Contrast Sensitivity Is Enhanced by Expansive Nonlinear Processing in the Lateral Geniculate Nucleus

Thang Duong and Ralph D. Freeman

Group in Vision Science, School of Optometry, and Helen Wills Neuroscience Institute, University of California, Berkeley, Berkeley, California

Submitted 8 August 2007; accepted in final form 20 October 2007

Contrast sensitivity is enhanced by expansive nonlinear processing in the lateral geniculate nucleus. J Neurophysiol 99: 367–372, 2008. First published October 24, 2007; doi:10.1152/jn.00873.2007. The firing rates of neurons in the central visual pathway vary with stimulus strength, but not necessarily in a linear manner. In the contrast domain, the neural response function for cells in the primary visual cortex is characterized by expansive and compressive nonlinearities at low and high contrasts, respectively. A compressive nonlinearity at high contrast is also found for early visual pathway neurons in the lateral geniculate nucleus (LGN). This mechanism affects processing in the visual cortex. A fundamentally related issue is the possibility of an expansive nonlinearity at low contrast in LGN. To examine this possibility, we have obtained contrast–response data for a population of LGN neurons. We find for most cells that the best-fit function requires an expansive component. Additionally, we have measured the responses of LGN neurons to m-sequence white noise and examined the static relationship between a linear prediction and actual spike rate. We find that this static relationship is well fit by an expansive nonlinear power law with average exponent of 1.58. These results demonstrate that neurons in early visual pathways exhibit expansive nonlinear responses at low contrasts. Although this thalamic expansive nonlinearity has been largely ignored in models of early visual processing, it may have important consequences because it potentially affects the interpretation of a variety of visual functions.

INTRODUCTION

The standard Weiner system, consisting of a linear component followed by a static nonlinearity, has been used extensively as a functional model for neurons in the retina, lateral geniculate nucleus (LGN), and primary visual cortex (Nykamp and Ringach 2002). This model is accurate for neurons in the retina and LGN, although other nonlinearities exist (Chichilnisky 2001; Victor 1987; Victor and Shapley 1979). Specifically, in the LGN and retina, a contrast gain control mechanism decreases the neuronal response gain with increasing stimulus contrast. This causes response saturation at high contrasts. A recent model proposes that the underlying mechanism for contrast gain control in LGN neurons is a suppressive region within the receptive field (Bonin et al. 2005). This model consists of three components: a linear classical receptive field, a nonlinear suppressive field, and a response rectification. The nonlinear suppressive field serves to compute local contrast of the stimulus. This local contrast then decreases the linear receptive field gain by divisive normalization. A final linear threshold function converts this signal to a positive spike rate.

In this suppressive field model, as stimulus contrast increases, the local contrast within the suppressive field also increases, which decreases the response gain. This decrease in gain causes a saturation of response with increasing stimulus contrast. An assumption of this model is that the static nonlinear component is simply a linear rectifying function. This is of primary significance because it implies an approximately linear response at low contrast levels where the suppressive field is minimally activated. If in fact there is a clear nonlinearity at low contrasts, this addition can improve the predictive power of the suppressive field model, and it also could have implications for the interpretation of visual function at low contrasts.

We have examined this issue by obtaining contrast–response functions from extracellular recordings of neurons in the cat’s LGN. We fit the data with a Naka–Rushton function (Naka and Rushton 1966). Results show significant expansive nonlinearities at low contrasts. Additionally, we have measured directly the static nonlinear function by comparing a linear prediction with actual spike responses. The linear predictions are generated from spatiotemporal receptive fields obtained by m-sequence stimulation. Static nonlinear functions for the majority of neurons of our sample exhibit power-law nonlinearities with a mean exponent of 1.58. These results show a clear expansive nonlinear component in LGN neurons for low-contrast visual stimuli. It is likely that this has important consequences for basic response properties of cortical neurons.

METHODS

Physiological preparation

All procedures complied with the National Institutes of Health Guide for the Care and Use of Laboratory Animals. Extracellular recordings were made using epoxy-coated tungsten microelectrodes in the LGN of anesthetized and paralyzed mature cats. Cats were initially anesthetized with isoflurane (1–4%). After catherization, a continuous infusion was given of a combination of fentanyl citrate (10 μg·kg⁻¹·h⁻¹) and thiopental sodium (6 mg·kg⁻¹·h⁻¹). Bolus injections of thiopental sodium were given as required during surgery. After a tracheal cannula was positioned, isoflurane was discontinued and the animal was artificially ventilated with a mixture of 25% O₂ and 75% N₂O. Respiration rate was manually adjusted to maintain an end-tidal CO₂ of 34–38 mmHg. Body temperature was maintained at 38°C with a closed-loop controlled heating pad. A craniotomy was performed to expose the LGN. To examine this possibility, we have obtained contrast–response data for a population of LGN neurons. We find for most cells that the best-fit function requires an expansive component. Additionally, we have measured the responses of LGN neurons to m-sequence white noise and examined the static relationship between a linear prediction and actual spike rate. We find that this static relationship is well fit by an expansive nonlinear power law with average exponent of 1.58. These results demonstrate that neurons in early visual pathways exhibit expansive nonlinear responses at low contrasts. Although this thalamic expansive nonlinearity has been largely ignored in models of early visual processing, it may have important consequences because it potentially affects the interpretation of a variety of visual functions.

