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31 Parallel Visual Pathways: A Comparative Perspective

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In spite of obvious differences between species, our knowledge of the structure and function of the human visual system is based almost entirely on extrapolation from studies in a very limited set of animal models. Although a variety of species have been used, the principal models for visual system studies continue to be domestic cats and macaque monkeys. These species, and others used less commonly, differ from humans in a number of respects including millions of years of separate evolutionary history, brain size, and visual behavior. The continued use of just a few species as models for the human visual system is defensible if the organization of the visual system is basically the same among species. Many differences, however, have been documented. For example, in cats the lateral geniculate nucleus (LGN) of the visual thalamus appears to have much more widespread connections with extrastriate cortex than in macaque monkeys or other primates. Differences in visual cortical architecture also have been documented among different primate groups including New and Old World simians and between Old World simians and apes and humans (Preuss, 2000).

Why have so few species been accepted as adequate models? Other than issues of practicality, one reason is that many features of the visual system of mammals appear to be shared across a range of species. Hubel and Wiesel, beginning with their seminal work more than 40 years ago, showed that the basic receptive field organization of cells in the LGN and the primary visual cortex (also called *striate cortex*, *area 17*, or *V1*) of cats and macaque monkeys is quite similar (see Hubel and Wiesel, 1998, for review). They demonstrated that LGN cells respond well to spots of light presented to one eye and, like their retinal ganglion cell inputs, demonstrate center-surround receptive fields of two types: ON or OFF center, with surrounds of opposing sign (Wiesel and Hubel, 1966). In V1 of cats and macaque monkeys, they demonstrated that cells tend to be binocular, are selective for the orientation of a bar of light, and exhibit a systematic organization of orientation preference across cortex. These same features have now been demonstrated in a wide range of mammals, suggesting that they represent general features of the mammalian visual system (LeVay and Nelson, 1991). Nevertheless, as we learn more details about the visual system, more species differences are being uncovered. The important issue is how we interpret both similarities and differences. In this chapter, we argue that species differences as well as similarities can provide important clues, not only about the organization of the human visual system, but also about how the visual system works. A comparative approach is particularly important in translating findings to humans since the possibilities of direct investigations in humans are so limited.

In this chapter, we focus primarily on primate relatives of humans. The chapter is further limited to an analysis of parallel pathways at the early levels of visual processing from the retina through the LGN to V1. The main purpose of the chapter is not to rehash current views on parallel visual pathways in primates but to point out key issues that remain to be resolved, as highlighted by the comparative perspective. Given that the cat visual system has been studied in the most detail, especially at these early levels of visual processing, comparisons with this species are included. Where relevant, other nonprimate species are also mentioned. Some issues have been omitted, and those concern the segregation of ocular inputs and the segregation of pathways concerned with ON-center and OFF-center responses.

This chapter is divided into six sections in addition to this introduction. The first of these sections highlights key findings from different schools that led to today's views of parallel processing within the visual system. The second section outlines how newer findings have altered the two-pathway view of retinogeniculocortical organization in primates. The third section discusses how the different pathways exiting the LGN are organized within the primary visual cortex (V1). The fourth section considers the functional significance of the parallel LGN pathways within V1. The final two sections are devoted to summaries of key points and an outline of the main questions that remain to be resolved.

Separate visual pathways: a little history

Beginning with the prize-winning work of Gasser and Erlanger (1929) on the somatosensory system, it has been recognized that different qualities within a sensory modality can be transmitted via parallel pathways that are

morphologically distinct. In the somatosensory system, it was suggested that different sensations within a single cutaneous nerve (e.g., pain and temperature versus light touch and pressure) might be carried via axons of different caliber and conduction velocity. By analogy, George Bishop (1933) subsequently argued that the three groups of axons that he identified in the optic nerve of the rabbit, based on axon size and conduction latency, were evidence of parallel processing for visual qualities, although he later changed his mind and argued that axon fiber size reflected evolutionary history (Bishop, 1959). Today the idea that separate retinal ganglion cell classes transmit different sensory messages to the brain is accepted as a basic organizational principle. The issues that remain controversial concern the number of pathways that exist, homologies among pathways across species, the exact content of these pathways, and how these pathways relate to different perceptual attributes.

How we think about parallel processing is a product of several distinct approaches to the problem. Outlined below are four lines of investigation that have strongly impacted our views of parallel processing in the visual system. First, in the mid-1960s, Enroth-Cugell and Robson (1966) sparked a revolutionary shift in thinking about visual processing. Approaching the visual system from an engineering standpoint, they proposed that the visual system works as a series of spatial filters, namely, as spatial-frequency analyzers. The idea was that the visual system represents objects by tuning different cells to different ranges of spatial frequency. Enroth-Cugell and Robson (1966) used this linear systems approach to analyze the responses of ganglion cells in the cat retina. This, in turn, was followed by the application to visual psychophysics of Campbell and Robson (1968). This general approach then led to a flood of studies based on the idea that the visual system's response to any pattern could be predicted from its response to more basic components. In their original work, Enroth-Cugell and Robson (1966) subdivided concentrically organized ON- and OFF-center retinal ganglion cells in cats into two types on the basis of their spatial summation properties. Those that summed luminance changes linearly across their receptive fields were called *X cells*, and those that did not were called *Y cells*. In their report, the authors also described other features that distinguished Y from X ganglion cells including the higher conduction velocities, sensitivity to higher speeds and lower contrasts, lower spatial frequency cutoffs, larger average receptive field center sizes, and more transient responses of Y versus X cells (Enroth-Cugell and Robson, 1966). More than a decade of studies on X and Y cells followed these seminal findings. These studies showed that X and Y cells also could be distinguished based on different distributions in the retina, parallel central projections, and morphology (reviewed by Stone, 1983). From this constellation of traits, it was proposed that X cells were part of a channel to cortex that subserves high-resolution pattern vision, while Y cells were part of a channel that subserves crude form and motion vision (Stone, 1983). Also during this time, other cell types were discovered in the cat retina that were collectively called *W cells*. From the beginning, it was clear that unlike X and Y cells, which show relatively low within-group variability, the response properties of W cells varied widely, having perhaps little more in common than the attribute of low conduction velocity (see Kaplan, 1991; Stone, 1983, for review). Because many W cells were shown to have heavy projections to the midbrain, it was proposed that they subserved a more primitive form of vision referred to as *ambient vision*. X and Y cells, by contrast, provided *focal vision*, or vision that required cortex. *Ambient vision* was seen as an almost unconscious primitive ability to orient to objects and move through the environment found in all vertebrates, while *focal vision* was seen as the conscious vision used to identify objects typical of primates. The analysis of X, Y, and W cells in cats also led later to a similar set of investigations in primates, where both similarities and differences between cats and primates were uncovered (see Casagrande and Norton, 1991, for review). We will return to the issue of species differences in parallel processing in the next section.

