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Local cortical lesions abolish lateral inhibition at direction selective cells in cat visual cortex

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Summary. Many cells in the cat visual cortex display a strong selectivity for the direction of motion of an optimally oriented stimulus. Postsynaptic inhibition has been suggested to generate this direction selectivity in simple cells, but the intracortical pathways involved have not been identified. While continuously recording from simple cells in layers 4 and 6, we have inactivated the superficial cortical layers in small regions 0.4–2.5 mm from the cortical column under study by using heat lesions, localized cooling or γ-aminobutyric acid (GABA) microiontophoresis. When inactivation affected cortical regions retinotopically representing motion in the non-preferred direction towards the receptive field, the responses to movement in this direction increased, and the recorded cells lost direction selectivity due to loss of inhibition. Our results indicate that direction selectivity of simple cells involves asymmetric inhibition of predictable cortical topography.

Key words: Striate cortex – Direction selectivity – Inhibition – Local inactivation

Introduction

Direction selectivity is rarely found in cells of the cat lateral geniculate nucleus (Kozak et al. 1965) but is well established in cat visual cortical cells (Hubel and Wiesel 1962). Inhibitory cortical mechanisms seem to participate in the cortical generation of direction selectivity as was shown with intracellular recordings (Benevento et al. 1972; Creutzfeldt et al. 1974; Innocenti and Fiore 1974) and with microiontophoresis of the γ-aminobutyric acid (GABA) antagonist bicuculline (Sillito 1977). The intracortical pathway of such an inhibition, however, has not been demonstrated. Inhibition causing direction selectivity should involve a spatial asymmetry of inhibitory elements, eliciting stronger inhibition during motion in the non-preferred direction. To test this hypothesis, in the present experiments we have applied local cortical lesions at some distance from the recorded cells in regions involved in signal processing during motion in the preferred and non-preferred directions, respectively.

Since it is generally agreed that direction selectivity is generated at an early stage of visual cortical signal processing, we have concentrated our first study on that cell class (simple cells) and those cortical layers (4 and 6) which receive direct inputs from lateral geniculate afferents.

Methods

Recordings were made with varnished or glass-coated tungsten electrodes from cells in area 17 of 20 adult cats paralysed with a mixture of d-tubocurarine and gallamine triethiodide and anaesthetized by artificial respiration with 0.2–0.5% halothane in nitrous oxide and oxygen (70 : 30). Blood pressure, expired CO_2 concentration and body temperature were continuously monitored and kept normal throughout the experiments. Atropine sulphate and phenylephrine were topically applied for mydriasis and retraction of the nictitating membranes. The cornea were covered with zero power contact lenses with artificial pupils.

Layer 4 and 6 neurons classified as simple cells (Kato et al. 1978; Henry et al. 1979) with receptive fields in the central visual area were stimulated by light bars moving back and forth with optimal velocity. Visual stimuli were generated with an image generator (Picasso by John Daugman) and presented on an oscilloscope 25 cm in front of the eyes. The image was focussed on the retina by appropriate lenses.

Local cortical inactivation was achieved by heat lesions, local cortical cooling or GABA microiontophoresis. The heat lesions were made during continuous recording from a given cell with a photocoagulator focused on the cortical surface about 2 mm away

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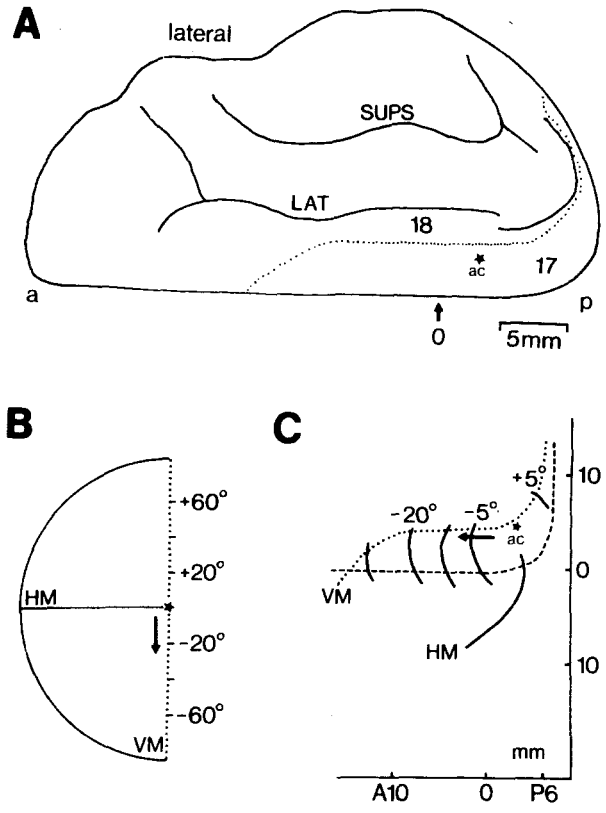

