PNAS RESEARCH ARTICLE | NEUROSCIENCE

Action-driven remapping of hippocampal neuronal populations in jumping rats

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Edited by Thomas Albright, Salk Institute for Biological Studies, La Jolla, CA; received December 7, 2021; accepted May 3, 2022

The current dominant view of the hippocampus is that it is a navigation "device" guided by environmental inputs. Yet, a critical aspect of navigation is a sequence of planned, coordinated actions. We examined the role of action in the neuronal organization of the hippocampus by training rats to jump a gap on a linear track. Recording local field potentials and ensembles of single units in the hippocampus, we found that jumping produced a stereotypic behavior associated with consistent electrophysiological patterns, including phase reset of theta oscillations, predictable global firing-rate changes, and population vector shifts of hippocampal neurons. A subset of neurons ("jump cells") were systematically affected by the gap but only in one direction of travel. Novel place fields emerged and others were either boosted or attenuated by jumping, yet the theta spike phase versus animal position relationship remained unaltered. Thus, jumping involves an action plan for the animal to traverse the same route as without jumping, which is faithfully tracked by hippocampal neuronal activity.

hippocampus | phase precession | theta oscillations | place cells | learning

Significance

A critical aspect of navigation is a sequence of planned, coordinated actions. We examined the neuronal activity of the hippocampus by training rats to jump a gap on a linear track. Stereotypic jumping was associated with phase reset of theta oscillations, predictable global firing-rate changes, and population vector shifts of hippocampal neurons. A subset of neurons ("jump cells") were systematically affected by the gap but only in one direction of travel. Novel place fields also emerged, and others were either boosted or attenuated by jumping. Thus, action plans and expectations also affect dynamic neural processes thought to underlie hippocampal-dependent memory.

An important operation of the brain is to evaluate the consequences of its own action. This function is usually framed in subjective terms, such as planning, foresight, anticipation, expectation, or decision-making (1). Several brain regions, including the hippocampus, have been implicated in the implementation of these hypothetical processes (2, 3). A natural behavior that succinctly encompasses such complex processing is jumping across a gap. Jumping is a risk-taking act with an approach–avoidance conflict, which requires evaluation of environmental signals, precise estimation of the gap distance, comparison of past experience with similar situations, assessment of the available resources of the animal, and temporal coordination of dozens of muscles for effective departure and landing (4). Yet, all animals with legs can jump seemingly effortlessly without the need for special handling, shaping, or training by the investigators (4–7). Vision is a critical input for absolute distance estimation, and movement of the visual field that is produced independently of the observer's movements yields very poor, absolute depth judgements (1). Thus, estimation of distance is an embodied process, requiring action-scaling cues.

For many rodent species, jumping, including prey capture and escape, is one of their most important survival skills (8, 9). Animals employ evolutionarily conserved, innate brain mechanisms for jumping, and the skill can be retained for the animal's lifetime after the task has been mastered (5, 6, 10, 11). In their native niche, rats can leap a distance more than five times their body length (12). Jumping behavior can be studied and described quantitatively in laboratory settings and correlated with brain activity. Yet, surprisingly few studies have employed jumping for physiological studies (13–15).

In the presented work, we examined electrophysiological correlates of jumping in the hippocampus. The current general view of the hippocampus is that it is a navigation "device" (16), guided by environmental cues (17, 18). Extensive work has shown that the hippocampus remaps in response to changes in sensory cues (17, 19–21). Yet, planned actions are just as important to navigation as sensory inputs (22). Jumping is a special form of action and includes defined sequences of movement patterns. In contrast to sensory cues placed on a track (23), the presence of a gap on a track requires a different action plan for the animal to traverse the same route surrounded by unchanging environmental cues. We report here that when rats jump a small gap on a running track, firing patterns are robustly reorganized not only for the gap part of the track but virtually for the entire travel. Thus, movement trajectory patterns depend both on environmental inputs and appropriate motor plans, and their conjunction is reflected by the sequential activity of hippocampal neurons.

RESULTS

We trained rats to walk back and forth on a linear track (1.8 m) for water reward, available at a platform at each end of the track (Fig. 1 *A* and *B*). After they moved

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Author contributions: G.B. designed research; D.T. performed research; J.R. contributed new reagents/analytic tools; L.G. analyzed data; and G.B. wrote the paper.

The authors declare no competing interest.

This article is a PNAS Direct Submission.

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This article contains supporting information online at http://www.pnas.org/lookup/suppl/doi:10.1073/pnas.2122141119/-/DCSupplemental.

Published June 22, 2022.

comfortably and steadily on the track, the rats were trained to jump a gap within the track. During this shaping period, the gap distance was gradually increased from 5 to 30 cm. Following these pretraining sessions (5 to 15 d in different rats), the formal jump sessions started. A 30-cm gap between two parts of the track were created at one of three fixed locations on the track (Fig. 1 *A* and *B*). After 7 to 20 prejump control trials (no gap), a gap was introduced, requiring the rat to jump across it in both directions of travel. After 20 trials, the position of the gap position was shifted, and after the second block of 20 trials, a third gap position was introduced. The order of the gap positions varied randomly across sessions. After the jump trials (a trial equaled travel in one direction), the rat ran post-jump control trials without a gap until it was satiated.

Each animal was implanted with silicon probes above the dorsal hippocampal CA1 pyramidal layer. The recording headstage also contained an accelerometer, which allowed continuous monitoring of the *x*, *y*, and *z* positions; velocity; and acceleration of the rat's head (Fig. 1*C*). In addition, head position was tracked with an OptiTrack system including six infrared cameras that allowed for the three-dimensional reconstruction of the animals' head position and head orientation to within 1 mm at 120 Hz (*Materials and Methods*).

Behavioral Observations. Traversing the track on control trials took a median of 2.0 s at a median speed of 76 cm/s ($n = 2{,}212$) (*SI Appendix*, Fig. 1). Rats learned to jump the gaps quickly at high efficiency. One rat fell once during training, but never during recordings, over the course of more than 3,000 jumps in 28 sessions across the 4 rats (1 m) from track to floor. Wild rats can fall from a height of 50 feet (~15 m) without getting hurt, corresponding to >99.9% efficacy (12).

The act of jumping can be divided into four distinguishable phases: preparation, takeoff, flight, and landing (Fig. 1*B*). During preparation, the rat aligned its four feet to the edge of the track, lowered its head steadily, or moved its head up and down a few times (Movie S1). Such self-generated head bobbing produces retinal motion of the image of the landing platform and is critical to determine the desired distance of the jump (9). The time of takeoff was determined from the accelerometer reading as the peak of the second derivative of the accelerometer reading of the horizontal direction (Fig. 1 *D*–*G*). Similarly, the location and time of landing on the track were determined from the minimum of the second derivative of the accelerometer reading of the vertical direction (Fig. 1*H* and *SI Appendix*, Fig. 1). The duration of preparation phase was longest at the middle gap (gap 2) in both directions of travel (Fig. 1*I*; mean ± S.D. wait time: gap 1 = 1.04 ± 0.52 s, gap 2 = 1.31 ± 0.49 s, and gap 3 = 1.14 ± 0.67). Flight time (i.e., time in air) was consistent across sessions and rats (Fig. 1*J*; mean ± S.D. flight time: jump 1= 0.17 ± 0.03 s [$n = 1{,}536$]; jump 2 = 0.18 ± 0.04 s; jump 3 = 0.19 ± 0.05 s). During the flight, the peak velocity exceeded 1.7 m/s, and the velocity and acceleration profiles varied stereotypically across trials (Fig. 1 *E*, *F*, and *I* and *SI Appendix*, Fig. 1; mean ± S.D. velocity: jump 1 = 176 ± 33 cm/s; jump 2 = 171 ± 48 cm/s; jump 3 = 164 ± 51 cm/s). The rat walked relatively slowly prior to jump and maintained momentum after jumping by galloping after landing (Fig. 1 *E*–*G*; before jump = 57 m/s; after jump = 120 m/s; $P = 0$ by Kruskal-Wallis test). Galloping was relatively stereotyped at ~5 Hz, as illustrated by the vertical bands in Fig. 1*D*. This difference in running patterns likely reflected the animal's increased confidence after the jump, since it did not depend on the available travel distance before or after the jump.

