

SHORT COMMUNICATION

Properties of place cell firing after damage to the visual cortex

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Abstract

Hippocampal place cells were recorded while rats with lesions of the striate visual cortex foraged for food pellets in a cylindrical arena. Compared to control rats, rats with striate damage had place cells whose firing was less well organized in space, according to a measurement of spatial coherence. More importantly, the spatial location of firing fields in rats with striate lesions was poorly controlled by three-dimensional objects, unlike the fields of either normal sighted rats or early blind rats. These findings suggest a possible contribution of the striate visual cortex to the selection of cues used for anchoring place cell firing fields in space.

Introduction

Place cells are hippocampal pyramidal cells that discharge when the freely moving rat is in a specific, restricted area of the environment called the firing field (O'Keefe & Dostrovsky, 1971). Previous work has shown that visual cues play a determinant role in the spatial control of place cell firing (e.g. O'Keefe & Speakman, 1987). For example, rotating a white cue card attached to the inner wall of the recording cylinder induces an equivalent rotation of firing fields. This effect indicates that the cue card exerts spatial control over place cell activity (Muller & Kubie, 1987). However, firing fields can remain spatially stable for some time even when the lights are switched off or all visual cues are removed (Quirk *et al.*, 1990; Markus *et al.*, 1994; Save *et al.*, 2000). Under these circumstances, rats seemingly use motion-related cues as well as olfactory and tactile information to maintain stable firing fields and possibly for orientating in space. Thus, visual information is important for place cell firing but its absence can be readily compensated for by relying on other sensory systems.

The above suggestion is partly supported by behavioural work showing that spatial performance in blind rats is not profoundly impaired in the reference memory water maze task (Lindner *et al.*, 1997). In view of the relatively preserved spatial skills in blind rats, it seems somewhat unexpected that ablation of the striate visual cortex induces a strong deficit in radial arm maze performance (Foreman & Stevens, 1982; McDaniel & Brown, 1984). A direct comparison of rats with striate cortical damage and blind rats revealed a much stronger impairment in the brain-damaged rats (Goodale & Dale, 1981). This impairment suggests that the striate cortex might accomplish some central function in spatial processing. If this assumption is correct, then one might expect visual cortical lesions to have a stronger effect on the firing properties of hippocampal place cells than simply blinding the rat. Because we have previously shown

that the activity of hippocampal place cells was not markedly altered in blind rats (Save *et al.*, 1998), the present study was aimed at recording place cells from rats with lesions of the striate visual cortex. Our experimental design was made as similar as possible to that used in Save *et al.* (1998) so as to compare the properties of place cell discharge in rats with striate lesions with those observed in normal sighted and early blind rats.

Materials and methods

Long-Evans male rats (Janvier, Le Genest-Saint-Isle, France) were housed one per cage in a temperature-controlled colony (20 ± 2 °C) on a natural light-dark cycle. Electrode implantation and screening for unit activity started when they were ≈ 90 days old and weighed between 300 and 350 g. They had water *ad libitum* during all phases of the experiment. Before electrode implantation, the rats were food-deprived to 85% of *ad libitum* body weight and then trained in a 'pellet-chasing' task (Muller *et al.*, 1987) for 10 days so as to permit estimation of positional firing rates everywhere in the cylinder. In this task, the rat had to retrieve 20-mg food pellets scattered into a grey cylinder 50 cm high and 76 cm in diameter. The cylinder where rats were tested was located in a room adjacent to the room containing the electrophysiological unit recording system. As in our previous studies (Save *et al.*, 1998), the cylinder was homogeneously painted grey and was visually isolated from the rest of the laboratory by a concentrically placed cylindrical curtain 250 cm in diameter and height. Similarly, during all phases of the study, a radio tuned to an FM station was fixed to the ceiling in a central position relative to the cylinder, producing background noise > 70 dB to mask accidental and non-controlled directional sounds. Three landmark objects similar to those used by Save *et al.* (1998) were used. The objects differed from each other in colour, size, shape and texture. Their locations relative to each other were fixed. Each object was against the wall of the cylinder, and their arrangement formed an isosceles triangle. All

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procedures complied with NIH guidelines (Principles of Laboratory Care, publication number 86–23, revised 1985).

