

THE NEURAL ARCHITECTURE OF BINOCULAR VISION

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The brain is remarkable in its ability to match images from the left and right eyes to produce a seamless single image of the world. Not only are the slightly different monocular views of the world fused to produce a single image, but also, in many frontal-eyed mammalian species, this single image provides additional information about stereoscopic depth. What components of neural architecture are responsible for these precepts? In this review we briefly outline current views of the neural hardware that have been proposed to be responsible for binocular single vision. We will focus primarily on the visual system of primates but mention other mammalian species where relevant. Within this framework we consider three issues which remain controversial. First, do all classes of retinal ganglion cells contribute signals that are important to stereoscopic depth perception or only one class? Second, does the dorsal lateral geniculate nucleus (LGN) play a special role in binocular vision? Finally, are ocular dominance columns of primary visual cortex important to binocularity?

OVERVIEW OF BINOCULAR CONNECTIONS

The visual pathways from the retina to the primary visual cortex (also called V1 or striate cortex) have been well studied in a variety of mammals.¹ As shown in Fig. 1, for a generic primate, the lateral geniculate nucleus (LGN) receives axons from ganglion cells in the nasal half of the contralateral retina and from the temporal half of the ipsilateral retina. All placental mammals segregate ocular input within the LGN although the number of layers varies from two in rodents to as many as eight in some humans. That there are more than two layers in the LGN in most species reflects the segregation within this structure not only of inputs from the left and

right eyes, but also of inputs from functionally distinct classes of retinal ganglion cells that exist in each retina. Thus, in primates there are two magnocellular (M) layers, one for each eye; two or more pairs of parvocellular (P) layers; and variably located sets of very small cells, which we will refer to here as the koniocellular (K) layers.²

The LGN also receives visual input from a variety of other sources including areas of the cortex, midbrain and brainstem.² Of these sources four provide retinotopically organised binocular input. The richest source of this input comes from V1. The binocular cells in layer VI of V1 send a heavy excitatory (aspartate) projection back to all layers of the LGN (see Fig. 1). In the cat LGN, synapses from this pathway, which ends on both excitatory relay cells and inhibitory (GABAergic) interneurons,³ outnumber those from the retina 10 to 1;⁴ this ratio has not been examined in primates. Other visually driven sources of excitatory axons to the primate LGN come from the superior colliculus and parabigeminal nucleus which terminate almost exclusively on K cells,^{5,6} and the pretectum, which targets all geniculate layers;⁷ all of the latter structures contain binocular cells.

The LGN projects ipsilaterally to the primary visual cortex (V1). Like the LGN, V1 in each hemisphere receives information about the contralateral visual field through both eyes and from three distinct classes of cells. For many primate species (but not all; see below), the same double segregation of eye and functional type that occurs in the LGN also occurs in V1, but whereas the LGN achieves both types of segregation by creating additional laminae, V1 utilises laminar segregation for functional class, and a *tangential* segregation for eye specificity.² (An interesting exception is the tree shrew visual cortex, which has a laminar segregation of both functional class and eye specificity.⁸⁻¹¹) Thus, in species that segregate ocular inputs, the primary input layer, layer 4, is not only split into M and P

