reflect this intermittent sampling. We conducted 2-3 instrument conditioning sessions (15-30 min duration) where mice were given water rewards (10  $\mu$ l delivered through a sip tube, Kent Instruments) in proportion to their forward velocity on the treadmill. This protocol rewarded walking and running movements on the treadmill, a behavior that most subjects continued to perform spontaneously even after they had received the requisite number of water rewards. The recording periods analyzed in this study did not include episodes with water rewards, either because spontaneous bouts of forward motion did not reach the threshold velocity required to trigger a reward or because the maximum possible water rewards had already been reached on that day.

Mice underwent a second surgical procedure to create a small craniotomy over the dorsal dentate gyrus (1-1.25 mm diameter; 3.1 mm caudal to bregma, 2.7 mm lateral ) prior to the first recording session using isoflurane anesthesia. To achieve reliable targeting of the granule cell layer (GCL), the dura was removed within the craniotomy and the brain tissue superficial to the dentate gyrus was aspirated to a depth of approximately 1-1.5 mm using a fine plastic pipette tip. The craniotomy was irrigated using warmed (40°C) artificial cerebral spinal fluid (ACSF, in mM: 124 NaCl, 3 KCl, 1.23 NaH<sub>2</sub>PO<sub>4</sub>, 1.2 MgSO<sub>4</sub>, 26 NaHCO<sub>3</sub>, 10 dextrose, 2.5 CaCl<sub>2</sub>, equilibrated with 95% O<sub>2</sub>/ 5% CO<sub>2</sub>). Except where noted, all compounds used in this study were obtained from Sigma. Patch clamp electrodes (1.2 mm OD, 0.96 mm ID borosilicate tubing containing a filament (WPI); typical resistance 6-8 MOhms) were advanced to within 500  $\mu$ m of the GCL using a motorized manipulator (MP-285, Sutter) while applying strong positive pressure (75-90 kPa) on the electrode tip. Pipettes were filled with an internal solution that contained (in mM): 140 K-methylsulfate (MP Biochemicals), 4 NaCl, 10 HEPES, 0.2 EGTA, 4 MgATP, 0.3 Na<sub>3</sub>GTP, 10 phosphocreatine, 0.1 Alexa594 (Invitrogen), pH 7.3 and

~290 mOsm. Once positioned near the GCL, the positive pressure applied to the electrode was reduced to 1.4 kPa and intracellular recordings were attempted using blind patch clamp methods and an Axopatch 1D amplifier (Molecular Devices). All recording sessions were conducted on the same day as the craniotomy surgery (range P28-42, mean P32). Subjects appeared to recover from anesthesia within 1-2 hours; the fraction of time they spent running > 2 cm/s during the initial 15 min of head-fixation immediately prior to the first attempted recording (37%) did not significantly differ from the last conditioning session prior to the craniotomy surgery (35%;  $P = 0.77$ ; paired t-test).

We also conducted a series of pilot *in vivo* recording experiments in head-fixed anesthetized mice (ketamine 100 mg/kg) placed on the same apparatus to optimize targeting of the dentate GCL. Alexa594 (100  $\mu$ M) was added to the intracellular solution in these experiments and neurons were visualized following brain fixation with 4% paraformaldehyde with an Axioskop2 fluorescence microscope (Zeiss). Body temperature was monitored using a rectal probe in recordings from anesthetized mice and maintained using a heat lamp or thermal blanket. Nearly all visualized neurons (31 of 34) in these pilot experiments from the same stereotaxic coordinates as the recordings in awake animals (-3.1 mm caudal, 2.7 mm lateral, between 2120 and 2310  $\mu$ m below the dura, mean  $2202 \pm 15 \mu$ m) were located in the dorsal dentate gyrus and corresponded to either granule cells or multipolar interneurons. The remaining recovered neurons were located in CA3 (n=2) or CA1 (n=1) subfields of the hippocampus. The large positive pressures necessary to reliably form patch clamp recordings and the low yield of successful recordings over multiple penetrations in the same subject precluded visualization of neurons in recordings in awake mice. Only two Alexa-filled neurons were recovered from awake intracellular recordings. Both neurons had somata in the GCL. (The processes of both cells were incompletely filled, preventing morphological classification of cell type.) All experiments were approved by the Case Western Reserve University Animal Care and Use Committee.

**Electrophysiology and data analysis.** Intracellular recordings were low-pass filtered at 5 kHz and acquired simultaneously with treadmill velocity signals at 10 kHz (ITC-18 interface, Instrutech) using custom VB.NET software. Plots of overall treadmill velocity (top traces in Figs. 1A-C and 2A) reflect the sum of the two absolute velocities acquired. The spectrogram in Fig. 2A was computed from moving 812 ms windows with 98% overlap. The three power spectra in Fig. 2B were each computed from one 812 ms window starting at -1, 0 or 1 sec relative to the onset of detected treadmill motion in each movement epoch. Basal power spectra were computed from 3-6 windows with no motion in the same episode; the resulting difference spectra was averaged across the population of 18 spontaneous movement epochs to generate the plots in Fig. 2B. Correlations of treadmill velocities and movement duration with  $\alpha$ -band oscillations (Fig. 3A-C) were computed over the initial 2 sec of the spontaneous movement epoch. Histograms of relative spiking timing

(Fig. 3D) were computed by comparing AP onset times with the depolarizing peaks of the intracellular voltage following band-pass filtering (zero phase sixth-order digital Butterworth; pass-band 8-15 Hz). Trial shuffled spike times in Fig. 3D3 were computed by calculating latencies between APs and intracellular oscillation cycles during motion onset from different movement epochs. Intracellular voltages presented were not corrected for the liquid junction potential. Data was analyzed using Matlab 2012a (Mathworks) and statistical tests were performed using Origin 8.5 (OriginLab). All data are presented as mean  $\pm$  s.e.m except where noted. Statistical significance was determined using Student's *t*-test unless specified otherwise.