

SYMPOSIUM REPORT

Structure and function of parallel pathways in the primate early visual system

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Parallel processing streams in the primate visual system originate from more than a dozen anatomically and functionally distinct types of retinal ganglion cells (RGCs). A central problem in determining how visual information is processed is understanding how each of these RGC types connects to more central structures, including the lateral geniculate nucleus (LGN) of the thalamus and (via the LGN) the primary visual cortex. Nevertheless, the available functional and anatomical evidence linking together specific cell types across these structures is surprisingly indirect. This review evaluates the available evidence and assesses the strength of the many inferences that can be made from these observations. There is strong evidence that parasol RGCs are the provenance of the magnocellular (M) visual pathway and that midget RGCs give rise to the parvocellular (P) pathway. Furthermore, the M and P pathways remain segregated up to the input layer of primary visual cortex. The relationships between the numerous other RGC types and cell types in the LGN remain less certain, and there remains ambiguity about how best to define additional pathways, such as the koniocellular (K) pathway, which probably arise from these other, less common, RGC types.

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The retina of the macaque monkey compacts the visual information received by more than 4 million cone photoreceptors and processed by millions of other retinal neurones into the trains of action potentials of about 1.6 million retinal ganglion cells (RGCs) (Rodieck, 1998). These signals pass through the bottleneck of the optic nerve to connect to a comparable number of neurones in the lateral geniculate nucleus of the thalamus (LGN), which in turn connect to more than 120 million neurones in the primary visual cortex (Van Essen *et al.* 1984; Beaulieu *et al.* 1992). It is generally believed that the RGC signals are optimized to provide a compact representation of the visual world, while the visual cortex extracts and reorganizes this information to convert it into the signals necessary to create a coherent percept (Van Essen *et al.* 1992). The strategies used to create compact visual representations are apparently reflected in the distinct properties of more than a dozen different types of RGCs that project in parallel to the LGN (Dacey *et al.* 2003). Understanding how these parallel signals

are deciphered by the visual cortex requires studies that link the structure and function of each pathway to the functional organization of visual cortex. Understanding how parallel visual pathways are generated and come together provides not only information about vision, but also a framework for understanding the mechanisms by which the brain integrates information from multiple sources to create a unified, coherent percept of the external world.

A central problem in understanding the organization and function of parallel visual pathways is to identify the structural and functional links between the component neurones at successive stages in the path. Our present evidence for such links, although in some cases strong, is surprisingly indirect. Here I focus on the evidence that links together parallel neuronal subsystems from the retina through the primary visual cortex of the macaque monkey.

Methods – their strengths and limitations

The quality of our understanding depends on the quality of the data that can be obtained to support our hypotheses. And, in turn, it also depends on the rigour with which the data are interpreted. Although there is ultimately no absolute certainty, the factors which influence our level

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of certainty are complex and also vary depending on the experience and data available to the observer. Therefore, rather than purveying a dogmatic view of what is known and what is left to be discovered, I prefer here to discuss hypotheses in the context of the available data and the methodological strengths and limitations related to those data. It is therefore useful to begin by considering some of the methods that have been commonly used to link structure to function and to link neural systems across distant structures.

The most common method for identifying the functional properties of individual neurones is extracellular recording with metal electrodes. This method allows unambiguous identification of the recorded neurone type only when the recording is made from within a structurally and functionally uniform population. In such cases, the location of the recording can be marked by making an electrolytic lesion. This method has been most useful in the LGN and V1, where relatively homogeneous populations can be found in distinct layers. When such an organization exists, it is possible to link structure and function across distant structures (e.g. retina, LGN, V1) using standard neuroanatomical tracers. If the tracer injection is confined to particular layers and this results in spatially localized anterograde or retrograde label, then the two functionally characterized compartments can be anatomically linked. It will be seen, however, that there are no locations with absolutely pure populations that are spatially separated. Thus, there is always some ambiguity about these links and our degree of confidence is therefore influenced by the degree of functional and morphological uniformity within an anatomical compartment.

