Visual Neuroscience (2000), **17**, 71–76. Printed in the USA.

Differential contributions of magnocellular and parvocellular pathways to the contrast response of neurons in bush baby primary visual cortex (V1)

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(RECEIVED May 1, 1997; ACCEPTED August 11, 1999)

Abstract

How neurons in the primary visual cortex (V1) of primates process parallel inputs from the magnocellular (M) and parvocellular (P) layers of the lateral geniculate nucleus (LGN) is not completely understood. To investigate whether signals from the two pathways are integrated in the cortex, we recorded contrast-response functions (CRFs) from 20 bush baby V1 neurons before, during, and after pharmacologically inactivating neural activity in either the contralateral LGN M or P layers. Inactivating the M layer reduced the responses of V1 neurons ($n = 10$) to all stimulus contrasts and significantly elevated ($t = 8.15$, $P < 0.01$) their average contrast threshold from 8.04 ($\pm$ 4.1)% contrast to 22.46 ($\pm$ 6.28)% contrast. M layer inactivation also significantly reduced ($t = 4.06$, $P < 0.01$) the average peak response amplitude. Inactivating the P layer did not elevate the average contrast threshold of V1 neurons ($n = 10$), but significantly reduced ($t = 4.34$, $P < 0.01$) their average peak response amplitude. These data demonstrate that input from the M pathway can account for the responses of V1 neurons to low stimulus contrasts and also contributes to responses to high stimulus contrasts. The P pathway appears to influence mainly the responses of V1 neurons to high stimulus contrasts. None of the cells in our sample, which included cells in all output layers of V1, appeared to receive input from only one pathway. These findings support the view that many V1 neurons integrate information about stimulus contrast carried by the LGN M and P pathways.

Keywords: Parallel pathways, Striate cortex, Vision, Receptive field, Primate

Introduction

The lateral geniculate nucleus (LGN) of primates receives anatomically and functionally segregated inputs from the retina and relays the signals contained in these parallel afferent sources to layer IV of the primary visual cortex (V1) (Casagrande & Norton, 1991). The parvocellular (P) and magnocellular (M) geniculocortical pathways differ markedly in their anatomy, physiology, and termination patterns within V1 (Norton & Casagrande, 1982; Derrington & Lennie, 1984; Derrington et al., 1984). Whether the signals relayed by these parallel pathways remain segregated or converge after their initial entry into V1 is an unresolved question (Merigan & Maunsell, 1993). Early studies in macaque monkeys, for example, found that the responses of V1 neurons to stimulus color, spatial detail, and/or motion reflect the properties of specific geniculate inputs (Livingstone & Hubel, 1984, 1988; Ts'o & Gilbert, 1988). These results led to the conclusion that LGN inputs are segregated within V1. More recent investigations describe a unified distribution of V1 cell response properties, implying that the LGN inputs become integrated within V1 (Lennie et al., 1990; Leventhal et al., 1995; Sawatari & Callaway, 1996).

Because the spatial properties of many stimuli excite both LGN pathways to varying degrees (Casagrande & Norton, 1991), distinctions between the M and P pathways are usually revealed only by stimuli at the extreme spatiotemporal limits. This makes identification of the contributions of the M and P inputs to V1 cell responses difficult and contributes to the inconsistent published results. One solution to this problem is to manipulate stimulus contrast. Magnocellular neurons in the LGN of both macaque monkeys and bush babies exhibit lower contrast thresholds and saturate at lower contrast levels than P cells (Kaplan & Shapley, 1982; Derrington & Lennie, 1984; Norton et al., 1988). The isolated contribution of the M pathway to responses of V1 neurons should therefore be revealed by low contrast stimulation. A second solution is to pharmacologically isolate *in vivo* individual LGN M or P layers (Malpeli et al., 1981; Nealy & Maunsell, 1994), then measure the activity of V1 neurons innervated by this layer. In com-

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bination, recording the contrast-response functions of V1 neurons while inactivating individual LGN layers offers a means of identifying the contribution to V1 cell responses made by both types of LGN inputs.

