

Path integration: how the head direction signal maintains and corrects spatial orientation

Stephane Valerio & Jeffrey S Taube

Head-direction cells have frequently been regarded as an internal ‘compass’ that can be used for navigation, although there is little evidence showing a link between their activity and spatial behavior. In a navigational task requiring the use of internal cues to return to a home location without vision (path integration), we found a robust correlation between head-direction cell activity and the rat’s heading error in the rat’s homing behavior. We observed two different correction processes that rats used to improve performance after an error. The more frequent one consists of ‘resetting’ the cell whenever the rat returns to the home location. However, we found that when large errors occur, the head-direction system has the ability to ‘remap’ and set a new reference frame, which is then used in subsequent trials. We also offer some insight into how these two correction processes operate when rats make an error.

Path integration, first hypothesized by Darwin¹, is an animal’s ability to continuously update its position, relying on self-motion cues (vestibular, proprioceptive or motor cues)². This strategy permits a direct return to the nest (homing) at any time and has been studied in a wide variety of species from insects to humans³. Various types of spatially tuned neurons (place cells⁴, head-direction cells⁵ and grid cells⁶) have been studied extensively in the limbic system, but it remains unclear how these cells continually compute distance and direction, and direct path integration^{7,8}. Head-direction cells, which provide a constant signal of the rat’s heading^{5,9} and are modulated by self-motion cues¹⁰ are commonly viewed as the best candidate for integrating angular head motion over time (angular integrator)^{11–15}. However, to fully establish that these cells underlie the neural basis for path integration, the head-direction signal should match the heading error, reflecting the accumulation of error over time that is characteristic of path integration^{16–18}, and reflect the reorientation process when a rat uses landmarks to take a ‘fix’ after making an error^{17–19}. Here we show that the head-direction signal recorded from the anterodorsal thalamic nucleus²⁰ of rats performing a path-integration task, satisfies these two conditions. First, we observed that the shift of the head-direction cell’s preferred firing direction (PFD) correlated with the heading error of the rat. Second, we report two types of correction processes used by the rats. After large heading errors the head-direction system ‘remaps’ and takes a new bearing for the subsequent trials, but in most cases, the head-direction cells’ PFDs are ‘reset’ to stable values anchored to the refuge. This coupling of the head-direction signal and homing behavior suggests that the head-direction signal provides the directional heading component to the path integrator.

RESULTS

We trained seven Long-Evans rats to perform the food-carrying task (forage for a large food pellet and bring it back to the refuge for

consumption²¹). Once the rats were proficient at performing the task with blindfolds, we implanted the rats with recording electrodes in the anterodorsal thalamus²⁰ (Supplementary Figs. 1 and 2). We recorded a total of 27 head-direction cells (Fig. 1a) in at least one foraging trial, and for each trial we evaluated the homing accuracy of the rats. We considered a trial ‘correct’ when the rat’s return trip reached the edge of the arena within the two barriers framing the refuge (Fig. 1b,c). We counted a trial as an error when the homing trip ended outside these two barriers. Among 263 trials, we classified 135 as correct trials (51.3%), with a mean heading error of $14.78 \pm 0.08^\circ$ (mean $\pm$ s.e.m.) from a straight return to the refuge, and 128 trials as errors, with a mean heading error of $39.43 \pm 2.41^\circ$. To determine whether the overall directional firing properties were different between correct and error trials, we calculated the Rayleigh r (mean vector length) for correct (mean, 0.632 ± 0.020) versus error trials (mean, 0.626 ± 0.019); we found no significant difference between these two types of trials (Student’s t -test, $P > 0.05$).

Are the rats using path integration?

To verify that the rats were using path integration, we analyzed whether the complexity of their outward trip (searching for the pellet) affected the accuracy of their return to the refuge (homing). We compared foraging excursions resulting in correct or incorrect (error) trials. Analyses revealed significant differences in the duration of the outward trip ($t_{261} = -2.07$, $P < 0.05$) and in the distance covered by the rats ($t_{261} = -3.14$, $P < 0.01$), suggesting that the longer the outward trip was, the higher the probability was of making an error on the return trip. Subsequent analyses showed that rats were more likely to make an error if the outbound trip contained more head turns, suggesting that errors may also result from inaccurate integration of head turns ($t_{261} = -2.12$, $P < 0.05$). We also observed that correct return trips usually followed outbound paths that had a higher mean angular head velocity ($t_{261} = -2.02$, $P < 0.05$),

Department of Psychological and Brain Sciences, Dartmouth College, Hanover, New Hampshire, USA. Correspondence should be addressed to J.S.T. (jeffrey.taube@dartmouth.edu).

Received 10 May; accepted 14 August; published online 16 September 2012; doi:10.1038/nn.3215

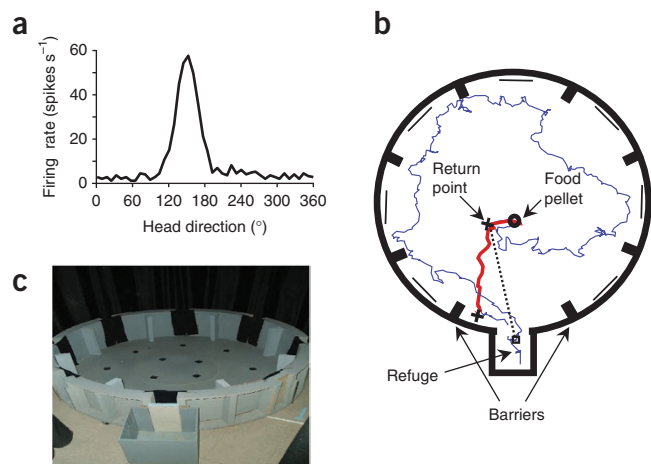


Figure 1 Head-direction cell and behavioral procedure. (a) Example of a head-direction cell tuning curve (8 min session in the cylinder, PFD = 150°). (b) Example of path taken by a rat: the foraging (outward) trip (blue) ends when the rat finds the food pellet. Heading of the return trip (red) was measured from the return point (first cross) to the edge of the arena (second cross). (c) The food-carrying task apparatus.

indicating that errors in path integration were more likely to occur after a succession of smooth large turns (Supplementary Fig. 3).

In sum, we did not find any defining characteristic of the outbound trip that predicted whether or not the rat would make a heading error (Supplementary Fig. 4). Nonetheless, the differences observed between correct and error trials confirmed that the complexity of their foraging trips influenced the rats' accuracy in their homing behavior and demonstrated that the rats were likely path-integrating when performing the task.

The shift in the cell's PFD correlates with heading error

To determine whether the amount of shift in PFDs of head-direction cells (Supplementary Fig. 5) correlated with the heading error of rats

during their homing behavior, we first calculated the amount the PFD shifted between the initial 6-min refuge session and each subsequent foraging trip. Our initial view was that for every trip, the refuge would serve as a unique and stable spatial reference point, which the rat would recognize and then use when resetting its orientation after each trial^{17,22} (Fig. 2a–e). In agreement with this hypothesis, there was a linear correlation (Pearson $r = 0.45$, $P < 0.0001$; Fig. 2f) between the amount the PFD shifted from the initial refuge session and the rat's heading error. Subsequent analyses, however, revealed a mean difference of $44.58 \pm 2.12^\circ$ between the heading error of the rats and the cells' PFD shifts, indicating that the PFD shift gave a poor prediction of the heading error.

We also found that the PFD shift in each trial compared to the initial refuge increased within a session ($F_{5,277} = 2.65$, $P < 0.05$; Fig. 2g), suggesting a gradual drift in the cell's PFD during the course of the session. Does this drift affect the rat's performance? The homing accuracy improved significantly within a session ($F_{5,257} = 2.28$, $P < 0.05$; Fig. 2h) and indicates that either there was a progressive disconnection between the head-direction signal and the rat's behavior within a session or that the resetting phenomenon is more complex than a simple return of the cells' PFDs to their initial refuge values.

Is the cell's PFD reset after each foraging trip?