Local Field Potential and Population Firing-Rate Correlates of Gap Jumping. The theta frequency in the local field potential (LFP) increased in frequency and power during jumping, reaching a maximum frequency (9.3 ± 1.0 Hz) during the flight phase and after landing, in time with the rat's velocity (Fig. 2*A*) and firing rates of interneurons (Fig. 2*B*). Interestingly, the rate changes of interneurons and pyramidal cells in CA1 preceded those in CA3 (*SI Appendix*, Fig. 2), implying that CA1 activity was not inherited from CA3 neurons. Theta frequency during postjump galloping was significantly higher than during prejump walking (Fig. 2*C*). The firing rates of interneurons showed a relatively linear relationship with speed during running (Fig. 2 *D* and *E*), but this relationship became nonlinear at very high speeds (24), associated with jumping (Fig. 2 *F* and *G*).

Jumping reset the phase of theta waves. The reliability of theta-phase reset was quantified by the phase consistency across trials (25) (*SI Appendix*, *Materials and Methods* and Fig. 10), referenced to the moment of takeoff (Figs. 1*E* and 2*F*). Theta-phase reset was also visible by the increased phase consistence of interneuron spiking (Fig. 2*G*). The consequence of theta-phase reset was the persisting phase consistency for several theta cycles, although significant phase consistency was present only for three cycles, surrounding the jump (Fig. 2*F*). Given this short duration and the presence of only one or two theta cycles during the jump itself, we could not determine precisely whether the reset occurred during the takeoff or the flight.

Neuronal Population-Measure Correlates of Gap Jumping. We examined encoding of the jump by computing population vector correlations, which tell us how hippocampal activity is correlated in space. The population vector correlations were computed by first *z*-scoring the trial-averaged firing rate for a given position of each neuron with mean activity above a noise threshold in at least one jump or control condition (Fig. 3*A*) and then calculating the correlation across neurons between their activities at each combination of position bins (Fig. 3*B*). Average population vector correlation decreased to near zero within <40 cm of separation (Fig. 3*C*), as reported previously in similar situations (26, 27). The population vector correlation analysis includes all active neurons and, therefore, is not biased by the definition of a place cell. In contrast to the high spatial correlation across control trials, spatial correlations of CA1 population activity between control and jump trials showed a strong reduction, except at the two extreme ends of the track. Importantly, the decreased spatial correlation was not restricted to the gap area; it was already low prior to the jump and persisted after the jump (Fig. 3 *D*–*G*).

Comparison of jump trials across different gap conditions also yielded low spatial correlations (Fig. 3 *H*–*K*), indicating remapping of neuronal activity across jump trials as well. Yet, in contrast to the steady near-zero correlations between control and jump trials (Fig. 3*G*), comparison of jump trials revealed a visible increase of population vector correlations at the jump location across gap positions (Fig. 3*K*). This observation implies the possibility of the presence of neurons, which represent different gaps similarly (see remarks about jump cells in *Discussion*). Population representation and changes across conditions were remarkably similar between CA1 and CA3 populations (Fig. 3 *L*–*O*). These findings are in agreement with previous analyses showing that population vector correlation is a more sensitive measure than population firing-rate analysis (compare Figs. 2*B* and 3*D*) (26).