Our objective in this study was to determine the contributions of the LGN M and P pathways to the contrast-dependent responses of individual V1 neurons. We chose to perform these studies in bush babies because the parallel pathways are well segregated in this species (Norton & Casagrande, 1982; Florence & Casagrande, 1987; Lachica & Casagrande, 1992; Casagrande, 1994). An additional advantage is that the bush baby LGN is structurally simpler than that of macaque monkeys with only two P layers and two M layers (see Fig. 1). This structure allowed us to test the contributions of M and P pathways to cells within the central vision representation of V1. Finally, the physiological properties of both LGN cells and V1 cells have been well studied in bush babies (Norton & Casagrande, 1982; Irvin et al., 1986; Norton et al., 1988; DeBruyn et al., 1993; Allison et al. 1993, 1995), making interpretation of the results less problematic.

Methods

Seven adult bush babies (*Galago crassicaudatus*; 1.0–1.5 kg) were anesthetized with Brevital (methohexital sodium; 1 mg/kg-h) and nitrous oxide (70%), paralyzed with Pavulon (pancuronium bromide; 1.9 mg/kg-h), and surgically prepared for recording from V1 and LGN. All surgical procedures and methods of monitoring anesthesia levels were identical to those used by DeBruyn et al. (1993) and adhere to those approved by NIH and the Vanderbilt University Animal Care Committee.

Prior to the initiation of this study, several control experiments were performed to determine empirically the magnitude of the spread of injectable agents in LGN and the time course of LGN inactivation. Recording electrodes were glued at distances of 50–500 μm from the centers of pipettes filled with 25 mM gamma-aminobutyric acid (GABA) in saline (pH 7.4). Recordings were then made in the LGN following injections of either 500 or 1000 nl GABA. With 500 nl of GABA, activity was completely blocked at a distance of 300 μm (diameter of 600 μm) but not 500 μm (diameter of 1000 μm) for 3.5–5.0 min. This volume of GABA was chosen for subsequent LGN layer blocking experiments because the predicted block would be expected to silence all of contralaterally innervated P layer 6 (200–300 μm in diameter) but not reach contralaterally innervated M layer 1 (800–1000 μm below the center of P layer 6) (see Fig. 1).

For layer blocking experiments, a microelectrode was lowered into V1 until a receptive field could be plotted. An injection/recording electrode (Needlecraft, Frederick Haer, Brunswick, ME) filled with 25 mM GABA was then lowered into either LGN M layer 1 or P layer 6, multiunit activity was recorded, and the electrode was repositioned until the LGN and V1 receptive fields were retinotopically aligned. The retinotopic alignment for each subsequently recorded V1 cell was checked to ensure that its receptive field overlapped with the LGN receptive field. After the electrodes were positioned, action potentials were isolated from a single V1 neuron and its contrast-response function (CRF) was measured by stimulating with spatiotemporally optimized, drifting sine-wave gratings (4-s duration) randomly interleaved in 0.15 log unit increments from 3 to 56% contrast (display was a Tektronix 608; mean luminance = 40 cd/m2; stimulus diameter = 10 deg; for details see Bonds, 1991; DeBruyn et al., 1993). Each contrast was presented three times for a total averaging time of 12 s. After measuring the baseline CRF, approximately 500 nl of GABA was pressure injected into either LGN M layer 1 or P layer 6 and the V1 cell's CRF was remeasured immediately. As described above, we chose to independently inactivate either LGN M layer 1 and P layer 6 because they lie at the ventral and dorsal edges, respectively, of the nucleus (Fig. 1). We could therefore reliably inactivate either layer independently without GABA spreading to the other layer. We confirmed the inactivation of local LGN neural activity by recording immediately after injection. Although we were unable to measure the size of the pharmacologically induced scotoma, inactivating *at least* a 600-μm diameter area of LGN tissue likely created a scotoma much larger than the average LGN receptive field (2 deg $\times$ 3 deg) recorded from the multiunit activity and was probably larger than the average cortical cell receptive field (2 deg $\times$ 5 deg). Furthermore, M layer 1 and P layer 6 are innervated by the contralateral retina. We ensured that V1 cells were stimulated only *via* the contralaterally innervated LGN layer by occluding the ipsilateral eye (schematically illustrated in Fig. 1 by the shaded areas through LGN layers 2, 3, and 4). The measurement was repeated until the V1 cell's CRF returned to baseline levels, usually within 20 min. Lesions marked each LGN and V1 electrode penetration. Cell locations in relation to V1 layers and cytochrome oxidase (CO) blobs and interblobs were determined.

