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Parallel pathways in macaque monkey striate cortex: Anatomically defined columns in layer III

(biocytin/cytochrome oxidase blobs/horseradish peroxidase/lateral geniculate nucleus/vision)

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ABSTRACT **Visual information reaching striate cortex comes from parallel pathways, and the information is organized, or processed, by the layers and columns of striate cortex. To better understand how this is accomplished anatomically, we asked whether parallel pathways originating in the lateral geniculate nucleus (LGN), and terminating separately in layer IV, remain separate in layer III of macaque monkeys. Layer III is of interest since it may play a special role in color and form vision but not in analysis of visual motion. The chief finding was that cells in "blobs" of layer III that stain densely for cytochrome oxidase receive indirect input, via layer IVC, from both LGN magnocellular (M) and parvocellular (P) cells. This is important because the P and M pathways may represent color/form and motion-processing channels, respectively. Interblob cells receive indirect input, via layers IVC and IVA, from the LGN P cells. Also, as suggested by others, our data demonstrate that layer III can be subdivided. The bottom tier, layer IIIB, receives direct projections from all cortical layers. Output from layer IIIB appears to remain intrinsic to striate cortex. In contrast, the top tier, layer IIIA, receives projections from layer IIIB as well as from layers IVA, IVB (blobs only), and V, but it receives no direct projections from LGN recipient layers IVC and VI. Unlike layer IIIB, the output of layer IIIA reaches extrastriate areas. Thus, impulses arriving from parallel LGN pathways may be recombined through serial stages in striate cortex to produce a set of parallel pathways that are qualitatively different from the original LGN set.**

The early stages of the primate visual system are characterized by the creation and segregation of functionally distinct pathways that work independently of, but parallel to, one another to provide a uniform perception of visual space (see refs. 1–6). In macaque monkey, three visual pathways have been described that arise from morphologically distinct cells located in the retina. These cells project to different layers of the lateral geniculate nucleus (LGN): the magnocellular (M), the parvocellular (P), and the interlaminar or S layers (7). It has been proposed that segregation of visual pathways in the LGN is preserved at higher levels in the visual system in separate layers and/or columns of primary visual cortex (striate cortex) and in separate hierarchies of extrastriate visual areas. According to one view (3), visual signals from the M LGN cells project via striate cortical layer IVCα to layer IVB and then out of the striate cortex. This pathway eventually terminates in areas within superior temporal and parietal cortex that are concerned with visual motion and orientation in space, respectively. Visual signals from the P pathway project to layer IVCβ where they are split and conveyed to layer II/III, either to the cytochrome oxidase-rich patches known as blobs, or to the lighter-staining spaces between these zones, the interblobs (8). These P pathways eventually contribute to color and form vision, respectively, in areas in occipital and inferior temporal cortex. The third visual pathway, which projects directly to the cytochrome oxidase blobs (9), is usually ignored because the physiology of the small LGN cells has not been examined in macaque monkeys. [Studies in another primate, *Galago crassicaudatus*, suggest that the physiological properties of small LGN cells are similar to cat W cells (10–12).]

The prevailing view suggests that pathways originating in the LGN interact minimally within striate cortex. This view is difficult to reconcile with evidence from anatomical studies showing the rich interconnections of the cortical layers (13, 14) and with an overall recognition that some interaction between parallel processing streams must occur. In fact, Livingstone and Hubel's (15) physiological recordings in the cytochrome oxidase blobs suggested that they may also receive M pathway input. One reason these data were ignored could be that anatomical investigations were never conducted in an appropriate manner to examine the type of segregation implied by earlier studies. For example, to our knowledge, there are no anatomical data demonstrating that cells in cytochrome oxidase blobs and interblobs in cortical layer III receive exclusive or even dominant projections from LGN P cells. Our goal was to examine the anatomical substrate for segregation and potential integration within striate cortex. Specifically, our study addressed the degree of interaction between LGN pathways in cortex beginning with layer III. Our results show that cells in cytochrome oxidase blobs and interblobs receive projections from the LGN P pathway but that cells in cytochrome oxidase blobs also receive projections from the M pathway. In addition, our data suggest that the LGN pathways are processed through serial stages in layer III, first in layer IIIB and then in layer IIIA, before being sent beyond striate cortex (Fig. 1).

