Contrast-Dependent Spatial Summation in the Lateral Geniculate Nucleus and Retina of the Cat


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INTRODUCTION

While it is known that the temporal properties of receptive fields in the early visual pathway can be influenced by the stimulus configuration (especially contrast, Reid et al. 1992), the spatial properties have been considered stimulus independent. Recently, this view has been challenged by several studies. In particular, it has been shown that spatial summation in the primary visual cortex of the monkey is strongly dependent on stimulus contrast; the area (length and width) over which responses summate increases as stimulus contrast decreases (Cavanaugh et al. 2002; Kapadia et al. 1999; Sceniak et al. 1999; see also Jagadeesh and Ferster 1990). Fitting summation curves to a difference of Gaussians model, we attributed this contrast-dependent effect to an actual change in the size of the center mechanism. Analogous changes in spatial frequency tuning were also observed, specifically increased peaks and cut-off frequencies with contrast. These effects were seen across the populations of both X and Y cell types. In a few cases, LGN cells were recorded simultaneously with one of their retinal ganglion cell (RGC) inputs (S-potentials). In every case, the RGCs exhibited similar contrast-dependent effects in the space and spatial-frequency domains. We propose that this contrast dependency in the retinal ganglion cells results directly from a reduction in the size of the center mechanism due to an increase in contrast. We also propose that these properties first arise in the retina and are transmitted passively through the LGN to visual cortex.

METHODS

General

Animals were prepared for single-unit recording as described in detail elsewhere (Palmer and Nafziger 2002). Briefly, adult cats were anesthetized with 3–4% halothane in a 70:30 mixture of N2O and O2. Venous catheters were placed in each hind limb, gas anesthesia was discontinued, and the animal was maintained on intravenous sodium thiopental as needed through the remainder of the surgery. A tracheotomy was performed, and the animal was placed in a stereotaxic frame and was paralyzed with an injection of gallamine triethiodide (Flaxedil, 60 mg). The animal was maintained on positive pressure ventilation that was adjusted to hold the end-expired CO2 at 4.0%. Two venous catheters were used so that anesthetic (2–8 mg/kg per hour thiopental) and paralytic (15 mg/h Flaxedil) could be infused throughout the experiment at independent rates. Cortical EEG was monitored continuously throughout the experiment, and the rate of anesthetic infusion was regulated to maintain the animal in a state similar to light sleep, characterized by frequent bursts of 7- to 10-Hz waves (spindles). Body temperature was also monitored and maintained at 38°C. A small craniotomy was made at Horsley-Clarke A6.0, L8.5. At the conclusion of the experiment, animals were given a lethal injection of sodium pentobarbital. This protocol was approved by the University of Pennsylvania’s Institutional Animal Care and Use Committee and conforms to guidelines recommended in Preparation and Maintenance of Higher Mammals During Neuroscience Experiments, publication 91–3207 of the National Institutes of Health.

Neutral contact lenses were placed in each eye, and spectacle lenses were added to bring the reflection of retinal vessels into sharp focus on the screen of a CRT mounted in front of the cat. Biprisms were also placed in front of each eye to allow approximate superposition of the lines of sight, although cells were studied monocularly. Pupils were dilated with 1% ophthalmic atropine, and the nictitating membranes were retracted with 1% phenylephrine hydrochloride. Animals were given intramuscular injections of glycopyrrolate (0.1 mg, im, once per day) to minimize secretions, dexamethasone (0.4 mg, im, once per day) to minimize cerebral edema, and ampicillin (10 mg/kg, im, twice per day) to prevent infection. Most experiments lasted 2 days.

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Recording and data acquisition

All recordings were made extracellularly with tungsten-in-glass electrodes. Signals were amplified and conditioned with a programmable amplifier (Alpha Omega, Nazareth, Israel). Amplified signals were routed to an oscilloscope, an audio monitor, and a spike sorter (Alpha Omega). The latter performed on-line template matching of action potentials and produced TTL pulses, which were detected at the computer interface during clock interrupt service routines every 100 or 1,000 µs, depending on the application. The electrode was advanced with a Burleigh Inchworm (Fishers, NY). Penetrations were limited to layers A and A1 based on electrophysiological criteria; no histology was performed. All receptive fields were limited to the central 10° of the visual field, and the vast majority was within 5°.