The costs of publication of this article were defrayed in part by the payment of page charges. The article must therefore be hereby marked “advertisement” in accordance with 18 U.S.C. Section 1734 solely to indicate this fact.
performed over the LGN and the dura was resected and covered with agar and wax to form a closed chamber. After completion of all surgical procedures, continuous injection of fentanyl citrate was discontinued, and thiopental sodium concentration was lowered gradually to a level at which the cat was stabilized for ≥1 h. The level of anesthetic used was determined individually for each cat. The range used was 1.0–3.0 mg·kg⁻¹·h⁻¹ and a typical level was 1.5 mg·kg⁻¹·h⁻¹. Once a stabilized anesthetic level was reached, it was kept constant throughout the experiment. To minimize eye movements during visual tests, animals were immobilized with pancuronium bromide (0.2 mg·kg⁻¹·h⁻¹). EEG, ECG, heart rate, temperature, end-tidal CO₂, and intratracheal pressure were monitored for the entire duration of the experiment. Contact lenses were used that were opaque except for a central 4-mm-diameter window to create an artificial pupil. To focus the eyes on the stimulation screen, ophthalmoscopic refraction was used to determine appropriate lens power.

For cortical data, electrode penetrations were made at Horsley–Clarke coordinates P4L2 angled 20° anterior and 10° medial. Electrodes were advanced until visually responsive cells were found. LGN electrode penetrations were made perpendicular to the cortical surface at Horsley–Clarke coordinates of approximately A6L9. Electrodes were then advanced until visually responsive cells with LGN response characteristics were found (typically around 12 mm below the cortical surface). Recordings were made from all layers of the LGN.

**Extracellular recording**

Single units were isolated in real time by the shape of their spike waveforms using custom software. An initial estimate of the tuning parameters was made qualitatively by computer-controlled manipulation of drifting sinusoidal gratings. The spatial extent of visual stimulation was kept larger than the receptive field size. Temporal frequency tuning curves were measured with drifting sinusoidal gratings at 50% contrast. Spatial frequency and contrast tuning curves were measured at optimal temporal frequencies determined for each cell, typically between 4 and 15 cycles/s.

**Visual stimulation**

Visual patterns consisting of sinusoidal gratings or noise patterns were presented on a large CRT at a frame rate of 75 Hz. The 47.8-cm-diameter CRT was positioned at an optical distance of 41.8 cm in front of the cat’s eyes, and split so that each half of the display stimulated the left or right eye. Luminance from the CRT was calibrated for a linear range with maximum and minimum values of 90 and 0.1 cd/m², respectively.

**Data analysis**

For contrast tuning data, the first harmonic (F1 component) was used for analysis. All data fitting was done by minimizing the sum squared error using *fminsearch* in Matlab (The MathWorks, Natick, MA), which implements the Nelder–Mead nonlinear minimization algorithm. For m-sequence analysis, spikes were first binned with a window of one m-sequence frame (typically ~13 ms). Reverse correlation was done using the fast m-transform method (Reid et al. 1997; Sutter 1991), and the first-order kernel was extracted from m-transformed data. Estimation of the static nonlinearity was done by comparing the linear prediction to the actual spike response. Linear predictions were generated by convolving the linear spatiotemporal receptive fields with the m-sequence stimulus. Details of this analysis are given elsewhere (Anzai et al. 1999a; Chichilnisky 2001).