After behavioural training, rats were anaesthetized with pentobarbital (40 mg/kg). Damage to the striate cortex was produced by thermocoagulation of the dura, a technique successfully used in past research to produce lesions limited to the cortical mantle (Save *et al.*, 1992; Baunez *et al.*, 1998). Briefly, two windows were opened in the skull to expose the brain at the following coordinates relative to bregma: AP (behind the bregma) –6.0 to –10.0 mm; L \pm 1.5 to \pm 6.0 mm (Zilles, 1985). Bilateral striate lesions were made by applying the tip (diameter, 0.5 mm) of a calibrated soldering iron (temperature 120 °C) directly on the dura for 0.5 s at each of five points of the cortical surface within the exposed brain area. A piece of sterile gelfoam was placed in the openings. In addition, a movable array of 10 electrodes (Kubie, 1984) was implanted in the hippocampus (P 3.8, L 3.0 mm to bregma, and 1.5 mm below dura; Paxinos & Watson, 1986; see Save *et al.*, 1998 for detailed description of electrode implantation). Miniature screws were placed over the right olfactory bulb, the left frontal cortex and the left cerebellar hemisphere to anchor the headstage. The screws and nut were then embedded in dental cement, and the bottoms of the three drive screw assemblies were cemented to the skull. The rats were then sutured and received an injection of antibiotic (terramycin, 60 mg/kg, i.m.) and of analgesic (tofedine, 0.06 mg/kg, s.c.).

One week after surgery, the activity from each electrode was screened while the rats performed the pellet-chasing task in the cylinder. If no recordable cell could be isolated, the electrode bundle was advanced 25–50 μ m. Recording was done with a system made of the electrode wires, a headstage with a unity gain preamplifier for each wire and a cable to lead the signals to a commutator. Signals were then amplified (\times 10 000 with 0.3–10 kHz band-pass filtering) and sent to a 250-kHz analogue-to-digital (A/D) board in a Pentium-based computer.

Spike discharges of single units were separated by using on-line and off-line clustering software (DataWave, Longmont, CO, USA). The most useful parameters for cluster cutting were spike height and amplitude, spike width and A/D-values at selected points of the waveforms. The rat's head position was tracked by locating a light-emitting diode (LED) that was secured \approx 1 cm above the head. The LED was tracked at 50 Hz with a digital spot follower connected to a camera fixed above the maze. The LED was detected in a grid of 64×64 square regions (pixels) of side 35 mm.

Once a waveform of sufficient amplitude ($>$ 100 μ V, i.e. $\approx 3 \times$ the background noise) was isolated, several successive 16-min recording sessions were run consecutively to establish whether the positional firing patterns were controlled by the position of the set of object landmarks in the recording arena. Only well-isolated cells with an overall firing activity $>$ 0.2 Hz and clear location-specific activity were included in the data set. The cluster boundaries for selected waveforms were refined during off-line analyses, using very conservative criteria for at least four out of the eight possible waveform parameters (generally, spike amplitude, spike width and A/D-value in the early and late portion of the waveform). This ensured that the signals were produced by single cells rather than by several cells with similar waveforms. In addition, only cells with similar spatial discharge properties during initial and final standard sessions (see below) were kept for the analysis so as to ensure that the same cell or set of cells was studied across the whole sequence of environmental manipulations.