Visual stimuli were presented on an Image Systems multisynch monochrome monitor at a frame rate of 125 Hz by means of a VSG-2/3 board (Cambridge Research Systems, Cambridge, UK). Mean luminance was 80 cd/m², and lookup tables were linearized for contrasts in the range of ±100%. The monitor was situated 24.7 cm from the eye such that 1 cm represents 2.3° of visual space. The screen subtended 36° horizontally and 27° vertically. Stimuli were presented with a resolution of 22 pixels/°. An identical monitor driven in series was situated at the experimenter’s console.

The responses of each cell were thoroughly characterized prior to any experimental procedures. This was accomplished in two phases. First, computer-assisted hand-plotting procedures were used to estimate the position and general properties of the receptive field in both the two-dimensional spatial and spatial-frequency domains (see Jones and Palmer 1987). In the second stage, the parameters of the receptive field were refined using a series of programs, each of which generated stacks of histograms as stimuli spanning some variable were presented in random order. Specifically, we generated at least one spatial-frequency tuning curve for each cell. In addition, several procedures were used to assure that the exact center coordinates of the receptive field were available for subsequent data acquisition. Along with the preliminary hand-plotting procedures, we used reverse correlation of the spike train with a dense two-dimensional spatiotemporal white noise stimulus (Reid et al. 1997). The position of the stimulus ensemble was modified to exactly center the most vigorous response. Additional details will be described in RESULTS.

Cells were classified as X, Y, and W according to a set of standard criteria (see Wolfe and Palmer 1998). For most cells, this included latencies to optic chiasm stimulation. Cells were also classified as lagged or nonlagged according to their responses to a spot covering both center and surround, which cycled between mean, bright, mean, dark contrasts or nonlagged according to their responses to a spot covering both center and surround, which cycled between mean, bright, mean, dark contrasts (usually 3–8 contrasts) (see Wolfe and Palmer 1998). For most cells, this included latencies at 0.5 Hz (Saul and Humphrey 1990; Wolfe and Palmer 1998).

RESULTS

A total of 69 LGN cells and 13 RGCs were studied in sufficient detail to be included in the results. For each cell, data were available for most or all of the multivariate protocols described. Cell counts and distributions will be given with each individual measurement.

Spatial summation is contrast dependent

The simplest demonstration of contrast-dependent spatial summation was achieved with a spatially homogeneous disk centered on the receptive field whose contrast was modulated sinusoidally in time, where the contrast \( C(t) \) at any given time \( t \) is given by

\[
C(t) = A \sin(2\pi ft)
\]

where \( A \) is the maximum contrast ranging between 5 and 80%, and \( f \) is the frequency. The disk was presented on a background of mean luminance, and its radius was varied in pseudorandom order. Each pass consisted of five cycles, presented at each of 26 sizes, and 5–10 passes constituted a dataset. There was no interstimulus interval, but the first cycle for each size was always discarded. Datasets were acquired for at least two contrasts (usually 3–5).

The response of an LGN X cell to a disk at three different contrasts is presented in Fig. 1. In Fig. 1A, the three sets of histograms correspond to responses to sinusoidal modulation at 10, 25, and 70% contrast at the radii shown on the left. The response amplitude (F1 in spikes/s) as a function of radius is plotted for each contrast in Fig. 1B. In each case, the response accrues as the radius of the disk increases up to some maximum and then falls slowly to a limiting value with further increases in radius. Error bars are SE, and the smooth curves are derived from the integral of the best-fitting DOGs (see next section). It is evident that response amplitude increases with contrast and that the disk eliciting the peak response shifts toward larger radii as the contrast decreases. Disk radii eliciting the peak response are plotted in Fig. 1C as a function of contrast. This value drops from 1.14° at 10% contrast to 0.65° at 70% contrast.