MATERIALS AND METHODS

The interlaminar connections of the blob and interblob zones were examined by making multiple small injections of horseradish peroxidase (HRP) or biocytin into the striate cortex of three adult monkeys (*Macaca fascicularis*). Surgical and histological protocols are described in detail elsewhere (16). Under sterile conditions, either 10% HRP or 5% biocytin was injected with glass pipettes (tip, 10 μm). Between 20 and 30 iontophoretic injections were made into each hemisphere. The subjects survived for 18–24 hr before being reanesthetized and perfused with lactated Ringer's solution and then with either 4% paraformaldehyde alone (biocytin cases) or with 0.5% glutaraldehyde added (HRP cases) in 0.1 M

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Abbreviations: LGN, dorsal lateral geniculate nucleus; M, magnocellular; P, parvocellular; HRP, horseradish peroxidase.

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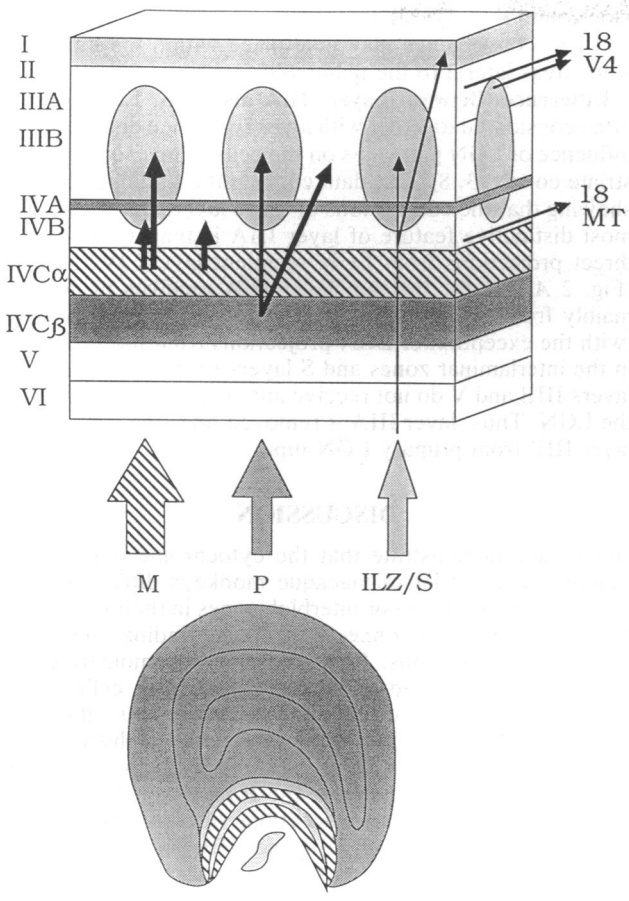

FIG. 1. Schematic diagram (*Upper*) of three parallel pathways from the layers of LGN (*Lower*) to and through striate cortex of macaque monkeys. The M pathway (thick arrows) projects via layer IVCα to layer IVB, which in turn sends information to hierarchies of extrastriate visual areas concerned with aspects of "where" an object is in visual space (2). The M pathway also projects via layer IVCα to the cytochrome oxidase blobs in layer IIIB. The P pathway (medium arrows) projects via layer IVCβ to both cytochrome oxidase blobs and interblobs in layer IIIB. The cells in the LGN interlaminar zones (ILZ) and S layers making up the ILZ/S pathway (thin arrows) project directly to the blobs in layer IIIB and to cortical layer I (9). Cells in cytochrome oxidase blobs and interblobs in layer IIIB send input to layer IIIA blobs and interblobs, respectively, which project this information into hierarchies of extrastriate areas concerned with object identification (2). MT, middle temporal visual area; V4, visual area 4; 18, visual area 18. See text for details.

phosphate buffer. The brains were sectioned frozen at 40 μm. Alternate sections were histochemically treated to visualize peroxidase or biocytin (17–19) and were stained for cytochrome oxidase (20).

