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THE LENGTH–RESPONSE PROPERTIES OF CELLS IN THE FELINE DORSAL LATERAL GENICULATE NUCLEUS

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SUMMARY

1. In this report we have systematically examined the length–response properties of a large population of cells recorded in the cat dorsal lateral geniculate nucleus (dLGN). The responses of A laminae dLGN cells were assessed by the use of conventional single-unit extracellular recording techniques. The length preference of these cells was examined by plotting multihistogram length tuning curves to moving bars of light. Bar length was randomized in an interleaved fashion under computer control. The other stimulus parameters were standardized within the limits of those routinely used to assess the length preference of cortical cells.

2. The majority of cells (186/198), whose length–response properties are considered in detail in this report, exhibited strong centre–surround antagonism and a mean degree of length tuning equivalent to, or exceeding, that seen in most cortical hypercomplex cells ($71 \pm 1{\cdot}18\%$, S.E.M., $n = 186$).

3. The values for X cells ($74 \pm 1{\cdot}41\%$, S.E.M., $n = 100$) and Y cells ($67 \pm 2{\cdot}13\%$, S.E.M., $n = 74$) were very similar, as were those of the on-centre ($71 \pm 1{\cdot}51\%$, S.E.M., $n = 123$) and off-centre ($71 \pm 1{\cdot}85\%$, S.E.M., $n = 63$) subgroups.

4. A distinct subgroup of the Y cell population was identified. These comprised the remaining twelve out of the 198 cells examined and their response properties were sufficiently distinct to merit classification as a discrete subpopulation of cells which we have termed nlY cells. They were characterized by very poor levels of both centre–surround antagonism and length tuning, and were most frequently encountered close to laminar borders. Their response properties have been described in detail elsewhere.

5. We quantitatively compared the degree of length tuning seen with moving bars to the strength of centre–surround antagonism assessed with flashing spots. The degree of length tuning did not necessarily follow the level of centre–surround antagonism.

6. Examination of the effects of unilaterally extending bar length to one or other side of the receptive field did not reveal the type of asymmetry frequently seen in cortical hypercomplex cells.

7. The high degree of length tuning seen in this study underlines the potential

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importance of geniculate response properties to the generation of the length–response properties of cortical hypercomplex cells. The findings are discussed in relation to the synaptic mechanisms contributing to the generation of length tuning at subcortical and cortical levels.

INTRODUCTION

Cells exhibiting length tuning were first described by Hubel & Wiesel (1965) in cat visual cortex. The primary characteristic by which these so-called 'hypercomplex' cells were distinguished from other cortical cells was that their responses to long bars were considerably reduced in magnitude in comparison to the response to a shorter bar of optimal length. Hypercomplex cells were originally proposed to exist at the top of a cortical hierarchical structure (Hubel & Wiesel, 1965, 1968), and the property of length tuning was regarded as a higher order feature generated through intracortical inhibitory mechanisms. More recent work has clearly demonstrated that hypercomplex cells can occur at the earliest stages in the cortical processing sequence (Dreher, 1972; Gilbert, 1977; Rose, 1977; Kato, Bishop & Orban, 1978), but length tuning is still widely regarded as a purely cortical phenomenon being generated through intracortical inhibitory mechanisms (e.g. Bolz & Gilbert, 1986).

It has, however, been suggested that relay cells in the dorsal lateral geniculate nucleus, which provide the input to the cortex, themselves exhibit some degree of length tuning. For example, Cleland, Lee & Vidyasagar (1983) noted that on average, cells in the A laminae of the cat dorsal lateral geniculate nucleus (dLGN) already exhibit a 50% decrement in response magnitude to long as opposed to short bars. Moreover, careful scrutiny of the literature reveals two further quantitative studies which, while predominantly examining the responses of cortical cells, briefly assessed the length–response properties of dLGN cells (Schiller, Finlay & Volman, 1976; Mustari, Bullier & Henry, 1982), and the results from these studies would also support the view that dLGN cells do show some degree of length tuning. These observations raise the possibility that at least part of the length tuning seen in cortical hypercomplex cells might merely reflect a decreased excitatory drive from geniculate inputs (Schiller *et al.* 1976; Rose, 1979; Cleland *et al.* 1983) as opposed to the action of specific cortical inhibitory mechanisms.

With the exception of the theoretical work of Rose (1979) the issue of the length preference of dLGN cells has been predominantly ignored in cortical literature. Indeed, the hypothesis that hypercomplex cell length tuning merely reflects an already length-tuned geniculate input is in direct conflict with the widespread belief that cortical inhibitory mechanisms are responsible for the generation of cortical 'end-inhibition' (Bolz & Gilbert, 1986). Resolving this issue is not a trivial matter, since it is clear that a length-tuned input from the dLGN would have very important implications for our understanding of the type of synaptic convergence and connectivity necessary to generate the varying patterns of spatial summation seen along the axis of the optimal orientation in cortical cells. Indeed, if nearly all cells in the dLGN are significantly length tuned, it is difficult to see how the very long receptive fields of the cells seen in layer VI of the visual cortex might be generated. Thus, it is particularly important to know the extent to which length tuning varies across the population of dLGN cells and whether, for example, it is significantly

lower in Y as opposed to X cells. In this work we have systematically examined the length tuning of dLGN cells utilizing the stimulus parameters routinely employed to assess the length tuning of cortical cells. Some cortical hypercomplex cells show marked asymmetries in the response reduction seen when a stimulus is extended to one or the other side of their field (Hubel & Wiesel, 1965; Dreher, 1972; Sillito & Versiani, 1977; Orban, Kato & Bishop, 1979*a*). We have checked whether such variations are apparent in the receptive fields of dLGN cells. A preliminary report of some of this work has previously appeared in abstract form (Jones & Sillito, 1987).

METHODS

Preparation and maintenance of animals

The experiments were carried out on adult, female cats in the weight range 2·0–3·0 kg. Anaesthesia was induced with a mixture of 70% N_2O, 30% O_2 and 5% halothane. Surgical procedures were carried out with 2–5% halothane in the N_2O–O_2 mixture. Anaesthesia was maintained throughout the course of the experiment with a mixture of 75% N_2O, 25% O_2 and 0·1–0·4% halothane. A solution of lignocaine hydrochloride (2% w/v) was applied to all wound margins, and the ear bars of the stereotaxic apparatus were coated with lignocaine hydrochloride gel. Animals were immobilized with an initial dose of 20–40 mg gallamine triethiodide (Flaxedil), followed by a continuous infusion at a rate of 10 mg kg^{-1} h^{-1}, in a solution of dextrose saline (4% w/v dextrose in 0·18% w/v saline). End-tidal CO_2 was monitored and the respiratory rate or volume adjusted to maintain a level of 3·8–4·2%. Temperature was maintained at 38 °C by use of a thermostatically controlled electric heating blanket. End-tidal CO_2, the ECG waveform, intersystolic interval and EEG waveform were monitored at all times through the experiment. Any perturbations of these parameters commensurate with a decline in the level of anaesthesia were immediately compensated for by an increase in the level of halothane.

Bilateral cervical sympathectomy was performed. Mydriasis and cycloplegia were achieved by topical application of atropine methonitrate (2% w/v), and the nictitating membranes were retracted using 1% (w/v) phenylephrine hydrochloride. The eyes were protected with plastic contact lenses, and brought to focus on a semi-opaque tangent screen 114 cm away using supplementary lenses. The positions of the optic disks and area centrales were determined by back projection (Fernald & Chase, 1971). Artificial pupils (3 mm diameter) were mounted immediately in front of the contact lenses.