To determine whether the cells' PFDs return to their initial refuge values after a foraging trip (during intertrial intervals; Fig. 2c–e), we assessed the cells' PFDs during 98 intertrial intervals. We found that the mean shift of the PFDs between the intertrial intervals and the initial refuge values was $36.76 \pm 3.41^\circ$ (mean shift after a correct trial was $36.64 \pm 4.73^\circ$ and after an error trial, $36.86 \pm 4.91^\circ$). This $\sim 36^\circ$ shift cannot be accounted for by shorter intertrial sampling times because a comparable analysis using subsampled data from the initial refuge session gave similar values (mean shift of $37.26 \pm 3.51^\circ$). If we used the cell's PFD during the intertrial interval preceding each foraging trip, we obtained a more accurate prediction of the rat's heading error (heading error versus cell PFD shift $\pm 20.48^\circ$) than if we used the initial refuge value (heading error versus cell PFD shift $\pm 43.35^\circ$, $n = 98$, paired

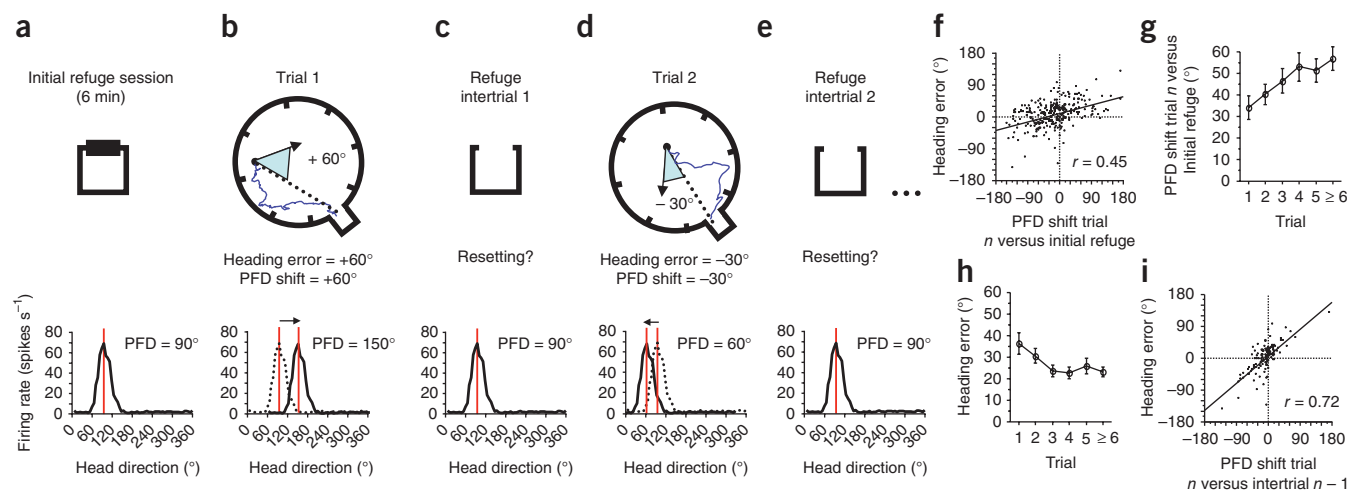


Figure 2 Hypothesized relationship between the PFD shift and the rat's heading error. (a–e) Depiction of two consecutive trials with intertrial intervals in the refuge (a, c, e) between each foraging trial (b, d). Below each figure are firing rate versus head-direction plots of a hypothetical head-direction cell, which has a PFD of 90° in the refuge. If the heading error of the rat on its return trip is $+60^\circ$ (b, blue triangle) the PFD recorded at the same time (trial 1) should show a $+60^\circ$ shift (PFD value, 150°) compared to the initial refuge value (dashed line in b, bottom). In c and e, the cell's PFD shifts back to the initial refuge value: 90° (previous theories suggested that a resetting of the cell's PFD would occur after each trip when the rat returns to the refuge). Vertical red line indicates the mean PFD of the cell. (f) Heading error as a function of the cell's PFD shift from its initial value in the refuge. (g) Mean PFD shift between trial n and the initial refuge value as a function of the trial number in the session. (h) Heading error as a function of the trial number in the session. (i) Heading error as a function of PFD shift on trial n compared to the previous intertrial interval ($n - 1$). Error bars, s.e.m.

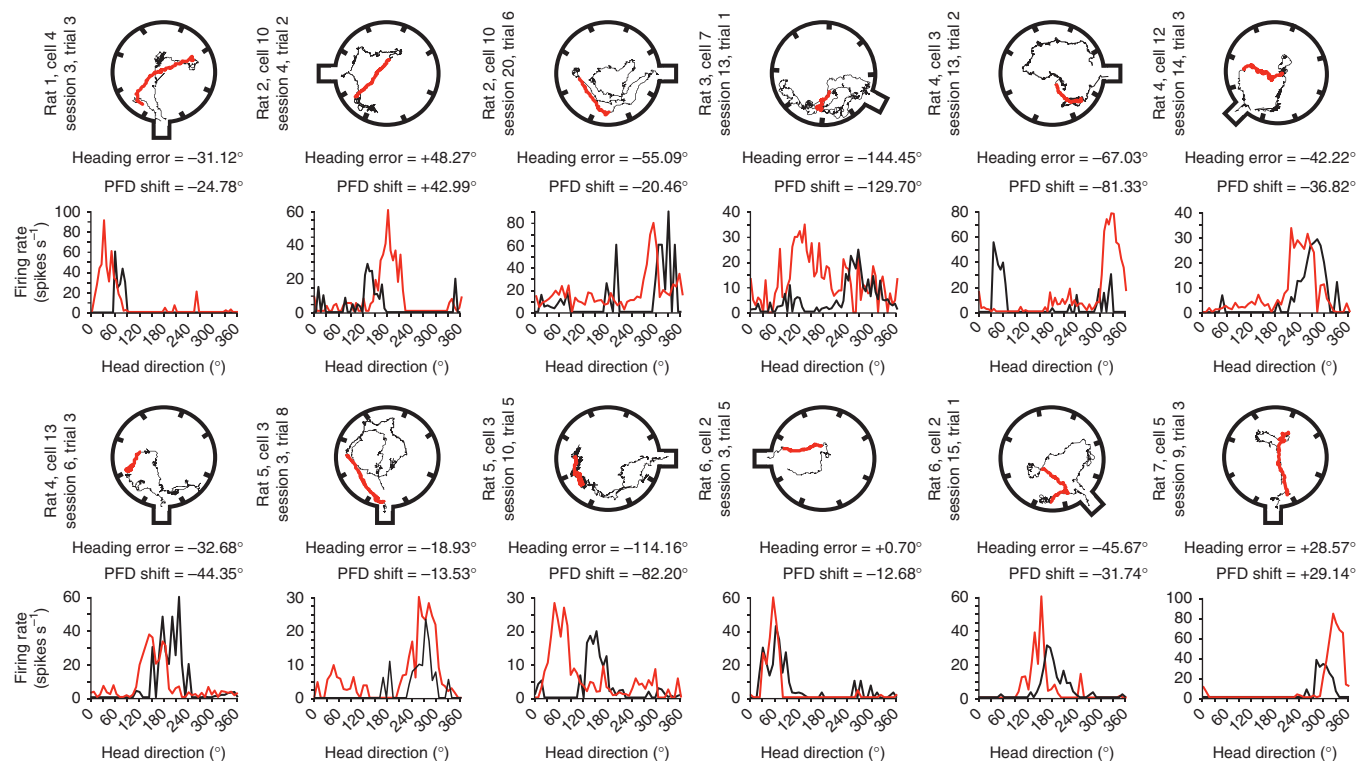


Figure 3 Examples of the foraging path with the corresponding tuning curve of a head-direction cell recorded during that trial. In each of these 12 examples, the return path of the rat (red), was used to calculate its mean heading error. The cell's tuning curve recorded during the entire foraging trip (red) is shown below the path maps and can be compared with the tuning curve for the preceding intertrial interval (black).

$t_{97} = 6.57$, $P < 0.0001$). Confirming that the intertrial interval PFD was a better reference for predicting the rat's behavior than the initial refuge value, we observed (Fig. 2i) a strong correlation between the heading error of the rat and the cell's PFD shift using the preceding intertrial interval value ($r = 0.72$, $P < 0.0001$; Fig. 3 and Supplementary Fig. 6). Supporting the view that disorientation in path-integration results from a slow accumulation of errors, we observed several examples where the cell's PFD slowly drifted away from its initial value and gave an accurate prediction of the rat's heading error (Supplementary Figs. 7–9).

In sum, we observed that using the preceding intertrial interval as a reference, as opposed to the initial refuge period, the PFD shift observed during foraging accurately reflected the heading error of the rat. But the question remains as to how the signal's preferred orientation can change from one inter-trial interval to another without altering the rat's performance (Fig. 2g,h).

Do head-direction cells remap during each refuge period?

What process accounts for the better correlation observed when using the refuge PFD from the preceding intertrial interval compared to the PFD in the original refuge (Fig. 2f,i)? Is the cell's PFD gradually drifting around the initial refuge value within a session, or is it characterized by large shifts (remapping)? The distribution of PFD shifts across successive intertrial intervals (Fig. 4a) revealed that in several refuge periods the cells' PFDs showed large shifts compared to the previous refuge period. To understand what caused these large shifts, we separated the distribution into two modes: mode 1, referred to as 'resetting', consisted of intertrial intervals in which the PFD shifted $< 35^\circ$ ($n = 50$), and mode 2, referred to as 'remapping', included the remaining cases where the PFD shifted $> 35^\circ$ ($n = 10$). We plotted the intertrial interval PFD shift against the intertrial order number in

the session (Fig. 4b). The separation of mode 1 from mode 2 for the first two trials of a session showed that large shifts only occurred in the first or second intertrial interval. Trials 3–5 only contained small PFD shifts (mode 1). Excluding the mode-2 shifts during the first two trials, there was a stable PFD shift from one refuge trial session to another (mean = 13.10°).