The ambient/focal vision idea was actually linked to a second very influential set of investigations that also began in the 1960s. In 1969 Schneider published an important article in which he proposed that there was an anatomical separation between visual coding of the location of a stimulus and its identification. Based on behavioral/lesion work in hamsters, he argued that there were two pathways: a *where* pathway involving the superior colliculus and a *what* pathway involving the visual cortex (Schneider, 1969). The *where* versus *what* or ambient versus focal pathways were subsequently modified and described as independent pathways, one involving a pathway from colliculus to pulvinar to extrastriate cortex and the other involving the geniculostriate pathway (Casagrande et al., 1972; Diamond and Hall, 1969). The idea that these pathways were capable of independent operation was demonstrated clearly in tree shrews, in which complete removal of striate cortex and complete degeneration of the LGN does not impair discrimination of simple patterns or acuity (Ware et al., 1972, 1974). The dual pathway idea more recently has been suggested as an explanation for the *blind sight* exhibited by humans in the absence of visual cortex (Ungerleider and Mishkis, 1982).

In 1982 the idea of two visual systems *where* versus *what*, took a different form. Ungerleider and Mishkin (1982) proposed that visual object identification (what) depended on connections to the temporal cortex, while object location (where) required the parietal cortex. They also argued that both areas required primary visual cortex (V1) based on their own data from other lesion studies in monkeys, as well as from human clinical studies. The cortical version of the

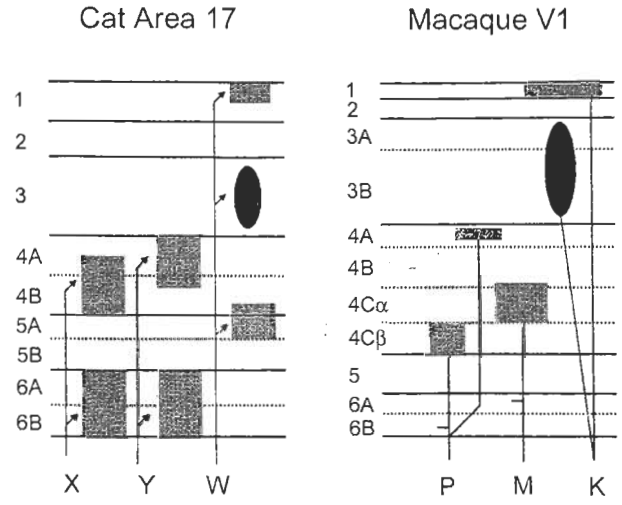

FIGURE 31.1 LGN projection patterns to visual cortex of cats and macaque monkeys. *Left*: projections from the LGN to V1 in the cat. X and Y LGN cells send axons to layers 4 and 6. W cells project CO blobs in layer 3 and to layers 1 and 5a. *Right*: projections from the LGN to V1 in macaque monkey. M and P cells project, respectively, to layers 4Cα and 4Cβ while K cells project to the CO blobs in layer 3 and to layer 1. In addition, P cells have been proposed to send axons to layer 4A. LGN projections to layer 4A are missing in apes, humans, and some other primates. Some M and a few P cells have collateral branches terminating sparsely in layer 6. See text for details. (*Left*: Data from Boyd and Matsubara, 1996; Humphrey et al., 1985; Kawano, 1998; *Right*: modified from Casagrande and Kaas, 1994, with permission of the publisher.)

what versus *where* hypothesis suggested that if the two visual systems originated subcortically, they must both pass through the LGN.

A third avenue of investigations involved efforts to define pathways based on anatomy. The advent of new technologies for tracing degenerating pathways in the 1950s and 1960s, and for anterograde and retrograde transport of tracers in the 1970s and 1980s, provided details of the connections of parallel pathways from retina to the LGN and from the LGN to the cortex in several species. For example, these studies clearly showed that different retinal ganglion cell classes projected to separate cell classes in the cat LGN and to separate layers of the LGN in all primate species examined (Casagrande and Norton, 1991). In addition, studies showed that the parallel arrangement of connections from the retina continued to V1, where X, Y, and W LGN cells in cats, and parvocellular (P) and magnocellular (M) and subsequently koniocellular (K) LGN cells in primates, were shown to project in parallel to separate layers of V1 (Fig. 31.1; see Casagrande and Kaas, 1994; Sherman and Guillery, 2001, for review).

Finally, in the 1980s, a set of studies was published by Livingstone and Hubel (1988) outlining their hypothesis that different attributes such as form, color, and motion were segregated within the layers and cytochrome oxidase (CO) blob compartments of V1 (Fig. 31.2). They linked various ideas described above together in a very satisfying model. According to this model, the P retinogeniculocortical pathway (form and color) projects ultimately to the what pathway ending in the temporal lobe and the M retinogeniculocortical pathway (motion) to the where pathway in the parietal lobe. Evidence to support the links between the P pathway and form/color and the M pathway and motion came primarily from physiology and connectional anatomy. Physiological studies had shown that P LGN cells exhibit chromatic opponency and have high spatial resolution, and that M cells are not selective for wavelength but exhibit high temporal resolution (reviewed in DeYoe and Van Essen, 1988; Livingstone and Hubel, 1988). Livingstone and Hubel and others provided evidence that linked the P pathway to the CO blob and interblob compartments in cortical layer 3 with appropriate output pathways to the what hierarchy of extrastriate visual areas, as well as evidence that the M pathway connected to the where hierarchy of visual areas via connections within V1 layer 4B (Fig. 31.2). The K pathway was ignored, in part, because it did not fit well with the model and had not been studied in as much detail (Casagrande, 1994; Hendry and Reid, 2000). Nevertheless, it was already known at that time that the K LGN pathway terminated in patches that appeared to coincide with the CO blobs in V1 in macaque monkeys (Livingstone and Hubel, 1982).

