

# THE KONIOCELLULAR PATHWAY IN PRIMATE VISION

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■ **Abstract** A neurochemically distinct population of koniocellular (K) neurons makes up a third functional channel in primate lateral geniculate nucleus. As part of a general pattern, K neurons form robust layers through the full representation of the visual hemifield. Similar in physiology and connectivity to W cells in cat lateral geniculate nucleus, K cells form three pairs of layers in macaques. The middle pair relays input from short-wavelength cones to the cytochrome-oxidase blobs of primary visual cortex (V1), the dorsal-most pair relays low-acuity visual information to layer I of V1, and the ventral-most pair appears closely tied to the function of the superior colliculus. Throughout each K layer are neurons that innervate extrastriate cortex and that are likely to sustain some visual behaviors in the absence of V1. These data show that several pathways exist from retina to V1 that are likely to process different aspects of the visual scene along lines that may remain parallel well into V1.

## INTRODUCTION

In the path from retina to primary visual cortex, the lateral geniculate nucleus (LGN) is most often seen as a stage of simple relay. For primates in general and for humans and macaques specifically, two populations of retinal ganglion cells are commonly held to drive the physiological properties of neurons in primary visual cortex (V1) by way of a relay through two sets of layers in the LGN. That is, midget and parasol ganglion cells send separate projections to parvocellular (P) and magnocellular (M) layers of the LGN (Leventhal et al 1981, Rodieck et al 1985). The LGN layers, in turn, innervate separate sublaminae in layer 4 of V1 (Hendrickson et al 1978, Hubel & Wiesel 1972). To a degree that appears to vary with time and experimental protocol, the M and P channels remain largely segregated in V1 (Livingstone & Hubel 1987, 1988) or converge early and often (Martin 1992, Merigan & Maunsell 1993). Yet in this debate about channels that lead from retina to V1, and from there to extrastriate cortex and visual perception,

few have argued about the basic principle that two channels leave the retina, relay through the LGN, and enter V1. What we review here is the evidence for a third koniocellular (K) channel, anatomically separate and physiologically distinct from the M and P pathways. Evidence for a K visual pathway has been ably summarized in a recent review (Casagrande 1994), with particular emphasis on results from the prosimian bushbaby (*Galago*). Because the data from studies of bushbabies have been widely interpreted in the light of the specialized niche and natural history of that genus, we emphasize findings from simian primates, particularly Old World macaques. We set the earlier findings into a context gained from more recent studies that show the third channel to be a robust and functionally distinct part of the visual system in all primates.

## HISTORY AND NOMENCLATURE

Among the early studies of cell architecture in the LGN of Old and New World monkeys, apes, and humans, several authors made note of “extremely small and lightly stained” somata outside the M and P layers (Le Gros Clark 1941a,b; Solnitzky & Harman 1943; Chacko 1948, 1954b,c, 1955a,b). The description by Le Gros Clark (1941b) for New World monkeys placed these somata in three locations that appeared to vary between species but that included a band between magnocellular layer 1 and axons of the optic tract, a band between the two magnocellular layers, and a band between the more dorsal magnocellular layer (layer 2) and the most ventral parvocellular layer (layer 3). Similar kinds of descriptions were offered for other species of monkeys and for apes and humans during the period when Nissl stains were the principal means for studying the thalamus. Most studies made clear how thin and variable were these populations of neurons when viewed in context with the much larger cells of the M and P layers. Differences in the number and placement of the small-celled layers were described in some detail (for reviews see Giolli & Tigges 1970, Kaas et al 1972), yet the various descriptive terms assigned to the layers indicated the role most investigators thought they played in visual perception. Whether they were referred to as minor, subsidiary, or even supernumerary, a term preserved in at least one modern study (Tootell et al 1988), the small-celled layers were viewed as an odd addition to an otherwise complete LGN or, more frequently, as a population in decline over the course of primate evolution.

### K Layers in Prosimians

A different story has been told for the LGN of prosimians. Studies of bushbabies (Campos-Ortega & Glees 1967, Campos-Ortega & Clüver 1968, Glendenning et al 1976, Ionescu & Hassler 1968, Tigges & Tigges 1970) and of lorises (Chacko 1954a, Laemle & Noback 1970) were able to detect well-formed M, P, and K layers. Thus, not only are there thin, small-celled layers among the M layers of

prosimian LGN, but there are also major populations of small neurons that form easily detectable layers inserted between the two P layers. In recognizing these layers of small cells as a separate population, Kaas and his colleagues (1978) proposed the name koniocellular to describe them. That term seems particularly apt, as it retains a style of naming layers of the LGN by the size of the somata within them and reinforces a description of these as being small enough to resemble grains of sand. To be sure, other names have been given and continue to be used when describing K layers. Because of the locations of some K layers, inserted as it were between pairs of more robust M and P layers, they have been called intercalated layers (Fitzpatrick et al 1983, Guillery & Colonnier 1970) and even interlaminar cells. As spelled out below, the neurons intercalated between all M and P layers and the small cells ventral to M layer 1 closely resemble the K neurons of bushbaby LGN, and thus the term koniocellular seems appropriate for them all. Regarding the term interlaminar, we agree with Fitzpatrick and his colleagues (1983) that there is some illogic in referring to neurons that form layers by a name that implies they exist outside layers. For these reasons, we find koniocellular to be the best term to use in referring to the small-celled layers of the primate LGN and we employ it throughout this review.

### What Are the S Layers?

There remains the name most commonly used to describe a subpopulation of K neurons. Sandwiched between M layer 1 and axons of the optic tract are two populations of neurons, one innervated by the contralateral retina and the other by the ipsilateral retina (Kaas et al 1978). Either or both were once called lamina 0 (Balado & Franke 1937, Hassler 1959), but in studies published over the past 20 years they have been referred to as S layers. That term was once restricted to a population of ventrally placed neurons innervated by the ipsilateral retina (Giolli & Tigges 1970), but its domain was enlarged in a seminal study of retinal innervation of monkey LGN to include neurons innervated by contralateral retina (Kaas et al 1978). Although Kaas and his colleagues (1978) took care to state they had assigned this term without reference to the sizes of the innervated cells, both the ipsilateral- and contralateral-recipient laminae have since been called "small-celled S layers" (Weber et al 1983, Harting et al 1991c). That description is accurate only for the population of cells innervated by the contralateral retina.