Experimental procedures

Single-unit activity was recorded in the A laminae of the dLGN using either single or multibarrelled glass micropipettes. Single pipettes and the recording barrels of multibarrelled pipettes were filled with either 2% (w/v) Pontamine Sky Blue in 0·5 M-sodium acetate, or 3 M-sodium chloride.

Visual stimuli were generated optically on the tangent screen or on a raster display (Peter Joyce Display, Joyce Electronics, Cambridge). Optically generated stimuli were used for preliminary mapping of receptive fields, and for the generation of flashing spots of varying size and contrast. The Peter Joyce display was utilized for generation of phase-reversing gratings and moving bars. Stimuli presented on the Joyce display were controlled using an Alpha LS1-2/502 interface computer system (Cambridge Electronic Design). Spike time sequences were stored on the computer generally with a resolution of 1 ms and data were displayed as peristimulus time histograms (PSTHs).

Classification of cells. We routinely determined receptive field centre–surround dimensions, strength of surround inhibition elicited by a large spot, eccentricity, response to standing contrast over field centre, presence or absence of the periphery effect and assessed the linearity of spatial summation. The latter was assessed from the responses to sinusoidally phase-reversing sinusoidal gratings presented at a range of spatial phases in a randomly interleaved sequence, when the highest spatial frequency compatible with a consistent response was used. A Fourier analysis was performed to extract the first and second harmonic components of the responses, which were then

plotted against spatial phase. The primary criteria used for the classification of X and Y cells was the distinction in linearity of spatial summation over their receptive fields, X cells showing linear spatial summation and Y cells showing non-linear summation. Hence, X cell responses were characterized by the strong phase dependence of the first harmonic response, exhibiting a clear null point at which no response to the grating could be revealed. Y cell responses included a second harmonic component which was not strongly phase dependent, and for these cells responses to the grating were elicited even at the position of least response. Additionally, X cells typically displayed sustained responses, strong centre–surround antagonism, small receptive field centres (as compared to Y cell fields at the same eccentricity), and exhibited no periphery effect. Y cells were usually characterized by their transient responses, larger receptive field diameters for a given eccentricity and a prominent periphery effect. Cells were thus classified as X or Y types (Enroth-Cugell & Robson, 1966; Cleland, Dubin & Levick, 1971; Hochstein & Shapley, 1976; So & Shapley, 1979). The non-dominant eye was occluded during all testing procedures.

Length tuning curves. Length tuning curves were constructed from the averaged responses to moving bars of varying length. Horizontal or vertical bars ranging in length from 0 deg (no stimulus condition, to give a measure of spontaneous activity) to 10 deg, were swept back and forth across the cell's receptive field, the differing bar lengths being presented in a randomized interleaved fashion, to minimize the effects of response variability. In general, ten bar lengths were used and the responses averaged over five or ten trials. Bar parameters were standardized (as far as possible) within the limits of the stimuli routinely used in the estimation of cortical length preference in this laboratory, only being taken outside these limits if it proved impossible to elicit a clear response from the cell with these parameters. For most cells, bar width was 0·5 deg (for very small receptive field centres, bar width was reduced to 0·3 deg; for cells having particularly large centre diameters, bar width was usually increased to the estimated receptive field centre diameter). Bar velocity was set to a value at which a clear response could be elicited as determined qualitatively (range 1·6–5 deg s^{-1}, though usually 3·3 deg s^{-1}). For on-centre cells, the bar contrast routinely used was 150 on 50 cd m^{-2}. Light or dark bars were routinely used to assess the length tuning of off-centre cells (150 on 50 or 50 on 150 cd m^{-2} respectively). In many cases, to check for possible effects of response saturation on our data, length–response curves were also generated using either a lower contrast ratio or the same contrast ratio on a lower background luminance level. These controls did not result in any marked change in the degree of length tuning observed. However, in all cases, stimuli used were in the photopic/high mesopic luminance range.

Comparison of length tuning and centre–surround antagonism. In order to quantitatively compare centre–surround antagonism and length tuning, detailed centre–surround tuning curves were constructed to flashed spots of light of varying diameter. To generate circular stimuli, a series of cut-out templates of varying diameter were used in conjunction with the optical projector and an electronic shutter (Ealing Beck Ltd). Peristimulus time histograms were collected over ten or twenty trials for each spot diameter. The various spot diameters used were presented in a random sequence (selected by the experimenter), ensuring that small and large diameters were intermixed. Spot luminance was adjusted using neutral density Wratten filters. In some cases spot luminance was matched to bar luminance; in others it was adjusted such that spot and bar responses to the stimulus sizes eliciting optimal responses were of equal magnitude.

Unilateral length tuning. The averaged response to presentation of a short bar of optimal length was determined (responses were usually averaged over twenty trials), followed by the response to a long bilaterally extended bar (the bar length used corresponded to that producing a large decrement in response magnitude as compared to the optimal response, as assessed from the length–response curve). The responses to a long bar which totally covered the receptive field centre (as extrapolated from the length tuning curve) but which extended only over one side of the remainder of the receptive field was then determined, and then repeated for a bar extending to the other side.

Data analysis. For each cell, the PSTHs collected at each bar length were used to plot length–response curves. Each curve was plotted as a percentage of maximum response against bar length. Responses were assessed from the accumulated count in the bins constituting the response area after subtraction of the background discharge level (assessed from the PSTH representing the no-stimulus condition). In the analysis, the response as the bar crosses the receptive field centre was used for on-centre cells stimulated with light bars or off-centre cells stimulated with dark bars, but the response as the bar leaves the centre and enters the surround was used for off-centre cells

stimulated with light bars, any secondary response peaks being ignored. The maximum value thus obtained was then regarded as 100% response, and all other values expressed as a percentage of this value. The percentage degree of length tuning was then calculated by subtracting the averaged response of all the points delineating the plateau from the peak. Length tuning curves were constructed for the two directions of bar motion and the response reduction seen in the two directions of motion was then averaged to give a mean value of length tuning for the cell.