**RESULTS**

To evaluate contrast tuning, we collected complete data for 168 LGN neurons. Visual stimulation by m-sequence tests was run on 250 LGN cells. These two populations of LGN neurons were partially overlapping. Additionally, we tested 96 simple cells in area 17 with m-sequence stimulation. Two visual stimulation protocols were used in this study. In the first protocol, responses to drifting gratings were measured with approximately optimal spatial and temporal frequencies at...
different contrast levels, as illustrated in Fig. 1A. Contrast in this experiment is defined in the standard way as

\[ c = \frac{L_{\text{max}} - L_{\text{min}}}{L_{\text{max}} + L_{\text{min}}} \]  

where \( L_{\text{max}} \) and \( L_{\text{min}} \) are maximum and minimum contrast values. Each condition consists of 1 s of stimulation followed by a 2 s rest period during which the screen is blank and is displayed at the mean luminance level. This protocol was run on 168 cells. The second protocol is a standard binary m-sequence stimulation procedure (Anzai et al. 1999a,b; Reid et al. 1997). Briefly, the stimulus field is split into a grid of either 8 \( \times \) 8 or 16 \( \times \) 16 elements centered on the receptive field. For cortical neurons, the dominant eye is stimulated. Luminance of individual grid elements was modulated at 75 Hz following a 14-bit binary m-sequence. An inverse repeat stimulation was always used (Reid et al. 1997; Sutter 1991), and multiple repetitions were presented per neuron as needed based on the signal-to-noise ratio of the response. The modulation was at 100% contrast so that each grid element can have a luminance of either 0.1 or 90 cd/m\(^2\). Spatiotemporal receptive fields were calculated using the fast m-transform (Reid et al. 1997; Sutter 1991). Figure 1C illustrates this stimulus. For both visual stimulation protocols, gratings or white noise patterns larger than the receptive field sizes were used.

The first issue is to characterize the manner by which neurons in the LGN increase their responses with stimulus contrast. This contrast–response data can be described by the Naka–Rushton function (Albrecht and Hamilton 1982; Naka and Rushton 1966; Solomon et al. 2004)

\[ r(c) = \frac{R_{\text{max}} c^n}{c^n + c_{50}^n} + b \]  

where \( r(c) \) is the neural response at contrast \( c \); \( n, R_{\text{max}}, c_{50} \), and \( b \) are free parameters. For \( n > 1 \), this function is expansive at low-contrast levels and compressive at high contrasts with an inflection point at

\[ c_i = c_{50}^{\frac{n-1}{R+1}} \quad n > 1 \]  

so that \( r(c) \) exhibits an expansive nonlinearity for contrasts \(< c_i \) and a saturation nonlinearity for contrasts \(> c_i \). Figure 1, B and

FIG. 2. Best Naka–Rushton fit values to the contrast–response data are presented in histogram form for a population of 168 LGN neurons. A, B, and C: histograms giving values for \( n, c_{50} \), and \( c_i \), respectively, as computed using Eqs. 2 and 3. Unfilled arrows indicate the mean values for each distribution. D–F: contrast–response data are presented for 3 representative LGN neurons tested at approximately optimal spatial and temporal frequencies. D: 0.4 cpd and 14 Hz. E: 0.1 cpd and 10 Hz. F: 0.5 cpd and 4 Hz. Solid and dashed lines denote, respectively, the best-fit Naka–Rushton functions (Eq. 2), and the condition in which the numerator exponent is set to 1 (Eq. 5). Corresponding \( R^2 \) values for the fits are: 0.96 and 0.80 (D); 0.98 and 0.73 (E); 0.98 and 0.94 (F).

FIG. 3. Contrast–response data are presented as histograms for our population of 168 LGN neurons. A: \( R^2 \) values of the best fits are given with (filled bars) and without (unfilled bars) an expansive nonlinearity. Filled and unfilled arrows indicate, respectively, mean values of conditions with and without an expansive nonlinearity. B: \( R^2 \) values are depicted of the best fits with expansive nonlinearity (x-axis) vs. without expansive nonlinearity (y-axis).
Figure 1B shows the Naka–Rushton function (solid curve) and, at low contrasts, the best-fit power-law function (dashed curve) given by

\[ r(c) = ac^n + b \]  (4)

where \( a \), \( b \), and \( n \) are free parameters. The dotted curve shows an example Naka–Rushton fit without the expansive component. Details of this function are subsequently given in Eq. 5. Filled and unfilled arrows denote \( c_{50} \) and \( c_i \), respectively. A comparison of \( n \) for Eqs. 2 and 4 shows that the best-fit power-law exponent \( n \) (y-axis) is lower than that of the corresponding Naka–Rushton value (x-axis) as shown in Fig. 1D. The data points here (open circles) are calculated by fitting Eq. 4 to the Naka–Rushton functions with various values of \( n \). The best-fit exponents in Eq. 4 are then plotted on the y-axis. The y = x unity solid line here is clearly above the data points.