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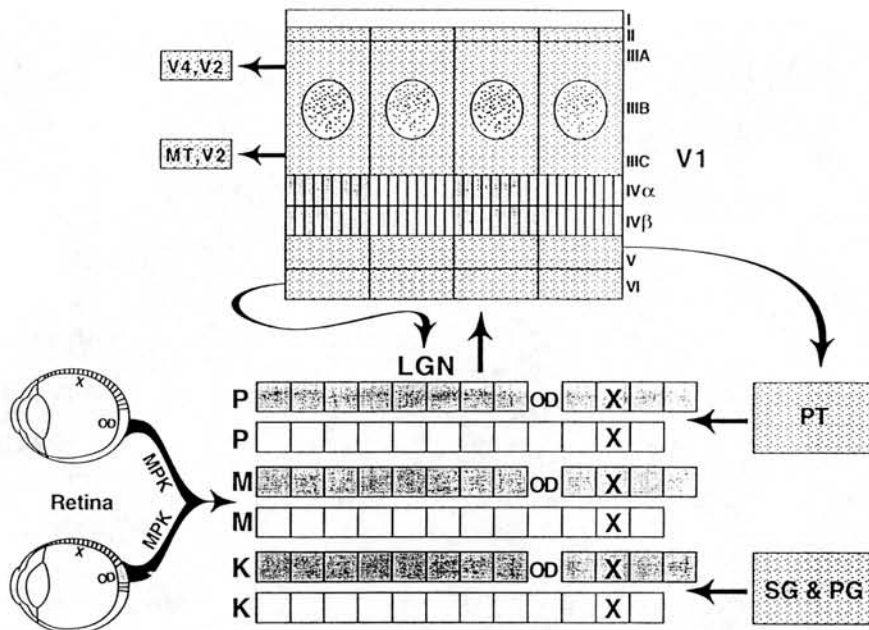


Fig. 1. A schematic diagram of the connections of the left and right eye with the right hemisphere in a primate. ON and OFF centre magnocellular (M), parvocellular (P) and koniocellular (K) retinal ganglion cells from the left nasal retina and right temporal retina send axons to different layers of the right lateral geniculate nucleus (LGN). The axons from each eye terminate retinotopically such that points on the retina (X) that represent the same point in visual space are in register in each layer. A gap appears in all LGN layers where the retina is blind at the optic disc (OD). The LGN also receive binocular visual input from other direct or indirect retinotopically organised sources including the pretectum (PT), the superior colliculus (SC), parabigeminal nucleus (PG) and primary visual cortex (V1). Projections to V1 from the LGN M and P layers end in layers IV α and IV β , respectively, and are also segregated into ocular dominance columns in some species. In bush babies and macaque monkeys, the cytochrome oxidase (CO) rich blobs (circles in layer IIIB) contain a higher percentage of monocular cells. The remaining layers contain a high percentage of binocular cells. Cells in layer IIIA project to visual areas 2 and 4 and cells in layer IIIC project to V2 and the middle temporal visual area (MT). Cells in layer VI project to the LGN and cells in layer V project to the SC and PT. Areas driven monocularly by the right eye are shown in white; areas driven monocularly by the left eye are shown in grey; areas driven binocularly are stippled. Roman numerals refer to cortical layers. See text and also Casagrande and Kaas¹ for further discussion.

sublayers but further divided into alternating columns that receive input from each eye, the ocular dominance (OD) columns (see also Fig. 1). Input from the K layers terminates within cortical layer III where it ends within zones which stain darkly for cytochrome oxidase (CO blobs), and within layer I. At present, it is unclear whether the K pathway also maintains ocular segregation in cortex, although this is likely since CO blobs are found in the centres of the OD columns and individual K LGN axons terminate only within a single CO blob^{12,13} (The higher percentage of monocular cells recorded within the centres of macaque and bush baby CO blobs^{14,15} may, in part, reflect this monocular LGN input.)

Although eye-specific inputs can be segregated within layer IV, they are combined at the next stage of cortical processing, and many binocular cells are found in V1 in all layers outside of layer IV.¹⁶ That is, while neurons in LGN and layer IV of V1 generally respond only to stimulation of one eye, neurons in supra- and infra-granular layers of V1 can be driven through either eye. Some of these binocular cells are sensitive to small horizontal disparities in retinal

position between the two eyes;¹⁷ such disparities occur for stimuli in front of or behind the fixation plane, making disparity-sensitive cells candidates for encoding depth information. Once ocular input is combined in V1 it remains together within all extrastriate visual areas regardless of specialisation. Thus, it seems reasonable to propose that interactions important to binocular vision and stereopsis are set up in cortical area V1.