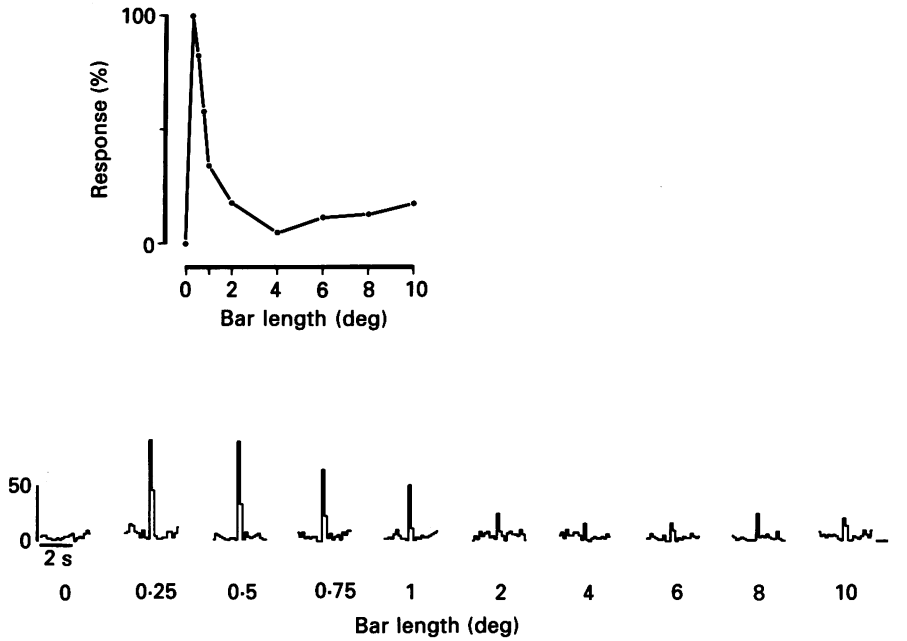

Fig. 1. Length tuning curve and PSTHs documenting the response of an on Y cell to light bars of different lengths being drifted over the receptive field. The bar lengths used were 0, 0·25, 0·5, 0·75, 1, 2, 4, 6, 8 and 10 deg. The stimulus velocity was 3·3 deg s^{-1}, width 0·5 deg and contrast 150/50 cd m^{-2}. Responses were averaged over five trials. For the PSTHs, the bin size is 150 ms. Vertical calibration 50 impulses s^{-1}; horizontal 2 s.

For centre–surround responses, the response to stimulus onset was used for on-centre cells, and the response to stimulus offset for off-centre cells. Response curves were constructed as described above for length tuning curves, but since the mode of presentation of stimuli did not allow stimulus presentation in a randomized sequence within each trial, the background discharge level subtracted from each PSTH was assessed from the baseline response seen in that PSTH.

Histology. Recording sites were marked by passing negative current of 8–15 μA through the Pontamine Sky Blue barrel for 6–10 min, resulting in the ejection of Pontamine Sky Blue from the electrode and its deposition in the tissue at the tip of the electrode. The brain was removed from the animal prior to fixation, and blocks containing the dLGN immersed in 10% (w/v) formal saline. After fixation, the tissue was impregnated with a solution of 1% gum arabic in 30% sucrose. The blocks were sectioned in the coronal plane on a freezing microtome, and serial sections, 50 μm thick, were mounted and stained with 1% Neutral Red. Sections were subsequently examined microscopically and the location of the blue spots marking the recording sites determined.

RESULTS

Length tuning

Quantitative length–response curves were constructed for 198 cells recorded from the A laminae of the dLGN. These comprised 129 on-centre and sixty-nine off-centre

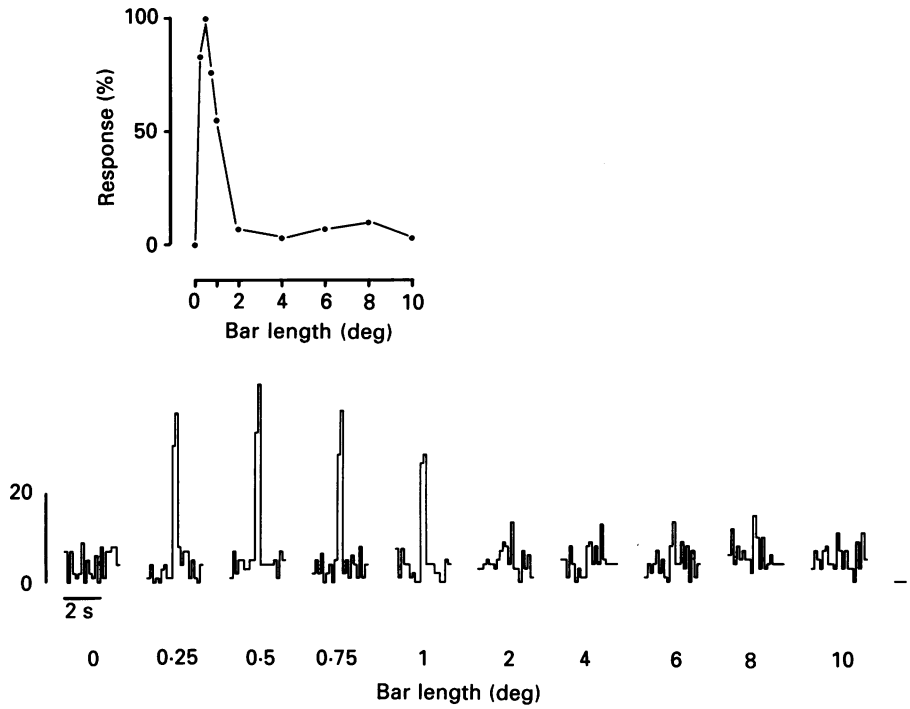

Fig. 2. Records documenting the response of an on X cell to light bars of different lengths being drifted over the receptive field. See Fig. 1 for further details, but vertical calibration 20 impulses s^{-1}. Bin size is 150 ms; five trials.

cells, 100 of which were unambiguously classified as X cells and eighty-six as Y cells, the remaining twelve cells being inconclusively categorized. All units were located within 15 deg of the area centralis, the majority being within 10 deg.

Cells with strong surround antagonism and length-tuned receptive fields

The majority of our population of cells (186) exhibited strong centre–surround antagonism, and the mean degree of length tuning for this population clearly matched or exceeded that seen in cortical hypercomplex cells. The records in Fig. 1 document the length tuning properties of a typical example. The length–response curve for this on Y cell is shown above, with the PSTHs from which it was constructed underneath. The PSTHs are arranged in order of increasing bar length from left to right, the PSTH on the extreme left-hand side representing the cell's response in the no-stimulus condition. This cell had a low level of spontaneous activity. When a 0·25 deg bar was drifted over the receptive field centre, the cell

responded with a clear increase in firing frequency, the response spanning two bins. As the bar length increased, the magnitude of the response decreased, so that the cell barely responded to a 4 deg bar. The length–response curve plots the change in unit response (expressed as a percentage of the maximum response seen) on the ordinate

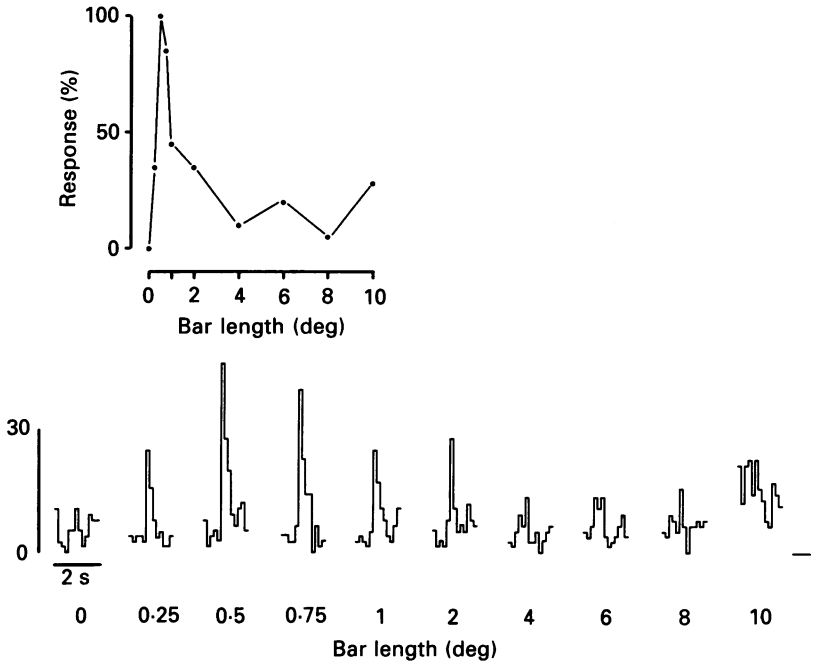

Fig. 3. Records documenting the response of an off Y cell to light bars of different length being drifted over the receptive field. See Fig. 1 for further details, but vertical calibration 30 impulses s^{-1}. Bin size is 150 ms; five trials.

against bar length (in degrees) on the abscissa. It illustrates that once the bar length was extended beyond the peak value seen for the cell, 0·25 deg in this case, the response magnitude decreased rapidly so that the response to a bar 1 deg in length was less than half that obtained with the 0·25 deg bar. With further increase in length, the response plateaued at a value of approximately 10% of the maximal response, giving a length tuning value of 90% for this cell. Thus, although the cell clearly responded to a 10 deg bar passing over the receptive field centre, the response magnitude was very much reduced in comparison to that seen to a short bar.