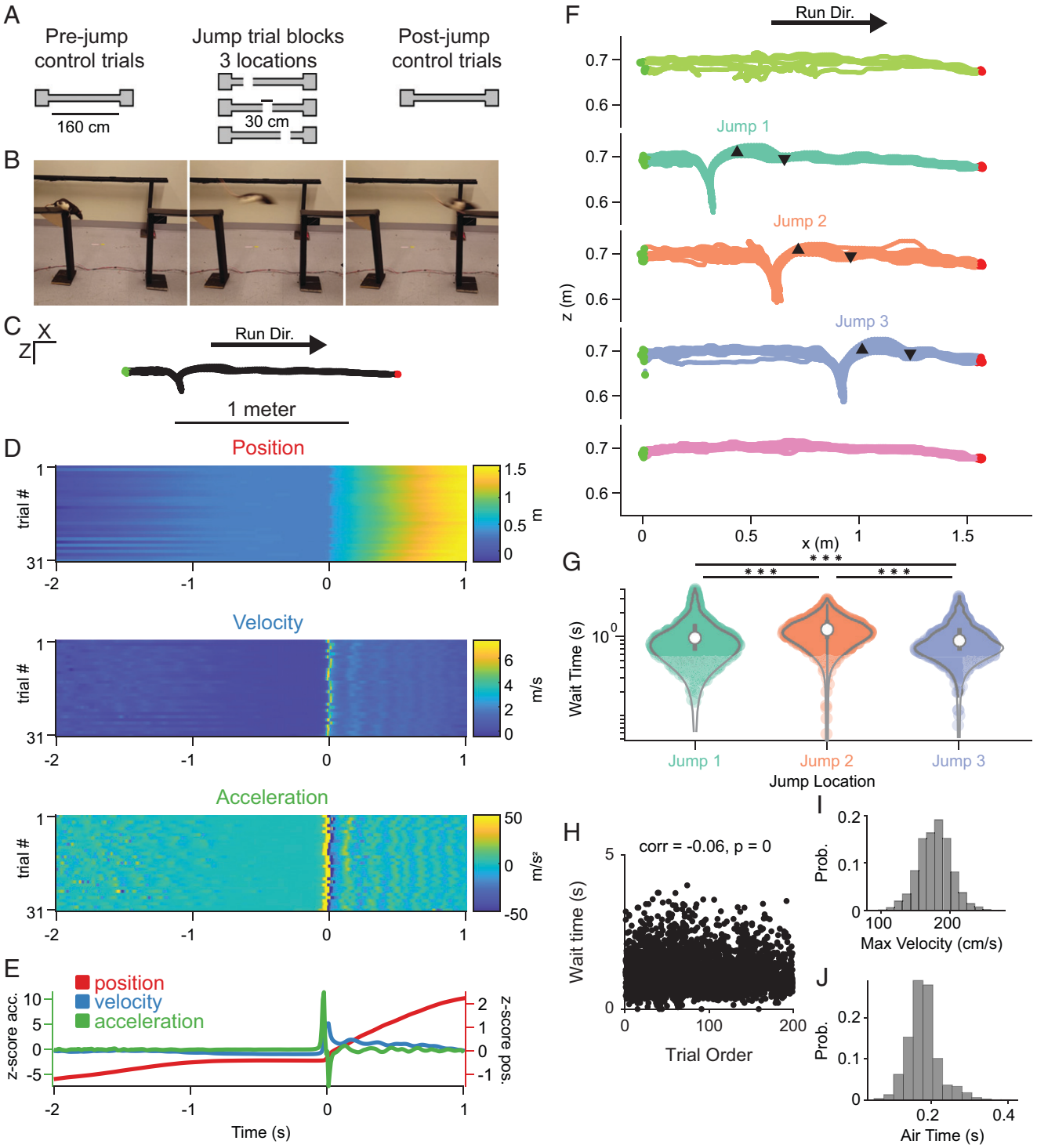

Fig. 1. Jumping a gap is stereotypic. (*A*) Running track and experimental schedule. In each session, the rat ran on the track back and forth for a water reward (prejump control trials), followed by three blocks of jump trials at three different locations. The gap locations (30 cm) on the track are indicated. The first, second, and third gaps are located 50 cm, 80 cm, and 110 cm after the start, respectively. After the jump blocks, the rat ran again on the uninterrupted track (postjump control trials). (*B*) Photographs of a rat prior to takeoff, during the flight, and landing. The rat crouches at the edge of track before jumping. Note the deep head position prior to takeoff. (*C*) Horizontal and vertical positions of a rat at the first jump location (n = 31 trials). Green: start location; red: end location. (*D*) Horizontal position (*Top*), velocity (*Middle*), and acceleration (*Bottom*) from 2 s before to 1 s after jump takeoff. Time 0 = jump takeoff. (*E*) The *z*-scored average horizontal position (red; *x*-axis), velocity (blue), and acceleration (green). The peak acceleration was used to determine the takeoff moment. (*F*) The *x* and *z* positions from a representative session. Takeoff and landing are indicated by upward and downward arrowheads. (*G*) Preparation (wait) times before jump for all sessions. Data from trials in the left and right directions are combined. ***$P < 0.001$ by Kruskal-Wallis test, with correction for multiple comparison. (*H*) Distribution of wait times is not correlated with the order of trials (as a proxy for fatigue). (*I*) Distribution of maximum flight velocity during jump (mean = 178 ± 25 cm/s). (*J*) Distribution of air times (flight) during jump (mean = 0.18 ± 0.04 s). acc., acceleration; corr, correlation; dir., direction; max, maximum; pos., position; prob., probability.

Single Neuron-Firing Correlates of Gap Jumping. The population analysis suggested that the act of jumping resulted in a combination of rate and global remapping of place fields (19, 20) of individual hippocampal pyramidal neurons in virtually the entire track, even though the distant spatial cues and motor behavior remained similar before and, to a great extent,

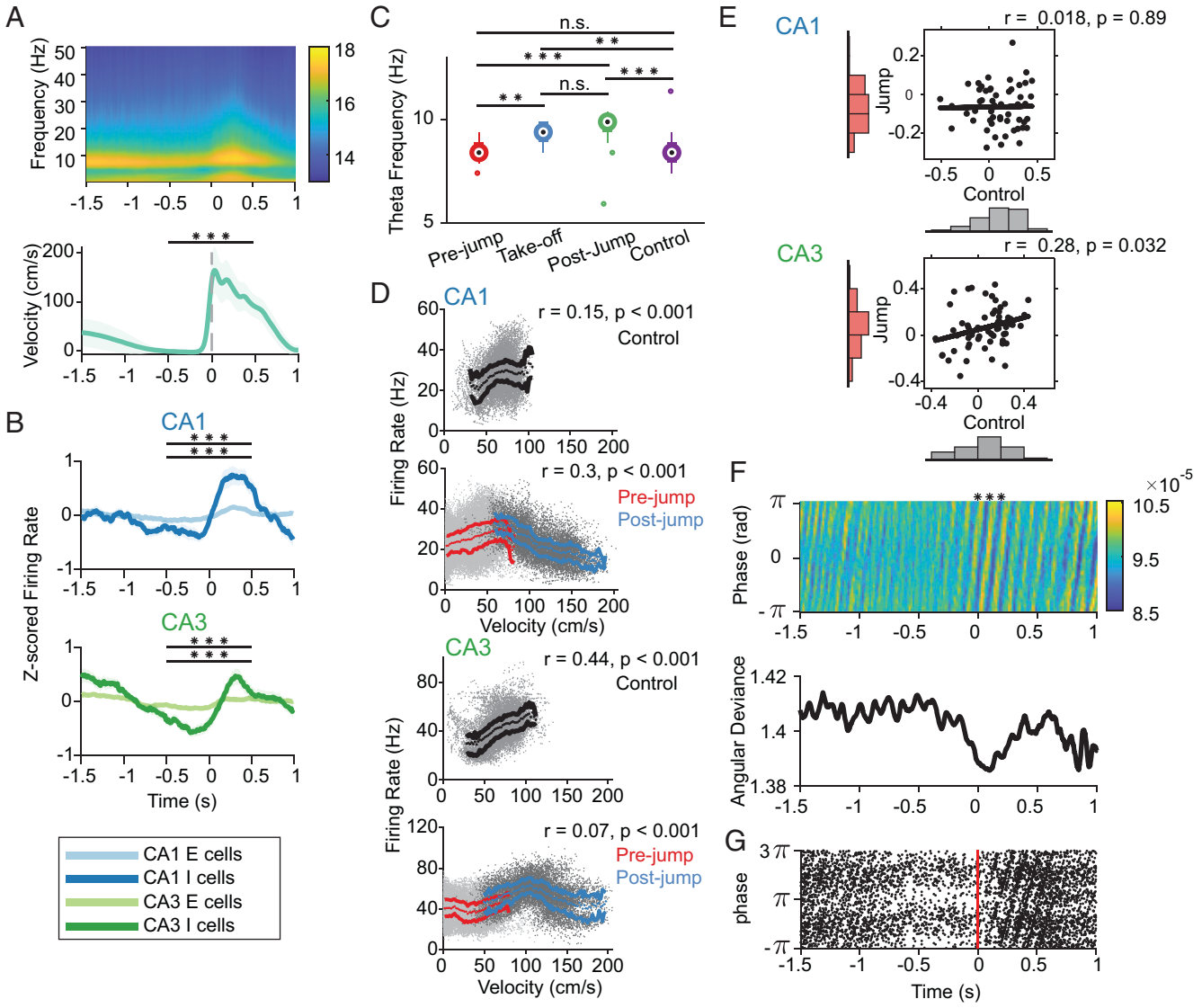

Fig. 2. LFP and population firing-rate correlates of gap jumping. (*A*) Time-resolved spectrogram of LFP before, during, and after jump. Note increased theta frequency and power during the jump. Time 0 s refers to the jump takeoff. Data are from an average of 2,727 trials from 28 sessions. *Bottom*, Average velocity during jump trials at jump location 2 (n = 28 sessions). (*B*) Average z-scored firing rates of CA1 (*Top*) and CA3 (*Bottom*) neurons during jump trials. Light blue: pyramidal cells; dark blue: interneurons. Shaded area shows SE. Firing rates from 0 to 0.5 s after the jump were significantly greater than firing rates at 0 to 0.5 s before the jump for both pyramidal cells and interneurons (CA1: $P = 4.6 \times 10^{-18}$ and $P = 0.006$, by Kruskall-Wallis test, n = 875 pyramidal cells and 59 interneurons; CA3: $P = 3.55 \times 10^{-9}$ and $P = 4.46 \times 10^{-5}$, n = 1,085 pyramidal cells and 73 interneurons, E cells: pyramidal cells; I cells: interneurons). (*C*) Mean theta frequency before, during, and after the jump. Red: 1.5 s before the jump; blue: time of jump takeoff (0 s in *A*); green: 0.3 s after landing; purple: halfway point of control trials. These values were obtained from the time-resolved spectra (as in *A*). $^{**}P < 0.01$ and $^{***}P < 0.001$ by Kruskall-Wallis test of median theta frequency values. Small symbols indicate outliers. (*D*) Firing rate versus animal velocity of an example CA1 interneuron (*Top*) during control trials (*Top*) and jump trials (*Bottom*). Black dots show mean firing rate and 25% to 75% quartile for each velocity bin. Red and blue dots show mean firing rate and 25% to 75% quartile for each velocity bin for prejump and postjump running, respectively. Note that that at highest speeds (jumps), the firing rate–speed relationship deviates from linearity. Inset shows the Spearman's rank correlation. The bottom two graphs report the same type of information but for an example CA3 interneuron. (*E*) Comparison of Spearman's rank correlation between firing rate and running velocity on control trials and jump trials (prejump only). There is an inconsistent relation between firing rate and speed in CA1 interneurons, and a weak but consistent relation with speed-specific CA3 interneurons across conditions. (*F*) Histogram of theta phase before and after jumps from all sessions. Theta phase is uniform before the jump but shows a phase reset after the jump (n = 2,727 trials), as reflected by the clustering of phases in the time-phase histogram. *Bottom*, Angular deviation of theta phase before and after jump. The angular deviation of theta phase (defined as the circular analog of SD) decreases after the jump. Asterisks represent time points when the distribution of phases is significantly nonuniform, from 25 ms before the jump to 144 ms afterward (Rayleigh test, $P < 0.05$), corresponding approximately to two theta cycles. (*G*) Example CA3 interneuron, showing a spike theta-phase alignment after the jump takeoff (n = 32 trials). Note diagonal bands at theta frequency after the jump in the average. n.s., not significant; std., standard.

