

Research report

# Non-dominant eye responses in the dorsal lateral geniculate nucleus of the cat: an intracellular study

Yifeng Zhou<sup>a,c,\*</sup>, Hongbo Yu<sup>a,1</sup>, Yupeng Yang<sup>a</sup>, Tiande Shou<sup>b,c</sup>

<sup>a</sup>Vision Research Laboratory, School of Life Sciences, University of Science and Technology of China, Hefei, Anhui 230027, China

<sup>b</sup>Center for Brain Science Research and Liren Lab, School of Life Sciences, Fudan University, Shanghai 200433, China

<sup>c</sup>Visual Information Processing Laboratory, Biophysical Institute of the Chinese Academy of Sciences, Beijing 100101, China

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## Abstract

While binocularity has been established as an important characteristic of cat visual cortical neurons, neurons in the dorsal lateral geniculate nucleus (LGNd) are commonly believed to be monocular. To test whether binocularity exists at the level of the LGNd, postsynaptic potentials (PSPs) of 101 cells were intracellularly recorded in eight normal and eight monocularly deprived cats while presenting stimuli to either the dominant or non-dominant eyes. The results showed that: (1) About 92% of neurons (45 out of 49) responded to a flashing spot presented to the non-dominant eye. In contrast to the dominant eye responses, the non-dominant eye PSPs usually exhibited the same polarization tendency (hyperpolarization or depolarization) to flashing spot stimuli of light increment or decrement, and most of them were inhibitory (hyperpolarization, 35 out of 45, 78%). (2) The response field (RF) of the non-dominant eye overlapped that of the dominant eye. (3) For most binocular cells, peak-to-peak amplitudes of non-dominant eye PSPs were about half the size (46%) of those of the dominant eye. The peak latencies and half-peak latencies of non-dominant eye PSPs were significantly longer than those of the dominant eye (mean differences were 5.4 ms and 5.6 ms respectively). (4) Most of the binocular cells responded well to contrast reversing gratings presented to the non-dominant eye, and the responses were clearly spatial-frequency tuned. No null phase could be found for non-dominant eye PSPs, no matter the neuron was classified as X or Y type according to dominant eye elicited responses. Some of the cells responded well to drifting gratings presented to the non-dominant eye. (5) We also recorded 52 cells in monocularly deprived cats, and found that 49 cells (94%) showed significant responses to flashing spots presented to the non-dominant eye, a similar percentage to that found in normal cats (92%). Conclusion: as strongly monocular neurons, most of LGNd cells could also be driven by the non-dominant eye. The responses evoked by non-dominant eye stimulation differ greatly from those evoked by dominant eye stimulation, and remain intact even without visual experience. These observations suggest an important role of the perigeniculate nucleus in providing binocular inputs to LGNd cells.

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## 1. Introduction

Excitatory responses of cells in the cat dorsal lateral geniculate nucleus (LGNd) are generally considered to be monocular, and binocular neurons mainly exist in the

visual cortex [3,16,18]. However, extensive evidence has shown that binocular interaction also occurs at the level of LGNd [2,23,25–27,30,33,37,46,52,55]. The responses of LGNd cells to non-dominant eye stimulation are mainly inhibitory [30,37,50], although evidence for excitatory inputs from the non-dominant eye could also be found [12,27]. The binocularity of LGNd neurons may result from the extensive corticofugal projections [4,14,15,36,43,51]. This projection provides the greatest number of inputs to the LGNd, which exceeded retinal

\*Corresponding author. Tel.: +86-551-360-1436; fax: +86-551-360-7014.

E-mail address: [zhouy@ustc.edu.cn](mailto:zhouy@ustc.edu.cn) (Y. Zhou).

<sup>1</sup>These two authors had the equal contribution.

afferents by an estimated factor of ten [40]. Furthermore, the recurrent feedback from the perigeniculate nucleus, which is considered to be binocularly driven [8,54], and feedforward inhibition from the intrageniculate interneurons may also contribute to the binocularity of cells in the LGNd [7,19,55]. Beyond the research on pathways, further investigations have been made on the response field properties of the non-dominant eye and the response properties to grating stimuli [26,34,47].

However, previous works have approached the above issue almost exclusively by using extracellular recording technique. Although some intracellular analysis was performed by Lindstrom et al. [24], the cells analyzed were all from interlaminar layers. Further, extracellular results were based on the analysis of the peristimulus time histograms (PSTHs), obtained from relatively prolonged periods of averaging. It is difficult for this extracellular method to measure the response properties of cells, such as amplitude and latency of a cell's weak response, especially when most of the responses are inhibitory. Usually, the background firing rates must be manually raised, in order to be able to detect inhibitory effects, which may cause the effect from dominant eye to non-dominant eye [50]. In the present study, we recorded postsynaptic potentials (PSPs) of LGNd cells in vivo for up to 3 h with patch clamp electrodes. This technique has been previously used in the visual cortex [10,28,31,58]. We compared PSPs evoked by stimuli presented to the non-dominant eye to those evoked by stimuli presented to the dominant eye, for using different kinds of visual stimuli (such as stationary flashing spots, reverse gratings, and drifting gratings). We also examined the non-dominant eye responses of LGNd cells in monocularly deprived cats to test the possible influence of visual experience on binocularity in LGNd.