Unlike the LGN and V1, which have clear laminar segregation related to the parallel pathways that emerge from the retina, the retinal ganglion cells themselves are extensively intermingled. There is therefore little hope of linking morphological cell types to functional properties based on extracellular recordings with metal electrodes. Fortunately, *in vitro* retinal preparations are amenable to intracellular recording and in these same preparations the circuits from photoreceptors to RGCs can be maintained. Thus, a good understanding of the links between RGC types and functional properties has emerged from such studies of the primate retina. Linking these RGC cell types across visual structures, from retina to LGN, is, however, more problematic. Making such links is dependent on the ability to make retrograde tracer injections that involve homogeneous populations of LGN neurones. Thus, these links are again only as good as the spatial separation within the LGN, and since the functional properties of RGCs are linked to anatomy based on dendritic morphology (see below), the quality of these links is also dependent on the quality of RGC dendritic filling. Methods for good filling of dendrites have gradually improved (e.g. Dacey *et al.*

2003), but we still rely on a good deal of data using older methods, such as retrograde labelling with HRP, which yield relatively poor cell filling.

Ganglion cell types and function in the primate retina

The earliest studies of retinal ganglion cell types used Golgi staining to distinguish neurones with distinct dendritic morphologies. These observations are exemplified by the work of Polyak (1941). The most common cell type is the midget ganglion cell, which is small and has a compact dendritic arbor (Fig. 1A). A second, relatively common cell type is the parasol cell, which has a much larger cell body and dendritic arbor (Fig. 1A). Based on these early Golgi studies, as well as more recent observations, it is clear that there are also more than a dozen other anatomically distinct cell types (Dacey *et al.* 2003).

The first recordings directly linking visual responses of primate RGC types to parallel pathways used recordings of S potentials in the LGN (Kaplan & Shapley, 1986). S potentials are recorded from RGC axons at their site of LGN termination, allowing the function of RGCs to be linked to both their laminar termination sites in the LGN and to the functional properties of the recipient LGN neurones (which can be recorded at the same time). These recordings clearly identified S potentials in two functionally distinct groups, those with high and low contrast sensitivity, that corresponded to recording sites in the magnocellular (M) and parvocellular (P) layers, respectively, of the LGN (Kaplan & Shapley, 1986). These RGC visual responses were therefore closely related to those of the recipient LGN neurones (see further below).

These two distinct RGC functional properties could then be linked to anatomical cell types in the retina based on the morphology of retinal ganglion cell types retrogradely labelled from tracer injections in the M *versus* P layers of the LGN (Leventhal *et al.* 1981; Perry *et al.* 1984; Rodieck & Watanabe, 1993). Midget ganglion cells comprise about 80% of the population of RGCs that project to the LGN and they project to the P layers. Parasol cells make up about 10% of the RGCs projecting to LGN and they project to M layers. These relationships provide strong evidence that the high contrast sensitive RGCs that project to M layers of the LGN are parasol cells while the less contrast sensitive cells that project to the P layers are midget ganglion cells (see also Michael, 1988). But these are not the only ganglion cell types that project to the LGN (Rodieck & Watanabe, 1993; Dacey *et al.* 2003), so the certainty of these relationships rests on the overwhelming abundance of the corresponding cell types and functional properties that are found in the retina and LGN. With these methods it was not possible (with the same level of certainty) to link cell types to function for the more rare RGC types, such as the P giant and bistratified RGCs (Fig. 1A) that also project to P layers of the LGN (Rodieck & Watanabe, 1993).