CONTRIBUTIONS OF PARALLEL PATHWAYS

How visual cortical cells put together the signals arriving from each eye is not a trivial problem. Visual cortical cells face the problem of combining inputs not just from each eye but also from different functional classes of ganglion cells. It is well recognised that retinal ganglion cells with different physiological properties represent common points in visual space.¹⁸ Presumably such an arrangement of functional parallelism allows the visual system to process separate aspects of the visual scene more efficiently since information from a single retinal

Binocular V1 Cells

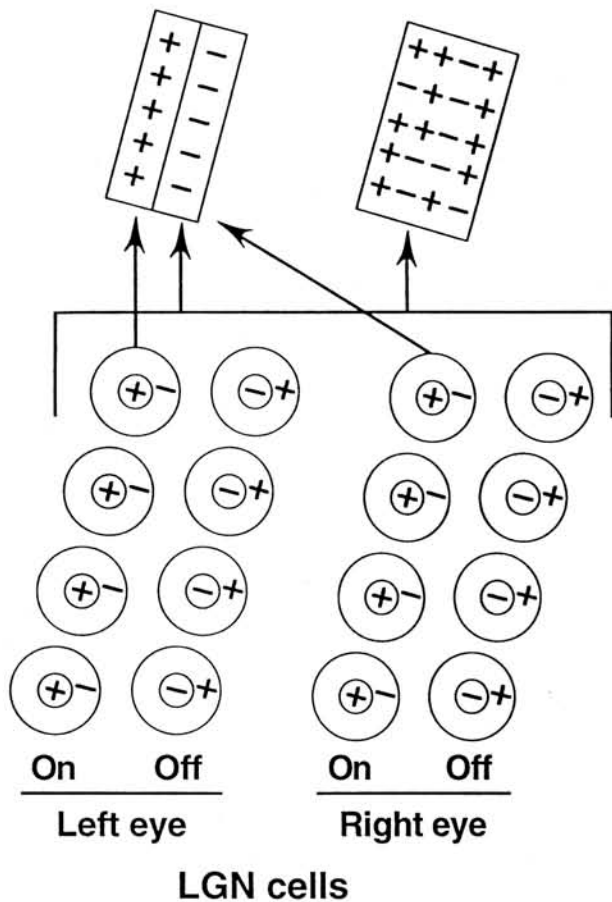


Fig. 2. Convergence of LGN inputs in V1 simple and complex cells. Shown are four concentric ON centre OFF surround LGN cells and four OFF centre ON surround LGN cells representing each eye. Cortical binocular cells are orientation selective and exhibit the same receptive field properties (with the exception of ocular disparity) for each eye. This requires that monocular LGN cells with different centre/surround and other properties projecting to the same cell match precisely to produce the appropriate cortical receptive field structure. This figure shows two types of direct convergence from LGN cells to cortical simple and complex cells that were identified by Tanaka.²⁵ Some simple and complex cells received projections from both ON and OFF LGN cells from both eyes. Other simple cortical cells received exclusively ON (as shown here) or OFF centre inputs. Convergence of the properties of primate LGN inputs to V1 cells has not been studied in detail. See text for discussion.

locus travels and is processed in parallel. However, such parallelism also presents problems for binding monocular signals in cortex. For example, it is well known that, except for disparity, binocular cells in V1 exhibit the same receptive field properties for each eye.¹⁹ Yet, at the level of the LGN, separate cells signal increments and decrements in brightness (ON centre and OFF centre cells) and populations of cells (M, P and K) with distinct chromatic, spatial and temporal properties are segregated, as mentioned

above, to separate LGN layers.² Is this convergence accomplished in one step or several serial steps?