Recent studies and models assume that contrast–response data for LGN neurons follow the Naka–Rushton function with \( n = 1 \) (Bonin et al. 2005; Li et al. 2006; Priebe and Ferster 2006). This assumption means that the contrast–response function of LGN neurons does not exhibit an expansive component. It implies that LGN neurons undergo saturation at all contrast levels above firing threshold. However, our measurements, as subsequently described, are at odds with this assumption. They demonstrate that LGN neurons exhibit expansive power-law nonlinearities when stimulated with low-contrast gratings. To quantify this observation, we measured the neural responses to gratings at different contrasts for our population of LGN neurons and calculated the best-fit Naka–Rushton function to these data. Figure 2, A, B, and C shows the best fits for \( n \), \( c_{50} \), and \( c_i \), respectively, for 168 LGN cells. The mean and SE values for \( n \), \( c_{50} \), and \( c_i \) are 2.47 ± 0.162, 68.89 ± 5.45, and 27.42 ± 2.01, respectively. The medians for \( n \), \( c_{50} \), and \( c_i \) are 2.03, 38.87, and 17.72, respectively, and the modes are 1.758, 27.51, and 13.91, respectively. Mean values are indicated by unfilled arrows above the histograms. Note that these distributions are non-normal.

To test whether an expansive nonlinearity is necessary to describe the contrast–response function, we also fit a modified Naka–Rushton function to the data. This modified function has no expansive component and is given by the following equation

[Graphs and Figures]
where the exponent in the numerator of the Naka–Rushton function is removed. Note that for the common case when the exponent of both the numerator and denominator is set to one, the fit is equal to or worse than that with Eq. 5. A plot of example functions from Eqs. 2, 4, and 5 is given in Fig. 1B (solid, dashed, and dotted, respectively). Figure 2, D–F shows the contrast–response data and best-fit Naka–Rushton functions for three representative LGN neurons. The dashed and solid lines denote best-fit functions with (Eq. 2) and without (Eq. 5) expansive nonlinearity, respectively. Clearly, for these cells, the data are better described with the expansive nonlinear component than without. The differences in fits may have substantial consequences as considered below. We quantify this relationship further in Fig. 3. For each cell, R² values are calculated using the best-fit function with and without expansive nonlinearity. Figure 3A shows the R² values of the fit with and without the expansive component. Mean and SE with and without expansive nonlinearity are 0.9233 ± 0.0110 and 0.8191 ± 0.0130, respectively. Mean values are indicated by unfilled and filled arrows, respectively. Of the two histograms, the one with expansive nonlinearity is more clearly weighted toward an R² value of 1. Figure 3B shows a scatterplot of R² values for our population of 156 neurons, which compares results with and without an expansive nonlinearity. The scatterplot is also weighted extensively toward the expansive nonlinearity side of the y = x line. Considered together, the data in Fig. 3 show clearly that an expansive nonlinearity provides a better explanation for the data.

Presumably for stimulation with low-contrast gratings, the effect of contrast gain control is minimal, and expansive nonlinearity is due entirely to static nonlinearity. To examine this possibility, we measured the static nonlinearity for 250 LGN neurons by comparing a linear prediction and actual spike response levels. The linear prediction is generated from the spatiotemporal receptive field by m-sequence stimulation (see METHODS). Figure 4 shows linear spatiotemporal receptive fields (A, B, D, E, G, and H) and static nonlinearities (C, F, and I) for three representative LGN neurons. Space–time (Fig. 4, A, D, and G) and x–y space (Fig. 4, B, E, and I) contour plots are shown. Green and red represent bright and dark excitatory responses, respectively. Greater color saturation represents higher response rate, and each contour line denotes a region of equal response level. For all three cells in Fig. 4, the relationship between actual response and linear prediction follows a power-law function with exponents of 3.03 (Fig. 4, A, B, and C), 2.64 (Fig. 4, D, E, and F), and 1.50 (Fig. 4, G, H, and I). For static nonlinearity plots given in Fig. 4, C, F, and I, the y-axis denotes actual response to m-sequence stimulation binned at one m-sequence frame window (13 ms) and averaged across all repetitions. For each actual response value, linear prediction varies through the course of the m-sequence stimulation. The x-axis denotes the average linear prediction for each corresponding actual response. Error bars denote SE of the prediction for each corresponding actual response.

We calculated the best-fit parameters to Eq. 4. Figure 5A shows the histogram of best-fit exponents for the LGN. The mean ± SE of the exponents across this population is 1.580 ± 0.004, and the median and mode are 1.408 and 1.319, respectively. The mean is indicated by the open arrow in Fig. 5A.