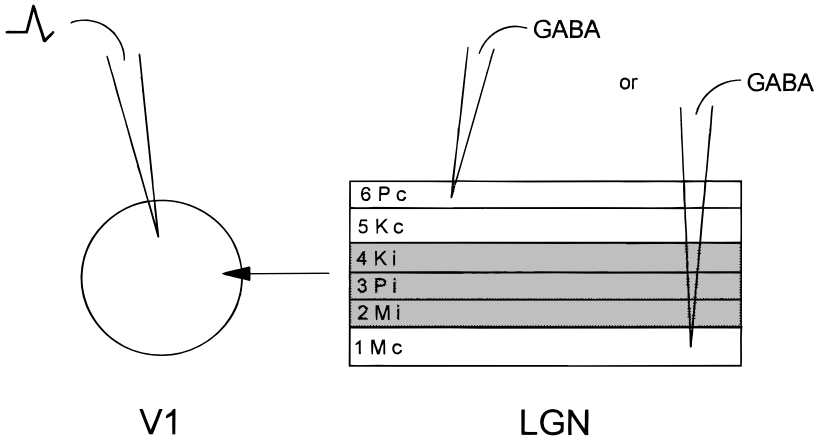

Fig. 1. Schematic diagram of the experimental paradigm showing the position of the GABA pipettes within the LGN and the recording electrode in V1. The anatomical organization of the bush baby LGN is ideal for this study because LGN M layer 1 and P layer 6 are at the edges of the nucleus more than 1.3 mm apart. In addition to the M and P layers, the LGN contains a third cell class that projects to V1, the koniocellular (K) cells (contralaterally innervated layer 5 and ipsilaterally innervated layer 4) (Irvin et al., 1986; Lachica & Casagrande, 1992). If K cells contribute to the contrast-response function of V1 cells, then the responses of V1 neurons after inactivation of M layer 1 possibly reflect the combined influence of the P and K pathways. Numerals refer to the LGN layers. c: contralateral; i: ipsilateral; M: magnocellular; and P: parvocellular. See text for details.

A linear regression analysis of the linear portion of the CRFs determined quantitatively each cell's average (±99% confidence interval) contrast threshold before, during, and after layer inactivation (Allison et al., 1993). The 99% confidence interval was also established for each cell's peak response to the highest contrast stimulus (i.e. 56%). A change in the contrast threshold or peak response of an individual V1 neuron was considered significant if it was beyond the 99% confidence interval of the baseline measure. For the two samples of V1 neurons (i.e. those recorded during M layer blocks and those recorded during P layer blocks), differences between control and experimental conditions were evaluated using a paired Students *t*-test.

Results

We recorded contrast-response functions (CRFs) from a total of 20 V1 neurons (seven simple cells and 13 complex cells). Inactivating neural activity in the LGN M layer 1 ($n = 10$) reduced the responses of V1 neurons to all stimulus contrasts. For example, CRFs recorded from a layer V simple cell under control (*filled circles*) and recovery (*open circles*) conditions were virtually identical (Fig. 2A). The neuron's baseline contrast threshold was 10.2 (± 2.1)% contrast. Injecting GABA into LGN M layer 1 (*filled squares*) reduced the neuron's response to each stimulus contrast and significantly elevated (i.e. beyond the 99% confidence interval) its contrast threshold to 25.2 (± 4.3)% contrast. Additionally, the neuron's peak response to 56% contrast was reduced significantly. The cell's response to stimuli above 20% contrast while M layer 1 was inactivated likely reflects the contribution of input from the P pathway.

Inactivating LGN M layer 1 increased significantly the contrast threshold of every cell tested (Fig. 2B). The average (± standard deviation) baseline contrast threshold of the sample was 8.04 (±4.01)% contrast. Inactivating M layer 1 elevated significantly ($t = 8.15$, $P < 0.01$) the average contrast threshold to 22.46 (±6.28)%. For eight cells, inactivating M layer 1 also reduced significantly their peak response to 56% contrast (Fig. 2C). The average peak response of these eight cells was reduced significantly ($t = 4.06$, $P < 0.01$) by 48.4 (±28.4)%. We must emphasize that the spatial and temporal frequencies of the stimulus were optimized for each recorded cortical neuron. The average spatial and temporal frequencies used during inactivation of the LGN M layer were 0.48 (±0.18) cycles/deg and 1.6 (±0.97) Hertz. These values are not "optimal" for LGN P neurons in *galago* (Norton et al., 1988), but are within the excitatory range. The eccentricity of this sample of V1 neurons ranged from 1 to 10 deg (mean = 6.1 deg).