Recordings were made first with the objects in a 'standard' position relative to the laboratory frame. Then, a second session with the objects in the standard position was made to ensure that the position

of the firing fields was stable under constant conditions. If this was the case, the third session was a 'rotation' session in which the set of objects had been rotated 90° counter-clockwise from the standard position. The fourth session was another 'rotation' session in which the object set had been rotated 90° clockwise back to the initial standard position. Between successive sessions 1–4, the rat was returned to its home cage, the floor and inner wall of the cylinder were cleaned, and finally the objects were placed at appropriate locations in the cylinder. In session 5, the three objects were removed from the apparatus while the rat remained in the cylinder. Care was taken not to disturb the animal during removal of the objects. Thus, the floor of the apparatus was not cleaned between sessions 4 and 5. Recording of session 5 was started immediately after the experimenter exited the recording room. Finally, when the session with the objects removed was finished, the rat was returned to its home cage and the floor of the cylinder was cleaned. The objects were returned to their standard positions so as to run a final standard session. This standard session was to test whether the original firing fields were still present at the same locations in the cylinder as during the initial standard session, regardless of whatever occurred during sessions 3–5. As in our previous studies, the cylinder itself was left in a constant position across all sessions.

The results were first analysed by visual inspection of positional firing rate maps associated with each session. To construct such maps, the total time the light-emitting diode was detected in each pixel (dwell time) and the total number of spikes in each pixel were accumulated for the session duration (i.e. 16 min). The rate in each pixel (i.e. the number of spikes divided by the dwell time) was then assigned a grey shade from a scale of four shades. Thus, white pixels represented locations where the firing rate was exactly 0.0 Hz for the whole session. Firing rates were shown as light grey, dark grey and black pixels from low to high. Because the in-field firing rates of place cells can vary over a large range, the values used as boundaries between grey categories were autoscaled for the map of the first session recorded for a given cell (Muller *et al.*, 1987). The rate categories used for subsequent sessions were the same as for the first session. A firing field was defined as a set of at least nine pixels contiguous by an edge and with firing rate just above the grand mean rate calculated over the whole apparatus.

Results

Cortical lesions were made in 10 rats in which the striate cortex (Oc1M and Oc1B) was completely destroyed. In some rats, the lesions encroached on the most lateral portions of mediolateral and mediomedial occipital cortex (Oc2ML and Oc2MM) and medial portion of the lateral occipital cortex (Oc2L; Zilles, 1985). Minimal damage was found in the most posterior part of the agranular retrosplenial area (RSA). No damage to the underlying hippocampus was observed in any rat. Figure 1 displays coronal sections of a rat brain showing the extent of cortical damage. Overall, the lesions were comparable in extent and location to those previously reported to result in spatial and visual deficits (e.g. Foreman & Stevens, 1982; McDaniel & Wall, 1988).

Five rats lost their headstage before any useful recording could be made. This happens frequently in rats whose skull has been partly removed for lesion purpose. The bone area after skull removal is reduced and consequently does not permit as tight anchoring of the headstage as in rats with an intact skull. The remaining five rats with striate cortex damage yielded 26 complete sequences of six recording sessions (36 hippocampal place cells recorded in a total of 156

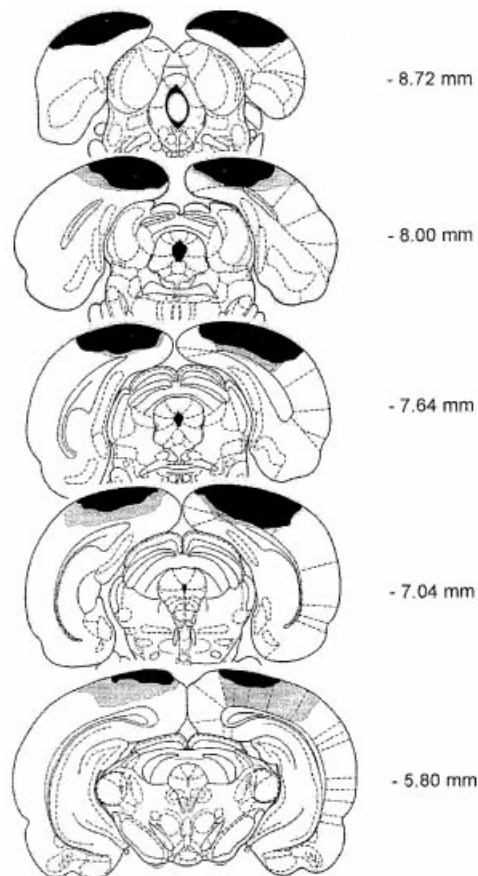


FIG. 1. Coronal sections of the rat brain showing the extent of damage to the visual striate cortex (minimum, dark shaded; maximum, light shaded). Numbers at the right of each brain section are the bregma-referred coordinates according to Paxinos & Watson (1986).