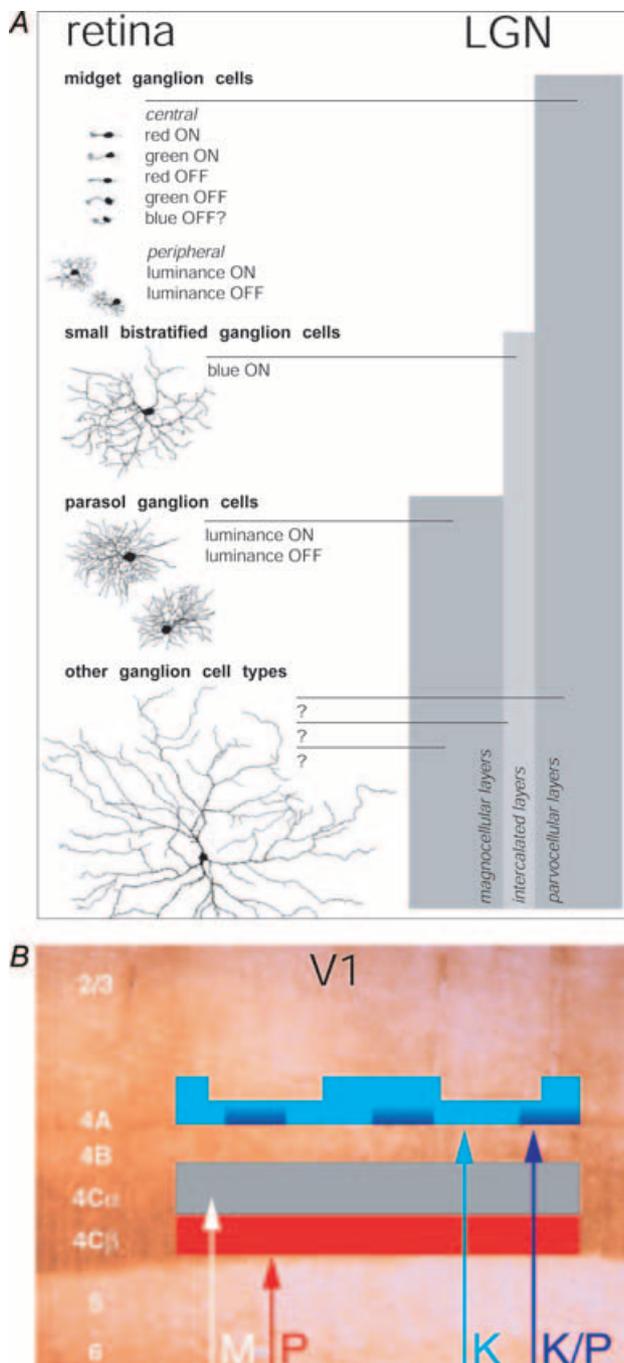


Figure 1. Connections of retinal ganglion cell types to LGN layers (A) and the functional organization of LGN afferents terminating in primary visual cortex (V1) (B)

A, as described in detail in the main text, midget ganglion cells have red–green colour opponency and connect to parvocellular (P) layers of the LGN. Some midget ganglion cells might have ‘blue–OFF’ receptive fields. Parasol ganglion cells carry luminance signals to magnocellular (M) layers of the LGN. Small bistratified ganglion cells have ‘blue–ON’ receptive fields and probably connect to koniocellular (K) neurones located mostly in the intercalated layers of the LGN. In addition there are numerous other ganglion cell types that connect to the LGN, but their functional properties and postsynaptic targets remain unidentified. B, recordings from the afferent axonal arbors of LGN

neurons at their sites of termination in V1 have revealed the functional organization of LGN input to V1. These observations also suggested further functional properties that might be expected for midget and parasol RGCs based on earlier recordings of neurones in M and P layers of the LGN. Neurones recorded in the four most dorsal, P layers usually have small, colour opponent receptive fields, while those in the ventral, M layers have larger, achromatic receptive fields (Wiesel & Hubel, 1966; Derrington *et al.* 1984; Michael, 1988). The great majority of neurones recorded in the P layers have red–green opponency, but cells with blue–yellow opponency and achromatic cells are also encountered. Thus, midget cells were inferred not only to have higher spatial frequency selectivity and lower contrast sensitivity than parasol cells, but also to carry colour-opponent signals. Parasol cells were inferred not only to have lower spatial frequency selectivity and higher contrast sensitivity, but also to be achromatic. A small proportion of the colour opponent cells recorded in the P layers were found to have coextensive ON and OFF regions (type II) rather than the more common centre–surround organization (type I) (Wiesel & Hubel, 1966). When Rodieck & Watanabe (1993) found that bistratified cells, with coextensive dendrites in both ON and OFF layers of the retina, project to P layers of the LGN, they proposed that these are the source of input to type II, LGN P cells.