In macaque monkeys there is physiological evidence that a number of cells within the M target layer, IV α , of visual cortex are monocular simple cells with ON and OFF responses,²⁰ suggesting that ON and OFF M LGN cells from one eye converge before ocular inputs are combined in more superficial cortical layers. Yet, the same may not be the case for the P LGN cells since many of their target cells in IV have been reported to have receptive fields that are identical to P cells in the LGN¹⁴ (see, however, Leventhal *et al.*²¹). Anatomically, layer IV sends projections to cells within layer III which are known to mix ON and OFF and ocular inputs. Thus, it is unclear in this primate species whether ON/OFF convergence and binocular convergence occur as a single event or serially. With respect to the latter it is noteworthy that recent psychophysical studies of humans indicate that, for some tasks requiring depth judgements, increments and decrements of brightness, presumably carried by the ON and OFF channels to cortex, are treated independently by each eye within the early stages (e.g. V1) of processing.²² This finding would indicate that at least some aspects of stereopsis involve cells that combine ocular pathways before ON or OFF pathways. It would be interesting to determine whether such cells can be found in the macaque monkey V1 and if so where they are located anatomically.

In tree shrews, close relatives of primates, anatomical and physiological studies^{9,11} have provided clear support for serial convergence of inputs such that ipsi- and contralateral ocular inputs are combined before the ON/OFF centre pathways. However, it is not clear whether tree shrews have stereopsis. Moreover, other primates in which there is evidence of stereopsis, such as bush babies²³ and squirrel monkeys,²⁴ have cells within input layer IV that are frequently ON/OFF and monocular (bush babies) or ON/OFF and binocular (squirrel monkeys). Moreover in cats, which have also been shown to exhibit stereopsis, direct physiological examination reveals that ON and OFF and left and right eye pathways can all converge at the first synapse in cortex²⁵ (see Fig. 2). Taken together, these findings suggest that different species may have different solutions to the problem of combining binocular inputs, although direct evidence is lacking.

Concerning parallel channels, a related issue is whether all channels participate equally in binocular single vision, particularly stereopsis. Livingstone and Hubel²⁶ have suggested that stereopsis is handled primarily by the M retinal channel. Evidence to support this comes from data which show that cells in IIC, which receive heavy input from the M pathway via IV α in V1, have a large number of binocular cells

that are selective for disparity. These cells project to bands of cells in V2 and to area MT that also contain a high proportion of disparity-selective cells.^{26,27} However, lesions of M LGN layers and area MT do not affect stereo vision in macaque monkeys.^{28,29} While the M pathway is thus not necessary for stereopsis, it probably does contribute to it, given the presence of good stereopsis under scotopic conditions³⁰ and the fact that M, but not P, retinal ganglion cells near the fovea receive input from rods.³¹ Psychophysical studies show that stereopsis is maintained at isoluminance,³² suggesting the involvement of the colour-opponent P pathway in stereopsis. Lesions of the LGN P layers do disrupt stereopsis but only when the image is made up of high spatial frequencies to which M cells respond poorly.²⁸ It seems likely that both M or P channels can contribute to stereopsis depending on the contrast and spatial frequency of the stimulus. This is not surprising, given anatomical evidence that the P and M channels (and possibly also the K channel) are combined early in V1.³³

BINOCULARITY AND THE LGN

The spatial maps of each of the LGN layers are in precise register such that a single point in visual space (e.g. X in Fig. 1) is represented as a line (the line of projection³⁴) running roughly perpendicular to the long axis of the layers. Given the segregation of ocular inputs in the LGN, the tight retinotopic register of each layer with its neighbours suggests the presence of binocular interactions in the LGN. In fact, the alignment of common points (e.g. X in Fig. 1) from layer to layer is so precise that cellular gaps appear at the laminar representation of the visually blind optic disc (OD in Fig. 1) in all contralaterally innervated LGN layers. This allows contralateral layers to remain exactly aligned with their ipsilaterally innervated LGN counterparts, such that projections across laminar borders would remain in the same location in the visual field. Dendrites of LGN cells have been shown to cross laminar borders in just such a fashion,^{35,36} as have the axons of the feedback pathway from layer VI of visual cortex.³⁷⁻³⁹ These pathways probably mediate the inhibition that is generated in many LGN neurons by stimulation of the non-dominant eye.⁴⁰⁻⁴⁴ Both short-latency inhibition that is not selective for orientation, and longer-latency, orientation-selective inhibition (but see Sengpiel *et al.*^{44a}) is seen: the former probably represents interactions at the LGN while the latter is probably the result of the corticogeniculate pathway.