sessions). Nineteen other place cells were excluded from further analysis because they were lost in the course of recording. That it was possible to record place cells from rats with striate cortex damage shows that such lesions do not totally disrupt hippocampal place cell activity. Similarly, striate cortex lesions did not appear to have a major impact on the proportion of place cells because the number of recorded cells per rat was in the range usually found for intact rats. Of the 36 place cells, 17 were from CA1 and 19 from CA3. Because no differences pertinent to this study were observed among the cells in these two regions, all place cells were treated the same.

The positional firing properties of place cells from rats with striate lesions were computed for the initial standard session and compared to the properties of a pool of 55 cells recorded from 10 rats with an intact striate cortex. Although eight of these 10 rats were used for a different purpose, their initial recording session was run under the same conditions and during the same period as for rats with striate lesions. The last two intact rats were also tested concurrently with the rats with striate lesions and were subjected to rotations of the object set on several sessions. The results of these comparisons revealed that, in many respects, place cells from rats with striate lesions were similar to place cells from intact rats. No significant difference was found in overall firing rate, in-field mean firing rate and peak firing rate (see Table 1; all t -values < 1.52 , d.f. = 89, NS). Similarly, no difference was found in field size ($t_{89} = 1.18$, NS) and position information content ($t_{89} = 1.77$, $P = 0.08$, NS). In contrast to the

TABLE 1. Main parameters of cell firing in control rats and rats with striate lesions

Parameter	Control rats ($n = 55$)	Rats with striate lesions ($n = 36$)
Overall firing rate (Hz)	1.38 ± 0.19	1.47 ± 0.46
Firing rate in field (Hz)	5.39 ± 0.55	4.01 ± 0.71
Peak firing rate in field (Hz)	9.61 ± 0.91	7.58 ± 1.15
Field size (pixels)	96.3 ± 8.1	82.2 ± 7.4
Positional information content	1.50 ± 0.08	1.28 ± 0.09
Spatial coherence	0.68 ± 0.02	$0.58^* \pm 0.03$
Mean interspike interval (ms)	35.1 ± 1.0	32.2 ± 1.2
Mode interspike interval (ms)	5.0 ± 0.2	5.4 ± 0.3

For each measurement, values are shown as means \pm SEM. Position information content (I) measures the amount of information (in bits) conveyed about spatial location by a single action potential emitted by a single cell (Skaggs *et al.*, 1993) and is calculated as: $I = \sum_i (\lambda_i/\lambda) \times \log_2 (\lambda_i/\lambda) \times P_i$, where λ_i is the mean firing rate in each pixel, λ the overall mean firing rate and P_i the probability of the animal being in pixel i (i.e. dwelling time in pixel i /total dwelling time). Spatial coherence estimates the local smoothness of firing fields (Kubie *et al.*, 1990), and is a nearest neighbour two-dimensional autocorrelation in which the firing rate in each element in the rate array is paired with the aggregate rate in the eight nearest pixels. The total size of the cylinder was ≈ 700 pixels (hence, the number of values entered for computing position information content and spatial coherence was ≈ 700). The mean and mode for interspike intervals were obtained by plotting discharge in 100 bins over 100 ms (1-ms bin width). *Significantly different from control cells (two-tailed Student's t -test on the z -transforms of the correlations: control $z = 0.89 \pm 0.04$, striate $z = 0.70 \pm 0.03$; $t_{89} = 3.14$, $P < 0.005$).