A direct comparison of the value of n for a static nonlinear power law versus the Naka–Rushton contrast–response function is difficult to make because other nonlinearities such as those imposed by a suppressive nonclassical receptive field may play a role in dissociating these two values (Bonin et al. 2005, 2006; Chander and Chichilnisky 2001). However, the data in Fig. 1D show that a mean power-law exponent of 1.58 approximates an n > 2 in the Naka–Rushton function. Therefore our results for both analysis approaches are consistent and show that LGN neurons exhibit an expansive nonlinearity at low stimulus contrast. The consistency between these two methods suggests that a static power-law nonlinearity as estimated by m-sequence stimulation contributes substantially to expansive nonlinearity in the contrast tuning data.

Finally, for comparison to data from visual cortex, we show the exponent distribution for an expansive static nonlinearity for 160 cortical neurons in Fig. 5B. This distribution has a mean (open arrow) and SE of 2.4 ± 0.2, which is higher than that for the LGN population. Note that many neurons in the cortical population have large exponents (>4). This reflects more pronounced expansive nonlinearities in visual cortex as compared with LGN.

**Discussion**

Nonlinearities exist in various forms at various stages of the early visual pathway. In retinal ganglion and LGN neurons, a gain control mechanism introduces distinct nonlinear response properties. First, a response phase advance is observed with increasing stimulus contrast. Second, the transfer characteristics and response gain are altered with stimulus contrast (Sclar 1987; Victor 1987). These properties also exist in the primary visual cortex and are attributed to a divisive normalization mechanism (Carandini...
and Heeger 1994; Carandini et al. 1997). Additionally, neurons in the primary visual cortex also exhibit a power-law expansive nonlinearity when stimulated at low contrasts (Albrecht and Geisler 1991; Carandini 2004; DeAngelis et al. 1993; Gardner et al. 1999; Miller and Troyer 2002).

Models of visual processing in the LGN and visual cortex have largely ignored any thalamic expansive nonlinearity (Bonin et al. 2005; Li et al. 2006; Priebe and Ferster 2006). In the current study, we show that neurons in the LGN also exhibit a power-law expansive nonlinearity when activated by low-contrast visual stimuli. This expansive nonlinearity is likely to be the origin of expansive nonlinearity in membrane potentials of cortical neurons when stimulated at low contrasts (Ahmed et al. 1997; Contreras and Palmer 2003). We suggest, therefore, that expansive nonlinearity in the visual pathway originates early and is enhanced at various stages.

Although our measurements were made in LGN, the origin of expansive nonlinearities is probably in the retina. However, we should point out that direct neural input is not the only way to produce an expansive nonlinearity. It can also be produced as a by-product of neural noise. Random fluctuations in the membrane potential can make subthreshold responses “visible” in the presence of a threshold spiking mechanism, which can cause spike responses near threshold to simulate an expansive nonlinearity (Miller and Troyer 2002). Given these factors, it is possible that an expansive nonlinearity originates in the retina and gradually increases in magnitude as transmission progresses in a feedforward manner along the visual pathway. Our data for LGN and visual cortex are consistent with this hypothesis.

The existence of a low-contrast expansive nonlinearity is not consistent with recent feedforward models of cross-orientation suppression in the primary visual cortex. In these models, response saturation with increasing contrast in LGN is thought to underlie cross-orientation suppression in primary visual cortex (Li et al. 2006; Priebe and Ferster 2006). For this to be true, the level of contrast saturation must match that of cross-orientation suppression at all contrast levels. We show here that, on average, the contrast–response function is expansive for contrasts < 27% (see Fig. 2C). This would cause cross-orientation facilitation, not suppression, in the visual cortex. However, cross-orientation suppression is present in the visual cortex even with gratings at contrasts < 27% (DeAngelis et al. 1992; Freeman et al. 2002; Li et al. 2006). Therefore at low stimulus contrast, another mechanism must be involved in cross-orientation suppression (Li et al. 2006).

Finally, it is relevant to consider possible consequences of a low-contrast expansive nonlinearity. In general, an expansive nonlinearity should contribute to a sharpening of tuning curves for different stimulus dimensions. This would occur by an increase in the slope of response functions so that small changes in the stimulus would generate relatively large changes in spike rates of neurons. This could apply to spatial frequency selectivity. It could also be relevant to orientation because tuning properties of orientation and spatial frequency of neurons in the visual cortex are related (Webster et al. 1990). In a similar fashion, an expansive nonlinearity at low-contrast levels could increase contrast sensitivity by steeper slopes in contrast tuning functions. This would also yield low thresholds or high-contrast sensitivities. This accentuation of sensitivity could be highly significant in a practical sense because most visual performance occurs in a relatively low contrast environment (Mante et al. 2005).

**Grants**

This work was supported by National Eye Institute Grants EY-01175 and EY-03176.

**References**