The records in Fig. 2 document the response of another typical cell, an on X cell in this case. In this example, the cell exhibited response summation for lengths increasing up to 0·5 deg. At this point, the response magnitude peaked, and further extension of the bar beyond this optimal length lead to a rapid decline in the cell's response, the response to a bar of 2 deg in length clearly forming part of the plateau. This cell had a plateau response level of 17% of the peak value, giving a length tuning value of 83%.

The responses of an off-centre Y cell are illustrated in Fig. 3. It is clear that this cell was highly length tuned; the response summated up to 0·5 deg but further bar

extension then produced a marked decrement in the response elicited, so that responses to long bars were only approximately 10% of the optimal response. It should be noted here that there appeared to be a clear increase in the maintained activity level of this cell when a 10 deg bar appeared at the edge of the visual display

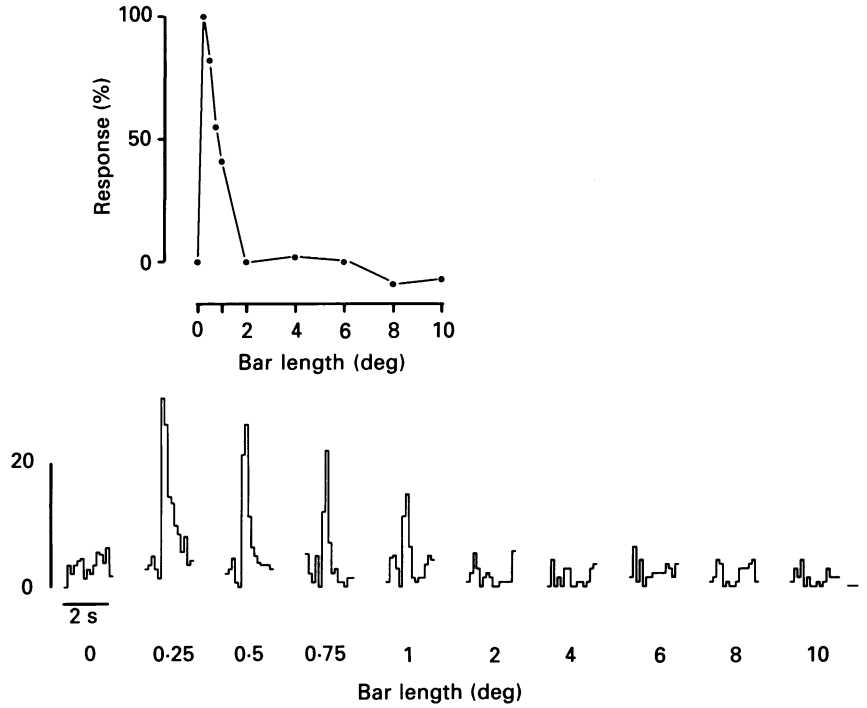

Fig. 4. Records documenting the response of an off X cell to light bars of different length being drifted over the receptive field. See Fig. 1 for further details but ten trials, vertical calibration 20 impulses s^{-1}. Bin size is 150 ms.

screen (well outside the receptive field centre), and that the increased response to a 10 deg bar suggested by the length tuning curve largely reflects this increase in maintained discharge, as opposed to a marked increase in the actual response elicited by bar motion over the receptive field centre.

In Fig. 4, the responses of an off-centre X cell are illustrated. This cell was very highly length tuned. The best response seen was to the shortest bar presented (0·25 deg), with lengthening of the bar producing a marked decrement in the responses elicited. Virtually no response could be discerned to the longer bars tested (2 deg or more), and in fact, movement of both the 8 and 10 deg bars across the response zone produced a small but distinct suppression of the spontaneous activity of the cell.

These length–response curves typified the profiles observed for most of the dLGN cells tested. The average degree of length tuning observed across this group of cells studied (186) was $71 \pm 1{\cdot}18\%$ (S.E.M.). In the histogram in Fig. 5, the population has

been subdivided into ten categories of length tuning, with cells having the weakest amount of length tuning placed in category 1 and those with the strongest in category 10. It illustrates that well over half the cells examined (115/186) showed a length preference of 70% or more, with only twenty cells (11%) exhibiting less than 50% length tuning.

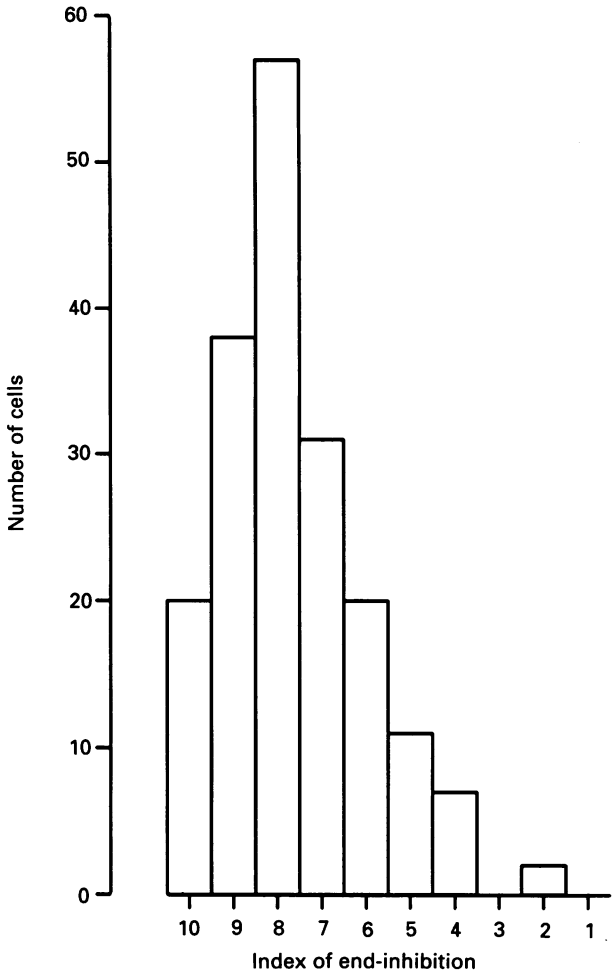

Fig. 5. Block histogram showing the distribution of length tuning in the dLGN cell population. The cells were subdivided into ten categories of length tuning. Cells having little or no length tuning were placed in category 1 (from 0 to 9%) with cells exhibiting almost total response suppression at longer bar lengths being placed in category 10 (from 90 to 100%). The mean value was $71 \pm 1{\cdot}18\%$ (S.E.M.); $n = 186$.