Fig. 1A–C. Topography of moving stimuli in the cat visual cortex (**A–C**). **A** View of the cat cortex from dorsal. a = anterior, p = posterior, SUPS = suprasylvian sulcus, LAT = lateral sulcus, O = interaural plane, ac = representation of the area centralis, the border between area 17 and 18 is indicated by a dotted line. **B** Visual half-field with horizontal (HM) and vertical meridian (VM). **C** Projection of the visual field onto the cortical surface. The arrows in (**B**) and (**C**) show the downward movement of a stimulus in the visual field and the corresponding response wave on the cortical surface

from the recording electrode. For cortical cooling we used a short, 0.35 mm diameter copper rod attached to a cooling chamber perfused with ethanol/water (1 : 1) of constant temperature. The cortical temperature below the cooling rod and at different distances was measured in control experiments with a thermistor probe (0.25 mm diameter). The temperature of the cooling chamber was set to obtain a cortical temperature of 9° C below the rod. For local inactivation by inhibition of cortical cell activity, GABA (0.5 M) was ejected microiontophoretically from a multibarrel electrode placed at a depth of 400 μm below the cortical surface and at different distances between 0.4 and 2.5 mm posterior to the tungsten recording microelectrode. With heat lesions only one cell per hemisphere was studied, reversible cooling and GABA inactivation allowed to test several cells consecutively in the same hemisphere. 104 cells were studied with one of the three inactivation methods. 23 of these were simple cells in layers 4 and 6 with appropriate direction selectivity to test our hypothesis and 18 were suited as control cells.

Peri-stimulus time histograms of the responses and polar plots of the peak responses to movement in different directions (30 degree steps) were compared before, during, and (in the reversible cases) after local inactivation. Changes of direction selectivity were assessed by computing directionality indices (DI = 100 · (rpd-rnpd)/rpd) from the peak responses to motion in the preferred (rpd) and non-preferred direction (rnpd).

The position and size of the heat lesions, the position of the GABA electrode tip in layers 2 or 3 and the recording sites of individual cells in layers 4 and 6 were histologically verified.

Results

To make specific lesions with respect to topography, the cortical representation of the visual field has to be considered (Bilge et al. 1967; Tusa et al. 1978; Fig. 1a–c). The upper visual field is represented posterior in the cortex and a stimulus moving down through the visual field (Fig. 1b) elicits a response-wave travelling from posterior to anterior on the cortical surface (Fig. 1c) and vice versa on return. If direction selectivity is based on inhibition during movement in the non-preferred direction, a lesion in the cortical region which retinotopically represents the response to stimuli moving in the non-preferred direction should lead to an increase of this response due to reduced inhibition from that region. If the larger response during motion in the preferred direction is due to asymmetrical excitation this should arise topographically from the opposite side and a specific lesion there should decrease the response in the preferred direction. Consequently, a lesion on one side of the recording electrode should affect the direction selectivity differentially depending on the underlying mechanism.

In a first approach we created heat lesions of 1–2 mm diameter with photocoagulations focused on the cortical surface. When recordings were made from layer 6, direct effects upon the recorded cell or its excitatory input from the dLGN were minimal. Figure 2b shows a cell with direction selectivity for upward movement in the visual field. According to the two possible hypotheses this could either be due to excitation from anterior or to inhibition from posterior. The heat lesion 2 mm posterior to the recording electrode affected layers 1–3 (Fig. 2a) and it completely abolished direction selectivity in the given cell (Fig. 2c). The response to the non-preferred direction increased thus indicating a loss of inhibition during movement in this direction.