## 2. Materials and methods

### 2.1. Animal preparation

Normal adult cats (eight) and monocularly deprived cats (eight) were used in our experiments. For monocular deprivation, we sutured one of the eyelids of postnatal kittens before eye opening and raised them for 10–12 months. The detailed methods for general preparation have been described in earlier publications [41,56]. Briefly, cats (2.5–3.8 kg) were initially anesthetized with ketamine (20 mg/kg). Intravenous and tracheal cannulae were inserted. Animals were placed in a stereotaxic apparatus (Nashiage, Japan), and all the pressure points were treated with lidocaine (1%). For the remainder of the experiment, light anesthesia was maintained with intravenous infusion of urethane given at an initial dose of 30 mg/kg, followed by continuous infusion of 20 mg/kg per h. This was supplemented with gallamine triethiodide (Flaxedil, 8–10 mg/kg/h) for paralysis. The ECG and EEG were continuously

recorded to monitor the level of the anesthesia. Neosynephrine (5%) was administered to retract the nictitating membranes. Pupils were maximally dilated with atropine sulphate (1%), and appropriate contact lenses were used to protect the cornea. Spectacle lenses were used for correction when needed. The animal's rectal temperature and end-tidal CO<sub>2</sub> were routinely monitored and kept within normal ranges.

The optic disks were projected repeatedly upon a tangent screen positioned 114 cm from the cat's eyes during the course of each recording session and were used to locate the positions of the area centralis. The clarity of the optics was checked routinely during all experiments.

### 2.2. Recording

The intracellular recording technique used in the present study is similar to that described in earlier studies [31,58]. Extracellular action potentials and intracellular postsynaptic membrane potentials were recorded from the LGNd cell by the same patch electrode. The electrode (tip diameter of 1–2 μm) was filled with a solution contained (mM): 130 K-gluconate, 5 NaCl, 10 EGTA, 10 Hepes, 1 ATP, 1 CaCl<sub>2</sub>, 2 MgCl<sub>2</sub>, pH 7.4 (KOH). The impedance of the electrode was 6–15 MΩ. Signals from the electrode were sent to the intracellular recording amplifier (Nihon MEZ-8201, Japan), and then fed into a window discriminator while recording extracellularly, which produced digital signals that were registered by a Compaq-486 computer. To ensure the accurate isolation of a single unit, the spike waveform was displayed on an oscilloscope to monitor its shape and time course. While recording intracellularly, postsynaptic membrane potentials were amplified, filtered (0–10 kHz), digitized and fed into a computer for further analysis.

### 2.3. Visual stimuli and receptive field mapping

The visual stimuli were usually flashing light circles (luminance of about 12.0 cd/m<sup>2</sup> with the background luminance of about 2.0 cd/m<sup>2</sup>) with different diameters and positions, as well as drifting sinusoidal and contrast-reversing squarewave gratings with mean luminance of 7.0 cd/m<sup>2</sup> and contrast of 0.5. The spatial frequency, temporal frequency and orientation of the gratings could be changed according to the properties of the cell being examined.

The stimuli were controlled by a computer running the Visual Stimulation System (CED, UK) program, and were displayed on an Innisfree 'Picasso' oscilloscope-based (Tektronix 608) optical display with a screen of 18×15 square degrees. The oscilloscope could be tangentially moved to any point in the animal's visual field, while maintaining a fixed distance of 38 cm (2 cm on screen equals to 3° of visual angle) between the display and the animal's eyes. Thus, we were able to study cells whose receptive field subtended any part of the visual field.

The receptive field centers of all the cells we recorded were located within  $10^\circ$  of the area centralis. The response fields of the non-dominant eye were first approximated according to the receptive fields of dominant eye, and then further refined during later recording. In most cases, the refined response fields overlapped significantly with the approximated ones.

#### 2.4. Experimental protocol and data analysis

Extracellular recording was obtained first. Receptive fields of isolated units were mapped by a handheld projector on a tangential white screen that was 114 cm from the cat's eyes, which were categorized as on- or off-center. The ocular dominance and the layer (A, A1, C) in which the cell located were determined. Cells were classified as X- and Y-types using the methods described previously [5,9,17,45]. After successful extracellular recording of an LGNd cell, we tried to obtain a stable intracellular recording by applying negative pressure and brief electrical shocks. The cell's membrane potential response to visual stimulation was measured while the resting potential was maintained at a level between  $-30$  and  $-60$  mV. In some cases, the electrode penetrations were reconstructed as described previously [42,57]. Special care was taken in distinguishing PGN cells from LGNd cells during the experiments. PGN cells are located above the LGNd, considered to be binocularly excited driven [8,34], and have on-off response to flashing stimuli as well [54].

The analysis of extracellular data was similar to that described in Shou et al. [41]. For the intracellular data, spikes were removed before the membrane potentials were averaged ( $n=20-100$ ). The peak-to-peak values of the averaged membrane potentials were taken as the cells' response amplitudes. The latencies of peaks and half-peaks were also measured.

### 3. Results

Forty-nine cells in the LGNd of normal cats were recorded intracellularly and extracellularly and quantitatively analyzed for investigation of binocular properties in LGNd of normal cats. Among these cells, 15 were in layer A, 27 in layer A1, and 7 in layer C (usually at the top of layer C). Twenty-two cells were on-center type and 25 cells were off-center type. Of the 45 identified cells, 14 were classified as Y cells and 31 as X cells.

