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Excitation and inhibition in orientation selectivity of cat visual cortex neurons revealed by whole-cell recordings *in vivo*

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Abstract

One striking transformation in response properties that occurs in the geniculo-cortical pathway is the appearance of a high degree of orientation selectivity in the cortex. This property may be conceived as arising purely from the excitatory inputs to the cell, as being structured largely by the inhibition a cortical cell receives or could be due to a combination of the two. We have studied the contributions of excitatory and inhibitory inputs to cortical cells' orientation selectivity by analyzing the postsynaptic potentials evoked in cat striate neurones by flashing stimuli of different orientations. We made these recordings using the *in vivo* whole-cell technique (Xing Pei et al., 1991), which provides more stable and reliable results than classical intracellular recording methods. Our results show that the cat striate cortex exhibits a variety of mechanisms to achieve orientation selectivity. Orientation selectivity of a particular cell can be created by excitatory, by inhibitory, or by a combination of both mechanisms.

Keywords: Cat visual cortex, *In vivo* whole-cell recording, Orientation selectivity

Introduction

A number of different hypotheses regarding the neural basis of the orientation selectivity of visual cortical cells have been advanced (Hubel & Wiesel, 1962; Benevento et al., 1972; Creutzfeldt et al., 1974; Shevelev et al., 1974; Sillito, 1975; Morrone et al., 1982; Vidyasagar & Heide, 1984; Leventhal, 1985; Vidyasagar, 1987; Eysel et al., 1990; Wörgötter & Koch, 1991). Even though each of them is based on substantial experimental evidence, they can be assigned into three different groups according to the role proposed for excitation and inhibition: relying exclusively on excitatory mechanisms, or on inhibitory mechanisms, or on a combination of the two. The model of excitatory convergence of the afferents of a number of dorsal lateral geniculate nucleus cells on to a single cortical cell (Hubel & Wiesel, 1962, 1965; Ferster, 1986; Ferster & Jagadeesh, 1992; Chapman et al., 1991) falls in the first category. The second attributes the selectivity to intracortical inhibition acting on a generally orientation nonspecific excitatory input (Benevento et al., 1972; Creutzfeldt et al., 1974; Shevelev et al., 1974; Sillito, 1975, 1984; Morrone et al., 1982). The third group of schemes incorporates both an orientation bias in the excitatory input to visual cortical cells as well as intracortical inhibition (Vidyasagar & Urbas, 1982; Leventhal, 1985; Vidyasagar, 1987, 1992; Ferster & Koch, 1987; Eysel et al., 1990; Wörgötter & Koch, 1991). Each group of hypotheses makes definite predictions regarding the postsynaptic potentials evoked in cortical cells when oriented stimuli are flashed on the receptive field. To test these predictions, we have done intracellular recordings from neurones in cat area 17 using *in vivo* whole-cell recording techniques (Sakmann & Neher, 1983; Xing Pei et al., 1991) and studied the orientation tuning of excitatory and inhibitory postsynaptic potentials. Our results indicate that the orientation selectivity in cat striate cortex does not exclusively depend on any one mechanism, but illustrative examples can be found for all of the three groups of schemes. These data have been reported in a preliminary form (Volgushev et al., 1992*b*).

Methods

Experiments were done on adult cats (2.0–4.5 kg) bred in the animal house of the Max-Planck Institute for Biophysical Chemistry. Anesthesia was induced with ketamine hydrochloride i.m. (Ketanest, Parke-Davis, Berlin, 25 mg/kg i.m.) or with Nembutal (Sanofi, Ceva, 35–40 mg/kg i.p.), and maintained with i.v. infusion of 3–4 mg/kg/h pentobarbitone (Nembutal) without nitrous oxide or with 1–2 mg/kg/h Nembutal and a gas