after the jump itself. In a typical session, only a small fraction of neurons displayed stable place fields with unaltered rates during jump trials, whereas the overwhelming majority of place fields were modified one way or another. In addition to the minority stable neurons (group 1), neurons with a modified firing pattern could be classified by subjective criteria into the following four major groups: group 2, novel place fields; group 3, neurons whose rates were decreased (truncated or attenuated); group 4, neurons with increased (amplified) rates during jump trials; and group 5, jump cells. Each of these changes could occur before, during, or after the jump (gap). Since hippocampal pyramidal cells form different place fields and sequences during opposite direction runs on linear tracks (28, 29), we evaluated place fields separately on left and right travels.

Stable place fields were found only at the start and end locations of the track (Fig. 4*A* and *SI Appendix*, Fig. 3). Neurons

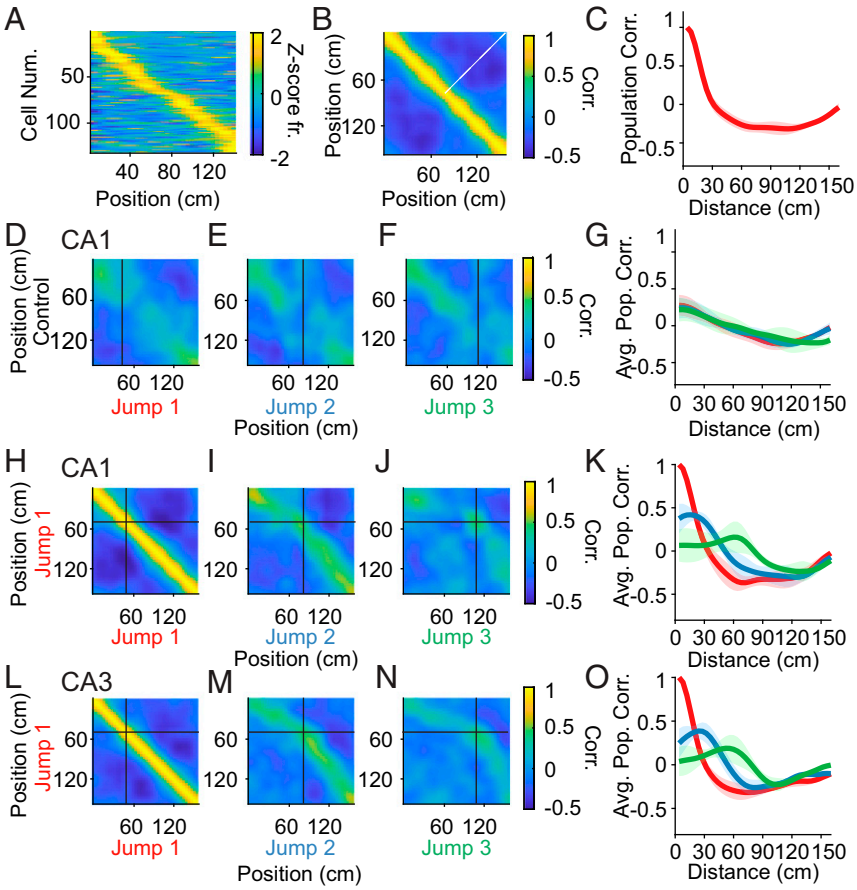

Fig. 3. Population vector representation of jumping. (*A*) Activity of CA1 pyramidal neurons (n = 132 neurons active in the control trials from all sessions). Neurons were ordered sequentially by the z-scored peak firing rates of their best place field. Neurons with mean firing rate <0.1 Hz were excluded. In the following analyses, only neurons with a mean firing rate >0.1 Hz in at least one condition were included (n = 209). (*B*) Population vector correlation matrix of firing rates of the same neurons as in *A*. (*C*) Average spatial correlation with respect to travel distance (average of the off-diagonal lines of the matrix in *B*). Neural activity shows a spatial scale of ~30 cm. (*D–F*) Correlation matrices of the correlations between control trials and jump trials 1 (*D*), 2 (*E*), and 3 (*F*) for all sessions. Note the decrease of correlation coefficients throughout the track, but especially at 50 cm, 80 cm, and 110 cm, respectively, corresponding to the location of the jumped gaps. (*G*) Average correlation with respect to distance between positions of the data in *C–G*. (*H–J*) Population vector correlation matrices of spiking activity (n = 209 CA1 pyramidal cells, active at least in one condition) during jump 1 (analogous to *B*), comparison between jumps 1 (*H*) and 2 (*I*) and comparison between jumps 1 and 3 (*J*). (*K*) Average population vector correlations with respect to distance. Note decreased correlations in the entire track but increased similarity between jumps (compare with *G*). (*L–O*) Same as *H–K* but for CA3 pyramidal cells (n = 275). avg., average; corr, correlation; num., number; pop., population.

with novel place fields (group 2) did not have a place field on prejump control trials, and the new field could occur anywhere on the track, including areas before, after, and even during the jump (Fig. 4 *B–D* and *SI Appendix*, Fig. 3). Neurons in group 3 had an existing place field in control trials, which was suppressed in jump trials. Spike suppression was typically largest when the place field coincided with the gap, but suppression was also present when the gap was either before or after the location of the place field (Fig. 5 *A–C* and *SI Appendix*, Fig. 4). When the gap coincided with the beginning of the place field, the remaining part of the place field was still expressed (Fig. 5*C*, fourth graph down in column). Firing rates of group 4 place cells increased on jump trials. Similar to the attenuated group, enhanced spiking could occur not only during gap jumping itself but also before or after the gap, and the center of the place field moved either toward or away from the gap (Fig. 5 *D–F* and *SI Appendix*, Fig. 5). The distinction of the attenuated and amplified groups is somewhat arbitrary since spike rate decrease and increase could be observed within the same place cell with different jump locations (*SI Appendix*, Figs. 4 and 5).

Neurons classified as jump cells (group 5) also meet the criterion of a novel place field, because firing at the future place field was absent during control trials. However, criteria for jump cells also included the requirements that they fired in relationship with two or three gaps and their fields moved with the gap on different trial types. Jumps cells occurred not only during jumping but could be present either before or after the gap (Fig. 6 *A–C* and *SI Appendix*, Fig. 6), suggesting that the motor action of jumping was not the necessary driver of spiking. When the jump cell occurred prior to departure, its duration lasted throughout the 1- to 1.5-s wait time, during which its spikes underwent a gradual phase precession (Fig. 6 *D–F*). With the exception of one neuron (*SI Appendix*, Fig. 7), jump fields occurred only during either left or right, but not both, direction of travel. Therefore, a more appropriate description of these neurons would be "conjunction cells of gap and head direction."