The degree of synthesis provided by Livingstone and Hubel's (1988) view of the visual system had a powerful impact on current thinking about the organization of the human visual system. Because of the simplicity of the model, things that did not fit were set aside as ever more streamlined diagrams of the original appeared in textbooks. Recently, however, as more data have been gathered in a variety of primates and in other species, findings have been presented that raise questions about the model. Examples are provided in the next section.

How many parallel retinal pathways project to and through the primate LGN?

In the parallel processing model of Livingstone and Hubel (1988), P and M pathways are the only pathways considered. Since their model was first published, a number of studies have appeared that have added controversy and complexity to the original proposal. Instead of just 2 major retinal pathways (P and M) to the LGN, as many as 10 morphologically distinct classes of ganglion cells have now been identified that project to the macaque monkey LGN, excluding ON-center and OFF-center cells of the same class (see Dacey, 1999, 2000; Dacey et al., 2001; Rodieck and Watanabe, 1993). Of these, at least five types have also been physiologically classified.

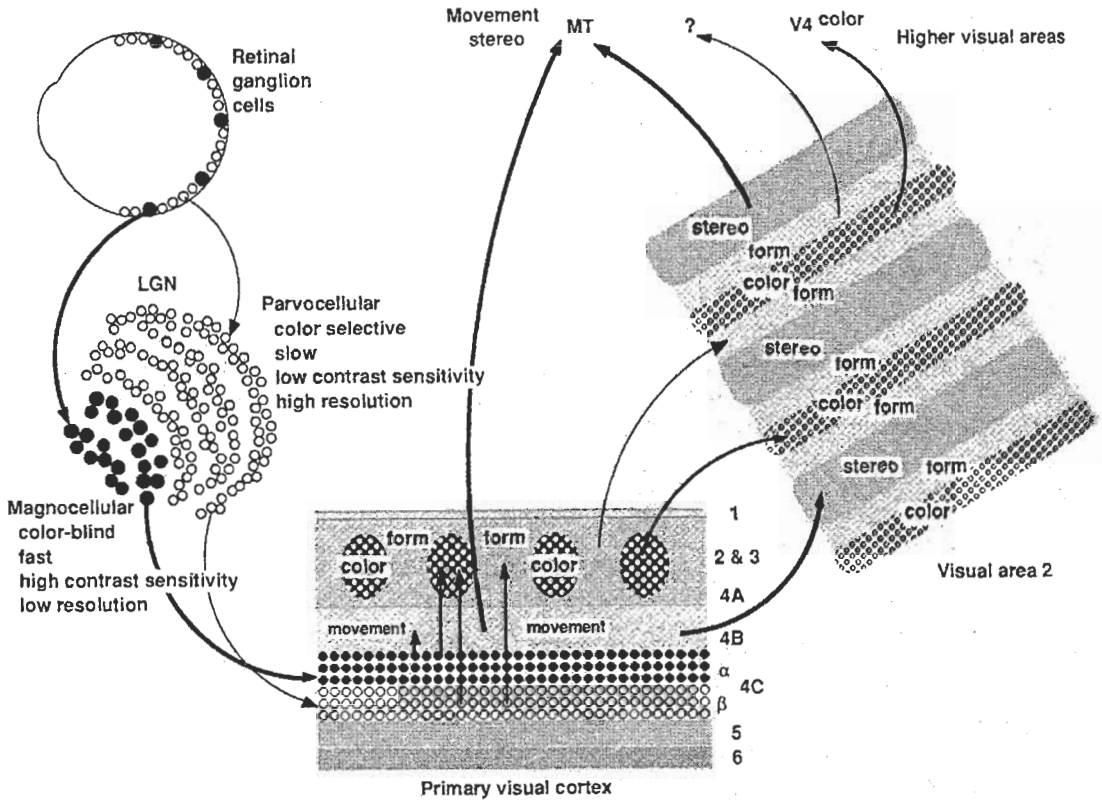

Figure 31.2 Diagram of the functional segregation of the primate visual system. *MT*, middle temporal area; *V4*, visual area 4; *LGN*, lateral geniculate nucleus. (From Livingstone and Hubel, 1988, with permission of the publisher.)

Within the LGN of all primates studied, it is now recognized that P and M LGN cells can be distinguished from K LGN cells based on their neurochemical signatures (Casagrande, 1994; Hendry and Reid, 2000). P and M relay cells contain the calcium binding protein parvalbumin, and K cells contain the calcium binding protein calbindin-D28k. K layers distinguished by the immunomarker calbindin are located primarily (although not exclusively) below all P and M layers. For ease of identification, these small cell layers are numbered from K1 to Kn beginning at the optic tract (Ding and Casagrande, 1997). It is now also established in several simian primates that cells in K3 project principally, although not exclusively, to the CO blobs in cortical layer 3, while those in K1/K2 project mainly to cortical layer 1 (Casagrande et al., 1997; Ding and Casagrande, 1997). It is not entirely clear, however, which LGN cell classes receive input from the 10 ganglion cell classes currently identified as projecting to the LGN from the retina in macaque monkeys. In a number of primate species, midget ganglion cells project to P LGN layers and parasol ganglion cells project to M LGN layers (Dacey, 1999; Rodieck and Watanabe, 1993; Yamada et al., 1998), but the question remains, which LGN cells receive input from the other ganglion cells that project to the LGN?