We found no difference between the PFD shift occurring after correct trials (mean = 13.90°) versus after error trials (mean = 12.57°) ($t_{48} = 0.56$, $P > 0.05$). These results suggest that in the majority of the intertrial intervals sampled (mode 1), the cell's PFD was stable from one refuge to another. The stability of the cell's PFD has been documented in previous studies^{20,23,24}. Thus, it is not surprising that in our experimental conditions, where rats were blindfolded²⁴ and had to rely on tactile and olfactory cues to recognize the refuge, resetting was less accurate than when rats have access to visual landmarks (mean = $4-8^\circ$; refs. 20,23). In the relatively small number of trials per session we recorded, we never observed remapping more than once per session. Notably, the mean PFD shift across intertrial intervals after remapping (mean = 21.09°) was significantly lower than the mean shift in mode-2 intertrial intervals ($\pm 68.18^\circ$, $t_{15} = 4.82$, $P < 0.001$), suggesting that after remapping the cell's PFD stabilized around the remapped refuge value.

In sum, we observed a relative stability of the cells' PFDs in the refuge, suggesting that resetting is the most common strategy used by the rats (Fig. 4). However, in some cases (mode 2) the cells' PFDs remapped to a new orientation (Fig. 4k) and thereafter became stable, such that on subsequent trials they reset to their new 'remapped values' (Fig. 4m). Although informative, the number of trials that we could analyze using this method (comparing two successive intertrial intervals) was limited to 21% of the trials because of frequent insufficient sampling in the refuge (that is, during short intertrial intervals, the rats sometimes

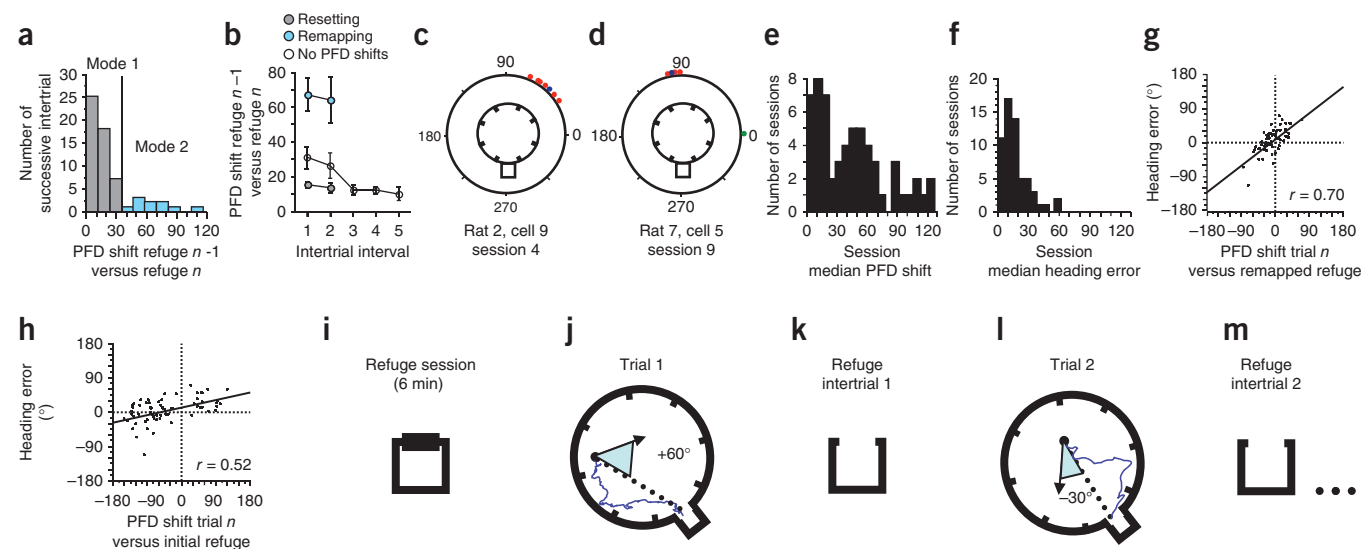


Figure 4 Variations of the PFD in the refuge. **(a)** Distribution of all PFD shifts between successive refuge periods across all trials. **(b)** PFD shift between successive refuge periods within a session. For intertrial intervals 1 and 2, mode-1 trials (resetting) and mode-2 trials (remapping; PFD shift > 35°) were separated. No mode-2 intertrial PFD shifts were observed for intertrial intervals 3–5. Error bars, s.e.m. **(c)** Circular histogram of PFD shifts for a session in which the cell's PFD remapped (shifted +51.2° from the initial refuge value; zero) after the first trial (blue dot); PFD shifts in subsequent trials are shown as red dots. **(d)** PFD shifts as in **c** for a session in which the cell's PFD initially reset to the refuge on the first trial (green dot) and then remapped after the second trial (blue dot) and then maintained this new value in all subsequent trials (red dots). **(e)** Distribution of the session median PFD shifts (from the initial refuge value). **(f)** Distribution of the session median heading errors. **(g,h)** Heading error as a function of the cell's PFD shift from its remapped refuge value **(g)** and from its initial refuge value **(h)**. **(i–m)** Schematic of the remapping hypothesis. As in **Figure 2a–e**: sequential behavioral episodes (top) and firing rate versus head-direction plots of a hypothetical head-direction cell (bottom). In the initial refuge the cell's PFD is 90° **(i)**. If the heading error of the rat on its return trip is +60° **(j, blue triangle)** the PFD recorded at the same time (trial 1) should show a +60° shift (PFD value of 150°) compared to the initial refuge value (dashed line in **j, bottom**). In **k**, the cell's PFD does not return to its initial refuge value, but remaps to a random PFD (for example, 270°). In **l**, the remapped PFD is the new reference and the amount the PFD will shift during trial 2 is compared to that remapped value (–30°). Thus, the amount the PFD shifts from the new remapped PFD value (–30° to 240°) predicts the heading error (–30°) of the rat. In the subsequent intertrial interval, the PFD resets to the value established after remapping (270°) **(m)**. The data partially confirmed this remapping hypothesis. One important difference was that the value of the remapped PFD is not random such as that shown in **k**, but was influenced by the shift observed in the previous foraging trip. In this case **(j)**, the remapped value would be ~150°.

only turned in one direction, and sampling was confined to ~180°. We therefore sought a second method to identify trials in which remapping occurred.

Identification of remapped sessions

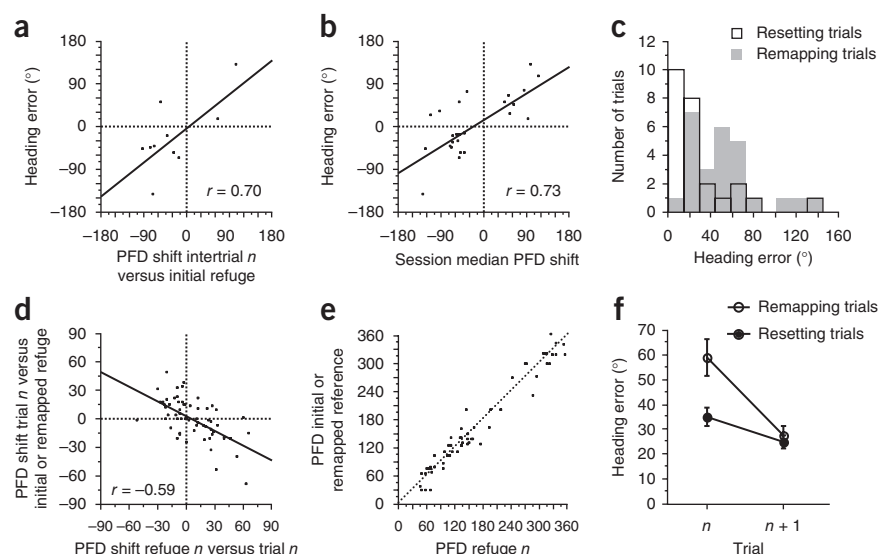
If remapping only occurred once during a session (**Fig. 4b**), then we should be able to identify the sessions in which it occurred. Using the amount the PFD shifted during foraging trips (compared to the initial refuge value), we sought instances in which there was a consistent bias in the distribution of PFD shifts across session trials. For example, if a cell's PFD remapped and shifted +70° compared to its initial refuge value after the first trial, then we should observe a consistent +70° shift in the cell's PFD for all foraging trips and intertrial intervals after remapping (**Fig. 4c,d**: +50° and +90°, respectively). To identify sessions showing this pattern, we examined the median PFD shift (to avoid the influence of outliers) for each session containing at least two trials ($n = 51$; see **Supplementary Fig. 10** for results using the session mean PFD shift). The distribution of the session median shifts (**Fig. 4e**) revealed a clear bimodality in the values (mode 1 around 0° and

mode 2 around 50°). Consistent with the results in **Figure 4a**, this pattern indicated that in mode-1 sessions (median PFD shift < 35°) the cell's PFD drifted around the initial refuge value, but that in mode-2 sessions (median PFD shift > 35°) the cell's PFD consistently shifted by about ±50° (for example, some PFDs shifted ≥ 90°). For comparison, the distribution of the session median heading errors (**Fig. 4f**) showed that behavior could not account for these large and consistent shifts.