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mixture of 70% N_2O plus 29.2% O_2 and 0.8% CO_2. Muscle relaxation was induced and maintained with gallamine triethiodide (Flaxedil, Davis & Geck, Pearl River, NY). End-tidal CO_2, body temperature, and EKG were continually monitored. The adequacy of the anesthesia was attested by the stable heart rate and synchronized EEG waves at the doses we used. Animals and the cortex were in good condition for 2–3 days. Application of the techniques used for patch-clamp recordings *in vitro* (Sakmann & Neher, 1983; Edwards et al., 1989) helped us to achieve stable and reliable recordings of postsynaptic potentials (PSPs) from neurones in the primary visual cortex *in vivo*. We used electrodes similar to those used for single-channel recording (Sakmann & Neher, 1983), with a tip diameter of 1–3 μm, pulled with a Patch-Clamp Pipette Puller L/M-3P-A (David Kopf). Their resistance was 2–7 MΩ when filled with a conventional patch pipette solution (Edwards et al., 1989): 130 mM K-gluconate; 5 mM NaCl; 10 mM EGTA; 10 mM HEPES; 1 mM ATP; 1 mM $CaCl_2$; 2 mM $MgCl_2$; pH 7.4 (KOH) (all drugs: Sigma Chemie GmbH, Deisenhofen). An electrode holder through which positive or negative pressures could be applied (Hamill et al., 1981) was connected to a hydraulic microdriver (David Kopf) that was mounted on the skull. On gaining access to the interior of a cell (for details see Xing Pei et al., 1991), stable recordings could be maintained over several hours with membrane potentials of usually −30 to −60 mV and cell resistances of 40–200 MΩ. Only occasionally were membrane potentials more negative than −60 mV. These values are in the range reported for visual cortical cells *in vivo* (Creutzfeldt et al., 1974; Douglas et al., 1991; Ferster & Jagadeesh, 1992). Further details of the experimental procedure and discussion of our recording situation are published elsewhere (Xing Pei et al., 1991).

Visual stimuli from a projector were presented using a computer (PC 386) on a screen positioned 57 cm in front of the animal, whose eyes were focused on the screen using appropriate hard contact lenses. Pupils were dilated by atropine and artificial pupils (3 mm in diameter) were used. Orientation of the stimuli or their position on the screen was changed in pseudorandom order. Receptive fields were classified according to conventional criteria. This was done first with the help of a hand-held projector and then the classification was checked during off-line analysis from the data collected with computer-controlled visual stimuli. Receptive-field profiles were plotted from responses to an optimally oriented bar flashed at 11 positions along the receptive field, or to a small spot (0.5 × 0.5 deg or 1 × 1 deg) flashed in 25 positions of a 5 × 5 matrix. ON and OFF zones were estimated as areas, where a postsynaptic potential was evoked by stimulus onset or offset. For testing orientation tuning, the stimulus was centered on the ON-excitatory zone. In some cases, responses to visual stimuli were recorded during the application of hyperpolarizing or depolarizing intracellular currents (0.1–1.3 nA). Ordinal position of the cell was estimated according to the latency of response to electrical stimulation of the lateral geniculate nucleus (LGN) (0.1 ms, 0.3–3.0 mA). Cells with latencies shorter than 2.0 ms were taken to be monosynaptically activated from the LGN (Bullier & Henry, 1979).

Results

We obtained reliable and stable whole-cell voltage recordings from 72 cells in area 17. Twenty of these were tested by flashing bars of different orientations on the center of the receptive field and in all but one significant PSPs could be evoked by these stimuli.

Forty-five percent of the cells (9/20; five simple, three complex, and one simple-like hypercomplex cell) exhibited excitatory postsynaptic potentials (EPSPs) that were already well tuned to the optimum orientation shown by the spike discharges. There were no significant inhibitory postsynaptic potentials (IPSPs) in the nonoptimum orientation. EPSPs were either not seen or weak in the nonoptimum orientation. As "nonoptimal" we considered the orientation which evoked the weakest excitation, if any, and differed from the optimal by 90 deg or 67 deg.

The two complex cells in Fig. 1 both show sharply tuned EPSPs. A cell shown in Figs. 1A–1C responded well to an optimally oriented stimuli, sometimes even with a burst of spikes (Fig. 1A, upper). Nonoptimally oriented bar flashed in the receptive-field center of this cell failed to evoke any response (Fig. 1A, lower). Excitatory input to this cell was already sharply tuned to orientation (Fig. 1B), and was much sharper than could be expected from the geometry of its excitatory receptive-field profile (Fig. 1C). However, this is not surprising as this cell was not monosynaptically driven from the LGN. The other cell had