Although the error-prone nature of subjective classification of firing pattern types are acknowledged, the distribution of these five groups was similar in both CA1 and CA3 regions (Fig. 7), implying a relatively random redistribution of the neuron pool in the context of a modified map. Overall, the analysis of individual neurons supports the remapping conclusion by the population vector analysis. In addition to the aforementioned five groups, the remaining putative pyramidal neurons either had very few spikes to quantify place fields or displayed scattered and nonpatterned firing throughout the track (group

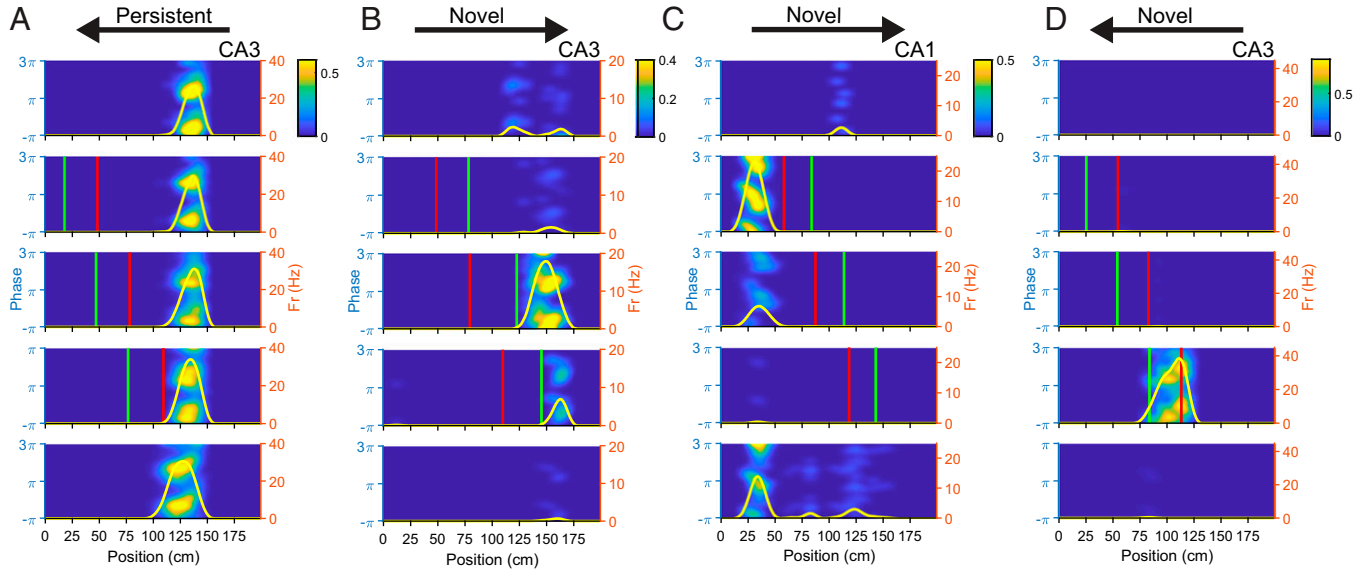

Fig. 4. Persisting and novel place fields. (*A*) Example of a persisting place field. (*B–D*) Example novel place fields after (*B*), before (*C*), and during jumping (*D*). Red and green lines mark takeoff and landing positions.

6: unclassified or "other" neurons; *SI Appendix*, Fig. 8). Yet, firing fields of some of these unclassified neurons were also modified in jump trials (*SI Appendix*, Fig. 8). Fast-firing, putative interneurons showed a correlation with running speed (Fig. 2 *B*, *E*, and *F*) (24), but the relationship between speed and interneuron firing rate was also modified by context, as demonstrated by the different interneuron firing rates at different gaps and the difference between left and right travels (*SI Appendix*, Fig. 9).

Remapping and the Internal Temporal/Theta-Phase Structure of Hippocampal Populations. Despite the large changes of firing patterns of individual neurons, the population structure of the neurons remained similar. First, we investigated the distance relationship between place fields and theta timescale-related timing of place-cell pairs by calculating the compression index (30). The compression index quantifies the relationship between travel distances (or travel time) between the peaks of overlapping place fields and the time (phase) offset of their spikes within theta cycles. When the time and phase offsets of the theta timescale cross-correlograms were plotted as a function of the distance between the peaks of their firing fields, we found that despite the extensive firing-rate modification of individual place fields, the compression index remained similar between control and jump trials (Fig. 8 *A–D*; for detection of theta phase, see *SI Appendix*, Fig. 10), indicating that fine-timescale properties of modified spike sequences within place-coding cell assemblies were preserved despite jump perturbation.

Another measure of place coding is the relationship between the animal's instantaneous position and the theta phase at which the neuron spikes [i.e., phase precession (31)]. Phase precession describes the association between a linear variable (position on track) and a circular phase (theta cycle) variable (32). Despite the prominent spike-rate modulation and moderate place-field shape distortions during jump trials, the position–theta-phase spike relationship remained invariant (Fig. 8 *A, C, G,* and *I*), implying that theta phase is a more reliable "code" of position than a firing rate code (33, 34). This invariance was a result of reduced place-field size and altered slope [radian or field width (32)] during jump trials (Fig. 8 *F* and *H*). The average reduction of place-field size was likely a result of the several place fields silenced midfield due to the presence of the gap (Fig. 5). In a few examples, the major part of the place field coincided with the gap, and spiking was completely suppressed during the jump. Despite the absence of spikes for the major part of the place field, when firing resumed at the end of the field, the theta-phase assignment of spikes was the same as during control trials (Fig. 8*E* and *SI Appendix*, Fig. 10) (25).

DISCUSSON

Jumping produced a stereotypic behavior associated with consistent electrophysiological patterns, including phase reset of LFP theta, global firing-rate changes, and population vector shifts of hippocampal neurons. These collective neuronal patterns were associated with modified firing of individual hippocampal neurons, appearance of novel place fields, and the emergence of jump-specific cells. Despite large changes in firing rates, the theta phase versus animal-position relationships of place cells remained stable. Thus, planning and executions of actions are as effective in altering hippocampal neuronal organization as are environmental cues.

Resetting Theta Oscillations. A reliable relationship between various motor actions and the phase of the theta cycle has previously been reported (35–42). In our experiments, jumping was associated with LFP theta-phase reset. The flight time itself was <0.2 s, typically one or two theta cycles, but the trial-to-trial phase consistency persisted for several theta cycles after the jump. The phase reset coincided with an increase of both theta amplitude and frequency, as also reported during vertical jumping (13, 15, 43, 44). The phase reset was also evident from the session average of interneuron firing, essentially tracking the phase-shifted LFP theta cycles. Finally, when the gap coincided with the place field of a pyramidal neuron, a sudden phase shift in spike–theta-phase preference was detected. This sudden shift may have occurred because in one or two theta cycles, the rat's head was displaced by 30 cm, thus maintaining the relationship between spatial cues and spike–theta-phase relationship (31). Alternatively, the phase shift might reflect the altered relationship between spiking of the jump cell and the phase-shifted reference LFP theta. Another issue that has remained ambiguous is whether jumping induced phase resetting of theta oscillations