There is evidence to suggest that the small bistratified ganglion cells, identified as excited by S cone inputs (blue ON), project to the K3 LGN layer in marmosets (Martin et al., 1997; White et al., 1998) and to layers K3 and K4 in macaque monkeys (Reid et al., 1997). Because K3 axons terminate within the CO blobs, a revision of the Hubel/Livingstone model has been proposed in which the K channel now becomes an S cone color channel (Dobkins, 2000). According to this revised view, S cone signals are sent directly to the CO blobs by means of the K pathway, while L and M cone signals reach the CO blobs indirectly from P channel projections that terminate within 4Cβ. This still leaves the P pathway performing both as a color and as a high-resolution spatial vision channel. Calkins and Sterling (1999) have gone one step further by proposing that midget ganglion cells that innervate P LGN cells cannot transmit chromatic opponency to the LGN due to the wiring of their surrounds (Dacey, 1996). The surrounds depend on either horizontal or amacrine cell connections; both horizontal and amacrine cells have been found to receive mixed-cone, not single-cone, connections (see Calkins and Sterling, 1999; Dacey, 2000, for review). According to this model, the L and M cone (red and green) channels must project through a nonmidget ganglion cell class: perhaps another type of ganglion cell that sends input to the K pathway. This could mean that all chromatic information reaching V1 travels through the K pathway to the CO blobs (Hendry and Reid, 2000). Other than evidence that the midget to P LGN cell

pathway may not carry pure cone opponent signals, this proposal has appeal if one assumes that true color opponent cells occur in low numbers in the retina and LGN (Calkins and Sterling, 1999). A sparse projection could force cortical terminations to become patchy in order to maintain retinotopic coverage in cortex, thus explaining the presence of CO blobs (Calkins and Sterling, 1999). It is noteworthy in this regard that, even in cats, the only chromatically sensitive retinal ganglion and LGN cells that have been identified are W cells, and these were found to be excited by S cones and inhibited by M cones (Cleland and Levick, 1974). W cells in cats also project to CO blobs (Boyd and Matsubara, 1996).

Appealing as this new proposal is, there are several issues that do not fit. The first concerns the number of K cells required to support both the S and L/M cone channels in macaque monkeys. Counts of K cells immunolabeled with calbindin and α CamII kinase (another marker for K cells in macaque monkeys) have provided evidence that there are too few cells within the foveal and parafoveal representation in macaque LGN to support the acuity of both color channels to cortex (Song et al., 2001). Moreover, in marmosets, only 20% of the total K cell population were found to carry S cone signals; none were found to be L/M cone opponent (White et al., 1998). In the same study, however, two types of P cells were identified: P cells that were L/M opponent and P cells that did not respond selectively to different cone inputs but had good spatial resolution. These same two classes bear a resemblance to the type I and type III P LGN cells identified originally by Wiesel and Hubel (1966). Taken together, these findings suggest that there are two classes of P cells and two classes of K cells but that both P and K channels transmit information related to one color channel, at least in Old World macaque monkeys and New World marmosets. Add to these data new findings indicating that there are two ganglion cell classes in addition to the small bistratified ganglion cells that transmit S cone signals to macaque LGN (Dacey et al., 2001), as well as older data showing that cells responding to S cones as well as to L/M cones have been identified in P layer targets in V1 in macaques and squirrel monkeys (reviewed in LeVay and Nelson, 1991), and the picture becomes even more complex.

Another issue concerns the function of K cells in nocturnal primates that lack S cones such as bush babies and owl monkeys (Jacobs et al., 1996). Both of these primate species have only a single cone type (an M cone), and both have morphologically identified midget and parasol ganglion cells as well as a variety of smaller ganglion cells, although owl monkeys apparently lack small bistratified ganglion cells that carry some of the S cone signals (Yamada et al., 2001). Both bush babies and owl monkeys have well-developed calbindin-positive K layers within the LGN that project to well-defined CO blobs (Casagrande, 1994; Johnson and Casagrande, 1995). Presumably some K cells and some CO blob cells perform noncolor functions that are defined by inputs from as yet to be classified ganglion cells. Additional evidence for heterogeneity among K cells comes from detailed physiological studies of their spatial and temporal properties in bush babies (Norton et al., 1988), owl monkeys (Xu et al., 2001), and marmosets (White et al., 2001). Given the proposal that diurnal primates arose from a nocturnal common ancestor, it seems likely that the origin of the K pathway and projections to the CO blobs arose originally to support some function or functions other than the processing of chromatic signals, with color processing being added later in evolution (Heesy and Ross, 2001). We consider this issue and the evidence for and against continued functional segregation of LGN parallel pathways within V1 in the next section.

How are parallel LGN pathways organized in V1?

As reviewed above, at least three classes of cells can be distinguished within the LGN based on morphology and physiology in all primates examined, as well as in cats. The basic patterns of geniculate to V1 projections that have been documented in primates and cats suggest that X/P medium-sized LGN cells send axons principally to the lower half of cortical layer 4, Y/M large LGN cells send axons principally to the upper half of cortical layer 4, and K/W small LGN cells send axons mainly to layers above 4 (Fig. 31.1). This pattern of projections appears to represent a basic mammalian plan since it has been identified (with some variation) in several other species (Casagrande and Kaas, 1994). Given the ever-increasing number of classes of retinal ganglion cells that project to the macaque LGN, as well as the many types that have been found previously to project to the LGN of cats (discussed in Isayama et al., 2000), it seems reasonable to suggest that more than three ganglion cell classes is the rule, not the exception. Unfortunately, markers such as the calcium binding proteins parvalbumin and calbindin, even combined with cell size, are not sufficient to distinguish the targets of all of these ganglion cells within the LGN of any species, assuming that the pathways remain separate at this level. Clues about how many pathways are maintained in parallel to and through the LGN to cortex can be gained by examining detailed termination patterns of these axons in primate V1 and connections within V1 itself. Several examples follow.

Within primate V1, there is evidence that projections within and among layers and compartments are more precise and complex than was originally believed. For example, in both macaque monkeys and owl monkeys there is evidence that the main recipient layer, Brodmann's (1909) layer 4C, consists of three, not two, tiers. Morphologically, this tripartite organization was originally noted in Nissl-stained sections in macaque monkeys (Lund, 1988). In owl