An important experiment to prove the topographical asymmetry of the underlying mechanism was to make different equidistant lesions while recording from a given cell. In a layer 4 simple cell with preferred direction downwards (Fig. 2e) the first lesion was made posterior to the recording electrode on the side of possible lateral excitation, i.e. opposite to the region of lateral inhibition possibly responsible for direction selectivity. This large lesion even

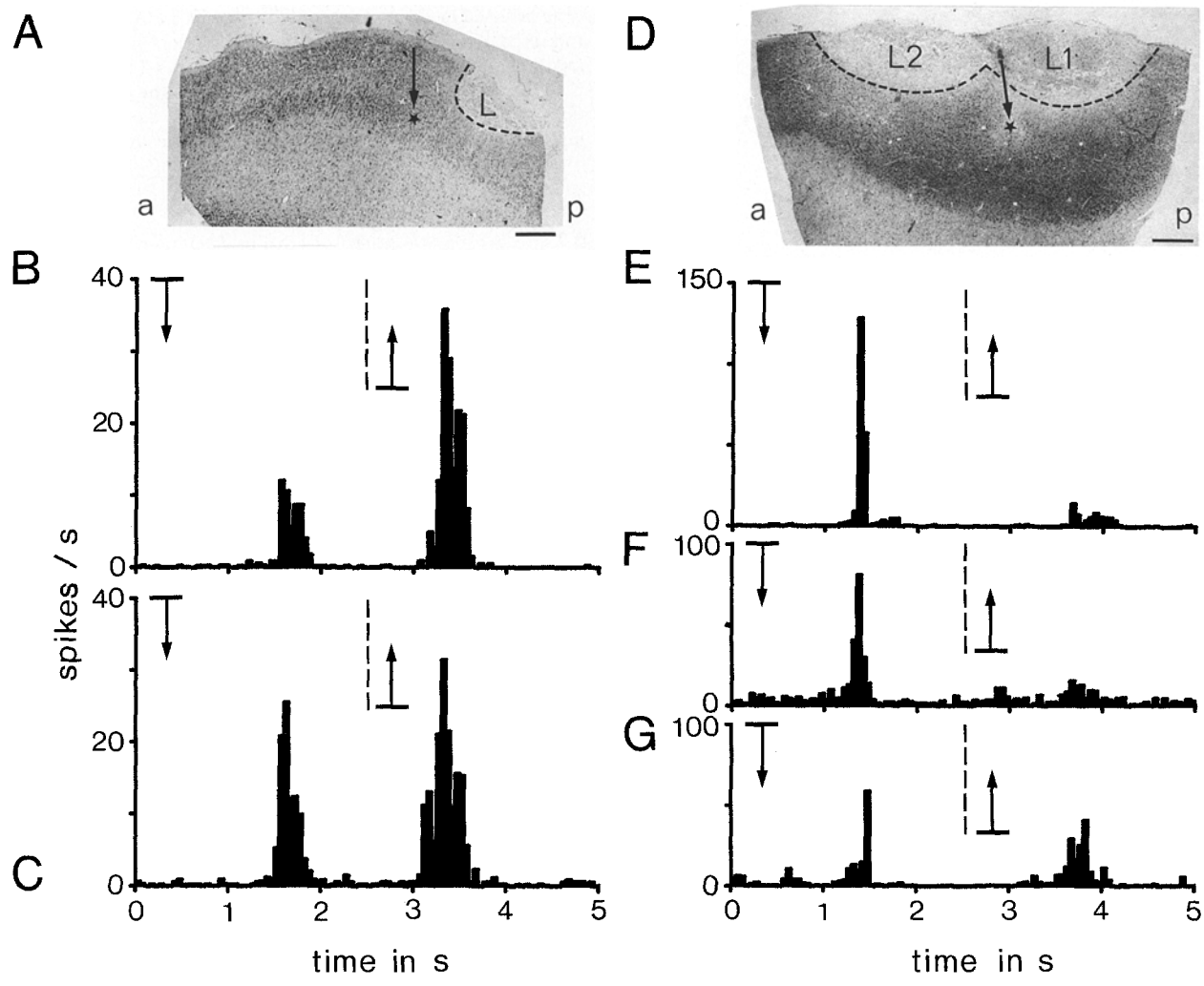

Fig. 2A–G. Changes in a layer 6 simple cell following a superficial heat lesion (**A–C**), and simple cell in layer 4 with anterior and posterior lesion (**D–G**). **A** Recording site of a layer 6 simple cell (asterisk) and histological control of the heat lesion (L) in a sagittal section. Scale bar 1 mm. **B** Peri-stimulus response histogram of the cell prior to the lesion. Bin width 50 ms, 64 trials averaged, upwards and downwards movement of a horizontal bar (upwards preferred). **C** Same cell after the cortical lesion with increased response to downward movement. Directionality indices, DI = (response in preferred direction – response in non-preferred direction) · 100/response in preferred direction: 70 in (**B**) and 20 in (**C**). **D** Sagittal section with recording site (asterisk) and lesions (L1, L2). Scale bar 1 mm. **E** Peri-stimulus time histogram from a simple cell with downward movement preferred (DI = 90). Bin width 50 ms, 32 trials averaged. **F** Histogram obtained following the large lesion (L1). DI = 81. **G** Histogram after lesion L2. Note the increase to upwards movement (DI = 28)