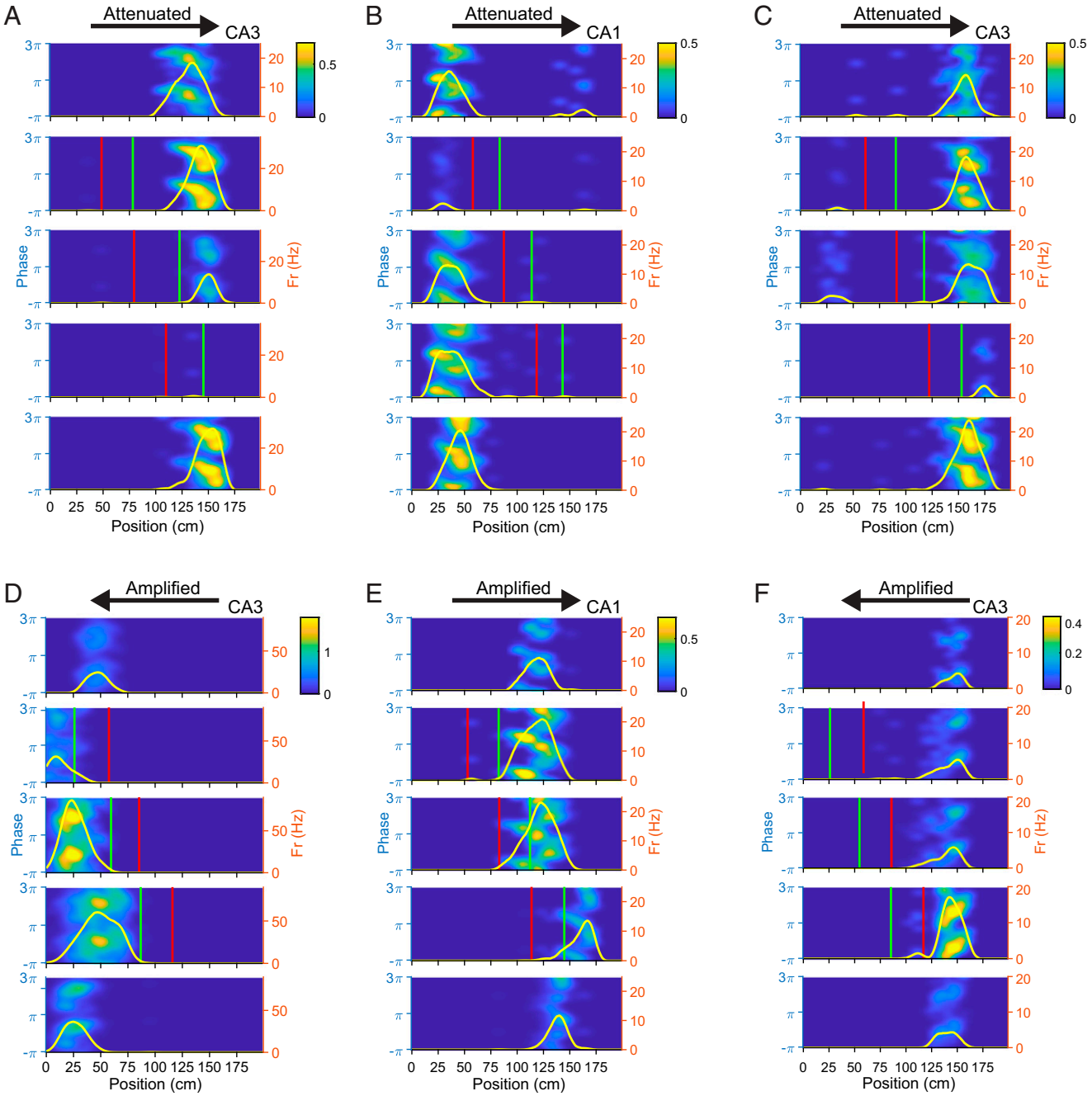

Fig. 5. Jump trials attenuate or amplify place-cell firing. (*A*–*C*) Attenuated firing within place fields during (*A*), before (*B*), and after jumping (*C*). (*D*–*F*) Amplified firing within place fields during and after (*D* and *E*) and before (*F*) jumping.

or whether the timing of jumping was biased by the phase of the theta cycle (35). The theta reset might have been brought about the corollary discharge from the motor command signal or from the sensory feedback of muscle activity (45, 46).

Reorganization of the Hippocampal Map by Jumping. Firing sequences of hippocampal neurons during exploration in one- or two-dimensional environments have been assumed to be driven by internal mechanisms (47, 48). For each environment, a new map with different firing patterns is retrieved, due to shifting from one neuronal "attractor" to another (49), known as remapping (19) or as an altered manifold (50). Under some conditions, reorganization is manifested as a change in firing rates at the same locations ("rate remapping"), whereas between distinct environments, new place fields appear and old ones disappear or move to a new location ["global remapping" (20)]. Remapping can be induced not only by spatial manipulations and changes in sensory inputs (19, 52–55) but also by motivational state (56–59), experience (60–62), and memory load (47, 63–66). In our experiments, nearly all place fields, with the exception of a small fraction of stable place fields at the two ends of the track, likely anchored to the start, or goal, platforms (67), were modified one way or another.

In jump trials, firing pattern reorganization occurred as a combination of global and rate remapping. To explain these changes, one can make the argument that the absence of a piece of the track is a local sensory cue and, thus, this experimenter-induced change is the sole explanation of remapping (17, 18). However, local cues are not expected to induce such a strong remapping as we have observed in our experiments. Several