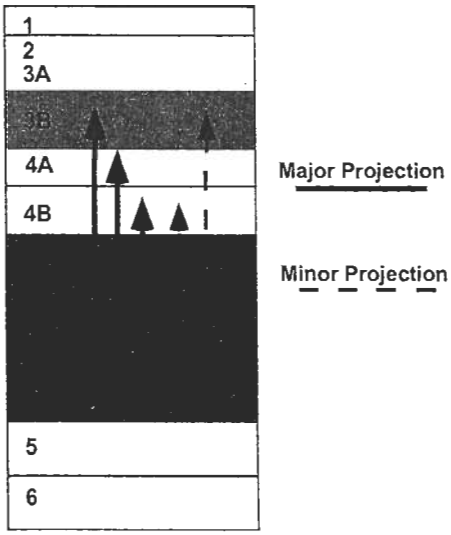

Figure 31.3 Projections from layer 4C to the supragranular layers in V1 of owl monkey. Layer 4Cα projects principally to layer 4B, 4Cβ to 4A, and 4Cctr to 3B. Layer 3A, the major output layer to area V2, receives signals from layer 4 only indirectly via other subdivisions of layer 3 and layer 5. See text for details. (From Boyd et al., 2000, with permission of the publisher.)

monkeys, in vivo studies tracing connections based on small laminar injections revealed that the three tiers of layer 4C projected to different targets within the upper cortical layers (Boyd et al., 2000). As shown in Figure 31.3, cells in 4Cβ project to 4A, cells in 4Cctr project to 3B, and cells in 4Cα project to 4B (Boyd et al., 2000). Intracellular filling of cells in slice preparations within layer 4C in macaque monkeys has identified similar sets of projections (Yabuta and Callaway, 1998). There is also evidence from axon reconstructions in macaque monkeys (Blasdel and Lund, 1983) and in bush babies (Florence and Casagrande, 1987) that axon arbors of some P and some M LGN cells extend through layer 4Cctr, while others are restricted to the top and bottom tiers. Add to these anatomical data the physiological evidence given above that there may be two P LGN cell classes, one color opponent and the other not, as well as evidence suggesting that there are two M LGN cell classes based on linearity of spatial summation in macaque monkeys (Kaplan and Shapley, 1982), and we now have four LGN cell classes projecting to layer 4C of V1.

Examination of LGN projections and connections to cortical layers above layer 4C adds to the complexity. Layer 4A has been proposed to receive input from P cells in macaque and squirrel monkeys (Lund, 1988), yet recent data suggest that cells in this layer in macaque monkeys are excited by S cone stimulation (blue ON), indicating that these cells also could receive input from a class of LGN K cells (Wandell et al., 2002). It is noteworthy that apes (e.g., chimpanzees) lack LGN input to layer 4A (Tigges and Tigges, 1979) and that humans probably also lack such input based on CO staining (Horton and Hedley-Whyte, 1984; Wong-Riley et al., 1993), even though 4A has been described cytoarchitectonically in humans (Yoshioka and Hendry, 1995). If this is true, it suggests that humans and apes lack this particular blue ON channel since they appear not to have LGN projections to cortical layer 4A, projections that are also lacking in the nocturnal primates owl monkeys and bush babies (Casagrande and Kaas, 1994). Why humans and apes that clearly have S cones would not also have a projection to layer 4A remains a mystery, although the recent discovery of a second class of blue ON ganglion cells in macaques suggests that S cones may not be restricted to a single channel (Dacey et al., 2001). Nevertheless, the fact that layer 4A is morphologically distinct and has unique intracortical projections, even in primates that lack direct LGN input to this layer, would indicate that layer 4A performs a specialized role in most primates.

Cortical layers above 4A receive projections from at least two classes of K axons, those that project primarily to the CO blobs within layer 3B and those that project primarily to cortical layer 1 from the ventral most K layers, LGN layers K1/2 (Ding and Casagrande, 1997). The latter pattern has been demonstrated in both nocturnal (owl monkeys) and diurnal (macaque and squirrel monkeys) Old and New World primates, so it appears to remain independent of lifestyle or phylogenetic history (Casagrande, 1994; Hendry and Reid, 2000). In prosimian bush babies, K layer connections also have been demonstrated to project to the CO blobs and layer 1 (Lachica and Casagrande, 1992). In bush babies, however, no investigations have been done of the more ventral K layers to see if these send axons primarily to layer 1. Given the heterogeneity of the axon projection patterns of K cells even in macaque monkeys (Fig. 31.4*A*) and the physiologically demonstrated heterogeneity of K cells in the LGN level in several primate species (see above and Fig. 31.4*B,C*), it seems likely that more than two K axon classes project above cortical layer 4A.

Combining the above data, the following picture of parallel input pathways emerges (Fig. 31.5*A*). Four classes of LGN axons project to cortical layer 4C, two P and two M classes. Layer 4A receives input from either K or P axons or both, but only in a subset of nonhominid primates. LGN axonal projections above layer 4A come from two or more classes of K axons. Interestingly, in other species such as cats, there also is evidence that X, Y, and especially W cells contain subgroups. Lagged and nonlagged X and Y cells have been described (Saul and Humphrey, 1990). Evidence also suggests that the Y cells within the C lamina and/or the medial interlaminar nucleus (MIN) in cats are not functionally equivalent to those in the A laminae (Boyd et al., 1998). W cells within the parvocellular C laminae of cats have been subdivided into at least two classes, W1/Q and W2, or tonic and phasic cell classes, as well as other types (Rowe and Cox,