As shown by the CRFs recorded from a layer IVB simple cell (Fig. 3A), injecting GABA into P layer 6 had no effect on responses to gratings below 20% contrast. The baseline contrast threshold of this cell (5.5% contrast) remained statistically unchanged during inactivation of P layer 6 (3.2% contrast). Inactivating P layer 6 did produce a premature saturation in the neuron's CRF which significantly reduced its peak response amplitude. Inactivating neural activity in LGN P layer 6 did not change the contrast-response threshold of any V1 neuron tested (Fig. 3B). The baseline average contrast threshold of this sample was 8.02% contrast. The average contrast threshold recorded during inactivation of P layer 6 (7.54% contrast) remained unchanged ($t = 0.88$, $P > 0.05$). All cells tested during P layer inactivation exhibited a reduced response to 56% contrast (Fig. 3C). Inactivating P layer 6 significantly reduced ($t = 4.34$, $P < 0.01$) the average peak response amplitude at high stimulus contrast by 47.4 (± 14.4)% in the ten cells tested. The spatial and temporal frequencies of the grating used during P layer block were optimized for each recorded cortical neuron, rather than being optimal for LGN M neurons. The

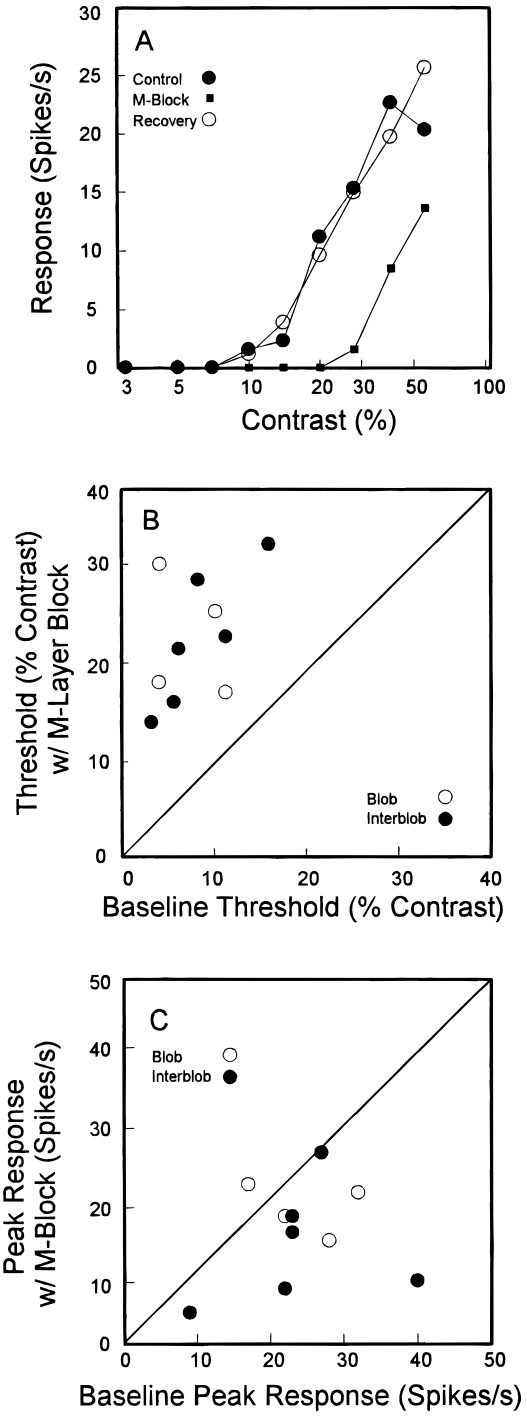

Fig. 2. Effects of inactivating LGN M layer 1 on the CRF of V1 cells. (A) CRFs recorded from a layer V simple cell under control (filled circles) and recovery (open circles) conditions were identical. Inactivating LGN M layer 1 (filled squares) elevated significantly this cell's contrast threshold and reduced its peak response to 56% contrast. All ten neurons tested during M layer inactivation displayed similar increases in contrast threshold (B) and eight neurons displayed significant decreases in their peak responses (C). Blob = cells located within or below a CO-blob. Interblob = cells located within or below an interblob.