When does remapping occur?

For each session, we identified the moment when the PFD shifted using the session median shift and searched for the trial after which the PFD shift occurred consistently. We then used the subsequent intertrial value (remapped refuge value) to test whether the remapped value was used as the new stable reference in the subsequent trials. For trials after remapping, we obtained a much better correlation between PFD shift and heading error using the remapped refuge value as a reference (**Fig. 4g**; $n = 66$, $r = 0.70$, $P < 0.0001$) than using the initial refuge value (**Fig. 4h**; $r = 0.52$). This result confirmed that after remapping occurs, a new 'zero' point (the remapped refuge value) was

Figure 5 The correction process. (a) Heading error as a function of the cell's PFD shift between the current intertrial interval and the initial refuge (mode-2 intertrials). (b) Heading error observed in remapping trials, as a function of the session median PFD shift compared to its initial refuge value. (c) Distribution of heading errors for resetting and remapping trials. (d) PFD shift in trial n (compared to initial or remapped refuge value) as a function of the PFD shift between trial n and the following intertrial interval (refuge n). (e) PFD recorded during the inter-trial interval n compared to the initial or remapped refuge value. (f) Comparison of heading errors made by the rats before (trial n) and after (trial $n + 1$) remapping and resetting. Error bars, s.e.m.



set for the remainder of the session (Fig. 4m). Together with the analyses of intertrial intervals above, these remapping trials explain the apparent discrepancy we observed between a good correlation when using the preceding intertrial interval as reference (Fig. 2i) versus a smaller correlation when using the initial refuge value (Fig. 2f).

What determines the remapped refuge value?

Did it result from a random shift in the PFD (Fig. 4k) or was there a reason that the cell remapped to a particular value? We addressed this issue using our two indicators of remapping: (i) the PFD shift between two successive intertrial intervals, when available ($n = 10$), and (ii) the session median shift ($n = 26$) to identify remapping trials (Fig. 5). Both measures revealed a significant correlation with the rat's heading error in the trial preceding remapping (Fig. 5a, $r = 0.70$, $P < 0.05$; Fig. 5b, $r = 0.73$, $P < 0.0001$; see Supplementary Fig. 11 for results using the session mean shift). These results indicate that the remapped PFD was not arbitrary but instead was influenced by the rat's behavior in the trial preceding remapping. In essence, this analysis suggests that the shifted PFD value which just lead to an error on trial n becomes the new reference point for the cell. This new PFD value is then maintained during the subsequent intertrial interval in the refuge and remains the new referenced value for the next trial ($n + 1$). An example of remapping is as follows: if a cell is initially tuned to 100° in the refuge and drifts 60° counterclockwise during the foraging trip, then the rat's return path will have a 60° counterclockwise error (PFD = 160°), but the cell's PFD in the subsequent intertrial interval will remain about 160° (Fig. 6a,b and Supplementary Fig. 12).

Beyond the simple description of this phenomenon, two questions remain about remapping. First, what causes remapping? Does remapping follow large heading errors? Second, why does the cell remap to the PFD observed in the preceding foraging trip? Why would a PFD that had just lead to an error be kept as the new reference for subsequent foraging trips? We observed (Fig. 5c) that remapping usually followed larger heading errors (remapping trials, $50.12 \pm 6.58^\circ$; resetting trials, $29.67 \pm 6.58^\circ$, $t_{49} = 2.27$, $P < 0.05$). However, we could make no systematic prediction about whether the rat would remap or reset solely based on the rat's behavior. Notably, most of the remapped values (PFD shifts; Fig. 4e) were situated around $\pm 50^\circ$, which is approximately the space that separates two consecutive doors in the arena (45°), suggesting that the structure of the apparatus influenced remapping.

In sum, our data suggest that the remapped PFD resulted from the state of the head-direction network in the preceding foraging trip (which in most cases lead to a large heading error) and from the geometry of the apparatus. The rat could reset its orientation upon return to the refuge, but apparently it ignored the cues in the refuge and resetting did not occur.

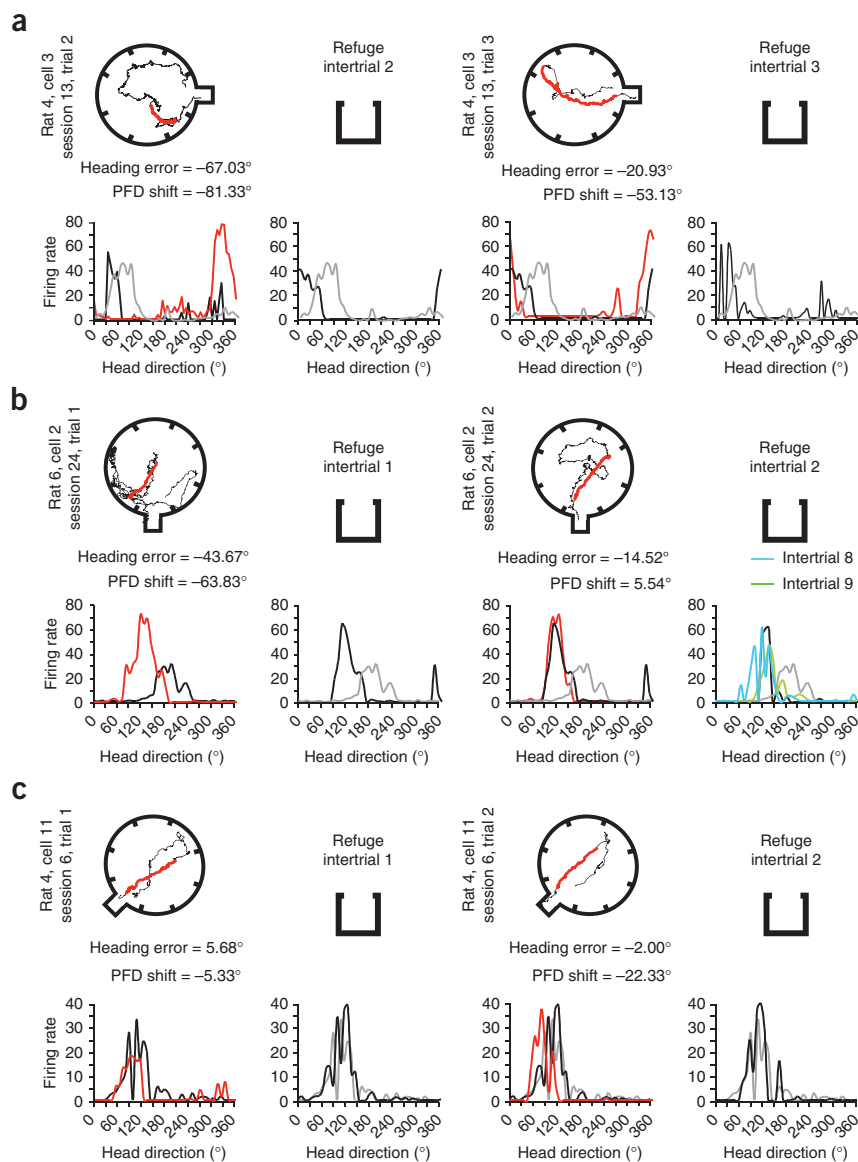
Resetting trials

Our analyses of successive refuge periods suggested that for most trials, the cells' PFDs remained stable from one refuge period to another (Fig. 4a). Even in sessions when remapping occurred, the cells' PFDs tended to reset to the new reference point for the remainder of the session (Fig. 4h). Another way to verify that resetting occurred is to determine whether a shift in the PFD that occurred during a foraging trip was corrected when the rat reached the refuge. For example, if the PFD shifted $+60^\circ$ during an outbound trip (PFD trial n – PFD refuge $n - 1$), would the PFD shift -60° in the subsequent intertrial interval in the refuge (PFD refuge n – PFD trial n)? We observed (Fig. 5d) that the amount the PFD shifted after a foraging trip negatively correlated with the amount of shift observed during that same trial (compared to the initial or remapped value; $n = 61$, $r = -0.59$, $P < 0.0001$). Thus, after each foraging trip for non-remapping trials, the cell's PFD returned either to a stable refuge value or to the remapped refuge value. Another way of representing this resetting phenomenon (Fig. 5e) showed that the PFD in refuge n was very close to the initial (mode-1 trials) or remapped (mode-2 trials) refuge value (mean difference = $\pm 17.31^\circ$; Fig. 6c and Supplementary Fig. 13).