included the layers above the recorded cell (Fig. 2d, L1), but the response in the preferred direction only slightly decreased, and direction selectivity remained (Fig. 2f). The second, anterior lesion (smaller and more distant, Fig. 2d, L2), specifically affected the cortical area appropriate for lateral inhibition causing direction specificity. Now the response to movement in the non-preferred direction increased (Fig. 2g). While both lesions affected the cell in terms of slightly reduced excitation, an inhibitory influence was specifically removed by lesioning the superficial layers anterior to the recorded cell; this finally abolished direction selectivity in this cell. Orientation specificity was not affected by remote heat lesions that abolished direction selectivity.

Heat lesions allow for histological control of the extent of damage and demonstration of the spatial relation of recording site and lesion; they are, however, irreversible. With local cortical cooling and GABA microiontophoresis we were able to reproduce our results with two reversible methods (Fig. 3).

In the first reversible approach we used local cooling with a copper rod gently positioned on the cortical surface posterior to the recording electrode. A continuously recorded simple cell in layer 6 with

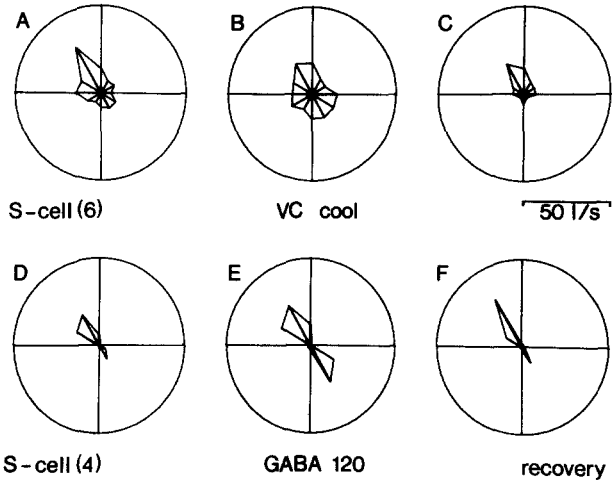

Fig. 3A–F. Effects of localized cortical cooling (**A–C**), and GABA microiontophoresis (**D–F**) on direction selectivity of simple cells in layers 4 and 6. Interleaved presentation of orientations in steps of 30 degrees, 5 trials averaged per orientation, bin widht 100 ms. **A** Polar plot from layer 6 cell with preferred direction upwards to the left (DI = 66). **B** After 2 min of cortical cooling 1.2 mm posterior to the recording electrode, direction selectivity was abolished by disinhibition (DI = 25). **C** Direction selectivity returned during recovery (DI = 85). **D** Simple cell in layer 4 with preferred direction upwards to the left (DI = 55). **E** GABA microiontophoresis with currents from four barrels of a multibarrel electrode (4 × 30 nA) abolished direction selectivity (DI = 7). **F** Complete recovery after GABA application (DI = 67)

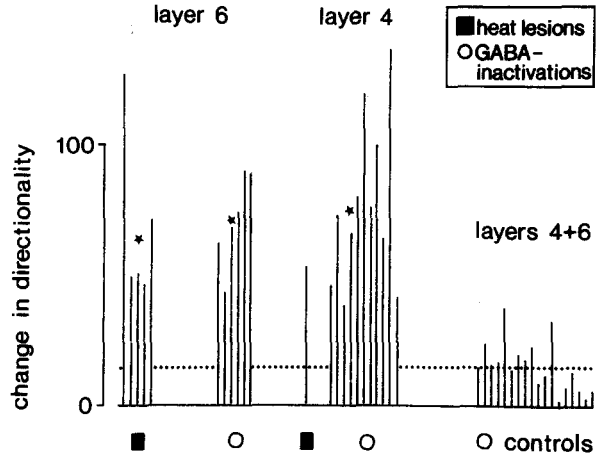

Fig. 4. Diagram displaying changes of direction selectivity in layer 4 and 6 simple cells. Differences of directionality indices before and after localized inactivation. Simple cells from layers 4 and 6 grouped according to the type of inactivation. For properties of control cells see text. Changes above 100 originate from cells with inverted directionality after inactivation. The asterisks indicate the mean changes of DI for each group of cells in layer 6 (64.0 ± 32.5 for heat lesions, 71.0 ± 17.7 for GABA inactivation) and layer 4 (74.58 ± 31.08 for GABA inactivation and heat lesion), which are in all cases significantly different (t-test, $p < 0.0005$) from the mean changes of DI (15.33 ± 9.78) observed in the control group of cells from layers 4 and 6 (dotted line)