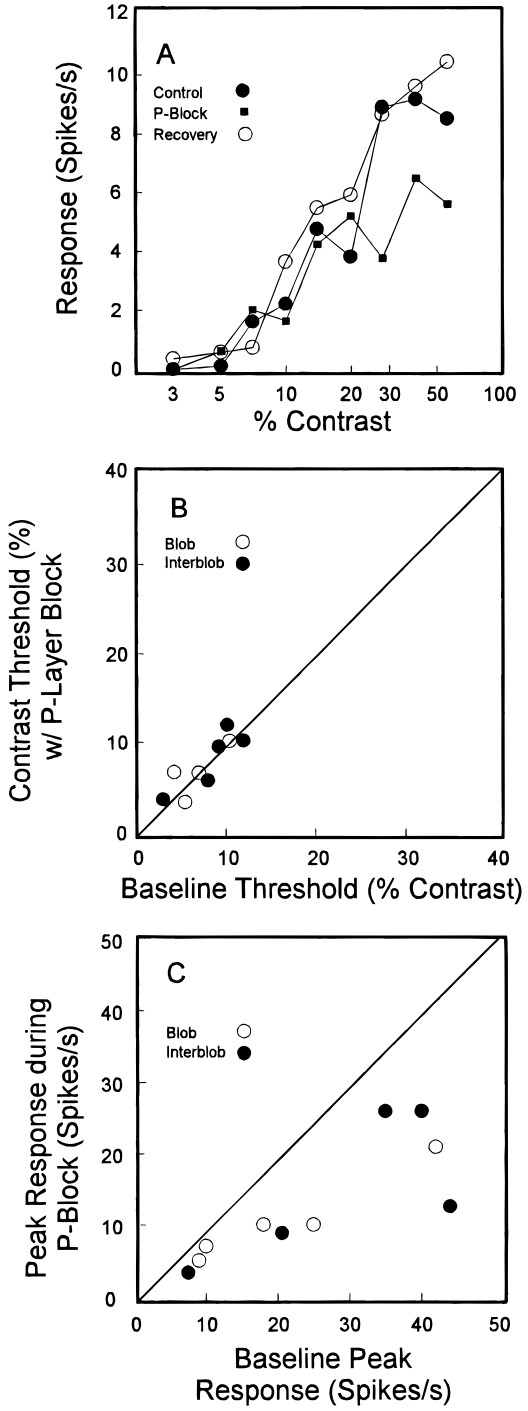

Fig. 3. Effects of inactivating LGN P layer 6 on the CRF of V1 cells. (A) CRFs recorded from a layer IVB simple cell show that inactivating LGN P layer 6 had no effect on this cell's contrast threshold. Inactivating P layer 6 reduced significantly its peak response to 56% contrast. None of the ten cells tested during P layer inactivation exhibited any significant change in contrast threshold (B), but all showed reduced peak responses to 56% contrast (C). Other conventions as in Fig. 2.

average spatial and temporal frequencies of the grating during P layer inactivation experiments were 0.37 (±0.12) cycles/deg and 2.1 (±0.74) Hertz. The eccentricities of this sample of neurons ranged from 5 to 10 deg (mean = 8.6 deg).

Anatomical reconstructions revealed that the recorded V1 cells were found in output layers or on the borders of output layers IIIA ($n = 1$), IVB ($n = 2$), IVB/IVCα ($n = 1$), IVCβ/V ($n = 3$), V ($n = 4$), V/VI ($n = 1$), and VI ($n = 7$). The location of one cell could not be determined. No cells recorded were centered within either P recipient layer IVCβ or M recipient layer IVCα, although four cells lay at the borders of these layers. Cells were found both in vertical columns containing cytochrome oxidase-rich blobs and cytochrome oxidase-poor interblobs, as indicated in Figs. 2 and 3.