#### 3.1. PSTHs and PSPs evoked by the non-dominant eye

Out of a population of 49 cells, 45 (92%) showed clear responses to flashing spot stimuli (on and off duration of 500 ms respectively, diameter  $3-13^\circ$ ) presented to the non-dominant eye. The remaining four (8%) had no or

unclear responses. Among those cells showing clear binocular responses, 35 (out of 45, 78%) were hyperpolarizing, seven (16%) were depolarizing, and three (6%) were mixed. These binocular cells could be different types (on, off, X, Y) and locate in different layers of the LGNd (A, A1 and C).

Fig. 1 shows the responses from an off-center X cell (Fig. 1A–D), recorded from layer A1, and an off-center Y cell from layer A (Fig. 1E–H). The dominant eye elicited PSPs showed a prominent hyperpolarized plateau throughout the on-light period for the X cell (Fig. 1B) but not for the Y cell (Fig. 1F), although there were transient hyperpolarized or depolarized PSPs following the onset and offset of stimuli for both cells. In contrast, when the same stimuli were displayed at the corresponding response fields of the non-dominant eye, both cells (Fig. 1C,G) showed transient hyperpolarized PSPs after the onset and offset of the stimuli. The hyperpolarized PSP duration was longer for the X cell than for the Y cell. As controls, when both eyes were covered, no prominent hyperpolarized or depolarized PSPs could be detected (Fig. 1A,E). For comparison, the averaged PSTHs obtained extracellularly from the same cells when the non-dominant eye was stimulated are shown (Fig. 1D,H). A clear decrease in the firing rate (inhibition, Fig. 1D) was evoked by the onset and offset of the light stimuli in the X cell. However, no obvious decrease was observed in the Y cell (Fig. 1H), even though the same stimulus evoked a hyperpolarized PSP response (Fig. 1G) in the Y cell. It should be mentioned that the lack of effects on firing rates of non-dominant eye stimulation was not cell-type-specific.

Unlike dominant eye responses, the non-dominant eye PSPs usually exhibited the same polarization (depolarized or hyperpolarized tendencies) for both the onset and offset of flashing light stimuli. Furthermore, almost all non-dominant eye PSPs (43 of 45) were transient for both X and Y cells.

#### 3.2. Response field of the non-dominant eye

LGNd neurons had non-dominant eye response fields whose positions overlapped the receptive field of the dominant eye. In 18 cells, we presented a small flashing spots, at various positions on CRT to the non-dominant eye. As the position of the flashing spot was moved away from the center of the response field, the peak-to-peak amplitudes of the PSPs decreased steadily, while the waveforms and latencies changed little (Fig. 2). It is notable that in no case did the PSPs reverse polarity with increasing eccentricity of the spot. This indicates that non-dominant eye response fields differ from the center-surround antagonistic structures of dominant eye receptive fields. Furthermore, the response field of the non-dominant eye was relatively large (diameter  $>8^\circ$ ) compared with the classical receptive field size of LGNd cells ( $1-2^\circ$ ).