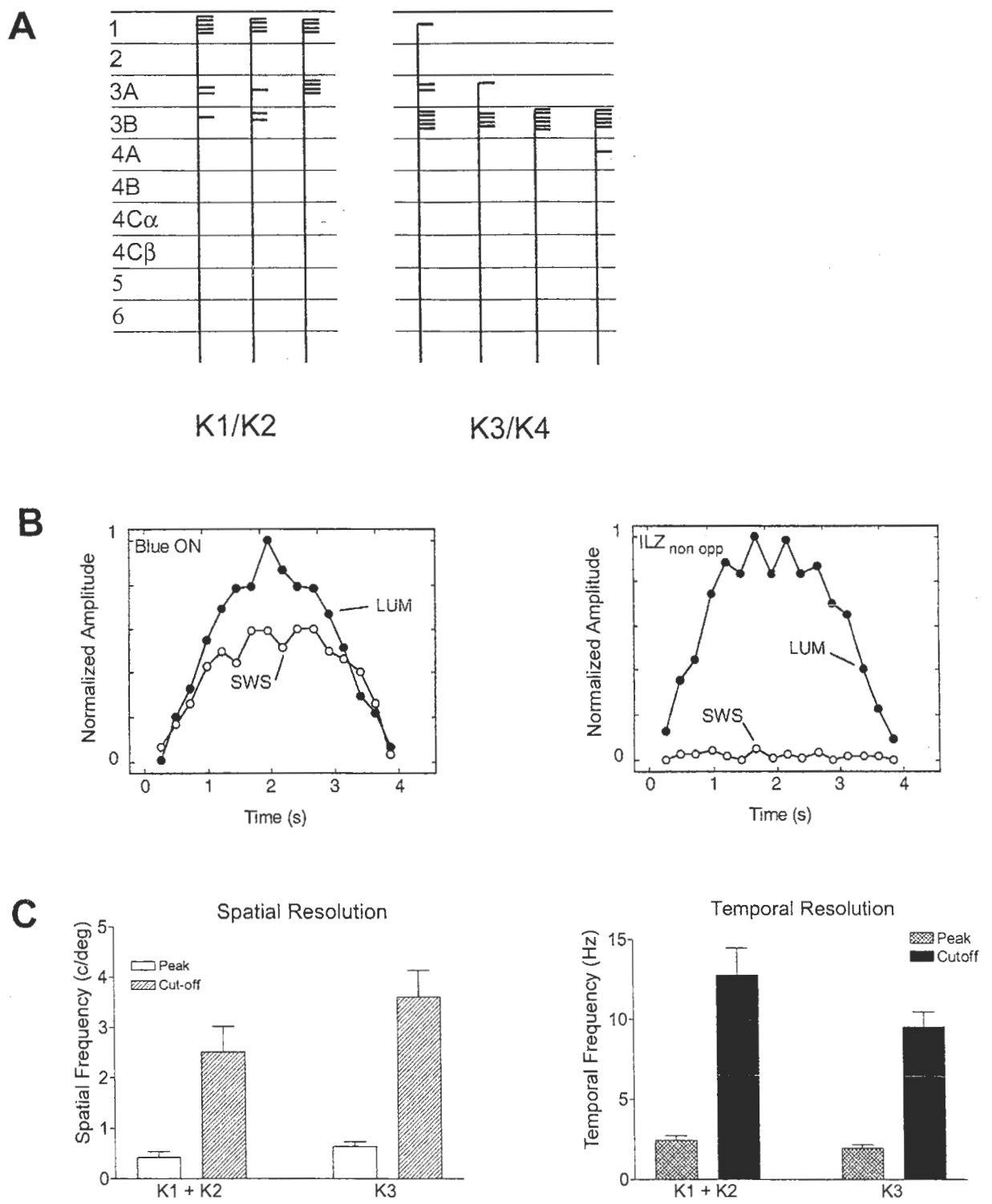

Figure 31.4 Morphological and physiological heterogeneity of K cells. *A*, In macaque monkeys, the geniculocortical projections from different K layers are morphologically distinct; axons from LGN layers K1/K2 mainly project to cortical layer 1, while axons from LGN layers K3/K4 chiefly terminate in cortical layer 3. *B*, Responses of different K cells to a short-wavelength-sensitive (SWS) isolating stimulus and luminance stimulus (LUM). About 20% of K cells in the marmoset LGN are blue ON cells (shown in *B* on the *left*) and respond to the SWS stimulus; 80% of K cells [also called *interlaminar zone* (*ILZ*) cells] show no response to the SWS stimulus (shown in *B* on the *right*). *C*, Spatial and temporal characteristics of owl monkey K cells in different LGN layers exhibited differences. Cells in K1/K2 tend to be selective for lower spatial frequencies and higher temporal frequencies than cells in layer K3. (*A* from Casagrande et al., 1997; *B* modified from White et al., 1998, with permission of the publisher; *C* modified from Xu et al., 2001, with permission of the publisher.)

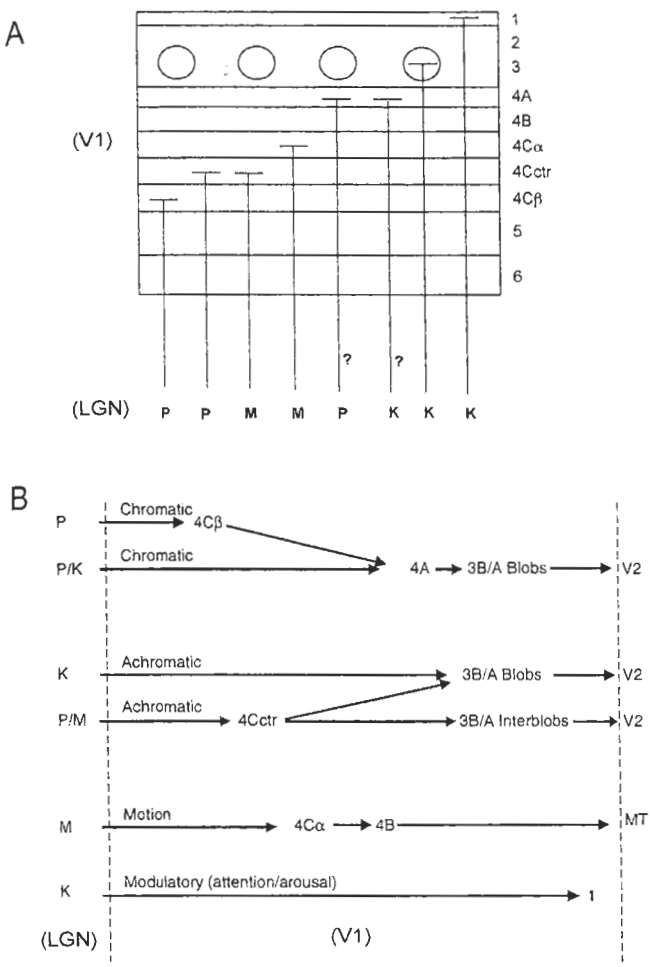

Figure 31.5 Parallel pathways from the LGN to V1 in macaque monkey. *A*, Eight parallel pathways are shown from the LGN to the cortical layers of V1 in macaque monkey: three P channels, three K channels, and two M channels. The circles within layer 3 represent the CO blobs. It is unclear whether K, P, or both pathways project to layer 4A. It is noteworthy that LGN projections to layer 4A represent a specialization of some simians only; chimpanzees, humans, owl monkeys, and bush babies lack this pathway. The layers of cortex are indicated by Arabic numerals. *B*, Connections and possible functions of the eight parallel pathways within V1. In this diagram the ventral to dorsal sequence of layers in V1 is laid out from left to right. V1 projects directly to different compartments within V2 and to area MT. Not shown: V1 also sends axons to DM/V3a which exit from CO blob columns, to area V3, and to area V4. See text for details.