Discussion

The present results demonstrate clear differences between the contributions of the LGN M and P layers to the contrast-dependent responses of V1 neurons (Fig. 4). In this sample of cells, input from the LGN M pathway accounted for V1 cell responses to low stimulus contrasts and contributed to responses at other contrasts measured. The LGN P pathway provided a substantial contribution to V1 cell responses only at contrasts above about 10%. These data demonstrate that some V1 cells (at least those within the output layers of V1) are capable of integrating information about stimulus contrast contained within the two parallel LGN inputs. Injecting GABA into the LGN modified the response of every V1 cell tested, regardless of which LGN layer was inactivated or where the V1 cell was located. No cell showed a complete loss of visually driven activity, thus none of the cells in our sample appeared to receive input from only one pathway. Previous LGN inactivation studies (Malpeli et al., 1981; Nealy & Maunsell, 1994) found that inactivating individual LGN layers completely silenced a small number of cells in all cortical layers. One possible explanation for the different results is that our data may be biased due to the low sample size. Another possibility is that cells receiving input solely from M or P LGN cells are located principally within cortical layer IVC.

In bush babies, as in other primates (Hendry & Casagrande, 1996), K cells lie between all of the main layers and below layer 1. Thus, K cells could contribute to the contrast sensitivity of V1 cells during inactivation of either the LGN M or P pathway. In bush babies the majority of K cells are found between the two P layers in K layers 4 and 5 (Fig. 1) This arrangement suggests that if K cells do contribute to the contrast sensitivity of V1 cells in bush babies, it should mainly be blocked during experiments blocking P layer 6 since contralaterally innervated K layer 5 is closest to 6 and far from M layer 1. However, preliminary data (unpublished) in which we directly blocked the K layers 4 and 5 with GABA failed to reveal any consistent changes in the contrast response of V1 cells, suggesting that this pathway may not contribute to the contrast response of V1 neurons that is measurable under these conditions.

Regardless of V1 cell location, inactivating the M or P layers produced consistent effects. These data suggest that either geniculate pathway is capable of influencing the responses of a variety of V1 neurons. The dominance of a particular pathway depends on the contrast of the stimulus. Thus, the present findings do not support the contention that V1 cells in different cortical layers process information about stimulus contrast due to exclusive input from a single LGN pathway (Livingstone & Hubel, 1984; DeBruyn et al., 1993). Admittedly our sample of cells was small and did not include cells within the centers of the input layers IVCα and IVCβ. However, even with a small sample of 20 cells one would expect to encounter at least one cell with exclusive input from the P or M pathway if this were a common feature. The present data are not inconsistent with our previous results showing

