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HOW PARALLEL ARE THE PRIMATE VISUAL PATHWAYS?

W. H. Merigan[1] *and J. H. R. Maunsell*[2,3]

Center for Visual Science and Departments of [1]Ophthalmology and [2]Physiology, University of Rochester, Rochester, New York 14642

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INTRODUCTION

The visual system, like all sensory systems, contains parallel pathways (see Stone 1983). Recently, much emphasis has been placed on the relationship between two subcortical and two cortical pathways. It has been suggested that the cortical and subcortical pathways are continuous, so that distinct channels of information that arise in the retina remain segregated up to the highest levels of visual cortex. According to this view, the visual system comprises two largely independent subsystems that mediate different classes of visual behaviors. In this paper, we evaluate this proposal, which has far-reaching implications for our understanding of the functional organization of the visual system.

The subcortical projection from the retina to cerebral cortex is strongly dominated by the two pathways (M and P pathways) that are relayed by the magnocellular and parvocellular subdivisions of the lateral geniculate nucleus (LGN) (see Shapley & Perry 1986). The importance of these pathways is demonstrated by the fact that they include about 90% of the axons that leave the retinas (Silveira & Perry 1991) and that little vision survives when both pathways are destroyed (Schiller et al 1990a). The P and M pathways maintain their sharp anatomical segregation through the termination of the LGN projection in layer 4C of V1 (striate cortex).

The complex network of connections in primate extrastriate visual cor-

[3] Present address: Division of Neuroscience, Baylor College of Medicine, Houston, Texas 77030.

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tex has also been described as dominated by two pathways. One pathway includes areas in parietal cortex and is thought to be important for assessing spatial relationships and object motion; the other includes visual areas in temporal cortex and is thought to be more involved in visual identification of colors, patterns, or objects (Ungerleider & Mishkin 1982). Differences between the parietal and temporal pathways can be seen in lesion-induced deficits, in neuronal response properties, and in anatomical connections (see Desimone & Ungerleider 1989; DeYoe et al 1990; Mishkin et al 1983; Ungerleider & Mishkin 1982; Van Essen & Maunsell 1983).

Cumulative anatomical, physiological, and behavioral evidence has suggested a relationship between these subcortical and cortical pathways, eventually leading to explicit proposals of a direct correspondence between them (Livingstone & Hubel 1987; Maunsell 1987). The major components of the parallel pathways involved in this proposal and their interconnections are shown schematically in Figure 1. It has been suggested that the contributions of the M and P pathways remain largely segregated in visual cortex, and each connects to one of the cortical pathways, with the M pathway and the parietal pathway forming one subsystem, and the P pathway and the temporal pathway forming the other. It has further been proposed that several specific visual functions, such as motion, stereopsis, and figure/ground discrimination, could each be attributed to a specific subsystem.

The notion of parallel visual subsystems has been broadly disseminated and popularized (e.g. Kandel et al 1991; Livingstone 1988), and has quickly become widely accepted, owing in part to its great explanatory power and its appealing simplicity. The idea is consistent with an extensive collection of observations, which has been reviewed in detail many times (Desimone & Ungerleider 1989; DeYoe & Van Essen 1988; Felleman & Van Essen 1991; Goodale & Milner 1992; Kaas & Garraghty 1991; Livingstone & Hubel 1987, 1988; Martin 1988; Maunsell 1987; Maunsell & Newsome 1987; Van Essen et al 1992; Zrenner et al 1990). However, a growing number of reports has called the idea of visual subsystems into question. The cortical pathways show appreciable anatomical cross-talk (Felleman & Van Essen 1991; Van Essen et al 1992), some of which is illustrated in Figure 1. Neurophysiological studies have demonstrated functional intermixing (Malpeli et al 1981; Nealey et al 1991), and behavioral studies have contradicted some of the proposed functional segregation (Schiller et al 1990a). Consequently, those outside the immediate field have found it increasingly difficult to know whether it is appropriate to consider the visual system as made up of subsystems.