Behavioral benefit of remapping and resetting

We compared the effect of remapping and resetting on the rat's performance for the subsequent foraging trip after an error (Fig. 5f). A two-way analysis of variance (ANOVA) with repeated measures (trial $\times$ process type) revealed a global effect for trial ($F_{1,76} = 24.34$, $P < 0.0001$), process type ($F_{1,76} = 7.59$, $P < 0.01$) and an interaction between the two factors ($F_{1,76} = 6.66$, $P < 0.05$). But when we separated the analyses based on remapping versus resetting, we observed an improvement in rats' homing behavior after both remapping ($F_{1,19} = 18.83$, $P < 0.001$) and resetting ($F_{1,57} = 5.41$, $P < 0.05$). Notably, performance reached a comparable level after both processes, with the mean heading errors after remapping and resetting of 27.72° and

Figure 6 Examples of remapping and resetting sessions. (**a–c**) Each pair of rows depicts successive behavioral trials in a single session, along with the responses of a head-direction cell recorded during the trial. As in **Figure 4**, columns 1 and 3 are examples of foraging paths (outbound trips, black; return trips, red) above the firing rate versus head-direction tuning curves recorded during the same trial (red) and the cell's response in the preceding refuge episode (black). Columns 2 and 4 show the cell's tuning curve during the intertrial interval after the foraging trial (black). To illustrate the changes of the cell's PFD in the refuge after remapping, the head-direction cell's response during the initial refuge period is shown for all plots as the gray trace. Column 4 shows the cell's response during the subsequent intertrial interval(s) in the refuge after the foraging trial shown in column 3 (black). In **a**, remapping occurred during trial 2. After shifting -81.33° during the second trial (compare red and black traces, column 1), the cell's PFD in the refuge during the next inter-trial interval (column 2) did not return to its initial refuge value (compare black and gray traces), but remained close to the value it had during the foraging trip (trial 2). In the next foraging trip (column 3) the shift of the cell's PFD to the remapped refuge value (black trace) gives a better prediction of behavior than the PFD shift calculated using the initial refuge value (gray trace). In the intertrial interval after trial 3, the cell's PFD returned to this remapped refuge value (column 4). In **b**, remapping occurred in trial 1 (column 1). In the next intertrial interval (column 2), instead of returning to the initial refuge value (gray trace), the cell's PFD (black trace) was closely aligned to the value recorded in the previous foraging trip (trial 1). As in **a**, after remapping, the cell's PFD reset to this new value in the refuge after subsequent foraging trials (column 4 shows intertrial intervals: two in black, eight in blue and nine in green) and shows that the cell's PFD maintained this new refuge value for the remainder of the session. In **b**, the initial refuge period is the same as the previous intertrial interval as column 1 depicts the first trial. In **c**, an example of resetting, the cell's PFD remained stable across all intertrial refuge periods.



25.15° , respectively ($t_{76} = 0.52$, $P > 0.05$). For comparison, recall that the mean heading errors before remapping and resetting were 50.12° and 29.67° , respectively. In sum, these results indicate that both processes corrected the rats' orientation after an error and improved rats' performance on the next trial.

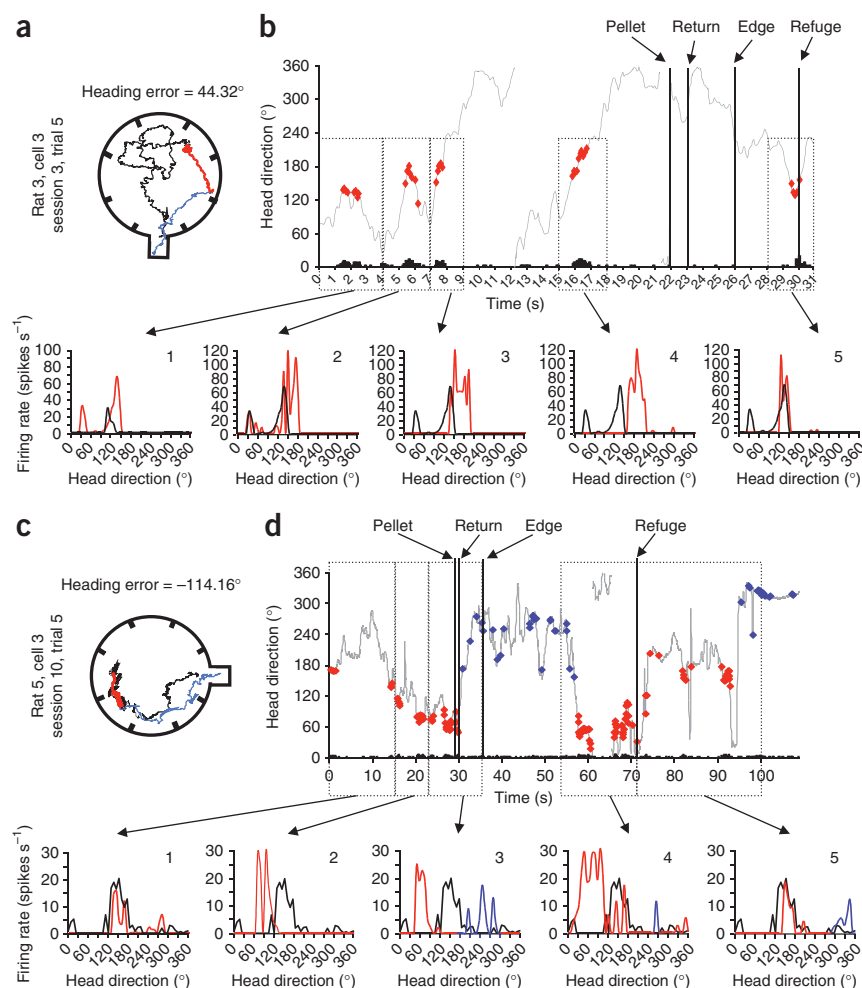
Error trials and cell resetting

In theory, resetting could result from two different strategies: (i) the cell's PFD could reset upon return to the refuge, where the rats can use the features of this familiar location to correct its orientation, or (ii) as the rats found the refuge despite their errors, it is possible that reorientation occurs 'on-line' on their way back to the refuge. Indeed, if the head-direction signal codes for the rat's behavior, then the active reorientation of the rat after reaching the apparatus edge and perceiving that it made an error should be coupled with a coherent shift in the cell's PFD.

When rats perceived they made an error by reaching the apparatus periphery without attaining the refuge, they quickly turned and changed their trajectory. These turns were sometimes in the correct direction of the refuge (**Fig. 3** row 1, examples 1 and 3) but sometimes were in the

wrong direction (**Fig. 3**, row 1, examples 2 and 4). (Note that turning in the wrong direction provides evidence that the rats were unlikely to be using olfactory cues emanating from the refuge to guide their return.) Although there were a limited number of trials to analyze in which the rat made a sizeable heading error and in which we also had sufficient sampling of different head directions during these short correction episodes, there were three trials that we could analyze sufficiently. We analyzed the moment-to-moment changes of the cell's PFD (**Figs. 7** and **8**) during trials that the rat made an error and had to change course on its return trip. In addition to the gradual drifting of the cell's PFD, which is apparent in the results shown in **Figures 7** and **8**, these trials suggest that the rat can use both strategies. In the first example (**Fig. 7a,b**), the cell's PFD appeared to reset before the rat reached the refuge, whereas in the second example (**Fig. 7c,d**), the shifted PFD was maintained throughout the course correction and was reset only when the rat entered the refuge upon its return. Consistent with the second hypothesis, in a remapping trial (**Fig. 8**), the cell's PFD appeared to change with the rat's orientation (the PFD shift reflected the rat's reorientation in the wrong direction) until the cell's PFD remained stable around a new value. This new PFD value was not reset in the refuge and remained the 'reference' for subsequent trials.

Figure 7 Moment-to-moment changes of the cell's PFD in two examples of resetting trials. (a–d) Foraging path separated into outbound (black), return from the pellet to the edge of the arena (red) and correction from the apparatus edge to the refuge (blue) paths (a,c). Moment-to-moment head direction (gray trace $\times$ time) of the rat coupled with cell activity (black bars (b,d). Red dots indicate head direction and time at which the cell's firing rate reached 50% of its maximum rate. Successive sampling episodes were isolated (insets), and tuning curves for each episode (red trace) were compared with a reference (black trace). In inset 1 in b, the cell's PFD at the beginning of the trial is compared to the only intertrial value that could be recorded in that session (intertrial value 2). In 2–4 the cell's PFD is compared to the PFD recorded at the beginning of the trial, and shows a slow drift counterclockwise while the rat foraged for food. In 5, the cell's PFD is shown while the rat was correcting its orientation after reaching the wall of the arena (blue path in a) and presumably first perceived that it made an error. At that time, the PFD was reset to its value at the beginning of the trip (black). The cell did not fire when the rat was facing 170° (29 s), which suggests that it was reset before reaching the refuge. In insets 1–3 in d, the cell's PFD drifts clockwise away from the preceding intertrial value (black curve). In 4, however the rat was correcting its error on its return to the refuge, the cell did not reset until the rat entered the refuge (5). In this example, a second cell is visible with a PFD around 260° (dark blue dots). These two cells shifted in register: when the rat reached the refuge, the first cell reset (red dots) and the PFD of the second cell (dark blue dots) shifted approximately the same amount.