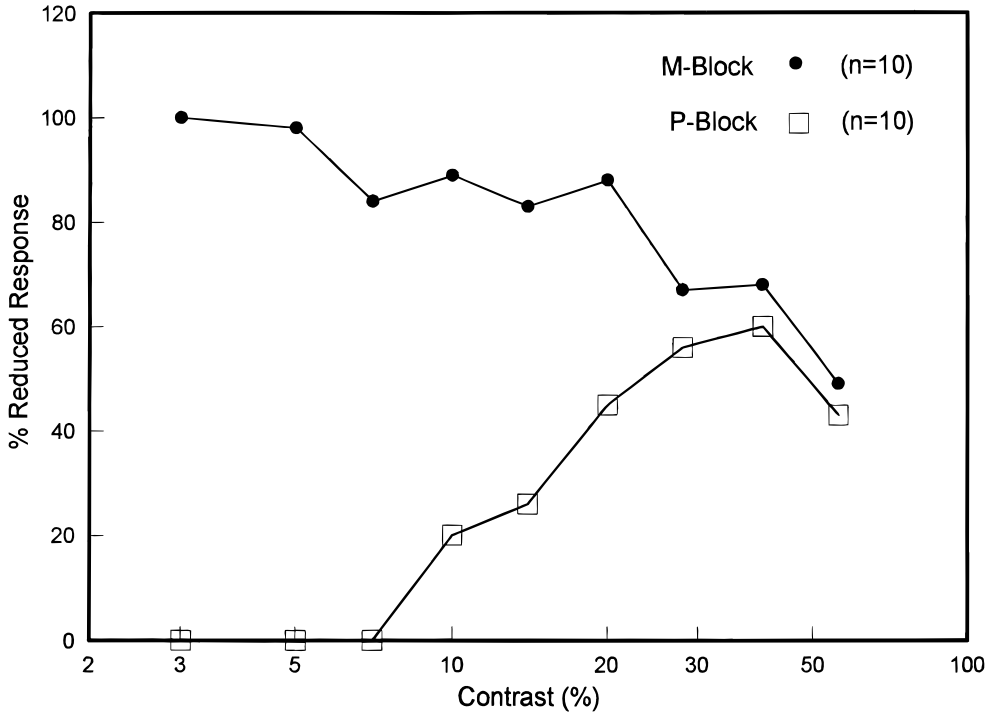

Fig. 4. The average percent reduction in response to each stimulus contrast following inactivation of either LGN M layer 1 (filled circles) or P layer 6 (open squares) pathway. For the cells recorded during M layer 1 inactivation, the amount of response reduction decreased when stimulus contrast was increased, especially above 20% contrast. Conversely, the magnitude of response reduction during inactivation of P layer 6 increased when stimulus contrast was increased, especially above 10% contrast. The contributions of each pathway to the contrast-dependent response of V1 cells are clearly distinguishable.

that cells belonging to different cortical layers exhibit differences in contrast sensitivity (DeBruyn et al., 1993). As can be seen in the summary data in Figs. 2 and 3, individual cells showed differences in overall responses to M or P blockade, suggesting that the proportionate contribution of M or P input to each cell can vary. Finally, it is important to bear in mind that we tested only one stimulus parameter while holding constant the spatial and temporal frequencies and orientation of our stimuli. Clearly, a complete picture of the contribution of M and P pathways would require systematic variation of all of these parameters which would be technically very challenging.

Although our results do not suggest that parallel LGN signals are processed separately by V1 neurons (Livingstone & Hubel, 1984, 1988), they do not diminish the importance of visual processing by parallel pathways. Signals arriving from an individual LGN layer strongly influence V1 cells. This effect is consistent with known physiology and connectivity of V1 cells (Casagrande & Kaas, 1994 for review), as well as with recent *in vivo* and *in vitro* data in macaque monkeys showing that V1 cells can be driven by M and P inputs (Nealy & Maunsell, 1994; Sawatari & Callaway, 1996), and behavioral observations about the relative contributions of each input (Langston et al., 1986). Many individual V1 cells, however, also appear capable of transmitting either M or P signals independently (or in combination), depending on the contrast of a stimulus. We therefore view cortical processing of parallel thalamic inputs as a dynamic process, with the proportionate role of each thalamic input adjusted continuously in relation to changes in stimulus features and conditions.

Acknowledgments

We thank J. Malpeli for much useful advice about LGN layer blocking; J. Dinger, J. Kabara, R. Snider, and B. Roig for assistance collecting and analyzing the data, and J. Mavity-Hudson, A. Wiencken, and C. Rehm for help processing the tissue. J. Kaas, J. Schall, T. Norton, K. Johnson, J. McKanna, R. Guillery, and J. Maunsell provided useful comments on the manuscript. This research was supported by NEI Grants F32 06410 (to J.D.A.), EY01778 (to V.A.C.), EY03778 (to A.B.B.), and core grants EY08126 and HD15052 (to Vanderbilt University).

References

Allison, J.D., Casagrande, V.A. & Bonds, A.B. (1995). The influence of input from the lower cortical layers on the orientation tuning of upper layer V1 cells in a primate. *Visual Neuroscience* **12**, 309–320.

Allison, J.D., Casagrande, V.A., DeBruyn, E.J. & Bonds, A.B. (1993). Contrast adaptation in striate cortical neurons of the nocturnal primate bush baby (*Galago crassicaudatus*). *Visual Neuroscience* **10**, 1129–1139.

Bonds, A.B. (1991). Temporal dynamics of contrast gain in single cells of the cat striate cortex. *Visual Neuroscience* **6**, 239–255.

Casagrande, V.A. (1994). A third parallel visual pathway to primate area V1. *Trends in Neurosciences* **17**, 305–310.

Casagrande, V.A. & Norton, T.T. (1991). The lateral geniculate nucleus: A review of its physiology and function. In *The Neural Basis of Visual Function*, ed. Leventhal, A.G., *Vol. 4, Vision and Visual Disfunction*, series ed. Cronley-Dillion, J.R., pp. 41–84. London, England: MacMillan Press.

Casagrande, V.A. & Kaas, J.H. (1994). The afferent, intrinsic, and efferent connections of primary visual cortex. In *Cerebral Cortex, Vol. 10, Primary Visual Cortex of Primates*, ed. Peters, A. & Rockland, K., pp. 201–259. New York: Plenum Press.