above measurements, the spatial coherence of firing fields (Muller & Kubie, 1989) was found to be somewhat lower in rats with cortical damage than in control rats (see Table 1). A possible basis for reduced coherence of firing fields is a change in the temporal dynamics of the discharge, e.g. cells tend to fire more unreliably or in irregular bursts. To test for this, normalized interspike interval histograms (100 bins; 1-ms bin width) were constructed for recordings with sufficient data (Table 1). No evidence was found for a shift in either mean interspike interval ($t_{85} = 1.83$, NS) or interspike interval mode ($t_{85} = 0.83$, NS), thus suggesting that cells did not discharge more unreliably or in bursts.

With regard to the effects of cue manipulations on cell firing, we first assessed changes between two successive sessions by visual inspection of the associated rate maps. To confirm this judgement, we then calculated the pixel-by-pixel cross-correlation as the positional firing patterns for the two sessions were rotated in 6° steps relative to each other. The rotation associated with the highest correlation (R_{MAX}) was taken as the rotation of the firing field between the two sessions. In all instances, simultaneously recorded cells reacted in the same way to cue manipulations so that it was always possible to draw unambiguous conclusions about the effects of a given cue manipulation. It should be added that, apart from the location of firing fields, none of the measured properties of cell firing (i.e. in-field firing rate, position information content and spatial coherence) was affected by any cue manipulation (all F -values < 1).

Figure 2 provides representative firing rate maps that show the most commonly observed patterns of change. For all place cells recorded from rats with striate lesions, firing fields were spatially stable when the object set was unchanged across the first two standard sessions as well as the final standard session (angular error $3 \pm 1^\circ$; $R_{\text{MAX}} = 0.56 \pm 0.02$; $n = 36$ cells).

Rotations of the object set were made on 52 sessions (i.e. 26 sessions with a $+90^\circ$ rotation and 26 sessions with a -90° rotation of