Direct observations of the relationships between RGC types and functional visual responses were finally made when Dacey (1996) used an *in vitro*, primate retinal preparation, in which intracellular recordings and dye-filling could be combined with measurements of visual responses. These studies have directly revealed that parasol RGCs are achromatic, midget cells are red–green opponent, and bistratified ganglion cells have blue–ON, yellow–OFF colour opponency (Dacey, 1994; Dacey & Lee, 1994; Martin *et al.* 2001; Dacey & Packer, 2003; Dacey *et al.* 2003; Diller *et al.* 2004). EM reconstructions of retinal circuits also suggest the possibility that a small proportion of midget ganglion cells might have blue–OFF, yellow–ON receptive fields (Ahmad *et al.* 2003; Klug *et al.* 2003).

neurons at their sites of termination in V1 have revealed the functional organization of LGN input to V1. Afferents recorded in layer 4C β of V1 have red–green colour opponency and arise from LGN P cells. Afferents recorded in layer 4C α are achromatic and arise from LGN M cells. Afferents recorded in more superficial layers have blue–yellow colour opponency. Blue–OFF afferents are encountered only in layer 4A and might arise from blue–OFF midget ganglion cells via LGN P cells. But it is also possible that they might have some other origin. Blue–ON afferents are encountered on layers 3 and 4A and therefore arise (at least in part) from α CAM kinase/calbindin-expressing LGN K cells. A is from Dacey (2000) and B is from Chatterjee & Callaway (2003).

The LGN – a mismatch of scale between anatomical and functional observations

Laminar functional organization of LGN. As introduced above, the primate LGN is divided into six layers. The two most ventral layers (layers 1–2) receive input primarily from parasol RGCs (Leventhal *et al.* 1981; Perry *et al.* 1984) and contain cells with large, achromatic, highly contrast sensitive receptive fields (Shapley & Perry, 1986), while the four dorsal layers (layers 3–6) receive input primarily from midget ganglion cells (Leventhal *et al.* 1981; Perry *et al.* 1984; Rodieck & Watanabe, 1993) and contain cells with smaller, colour-opponent, poorly contrast sensitive receptive fields (Shapley & Perry, 1986). These relationships can be relatively easily identified because they exist at the spatial resolution with which extracellular single unit recordings can be localized. Nevertheless, there are smaller populations of neurones encountered in both the M and P layers whose functional properties differ from the most commonly encountered varieties.

Although, these observations successfully linked structure to function from retina to LGN, the spatial resolution of both anatomical tracer injections and single-unit recordings in the LGN have been limited to the spatial scale of these six layers. But it is clear from the diversity of RGC cell types that are retrogradely labelled from the LGN (Rodieck & Watanabe, 1993; Dacey *et al.* 2003), as well as from the diversity of functional properties found in the LGN (see above), that, beyond midget and parasol cells, the laminar organization of the LGN is insufficient to allow unambiguous links between structure and function, or across multiple stations in the visual pathways. Therefore, at the level of the LGN, additional observations have aided in the resolution of cell types and function on a finer scale. The most useful of these have been staining for neurochemical markers and labelling of afferent axons to reveal anatomical distinctions of the arbors within V1.

Anatomical and neurochemical heterogeneity. The most useful markers for distinguishing heterogeneity within the primate LGN are parvalbumin, α CAM kinase, and calbindin. At a cellular level, calbindin and α CAM kinase are nearly completely overlapping, while these markers are complementary to parvalbumin (Hendry & Yoshioka, 1994). Individual LGN neurones express either parvalbumin or α CAM kinase/calbindin, but not both. The great majority of neurones in both the M and P layers of the LGN express parvalbumin. Staining for α CAM kinase/calbindin reveals a small subpopulation of LGN neurones concentrated in the intercalated zones between the main M and P layers (Fig. 1A), and also scattered within these layers.

The possible relevance of these markers to anatomy and function was highlighted by the observation that retrograde tracers injected into superficial layers of V1 label only α CAM kinase/calbindin positive cells (Hendry & Yoshioka, 1994). Injections in deeper cortical layers also labelled parvalbumin positive neurones. Thus, these neurochemically distinct cell types also differ in their laminar patterns of projection to V1. This suggests that they should also differ from parvalbumin cells both functionally and in their sources of retinal input.