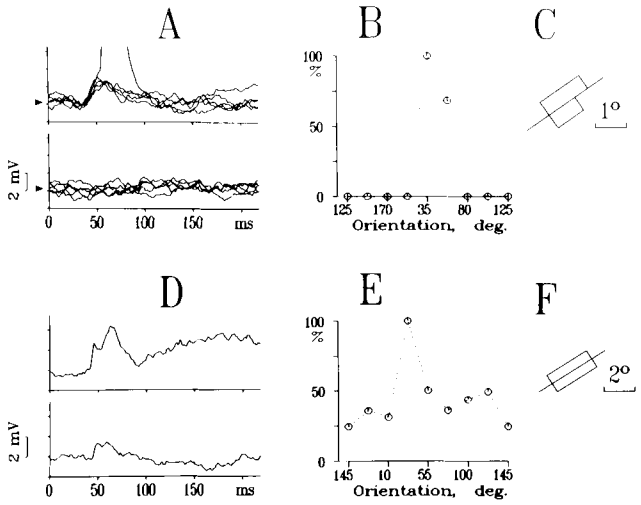

Fig. 1. Orientation sensitivity derived largely from excitatory inputs. Two complex cells, with (D–F) and without (A–C) direct input from the LGN are shown. A,D: PSPs evoked by a bar flashed (1 s ON, 2 s OFF) on the receptive-field center in optimal (upper) and nonoptimal (lower) orientations. In A, responses for five presentations are superimposed. In D, averaged responses ($N = 5$) of another cell are shown for optimal (upper) and nonoptimal (lower) orientations. The time scale (abscissa) starts with stimulus on. B,E: Orientation tuning of PSPs. Abscissa: Orientation of flashed bar. Ordinate: Response strength, normalized to maximum. For this and following tuning curves, response strength was estimated within a certain temporal interval (window) as the integral area of averaged PSP which differed from the mean resting membrane potential by more than one standard deviation. Readings made just before each stimulus were used to calculate mean resting potential and its standard deviation. Window for measuring response strength in A was 35–75 ms and in B was 45–65 ms. Resting potentials were −50 ± 2.6 mV (A, dotted line and arrowheads) and −30 ± 1.65 mV (D, dotted line). C,F: ON region of receptive field giving excitatory responses when plotted with a 0.5 × 0.5-deg spot. Optimal orientation is shown by the line through the receptive field. OFF region was overlapping the ON region for both cells. Latencies to LGN stimulation were 5.8 ms (cell A–C) and 1.8 ms (cell D–F).

a latency of 1.8 ms to LGN stimulation and thus driven monosynaptically (Hoffmann & Stone, 1971; Bullier & Henry, 1979). Strongest responses were evoked in this cell by stimuli whose orientation coincided with the long axis of the excitatory receptive field. Other orientations evoked weaker, but clearly distinguishable responses. We also recorded from a monosynaptically driven simple cell which showed PSP responses like those in Fig. 1. Fifteen percent of cells (3/20) were monosynaptically driven, and belonged to this group.

Fig. 2 shows a cell whose EPSPs were very broadly tuned to orientation, and whose selectivity was largely due to its inhibitory input. This cell had a simple receptive field and was monosynaptically driven from the LGN (latency 2.0). Optimally oriented stimuli evoked in the cell a strong excitatory response always leading to a burst of spikes. With nonoptimal orientation, some initial excitation can also be distinguished, but it was sharply cutoff and did not evoke spikes. This suggests that inhibition may be activated by nonoptimally oriented stimuli. Indeed, on applying a hyperpolarizing current that suppresses spikes and reduces or removes the effects of the IPSPs, there is little difference between the first burst of EPSPs evoked by the optimal and nonoptimal orientations (Fig. 2B). Therefore, both optimal and nonoptimal orientations produced nearly the same excitatory input to the cell, but inhibition seems to prevent the development of excitatory response to nonoptimally oriented stimuli. It should be noted here that some inhibition might be present also between the two bursts evoked by optimally oriented stimuli. But this is likely to be largely recurrent inhibition since it was stronger when the number of discharges in the first burst was greater and thus contributing little to the orientation selectivity of the first burst. Another interesting point to note is that the second excitatory component of the response (around 120–150 ms), which was not affected by the hyperpolarization and although of a cortical origin, had the same orientation preference as the first excitatory burst.