There appeared to be no difference in the degree of length tuning exhibited by on- and off-centre cells ($71 \pm 1{\cdot}51\%$, S.E.M., $n = 123$ and $71 \pm 1{\cdot}85\%$, S.E.M., $n = 63$, respectively; not significantly different at the $P < 0{\cdot}05$ level when tested using the

Mann–Whitney U test). There was only a small difference in the degree of length tuning seen in X and Y cells ($74 \pm 1{\cdot}41\%$, S.E.M., $n = 100$, and $67 \pm 2{\cdot}13\%$, S.E.M., $n = 74$), though this did appear to be statistically significantly different at the $P < 0{\cdot}01$ level (Mann–Whitney U test). The histograms in Fig. 6 show the index of

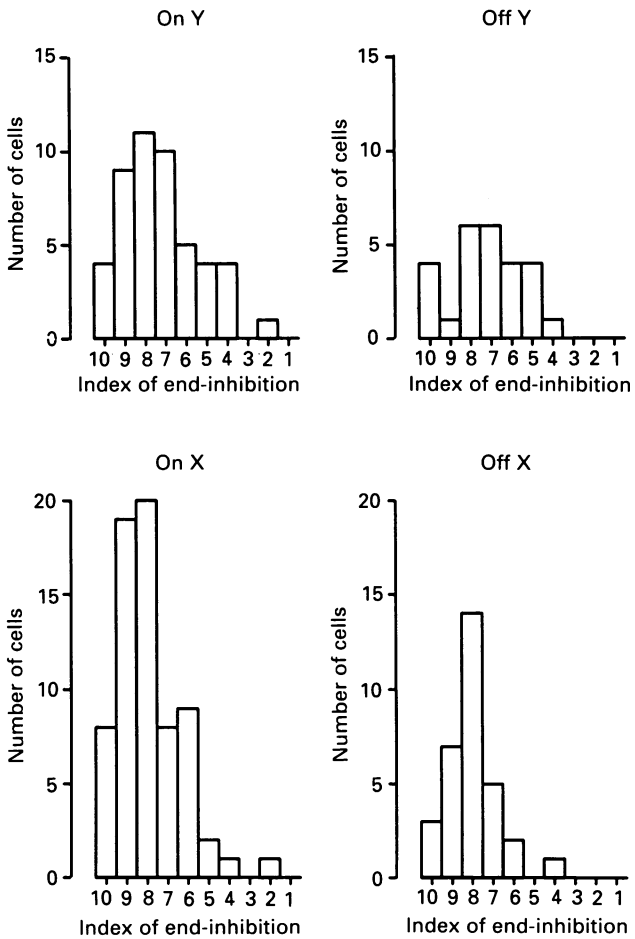

Fig. 6. Block histograms calculated as described in Fig. 5, showing the distribution of length tuning for dLGN cells, subdivided into cell classes. For on Y cells, mean = $68 \pm 2{\cdot}79\%$, S.E.M., $n = 48$; on X cells, mean = $73 \pm 1{\cdot}83\%$, S.E.M., $n = 68$; off Y cells, mean = $67 \pm 3{\cdot}29\%$, S.E.M., $n = 26$; off X cells, mean = $75 \pm 2{\cdot}06\%$, S.E.M., $n = 32$.

length tuning (calculated as described above for the overall population) against numbers of cells for on X, on Y, off X and off Y cell classes.

Cells lacking significant surround antagonism and length tuning

In our population we observed twelve quantitatively documented cells, all of which could be classified as Y cells on the basis of non-linear spatial summation, and yet differed sufficiently in their responses to be tentatively regarded as a discrete

population of cells, which have been termed nlY cells. The responses of these cells were characterized by very poor levels of centre–surround antagonism and length tuning, and they were most frequently encountered close to laminar borders. An example of the responses of one of these is illustrated in Fig. 7. This cell showed

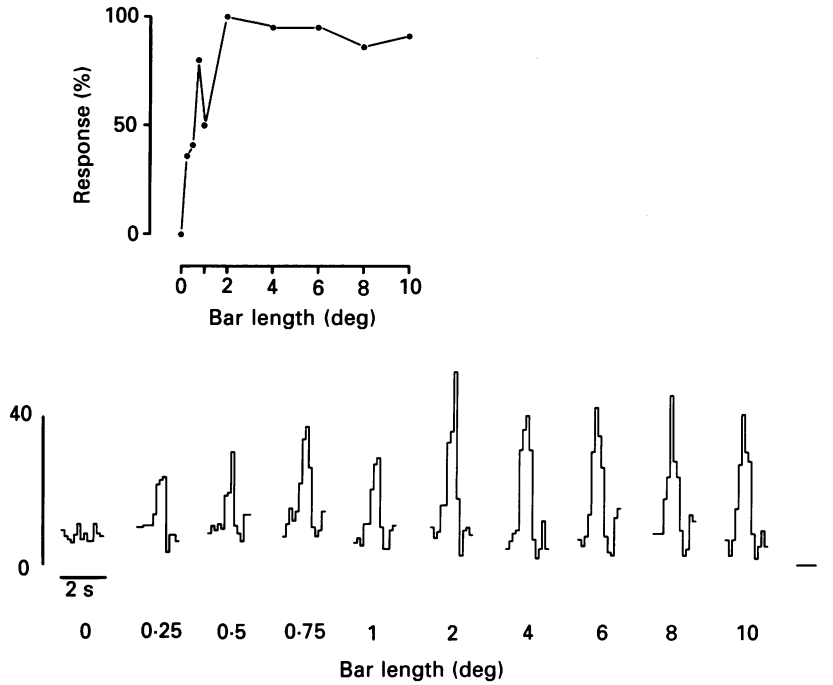

Fig. 7. Records documenting the response of an on-centre nlY cell to light bars of different length being drifted over the receptive field. See Fig. 1 for further details but ten trials, vertical calibration 40 impulses s^{-1}. Bin size is 150 ms.

length summation for stimuli increasing up to 2 deg in length. However, in contrast to the length tuning curves illustrated above, no significant decline in the response magnitude was observed with further lengthening of the bar. The responses of these nlY cells have been described in detail in a previous paper (Jones & Sillito, 1990), and they are not considered further in this report.

Comparison of length tuning and centre–surround antagonism

It is important to know whether the length tuning of dLGN cells as tested with moving bars is linked to the degree of centre–surround antagonism as tested with stationary flashing stimuli. We have therefore quantitatively compared the degree of centre–surround antagonism and length tuning exhibited by dLGN cells.

In many cases, the degree of centre–surround antagonism and length tuning exhibited by cells were very similar. A typical example is illustrated in Fig. 8. This on Y cell exhibited clear response summation to spots and bars up to 1 deg; further increase in stimulus size resulted in a substantial decrement in the responses observed, with plateau levels of response achieved by 4 deg in both cases.