In squirrel monkeys (Fitzpatrick et al 1983), bushbabies (Casagrande & DeBruyn 1982), and rhesus monkeys (Yoshioka et al 1999), ipsilateral retinal axons ventral to M layer 1 innervate a population of large neurons that make up a displaced part of magnocellular layer 2. In contrast, the contralateral retina innervates a much more numerous and widely distributed population of small neurons. Only this contralateral-recipient group is neurochemically identical to K cells of other layers (Yoshioka et al 1999b). Apparently, then, the term S layers has been used to name two very different groups of neurons, one magnocellular and the other koniocellular. One might argue that S layer could be retained as a

name for the magnocellular group, but its continued use to designate the most ventral K layer unnecessarily divides it from layers of similar structure, function, and connectivity while lumping it into the same population as a very different group of displaced M cells.

## A Contribution from Chemistry

As recently as five years ago, only the K layers of bushbabies were well studied and well understood. In studies conducted by Casagrande and her colleagues, the aggregation of K cells into two wide layers was exploited to determine the connectional and physiological properties of these neurons (Casagrande & DeBruyn 1982, Casagrande 1994). As is described below, much of this work had strong predictive value for what has been found for K layers across the primate Order. Other studies in squirrel monkeys established several essential truths about K layers in simians (Fitzpatrick et al 1983, Weber et al 1983). Yet these studies were either ignored or dismissed as largely irrelevant to the visual system of Old World primates, because in these latter species the K layers were held to be too thin and patchy to support the function of any neuronal group in visual cortex.

Changes in the perception of K layers as tenuous or gossamer thin are due in part to the discovery that K cells in macaques and all other species of primates differ neurochemically from the neurons of the M and P layers. In adult monkeys there are three proteins, in fetal monkeys a fourth, that among the neurons of the LGN are expressed exclusively by K cells. In adults, the 28-kDa calcium binding protein, calbindin (Jones & Hendry 1989, Diamond et al 1993, Johnson & Casagrande 1995, Goodchild & Martin 1998), the alpha subunit of type II calmodulin-dependent protein kinase (Benson et al 1991, Hendry & Yoshioka 1994), and the gamma subunit of protein kinase C (Fukuda et al 1994) are expressed by K cells and not by M and P cells. In fetuses of middle to late gestation, a second calcium binding protein, calretinin, is expressed transiently (Yan et al 1996). These molecular tags have allowed K cells to be viewed and studied in isolation, much as similar tags have permitted specific retinal populations to be examined without interference from neighbors of other types (Hendry & Calkins 1998). Recent studies that describe something of the organization and function of the K layers in macaques and other monkeys were achieved because these cells are neurochemically distinct.

There is no easy answer to the question of what K cells can do, with their molecular makeup, that M and P cells cannot. So far as calbindin expression is concerned, neurons in many nuclei of the macaque thalamus are divided cleanly into those immunoreactive for calbindin and those immunoreactive for a second calcium binding protein, parvalbumin (Jones & Hendry 1989). Yet as studies of cortical inhibitory interneurons have shown, it is not obvious how one protein affects  $\text{Ca}^{2+}$ -dependent processes in ways that the other does not (Andressen et al 1993). That leaves a situation in which the difference in protein immunoreactivity is convenient but not predictive of the way two populations differ physio-

logically. Selective K cell expression of  $\alpha$ CaM II kinase ought to be more easy to interpret, because this is a protein whose function at the cellular level is well understood (Hanson & Schulman 1992). What stands out, however, in studying kinase expression across the macaque thalamus is not how the K cells differ from the majority of thalamocortical neurons but how the M and P cells do. That is, of the major populations of relay neurons in macaque thalamus, only M and P cells fail to express  $\alpha$ CaM II kinase (Benson et al 1991, Yoshioka & Hendry 1999a). From such a pattern that spans nuclei we anticipate that K cells would act much like other thalamocortical neurons, whereas M and P would not.

## ORGANIZATION OF K LAYERS

### Qualitative Observations of K Cell Distribution in Macaques

Through much of the macaque LGN, K neurons form six layers. Thus, caudal to the representation of the optic disk and rostral to the extreme posterior pole of the nucleus, six thin bands of K cells are apparent (Yoshioka & Hendry 1999a). That is, wherever there are found two M and four P layers, there exists a K layer ventral to each. Where the number of principal layers is reduced to four (rostral to the representation of the optic disk) or two (the representation of the temporal crescent or the extreme caudal pole of the LGN), the number of K layers is similarly reduced. So long as they are immunocytochemically stained for  $\alpha$ CaM II kinase, sagittal sections through the macaque LGN provide a clear view of the fusion of K layers as the representation of central retina gives way to a representation of paracentral and finally peripheral retina. More important than the details, however, is the simple observation from immunostained sections that K layers exist from the rostral pole to the caudal pole of the LGN (Hendry & Yoshioka 1994, Yoshioka & Hendry 1999a). Moreover, each of the K layers is particularly robust in the caudal one third of the LGN. These findings demonstrate that K layers of the macaque LGN are present through the full representation of the visual hemifield and that they are especially abundant in the representation of the fovea.

K cells are not restricted to K layers. In each part of the macaque LGN, small neurons that display the neurochemical signature of K cells are present in M layers and, most frequently, in P layers (Hendry & Yoshioka 1994, Yoshioka & Hendry 1999a). Because these cells also innervate the same cortical targets as neurons in the K layers proper (Hendry & Yoshioka 1994, Yoshioka & Hendry 1999b), it is reasonable to conclude that small,  $\alpha$ CaM II kinase-immunostained neurons in the M and P layers are a subpopulation of K cells. The greatest number of displaced K cells is present in the middle two principal layers of the LGN, P layers 3 and 4. There they often occupy distinct clusters that span the distance between two adjacent K layers and, by doing so, form a K cell bridge across a P layer

(Hendry & Yoshioka 1994). The largest such bridge, however, is found in M layer 1 at the representation of the optic disk. As much as 0.5 mm in diameter, this group of K neurons includes several hundred neurons (Yoshioka & Hendry 1999a). In addition to forming clusters or bridges, K cells are found in small groups, in pairs, or as isolated individuals in any principal layer. Cells of this sort are particularly abundant in the two most dorsal P layers.