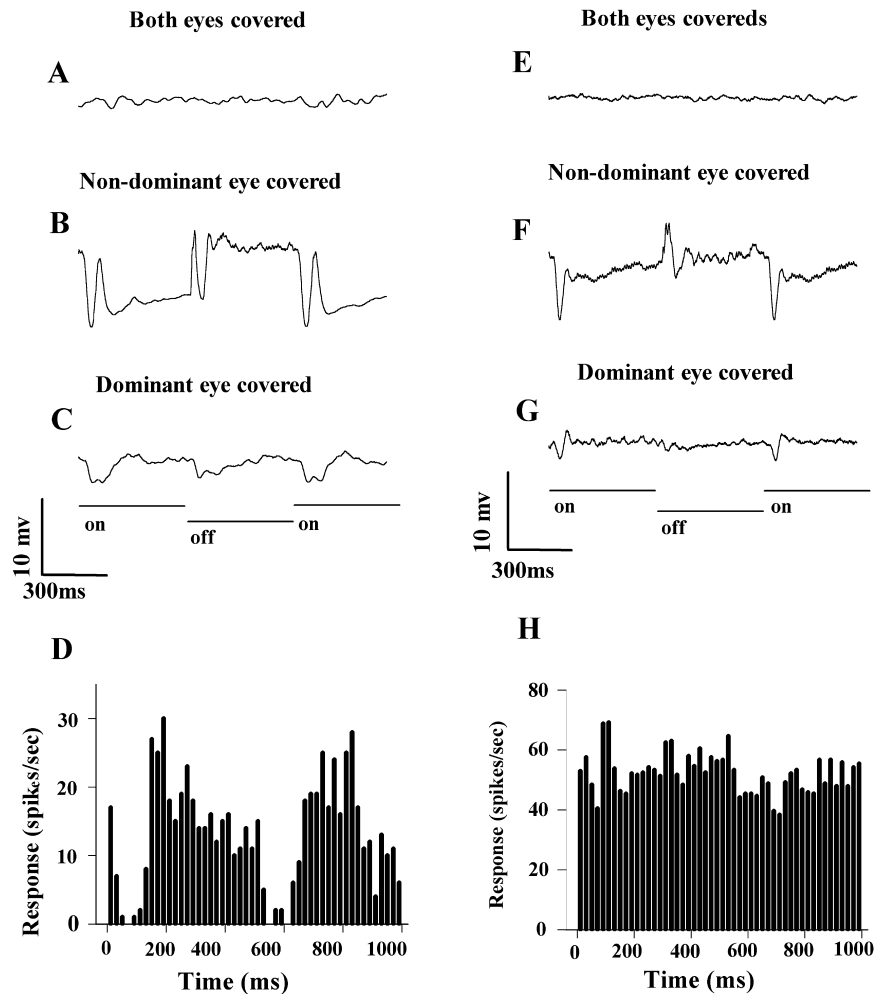


Fig. 1. Averaged PSPs and PSTHs evoked by flashing spot stimuli (on and off duration were each 500 ms, spot diameter=6° for A–D, 9° for E–H) for two cells of the LGNd. PSPs were averaged for 80 trials, and PSTHs for 100 trials. (A–D) Responses of an off-center X cell in layer A1, the cell's resting potential was  $-39$  mv. (E–H) Responses of an off-center Y cell in layer A, the cell's resting potential was  $-48$  mv. (A and E) Contrast PSPs (covering both eyes); (B and F) PSPs in response to dominant eye stimulation (covering non-dominant eye); (C and G) PSPs in response to non-dominant eye stimulation (covering dominant eye); (D and H) PSTHs of spikes recorded extracellularly (covering dominant eye). A clear inhibitory response (D) was evoked by the onset and offset of the light stimuli for this X cell. No inhibitory response to the stimulus, which had evoked hyperpolarized PSP response to the non-dominant eye (shown in G), could be observed in the Y cell (H).

### 3.3. Latency and amplitude of evoked PSPs

The peak-to-peak amplitudes of PSPs were measured in response to flashing spots presented to either the dominant eye or non-dominant eye. The non-dominant eye responses were always weaker than dominant eye responses (Fig. 3A). Comparing the ratio of responses between the two eyes (amplitude of the non-dominant eye/amplitude of the dominant eye, Fig. 3B), we found that eight out of 34 cells (24%) showed weak binocular responses (the response ratio  $< 0.25$ ), 17 cells (47%) had medium binocular responses ( $0.25$ – $0.7$ ), and the remaining nine cells (29%) had strong binocular responses (ratio  $> 0.7$ ). The mean ratio was  $0.46$ . We also measured the peak latencies of evoked PSPs for 33 cells (Fig. 3C), and half-peak latencies for 28 cells (Fig. 3D). Comparing these results between stimulation of the two eyes, we found that the non-dominant eye

latencies of most cells were longer than those of the dominant eye (Fig. 3C,D). The mean latency of the non-dominant eye PSPs was significantly longer than that of the dominant eye PSPs, with a difference of  $5.6$  ms in half-peak latency and  $5.4$  ms in peak latency ( $t$ -test,  $P < 0.002$  and  $P < 0.05$  respectively).

### 3.4. PSPs evoked by grating stimuli presented to the non-dominant eye

We also used contrast-reversing squarewave gratings to assess the binocularity of cells in the LGNd, and the effect of the grating spatial phase on five X cells. The spatial phase of gratings presented to the non-dominant eye did not affect the shape of PSPs. For the dominant eye PSPs (Fig. 4A–F), from an on-center X cell, a clear reversal of the polarity appeared at the phase of  $75^\circ$ , which was

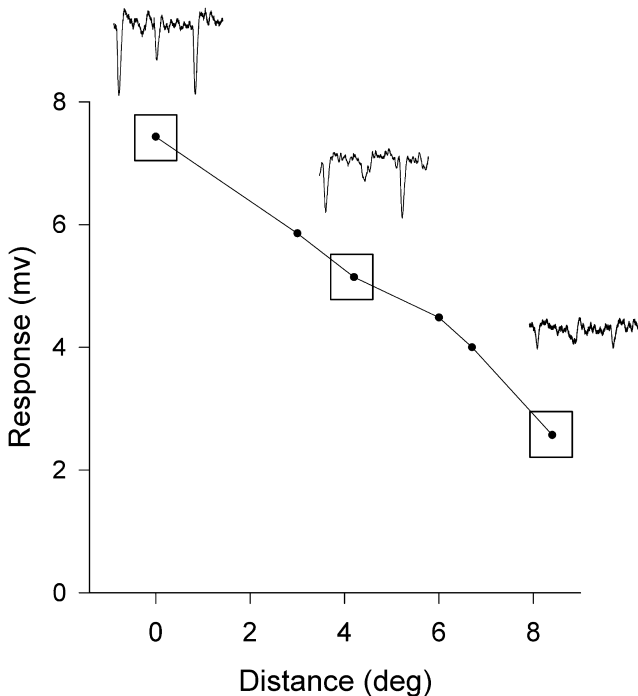


Fig. 2. Tuning curve for the averaged PSPs of an on-center X cell in layer A evoked by a flashing spot (temporal frequency 1 Hz, diameter =  $3^\circ$ ) presented at different distances from the center of the response field of the non-dominant eye. Three typical traces of the PSPs are shown above the curve, and their peak-to-peak amplitudes are represented as filled points in the three-dashed square, respectively.

defined as the null position. The responses during the first 500 ms became more depolarized when decreasing the spatial phase from  $75^\circ$ , and more hyperpolarized when increasing the spatial phase. In contrast, when stimulating non-dominant eye alone, no null position could be observed from the same cell (Fig. 4G–J), although the amplitudes of the hyperpolarized PSPs varied along with the shift of spatial phase.