While recording from LGN cells, we were often able to simultaneously record S-potentials, the extracellularly recorded excitatory postsynaptic potentials (EPSPs) resulting from the firing of an RGC input to the relay cell (Kaplan and Shapley 1984). Using on-line template matching, we were able to simultaneously and independently extract both LGN and S-potential events. Twenty-five superimposed traces from such a simultaneous recording are shown in Fig. 2A. The large waveform is that of the LGN cell, and the small, earlier waveform is from an RGC. The corresponding correlogram is shown in Fig. 2B. The receptive fields of the LGN cell and its associated S-potential were precisely superimposed in every case. This was established by simultaneous reverse correlation of their spike trains with a dense two-dimensional spatiotemporal white noise stimulus (Reid et al. 1997) (Fig. 2, C and D). These data were collected for eight pairs of cells. The normalized inner product of the first-order responses obtained from the cells of the eight pairs ranged from 0.91 to 0.99 (mean, 0.96 ± 0.02), indicating that the receptive fields were not only co-spatial but also nearly identical.

Since the fields were superimposed, we looked at contrast-dependent spatial summation in both cells with a single stimulus, as shown in Fig. 2, E and F. Spatial summation curves, like those in Fig. 1, are shown for the retinal ganglion cell and the LGN cell for contrasts of 10, 20, and 40%. Plots of the disk radii eliciting the maximal response as a function of contrast are shown in Fig. 2, G and H. These data show that the radii eliciting the peak response shift toward higher values as contrast decreases for both members of the pair.

These data are typical of those obtained from all cells in our population. Figure 3 compares the disk radii at which the peak response was elicited for the highest (x-axis) and lowest (y-axis) contrasts available for each cell in our population. The high contrast was, on average, 6.7 times that of the low contrast. Data for LGN cells are shown in Fig. 3A (n = 69), and data for S-potentials are shown in Fig. 3B (n = 13). Nearly all the points are positioned above the line of unit slope,
showing that spatial summation extended to larger disks at lower contrast. The population mean is significantly different from 1.0 in both cases (1-sample t-test: LGN, $P < 0.0001$; RGC, $P < 0.0001$). The mean ratio of the disk radii eliciting maximal responses ($\text{Peak}_{\text{low contrast}} / \text{Peak}_{\text{high contrast}}$) was 1.49 for RGCs and 1.56 for LGN cells.

**Fitting with a DOG model**

The position of the peak in the spatial summation curve is not a direct measure of the size of the center mechanism (although it is highly correlated with it). Assuming a DOG receptive field model, it is actually the point at which the surround suppression overtakes the center excitation. To establish what spatial parameters are changing as the contrast changes, we fit our spatial summation curves to the two-dimensional, polar integral of the DOG (IDOOG) with four free parameters

$$F(r) = 2\pi K_c \int_0^r r e^{-\pi K_c r^2} dr - 2\pi K_i \int_0^r r e^{-\pi K_i r^2} dr$$

where $K_c$ and $K_i$ are the strengths, and $R_c$ and $R_i$ are the radii of the center and surround Gaussians, respectively. Fits were achieved with a simplex method modified from Press et al. (1988). Examples can be seen as the continuous lines in the spatial summation curves of Figs. 1 and 2.

We compared the values of these four parameters as extracted from high and low contrast spatial summation curves under the assumption that one or more should show a systematic relationship with contrast. These data are shown in the four parts of Fig. 4 (LGN (■), $n = 69$; RGC (○), $n = 13$). Here it is evident that the strongest correlation with contrast is the radius of the center, with almost all points lying above the line of unit slope, showing a larger center size at lower contrasts. The mean $R_c$ ratio ($R_c^{\text{high contrast}} / R_c^{\text{low contrast}}$) is significantly larger than 1.0 as expected (1-sample t-test: LGN, $P < 0.0001$; RGC, $P < 0.0001$). There is a weaker but still significant correlation between the strength of the center and contrast, with $K_c$ being smaller at higher contrasts (1-sample t-test: LGN, $P < 0.0001$; RGC, $P < 0.013$). The latter may be some
form of adaptation since the center is relatively less sensitive at higher contrasts. Neither parameter for the surround is significantly correlated with contrast (1-sample t-test: LGN, $K_i$: $P > 0.05$; $R_i$: $P > 0.05$; RGC, $K_i$: $P > 0.05$; $R_i$: $P > 0.05$).