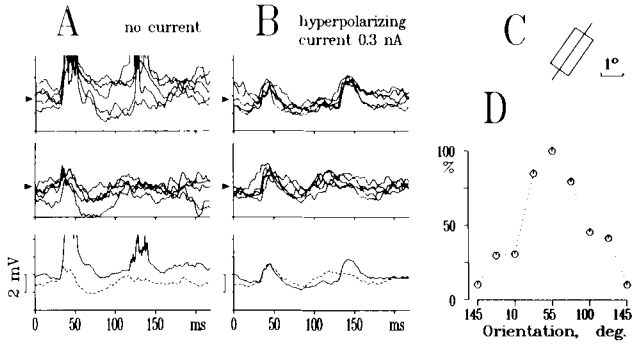

Fig. 2. Orientation sensitivity derived largely from inhibitory inputs in a simple cell. A: PSPs evoked in the cell by stimuli of optimal (upper traces) and nonoptimal (middle traces) orientations. Traces at the bottom are averaged responses in the optimum (solid line) and nonoptimum (broken line) orientations. Resting membrane potential of this cell was -35 ± 1.2 mV. B: Responses to the same stimuli as in A, but with 0.3 nA of hyperpolarizing current. Strength of the current was adjusted to the threshold value when no more hyperpolarization could be evoked by stimulating the inhibitory zone of the receptive field. Under these conditions, the PSPs evoked in the two orientations are very similar. C: Profile of ON region of the receptive field. D: Orientation tuning of the PSPs, measured within a window 25–70 ms. Latency to LGN stimulation was 2.0 ms. Conventions are as in Fig. 1.

Ten percent of the cells (2/20; one simple and one complex, both monosynaptically driven from the LGN) had an excitatory input that was either nonoriented or only poorly tuned to orientation but with a tuned inhibitory input. Cells of this type support the inhibitory scheme of orientation selectivity (Benevento et al., 1972; Creutzfeldt et al., 1974; Shevelev et al., 1974; Sillito, 1975, 1984).

Fig. 3 provides an example of a cell in which both excitatory and inhibitory inputs were responsible for the final orientation selectivity. In this simple cell with a direct monosynaptic drive from the LGN, EPSP amplitudes are highest at the optimal orientation. Primary IPSP amplitudes (defined as those occurring within 75 ms) appear to be weakest at this orientation and at the orthogonal orientation they are most pronounced. The late inhibition, measured after 100 ms, however, could be strong even at the optimal orientation. But excitation and primary inhibition are tuned to quite different, almost orthogonal, orientations. From this data, it is not possible to assess with certainty the tuning of EPSPs, which might in fact be much broader if the strong cross-orientation inhibition can be eliminated.

Forty percent of the cells (8/20, five simple, one complex, and two simple-like hypercomplex; four of these cells were first order) exhibited primary inhibition, which was tuned to an orientation quite different from the optimum orientation of the EPSPs. This group of cells supports the scheme where both excitatory and inhibitory inputs contribute to the final orientation selectivity of the cortical cell (Vidyasagar, 1987, 1992; Ferster & Koch, 1987; Eysel et al., 1990; Wörgötter & Koch, 1991).

Discussion

Two main conclusions may be drawn from our data. First, both excitatory and primary inhibitory input to the cell can be orientation selective. Second, if both of them are orientation selective, then they have different "preferred" orientations. Maximal excitation and strongest inhibition are most often evoked by stimuli of different orientations. Therefore, orientation selectivity of a particular cell might be created by excitatory mechanisms, by inhibitory mechanisms, or by a combination of both.

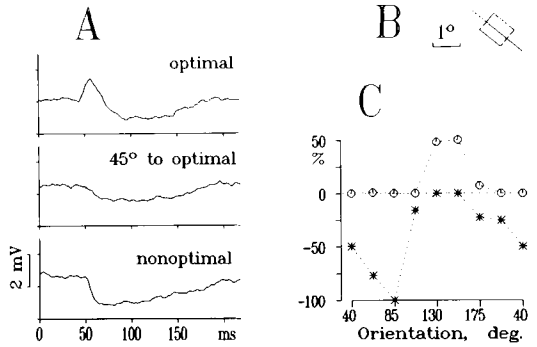

Fig. 3. Orientation sensitivity derived from both excitatory and inhibitory inputs in a simple cell. A: Averaged PSPs evoked in the cell by stimuli of optimal (upper), 45 deg to optimal (middle) and nonoptimal (lower) orientations. Resting membrane potential of this cell was -30 ± 0.83 mV. B: Profile of ON region of receptive field. C: Orientation tuning of excitatory (circles) and primary inhibitory (asterisks) PSPs, both measured within a window 50–75 ms. Conventions are as in Fig. 1.

We estimated orientation selectivity from the early, primary components of a cell's response. Thus, we considered as "primary," events occurring within a latency comparable to that of the spike response (within 5–10 ms), with some of them obviously affecting the spikes. Other excitatory and inhibitory influences, occurring after another 30–50 ms or later, were not considered here. They might contribute, for example, to directional selectivity of a cell.