When using contrast-reversing squarewave gratings, 19 out of 26 cells (73%) showed prominent non-dominant eye PSPs. They were spatial-frequency tuned with generally low (0.5–0.8 cycle/degree) cut-off spatial frequencies. There were also a few cases with relatively high cut-off spatial frequencies. A typical example is shown in Fig. 5A–F. The waveforms of the PSPs were similar for different spatial frequency stimuli, with the optimal spatial frequency around 0.35 cycle/degree. Within a fairly broad range (0.14–2.0 cycles/degree), the response amplitudes sustained a relatively high level ( $>4$  mV), and decreased sharply near the cut-off spatial frequency (about 2.7 cycles/degree, unfilled circles in Fig. 5K).

We also tested the response properties of cells using drifting sinusoidal gratings presented to the non-dominant eye. Fifty-four percent of cells (seven out of 13) showed sinusoidal PSPs to drifting sinusoidal gratings and their cut-off spatial frequency were relatively low ( $<0.5$  cycle/degree, Fig. 5G–J). Other six cells did not show clear

responses to drifting sinusoidal gratings. In contrast to the cut-off spatial frequency of about 2.7 cycles/degree for contrast-reversing squarewave gratings (unfilled circles in Fig. 5K), the cut-off frequency for the same cell in response to drifting sinusoidal gratings (unfilled squares in Fig. 5K) was fairly low (about 0.5 cycle/degree). Out of these seven cells, none had a higher cut-off spatial frequency for drifting gratings than for contrast-reversing squarewave gratings.

### 3.5. Monocularly deprived cats

We also recorded 52 cells from LGNd in monocularly deprived cats. Forty-nine cells (94%) showed significant non-dominant eye PSPs evoked by flashing spots, similar to the percentage found in normal cats (92%). Most of the cells (43 out of 49) showed hyperpolarized and transient PSPs, whereas four cells showed depolarized PSPs. No significant difference between the LGNd cells in the deprived and non-deprived layers (A, A1) were found. This suggested that monocular deprivation did not affect the response of LGNd neurons evoked by flashing stimuli presented to the non-dominant eye.

However, no neurons (out of nine) in the deprived layers responded to drifting sinusoidal grating stimuli presented to the non-dominant eye, while half of the neurons (eight out of 17) in the non-deprived layers did. Comparing these results with those obtained in normal cats (seven cells out of 13 cells responded to the drifting sinusoidal grating stimuli presented to the non-dominant eye), it suggests that visual experience might be necessary for the development of grating responses to non-dominant eye stimuli.

## 4. Discussion

Previous studies have investigated the binocularity of LGNd neurons almost exclusively with extracellular recordings [2,12,25,27,33,37,46,52], a method which has several limitations. Since most of the responses elicited by non-dominant eye stimuli are inhibitory [26,30,37,47,50], it is hard to detect from looking for a decrease of the spontaneous discharge rate, especially when this rate is very low. In some previous studies [22,34,35,44,50], the baseline was manually increased by presenting a stimulus to the dominant eye, and then detecting the dip effect (a decrease in the maintained discharge rate). However, this method is problematic. The response properties obtained in this way come from the influence of two sources rather than purely from the non-dominant eye. In fact, Wang et al. [50] showed that increasing the maintained discharge rate enhanced the dip effect nonlinearly, while Pape and Eysel [30] demonstrated that the discharge evoked by the dominant eye stimuli could diminish the inhibition from the non-dominant eye. Thus, it is interesting to re-evaluate the response properties of LGNd neurons elicited from non-