1993; Sur and Sherman, 1982; Troy et al., 1995). All of these data argue against a simple two-channel model for parallel pathways to and through the LGN.

Functional implications of multiple LGN channels to V1

WHY HAVE TWO M CHANNELS? If primates have multiple M, P, and K channels to V1, what are the functional implications of such a model? First, beginning with the M LGN cells, Blasdel and Lund (1983) found two classes of M axons terminating in layer 4Cα in macaque monkeys. The majority of axons arborized throughout layer 4Cα, with minor or no collateral input to layer 6, while the arborizations of the others were restricted to the upper half of layer 4Cα, with extensive collaterals in layer 6. Later, it was theorized that differences in receptive field sizes and contrast sensitivities in these two populations could account for changes in these properties with depth in layer 4C (Bauer et al., 1999; Lund et al., 1995). Cells at the top of 4Cα have larger receptive fields and higher contrast sensitivity than cells deeper within 4Cα. In addition, like cells in 4B, many cells at the top of 4Cα are orientation and direction selective. This arrangement suggests that the M LGN cells projecting to 4Cα are part of a channel allowing for rapid transmission of information important to stimulus motion directly to dorsal stream areas like the middle temporal (MT) visual area via cortical layer 4B or possibly even via synapses on dendrites of large 4B pyramidal cells that dip into 4Cα (Fig. 31.5*B*). In cats, the class of Y cells that projects beyond area 17 may play a similar role, providing rapid transmission of motion information to higher areas. One could speculate that in the latter case the transmission is even more direct and unfiltered simply because the lifestyle of cats requires a very rapid reaction to motion.

Why then would one need two M channels? In the most basic sense, P and M populations of LGN cells represent two ends of a continuum in the spatial and temporal frequency domains (Schiller et al., 1990). Either channel alone allows for a much more limited sampling of spatial and temporal frequencies, a limitation imposed at the retina simply because ganglion cells cannot have both small and large dendritic fields simultaneously. The second M channel terminating within layer 4Cctr could, along with an achromatic P channel, provide the full range of basic spatial and temporal information to cells in the ventral stream concerned with object identification through connections with cortical layer 3 cells.

WHY HAVE TWO P CHANNELS? Dacey (2000) has proposed that the unique midget cell architecture of the fovea evolved first to permit high spatial resolution, which demands cones, and only recently evolved to carry color opponent L/M channels in Old World primates. If one P channel is transmitting information about spatial detail, the other is presumably a color channel. P input in 4Cβ, with its heavy projections to layer 4A, could represent such a chromatic channel, at least in those primate species that have color vision (Fig. 31.5*B*). Color-selective nonoriented cells have been reported in layers 4Cβ and 4A, at least in macaque monkeys (see LeVay and Nelson, 1991, for review). As mentioned earlier, it is unclear whether layer 4A also receives input from a subset of K cells, from P cells, or from

both cell classes. Interestingly, physiological studies have noted two populations of cells within layer 4A (Blasdel and Fitzpatrick, 1984). Perhaps both P and K LGN cells project to 4A. Unfortunately, older studies claiming that P LGN cells project to layer 4A were based on tracer injections that included the K layers lying below each P layer (Lund, 1988).

WHY SEGREGATE K AXONS TO THE CO BLOBS? The only documented direct input (based on axon reconstruction) to the cortical layers dorsal to layer 4A are K axons in primates. These K axons are restricted to CO blobs within layer 3B, leaving the interblob columns in layer 3 free of any direct LGN input; K axons also appear to project to all of layer 1 (Casagrande et al., 1997; Ding and Casagrande, 1997; Lachica and Casagrande, 1992). This arrangement certainly suggests that CO blobs and interblobs within layer 3 perform different functions. Also, the fact that CO blobs can be distinguished neurochemically in several other ways from interblobs (Wiencken and Casagrande, 2000) argues that these two compartments do different things. The original proposal from Livingstone and Hubel (1988) was that the CO blobs send signals to extrastriate areas that are relevant to color, while the interblobs are concerned with putting together signals to support form vision. Although this scenario would fit data from diurnal monkeys, it does not fit data from nocturnal primates that appear to have the same direct K LGN projections to CO blobs and the same basic arrangement of intracortical connections in V1 (Casagrande and Kaas, 1994). Interestingly, one difference was found between diurnal and nocturnal simians in the microcircuitry of the CO blobs (Shostak et al., 2002). The subset of K axons that synapse on dendritic shafts in diurnal macaque and squirrel monkeys synapse on significantly larger shafts than in the nocturnal owl monkey. Shostak et al. (2002) have argued that it is possible that the latter shift represents a loss of K axons that carry S cone signals in owl monkeys. Since the majority of K LGN cells probably do not carry color signals anyway (see above) and cannot carry these signals in nocturnal species, we are still left with the question of why the CO blobs exist. Clearly, CO blobs are not necessary for good color vision since many species, such as tree shrews and ground squirrels, have excellent color vision but no CO blobs (Jacobs, 1993; Wong-Riley and Norton, 1988). CO blobs, however, appear to be a universal feature of primate V1. What primates do, more than other species, is to use vision to analyze objects that they manipulate. Cortical layer 3, which sends information into the ventral stream, is particularly well developed in primates. One possibility is that the special demands of high-level object vision require that stimulus attributes be combined in several different ways to support the variety of analyses performed by the ventral stream hierarchy of areas. The compartments within the CO blobs and interblobs that extend throughout layers 2 and 3 may provide the substrate of the variety of combinations necessary. Further support for this scenario comes from the fact that so many distinct extrastriate visual areas receive input directly from cells located within cortical layer 3, including different compartments within area V2 and areas V3 and V3a and its presumed homolog, the dorsal medial area (DM) (Beck and Kaas, 1999).