However, for some cells, the degree of length tuning and centre–surround antagonism varied considerably. In all such cases, the cells exhibited far better centre–surround antagonism than length tuning. One example is documented in Fig. 9. This on X cell exhibited clear response summation with increase in spot diameter

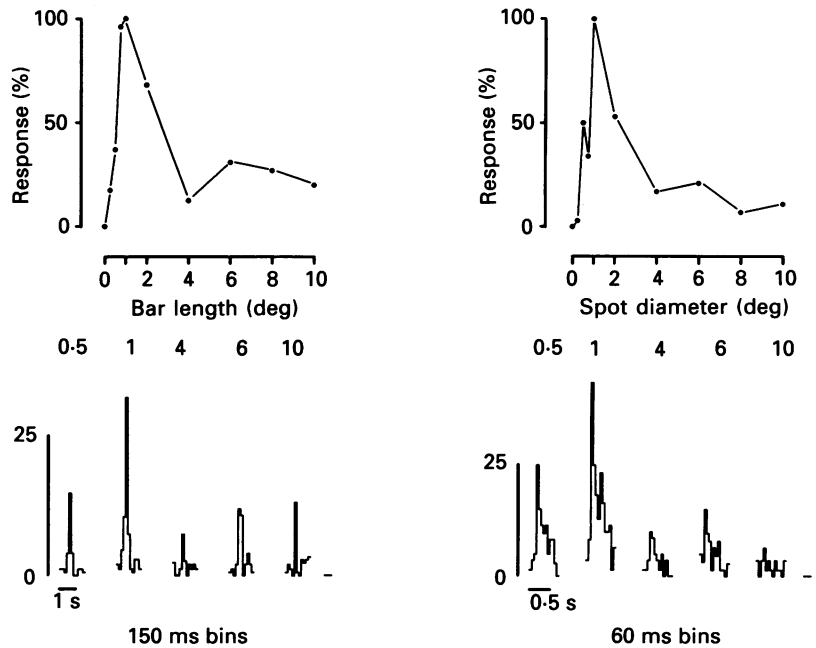

Fig. 8. Records documenting the length–response and centre–surround properties of an on Y cell. The left-hand side documents length–response properties and the right-hand side documents centre–surround properties. For the length tuning curve, stimulus velocity was 3·3 deg s^{-1}, width 0·5 deg and contrast 150/50 cd m^{-2}. Spot luminance was matched to bar luminance. The left-hand side PSTHs illustrate responses to moving bars and the right-hand side PSTHs responses to flashed spots. The bar lengths illustrated are 0·5, 1, 4, 6 and 10 deg. Bin size for these PSTHs is 150 ms. Calibration bars: vertical, 25 impulses s^{-1}; horizontal, 1 s. Spot diameters illustrated are 0·5, 1, 4, 6 and 10 deg. Bin size is 60 ms. Calibration bars: vertical, 25 impulses s^{-1}; horizontal, 0·5 s. All responses are averaged over ten trials.

up to 0·75 deg, with further increase in diameter leading to a pronounced decline in response magnitude. The response plateaued at spot sizes of 4 deg and upwards; the response to a 4 deg flashing spot was only 8% of the optimal response seen. An increase in bar length beyond 1 deg also caused some decrement in the responses seen. However, the degree of response reduction obtained was much smaller; the response to a 4 deg bar was 50% of the optimal bar response seen.

Unilateral bar extension

Following from the fact that cortical hypercomplex cells can exhibit marked asymmetries in the degree of response reduction when a bar is extended to one or the other side of their receptive field, we have tested dLGN cell responses with the

unilateral extension paradigm. The records in Fig. 10 illustrate the results observed with this procedure. The top trace documents the response of an on Y cell (records are from the same cell whose length tuning curve was shown in Fig. 1) while the bottom trace documents the response of an on X cell. The response to a bar of

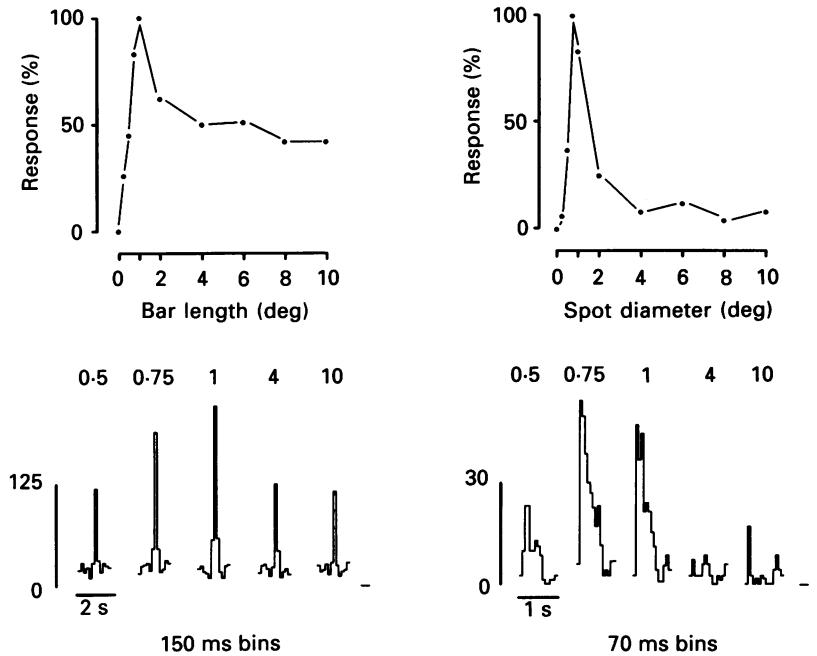

Fig. 9. Records documenting the length–response and centre–surround properties of an on X cell. Broad details as in Fig. 8. The bar lengths illustrated are 0·5, 0·75, 1, 4 and 10 deg. Bin size for these PSTHs is 150 ms. Calibration bars: vertical, 125 impulses s^{-1}; horizontal, 2 s. Spot diameters illustrated are 0·5, 0·75, 1, 4 and 10 deg. Bin size is 70 ms. Calibration bars: vertical, 30 impulses s^{-1}; horizontal, 1 s.

optimal length is shown in the PSTH on the extreme left, while the response to a bilaterally extended long bar is shown on the extreme right. The middle records illustrate the response to a long bar extended first to one and then the other side of the receptive field centre. Two major points are clearly illustrated by these results. Firstly, for both cells, there is virtually no difference in the responses elicited by bars extended to opposing sides of the receptive field centre. Secondly, the response decrement observed to a bilaterally extended bar is nearly exactly that which one would predict if the response reductions produced unilaterally are simply summated linearly for the bilateral bar. In the upper trace, the response elicited by the bilaterally extended bar was 20% of that obtained with a bar of optimal length (the percentage values quoted here, and in the remainder of this paragraph, have not been adjusted to take account of spontaneous activity levels). The responses elicited by the unilaterally extended bars were 60 and 61% of the optimal respectively. Thus bilateral extension resulted in a reduction in response magnitude of 80%, whilst unilateral extensions resulted in reductions of 40 and 39%. Hence the combined

response reduction produced unilaterally at 79% is nearly identical to the 80% reduction seen with the long bilaterally extended bar. For the cell in the lower trace, the response reduction to the bilaterally extended bar was 72% compared to a combined value of 80% (39 and 41%) for the two unilateral responses.