The size of the excitatory region of the classical receptive field is often estimated by the peak of the spatial summation tuning curve (e.g., Kapadia et al. 1999). Here we have extracted the size of the excitatory region using fits with an IDOG.
and compared this value with the classical estimate of receptive field size. The two measures of excitatory receptive field size are not equal (2-sample t-test: LGN, $P < 0.001$; RGC, $P < 0.001$); however, they are significantly correlated (LGN: $r^2 = 0.86$, $P < 0.001$; RGC: $r^2 = 0.83$, $P < 0.0001$).

**Dependence of spatial-frequency cut-off and peak on contrast**

If the radius of the excitatory Gaussian is in fact changing with contrast, as the analysis suggests, this should be evident in other response properties of the cell. In particular, we would expect that the high spatial frequency cut-off should be higher at high contrasts when the radius of the excitatory Gaussian is smaller. Accordingly, we obtained spatial frequency tuning curves at a range of contrasts for each cell using a stimulus approximately three times the size of the excitatory center of the receptive field. The curves were fit using a DOG model, and the cut-off and peak for each curve was determined. Cut-off frequencies for the LGN cells are compared at high and low contrasts in Fig. 5A. As expected, nearly all the points lie below the line of unit slope, showing that cut-offs tend to be higher at the high contrasts, with the mean cut-off ratio ($SF_{\text{Cut-off high}}/SF_{\text{Cut-off low}}$) equal to 1.12. This population is significantly different from 1.0 (1-sample t-test, $P < 0.0003$). We also examined the peaks of the spatial frequency tuning curves at high and low contrast, and found that the mean peak ratio ($SF_{\text{Peak high}}/SF_{\text{Peak low}}$) is equal to 1.15 (Fig. 5B). This change is also significant (1-sample t-test, $P < 0.003$), and again, the peak increases with contrast. The RGCs also show significant increases in cut-off and peak spatial frequency as contrast is increased (1-sample t-test, cut-off: $P < 0.006$; peak: $P < 0.01$), although the spatial frequency data were only collected for six RGCs. These results are in partial agreement with a similar study done in primate V1 (Sceniak et al. 2002). That study showed a significant increase in spatial frequency cut-off with contrast but no significant change in the peak.

**Surround strength**

One may suspect that, as contrast increases, the relative strength of the surround suppression would increase, and this could provide an explanation for changes in spatial summation observed as contrast is increased. Surround suppression is often estimated using the percent reduction of the peak response for a given spatial summation tuning curve (e.g., DeAngelis et al. 1994). However, this measurement does not consider the spatial scale of the center and surround regions (Sceniak et al. 2001). Using the DOG model, we calculated a suppression index (SI) as a measure of the relative strengths of the surround and the center at any given stimulus contrast. The approximate areas under the excitatory ($K_e R_e$) and inhibitory

![Fig. 3](http://jn.physiology.org/)

**FIG. 3.** Spatial summation peaks. A and B: disk radii at which the peak response was elicited for the high (x-axis) and low (y-axis) contrasts for each cell in our population. LGN cells are shown in A ($n = 69$) and S-potentials in B ($n = 13$). Nearly all points are positioned above the line of unit slope, showing that spatial summation extended to larger disks at lower contrast. Average ratio of the disk radii eliciting maximal responses ($Peak_{\text{low contrast}}/Peak_{\text{high contrast}}$) was 1.49 for retinal ganglion cells and 1.56 for LGN cells. C: peak ratios ($Peak_{\text{low contrast}}/Peak_{\text{high contrast}}$) for RGC-LGN cell pairs plotted with RGC peak ratio on the y-axis and LGN peak ratio on the x-axis. On average, peak ratio for the RGC is similar to peak ratio of the LGN cell, although the population is small.
components are compared directly using the Gaussians identified from the model. As a result, the SI gives an index of inhibitory strength of the surround in the range of 0–1, with 0 signifying a complete lack of suppression and 1 signifying equal center and surround strengths. The difference between the SI at low and high contrasts measures the difference in surround suppression strength as contrast changes. If $D$ is positive, less surround suppression is present at high contrast than at low contrast, whereas if $D$ is negative, more surround suppression is present at high contrast than at low contrast.