The six K layers found through much of the macaque LGN are not equal in size. By far the largest is the one ventral to M layer 1 (in a nomenclature that assigns each K layer the same number as the principal layer immediately dorsal to it, referred to as layer K1). Layers K2, K3, and K4 are thinner but nonetheless substantial bands of cells, whereas layers K5 and K6 are seldom more than monolayers, often interrupted by cell-free gaps as wide as 100  $\mu\text{m}$ . There are hints in this distribution of a paired arrangement, in which the two most ventral K layers form one unit with a similar organization, as do the two middle and the two most dorsal layers (Yoshioka & Hendry 1999a).

### Quantitative Analyses of K Cells

Although they have something of a checkered past, quantitative studies of primate LGN have highlighted the relative contributions of M and P cells to visual function. These studies indicate that neurons of the P layers outnumber those of M by one order of magnitude (1 million versus 100,000) (Ahmad & Spear 1993, Suner & Rakic 1996), that one fourth of neurons in P layers and one third in M layers are GABAergic interneurons (Montero & Zempel 1986, Hendry 1991), and that the divergence ratio for M cells (i.e. the number of neurons in layer IVC $\alpha$  versus thalamocortical neurons in the M layers) is 1:300, whereas the ratio for P cells is 1:50 (Peters et al 1994). These and related findings of axonal branching in layers IVC $\alpha$  and IVC $\beta$  demonstrate that the precision of the P cell input to V1 is contrasted with the greater amplification of M cell input.

A similar sort of analysis for K cells is instructive of how they compare with the other major LGN populations. When immunoreactivity for  $\alpha\text{CaM}$  II kinase is used as the default marker for K cells, their number (100,000 per rhesus monkey LGN) is found to approach that of M neurons and to be one tenth that of P cells (Hendry 1994, Hendry & Meszler 1999). Yet none of the immunoreactive K cells is GABAergic (Hendry 1994), as  $\alpha\text{CaM}$  II kinase and the GABA synthesizing enzyme, glutamate decarboxylase, are expressed by nonoverlapping neuronal populations throughout the macaque forebrain (Benson et al 1991). Thus, all 100,000 kinase-immunoreactive K neurons are most likely thalamocortical cells, whereas the equally populous M layers include some 30,000 interneurons (Montero & Zempel 1986, Hendry 1991) and only 70,000 thalamocortical cells. If we take the targets of macaque K cells to be the cytochrome oxidase-rich patches or blobs in layers II and III of V1 (see below), the divergence of K cells to potential target cells (i.e. the number of cortical neurons within CO-rich blobs of layers II and III) is roughly 1:50, a figure close to that of the P layers (Hendry 1994,

Hendry & Meszler 1999). These figures place an upper limit on the relative breadth of the role of the K pathway in vision. Because thalamocortical neurons of this pathway are only slightly more numerous than those of the M pathway but lack the power to dominate cortical circuits that comes from the high divergence factor and large axons displayed by M cells, we can anticipate roles for K cells that are, themselves, limited in scope.

## A General Plan for K Layers in Primates

An organization of K layers, similar to that of macaques, can be detected across the primate Order; and in every case, the K layers display the same neurochemical attributes as homologous neurons in the macaque LGN. Specifically, neurons that are appropriately considered K cells are calbindin immunoreactive in species that include prosimians, nocturnal and diurnal New World monkeys, macaques, apes, and humans (Hendry & Casagrande 1996). The default condition for primates appears to be a four-layered organization, much like the basic organization of the principal layers, in which K cells are present ventral to both M layers and both P layers. Where P layers are split into leaflets, thereby producing the textbook example of a six-layered LGN, the dorsal K layers are similarly divided. This four-layered organization is particularly clear in the unusual simian genus *Tarsier*, whereas six layers are found in each of the Old World primates, including macaques, orangutans, and humans (Hendry & Casagrande 1996).

There are variations in the relative size of K layers, so that the most ventral is also most robust in Old World primates (Hendry & Yoshioka 1994, Yoshioka & Hendry 1999a), whereas the equivalent of K4 is greatly enlarged in bushbabies (Diamond et al 1993, Johnson & Casagrande 1995). The greatest variation is seen in, rather than across, suborders of primates, as species of New World monkeys display several unusual patterns. Perhaps most unusual is the pattern seen for capuchin monkeys (*Cebus apella*), in which K cells with stereotypical neurochemical and neuroanatomical attributes of K cells form no distinct layers dorsal to the first P layer. These cells, which would otherwise form layers K4–6, are instead richly and widely distributed in the P layers (Hendry & Casagrande 1996). Few of these variations make intuitive or predictive sense, with respect to niche or family. The exception, perhaps, is the complete absence of layer K4 in owl monkeys (Hendry & Casagrande 1996), the one nocturnal simian species, which does not express the S-cone pigment gene (Jacobs et al 1993). Such a discovery suggests that for simians, layer K4 may be part of an S-cone-dominated pathway (see below).

## DEVELOPMENT AND PLASTICITY OF K LAYERS

How the pattern of K layers comes about during normal development and how it might be perturbed by manipulation or the visual periphery is understood only in its most rudimentary details. Studies of neuronal birthdating in rhesus monkey

LGN suggest that K cells are generated contemporaneously with neighboring M and P cells (Rakic 1977a,b). The first-born geniculate neurons come to occupy the most ventral aspect of the nucleus, the last born the most dorsal. Because there is no evidence of a period in which K cells are selectively generated, these findings suggest that neurons of layer K1 are born close to the time of final mitosis for cells in M layer 1, whereas cells of layer K6 are born at or slightly before the time of cells in P layer 6. The birthdates for neurons in the rhesus monkey LGN are packed into a 2-week period, however, and the gradients in cell generation are not nearly so steep as those seen for cells in primary visual cortex (Rakic 1977a,b). Thus, a difference in a day or two between the birth of cells in a K layer and cells in an overlying principal layer would not be detected. What these data provide is a strong indication that within a very short period of time, K cells are generated and migrate with M and P cells.

### LGN Lamination

In the period between the first generation of LGN neurons at embryonic day 38 (E38) and the midpoint of gestation in rhesus monkeys (E83), the aggregate that would become the adult LGN displays no sign of lamination or of differences in the sizes of neuronal cell bodies (Rakic 1977a,b). Studies of Nissl-stained LGNs indicate that beginning first in caudal and ventral LGN, among the M neurons of what will become the foveal representation, a division of cells into layers is seen and the relative increase in size of M neurons can be detected. The earliest date for initial lamination is approximately E90. From there, differentiation of M and P cells and their separation into distinct layers proceeds rostrally and dorsally. The event causing the mass of LGN neurons to crystallize into a laminated pattern is unknown, although it is logical to anticipate that K cells play some role in this process because these are, after all, the neurons that separate M and P layers from one another.