These observations vitalized investigation of a third, koniocellular (K) pathway, that had been described in new world monkeys, but relatively ignored in macaque monkeys (Casagrande, 1994). But they did not reveal the functional properties of the component LGN neurones or the RGC types that provide their input, and although they revealed that at least some of the α CAM kinase/calbindin neurones project to superficial layers (Hendry & Yoshioka, 1994), others might well project to other layers. Thus, separate definitions of the K pathway have informally emerged at different levels in the visual pathway. At the level of V1, the K pathway has come to be defined as the pathway that terminates in superficial layers, while at the level of the LGN the definition tends to be cells that express α CAM kinase/calbindin. There is not necessarily reciprocity, however, between these definitions. For example, recently a population of LGN cells has been revealed which expresses α CAM kinase and projects to area MT, but not to V1 (Sincich *et al.* 2004) and the possibility that some α CAM kinase/calbindin expressing LGN cells might project to deeper layers in V1 (4A, 4C, or 6) or to other cortical areas has not been ruled out.

At the level of the retina, the definition of the K pathway is even less clear. If all parvalbumin expressing LGN cells were to receive input from either midget cells (in the P layers) or from parasol cells (in the M layers), then the K pathway could be defined as RGCs that connect to α CAM kinase/calbindin cells in the LGN, and the RGC types giving rise to this pathway might then be defined as all LGN-projecting cells that are not midgets or parasols. However, based on the available evidence, it remains plausible that some parvalbumin-expressing LGN neurones receive input from RGCs other than midgets or parasols. Thus, the present definitions may lack congruency across levels.

It does seem likely, however, that bistratified RGCs give rise to at least part of the K pathway, as defined by both α CAM kinase/calbindin expression and projections to superficial layers of V1. There are several observations that provide strong, indirect evidence for this relationship. Small and large bistratified cells have blue-ON receptive fields (Dacey & Lee, 1994; Dacey *et al.* 2003) and small diameter axons. The RGC axons that terminate in the

LGN intercalated zones are also of small diameter (Conley & Fitzpatrick, 1989), and in the new world (marmoset) monkey, cells with blue–ON receptive fields tend to be encountered most frequently in these same zones (but are also found elsewhere) (White *et al.* 1998). Finally, the LGN afferents that terminate in layer 3 of macaque V1 have blue–ON receptive fields (Chatterjee & Callaway, 2003) – these same cells must be LGN K cells that express α CAM kinase/calbindin (Hendry & Yoshioka, 1994).

Linking retinal ganglion cell types to primary visual cortex via the LGN

Connectional heterogeneity. Another method that has revealed heterogeneity of LGN cell types is reconstruction of their individual axonal arbors within V1. This method not only reveals diversity in the LGN population, but also provides information about the likely connectivity of each cell type within V1. Recently, these differences were also exploited to identify the functional properties of LGN cells with axons terminating in distinct locations within V1 (Chatterjee & Callaway, 2003). It was possible to record from the small electrical signals in the terminal arbors of LGN cells by inactivating, with muscimol, the larger ‘background’ spikes of V1 cortical neurones (Chapman *et al.* 1991). The functionally disparate LGN populations proved to have sufficiently uniform axonal arbors within cell types and segregation across cell types to allow cell types to be correlated with function based on extracellular recordings and localization of electrolytic lesions (Fig. 1B) (Chatterjee & Callaway, 2003).

There is very strong evidence that the M and P pathways, which originate with parasol and midget RGCs, respectively, remain segregated at the level of their afferent terminations in V1. Injections of anterograde tracer into the LGN P layers results in label in layers 4A and 4C β of V1, while injections into the M layers results in label in layer 4C α (Hendrickson *et al.* 1978). These observations imply that the M pathway connects to layer 4C α of V1, while the P pathway connects to layer 4C β and possibly also to layer 4A; reconstructions of individual axons suggest that these inputs come from separate populations (Blasdel & Lund, 1983) (see below). But these labelling studies do not rule out the possibility that other types of LGN cells receiving input from other RGC types might connect to these same layers of V1. Apparently these tracer injections lacked the sensitivity required to reveal additional LGN input to layer 1 and cytochrome oxidase (CO) blobs in layer 2/3 (Livingstone & Hubel, 1982; Blasdel & Lund, 1983; Hendry & Yoshioka, 1994).