DISCUSSION

Here we demonstrated that in a path-integration task, the head-direction cells' PFD shifts correlated with the rat's behavioral trajectory. The apparent disconnection that we occasionally observed between the cells' PFD shifts and behavior resulted from a remapping phenomenon, which appears to be one way the rats reorient themselves after making an error. Our data provide evidence for two correction processes that rats use to avoid accumulation of errors and maintain accurate orientation: remapping and resetting. Most of the time the cells' PFDs reset, either to the initial or to the remapped refuge value, when the rat returned to the refuge, but we also observed that after large heading errors, the head-direction cells shifted to a new PFD that became the new 'zero value' for the subsequent foraging trips.

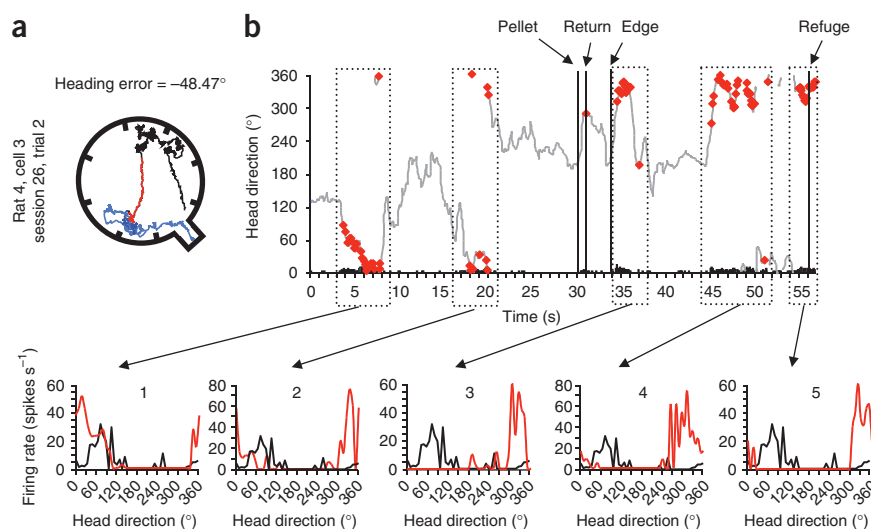
What differentiates remapping from resetting? If resetting is the return of the cells' PFDs to their initial or remapped values, is it possible that remapping is simply the failure of the resetting process and we artificially separated them into two different processes? We feel this possibility is unlikely for two reasons. First, remapping was beneficial to the rats' performance, which suggests that remapping was more of an active correction process than an uncontrolled shift of the cell. Second, we observed that remapping was not a random shift but rather the rat's new reference was influenced by two elements: the PFD observed in the preceding foraging trip and the geometric structure of the apparatus. Previous studies have shown that the geometry of the environment can drive both the head-direction signal^{25,26} and the rats' spatial behavior²⁷. Our finding suggests that the rats reoriented

using external landmarks during remapping trials to establish a new bearing for the subsequent trials.

This sequential use of internal and external information is necessary to perform path integration, and it has been observed in several other species^{17,18,28}. The use of fixes has usually been referred to as resetting²⁹ and viewed as a process by which the path integrator adjusts to the perceived environment. Notably, a resetting phenomenon comparable to the one reported here, has been observed in place cells^{30,31}, and a recent report on grid cells could also be interpreted as containing home-based resetting³². Additionally, in place cells, two types of adjustment have been reported when idiothetic and external cues gave conflicting information³⁰: in cases of small mismatches between idiothetic and external cues, "the internal representation caught up with the real world coordinate." But in addition to this smooth correction process, in cases of large mismatches the authors observed³⁰ "abrupt shifts in the hippocampal representation." These observations clearly parallel the two correction processes we describe in head-direction cells. The observed remapping of the head-direction cell can be viewed as a result of a failure to reach the refuge using idiothetic cues. Indeed, it appears that after large errors and experiencing 'misorientation', the rats established and maintained a new reference point: they set a new 'zero' that anchored their current PFDs to the available landmarks.

The fact that the rats use external information is not what differentiates remapping from resetting. In both cases, the rats use a fixed point to anchor their spatial representation: the refuge in the case of resetting versus some tactile or geometric features of the apparatus in the case of

Figure 8 Moment-to-moment changes of the cell's PFD in an example of a remapping trial. (a) As in **Figure 7**, outbound (black), return (red) and correction (blue) paths of the rat. (b) As in **Figure 7**, moment-to-moment head-direction of the rat coupled with the cell's activity (firing rate $\times$ head-direction $\times$ time; top) and tuning curves for the successively sampled episodes (bottom). Red dots indicate firing rates $>50\%$ of maximum firing rate. Insets 1 and 2 show the cell's PFD drifting clockwise away from the value recorded in the preceding intertrial value (black). The cell's PFD after the rat reached the edge of the arena is shown in inset 3; in this trial and visible in a, the rat went in the wrong direction (southwest door) while trying to find the refuge (located in the southeast). The cell's PFD recorded at the same time (3) reflected this error in the reorientation process by drifting away from its original value on the outbound foraging trip. Therefore, the reorientation of the rat appeared to be coupled with a predictable change in the cell's PFD. Insets 4 and 5 show a representative example of the remapping process, in which the cell's PFD was not reset and remained at the shifted value around 325° even after the rat returned to the refuge. In this case, this value (325°) will be the new reference for subsequent trials.



remapping. It is important to note that even if the head-direction signal is strongly influenced by self-motion information, it does not code for an egocentric representation of space but rather for an allocentric one³³. Therefore, what characterizes remapping is the fact that the allocentric-based representation of the initial refuge is abandoned and that the rats establish a new reference, a new zero, which they maintain in the refuge and during subsequent trials. A key characteristic of remapping is the absence of resetting when the rats reach the refuge, suggesting that the spatial representation based on the initial refuge has been discarded and replaced by a new reference frame. Our data suggest that in this highly familiar environment, this new reference frame is influenced by the geometry of the apparatus and, in most occasions, consists of a simple rotation of the initial reference frame by 45° (one doorway). Similar to our results, this change in the reference frame could explain the apparent disconnection between behavior and the head-direction signal observed in previous studies^{13,26}.

How is the remapping process we observed related to the remapping phenomenon usually evoked for hippocampal place cells? Three types of remapping have been observed in place cells: (i) rotational remapping, when place cells keep their spatial characteristics and relationships but all the place fields rotate together, (ii) global remapping, which consists of a complete reorganization of the place code after substantial changes in the environment³⁴ or in task demand³⁵, and (iii) rate remapping, when place cells maintain their place field but show firing-rate changes based on subtle changes in a given environment³⁶. A tight relationship between head-direction cells and place cells has been observed in two studies where they had been recorded simultaneously^{37,38}. These studies show that when idiothetic cues are in conflict with visual cues, place and head-direction cells show rotational remapping until the conflicting information reaches a difference of 45° , at which point global remapping occurs. These results parallel our observations and suggest that place cells would show rotational remapping in our resetting trials but would show global remapping when we observed large PFD shifts (remapped trials).

Our results showing that rats used two different correction processes, presumably depending on the size of the most recent heading error, suggests that the head-direction system receives information feedback about the outcome of its current performance. Many studies exploring the neural processes involved in reward, particularly in the

mesolimbic dopaminergic system³⁹, have shown that the dopamine signal encodes for 'prediction error'⁴⁰. Is this signal somehow conveyed to the head-direction system? One possibility is that this signal is conveyed via the lateral habenula, which projects to the dorsal tegmental nucleus, a key point in the head-direction circuitry^{41,42}. Unexpected omission of an anticipated reward results in a transient cessation of dopaminergic activity in the ventral tegmental area (VTA)⁴³, which is mediated by an indirect inhibitory projection from the lateral habenula to the VTA⁴⁴. In the food-carrying task, an error on the return trip could be viewed as an omitted reward, which would require a reconsideration of the rat's orientation. Alternatively, the error signal could be conveyed from the anterior cingulate cortex^{45,46} to the postsubiculum via the retrosplenial cortex. In agreement with this hypothesis, some studies have shown that lesions of retrosplenial cortex induce impairments in the food-carrying task⁴⁷. Another possibility is that given the tight relationship between the hippocampus and the head-direction network³⁷, remapping in the head-direction network results from remapping in the hippocampus. Indeed, the locus coeruleus, which is also activated by unexpected outcomes³⁹, has been hypothesized to initiate remapping in the hippocampus⁴⁸. Understanding what causes this switch in the reference frame, which permits reorientation, as well as the brain areas involved, is critical for discerning how navigational errors are corrected.