This question cannot be answered adequately with a simple yes or no. A careful assessment of the available evidence suggests that the con-

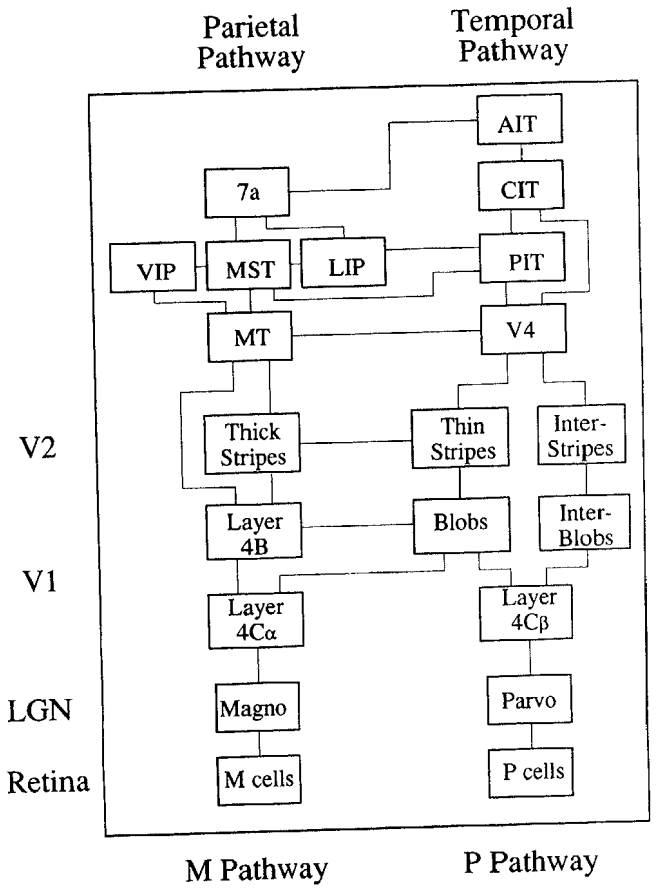

Figure 1 Parallel pathways in the primate visual system. The visual system is shown in schematic form from the retinal ganglion cells (*bottom*) to the higher levels of visual cerebral cortex (*top*). The components of the magnocellular and parietal pathways have been grouped to the left; those of the parvocellular and temporal pathways have been grouped to the right. Lines show established connections between the illustrated components. As in other summaries of visual pathways, many cortical areas and connections have been omitted. Abbreviations: AIT, anterior inferotemporal area; CIT, central inferotemporal area; LIP, lateral intraparietal area; Magno, magnocellular layers of the LGN; MST, medial superior temporal area; MT, middle temporal area; Parvo, parvocellular layers of the LGN; PIT, posterior inferotemporal area; VIP, ventral intraparietal area.

tributions of the M and P subcortical pathways retain some degree of segregation in visual cortex, but that their separation is far from complete and may not justify viewing the components as subsystems. This review elucidates the degree of parallel organization in the visual system through

a critical evaluation of the anatomical, physiological, and behavioral evidence for and against parallel visual subsystems. Because the independence of the proposed visual subsystems is contingent on the independence of the M pathway from the P pathway and of the parietal pathway from the temporal pathway, we first consider the degree of segregation of the M and P pathways up to the level of layer 4C in V1. Segregation between the parietal and temporal pathways is then addressed, starting from the level of area V4 and the middle temporal visual area (MT). Finally we address the question of the linkage between the subcortical and cortical pathways, specifically the extent to which they can be considered to correspond. We restrict our discussion to the primate visual system, principally that of the macaque monkey.

THE SEGREGATION OF THE M AND P PATHWAYS

Neurons in the P and M pathways have been extensively studied (see Derrington & Lennie 1984; Ingling & Martinez-Uriegas 1983; Lee et al 1989; Purpura et al 1988; Shapley & Perry 1986). We focus here on observations that bear on the independence of the pathways.

Anatomical Evidence

A striking feature of the P and M pathways is the apparently strict anatomical independence they maintain. Several studies have looked for anatomical evidence of mixing between P and M pathways in the lateral geniculate (Conley & Fitzpatrick 1989; Michael 1988), but have found axonal arbors and dendritic fields in parvocellular and magnocellular layers to be separated. Likewise, the projections from parvocellular layers terminate primarily in V1 layers 4A and 4Cβ, whereas those from magnocellular geniculate terminate in layer 4Cα (Fitzpatrick et al 1985). Both subdivisions of the LGN have connections with layer 6 of VI; however, the cortical projections to the magnocellular and parvocellular layers appear to arise from separate subdivisions within that layer (Fitzpatrick & Einstein 1989; Lund & Boothe 1975). Thus, the anatomical segregation of the P and M pathways appears to be essentially complete.

Physiological Evidence

The physiological responses of cells in the P and M pathways are virtually identical on some dimensions and profoundly different on others. The most striking difference is sensitivity to color. P pathway neurons show color opponency of either the red/green or blue/yellow type, which means

that they respond to color change regardless of the relative luminance of the colors (Derrington & Lennie 1984). M neurons, on the other hand, are considered insensitive to color, because they have virtually no response to color alternation when the luminance of the color is balanced. However, even when luminances are balanced (i.e. at isoluminance), M cells show a nonselective response to color transitions (a nonsigned, frequency-doubled response) (Lee et al 1989; Schiller & Colby 1983) that might be used for detecting temporal change.