Through use of calcium binding protein immunoreactivity to mark K cells selectively, Ty and his colleagues (1998) found the initial event in laminar organization of the macaque LGN is invasion by an acetylcholinesterase-rich population of afferent axons on or near E80. Within a period of 5–7 days after these axons appear, K cells go from what appears as a randomly distributed population to one that is grouped into a series of thin bands. Only later, delayed by a period of 3–5 days, do M then P layers become organized as layered groups of cells. These data indicate that of the three major neuronal populations in the macaque LGN, K cells are the first to laminate, and they appear induced to do so by the invasion of an afferent system, perhaps arising from a subpopulation of retinal ganglion cells.

### Developmental Plasticity in the LGN

When the retina that innervates them is deprived of pattern vision, M and P layers of the macaque LGN shrink dramatically (Headon & Powell 1973, Hubel et al 1977, von Noorden 1973) and retract their axonal arbors in V1 to a series of

narrow stripes (Hubel et al 1977, LeVay et al 1980). This effect can be attributed to competition among neurons in the LGN and among their axons in V1. What then of the K layers? Strong competition among K cells is unlikely because K layers are separated from one another by an intervening M or P layer, whereas their patchy terminations in V1 are separated by a large expanse of noninnervated tissue. Both of these factors lead to a prediction that neither K cells nor their axonal domains would be much affected by a loss of patterned visual input.

That prediction has been supported by two observations. (a) Immunostaining for  $\alpha$ CaM II kinase and calbindin is robust in adult macaques deprived of vision in one eye from birth (Ty et al 1998). The immunostaining is not just normal but unusually rich in oddly distributed clusters of cells. These clusters are also seen in some late fetuses and early neonates. The data demonstrate, then, that K cells display no reduction in size or number when deprived of patterned visual input and suggest that deprivation has the effect of stabilizing an immature pattern of immunoreactivity in the LGN. (b) Not only the K cell somata but also their axons in V1 remain overtly normal in monocularly deprived monkeys. Horton & Hocking (1998) found that patchy terminations of K cell axons are equally robust at the centers of deprived-eye and normal-eye columns. So although the M and P cell arbors in deprived-eye columns shrink to a fraction of their normal size, K cell arbors appear to be normal in size and terminal density.

## FUNCTIONAL CIRCUITS OF K LAYERS

### Retinal Innervation

Although it was once thought unlikely that K cells of the simian LGN are innervated by retinogeniculate axons, recent studies have found that all K layers of rhesus monkeys receive retinal inputs from either contralateral or ipsilateral retina (Yoshioka et al 1999). To a first approximation, each K layer is innervated by the same retina as that which innervates the overlying M or P layer. Thus, layers K1, K4, and K6 receive contralateral retinal inputs, and layers K2, K3, and K5 ipsilateral. The one complexity to this picture is layer K2. It is split into separate tiers of immunostained neurons in rhesus monkeys, the more dorsal of which is innervated by ipsilateral retina and the more ventral by contralateral retina. In this respect, K2 of macaques resembles the extremely robust K4 of bushbabies, itself divided into dorsal and ventral tiers of calbindin-immunoreactive neurons (Johnson & Casagrande 1995), innervated separately by the ipsilateral and contralateral retinae (Campos-Ortega & Glees 1967, Campos-Ortega & Clüver 1968, Tigges & Tigges 1970). Examinations of gibbon (Tigges & Tigges 1987) and chimpanzee (Tigges et al 1977) LGN suggest that the contralateral input to K2 persists in anthropoid apes.

Two other regions of unusual retinal innervation are apparent in the macaque LGN. Although each overlaps the location of K layers, neither includes K neurons

as defined by the size and immunocytochemical staining of their somata. As described above, the most prominent is found ventral to M layer 1 (Hubel et al 1977, Kaas et al 1978) and includes large, displaced neurons of M layer 2, innervated by axons of the ipsilateral retina (Fitzpatrick et al 1983, Yoshioka et al 1999). The second is a contralateral-recipient zone stuck between M layer 2 and P layer 3 (Hubel et al 1977, Kaas et al 1978); it is a zone filled not with K cells but with displaced P cells from layer 4 (Yoshioka et al 1999). Thus, layer K2 is the only population of K cells that receives input from both retinae, although even here the axons from the two eyes terminate in separate tiers rather than among the same neurons.

Retinogeniculate axons innervating K layers are thin and poorly branched and give rise to small, en passant boutons (Conley & Fitzpatrick 1989, Conley et al 1987). These are the conclusions, however, from studies in which very few axons, destined for ventral K layers, have been filled. Of the several dozen physiologically characterized retinogeniculate axons that have been filled and studied in primate LGN, those whose terminations are most closely matched to the location of K cells display center-only blue-ON, yellow-OFF receptive fields. Michael (1988) states that axons of this type terminate at the extreme dorsal or ventral borders of P layers 3 and 4. Subsequent studies of LGN receptive fields and of the ganglion cell types that innervate specific layers suggest that these axons terminate, instead, in layers K3 and K4 (see below).

By far the largest number of retinogeniculate axons that innervate K layers have been studied in bushbabies (Lachica & Casagrande 1993). The complexity of these axons greatly exceeds that seen in macaques, even though their terminal morphology (small, en passant swellings that are difficult to resolve at the light microscopic level) is similar in the two species. In addition, Lachica & Casagrande (1993) observed a heterogeneity in the size and shape of retinogeniculate axons ending in bushbaby K layers, with those of the ventral K layers more closely resembling the morphology of axons studied in macaques. Thus, the principal difference in retinogeniculate morphology that has been documented for bushbabies and macaques is less likely to be a species-specific variation and more likely to be a function of which K layers have been examined in the two species. When homologous layers are examined, the morphology of individual axons in macaques closely resembles that seen in bushbabies [e.g. compare Conley & Fitzpatrick (1989: Figure 9) with Lachica & Casagrande (1993: Figure 6)]. It may be safe to anticipate, then, that retinogeniculate axons ending in the middle K layers of macaques are more robust and more variable in their morphology than are axons of ventral K layers.