preferred upwards movement (Fig. 3a) lost its direction selectivity (and part of the orientation specificity) when the cortex was cooled locally in the region predicted as origin for direction selective inhibition (Fig. 3b, cooling device centered 1.2 mm posterior to the recording electrode). Upon recovery, direction and orientation selectivity returned (Fig. 3c).

The second reversible approach was GABA-microiontophoresis with a micropipette placed in the superficial cortical layers anterior or posterior to the recording site. GABA reversibly inactivates the surroundings of the injection site (Bolz and Gilbert 1986). In a layer 4 simple cell with direction preference for upward movement (Fig. 3d) GABA microiontophoresis (Fig. 3e, 120 nA) abolished direction selectivity without reducing orientation specificity or the overall responsiveness of the cell. With longer application times or further increased ejection currents a depression of response rates was observed in most cells, and currents were adjusted to avoid such direct GABA effects. During the recovery period after GABA application the effects were fully reversible (Fig. 3f). Comparable results were obtained from layers 4 and 6 with GABA microiontophoresis (Fig. 4).

To test the inhibition hypothesis, we have selected 23 direction selective simple cells from layers 4 and 6 with inactivation in the region of predicted lateral inhibition and a control group of 18 cells with preferred orientation and direction of movement perpendicular to the test group to compare possible non-specific effects. Both cells groups were investigated under comparable conditions. Directionality indices (DI) were computed and the maximal changes in directionality following irreversible heat lesions, or reversible GABA inactivation of the nearby visual cortex were tested statistically against the maximal changes observed in the control group. The changes in directionality found in cells of the test group with both inactivation methods were significantly larger than those observed in the control group (t-test, $p < 0.0005$, Fig. 4). Complex cells recorded in layers 2 to 6 displayed similar changes in directionality when subjected to the same experimental procedures.

Discussion

Local inactivation with three different methods was used during continuous recording of single cells in the present experiments. The heat lesions combined the advantage of histological demonstration of site and size of the lesions with the disadvantage of being irreversible with uncertain functional extent relative to the histological damage. To avoid influences of tissue shrinkage that would lead to a loss of the

recorded cell and to minimize direct effects that would silence the recorded cells we have recorded from layer 6 cells and lesioned the supragranular layers at some lateral distance. In addition, the experiments were ended immediately after a successful heat lesion, to assure that histology would reflect the situation during recordings as closely as possible. One of the experiments (Fig. 2d–g) showed that nonspecific changes due to a large heat lesion including parts of the cortex above the cell were in fact observable, but loss of inhibition was seen only with a smaller lesion in a predictable cortical region on the other side of the recording electrode. This shows that the heat lesions do not produce merely unspecific effects.

While local cortical cooling has the advantage of reversibility, the effects spread over longer distances and no histology of the site and size of inactivation can be obtained. Therefore, we abandoned this method of reversible inactivation after having reproduced in two experiments the results obtained with irreversible heat lesions.

Local GABA inactivation of parts of the visual cortex has been applied before (Bolz and Gilbert 1986). These authors reported a local inactivation after microinjections of GABA that did not affect the recorded cells in a distance of about 1 mm. In our hands, dependent on duration and ejection currents of microiontophoretic application, different effects including direct inhibition of the recorded cells could be obtained. Only with appropriate selection of variables, GABA microiontophoresis did specifically and reversibly reproduce the results obtained with heat lesions and local cortical cooling.

There is an apparent contradiction of our results with the experiments of Schwark et al. (1986), who reported not to have found effects on response specificities of cells in the deep cortical layers when cooling the superficial layers. However, to a certain degree the data of Schwark et al. seem to show a weak (though statistically not significant) decrease of directionality in layer 6 cells below supragranular cryogenic lesions. More importantly, there are some methodical differences between the two sets of experiments. Large cortical regions were inactivated above the recorded cells with receptive fields in the periphery of the visual field as opposed to focal lesions asymmetrically located on one side of the recorded cells with receptive fields close to the area centralis. If the dissimilarities are not due to the use of barbiturate anaesthesia in the study of Schwark et al., the different results indicate differences between the center and the periphery of the visual field or between the effects of symmetrical and asymmetrical lesions.