An interesting proposal by Allman and Zucker (1990) on the function of CO blobs suggested that cells in CO blobs are designed to analyze scalar variables related to the intensity of the stimulus such as brightness, color contrast, and texture, while interblobs are designed for analysis of geometrical variables such as orientation preference and possibly more complex binocular interactions. From a phylogenetic perspective, this proposal still has considerable appeal since this segregation of function could have evolved first in the absence of color vision, with color contrast added later. CO-blob cells are in a position to integrate the full range of information from different K axons directly and P and M axons indirectly. In this respect it is noteworthy that in cats Cleland and Levick (1974) recorded from an S cone–driven W cell that appeared to substitute rod input for S cone input under scotopic conditions. There have also been other reports in primates that LGN cells carrying S cone signals exhibit rod input under dark-adapted conditions (Virsu et al., 1987). M cells also carry rod signals (Virsu et al., 1987) and tend to have a larger input to CO blobs than to interblobs. Taken together, all of these inputs could provide CO-blob cells with the ability to maintain constancy over a very broad range of stimulus intensities and wavelengths under both scotopic and photopic conditions. From this perspective, it would be interesting to know whether there are any differences in the responses of CO-blob and interblob cells under photopic verses scotopic conditions, as would be predicted by such a model.

WHY SEND LGN SIGNALS TO CORTICAL LAYER 1? As reviewed earlier (Casagrande, 1994), the K axons that project to cortical layer 1 are in a position to modulate activity within many pyramidal cells throughout the depth of V1 since the apical dendrites of some cells in almost all layers extend into cortical layer 1 (Lund, 1988). The K axons that have a branch in cortical layer 1 have been described following injections involving almost all of the K layers (Fig. 31.4*A*; also see Ding and Casagrande, 1997), but the bulk of these projections appear to come from K layers 1 and 2. Jones (1998) has stated that there is a correlation between calcium binding protein content in thalamic relay cells and projection patterns to cortex, with the calbindin-positive cells having projections above layer 4 and being part of a more diffuse system serving a role in recruitment of more widespread areas of cortex, in contrast to the parvalbumin-

containing cells, which are dedicated to sensory transmission. In the K pathway, this scenario would hold only for cells projecting to layer 1 since other K cells clearly project to very limited areas of cortex. Regardless, this hypothesis is of interest given the long history of proposals implicating cortical layer 1 in attention, arousal, and other forms of modulation (Vogt, 1991). Interestingly, both K LGN cells and cells in the adjacent inferior pulvinar are immunoreactive for calbindin, receive input from the superficial colliculus (also implicated in attention), and send axons to cortical layer 1 of V1 (Ogren and Hendrickson, 1977). Evidence for the pulvinar's involvement in attention has a long history (Robinson and Petersen, 1992).

Do Differences in Visual Latencies Offer Functional Clues? One issue that has not been considered is that of differences in visual latencies between pathways and the impact this may have on the responses of cortical neuron targets. Clear differences in the visual onset latencies of K, M, and P cells have been demonstrated at the level of the LGN (e.g., Irvin et al., 1986). As discussed by Maunsell et al. (1999), it is difficult to predict the cortical impact of these differences because stimulus attributes, stimulus intensity, and degree of convergence of inputs on the postsynaptic cell all affect response latency. It is still possible, however, that V1 takes advantage of these timing differences to increase the probability that cells reach threshold. For example, one could imagine that within the CO-blob cells might receive signals from the slower K axons that have direct projections at the same time as signals from the faster M and P pathways that must traverse several synapses. Due to greater convergence from larger numbers (Maunsell et al., 1999), signals from P cells would arrive at about the same time as those from M cells. From this perspective, it would be of interest to know how removal of one of these pathways affects the thresholds of cells within the output layers of V1. The timing differences may also be important in combining signals from the feedforward parallel pathways and the multiple pathways that feed back to V1.

Conclusions

Parallel pathways from the retina via the LGN to V1 presumably act to maintain the integrity of signals that can only be combined at later stages of processing. Since V1 is concerned with local feature analysis, the layer and compartmental geometry of V1 provides a substrate for such independent processing and for the creation of different combinations of output pathways. The evidence reviewed above suggests that there may be as many as 10 pathways that pass through the LGN that could impact V1 processing in different ways; good evidence for at least 8 now exists. The puzzling question is why there are so many. In some cases, evolutionary history may offer a possible explanation. Cones would be expected to provide input to any system designed for high-resolution vision, like midget ganglion cells. The addition of another cone type would simply provide a potential substrate for transmission of color signals along the same pathway. It is less easy to explain why K cells might carry S cone signals, except that this appears to happen earlier in evolutionary history if one considers that W cells also carry these signals. The coevolution of M and P cells or X and Y cells (and perhaps other subtypes as well) also may be, as mentioned earlier, the result of incompatibility (e.g., sensitivity often is incompatible with spatial resolution). The variety of conventional and unconventional responses of different K cell populations to visual stimulation suggests that LGN channels may do more than perform a simple analysis of spatial and temporal frequencies. The challenge for the future is to understand how V1 cells use the different views of the visual world that each of the parallel pathways provide. This can only be done by taking into account the dynamic aspects of the system by sampling from multiple cells at different levels within the system while the system is exposed to a variety of visual stimuli.

Unanswered questions

- How many pathways project from the retina to and through the LGN? How many of these pathways generalize across primates or across other mammals like the cat?
- If 10 ganglion cell classes project to the LGN in macaque monkeys, are there 10 separate target cell classes in the LGN? If so, what criteria can be used to distinguish them?
- To what extent do different LGN cell classes carry redundant information to V1?
- What is the functional significance of different calcium binding proteins within the different layers of the LGN?
- Why are there so many types of K cells?
- Why do K and W cells get input from the superior colliculus?
- Do LGN K cells and P cells both carry chromatic signals in diurnal primates?
- Why are LGN projections to layer 4A seen in only some primates?
- Why are S cones missing in nocturnal primates?
- Do CO-blob cells function differently in diurnal and nocturnal primates or in cats?
- Why do K cells in primates and W cells in cats send axons to the CO blobs and not to the interblobs?
- V1 projects to three compartments in V2 and also to compartments in V3, V3a/DM, MT, and V4. Do all of these projections arise from separate groups of cells in V1?
- How many extrastriate target areas of V1 are homologous across primates?

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