Although the function of binocular interactions in the LGN is unclear, several investigators have suggested that they aid in the process of binocular fusion for stereopsis.^{41-43,45} Mellwain⁴⁵ nicely summarises these views in a recent theoretical paper.

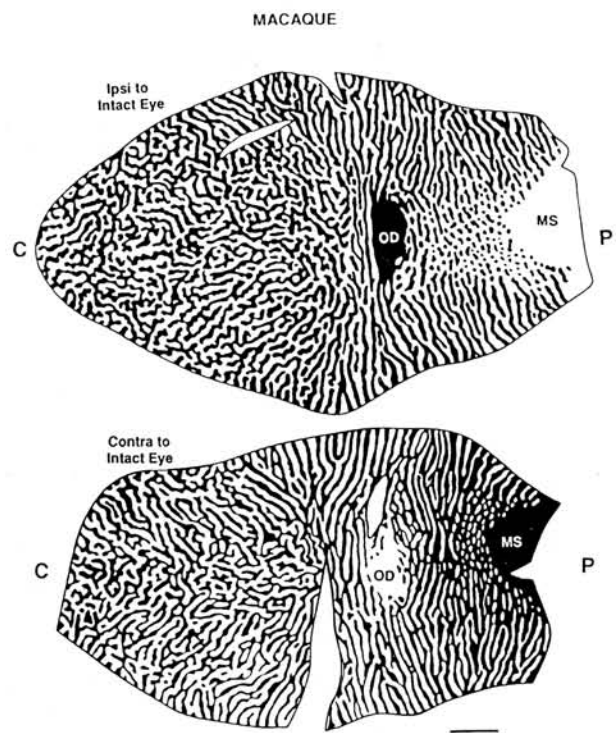


Fig. 3. Distributions of ocular dominance columns in a macaque monkey reconstructed from a montage of sections cut through layer IV of area V1 after flattening the cortical tissue. The columns are demonstrated in a cytochrome oxidase stain following blockade of activity from one eye. The dark bands present the intact eye. The patterns ipsilateral (above) and contralateral (below) to the intact eye are similar but not identical. The entire visual field is represented from central (C) to peripheral (P). The representation of the optic disc (OD) of the nasal retina is centred 17 degrees from the fovea. The unbanding segments to the right correspond to the monocular segment (MS) of the visual field. Scale bar represents 5 mm. From Florence and Kaas⁴⁸ with permission of the publishers.

Basically, the visual system is faced with a geometric problem. The problem is that two retinal loci that are optically in register at one visual fixation distance are not in register at other fixation distances. It has been proposed that visual cortical neurons that code disparity are important for stereopsis. Since these cells are known to respond to a very narrow range of disparities, either the visual cortex needs a lot of these cells tuned to each disparity at each locus or the disparity tuning of individual cells is dynamically adjusted. Originally Schmielau and Singer⁴¹ suggested that dynamic disparity tuning could be accomplished within the LGN via binocular feedback from V1 corticogeniculate axons.