Excitatory convergence (Movshon et al. 1978) and intracortical inhibition (Benevento et al. 1972; Creutzfeldt et al. 1974; Innocenti and Fiore 1974; Goodwin et al. 1975; Emerson and Gerstein 1977; Sillito 1977; Ganz and Felder 1984) have been proposed as possible mechanisms underlying direction selectivity in the cat visual cortex. Accumulating information about structure and function of the visual cortex provides the background for increasingly detailed analyses of cortical circuitry: cell types have been classified by functional properties (Hubel and Wiesel 1962; Kato et al. 1978; Henry et al. 1979), correlated with morphological features (Gilbert and Wiesel 1979; Martin and Whitteridge 1984), and excitatory connections – vertical as well as horizontal – have been demonstrated (Ferster and Lindström 1983; Gilbert 1983, 1985; Gilbert and Wiesel 1985). On the other hand, comparatively little is known about inhibitory pathways within the visual cortex. Recently, Bolz and Gilbert (1986) have provided functional evidence that interlaminar, inhibitory connections from layer 6 to layer 4 generate end-inhibition in layer 4 cells. This function was discussed in relation to identified anatomical connections between layer 6 cells and inhibitory interneurons in layer 4 (McGuire et al. 1984). Direction selectivity was not influenced by this mainly vertically oriented pathway, supporting our results which suggest horizontal rather than vertical connections to be involved in direction selectivity.

Several classes of inhibitory interneurons in different cortical layers and with different ranges of vertical and horizontal connections have been characterized and shown to be GABAergic with respect to immunohistochemistry (Somogyi et al. 1983; Kisvarday et al. 1985; Somogyi 1986) and neurophysiological observations indicate lateral inhibitory processes in the visual cortex (Benevento et al. 1972; Creutzfeldt et al. 1974; Innocenti and Fiore 1974). Without doubt, short range effects are involvd in the generation of direction selectivity since it can be elicited by stimulation within the excitatory field of cortical cells (Goodwin et al. 1975; Emerson and Gerstein 1977; Ganz and Felder 1984). On the other hand, in experiments with visual stimuli outside the classical receptive field long-ranging lateral inhibition has been shown in the visual cortex (Jones 1970; Glezer et al. 1982; for a review see Allman et al. 1985). Lateral inhibition has been elicited by microiontophoretically applying glutamate (presumably to inhibitory interneurons) at a distance of 400 μm from cells recorded in the visual cortex of the cat (Hess et al. 1975), and electrical microstimulation in the motor cortex yielded lateral inhibition up to 1000 μm (Asanuma and Rosen 1973). Our results

suggest that inhibition extending over similar distances is contributing to direction selectivity in the visual cortex.

Long ranging horizontal connections disclosed by HRP injections (Gilbert 1985; Rockland and Lund 1983) need not necessarily indicate horizontal excitatory signal processing. Horizontal excitatory connections as described between columns of equal optimal orientation (Gilbert 1985; Ts'o et al. 1986) might innervate inhibitory interneurons and contribute to direction selectivity, while the shorter lateral connections with columns of perpendicular orientation shown by Matsubara et al. (1985) could connect with inhibitory interneurons involved in orientation selectivity, as suggested by these authors and earlier by Sillito (1979). Alternatively, the GABAergic visual cortical basket cells with axons extending laterally up to 1500 μm (Somogyi 1986; Somogyi et al. 1983) could represent a morphological basis for the type of lateral inhibition that was affected by lesions and inactivation in our study. In area 18 of the cat, Matsubara et al. (1987) have shown GABAergic connections long enough to mediate lateral inhibition between different cortical columns.

According to our evidence, direction selectivity of cells in the visual cortex seems to involve lateral inhibitory mechanisms of predictable topography, asymmetrical in relation to the direction selective cell and mediated by a pathway which includes the upper cortical layers. Orientation specificity apparently involves different cortical circuits than those participating in direction selectivity (Hammond 1978). This is supported by our finding that orientation selectivity remained unchanged following the heat lesions and many of the GABA experiments which abolished direction selectivity (Fig. 3d–f). Our findings support a model of direction selectivity in which a wave of inhibition moves across the cortex ahead of the stimulus sweep, selectively blanking the responses of cells whose non-preferred directions correspond to the direction of the wave.

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