## Corticogeniculate Axons

The cerebral cortex provides the quantitatively dominant input to M and P layers of the LGN (Guillery 1971, Wilson 1993). The same is true for K layers, as corticogeniculate axons appear to overwhelm all other inputs. K layers are among

the most heavily labeled parts of the LGN when V1 is injected with anterograde tracer, and they are the one recipient of corticogeniculate axons arising from extrastriate areas (Lin & Kaas 1977, Symonds & Kaas 1978). As a result, terminals of the type given off by corticogeniculate axon are by far the most frequently encountered profiles in macaque (Guillery 1971) and bushbaby K layers (Feig et al 1992). These axon terminations in macaque K layers are likely to arise from a population of layer 6 neurons in V1 that is separate from the neurons innervating M or P layers (Fitzpatrick et al 1994).

### Brainstem Afferents

K layers are the only regions of the primate LGN innervated by the superior colliculus (Harting et al 1973, 1978, 1980, 1991a). Numerous studies have shown that tectogeniculate axons arising from the superficial gray layer of the superior colliculus terminate in each K layer of macaques, squirrel monkeys, owl monkeys, and bushbabies. Variations in the density of tectal inputs appear to exist from one species to the next. Thus, layers K1 and K2 receive the densest terminations in squirrel monkeys and rhesus monkeys, whereas the equivalent of layer K4 is most densely innervated in bushbabies [see Harting et al (1991a: Figure 1)]. The evidence for a richer tectal innervation in the more ventral K layers of macaques suggests that these neurons may be more closely allied with the functions of the superior colliculus, including the reflexive control of eye movements.

A second brainstem afferent, selective for K layers, arises from the parabigeminal nucleus of the midbrain (Harting et al 1991b). Often viewed as an auxiliary component of the superior colliculus, the parabigeminal nucleus sends cholinergic axons to the LGN (Fitzpatrick et al 1988). These axons have been illustrated as terminating more densely in the ventral K layers than in the more dorsal [see Harting et al (1991a: Figure 15)]. Thus, both tectogeniculate and parabigemino-geniculate axons in macaques appear to share a preference for K cells near the M layers.

### Intrinsic Elements

The axons of thalamocortical neurons in macaque LGN give rise to few sparsely branched intranuclear collaterals (Wilson 1989, 1993), as part of a pattern seen for thalamic nuclei in general. Golgi studies of what are most likely K cells of macaque and human LGN indicate that the pattern holds for them as well (Garey & Saini 1981, Hickey & Guillery 1981, Saini & Garey 1981, De Courten & Garey 1982), which leaves GABAergic interneurons as the major source of synaptic terminals intrinsic to the K layers. Although immunoreactivity for  $\alpha$ CaM II kinase excludes all neurons that express the GABA-synthesizing enzyme glutamate decarboxylase (Benson et al 1991), that is not to say K layers contain no GABA interneurons. GABA neurons of the K layers are not of the density found in either M or P layers, but they are hardly rare. Their axons and those from the thalamic reticular nucleus often outline the surfaces of kinase-immunostained K neurons

(Hendry 1991). Because K neurons express both  $\alpha$  and  $\beta$  subunits of the GABA<sub>A</sub> receptor, as do thalamocortical neurons of the M and P layers (Hendry & Miller 1996), they appear to be as easily regulated by synaptically released GABA or exogenously applied GABA agonists as are neurons in the principal layers.

## Synthesis of Functional Circuits

Electron microscopic studies of the K layers provide a means to view the relative contributions of various synaptic inputs, to see what is common among M, P, and K circuits, and to determine what is unique. Only in bushbabies have K layers been examined in detail (Feig et al 1992, Feig & Harting 1994); in that species, what stands out is a fundamental rearrangement of retinogeniculate input to K neurons. In M and P layers, retinal afferents form only a minor fraction of the total synaptic input, but the large size of the terminals and of the synapses they form, coupled with the placement of synapses on large, primary dendrites, leads to the production of powerful excitatory postsynaptic potentials in thalamocortical neurons (Guillery 1971, Wilson 1993). The more numerous corticothalamic and brainstem inputs on M and P cells terminate on finer dendritic branches and, by this arrangement, are thought to be a much weaker influence on thalamocortical output. That is not the case for K layers in bushbabies. Retinal axons end in rows of small terminals that form a series of small synaptic contacts on fine dendrites (Feig et al 1992, Feig & Harting 1994). In this regard, the retinal input to K cells differs little from brainstem inputs, and each might be equally effective in driving thalamocortical neurons of bushbaby K layers.

The pattern seen in bushbabies may not be repeated in K layers of other species. Many of the same elements in much the same pattern are present in the more ventral K layers of macaques (Guillery & Colonnier 1970), yet the pattern in the more dorsal K layers appears to differ. In particular, the middle pair of K layers, ventral to parvocellular layers 3 and 4, is innervated by retinal axons of the same size and shape as those terminating in M and P layers. They also form synapses of the caliber typical of the principal layers (SHC Hendry & LB Meszler, unpublished observations). This result suggests that retinal inputs to the middle K layers of macaque LGN may be as influential in controlling the activity of thalamic neurons as those that innervate M and P cells.

A summary of K cell circuits accents the heterogeneity among these neurons both in different species and, for macaques, in different layers. For each K layer that has been studied, retinal inputs compete with a quantitatively dominant corticothalamic innervation and a rich innervation from brainstem nuclei. For the ventral K layers of macaques and for the greatly enlarged K layers of bushbabies, the morphology and location of retinal afferents suggest they possess no advantage over the other afferents in driving neurons. Yet in the middle layers of macaque LGN, retinal terminations are of the right size and location to control physiological properties as thoroughly in K layers as they do in P layers. Thus, no single circuit can be drawn to illustrate how input leads to output in primate

K layers, and the data already available suggests that circuits, and thus physiological properties, vary from one layer to the next.

## PHYSIOLOGICAL PROPERTIES OF K CELLS

### Heterogeneity in Bushbaby K Layers

The receptive-field properties of K neurons have been thoroughly explored only in bushbabies. A series of studies makes a strong case for a population whose properties vary markedly (Norton & Casagrande 1982, Irvin et al 1986, Norton et al 1988). As an example, if the mean values for spatial frequency cutoff and contrast sensitivity are compared, then K cells occupy a position midway between M and P cells (Norton et al 1988), but within the mean are individual K cells at the extremes. As a group, bushbaby K cells respond at long latencies to orthodromic activation of optic tract axons, to the onset of a visual stimulus, and to antidromic stimulation of the optic radiations. The neurons respond with low rates of maintained discharge and with low peak firing rates. Moreover, few display the kind of spatially opponent-receptive fields that are so commonly found in M and P layers, and most display some change in firing rate to acoustic or tactual stimuli. Responses of this sort can be recorded not only from the robust K layers, located between the two P layers (Norton & Casagrande 1982, Norton et al 1988), but also from the much thinner K layers that occupy positions ventral to M layers (Irvin et al 1986). These averaged findings serve to distinguish the physiological properties of bushbaby K cells from those of either M or P populations, but the variation within the group of K cells far exceeds that seen for the other two LGN populations.