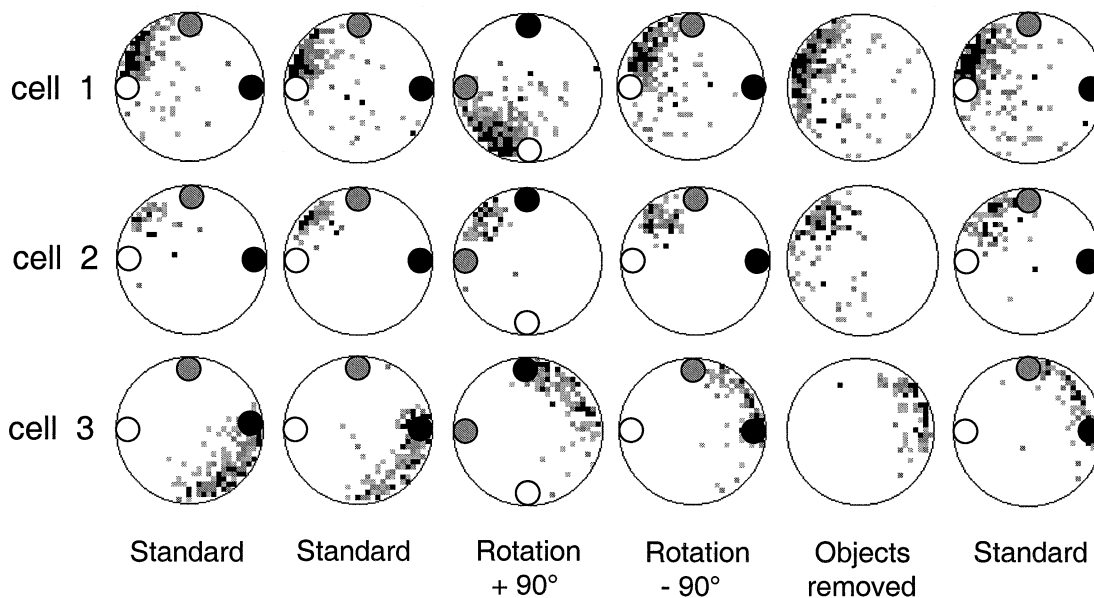


FIG. 2. Firing rate maps of three place cells during six recording sessions. These maps show the three observed patterns of change in place cell spatial firing after cue manipulations. Cell 1 had a firing field controlled by the location of the object set during both rotations, its field rotated $+90^\circ$ after the first rotation and -90° after the second rotation (a pattern found in 6/26 instances). Cell 2 had a firing field never controlled by the location of the object set; its field was stable after both rotations (a pattern found in 18/26 instances). Cell 3 had a firing field partly controlled by the location of the object set; its field rotated $+90^\circ$ after the first rotation but was stable after the second rotation (a pattern found in 2/26 instances). In all maps, pixels in which no firing was observed are white. Pixels in which firing did occur are shown as grey-shaded squares. The highest firing is coded as black, and intermediate rates are shown as light grey and dark grey from low to high. The three objects in the cylinder are shown as white, grey and black circles. Median firing rates for grey shades are Cell 1: white 0.0, light grey 0.7, dark grey 2.8 and black 7.5 AP/s; Cell 2: 0.0, 0.6, 1.8 and 5.9 AP/s; Cell 3: 0.0, 1.7, 5.5 and 12.5 AP/s.

the objects). Of these 52 sessions, 14 led to appropriate field rotation. The first ($+90^\circ$) rotation of the object set was followed by an equivalent rotation of the fields in 8/26 sessions (rotation error $2 \pm 6^\circ$; $R_{MAX} = 0.48 \pm 0.03$; $n = 16$ cells). In the remaining 18 sessions, fields were stable relative to the room because they shifted by only 10° (rotation error $80 \pm 3^\circ$; $R_{MAX} = 0.39 \pm 0.05$; $n = 20$ cells). The second (-90°) rotation of the object set was followed by a 90° angular shift in 6/26 sessions (rotation error $-6 \pm 4^\circ$; $R_{MAX} = 0.36 \pm 0.04$; $n = 8$ cells). In the remaining 20/26 sessions, fields remained stationary relative to the room (rotation error $-83 \pm 2^\circ$; $R_{MAX} = 0.47 \pm 0.04$; $n = 28$ cells). In two instances, fields that had rotated appropriately after the first rotation did not return to their initial standard position when the object set was back in the standard configuration. Overall, the firing fields of rats with striate lesions were consistently controlled by the object set in the two rotation sessions in only 6/26 occasions (23%).

Importantly, no systematic effect of experience was found on the ability of the objects to exert cue control over firing fields; in all rats, the occurrence of sessions in which the fields were seen to rotate with the objects was unpredictable across successive exposures. For example, one rat recorded for seven complete sequences was thus subjected to 14 rotations of the object set over a period of 3 weeks, which resulted in the following sequence of controlled (C) and uncontrolled fields (U): U-U-C-U-C-C-U-U-U-U-U-U-C-U. Similarly, no difference was found in the characteristics of fields consistently controlled and fields poorly controlled by the object set. All discharge properties of both classes of cells were very similar (overall firing rate: $t_{34} = 0.16$, NS; in-field mean firing rate: $t_{34} = 1.67$, NS; peak firing rate: $t_{34} = 1.37$, NS; field size: $t_{34} = 0.30$, NS; position information content: $t_{34} = 0.49$, NS; spatial coherence: $t_{34} = 0.01$, NS).

Rotation of the object set performed in the two intact rats resulted in appropriate field rotation in 10/10 sessions (rotation error $4 \pm 3^\circ$; $R_{MAX} = 0.44 \pm 0.03$; $n = 16$ cells). Thus, failure to get cue control in rats with striate lesions did not appear to be caused by some idiosyncratic, uncontrolled aspects of the experiment because explicit cue control was observed in intact rats tested under identical conditions. Lastly, a comparison of this data with the results of Save *et al.* (1998), who recorded place cells under strictly similar conditions, revealed that the control exerted by the object set in rats with striate lesions was much diminished compared to either normal sighted or early blind rats (90%, $\chi^2 = 21.2$, $P < 0.001$ and 100%, $\chi^2 = 33.2$, $P < 0.001$, respectively).