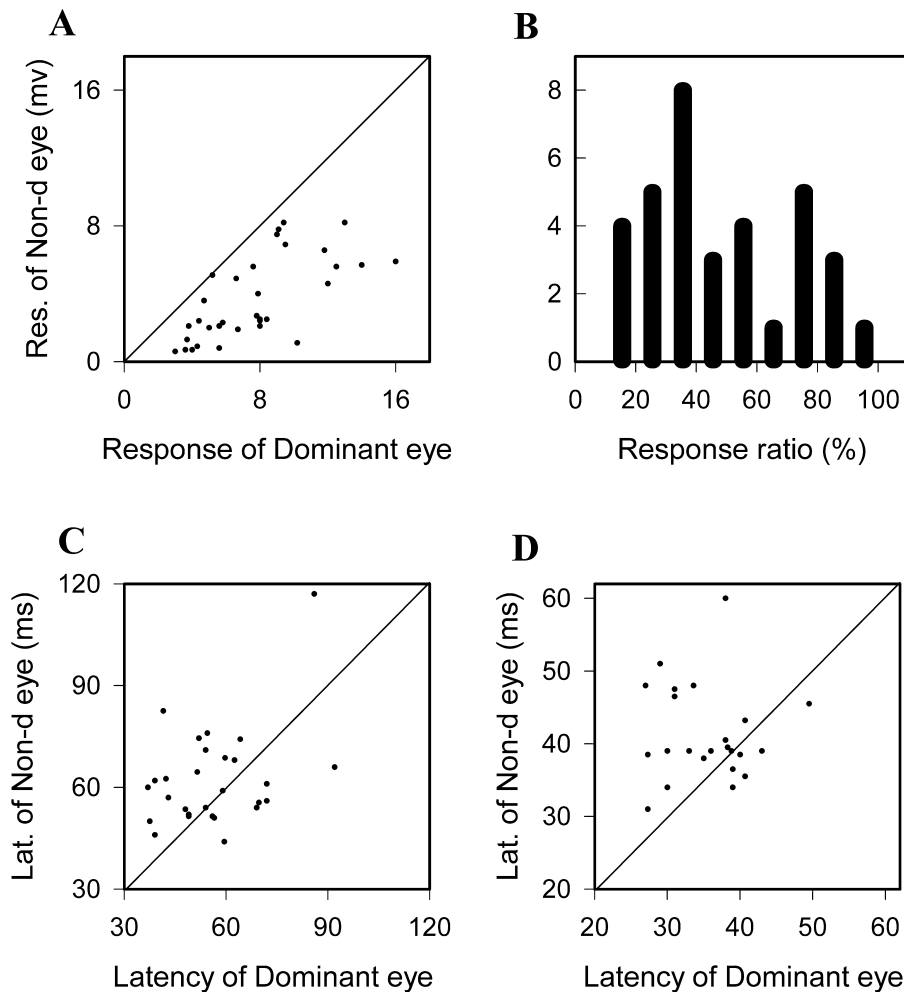


Fig. 3. Distribution of amplitudes and latencies of binocular LGNd cell responses. Flashing spots were used as stimuli (temporal frequency 1 Hz, diameter=3–13°). (A) Comparison of peak-to-peak amplitudes between dominant eye (*X*-axis) and non-dominant eye (*Y*-axis) PSPs; (B) distribution of the non-dominant/dominant eye response ratio (amplitude of the non-dominant eye/amplitude of the dominant eye); Comparison of peak latencies (C) and half-peak latencies (D) between dominant eye (*X*-axis) and the non-dominant eye (*Y*-axis) stimulation. In (A), all of the points lie below the line with slope of 1, indicating that the responses to the non-dominant eye were always weaker than those to the dominant eye. In (C and D), the points below the line with slope of 1 represent those cells with longer latency responses to dominant eye stimulation, whereas points above the line represent those cells with longer latencies to non-dominant eye stimulation. Notice that most cells have longer latencies to non-dominant eye stimulation.

dominant eye stimulation using a different method. Intracellular recorded PSPs provide a direct and objective way to approach this issue. Strong binocularity of neurons in LGNd [24] has been demonstrated using intracellular recordings in five interlaminar cells, which connect with both layer A and A1. However, the binocularity of the cells classified as strongly monocular cells (majority of LGNd neurons) is still not well understood.

Our intracellular recording results demonstrate that almost all the neurons (at least 92%) in the LGNd receive binocular afferents. This ratio is higher than that found in previous works (82% for Sanderson [34]; 75% for Murphy and Sillito [27]; and also see 55% for Guido et al. [12]; 41% for Sengpiel et al. [37]). The different ratios found between our work and previous works might come from methodological differences. The high sensitivity of our intracellular recordings might help to detect weak re-

sponses to non-dominant eye stimuli when the responses are inhibitory and background firing rates are low. This would lead to a higher estimate than in previous studies of the percentage of neurons receiving binocular afferents. Our data suggest that binocularity is a general property in LGNd. The size of the non-dominant eye response was large enough to modulate the binocularity of a neuron. The mean peak-to-peak value of PSPs evoked from non-dominant eye stimulation were about half the size of those elicited by the dominant eye, and thus could underlie the 20–70% decrease of the maintained discharge rate found in previous extracellular recording works [13,22,33,34,50].

Our results confirm that the positions of the response fields of LGNd cells in response to non-dominant eye stimulation match the dominant eye receptive fields. However, in contrast to the dominant eye receptive fields, the non-dominant eye response fields have no center-