### S-Cone Responses in Simians

***Retinal Circuits for S-Cone Responses*** Recent studies in species other than bushbabies have accented the contribution of cone-specific inputs to certain K layers, specifically those arising from the population of S or blue cones. One of the earliest studies of chromatic selectivity in the LGN of rhesus monkeys found neurons with blue-ON/yellow-OFF responses associated with P layers 3 and 4 (Schiller & Malpeli 1978). Only recently has the retinal circuitry for such a response been firmly established in macaque retina (see Calkins et al 1998). Beginning with a population of S cones (de Monasterio et al 1981; Ahnelt et al 1987, 1990) relayed through a structurally (Mariani 1984, Kouyama & Marshak 1992) and neurochemically distinct (Marshak et al 1990) population of blue bipolar cells, short-wavelength signals arrive in a group of ganglion cells with a unique dendritic field. These blue-ON ganglion cells possess two strata of dendritic branches, one in the inner half and the other in the outer half of the inner plexiform layer (Dacey & Lee 1994, Dacey 1996). By this arrangement, ON and OFF signals

arrive from overlapping parts of the visual field, producing a center-only (type II), blue-ON/yellow-OFF response.

**Retinogeniculate Relay of S-Cone Responses** Ganglion cells that possess blue-ON receptive fields, referred to as small bistratified cells, were first identified by their terminations in the dorsal LGN following injections that overlapped both P and K layers (Rodieck & Watanabe 1993). In a recent study of ganglion cell innervation of rhesus monkey K layers, the terminations of small bistratified cells were traced to the two middle K layers, ventral to P layers 3 and 4 (Calkins & Hendry 1996, Calkins et al 1997, Hendry & Calkins 1999). Small amounts of fluorescent tracers injected into either layer K3 or layer K4 produced retrograde labeling in ganglion cells that, when filled by intracellular injection were found to be small bistratified cells. By contrast, injections confined to the overlying P layers produced retrograde labeling of midget ganglion cells. These anatomical data indicate that very different types of ganglion cells, each of which is known to be chromatically opponent, innervate different layers in the dorsal LGN. The findings provided a reasonable expectation for recording type II, blue-ON responses in layers K3 and K4 of macaque LGN.

**Blue-ON Receptive Fields in Marmoset LGN** The first evidence for such a blue-ON response among neurons of a K layer has been documented for marmosets (*Callithrix jacchus*). In recording from the LGN of this New World monkey, Martin and his colleagues firmly established the presence of blue-ON responses in a region between P layer 3 and M layer 2 (Martin et al 1997, White et al 1998). In displaying a 180° phase difference in response to short-wavelength stimulation and luminance modulation, the response of the neurons closely resembled that recorded from small, bistratified ganglion cells (Dacey & Lee 1994). These and subsequent studies of marmoset LGN document the presence of calbindin-immunoreactive neurons in the region of the blue-ON responses, yet they also reveal the presence of larger, nonimmunoreactive cells in the same regions (Goodchild & Martin 1998). Goodchild & Martin (1998) took advantage of the fact that two neuronal populations are offset slightly, with the unstained neurons dorsal to the immunostained, to suggest that blue-ON responses in marmoset may arise from the unstained group. If that were so, it would lead to the conclusion that K cells, as defined neurochemically, are not the target of axons arising from small, bistratified ganglion cells and that they are not the source of blue-ON responses in the marmoset LGN. This chemical distinction among neurons is not trivial, because studies in Old World and New World monkeys demonstrate only that the immunostained population innervates CO-rich blobs (Hendry & Yoshioka 1994, Hendry & Casagrande 1996, Hendry et al 1997, Yoshioka & Hendry 1999b).

**Blue-ON Responses in Macaque K Layers** Unlike the situation in marmosets, experiments in macaque LGN indicate that blue-ON responses occur among cal-

bindin- and  $\alpha$ CaM II kinase-immunoreactive K cells. The chromatic properties of macaque LGN neurons could be efficiently characterized with white-noise analysis with stimuli that selectively modulated one cone type at a time (Reid & Shapley 1992). As noted previously, type II blue-ON responses can be recorded near the ventral borders of both P layers 3 and 4 (Wiesel & Hubel 1966, Schiller & Malpeli 1978). When electrode penetrations were aimed so as to run for several hundred microns parallel to laminar borders, a sequence of blue-ON cells could be isolated (Reid et al 1997). These penetrations were discovered on histological reconstruction in immunostained sections to enter and remain within a kinase-positive band of cells, either in layer K4 or layer K3. The former was responsive to stimulation of the contralateral eye, the latter to stimulation of the ipsilateral eye. Thus, in macaque LGN, blue-ON responses have been recorded from neurons that are considered K cells by their location, size, and immunoreactivity for  $\alpha$ CaM II kinase. These were the same locations from which small, bistratified ganglion cells could be labeled by the placement of retrogradely transported tracers.

### Heterogeneous Responses in Other Macaque K Layers

Different receptive fields were detected among immunostained K cells in other layers of the macaque LGN (Reid et al 1997). These include broad-band responses in both the ventral-most and dorsal-most pairs of K layers. Among the responses recorded in dorsal K layers are receptive fields much larger than those of M and P neurons at a comparable eccentricity. Such receptive fields are to be expected from a population of LGN neurons innervated by a type of ganglion cell with dendrites that, themselves, span the equivalent of  $15^\circ$  of the visual field (Hendry & Calkins 1999). Rodieck & Watanabe (1993) named this type P giants but they correctly surmised that these neurons innervate the sparsely populated K layers in dorsal-most LGN. The ventral-most K layers, however, are innervated by types of  $\gamma$  and  $\epsilon$  ganglion cells, similar in several ways to the types of cells that send their axons to the superior colliculus and pretectum.