The mean value of $D$ for the 69 LGN cells is 0.19, and for 84% of the LGN cells, $D > 0$. Thus for most LGN cells, the surround is actually weaker relative to the center at high contrasts (Fig. 6). Results for RGCs were virtually identical, with 85% having $D > 0$. Therefore surround suppression generally decreases with contrast rather than increasing as might be expected. We conclude, as did Sceniak et al. (2001), that changes in spatial summation with contrast are not the result of contrast-dependent changes in surround suppression.

Analysis by cell types

We examined the cell populations for any differences between cell types within the LGN and retina. The X, Y, and W
cell populations show no differences in contrast-dependent changes in the peaks of spatial summation curves, excitatory center sizes, and spatial frequency cut-offs and peaks (Table 1). Two exceptions were found, which concern the RG-X cells and the LGN-W cells, although they are most likely due to the small number of cells in those populations. For the four RG-X cells, the summation curve peak ratio and excitatory center size ($R_e$) ratio were $>1.0$ in each case; however, the means were not significantly different from 1.0 ($P > 0.05$). We also found that, for the three LGN-W cells for which spatial-frequency data were obtained, none showed a significant change in the peak or cut-off spatial frequency as contrast was varied. Excluding the mean $R_e$ ratio of the RG-X cells, the means of the X, Y, and W cell populations for each category examined were not significantly different from each other (ANOVA, $P > 0.05$, for each case). Spatial frequency data were only collected for six S-potentials and are not included in Table 1.

**DISCUSSION**

In this study, we showed that spatial summation in the LGN cells and RGCs is contrast dependent. The effect seems to be largely accounted for by a change in the actual size of the center mechanism. This conclusion is similar to that reached by Solomon et al. (2002) in the LGN of the marmoset. It is also similar to results obtained in primary visual cortex of primates, although the effect there is considerably larger (Cavanaugh et al. 2002; Kapadia et al. 1999; Sceniak et al. 1999).

**Simultaneous recording of LGN cells and RGCs**

For a few of our recordings from LGN cells (13/69), we were able to record from the associated S-potentials as well. S-potentials are known to be the extracellular manifestation of the EPSPs produced by a single RGC (Kaplan and Shapley 1984). In every instance, the receptive fields of the LGN–S-potential pair were exactly superimposed, and in fact, were nearly identical. This was established by simultaneous reverse correlation of the two spike trains with a single presentation of two-dimensional spatiotemporal white noise. The average normalized inner product of the two receptive fields was 0.96. Furthermore, the effects of contrast on spatial summation exhibited by each member of each pair were virtually identical. These data strongly imply that contrast-dependent spatial summation has its origins in the retina and is passed onto LGN cells by virtue of the powerful synapses between them (Mastronarde 1987a,b).

**Receptive field changes related to contrast**

Spatial summation was explored in both the space and spatial frequency domains. Since the fields of the LGN and RGC were superimposed, this allowed for simultaneous collection of spatial summation and spatial frequency tuning curves when paired recordings were obtained. In the space domain, responses were acquired to sinusoidal contrast modulation of a disk of varying radius. These summation curves were very detailed (26 radii) and independently fit with a two-dimensional IDOG function for each of several contrasts. From these fits, we extracted measures of the size and strength of both the center and surround and found that it is primarily the center size that changes consistently with contrast. In most cases, the apparent size of the center decreased with an increase in contrast for both LGN cells and RGCs. The center size was an average of 1.75 times greater at low contrast than at high contrast for the LGN cells and 1.99 times greater for RGCs. The curve fitting confirmed our observation at the time of data acquisition that the peak of the spatial summation curve moved toward lower values as the contrast increased (see Fig. 1).