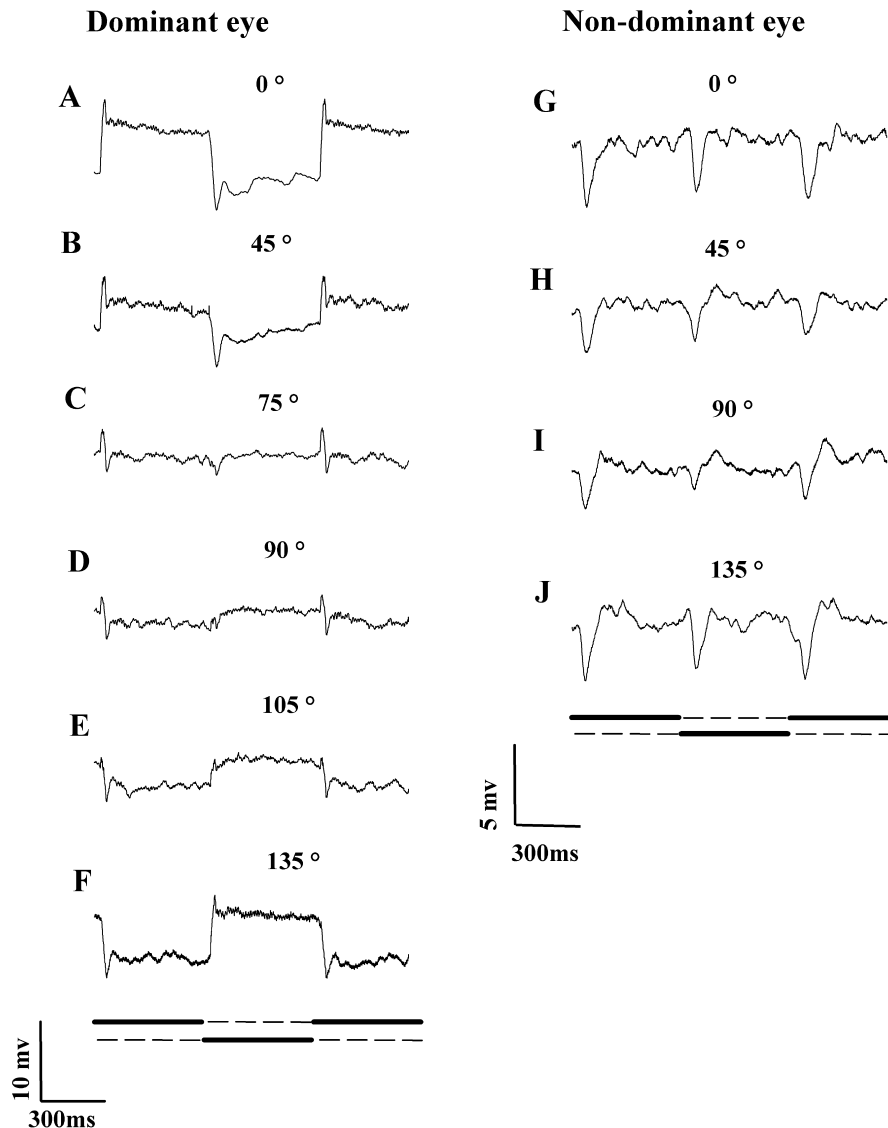


Fig. 4. Averaged PSPs (recorded from an on-center X cell, in layer A) evoked by reversing gratings (temporal frequency 1 Hz, contrast 0.5, diameter=13°) of different spatial phases. (A–F) Responses to the dominant eye; (G–J) responses to the non-dominant eye. The spatial phases are marked up each curve. The spatial frequencies were 0.5 and 0.2 c/d for A–F and G–H, respectively.

surround structure, and the responses are evoked by both on and off stimuli. These observations are consistent with most of the previous studies that used dark and bright moving spots or bars as stimuli [27,34,37,50]. The results suggest that the responses to non-dominant eye stimulation may come from a different mechanism compared with the responses to dominant eye stimulation.

Our study reveals further differences of the response properties between non-dominant and dominant eye stimuli. First, with flashing on–off spots as stimuli, almost all of the responses to the non-dominant eye (43 out of 45) are transient regardless of the classification of the cell as X and Y types by the responses to the dominant eye. Further, the PSPs usually show the same polarization (either depolarization or hyperpolarization) to stimulus onset or offset. Second, by using contrast reversing gratings, no

‘null position’ effect can be found in response to non-dominant eye stimulation, even though this effect is clear for X cells in response to dominant eye stimulation. Third, for most cells, the latencies of the response to non-dominant eye stimulation are significantly longer than those to dominant eye stimulation (the mean difference between them is around 5.5 ms). The above findings strongly suggest that the pathway giving rise to the non-dominant eye response differs from that giving rise to the dominant eye response.

A direct pathway of non-dominant eye responses showed that one LGNd cell received inputs from retinal axons from both eyes [32], but those may be the binocularly excitable interlaminar cells [24], which is not the majority of LGNd cells. Three possible indirect pathways have been postulated in previous works. They are feedfor-