### Conclusions of K Cell Physiology

From these studies of retinal innervation receptive fields come the conclusion that K layers are unlike M and P layers in that a marked heterogeneity exists among their constituent neurons. The K layers appear as an amalgam of cells with different properties and, so it would seem, with different functions. Yet in the LGNs of species that have been examined, heterogeneity can appear either organized or chaotic. In the well-studied prosimian, bushbaby, the two major K layers possess a thoroughly mixed population of receptive-field types. By contrast, in the LGN of macaques and perhaps marmosets a more orderly arrangement exists. As a group, the K cells of macaques display as great a range in physiological properties as do the K cells of bushbabies, but for each pair of K layers in macaques there is a pattern of ganglion cell innervation and of receptive-field physiology that is characteristic and unique. It appears, then, that adaptations to niche have played

a strong role in determining how K layers in a particular species are organized and how the neurons within them come to function in visual behavior.

## GENICULOCORTICAL RELAY BY K CELLS

### Innervation of V1

**Terminations in Superficial Layers** The existence of geniculocortical projections by K cells was anticipated from early studies that showed retrograde shrinkage of all neurons, including Nissl-stained K cells, following focal ablation of V1 (Kaas et al 1972). Yet the precise site of their terminations had been elusive until well-placed injections of horseradish peroxidase in superficial layers of squirrel monkey V1 led to retrograde labeling of neurons intercalated between M and P layers (Fitzpatrick et al 1983, Weber et al 1983). Because the only sites of thalamocortical terminations above layer IV are layer I and the CO-rich blobs of layer III (Itaya et al 1984, Hendrickson 1985), it was possible to conclude that small intercalated neurons (K cells) send their axons to the superficial blobs of V1.

Subsequent studies have identified the neurochemical properties of LGN neurons that innervate macaque V1 above layer IV and found them to be immunoreactive for  $\alpha$ CaM II kinase (Hendry & Yoshioka 1994, Yoshioka & Hendry 1999b). Thus, by the criteria of size, location, and immunoreactivity, the geniculate neurons innervating blobs and layer I of V1 have been identified as K cells. Much the same has been documented for K cell terminations in bushbabies and capuchin monkeys (Hendry & Casagrande 1996). To a certain degree, separate populations innervate layer 1 and the blobs, with neurons in the dorsal-most pair of K layers sending their axons to layer 1 and the others sending a major axonal branch to the blobs. It has been demonstrated, however, by both anterograde (Ding & Casagrande 1998) and retrograde transport experiments (Yoshioka & Hendry 1999b) that neurons in the ventral-most K layers provide at least a minor collateral to layer 1 of V1.

**K Cell Innervation of Specific Blobs** Most studies of K cell terminations have relied on the placement of injections or deposits of tracers that could be confined to superficial layers. A more elegant and satisfying approach has been the application of optical imaging to locate the territory of blobs and of electrophysiological methods to record from neurons in them prior to injecting these blobs with retrogradely transported dyes (Hendry et al 1997). At its most basic, this approach can assign to any blob its location in the ocular dominance domain, because blobs in macaque V1 line up at the centers of ocular dominance columns (Horton & Hubel 1981, Horton 1984, Livingstone & Hubel 1984). Results of such a study demonstrate a pattern consistent with the picture seen from retinogeniculate terminations: Neurons in layers K1, K4, and K6 send their axons to blobs at the centers of contralateral-eye columns, neurons in K3 and K5 to ipsi-

lateral-eye columns, and neurons in K2 to both, with separate tiers of cells innervating contralateral and ipsilateral eye blobs. Furthermore, through injection of separate tracers into adjacent blobs, the degree to which individual K cells send collaterals to two or more blobs can be determined. That degree approaches zero, as there is little evidence for cells retrogradely labeled from separate injections into neighboring blobs. Finally, a basic question of convergence can be addressed by this approach: How many K cells innervate a single blob? An estimate for blobs in the foveal representation is that something on the order of 25–30 K cells send their axons to an individual blob. Because the number of blobs in macaque V1 is estimated to be 3000 (Horton 1984) and the number of K cells in macaque LGN 100,000 (Hendry & Meszler 1999), a figure of 30 K cells per blob is perhaps accurate across all of V1.

An impressive body of evidence indicates there is no such thing as “a blob.” That is to say, a wealth of data suggests that the connections of an individual blob vary with depth and the physiological properties vary from one blob to the next. The latter is a result seen when highly saturated chromatic stimuli are used to examine the response of blob neurons (Ts'o & Gilbert 1988). In some blobs, neurons display a blue/yellow antagonism, in others a red/green. These are innervated by separate populations of K cells in the middle layers of the LGN (Hendry et al 1997). Thus, injections of blue/yellow blobs produce retrograde labeling of  $\alpha$ CaM II kinase-immunostained cells in layers K3 and K4. Such a pattern is consistent with the blue-ON properties recorded from these cells. By contrast, injections of red/green blobs lead to labeling of displaced immunostained K cell populations in P layers 3 and 4. These neurons are as numerous as the cells of layers K3 and K4 proper (Hendry & Meszler 1999) and are otherwise similar to the cells in the K layers in size, morphology, and neurochemical properties. From these data a picture emerges of a further subdivision in the K cell population, with anatomically separate groups innervating different types of blobs. Perhaps the key remaining question is how the displaced K cells in the P layers differ from those of the K layers in retinal innervation and receptive-field properties.

### Innervation of Extrastriate Cortex

Not all geniculocortical neurons in macaque LGN innervate V1. As first demonstrated by Wong-Riley (1976) and subsequently confirmed and expanded on by several groups, a population of relatively large cells scattered throughout the LGN sends its axons to V2 (Benevento & Yoshida 1981, Fries 1981, Bullier & Kennedy 1983, Kennedy & Bullier 1985), V4 (Lysakowski et al 1988, Tanaka et al 1990), and inferotemporal cortex (Hernández-Gonzalaz et al 1994). Because these cells tend to occupy positions between principal layers, they could be thought of as K cells, yet the size of their somata suggests that they are not. Studies of K cells immunostained for  $\alpha$ CaM II kinase or calbindin make the point, however, that the majority population of small neurons is seeded with a minor group of large neurons (Hendry & Yoshioka 1994, Hendry 1995, Yoshioka &

Hendry 1999a). Double-labeling studies demonstrate that it is from this population of large immunostained neurons that LGN projections to extrastriate arise (Hendry 1995, Yoshioka & Hendry 1999b). For the most part the K neurons innervating any of the extrastriate areas studied are few in number, sparse and broadly distributed across the LGN, and present in all K layers. The principal exception to the rule is found in the foveal representation of V2, which is innervated by a densely packed population of K neurons along the caudal and medial margin of the LGN. So numerous are the larger K neurons in this region that the usually sharp borders between M and P layers become blurred in standard histological preparations.