Removal of the objects was made on 26 sessions. In all instances, the firing fields were observed to remain fairly stationary relative to the room with a mean drift of $15 \pm 3^\circ$, a value not different from the drift observed in normal rats recorded under comparable circumstances ($13 \pm 3^\circ$, $t_{38} = 0.54$, NS; Save *et al.*, 2000; data from group 'light/no cleaning'). In addition, none of the measured properties of cell firing was affected by object removal (in-field firing rate 1.1 ± 0.3 action potentials/s (AP/s) vs. 1.3 ± 0.5 AP/s, $t_{25} = 0.4$, NS; position information content 1.2 ± 0.1 vs. 1.0 ± 1.0 , $t_{25} = 1.25$, NS; spatial coherence 0.57 ± 0.03 vs. 0.53 ± 0.03 , $t_{25} = 0.97$, NS).

In the last standard session, the fields were observed in the same location as during the first standard session in 24/26 instances. The two remaining instances came from the cells that had not returned to their standard position after the second rotation, and whose fields were therefore offset by $+90^\circ$ relative to the first session (see Fig. 2, cell 3).

Lastly, no difference in behaviour was found between rats with cortical damage and control rats. Both groups moved at similar speeds and displayed similar patterns of object exploration, as shown

by measurements of the duration and number of contacts with the objects (all F -values < 1).

Discussion

The present results indicate that damage to the rat primary visual cortex did not disrupt some of the basic properties of hippocampal place cells. Thus, rats with visual cortical lesions had functional place cells that were similar, on several measurements, to place cells from control rats with an intact cortex. Also, fields were as stable across time in rats with striate lesions as in control rats. Even though our study did not directly address this issue, we anecdotally observed that the fields of cells recorded over successive days were consistent in shape and location within the cylinder. This observation suggests a different contribution of the striate cortex to place cell firing compared to the perirhinal cortex whose lesion was recently found to result in pronounced instability of firing field locations (Muir & Bilkey, 2001).

One major deviation from the similarity of firing fields in rats with striate lesions and control rats concerns the spatial coherence of firing fields, which was found to be diminished in brain-damaged rats. A possible explanation for this effect relies on the idea that, although visual input provides the necessary information for initial anchoring of spatial firing patterns relative to the environment, it also might be used for on-line recalibration of position. In this view, place cell spatial firing is thought to primarily rely on the dynamic use of internally generated, motion-related information (e.g. kinaesthetic and proprioceptive signals) to update the rat's position in the environment (McNaughton *et al.*, 1996). Such signals, however, are known to accumulate errors across successive movements in space. Current hypotheses therefore make the additional assumption that errors generated during position computations based on motion-related cues need to be corrected by recalibration based on visual cues. Presumably, if rats with striate lesions are impaired in processing visual information, their recalibration process may also be defective. Therefore, organization of place cell firing might be less precise than in intact rats, thus making firing fields less coherent after striate lesions damage.

Another major result from this study is that, contrary to both sighted rats (Cressant *et al.*, 1997 and present study) and early blind rats with an intact striate cortex (Save *et al.*, 1998), place cells in rats with striate lesions were demonstrated to use three-dimensional objects much less efficiently for the spatial anchoring of firing fields. In rats with an intact striate cortex, rotation of the objects is followed by equivalent rotation of firing fields in a vast majority ($\approx 90\%$) of instances. That is, the object set exerts almost ideal control on firing field locations. This control was found to be dramatically reduced in rats with striate lesions in which rotations of the object set induced appropriate field rotations only in a minority of instances ($\approx 27\%$). Though small, this fraction suggests that the inability of the objects to control the angular firing position in rats with striate lesions was not absolute, however. Noticeably, all fields that did not rotate with the object set were stationary relative to the laboratory. Thus, rats with striate cortex damage tended to use the laboratory as the reference frame for anchoring the spatial firing of their place cells, contrary to control rats which used the objects intentionally placed into the recording cylinder to serve as spatial landmarks. Together, these observations indicate that the selection of the spatial reference frame by place cells is a complex process that seems to rely on a subtle balance between several influences. The effects of striate cortex

lesions can be best described as an alteration of this balance than as a total disruption of the cue control process.