In primate V1, Cavanaugh et al. (2002) have shown that contrast-dependent spatial summation is best accounted for...
TABLE 1. Comparisons of X, Y, and W cell populations with respect to contrast-dependent changes in the peaks of spatial summation curves, excitatory center sizes, and spatial frequency cutoffs and peaks

<table>
<thead>
<tr>
<th>Lateral Geniculate Nucleus</th>
<th>Spatial Summation</th>
<th>Retinal Ganglion Cells</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Mean Peak Ratio</td>
<td>Mean ( R_c ) Ratio</td>
</tr>
<tr>
<td>X (N = 32)</td>
<td>1.49 ± 0.43</td>
<td>1.66 ± 0.64</td>
</tr>
<tr>
<td>Y (N = 32)</td>
<td>1.48 ± 0.46</td>
<td>1.81 ± 0.69</td>
</tr>
<tr>
<td>W (N = 5)</td>
<td>1.70 ± 0.29</td>
<td>1.92 ± 0.51</td>
</tr>
</tbody>
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<table>
<thead>
<tr>
<th>Spatial Frequency</th>
<th>Mean Peak Ratio</th>
<th>Mean Cutoff Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>X (N = 22)</td>
<td>1.11 ± 0.23</td>
<td>1.09 ± 0.11</td>
</tr>
<tr>
<td>Y (N = 29)</td>
<td>1.18 ± 0.42</td>
<td>1.16 ± 0.27</td>
</tr>
<tr>
<td>W (N = 3)</td>
<td>1.03 ± 0.03</td>
<td>0.89 ± 0.10</td>
</tr>
</tbody>
</table>

Mean peak ratio = (Peak low contrast/Peak high contrast)
Mean \( R_c \) ratio = (\( R_c \) low contrast/\( R_c \) high contrast)
Mean cutoff ratio = (Cutoff low contrast/Cutoff high contrast)

Values are means ± SD. Spatial frequency data were only collected for 6 S-potentials and therefore are not included in the table.

were inferior to those obtained with the DOG model. Furthermore, attempts to fit multiple datasets obtained for a range of contrasts while specifying that \( R_c \) and \( R_e \) must be the same were hopelessly unacceptable (data not shown). This is not surprising since the two situations are quite different. In the cortex, the extraclassical receptive field is silent, whereas in the LGN and retina, the surround is actually part of the receptive field, and responses can be elicited from it. We infer from this exercise that the subtractive DOG model is more appropriate for the center-surround interaction of the RGCs and LGN cells.

In the spatial frequency domain, we acquired spatial frequency tuning curves at several contrasts. These were also fit to DOGs, and we found that the cut-off and optimal frequencies increased with contrast as expected if the receptive field center was getting smaller with contrast. These effects were seen in both LGN cells and RGCs. Thus the results from spatial and spatial-frequency measurements are in agreement. Sceniak et al. (2002) did not find changes in the peak spatial frequency as contrast was varied for cells in primate V1. In cortex, the optimal spatial frequency of a cell is most likely determined by the periodicity in the spatial weighting function, i.e., the distribution of its inputs rather than by the detailed characteristics of those inputs such as the absolute size of their centers. Therefore the peak spatial frequency of LGN cells could change with contrast and have no effect on the optimal spatial frequencies of cortical cells to which they project. We note that the effects of contrast are not likely to be caused by light scatter. Light scatter would increase with contrast, thus tending to decrease the cut-off and peak frequency and enlarge the apparent center radius, effects exactly opposite to those observed here.

Solomon et al. (2002) also found contrast-dependent changes in the size of the receptive field center of marmoset LGN cells. They reported a 31% increase in the center size as contrast changed from high to low. Our value in the cat is somewhat higher (75%). This could be a genuine species difference or may have arisen due to significant differences in technique.

In primary visual cortex of awake rhesus monkeys, Kapadia et al. (1999) reported a fourfold increase in receptive field length as contrast drops from high to low. Sceniak et al. (1999) and Cavanaugh et al. (2002), working in VI of anesthetized marmosets, reported a twofold increase.
macaque monkeys, reported an average 2.3- and 2.5-fold increase, respectively, as contrast drops from high to low. They observed this change for both the length and width dimensions of cortical receptive fields and based their findings on IDOG and ROG fits to spatial summation curves, respectively.