The role of geniculo-extrastriate neurons is most often addressed not for the normal state of vision but in the pathological state of cortical blindness. Bilateral destruction of V1 eliminates visually driven activity in V2 (Schiller & Malpeli 1977, Girard & Bullier 1989) and V4 (Girard et al 1991) but not MT/V5 (Girard et al 1992, Rodman et al 1990), and it leaves humans and macaques with greatly reduced but detectable vision (Covey & Stoerig 1995, Stoerig & Covey 1997). This phenomenon of vision without V1 is called blindsight. Many have suggested that an alternate path from retina to extrastriate cortex that passes through the superior colliculus and pulvinar complex could be the anatomical substrate for blindsight (Brindley et al 1969, Pasik & Pasik 1971). Zeki (1995) has proposed, however, that a direct geniculo-extrastriate path could account for the same phenomenon. That proposal is particularly attractive given the robust innervation of the foveal representation of V2 and the likelihood that this region has been little explored in studies of V1 ablation or cooling because of its proximity to V1. Thus, a relatively rich innervation of V2, with its feed-forward projections to most other extrastriate areas, as well as a much poorer direct geniculocortical innervation of those other areas, could well give the K layers a central role in blindsight.

## K CELLS IN THE FUNCTIONAL ORGANIZATION OF THE VISUAL SYSTEM

### Homology with W Cells

The standard operating model of cat LGN divides neurons into X, Y, and W populations, based on differences in their receptive-field properties (Casagrande & Norton 1992). These make up anatomically separate groups in cat LGN, with characteristic patterns of cortical innervation. As M and P layers are often assigned the roles of Y-like and X-like populations in monkeys, so K cells in bushbabies have been considered the primate equivalent of W cells (Casagrande 1994). The reasons for proposing such a homology are compelling in both anatomical and physiological terms. The cluster of receptive-field and other physiological properties displayed by bushbaby K neurons closely resembles those

displayed by the heterogeneous W cells of cat LGN (Norton & Casagrande 1982, Norton et al 1988, Casagrande 1994). Although not for the monochromatic bush-babies, those properties include the response to S-cone stimulation in W cells of cats and K cells of marmosets and macaques. Cat W cells, clustered in the parvocellular C laminae of the LGN, are innervated by small retinogeniculate axons and send their axons to cytochrome oxidase-rich blobs in superficial V1 (Boyd & Matsubara 1996). Those, of course, are the patterns of innervation displayed by K neurons in the LGNs of at least three primate species. We conclude, then, that the reasons for assigning to K cells in primates the role taken by W cells in cats are well justified.

## K Layers and Parallel Pathways

Models of parallel processing in primate visual system have focused on the innervation and contribution of M and P layers in the LGN. In their strictest form, these models propose that surface features of color and form are carried along the P pathway and divided in V1 into color and form subsystems, corresponding to blobs and interblobs (Livingstone & Hubel 1987, 1988). From there the path leads to ventral areas of cerebral cortex, eventually reaching inferotemporal cortex. The M pathway, by contrast, carries information relevant to motion and spatial aspects of vision through the upper part of layer IV in V1 to eventually reach areas of parietal lobe. Many cogent arguments have been marshaled to dispute the strict bipartite division of labor in the visual system, most of which are centered on what happens in V1 and extrastriate cortex. The existence of a robust K pathway directly challenges these models from a different viewpoint, in that more than two pathways carry visual information from the retina to a first synaptic stage in V1. In a minimally revised scheme, then, the geniculocortical input to blobs can be seen as part of a separate and functionally distinct K pathway rather than as a subdivision of the P pathway.

A major part of the information carried from retina to blob deals specifically with short wavelengths. From photoreceptor to ganglion cell, an S-cone pathway exists in retina that is unlike the midget system for L- and M-cone signals. Studies of K cells in the past couple of years strongly suggest that an S-cone pathway, still segregated from an L- and M-cone-dominated path, is relayed to a distinct collection of CO-rich compartments in V1. As Martin and his colleagues (1997) point out, these data suggest that one contribution to the eventual perception of color has its origins in a K system.

Despite their challenge to a model of primate vision based solely on M and P channels, studies of the K pathway support the concept of parallel visual processing. Existence of K layers in macaque LGN certainly makes more difficult the interpretation of studies that have best contributed to the idea of early convergence among retinogeniculate channels. In these studies, the contribution of M or P layers to visual behavior or to the response properties of cortical neurons was tested by chemically ablating or pharmacologically silencing either one set

of layers or the other. Even the cleanest of the results are contaminated by the presence of a distributed K population. Thus, the discovery that ablation of the dorsal layers in the LGN obliterates color discrimination has been attributed to the obligatory role played by P layers in color vision (Schiller & Logothetis 1990, Schiller et al 1990a,b). Yet these lesions would have also eliminated K cells in and among the P layers, neurons that are responsive to stimulation of S cones. The very strong possibility exists then that the removal of both P and K cells is necessary to eliminate discrimination based on chromatic content.

A similar line can be taken for studies that have compared the response of neurons in V1 before and after the silencing of dorsal and ventral layers in macaque LGN. Neurons in blobs of V1 have their visual response reduced but not eliminated by injections of muscimol designed to silence either M or P layers (Nealey & Maunsell 1994). These data have been interpreted as evidence for an early convergence of M and P signals in blobs. A different interpretation would have the reduction in blob visual response occurring because the input from some but not all K cells is silenced by injections aimed at either M or P layers. By this interpretation, the early cortical components of the M, P, and K pathways could remain segregated to a greater degree than is commonly believed.

## SUMMARY

The existence of a robust and physiologically distinct K pathway introduces a complexity into a field only recently viewed as clean and simple. The presence of a K pathway and its division into three separate populations in the macaque LGN, represented by dorsal, middle, and ventral pairs of K layers, implies a division of labor in the primate visual system not just into two pathways but into at least five and most likely more. Such a division appears as a part of the visual system in all primates that have been examined, from prosimians to humans. By its presence and by what is known of its physiological and anatomical properties, the K pathway permits a reinterpretation of how the various aspects of visual scene are processed. The data suggest something of a compromise among the various models of primate visual system organization, namely that many paths exist for the relay of information from retina to V1 but that these paths may remain anatomically separate and physiologically distinct.

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