Low-power reconstructions of all injection sites and blob/interblob borders were made with a projection scope. Only injections centered within a blob or an interblob and centered within layer IIIA or IIIB were reconstructed. Of the 124 injections that resulted in either a recognizable injection site or adequate retrograde label, only 33 fulfilled the latter criteria and were analyzed further. The laminar distribution of labeled cells resulting from these injections was plotted through serial sections at higher magnification (×16 to ×25) using a camera lucida. The number and density of labeled cells within individual layers were noted qualitatively for all injections. Quantitative estimates were not attempted because of complications involved in comparing the density of labeled cells where layers vary in cell density and where injection sites vary in size and density of label. It is important to emphasize that analysis focused on consistent differences in the patterns of projections and not on the exact number of cells that were labeled; in our material, even injections of comparable size and location showed variability in the density of retrogradely labeled cells. Also, although our material contained clear evidence of anterogradely labeled axons, many of which could be seen running horizontally in layer III, we confined the present analysis to the pattern of retrogradely labeled cell bodies. Our analysis of the anterograde pattern of connections will appear as a separate communication.

Eighteen injection sites restricted to cytochrome oxidase blobs were reconstructed; 10 were in layer IIIA and the remainder were in layer IIIB. Fifteen interblob injection sites, 9 in layer IIIA and the remainder in layer IIIB, were reconstructed. Laminar distinctions (with the exception of layer V) were made according to Lund and Boothe (14). All injections analyzed were restricted to the dorsal bank of striate cortex, which represents 0°–5° of visual space (21).

There are two potential methodological concerns. First, with multiple injection sites it is possible that injections could overlap. This is unlikely since all injections were separated by 2 mm or more. Second, HRP is known to be taken up both actively and passively (damage) by dendrites and axons. Thus, we cannot rule out the possibility that cells could have been filled via cut axons or apical dendrites. We think that such passive filling of cells did not make a significant contribution to our HRP results because (*i*) injections with biocytin produced identical results, and previous studies have shown that biocytin is not taken up by fibers of passage (19); (*ii*) patterns of labeled cells differed between blob and interblob injections; (*iii*) no evidence exists to suggest that apical dendritic patterns differ with cytochrome oxidase density in cortex.

RESULTS

Blobs and Interblobs: Layer IIIA. The connections of layer IIIA vary between blob and interblob zones. In layer IIIA, blob cells receive a substantial number of projections from layer IVB. These projections are absent from the layer IIIA interblobs (compare Fig. 2 *A* and *B*). The difference suggests that layer IIIA blob cells may be dominated more by the LGN M pathway than are interblob cells, since layer IVB receives its major projections indirectly from LGN M cells via layer IVCα. In addition, a broader zone of layer IVB, well outside the main column of label, is consistently labeled after injections into layer IIIA blob zones, but not after injections of comparable size into interblob zones. Thus, there appears to be greater convergence of projections from layer IVB onto layer IIIA blob cells than onto interblob cells (Fig. 2*A*). By contrast, all other projections to layer III blob or interblob zones appear confined to areas directly beneath the injection site. A final difference between layer IIIA blob and interblob zones is in the distribution of projections from layer V. Retrograde label is clearly more confined to the top tier of layer V (VA) after injections into the interblob zones than it is after injections into the blob zones.

Blobs and Interblobs: Layer IIIB. Injections centered within blob zones in layer IIIB produce retrogradely labeled cells in all layers except layers II and I (Figs. 2*C* and 3). This observation implies that cells within blob zones may receive impulses from both M and P pathways after only one synapse in layer IVC. Furthermore, although both layers IVCα and IVCβ contain labeled cells, the majority of labeled cells are found in layers IVCα (i.e., the M pathway), V, and VI, and not in layer IVCβ (i.e., the P pathway) as originally predicted (8, 22). The fewest labeled cells are located in layers IIIA, IVA, and IVB.

Injections centered in interblob zones within layer IIIB label a different, more restricted, subset of cells (Fig. 2*D*).