METHODS

Methods and any associated references are available in the online version of the paper.

Note: Supplementary information is available in the online version of the paper.

ACKNOWLEDGMENTS

We thank R. Leaton, P. Trifileff and E. Lesburgueres for their valuable comments on the manuscript. These experiments were supported by US National Institutes of Health grants DC009318 and NS053907.

AUTHOR CONTRIBUTIONS

S.V. and J.S.T. conceived and designed the study, discussed the findings, and wrote the manuscript. S.V. performed the experiments and analyzed the results.

COMPETING FINANCIAL INTERESTS

The authors declare no competing financial interests.

Published online at <http://www.nature.com/dofinder/10.1038/nn.3215>.

Reprints and permissions information is available online at <http://www.nature.com/reprints/index.html>.

1. Darwin, C. Origin of certain instincts. *Nature* **VII**, 417–418 (1873).
2. Mittelstaedt, M.L. & Mittelstaedt, H. Homing by path integration in a mammal. *Naturwissenschaften* **67**, 566–567 (1980).
3. Etienne, A.S. & Jeffery, K.J. Path integration in mammals. *Hippocampus* **14**, 180–192 (2004).
4. O'Keefe, J. & Dostrovsky, J. The hippocampus as a spatial map. Preliminary evidence from unit activity in the freely-moving rat. *Brain Res.* **34**, 171–175 (1971).
5. Taube, J.S., Muller, R.U. & Ranck, J.B. Head-direction cells recorded from the postsubiculum of freely-moving rats. I. Description and quantitative analysis. *J. Neurosci.* **10**, 420–435 (1990).
6. Haftting, T., Fyhn, M., Molden, S., Moser, M.B. & Moser, E.I. Microstructure of a spatial map in the entorhinal cortex. *Nature* **436**, 801–806 (2005).
7. Muir, G.M. & Taube, J.S. The neural correlates of navigation: do head direction and place cells guide spatial behavior? *Behav. Cogn. Neurosci. Rev.* **1**, 297–317 (2002).
8. Save, E. & Poucet, B. Do place cells guide spatial behaviour? in *Hippocampal Place fields, Relevance to learning and memory* (ed., Mizumori, S.J.) 138–149 (Oxford University Press, New York, 2008).
9. Wiener, S.I. & Taube, J.S. (eds.). *Head Direction Cells and the Neural Mechanisms of Spatial Orientation* (MIT Press, Cambridge, Massachusetts, USA, 2005).
10. Taube, J.S. The head direction signal: origins and sensory-motor integration. *Annu. Rev. Neurosci.* **30**, 181–207 (2007).
11. McNaughton, B.L., Chen, L.L. & Markus, E.J. "Dead reckoning", landmark learning, and the sense of direction: a neurophysiological and computational hypothesis. *J. Cogn. Neurosci.* **3**, 190–202 (1991).
12. Taube, J.S. Head direction cells and the neurophysiological basis for a sense of direction. *Prog. Neurobiol.* **55**, 225–256 (1998).
13. van der Meer, M.A., Richmond, Z., Braga, R.M., Wood, E.R. & Dudchenko, P.A. Evidence for the use of an internal sense of direction in homing. *Behav. Neurosci.* **124**, 164–169 (2010).
14. McNaughton, B.L., Battaglia, F.P., Jensen, O., Moser, E.I. & Moser, M.B. Path integration and the neural basis of the 'cognitive map'. *Nat. Rev. Neurosci.* **7**, 663–678 (2006).
15. Hasselmo, M.E. Grid cell mechanisms and function: contributions of entorhinal persistent spiking and phase resetting. *Hippocampus* **18**, 1213–1229 (2008).
16. Gallistel, C.R. *The organization of learning* (MIT Press, Cambridge, Massachusetts, USA, 1990).
17. Souman, J.L., Frissen, I., Sreenivasa, M.N. & Ernst, M.O. Walking straight into circles. *Curr. Biol.* **19**, 1538–1542 (2009).
18. Etienne, A.S., Maurer, R., Boulens, V., Levy, A. & Rowe, T. Resetting the path integrator: a basic condition for route-based navigation. *J. Exp. Biol.* **207**, 1491–1508 (2004).
19. Knaden, M. & Wehner, R. Ant navigation: resetting the path integrator. *J. Exp. Biol.* **209**, 26–31 (2006).
20. Taube, J.S. Head direction cells recorded in the anterior thalamic nuclei of freely moving rats. *J. Neurosci.* **15**, 70–86 (1995).
21. Whishaw, I.Q., Coles, B.L. & Bellerive, C.H. Food carrying: a new method for naturalistic studies of spontaneous and forced alternation. *J. Neurosci. Methods* **61**, 139–143 (1995).
22. Etienne, A.S., Boulens, V., Maurer, R., Rowe, T. & Siegrist, C. A brief view of known landmarks reorientates path integration in hamsters. *Naturwissenschaften* **87**, 494–498 (2000).
23. Goodridge, J.P. & Taube, J.S. Preferential use of the landmark navigational system by head direction cells in rats. *Behav. Neurosci.* **109**, 49–61 (1995).
24. Goodridge, J.P., Dudchenko, P.A., Worboys, K.A., Golob, E.J. & Taube, J.S. Cue control and head direction cells. *Behav. Neurosci.* **112**, 749–761 (1998).
25. Clark, B.J., Harris, M.J. & Taube, J.S. Control of anterodorsal thalamic head direction cells by environmental boundaries: Comparison with conflicting distal landmarks. *Hippocampus* **22**, 172–187 (2012).
26. Golob, E.J., Stackman, R.W., Wong, A.C. & Taube, J.S. On the behavioral significance of head direction cells: neural and behavioral dynamics during spatial memory tasks. *Behav. Neurosci.* **115**, 285–304 (2001).
27. Cheng, K. Whither geometry? Troubles of the geometric module. *Trends Cogn. Sci.* **12**, 355–361 (2008).
28. Steck, K., Hansson, B.S. & Knaden, M. Desert ants benefit from combining visual and olfactory landmarks. *J. Exp. Biol.* **214**, 1307–1312 (2011).
29. Collett, T.S. & Graham, P. Animal navigation: path integration, visual landmarks and cognitive maps. *Curr. Biol.* **14**, R475–R477 (2004).
30. Gothard, K.M., Skaggs, W.E. & McNaughton, B.L. Dynamics of mismatch correction in the hippocampal ensemble code for space: interaction between path integration and environmental cues. *J. Neurosci.* **16**, 8027–8040 (1996).
31. Gothard, K.M., Skaggs, W.E., Moore, K.M. & McNaughton, B.L. Binding of hippocampal CA1 neural activity to multiple reference frames in a landmark-based navigation task. *J. Neurosci.* **16**, 823–835 (1996).
32. Derdikman, D. et al. Fragmentation of grid cell maps in a multicompartment environment. *Nat. Neurosci.* **12**, 1325–1332 (2009).
33. Taube, J.S., Muller, R.U. & Ranck, J.B. Jr. Head-direction cells recorded from the postsubiculum in freely moving rats. II. Effects of environmental manipulations. *J. Neurosci.* **10**, 436–447 (1990).
34. Muller, R.U. & Kubie, J.L. The effects of changes in the environment on the spatial firing of hippocampal complex-spike cells. *J. Neurosci.* **7**, 1951–1968 (1987).
35. Markus, E.J. et al. Interactions between location and task affect the spatial and directional firing of hippocampal neurons. *J. Neurosci.* **15**, 7079–7094 (1995).
36. Leutgeb, S. et al. Independent codes for spatial and episodic memory in hippocampal neuronal ensembles. *Science* **309**, 619–623 (2005).
37. Knierim, J.J., Kudrimoti, H.S. & McNaughton, B.L. Interactions between idiothetic cues and external landmarks in the control of place cells and head direction cells. *J. Neurophysiol.* **80**, 425–446 (1998).
38. Knierim, J.J., Kudrimoti, H.S. & McNaughton, B.L. Place cells, head direction cells, and the learning of landmark stability. *J. Neurosci.* **15**, 1648–1659 (1995).
39. Schultz, W. & Dickinson, A. Neuronal coding of prediction errors. *Annu. Rev. Neurosci.* **23**, 473–500 (2000).
40. Fiorillo, C.D., Tobler, P.N. & Schultz, W. Discrete coding of reward probability and uncertainty by dopamine neurons. *Science* **299**, 1898–1902 (2003).
41. Sharp, P.E., Tinkelman, A. & Cho, J. Angular velocity and head direction signals recorded from the dorsal tegmental nucleus of guinea in the rat: implications for path integration in the head direction cell circuit. *Behav. Neurosci.* **115**, 571–588 (2001).
42. Bassett, J.P. & Taube, J.S. Neural correlates for angular head velocity in the rat dorsal tegmental nucleus. *J. Neurosci.* **21**, 5740–5751 (2001).
43. Matsumoto, M. & Hikosaka, O. Lateral habenula as a source of negative reward signals in dopamine neurons. *Nature* **447**, 1111–1115 (2007).
44. Balcita-Pedicino, J.J., Omelchenko, N., Bell, R. & Sesack, S.R. The inhibitory influence of the lateral habenula on midbrain dopamine cells: ultrastructural evidence for indirect mediation via the rostromedial mesopontine tegmental nucleus. *J. Comp. Neurol.* **519**, 1143–1164 (2011).
45. Ito, S., Stuphorn, V., Brown, J.W. & Schall, J.D. Performance monitoring by the anterior cingulate cortex during saccade countermanding. *Science* **302**, 120–122 (2003).
46. Michelet, T., Bioulac, B., Guehl, D., Goillandeau, M. & Burbaud, P. Single medial prefrontal neurons cope with error. *PLoS ONE* **4**, e6240 (2009).
47. Whishaw, I.Q., Maaswinkel, H., Gonzalez, C.L. & Kolb, B. Deficits in allothetic and idiothetic spatial behavior in rats with posterior cingulate cortex lesions. *Behav. Brain Res.* **118**, 67–76 (2001).
48. Bouret, S. & Sara, S.J. Network reset: a simplified overarching theory of locus coeruleus noradrenaline function. *Trends Neurosci.* **28**, 574–582 (2005).