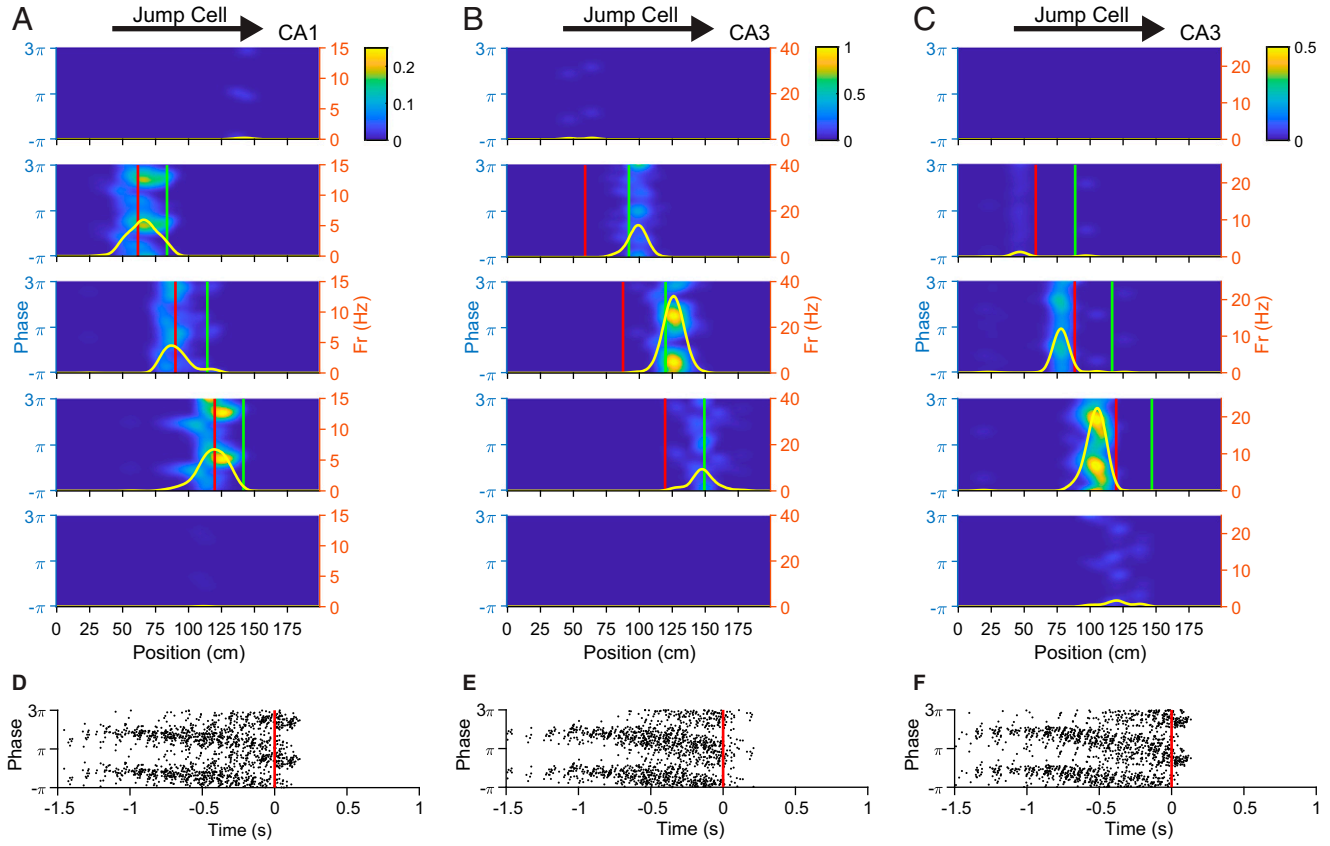

Fig. 6. Jump-specific cells. Jump cells moved reliably with gaps and could emerge during (*A*), after (*B*), or before (*C*) the gap. Note different firing rates at different gap positions. Jumps cells were context specific and fired only during left or right travels (*SI Appendix*, Fig. 6). Note also the sudden theta-phase shift of spiking from takeoff to flight (*A*) and from flight to landing (*B*). See more examples in *SI Appendix*, Fig. 6 *E–G*. (*D–F*) Phase precession in time during wait time prior to departure at gaps 1, 2, and 3 from the cell shown in *A*.

previous studies have examined the impact of overt or hidden local cues on hippocampal neuronal firing. These experiments vary from reporting no effect to reporting moderate effects on place-field activity, including place-field enrichment, smaller place fields, increased spatial tuning (i.e., firing-rate increase), altered phase precession, and increased or reduced spatial encoding (51, 68–72). Common to these previous experiments (but see refs. 68 and 69) is that the described firing-pattern alterations were confined to the particular segment of the environment where the density of visual and tactile cues was enriched. In contrast, in our experiments, remapping occurred virtually on the entire track. In fact, firing-pattern changes were similar at, near, and far from the gap, implying that, in jump trials, the brain regarded the track differently from the same track in control trials without a gap. The implication is that from the beginning of each trial, a unique attractor, containing partially the same neurons, was retrieved, depending on whether it was a control trial or a jump trial with different gap locations. This hypothesis is supported by the relatively equal probability distribution of the five groups of place fields of both CA1 and CA3 neurons on jump trials, including persisting place fields, novel place fields, place fields with increased or decreased firing rates, and jump cells. These findings suggest that the expected different action plan (i.e., preparation for jumping) from the beginning of the trial exerts a stronger internal impact on the hippocampus than do changes in local cues.

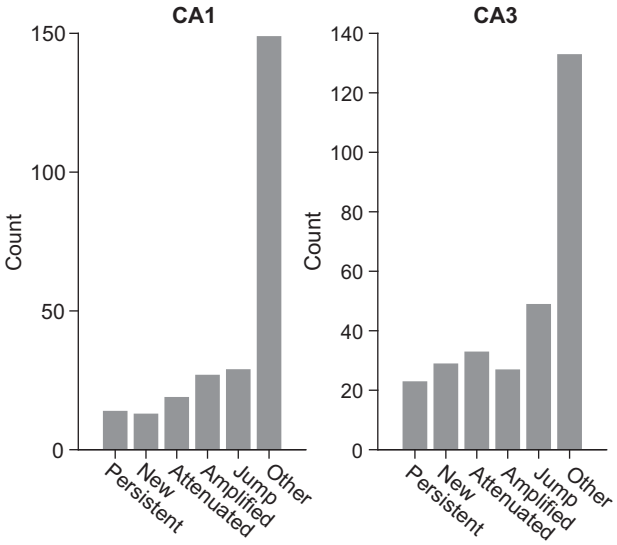

Fig. 7. Distribution of single cell types. Distribution of firing features of CA1 and CA3 pyramidal neurons by categories (groups 1–5). Note that the different categories were evenly distributed in both hippocampal regions. "Other" cell types had low firing rates (<1 Hz) or did not have definable place fields (see examples in *SI Appendix*, Fig. 8).

Neurons whose firing rates were attenuated (group 3) or amplified (group 4) during jump trials may be related (or identical) to "object-location memory" cells (73). When objects are removed from an environment or repositioned, object-location memory cells, located in both CA1 and CA3 regions, increase their firing at the locations where the objects used to be (74). Similar to previous interpretations (73), rate remapping is an indication of conjunctive features of neurons, the ability to simultaneously encode multiple types of information (75, 76).