The majority of neurones in our sample have orientation-dependent excitatory inputs, as can be concluded from orientation dependence of excitatory PSPs. The tuned excitatory input is likely to be due to excitatory convergence as proposed by Hubel and Wiesel (1962, 1965) and/or due to a biased geniculate input (Vidyasagar & Urbas, 1982; Leventhal, 1985; Vidyasagar, 1987, 1992). It provides a major framework for the orientation selectivity in the visual cortex and evidence for the excitatory models of orientation selectivity. However, a pure excitatory mechanism, without participation of inhibition, seems to be more characteristic of the second-order than of the first-order cells. At least all cells with a disynaptic or polysynaptic input from the LGN belonged to this category. Among second-order cells, therefore, orientation selectivity was achieved by an excitatory input tuned to orientation, with inhibition playing no part. On the other hand, in only a minority of first-order cells (latency of 2.0 ms or less), we fail to see inhibitory potentials associated with stimuli of nonoptimal orientation. Their excitatory inputs may be tuned either due to an excitatory convergence from LGN afferents (Hubel & Wiesel, 1962, 1965; Chapman et al., 1991) or due to inputs from an LGN field already biased for orientation (Vidyasagar, 1987). However, if the inputs were only biased for orientation like single LGN cells, then the sharp orientation tuning and highly elongated receptive fields can be explained only by assuming an inhibitory input that is of a shunting nature (Koch & Poggio, 1985) and not visible in soma recordings as IPSPs (Vidyasagar, 1987). If indeed shunting inhibition does not operate in the cat striate cortex as it has been suggested in some papers (Douglas et al., 1988, 1991; Berman et al., 1991; Ferster & Jagadeesh, 1992), then cells such as these provide support for an excitatory convergence of the type proposed by Hubel and Wiesel (1962, 1965).

Orientation selectivity of the inhibitory input to a number of cells in our sample and the cross-orientation nature of primary inhibition support the schemes that rely on inhibitory mechanisms of orientation selectivity (Benevento et al., 1972; Creutzfeldt et al., 1974; Shevelev et al., 1974; Sillito, 1975, 1984; Morrone et al., 1982; Vidyasagar & Heide, 1984; Koch & Poggio, 1985; Eysel et al., 1990), showing that inhibition often sharpens, and sometimes even creates, the orientation selectivity of the cell. For example, the excitatory input the cell in Fig. 2 receives is almost equal during stimulation by stimuli of either optimal or nonoptimal orientation, as seen in the similarity of evoked responses when effects of inhibition on the membrane potential were removed by hyperpolarization of the cell. Therefore, orientation selectivity of this cell in the normal, nonpolarized state could have been created only by inhibition, that prevents the development of excitatory response to nonoptimal stimuli, leading to spikes. However, such a situation, where the excitatory input to the cell was not tuned to orientation at all, was not very common in our sample. More often, excitation and inhibition complemented one another, with inhibition sharpening orientation selectivity already present in the excitatory input. In such a case, inhibition and excitation would be tuned to (i.e. maximal at) different orientations. We did indeed find that primary inhibition, when apparent, was tuned to orientation different by 45–90 deg from the optimal for excitation.

The difference in the effects of removal of GABAergic inhibition on orientation selectivity of simple and complex cells (Sillito, 1975, 1984) suggests that inhibitory mechanisms may be more important for orientation selectivity of simple cells. But we found no significant correlation between the presence of primary orientation-selective inhibition and receptive-field type, as was also found in another study (Eysel et al., 1990).

Contrary to recent reports based on responses to moving stimuli (Ferster, 1986; Douglas et al., 1988; Ferster & Jagadeesh, 1992), we have found primary inhibition contributing to orientation selectivity in at least half of the cells in cortex, and among first-order cells, the percentage is even higher (6/9 cells, 66%). This discrepancy is likely to be related to our use of flashing stimuli, which avoids the successive activation of different zones of the receptive field as well as intracortical interactions induced by moving stimuli. Flashing stimuli permit the analysis of individual temporal components of responses evoked from a specific region of the receptive field.

In conclusion, orientation selectivity of striate cortical neurons primarily arises from the balance of excitatory and inhibitory influences on the cell, and can be enhanced by amplification of the resultant excitation by intracortical circuitry (Douglas et al., 1991) or by the involvement of voltage-dependent channels (Volgushev et al., 1992*a*). Thus, the cat striate cortex exhibits a variety of mechanisms to achieve orientation selectivity. In a particular cell, orientation selectivity can be generated by purely excitatory mechanisms, largely by inhibitory mechanisms, or by a combination of the two.

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