Several possibilities are in order to explain the observed alteration. Firstly, although striate cortex lesions dramatically impair visual processing, they do not remove all visual pathways. For example, it is known that the pathway from the retina to the striate cortex via the dorsal lateral geniculate nucleus conveys information about high spatial frequencies while the pathway reaching the superior colliculus conveys information about lower frequencies (Dean, 1981). Thus, damage to the visual cortex might impede the processing of fine-grain cues (such as small objects) so that the rat can rely only on coarse-grain cues for its localization. In our situation, such coarse-grain cues eventually would be provided by distant uncontrolled landmarks such as, for example, patterns of light reflected from the curtains enclosing the recording cylinder.

Another possibility is simply that rats with striate damage may rely on nonvisual distant information (such as odours or sounds) to set the reference direction for firing fields. Such information, in combination with motion-related cues, might help the rat to know its position everywhere in the apparatus in the absence of explicit local cues. This possibility was bolstered by the observation that object removal did not induce a major change in place cell firing in rats with cortical damage. As in control rats, fields were seen to be relatively stable, thus suggesting the use of a combination of intra- and extra-apparatus cues (such as remaining odours) in conjunction with motion-related cues.

Lastly, a less specific though quite interesting idea is that the visual cortex would be involved in the initial calibration of the spatial reference frame used by the place cell system. For example, it might be required for selecting the visual cues to use as landmarks. Interestingly, disrupted cue control over place cell firing fields is also observed after lesions of the postsubiculum (J. P. Goodridge, B. W. Stackman, W. B. Archey and J. S. Taube, unpublished observations, reported in Taube, 1998, p. 240), thus raising the possibility that both the striate cortex and postsubiculum work together to provide the angular reference direction for orientating the hippocampal spatial map. Such function would explain why some properties of place cells, especially in terms of cue control, are strongly altered after lesions of the striate cortex but much less so after enucleation (Save *et al.*, 1998). Note, however, that a simpler interpretation of this difference regards the fact that, while the enucleated animals in the study of Save *et al.* never had vision, the rats with striate cortex damage were sighted during pretraining. Thus, reduced cue-control by the object set could result from the shorter period available to the rats with striate lesions to learn nonvisual strategies for orientating on the basis of intra-apparatus objects. Although this explanation is appealing, the extended postsurgery period (10 weeks on average) during which rats were daily screened for units to be recorded (and thus were daily exposed to the object) militates against it. Nevertheless, it must be acknowledged that compensatory mechanisms cannot be discarded as a possible explanation of the dissociation between enucleated and striate cortex damaged rats. One way to address this issue in further research will be to record place cells from rats with early lesions of the striate cortex. Interestingly, however, the idea that the striate cortex contributes in an essential way to place cell firing by setting a coherent frame of reference based on available visual cues in the environment is coherent with the observation that striate cortex lesions induce much deeper behavioural deficits in spatial tasks than blindness (e.g. Goodale & Dale, 1981; see also Introduction above).

In conclusion, the present study provides evidence that lesions of the visual striate cortex alter the cue control normally exerted by

stimulus cues placed in the recording apparatus. We believe that this effect reveals a fundamental contribution of the visual cortex to place cell spatial firing, an idea supported by behavioural data. Uncovering the type of spatial preprocessing achieved by the striate cortex will probably rely on the recording of the discharge correlates of its neurons under conditions similar to those used for recording hippocampal place cells.

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Abbreviations

A/D, analogue-to-digital; AP/s, action potentials/s; R_{MAX} , highest correlation between two rate maps.

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