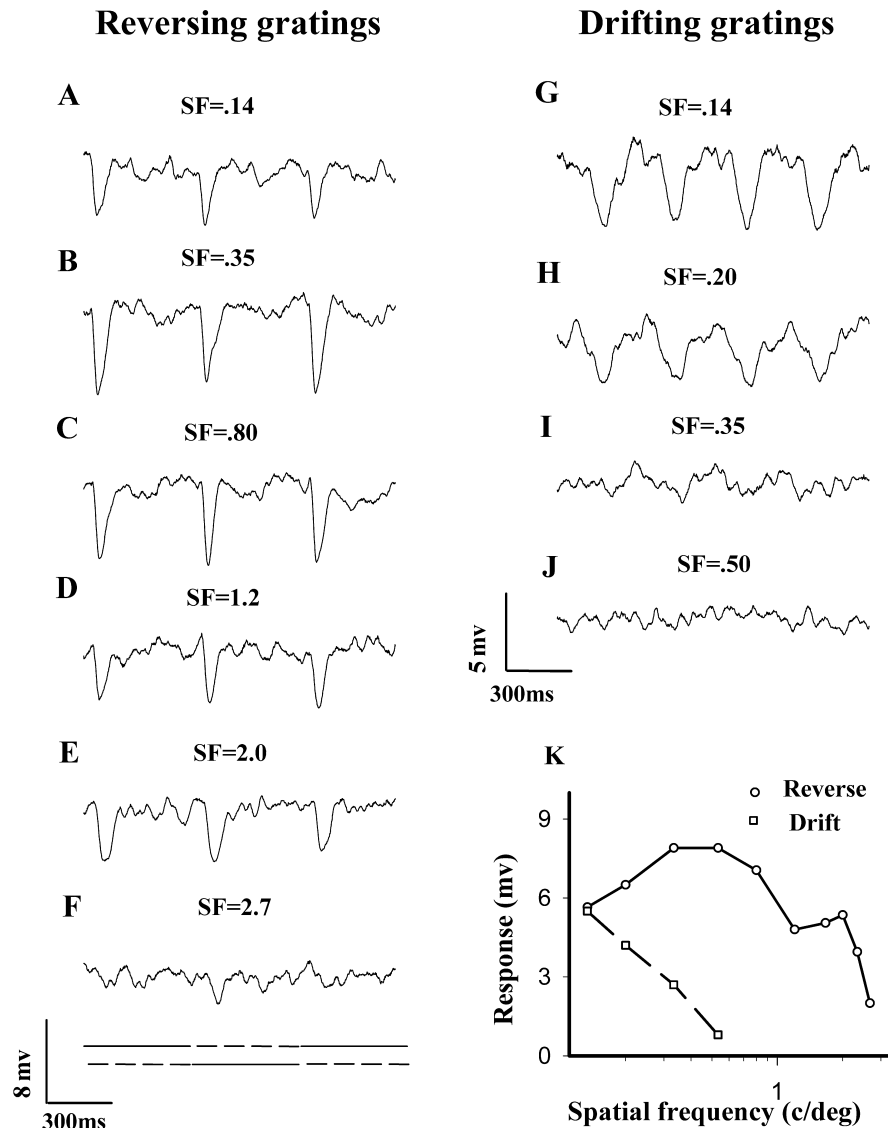


Fig. 5. Averaged PSPs (recorded from an on-center X cell in layer A) evoked by reversing grating stimuli of different spatial frequencies (A–F, temporal frequency 1 Hz, contrast 0.5, diameter=13°) and by sinusoidal drifting gratings (G–J, temporal frequency 3 Hz, contrast 0.5, diameter=13°). (K) The spatial frequency tuning curves.

ward inhibition through the intrageniculate interneurons, recurrent inhibition through the PGN cells, and corticofugal feedback [1,12,26]. The present observations suggest that the PGN pathway may be the main source of binocularity of LGNd cells, because the response properties of cells (PSPs) to non-dominant eye stimulation are similar to those of PGN neurons in a number of ways [8,35,36,54]. The receptive fields of PGN neurons have no center-surround antagonist structure [8]. PGN neurons are binocularly driven, although they prefer to contralateral eye inputs [8,34,54]. They respond to both on and off stimuli [8], tend to have transient responses, and exhibit no linear spatial summation. PGN neurons prefer to fast moving, low spatial frequency stimuli [54], which are the stimuli that we found to elicit the strongest non-dominant eye responses. The latter property is also consistent with

previous extracellular studies in LGNd [13,26,47,54]. In fact, anatomical evidence of PGN projections to LGNd has been found [7,20]. Furthermore, the longer latency responses to non-dominant eye stimulation are consistent with the responses being the result of recurrent feedback. In contrast, the receptive fields of the intrageniculate interneurons are known to be center-surround and both sustained and transient categories of interneurons exist in LGNd [8], and this is not a consistent property of the non-dominant eye driven response.

The response properties we found in LGNd are less comparable to those in visual cortex, where most of the neurons prefer to drifting gratings with much higher spatial frequencies. Also, responses to large flashing spots are not as robust as those to drifting gratings. In contrast, the percentage of LGNd neurons responding to non-dominant



eye flashing spots and drifting gratings is 92% and 54%, respectively. Evidence for the role of corticofugal feedback on non-dominant eye responses is conflicting. By ablating or reversibly deactivating the primary visual cortex and other extrastriate cortex, some groups found that the non-dominant eye responses in LGNd remained intact [34,44,48]. However, the contribution of cortical feedback undoubtedly existed in some other studies [30,36,49]. In monocularly deprived cat, whose visual cortex is expected to be mostly monocularly driven [29,53], the cortical feedback should be largely non-deprived eye driven. However, there was no difference between the percentage of binocular neurons in deprived and non-deprived layers of LGNd. This finding indicates that cortical feedback is not a major source of non-dominant eye responses. However, in contrast to the intact responses to flashing spots, the response to drifting gratings was clearly changed by visual deprivation, as no neurons in the deprived layer of the LGNd responded to drifting gratings presented to the non-dominant eye. This is most likely due to the weakening of the feedback from deprived eye driven cortical neurons. At the time of birth in the cat, the retinogeniculate projection has already formed layer specific connections [21,38]. Geniculocortical connections and intracortical connections are formed much later [11,39], and till post-natal day 14, the ocular dominance columns are formed functionally [6]. Developmentally, cortical properties seem to be more easily modified by visual experience than geniculate properties. This has been demonstrated by showing a differential change of orientation bias in visual cortex and LGNd in dark reared cats [57]. Therefore, it is possible that some fine response properties from the non-dominant eye are modified by cortical feedback, while the major source of inputs responsible for the non-dominant eye responses comes from the PGN.

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