Reconstructions of individual LGN axonal arbors within V1 reveal five common laminar patterns. Cells with the characteristic response properties of the P pathway terminate in layer 4C β , while those with functional

characteristics of M cells terminate in layer 4C α (Blasdel & Lund, 1983; Freund *et al.* 1989). Since these samples are small, it should again be pointed out that these observations do not preclude smaller populations with other functional properties that might also terminate in these same zones. Three additional patterns of afferent arborization have been reconstructed and these terminate in either layer 4A, layer 3 blobs, or layer 1 (Blasdel & Lund, 1983; Ding & Casagrande, 1997). One afferent axon terminating in layer 1 was functionally characterized and had a blue–ON receptive field (Blasdel & Lund, 1983).

To identify the visual responses of LGN afferents terminating in layers 3 and 4A, and to better determine whether there might be previously undetected heterogeneity in the inputs to layers 4C α and 4C β , Chatterjee & Callaway (2003) recorded from these afferents at their sites of arborization within V1 (see above). Functional characterizations were obtained for 46 afferents recorded in layer 4C β , 98 in layer 4C α , and 77 in layers 3 and 4A (a summary of these results is found in Fig. 1B). The sizes of these samples are far larger than the small handful (less than 10) of axons previously characterized functionally during intracellular recording and labelling of axons (Blasdel & Lund, 1983; Freund *et al.* 1989). Afferents recorded in layer 4C β had only red–green colour opponency – none were blue–yellow opponent (Chatterjee & Callaway, 2003). This suggests that only red–green opponent midget RGCs connect through the LGN to layer 4C β of V1. All of the afferents recorded in layer 4C α were achromatic and had properties similar to the parasol RGC recipient LGN M cells. Since nearly 100 LGN afferents were functionally characterized in layer 4C α , other cell types connecting to this region are probably nonexistent, extremely rare, or arborize too sparsely to detect.

All 77 of the afferents recorded in superficial layers of V1 (layers 3 and 4A) had blue–yellow colour opponent receptive fields. Furthermore, blue–ON and blue–OFF afferent recordings were spatially segregated. All of the afferents recorded in layer 3 (presumably in blobs) had blue–ON receptive fields suggesting that their functional properties arise from bistratified RGCs (Dacey & Lee, 1994; Dacey *et al.* 2003). (An interesting possibility is that the small bistratified cells are the source of input to LGN cells terminating in layer 3, while large bistratified cells connect to blue–ON cells with more widespread axons in layer 1; Blasdel & Lund, 1983.) Perhaps the most surprising finding of Chatterjee & Callaway (2003), was the relatively frequent encounter of blue–OFF cells (17 cells). These had only rarely been encountered in previous recordings from the LGN (Derrington *et al.* 1984; Valberg *et al.* 1986). All of these afferents were recorded in layer 4A, suggesting that they correspond to afferents with dense, but tightly circumscribed arbors restricted to layer 4A (Blasdel & Lund, 1983). The most likely candidate RGC type contributing to the blue–OFF pathway to layer

4A is the subset of midget RGCs that connect to S-cones via OFF midget bipolars (Ahmad *et al.* 2003; Klug *et al.* 2003), but this will need to be verified with other methods. The receptive field sizes of melanopsin-expressing RGCs that also have blue-OFF responses (Dacey *et al.* 2005) are probably too large to correspond to the afferent recordings made in layer 4A (unpublished observations) and the two populations also differ markedly in the relative strength of S cone *versus* L + M cone responses (Chatterjee & Callaway, 2003; Dacey *et al.* 2005).

Finally, it should be noted that the number of LGN cell types identified with unique patterns of axonal arborization within V1 (5 types, see above) is far smaller than the number of RGC types that project to the LGN (about a dozen; Dacey *et al.* 2003). There are several possible explanations for this discrepancy. It is clear that at least part of the difference can be explained by the observation that some LGN cell types do not project to V1, but instead project to extrastriate cortical areas (Sincich *et al.* 2004). Another possibility is that one of the basic assumptions inherent in most interpretations of these pathways does not always hold – that is the assumption that each RGC type connects one-to-one to a unique LGN cell type. There are at present no strong data to rule out the possibility that some LGN cell types might receive input from more than one type of RGC or that some RGC types might connect broadly to many LGN cell types and have only a modulatory influence. Lastly, there could be some LGN cell types that project to V1 and have not been uniquely identified.

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