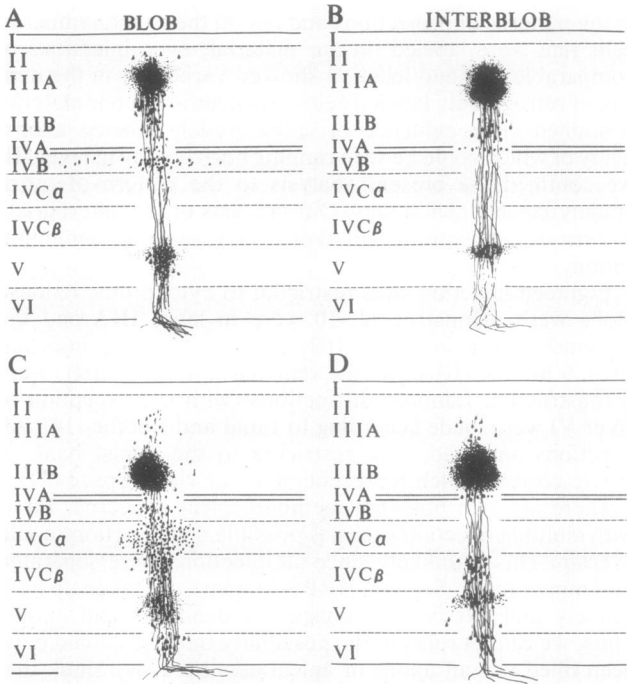

FIG. 2. Examples of reconstructions of injection sites in cytochrome oxidase blobs and interblobs in cortical layer III of macaque monkey and the resulting pattern of labeled cells and axons. (*A* and *B*) Injections in a blob and an interblob in layer IIIA are shown. (*C* and *D*) Injections in a blob and an interblob in layer IIIB are shown. Roman numerals indicate cortical layers according to Brodmann's nomenclature (23). (Bars = 250 μm.)

The majority of labeled cells are found in layer IVCβ and in the upper tier of layer V (VA). In addition, labeled cells are seen in layer IVA, both tiers of layer VI, with a few cells also labeled in layer IIIA. This pattern suggests that, in contrast to blob cells, interblob cells could be under more influence from the P cell pathway. In addition, the limited lateral spread of connections within interblob columns as opposed to the blob columns in layer IIIB (compare Fig. 2 *C* and *D*) indicates that less convergence may take place within the interblobs from areas lateral to the main column.

Differences Between Layers IIIA and IIIB. Layer IIIA is often considered together with layer IIIB when discussing the influence of LGN pathways on the cells of macaque monkey striate cortex (3, 8). Our data confirm the findings of others showing that the connections of these layers differ (24). The most distinctive feature of layer IIIA is that it receives no direct projections from LGN recipient layers IVC and VI (Fig. 2 *A* and *B*). Instead, layer IIIA receives projections mainly from layers IIIB and V. This is important because [with the exception of LGN projections from the small cells in the interlaminar zones and S layers to the IIIB blobs (9)] layers IIIB and V do not receive any direct projections from the LGN. Thus, layer IIIA is removed one step further than layer IIIB from primary LGN input.

DISCUSSION

Our results demonstrate that the cytochrome oxidase-rich "blobs" in layer III of macaque monkeys differ from the cytochrome oxidase-poor interblob zones in their patterns of interlaminar cortical connections. These findings are significant for three reasons. First, the results demonstrate that cells within blobs can be distinguished from cells within interblobs based on differences in projections (direct or indirect via layer IVC) from the three LGN pathways; blob cells receive projections from all three pathways; interblob cells receive projections from the P pathway. Second, the results demonstrate that connections of blob and interblob cells are different in layers IIIA and IIIB. The differences in these patterns of connections suggest that signals from the separate LGN pathways may be processed serially first in layer IIIB and then in layer IIIA before being transmitted to extrastriate areas. Finally, our data on the intrinsic cortical connections of cells in both the blob and interblob zones support the view that signals from separate LGN pathways could be combined in several ways before these signals leave striate cortex. These conclusions are considered in more detail below.