DeBruyn, E.J., Casagrande, V.A., Beck, P.D. & Bonds, A.B. (1993). Visual resolution and sensitivity of single cells in the primary visual cortex (V1) of a nocturnal primate (bush baby): Correlations with cortical layers and cytochrome oxidase patterns. *Journal of Neurophysiology* **69**, 3–18.

Derrington, A.M., Krauskopf, J. & Lennie, P. (1984). Chromatic mechanisms in lateral geniculate nucleus of macaque. *Journal of Physiology* **357**, 241–265.

Derrington, A.M. & Lennie, P. (1984). Spatial and temporal contrast sensitivities of neurones in lateral geniculate nucleus of macaque. *Journal of Physiology* **357**, 219–240.

Florence, S.L. & Casagrande, V.A. (1987). Organization of individual afferent axons in layer IV of striate cortex in a primate. *Journal of Neuroscience* **7**, 3850–3868.

Hendry, S.H. & Casagrande, V.A. (1996). A common pattern for a third visual channel in the primate LGN. *Society for Neuroscience Abstracts* **22**, 1605.

Irvin, G.E., Norton, T.T., Sesma, M.A. & Casagrande, V.A. (1986). W-like response properties of interlaminar zone cells in the lateral geniculate nucleus of a primate (*Galago crassicaudatus*). *Brain Research* **362**, 254–270.

Kaplan, E. & Shapley, R.M. (1982). X and Y cells in the lateral geniculate nucleus of macaque monkeys. *Journal of Physiology* **330**, 125–143.

Lachica, E.A. & Casagrande, V.A. (1992). Direct W-like geniculate projections to the cytochrome oxidase (CO) blobs in primate visual cortex: Axon morphology. *Journal of Comparative Neurology* **319**, 141–158.

Langston, A., Casagrande, V.A. & Fox, R. (1986). Spatial resolution of the Galago. *Vision Research* **26**, 791–796.

Lennie, P., Krauskopf, J. & Sclar, G. (1990). Chromatic mechanisms in striate cortex of macaque. *Journal of Neuroscience* **10**, 649–669.

Leventhal, A.G., Thompson, K.G., Liu, D., Zhou, Y. & Ault, S.J. (1995). Concomitant sensitivity to orientation, direction, and color of cells in layers 2, 3, and 4 of monkey striate cortex. *Journal of Neuroscience* **15**, 1808–1818.

Livingstone, M.S. & Hubel, D.H. (1984). Anatomy and physiology of a color system in the primate visual cortex. *Journal of Neuroscience* **4**, 309–356

Livingstone, M. & Hubel, D. (1988). Segregation of form, color, movement, and depth: anatomy, physiology, and perception. *Science* **240**, 740–749.

Malpeli, J.G., Schiller, P.H. & Colby, C.L. (1981). Response properties of single cells in monkey striate cortex during reversible inactivation of individual lateral geniculate laminae. *Journal of Neurophysiology* **46**, 1102–1119.

Merigan, W.H. & Maunsell, J.H. (1993). How parallel are the primate visual pathways? *Annual Review of Neuroscience* **16**, 369–402.

Nealy, T.A. & Maunsell, J.H. (1994). Magnocellular and parvocellular contributions to the responses of neurons in macaque striate cortex. *Journal of Neuroscience* **14**, 2069–2079.

Norton, T.T. & Casagrande, V.A. (1982). Laminar organization of receptive-field properties in lateral geniculate nucleus of bush baby (*Galago crassicaudatus*). *Journal of Neurophysiology.* **47**, 715–741.

Norton, T.T., Casagrande, V.A., Irvin, G.E., Sesma, M.A. & Petry, H.M. (1988). Contrast-sensitivity functions of W-, X-, and Y-like relay cells in the lateral geniculate nucleus of bush baby, *Galago crassicaudatus*. *Journal of Neurophysiology* **59**, 1639–1656.

Sawatari, A. & Callaway, E.M. (1996). Convergence of magno- and parvocellular pathways in layer 4B of macaque primary visual cortex. *Nature* **380**, 442–446.

Ts'o, D.Y. & Gilbert, C.D. (1988). The organization of chromatic and spatial interactions in the primate striate cortex. *Journal of Neuroscience* **8**, 1712–1727.