Extensive data for cat visual cortex are not currently available (but see Jagadeesh and Ferster 1990). This means that data comparing the contrast-dependent spatial summation in LGN and cortex are not available in any single animal. This is unfortunate since it makes it difficult to surmise on the significance of contrast-dependent spatial summation in retina and LGN for similar behavior seen at the level of cortex. Those working in cortex assume that cortical networks are exclusively responsible for the expansion of the receptive field at low contrasts. This may be largely true since the effect seems to be considerably larger in cortex than in the LGN (comparing cats and primates), but a significant contribution from the LGN cannot be ruled out given the findings reported here and by Solomon et al. (2002).

**Origins of contrast-dependent spatial summation**

The ratio of center sizes taken at low contrasts compared with high contrasts was 1.75 for LGN cells and 1.99 for RGCs. These are not significantly different (2-sample t-test: \( P > 0.05 \)). This fact, coupled with the observation that most action potentials produced by LGN cells are attributable to an antecedent spike in its RGC inputs (Cleland and Lee 1985; Mastronera 1987a,b), suggests strongly that contrast-dependent spatial summation first arises in the retina and is transmitted passively through the LGN to visual cortex. Furthermore, the effect was seen in all LGN and RGC cell types, and the magnitude of the effect did not differ significantly among cell types. Several studies have reported that the antagonistic effect of the surround on the center of the receptive field is stronger in LGN cells than in the RGCs (Hubel and Wiesel 1961). On average, we also found the mean \( S_{RGC} > S_{LGN} \). As a result, if the receptive field surround was responsible for contrast-dependent spatial summation, one would assume that the change in receptive field size from high to low contrast would be greater for the LGN cells compared with the RGCs. Our results show that this is not the case.

It is possible that the center and surround of the receptive field display contrast response functions with different slopes. If true, this could produce the contrast-dependent changes in center size that we see here. However, this seems unlikely given that the majority of our data show surround suppression is actually lower at high contrast \( (D > 0) \), which would tend to increase the apparent center size as contrast increases. Given this observation, we elected not to measure the contrast gain of the center and surround, a difficult measurement under the best of circumstances.

Since contrast-dependent spatial summation is seen in all cell types, it must arise within the retina somewhere prior to the ganglion cells. Since we see changes only in the size of the center, it is unlikely that the effect depends on horizontal cells since these contribute mainly to the surround (Lankheet et al. 1993). The centers of beta (X) ganglion cells are formed from bipolar cells, as are the subunits comprising the more distributed receptive fields of alpha (Y) cells. This center (or the subunits) is largely determined by the convergence of a small number of photoreceptors onto each bipolar cell, a connection that is modulated by horizontal cells (Demb et al. 1999). The photoreceptors themselves are coupled by gap junctions. Contrast-dependent coupling through these gap junctions or contrast-dependent horizontal cell modulation of the photoreceptor to bipolar synapse could theoretically serve to make center sizes contrast dependent.

It is also conceivable that contrast-dependent spatial summation arises from circuitry residing in the inner plexiform layer. A specific candidate here is the AII amacrine cell, which is coupled to cone Bipolars and to itself. This coupling is known to vary with the background illumination (Bloomfield et al. 1997; Smith and Vardi 1995; Vardi and Smith 1996), although an actual contrast dependence has not been shown.

**Functional implications**

There is logic to receptive field expansion at low contrasts—gathering information from more extensive regions of visual space at the cost of reduced spatial resolution. This is the standard explanation for the dramatic changes in receptive field dimensions seen in cortex as contrast (or signal-to-noise ratio) changes (Kapadia et al. 1999; Sceniak et al. 1999, 2001). The smaller effect seen at the level of retina and LGN may serve a similar purpose, but in the nature of signal conditioning prior to cortical processing. Just as the effects of LGN surrounds are not manifestly obvious at the level of cortex and are swamped by convergent inputs from many LGN cells, so also may the small changes in receptive field center sizes at the level of the LGN be overwhelmed by contrast-dependent intracortical processing.

**References**