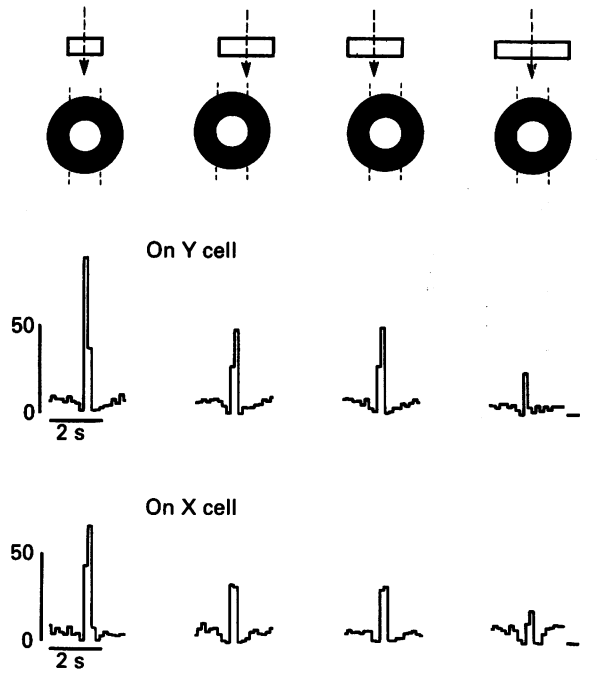

Fig. 10. PSTHs documenting the responses of two dLGN cells to bars of optimal length, a long bar extended bilaterally, and to long bars extended unilaterally to one or other side of the receptive field centre, being drifted over the receptive field at a velocity of 3·3 deg s^{-1}. The diagrams above each record schematically summarize the stimulus condition. The upper records show the responses of an on Y cell, the lower those of an on X cell. Bar width was 0·5 deg; contrast 150/50 cd m^{-2}. Bar lengths were (from left to right): upper records, 0·5, 4·25, 4·25 and 8 deg; lower records, 0·75, 2·35, 2·35 and 4 deg. Bin size was 150 ms. Number of trials was twenty. Calibration bars: vertical, 50 impulses s^{-1}; horizontal, 2 s.

Eighteen cells were tested in this way, comprising seven on X cells, five on Y cells, four off X cells and two off Y cells. In no case was it possible to reveal a marked difference in response magnitude to unilateral bar extension to one or other side of the receptive field centre. The small differences seen in some cells (the mean difference in the percentage response reduction with respect to the optimal response seen to the unilaterally extended bars was $5 \pm 1{\cdot}12\%$, S.E.M., $n = 18$) were in no way comparable to the large asymmetries that have previously been documented for cortical cells. Indeed, since the unilateral data were gathered in a non-randomized manner, even the small differences occasionally observed cannot with any confidence be regarded as a real bias, but more probably arise from a small shift in cell responsiveness with time.

DISCUSSION

These results demonstrate that when dLGN cells are driven by stimuli routinely used to study cortical cell responses, a large proportion of cells exhibit a high degree of length tuning. Overall, the mean degree of length tuning across all the dLGN cells for which quantitative length–response curves were generated (excluding the small subgroup of non-length-tuned Y cells) was $71 \pm 1{\cdot}18\%$ (S.E.M., $n = 186$). Even if the data pertaining to nlY cells are included in this analysis, the mean degree of length tuning seen was $68 \pm 1{\cdot}48\%$ (S.E.M., $n = 198$) which is considerably greater than values obtained in previous studies of cat dLGN (Mustari *et al.* 1982; Cleland *et al.* 1983). Indeed, our data clearly demonstrate that for many dLGN cells, the degree of length tuning matches that obtained for the most tightly tuned cortical hypercomplex cells. Though it is universally accepted that for a cortical cell to be regarded as hypercomplex, its response to a long stimulus should be considerably less than that elicited by a short stimulus, there is no general agreement on the actual degree of response reduction which should be exhibited before a cell is placed into the hypercomplex category (Rose, 1977; Kato *et al.* 1978; Yamane, Maske & Bishop, 1985). Using a cut-off value of 50%, Yamane *et al.* (1985) classified nearly half the cells tested in their work as hypercomplex. Using the same cut-off point, 89% of the dLGN cells whose responses are described in this report would have fallen into the hypercomplex cell category were they to have been recorded in the cortex. However, none of the nlY cells whose responses have been described previously (Jones & Sillito, 1990) would have been regarded as hypercomplex, and these cells appear to comprise roughly 13% of the total population of dLGN cells. Even taking this into account, it appears that only about a quarter of all dLGN cells exhibit less than 50% length tuning. Thus, 75% of dLGN cells (as opposed to 50% of cortical cells) could be classified as hypercomplex. These data therefore support the proposal that the length tuning seen in cortical hypercomplex cells may be initiated by a length-tuned input (Rose, 1979; Cleland *et al.* 1983). In fact, the present results could be interpreted as suggesting that the magnitude of subcortical length tuning is sufficient to underlie all of the length tuning seen in cortical hypercomplex cells. This idea is highly controversial, since it strongly questions the widely held belief that intracortical processing is necessary to produce much of the length tuning seen in the cortex (Hubel & Wiesel, 1965; Bolz & Gilbert, 1986). In view of the present evidence, this latter theory would, however, imply that the cortex first eliminates the very considerable length tuning present in its geniculate input, and then regenerates this property. This interpretation seems to add unnecessary complexity and is not immediately attractive. Nevertheless, it is fair to comment that consideration of some facets of cortical hypercomplex cell length tuning does question the extent to which geniculate length tuning could underlie all the length tuning seen in hypercomplex cells.

For example, hypercomplex cells exhibiting unilateral response asymmetries in the strength of the end-zones to either side of the excitatory discharge zone have been extensively documented in the cortex (Hubel & Wiesel, 1965; Dreher, 1972; Sillito & Versiani, 1977; Henry, Goodwin & Bishop, 1978; Orban *et al.* 1979*a*). As might be predicted from the essentially symmetrical receptive fields of dLGN cells, response

asymmetries of the magnitude seen in cortical cells could not be detected in the dLGN cells tested in this study. In this respect, the present data provide clear evidence for the view that the asymmetrical end-zones seen in hypercomplex cells arise through cortical interactions, thus suggesting that intracortical mechanisms do contribute in part to the generation of hypercomplex cell properties. This viewpoint is supported by data documenting the effects of ionophoresis of the $GABA_A$ antagonist bicuculline on superficial layer hypercomplex cells (Sillito & Versiani, 1977). This procedure did not reveal a marked reduction in the degree of length tuning to bilaterally extended bars, which would have been predicted were a specific GABAergic inhibitory process at this level responsible for the generation of length tuning in these cells, but its application did uncover an increased response to unilaterally extended bars. Many of the cells clearly exhibited asymmetrical end-zones to the unilaterally extended stimuli prior to bicuculline ionophoresis, and the asymmetries in the responses were reduced (though not entirely abolished) during bicuculline ejection.

Furthermore, some (though not all) studies have concluded that hypercomplex cells exhibit orientation-specific end-zones (Hubel & Wiesel, 1965; Orban, Kato & Bishop, 1979*b*) which has led some authors to conclude that hypercomplex cell length tuning is therefore necessarily cortically generated (Orban *et al.* 1979*b*). None the less, as pointed out by Cleland *et al.* (1983), the demonstration of such an effect in the presence of an excitation which is, of course, itself orientation dependent, is difficult, and could be misleading if there is significant overlap of the two zones. Indeed, since the strongest part of the end-zone has been shown to overlap with the central excitatory zone (Kato *et al.* 1978; Henry *et al.* 1978), this is obviously a problem. Thus it is plausible that at least a component of the effect seen in the work of Orban *et al.* (1979*b*) might result from a lack of contiguity between the stimuli covering the excitatory discharge zone and the end-zone, which might give rise to the appearance of orientation tuning, since contiguity would only be achieved when the orientation of the stimulus covering the end-zone matched that used for the discharge zone. The situation is further complicated by the fact that intracortical connections, both excitatory and inhibitory, will of course influence the responses of hypercomplex cells even if their basic length tuning is established by the geniculate input. It is this type of connection that may generate the asymmetries in the field.