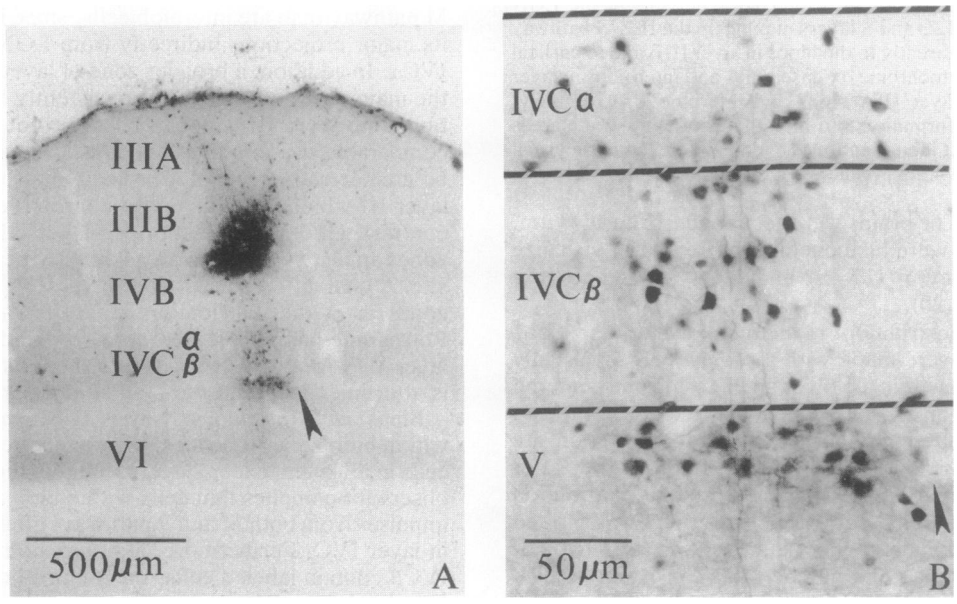

FIG. 3. Photomicrograph of a biocytin injection site in layer IIIB of a blob zone (as determined by the adjacent section stained for cytochrome oxidase and resulting retrogradely labeled cells. All layers of cortex except layers II and I contain labeled cells (*A*). At higher magnification, labeled cells can be seen in both layers of LGN-recipient layer IVC (*B*). Arrowheads indicate a common blood vessel. This injection represents one of our largest injections and was selected because both injection and label are visible in the same section; labeled cells resulting from the majority of our injections were distributed over several sections. (*A*, bar = 500 μm; *B*, bar = 50 μm.)

Blobs and Interblobs. Hubel and Livingstone (25) originally proposed the idea that the LGN P and M cell pathways remain segregated through the striate cortex and enter separate hierarchies of visual cortical areas subserving form/color and motion information, respectively. This model, based mainly on physiological data, suggests that information about color and form conveyed by the P channel is split into two channels that are represented by cells in the blob zones (color) and by cells in the interblob zones (form) before leaving the striate cortex (2).

The problem with this model is that unless one is able to test the influence of pathways independently, determining the influence of a LGN pathway on a cortical cell's response depends critically on each LGN pathway having a unique signature. With the possible exception of the chromatic signature of the P pathway, such signatures do not exist.

Our anatomical results clearly show that blob cells, as a group, receive projections from both P and M pathways. In one sense, this result should not come as a surprise since an earlier study (24) showed that, after some injections into layer III of striate cortex in macaque monkeys, both divisions of layer IVC contained retrogradely labeled cells. However, the issue of where these injections were located with respect to the cytochrome oxidase blob zones was not addressed. Data in a 2-deoxyglucose study suggested that the M pathway makes a small contribution to blob cell activity (22). However, the argument presented in that study insisted that blob cells are dominated by the P pathway. Our anatomical findings suggest the opposite—namely, that the M pathway could contribute more substantially to blob cell activity (Table 1). More importantly, we would argue that there is more convergence of M and P channel input to blob zones than was originally proposed (Fig. 1). Obviously, as mentioned earlier, a good test of signal convergence at the single cell level would require measuring the contribution of each pathway separately. In fact, more than a decade ago, Malpeli *et al.* (27) used lidocaine and cobalt to block appropriate areas of the M and P LGN layers to demonstrate that over one-third of the cells in macaque monkey striate cortex are influenced by both pathways. Although the latter study did not directly address the issue of influence on neurons in blobs, a recent preliminary report in which similar techniques were used finds that individual blob cells can be driven by both P and M cells (28). Moreover, studies using acrylamide to selectively eliminate the P pathway are consistent with this hypothesis (29).