Verela and Singer,⁴³ using dichoptic stimulation in which a horizontal square-wave drifting grating was used to activate cat LGN cells through the dominant eye and intermittent stimulation using another grating was used to stimulate the non-dominant eye, showed that suppression caused by stimulation of the non-dominant eye was strongest when the two

Ocular Dominance Columns in V1

Primates				Archontans
prosimians	new world simians	old world simians	apes/man	(tree shrew)
bush baby	squirrel monkey owl monkey † marmoset † cebus albifrons cebus apella saki monkey spider monkey	macaque talapoin	chimpanzee man	Carnivores cat ferret mink
				Rodents squirrel rat mouse
present				
absent				
† weakly present				
‡ only found after deprivation or in neonates				

Fig. 4. Presence or absence of ocular dominance columns for a selection of primate and non-primate species. Names of species without ocular dominance columns are in grey rectangles. Ocular dominance columns have been found in all Old World monkeys and apes examined, but are found in only some New World monkeys. In some of these species (marked with a dagger or asterisk, respectively) columns are only weakly present, or their formation can be induced (or stabilised) with early deprivation. Note that ocular dominance columns are also present in certain non-primates such as carnivores.

gratings differed most in orientation. Such pairs of grating stimuli are known to cause binocular rivalry. On the basis of these experiments Vela and Singer⁴³ hypothesised that the afferents from the visual cortex play an important role in modifying the signal-to-noise ratio based on congruency between ongoing cortical activation and retinal activation. In the light of this hypothesis it is interesting that local iontophoresis of glutamate within layer VI of striate cortex has been shown to produce facilitation of LGN cell responses if the receptive field of the LGN cell is aligned within 1–3 degrees of the retinotopic location of the cortical stimulation. If the field is misaligned with the cortical stimulation site, inhibitory effects occur.⁴⁶ If the corticogeniculate pathway is involved in dynamic disparity tuning of cortical cells, then one would predict that the receptive fields of LGN and binocular cortical neurons would not be fixed on the retina during active viewing conditions with varying fixation requirements. Although some evidence of such dynamic changes in the receptive field properties of cortical neurons exists, there are no data showing such changes in LGN receptive fields. Specifically, one would predict that changes in vergence should produce small displacements in the LGN receptive fields relative to the retinal receptive field.⁴³ No one has yet looked for such changes.

One anatomical prediction of such a hypothesis is that corticogeniculate axons be restricted to, or be more heavily represented within, the binocular segment of the nucleus. This does not appear to be the case, although systematic examination of corticogeniculate innervation of the two segments of the

LGN has not been done. Moreover, it seems likely that this feedback pathway plays several roles. In the light of our earlier discussions of the relative roles of different functional channels (e.g. ON, OFF or M, P and K) it is noteworthy that corticogeniculate axons have been found within all LGN layers as well as the interlaminar zones.

BINOCULARITY IN CORTEX: WHY DO WE HAVE OCULAR DOMINANCE COLUMNS?

The presence of ocular dominance bands or columns in the visual cortex of some primates (see Fig. 3) has been assumed, logically, to play some important role in binocular interactions, perhaps stereopsis. These columns have been featured as a centrepiece of functional organisation of V1 in most neurobiology textbooks. Yet their functional role has never been established. As shown in Fig. 4, ocular dominance columns occur in all Old World prosimians and simians that have been examined, as well as in chimpanzees and man; other apes have not been examined.^{47,48} They appear in some but not all New World primates. And in some cases, such as in marmosets, ocular dominance columns are only evident either in early development⁴⁹ or if vision in one eye has been compromised early in development.⁵⁰

Ocular dominance columns have also been identified in non-primates including several carnivores (e.g. cat, mink, ferret) and perhaps also in sheep,⁵¹ but are not present in other species with good vision such as squirrels⁵² and tree shrews.⁵³ Attempts to correlate the presence or absence of ocular dominance columns with other features of an animal's visual life style have so far failed.^{47,48} The presence of ocular dominance columns does not appear to be correlated with diurnal or nocturnal vision, presence of trichromatic colour vision, or body size. However, New World primates that have ocular dominance columns do tend to be larger with more widely separated eyes, suggesting a link with stereopsis. A direct test of the hypothesis that ocular dominance columns are important for stereoscopic vision yielded negative results. Squirrel monkeys lack anatomically defined ocular dominance columns⁵⁴ and have few monocularly driven cells in V1.²⁶ Yet, a recent study using evoked potentials to measure the responses of awake squirrel monkeys to the presence of random-dot stereograms suggests that these primates do have stereoscopic vision.²⁴ This casts doubt on the assumption that ocular dominance columns play an essential role in stereopsis.