Bolz & Gilbert (1986) have suggested that the projection from long field layer VI cells to inhibitory interneurones in layer IV is responsible for the generation of hypercomplex cell length tuning since they found that length tuning of cells in layers II, III and IV of the visual cortex was abolished during focal pressure ejection of GABA in layer VI. This interpretation of their results is, of course, incompatible with the view that the length preference seen in layer IV hypercomplex cells reflects that of their geniculate input. However, in view of data demonstrating that a component of the length tuning seen in dLGN cells is dependent on the corticofugal projection (Murphy & Sillito, 1987), the effect seen by Bolz and Gilbert might in part be attributed to the fact that as well as inactivating the layer VI to layer IV pathway, their GABA injections would also have switched off the corticofugal projection.

Previous reports addressing dLGN length tuning have attributed this reduction in response to long bars as a consequence of the concentric, antagonistic centre–

surround receptive field properties of retinal ganglion and dLGN cells (Schiller *et al.* 1976; Mustari *et al.* 1982; Cleland *et al.* 1983). It is of course self-evident that both the antagonistic surround of retinal ganglion cells (Kuffler, 1953) and the enhanced level of surround antagonism seen in the dLGN (Hubel & Wiesel, 1961; Cleland *et al.* 1971; Singer, Poppel & Creutzfeldt, 1972; Sillito & Kemp, 1983) will lead to some decrement in the responses to long as opposed to short stimuli, since a short, centrally located bar will move only over the receptive field centre while a long bar will also encroach into, and thus activate, the antagonistic surround. However, though the length tuning profiles observed for many dLGN cells did appear to mirror the centre–surround profiles obtained, which might superficially be regarded as support for the view that the length tuning observed in dLGN cells merely reflects the centre–surround properties of these cells, the situation is somewhat complicated by the fact that in a few cases, we were able to observe poor length tuning from cells exhibiting excellent centre–surround antagonism (as illustrated in Fig. 9). Thus, at least in some cases, excellent centre–surround antagonism is not, in itself, sufficient to generate high levels of length tuning. Indeed, detailed consideration of the geometric differences between the respective stimulus protocols itself leads to the conclusion that it would be difficult to ascribe the generation of the very high degree of length tuning documented here solely to the centre–surround receptive field structure of dLGN cells. The actual amount of surround coverage produced by a long bar whose width is roughly equivalent to the cell's centre dimensions will be far less than that produced by a spot of light whose diameter is equivalent to the length of the bar stimulus used. However, the proportional difference in the coverage of the centre by small spots of optimal diameter, as opposed to short bars of optimal length, will be much less. Hence, on purely geometric considerations, there will be a large difference in the balance of centre–surround antagonism invoked by the two stimulus conditions, thus presumably necessitating some other additional factor to account for the high degree of length tuning seen in most dLGN cells, serving to compensate for the reduced amount of surround inhibition evoked by bar stimuli. Indeed, since recent work from this laboratory has demonstrated that the generation of a considerable component of the length tuning seen in dLGN cells is dependent on an intact corticofugal projection to the dLGN (Murphy & Sillito, 1987), it would appear that dLGN cell length tuning arises only partly through centre–surround interactions and partially through the influence of the corticofugal projection. Presumably, those cells recorded in this study, from preparations receiving an intact corticofugal feedback, but which displayed a considerably lesser degree of response attenuation to long bars than to large flashed spots, might reflect the fact that, for these particular cells, the stimulus protocol utilized failed to activate the corticofugal system, or that for these cells, either the number or the efficacy of the synapses through which the corticofugal influence is mediated was considerably reduced.

Our data clearly underline the fact that most cells in the dLGN are far more sensitive to stimulus length than has hitherto been imagined, and that for many cells the degree of selectivity seen matches that of the most tightly tuned cortical cells. While some reports have indicated that dLGN cells exhibit some degree of orientation bias in their responses (Daniels, Norman & Pettigrew, 1977; Vidyasagar & Urbas, 1982; Albus, Wolf & Beckman, 1983; Vidyasagar & Heide, 1984; Soodak,

Shapley & Kaplan, 1987), these are small in comparison to the degree of orientation selectivity displayed by cortical cells, and appear largely to reflect a small orientation bias already present in the retina (Soodak *et al.* 1987). The high degree of length tuning present in cells having only a slight orientation bias raises several interesting issues. Firstly while dLGN cells themselves show only a small degree of orientation selectivity, cells in layer VI of the visual cortex which give rise to the corticofugal projection are of course highly selective with respect to stimulus orientation. The possibility of an orientation bias in the corticofugal feedback raises a number of interesting questions. For example, as the corticofugal influence is responsible for the generation of a significant component of dLGN cell length tuning, it suggests the possibility of an orientation-dependent component to this length tuning, superimposed on an otherwise virtually non-orientationally selective response. However, recent data from our laboratory indicate that the corticofugal influence to a given location in the dLGN is derived from an entire subset of orientation columns (Sillito, Murphy & Cudeiro, 1991). Thus it appears that this projection provides a feedback sensitive to the motion of an elongated edge but not to its absolute orientation. Secondly, an input to non-length-tuned cortical cells derived from geniculate inputs having a high degree of length tuning at all stimulus orientations has important implications for our understanding of the synaptic convergence generating spatial summation along the axis of optimal orientation in cortical cells. The elongated fields and orientation tuning of cortical cells have frequently been attributed to convergent input from an array of geniculate cells whose receptive fields are arranged in an elongated row in visual space (Hubel & Wiesel, 1965; Cleland *et al.* 1983; Ferster, 1987). However, it is immediately clear that if these input cells are themselves highly length tuned, increasing stimulus length along the plane generating orientation preference while undoubtedly recruiting additional geniculate inputs will also cause a decrease in response magnitude of the centrally placed geniculate inputs. While appropriate weighting functions can be assigned to the input cells so that an elongated, length-summating field can be generated (Cleland *et al.* 1983), such manipulations have the caveat that a short, centrally placed bar moved over the receptive field at the orthogonal orientation would be expected to elicit a strong response. Thus it appears difficult to construct an elongated, orientation-selective field solely on the basis of excitatory convergent input from highly length-tuned geniculate cells.

The balance of current evidence would suggest that the situation with respect to the generation of length tuning in the visual pathway may be considerably more complicated than has previously been envisaged. Hence, a large component of cortical length tuning may well reflect that present at subcortical levels, but this is then in turn modified to some extent by cortical mechanisms. However, since the length tuning seen in the dLGN is itself partially dependent on cortical mechanisms (via the corticofugal projection) the generation of length tuning in the visual system involves the operation of a neuronal circuit, and not merely a hierarchical progression. Furthermore, from our earlier demonstration of a distinct subpopulation of non-length-tuned relay cells in the dLGN (Jones & Sillito, 1990) it is interesting to speculate on the possibility of two separate streams of input to the cortex, one from the highly length-tuned cells described here, and one from the nlY

cells, which we have previously suggested could underlie the response properties of the long field cells of layer VI.

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