The patterns of connections to blobs that we report here are not unique to macaque monkeys. Identical patterns of connections to the cytochrome oxidase blobs have been found in other primates. We have examined this issue directly in a nocturnal prosimian, *G. crassicaudatus* (30), and in a diurnal New World simian, squirrel monkey (31), and found that blob zones in layer IIIB receive projections from both M and P pathways based on labeling patterns in layer IV. In each species examined, the M pathway projections appeared to be stronger. Thus, data from several primate species suggest that if any LGN cell pathway has a dominant contribution to blob cell receptive field physiology, the connections would favor the M LGN cell pathway. However, if density of the labeled cells is used as an index to determine which cells might have the greatest effect on blobs, the anatomy in all of these primates favors the cells of infragranular layers V and VI. In addition, although not emphasized here, it is clear that the blob zones in macaque monkeys and other primates receive their only direct LGN projections from the third pathway originating in the LGN S layers, LGN interlaminar zone cells, or their LGN equivalent (9, 32–35).

Table 1. Summary of inputs to striate cortex layer III

	Layer IIIA		Layer IIIB	
Layer	Blobs	Interblobs	Blobs	Interblobs
IIIA	*	*	+	+
IIIB	+	+	*	*
IVA	+	+	+	+
IVB	+	−	+	−
IVCα	−	−	+	−
IVCβ	−	−	+	+
VA	+	+	+	+
VB	+	−	+	−
VIA	−	−	+	+
VIB	−	−	+	+

Roman numerals refer to layers of cortex as defined by Brodmann (23). Sublayer VA is defined as the top half of layer V. Lund (26) defines this region as the top one-third of the layer. *, Injection site; +, presence of labeled cells; −, absence of labeled cells.

Taken together, three conclusions can be drawn from these results. First, the pattern of intrinsic interlaminar connections of blob zones is a conserved property in primate evolution. All primates, including humans, are likely to have similar connections. Second, since identical blob zone connections can be demonstrated in three very distantly related primate species, cells in blob zones must play a basic role in vision in all primates regardless of niche. Finally, the anatomy of blob zone connections suggests that they could be zones of pathway integration, not pathway segregation.

Using similar logic, one could argue from our data that interblob zones in macaque monkeys are dominated more by the P pathway. Even in the case of the interblob zones, comparisons between the densities of labeled cells indicate that the infragranular layers provide more input than LGN recipient layer IVCβ. In fact, as reported for blob cells, investigation of the response properties of individual interblob cells following the block of activity in appropriate patches of either M or P LGN layers has shown that some interblob cells in macaque monkeys are driven by either the M or the P pathway (28). These observations also suggest that considerable integration of pathway information also takes place within interblob zones.

Differences Between Layers IIIA and IIIB. Our data add a new dimension to previous data demonstrating that the two divisions of layer III (IIIA and IIIB) in macaque monkeys have different interlaminar connections (24, 36). In this report, we show that interlaminar connectional differences between blob and interblob zones can be found in both layers IIIA and IIIB. This finding suggests that cytochrome oxidase blob and interblob zones may define columns that function as separate units or modules through several cortical layers. Moreover, the fact that the blob zones in layer IIIA receive strong projections from M dominated layer IVB, while interblob zones receive no projections from this sublayer, reinforces the idea that blob zones may be more under the influence of the M pathway than interblob zones. We also confirm the finding that IIIA cells receive no projections from the main target layers of the LGN—namely, layers IV and VI. Thus, cells in layer IIIA receive LGN signals after at least an additional processing step. Thus, layer IIIA can be considered at a higher level than layer IIIB in the information processing hierarchy. The importance of this finding, however, can only be appreciated by noting an important difference in the output of layers IIIA and IIIB. An examination of the relevant anatomical literature reveals that within layer III, only layer IIIA cells send axons beyond striate cortex (37–39). Thus, layer IIIB cells appear to serve as interneuronal integration units within their cortical column.

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