It has been suggested that ocular dominance columns are the developmental outcome of competitive interactions between cells with different firing patterns, and a tendency for cells with common firing patterns to connect with one another.⁵⁵ The

production of ocular dominance stripes from two eyes forced to share space in the optic tectum of three-eyed frogs⁵⁶ certainly supports such a possibility. In species with ocular dominance columns, activity-dependent competitive interactions in the cortex between terminals driven by the two eyes have been well studied.⁵⁷ After early monocular lid-suture, ocular dominance columns of the deprived eye become smaller, and the loss of terminal space in cortex is reflected by the shrinkage of deprived eye-driven LGN neurons.^{58,59} In monocularly deprived squirrel monkeys, a similar shrinkage of LGN neurons occurs,⁶⁰ suggesting that competition within cortex of geniculate fibres representing the two eyes does occur in species that do not have ocular dominance columns. Apparently, competition between two sets of innervating axons is not, in itself, a sufficient condition for the development of columns.

Moreover, if ocular dominance columns are only an epiphenomenon of early competitive interactions between monocular LGN axons in layer IV of V1, then it is curious that the ocular bias in organisation is replayed within V1 cells above and below this layer. This can easily be appreciated from single unit recording or functional maps of the uptake of 2-deoxyglucose (2DG) following monocular stimulation or from the rapid downregulation of immediate early gene expression following monocular compromise of vision.^{61,62} Evidence suggests that ocular dominance columns in macaque layer III are, in part, sharpened by inhibitory intracortical connections.⁶³ In addition, other functionally defined compartments of V1 such as CO blobs appear aligned with ocular dominance columns,⁶⁴ suggesting that these columns have functional significance. Taken together, these findings suggest that there may be different evolutionary solutions to the binocular fusion problem, although essential details on the binocular visual abilities of different species are still lacking.

CONCLUSIONS

At the beginning of this article we posed three questions related to the anatomy of binocular vision. First, we asked whether all classes of retinal ganglion cells contribute signals that are important to stereoscopic depth perception or only one class. Data support the conclusion that at least the P, M, ON and OFF channels set up at the retina contribute to the perception of depth. However, it is presently unclear how the cortex combines the monocular signals from each of these channels. It would be particularly interesting to know how deletion of individual channels at the retina or LGN would affect binocular receptive fields within V1. Second, we asked whether the dorsal lateral geniculate nucleus (LGN) plays any special role in binocular vision. Although some

findings and theories support such a view, the data are not sufficient at present to be certain whether this is the case. Selective deletions of the corticogeniculate pathway have not been tried although such an experiment might be possible in cats where it has been shown that axons from V1 to the LGN travel in a discrete bundle in the white matter. Also, careful analysis of the interactions between individual corticogeniculate axons with LGN layers, and LGN interneurons and relay cells in primates might prove useful in this regard. Finally, we questioned whether ocular dominance columns of primary visual cortex are important to binocularity and stereopsis. Evidence suggests that such segregation of ocular inputs in V1 is not essential to stereopsis. However, the behavioural data are limited. The idea that ocular segregation is an epiphenomenon of developmental Hebbian synaptic rules is not satisfying since it fails to explain why ocular segregation does not always occur; nor does it explain what allows binocular cells to develop. Studies examining the binocular visual abilities of different primates with and without ocular dominance columns could help to resolve this issue since it still may be the case that the presence and absence of ocular dominance columns are associated with specific aspects of stereopsis.

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Key words: Lateral geniculate nucleus, Ocular dominance columns, Parallel pathways, Retina, Visual cortex.

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