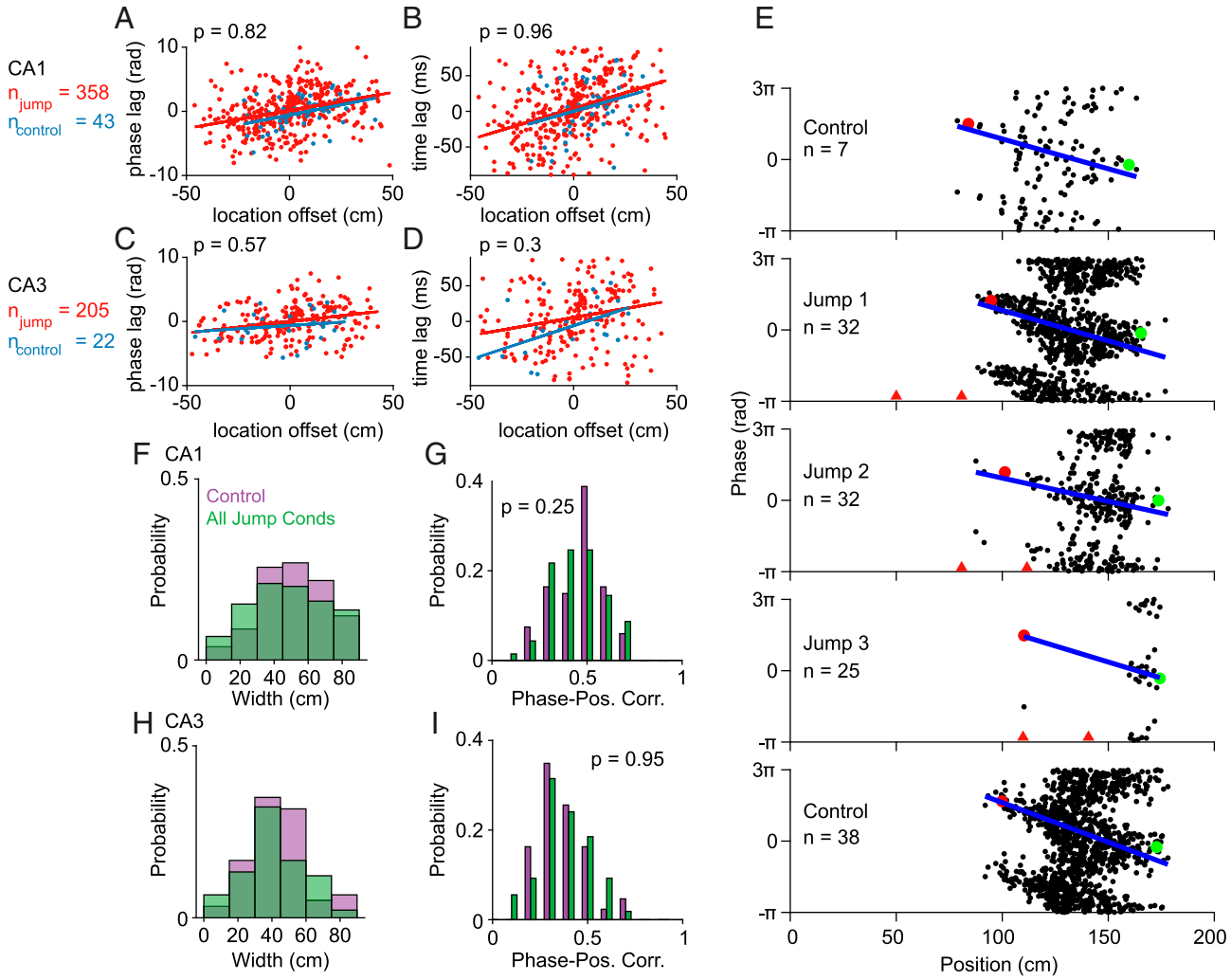

Fig. 8. Preserved properties of hippocampal neurons during jumping. (*A* and *B*) Distance to time/phase compression of CA1 place cell pairs. Distances between peaks of place-field pairs as a function of theta scale-phase difference (*A*) and theta time difference (*B*). (*C* and *D*) Same as *A* and *B* but for CA3 pairs. Note preserved relationships between control and jump trials for both CA1 and CA3 neurons. (*E*) Spiking activity with respect to phase and position of an example neuron in CA1 during control and jump trials. The blue line is the slope (32). Note that during jump 3 trials, the neuron was silent, yet at the end of the place field, it fired at the same theta phase as in other trials. (*F* and *H*) Distribution of CA1 and CA3 place-field widths. While the place fields can both stretch and shorten, the mean place-field size is smaller during jump trials, since parts of the place fields are sometimes cut off during jumps (Fig. 5). (*G and I*) Comparison of the distribution of phase-position correlations (phase-pos. corr.) between control and jump trials.

An explicit mechanism offered for such conjunctive coding is that the spike phase of theta is primarily responsible for locating the animal in the environment (34), whereas the independent firing rate is available to code for other features, such as speed, objects, goals, or motivation (33, 58, 77). This hypothesis is supported by our observations that despite the strong changes in firing rates, the theta-spike phase versus animal-position relationship remained unaltered during jump trials.

According to the classic theory, place fields are induced and maintained by a combination of sensory inputs (16) and intracellular mechanisms (78–80). An alternative view is that neuronal sequences are self-organized at the circuit levels and such preexisting sequences are matched to particular environments (47, 49, 81, 82). Our observation that the compression of distance to theta-phase (time) offset between place-cell pairs remained unaltered during jump trials, despite changes in firing patterns of individual place cells, supports the internally organized hippocampal model. Previous experiments also found that theta time offset of spikes between place-cell pairs remains fixed, despite increasing distances between place-field peaks in larger environments (2), running speed differences (83), or different temporal requirements (84). Yet, action- and environment-anchored reference frames can coexist in the hippocampus (85).

Cases where the preexisting place field coincided with the location of jumping provide insight into the triggering mechanisms and maintenance of place fields. In a few group 3 neurons, the beginning or a large part of a place field was completely obliterated by the jump. Despite the absence of spikes in a large portion of the place field, spiking resumed after the jump and at the expected theta phases. These findings also support the internally organized model, which assumes that that spiking of hippocampal place cells within their fields is an assembly product (86). From this assembly point of view, we hypothesize that the place field in group 3 neurons was not abolished but suppressed by inhibition, and when the neuron was released from inhibition after the jump, it continued to fire together with its less-affected recorded and nonrecorded assembly peers. This hypothesis can explain how neurons that were silenced during the jump continued to fire at the same theta phase as during control trials.

Sentinel Function of the Hippocampus. Remapping of place fields is often interpreted as an example of an operation in a

dynamically changing circuit capable of updating neuronal assembly sequences and needed to support episodic memory (87). One can assume that jumping a gap and traveling back and forth on a linear track do not need the hippocampus (although we have not tested this directly); therefore, the observed sequential changes are not relevant to behavior. Yet, in line with our findings, previous studies have already shown that hippocampal neuron firing patterns do change in response to various environmental, motivation, and motor variables, even in situations which do not require the hippocampus or its allied structures (16, 19, 20, 31, 58, 67). These studies are compatible with the hypothesis that a main function of the hippocampal system is to continuously monitor the activity of the neocortex and respond selectively to unexpected changes with appropriate selection of assembly sequences. In this sentinel function, the hippocampus perpetually compares the difference between neocortical neuronal messages and the reconstructed, predicted versions of those messages by the hippocampus (88–91). Only when the discrepancy between inputs and planned actions ("error") is large does the hippocampal circuit induce new neuronal trajectories (92).

Jump Cells. In a previous study, rats were trained to avoid an electric shock by jumping up onto the rim of a box with 33-cm walls (15). A fraction of hippocampal pyramidal neurons fired selectively during the vertical jump, and the authors suggested that these jump cells corresponded to place cells in the z (i.e., vertical) dimension. This contention is further supported by a report showing that during rearing, specific cells may become active at particular locations (93). Our findings allow for a different interpretation. First, in our experiments, the rats jumped horizontally and the elevation of their head during the flight was less than the length of the rat (Fig. 1). Second, while jumps cells were active at the same z distance from the track, their x coordinate was different, yet neurons repeatedly fired at two or three gap locations and at different rates. Third, the majority of our jump cells did not coincide with the jump itself but were active during running either before or after the gap. Finally, virtually all jump cells were active in only one travel direction. These findings eliminate the possibility that jump cells were linked strictly to motoric actions. Such direction (or "context") and location specificity suggests that jump cells are not fundamentally different from place cells but require the conjunction of a cue and an appropriate action plan.

In the few jump cells whose fields coincided with the act of jumping itself, the phase preference of spikes moved from the peak to the trough of the theta waves in just one or two cycles. This observation is consistent with the hypothesis that jump cells possess the essential features of place cells (15), since the magnitude of a full theta-cycle phase precession corresponds to the length of the place field (31), irrespective how many theta cycles it takes for the animal to traverse the field [i.e., speed (86)].

Neurons designated as jump-specific cells in our study share several features of a related (or identical) class of neurons known as landmark-vector cells (73). Landmark-vector cells fire at a similar distance and direction from a landmark and may follow the landmark when moved. Similarly, our jump cells also displayed vectorial features, since they fired selectively at the gap or at a constant distance from the gap but only during either left- or right-bound travels.

Materials and Methods

All experiments were approved by the Institutional Animal Care and Use Committee at New York University Medical Center. Details about of surgery, locations of the probes, and recordings are available in ref. 34 and in the *SI Appendix, Materials and Methods*. The takeoff during jumping was determined by the peak of the horizontal acceleration, and the landing was determined by the peak of the negative horizontal acceleration. Wait time was determined by the time spent at a speed less than 9 cm/s before the jump takeoff. Theta phase was extracted by filtering the LFP signal with a third order Butterworth bandpass filter (6 to 13 Hz), then applying the Hilbert transform to extract just the phase. Circular deviance, the circular analog of SD, was measured across each time bin. Place fields were determined as described in ref. 34. Jump-specific cells were identified by eye. Circular–linear correlations, circular deviance, and the Rayleigh test were performed using the CircStat toolbox for circular statistics (94).

Data Availability. Electrophysiological data have been deposited at https://buzsakilab.com/wp/database/ and on Zenodo (95). All study data are included in the article and/or *SI Appendix*.

ACKNOWLEDGMENTS. We thank James Knierim and members of the G.B. laboratory for feedback on the manuscript. This work was supported by NIH grants MH122391, U19 NS107616, and U19 NS104590 (G.B.). L.G. was supported by an NIH Training Grant for Computational Neuroscience (R90DA043849).

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