ONLINE METHODS

Subjects and apparatus. Seven food-deprived Long-Evans female rats (250–400 g) were placed on a food-restricted diet (10–15 g/d) and trained in a food-carrying task, which involves searching for food in an open field and returning to a refuge to consume it²¹. The food-carrying task apparatus consisted of a large gray, circular (1.83 m diameter) open field with 12 black food cups placed uniformly around the surface (Fig. 1c). The open field was surrounded by a wall (38 cm high) containing eight uniformly distributed doorways, each separated by 45° from the ones adjacent to it. There was a refuge (29 × 30 cm) behind one doorway. The other seven doorways served as false refuges and were closed. All screening for head-direction cells took place in a gray cylinder (76 cm diameter, 51 cm high) containing a white cue card covering 110° of arc. All procedures involving the rats were performed in compliance with institutional standards as set forth by the National Institutes of Health Guidelines for the Care and Use of Laboratory Animals.

Behavioral procedure. Before being implanted with an electrode, the rats were trained in the food-carrying task. This task relies on the rat's proclivity to carry large food pellets back to a shelter for eating, rather than consuming them out in the open field²¹. The aim of this task is to test how accurately rats can return to the refuge (homing) after a random search for a food pellet when they are deprived of visual cues and must rely on self-motion cues and path integration for their homeward trip. Detailed training procedures have been described previously⁴⁹. There were three training stages. In the first stage (3 d), rats were acclimated to the apparatus by placing them in the refuge for 10 min with the door closed and providing them with ten food pellets. In the second stage, rats were trained in the task with visual cues available. In the third stage, rats were blindfolded, and they had to complete the task without the use of visual cues. A typical session started with the rat placed in the refuge. Then the door was opened, and the rat eventually left the refuge and went out into the open field to forage for large sugar pellets (750 mg), which were randomly placed in a food cup on the arena floor. Once the rat found a food pellet, the rat had to bring it back to the refuge to consume it there. To help motivate the rats that the only safe way to consume the food was to bring it back to the refuge, the experimenter took the food pellet away from the rat whenever the rat tried to consume it in the arena. The homing accuracy of the rats was not assessed during the training phase. The behavioral criterion to go to the third stage was the completion of four trials (four pellets brought back to the refuge) in <10 min for two consecutive training sessions. The rats required 12.57 ± 0.68 sessions to complete this second training stage (range, 9 to 14 sessions). At the end of the first training stage, the rats were habituated to wear the blindfold: 20 min/d in their home cage for at least 1 week. Once habituated to the blindfold and able to complete four trials in <10 min with the visual cues available, the rats were trained to perform the task blindfolded⁵⁰. The rats required 16.29 ± 1.21 training sessions to complete this third training phase (range, 12 to 22 sessions).

Surgery and electrophysiological recordings. When proficient at performing the task, the rats were implanted with an array of recording electrodes just dorsal to the anterodorsal thalamic nucleus (1.5 mm posterior to bregma, 1.3 mm lateral to bregma and 3.7 mm ventral to the cortical surface). After recovery (7 d), the activity on each of the ten electrode wires was monitored while the rat foraged for food pellets thrown randomly inside the gray cylinder apparatus. When a cellular waveform was well-isolated from the background noise (signal-to-noise ratio > 2:1) of an identified head-direction cell, a standard session (8 min) was recorded in the cylinder. Two light-emitting diodes (LEDs), spaced ~12 cm apart and positioned above the rats' head along its longitudinal axis, were used to indicate the rat's directional heading. Their positions were monitored at 60 Hz with a two-spot video tracking system. The firing rate of the cell in relation to the rat's head direction was computed and analyzed offline (LabView, National Instruments).

If the cell was classified as directional (Rayleigh $r > 0.4$), the rat was blindfolded and underwent a disorientation procedure, which consisted of gently spinning them back-and-forth in an opaque cardboard box for about 1 min, while the experimenter walked around the periphery of the curtain and cylinder. The rat was then placed in the refuge area, and the cell was recorded for 6 min with the door closed. A second video camera was positioned above the refuge in order to track the rat's directional heading while in the refuge. In cases when there was an obvious shift in the cell's PFD during the 6-min refuge session, the PFD value during the last minute was taken as the refuge value for the next session and used to calculate the PFD shift. Then the door of the refuge was opened and the rat was allowed to forage for food pellets for 10 min. Twelve food cups were randomly scattered on the floor of the apparatus away from the walls. The pellet locations were varied pseudo-randomly, and only one cup was baited at a time. Rats typically completed 2–10 trials in a 10-min recording session.

Head-direction cell analyses. The cell's PFD was analyzed and plotted offline (firing rate × head direction) for each recording condition (cylinder, refuge and arena). Analysis of directionality was assessed using the Rayleigh test. A cell was classified as a head-direction cell in the cylinder if the Rayleigh r was ≥ 0.4. All head-direction cells were then recorded in the food-foraging task. To determine the cell's mean PFD on each trial (foraging trip) or during inter-trial intervals, the mean firing direction was calculated from the firing rate versus head-direction tuning function by determining the center of mass (COM) using ± 8 bins (64°) on either side of the bin with the maximal firing rate. $COM = (\sum (f_i \times \alpha_i)) / FR$, where f_i is the firing rate in the i th bin, α_i is the angle of the i th bin, and FR is the sum of the firing rates in all bins. The PFD shift was defined as the difference in the cell's PFD between one episode and the previous episode (Supplementary Fig. 5). On five occasions (5 sessions, 20 trials), two head-direction cells were recorded simultaneously during the task; the analyses of the coherence of these simultaneously recorded cells are shown in Supplementary Figure 14. A complete excursion included both an outbound and an inbound trip. The outbound trip was defined as the rat's path from the refuge to the cup containing the food. The return trip was defined as the rat's path from the food cup to the wall of the apparatus, but not its path from the peripheral wall to the refuge when it made an error (Supplementary Fig. 3).

Behavioral analyses. The behavioral analyses (errors, mean head direction of the return trip and number of turns) were measured offline using LabView software (Supplementary Fig. 3). A trial was counted as correct, if the rat reached the periphery of the arena between the two vertical barriers that framed the refuge (Fig. 1c). For all analyses, clockwise heading errors and PFD shifts were defined as negative, counterclockwise deviations were defined as positive. In some trials the rats returned to the refuge without having found the food pellet; these trials were included in the analyses when a clear return path was identified ($n = 64$; Supplementary Fig. 15).

Statistical analyses. We used unpaired Student t -tests to test for behavioral differences between correct versus incorrect trials, and used a one-way ANOVA to test for within-session effects of heading error and PFD shift. Correlations between different measures were computed using a Pearson correlation (Supplementary Fig. 16).

49. Frohardt, R.J., Bassett, J.P. & Taube, J.S. Path integration and lesions within the head direction cell circuit: comparison between the roles of the anterodorsal thalamus and dorsal tegmental nucleus. *Behav. Neurosci.* **120**, 135–149 (2006).
50. Whishaw, I.Q. & Maaswinkel, H. Rats with fimbria-fornix lesions are impaired in path integration: a role for the hippocampus in "sense of direction". *J. Neurosci.* **18**, 3050–3058 (1998).