Cells in the P and M pathways also differ greatly in the time course of their response to visual stimuli. In response to step changes in illumination, P cells show a more tonic response than M cells (Purpura et al 1990; Schiller & Malpeli 1978). P and M pathway neurons are also different in conduction velocity, with the stouter M pathway cells conducting impulses more rapidly (Gouras 1969; Kaplan & Shapley 1982; Schiller & Malpeli 1978). It is not clear if this difference has functional significance, given that total transmission times between retina and visual cortex differ by only a few milliseconds (Lennie et al 1990; Sherman et al 1984). Another physiological difference between P and M cells is their sensitivity to stimulus contrast. The contrast sensitivity of M cells is typically many times that of P cells. P cells rarely respond well to luminance contrasts below 10%, whereas M cells often respond to stimuli with contrasts as low as 2% (Purpura et al 1988; Sclar et al 1990; Shapley et al 1981). This difference in sensitivity might stem from M cells having larger receptive fields (de Monasterio & Gouras 1975), which would indeed result in greater sensitivity (Lennie et al 1990). However, recent studies do not show a marked difference between P and M cells in receptive field center size (Blakemore & Vital-Durand 1986; Crook et al 1988; Derrington & Lennie 1984), a finding that is certainly surprising given the large difference in the size of the dendritic fields of P and M retinal ganglion cells (Perry & Cowey 1985; Rodieck et al 1985) and their great difference in contrast sensitivity.

Along other stimulus dimensions, including spatial response, temporal response, and luminance response, neurons in the P and M pathways have different, but largely overlapping, ranges of sensitivity. M pathway cells are often reported to be responsive to higher temporal and lower spatial frequencies than P cells (Derrington & Lennie 1984; Hicks et al 1983). However, this difference is small, perhaps 15% in peak temporal frequency, cutoff temporal frequency, and peak spatial frequency, and there is a substantial overlap along all of these dimensions between the two classes of cells (Blanckensee 1980; Sherman et al 1984). There also appears to be a large overlap in the range of luminances over which neurons in P and M pathways respond. M pathway cells responded to somewhat lower luminance levels (Purpura et al 1990), but neurons in both pathways

respond at rod mediated (scotopic) light levels (Virsu & Lee 1983; Wiesel & Hubel 1966). The possibility that the M pathway may dominate vision under scotopic conditions remains controversial (see Purpura et al 1990).

Some response properties of the P and M pathways show little difference. The most prominent is spatial resolution. As with other properties described above, spatial resolution in both the P and M pathways varies with eccentricity from the fovea and differs in temporal and nasal portions of the visual field. But, at a given eccentricity, neurons in both pathways have virtually the same spatial resolution (Crook et al 1988). This is surprising, because early studies described an approximately threefold difference in the size of receptive fields centers in P and M cells (deMonasterio & Gouras 1975), and spatial resolution is inversely related to receptive field center size among cells that show linear spatial summation, which includes virtually all P and M cells (Shapley et al 1981). However, more recent measurements and calculations have shown that there is little difference in the size of receptive field centers in the two pathways (Blakemore & Vital-Durand 1986; Crook et al 1988; Derrington & Lennie 1984).

Thus, P and M cells in retina and LGN are qualitatively different on only a few dimensions—color opponency, time course of response, and contrast gain. Along some other important dimensions, such as luminance, temporal frequency and spatial frequency (Shapley & Lennie 1985), they each cover a wide range of values, with differences in their mean response, but a large overlap in effective stimuli. Moreover, they appear to show almost no difference in spatial resolution or receptive field center size at any given eccentricity. Thus, although anatomy indicates clear segregation between P and M pathways, physiological properties reveal both differences and similarities. In the following section, we consider behavioral evidence that addresses the functional significance of the above physiological properties.

Behavioral Evidence from Lesions of M or P Pathways

In the past few years, techniques to create selective, localized lesions in the P and M pathways have been developed. These include the use of excitotoxins, such as ibotenic acid, that damage cell bodies, but spare fibers of passage (Schwarcz et al 1979), and acrylamide, which selectively lesions the P pathway (Lynch et al 1992). When combined with careful behavioral measures of visual capabilities, these approaches provide valuable insights into the functional specialization of the P and M pathways. The results of these lesion studies can easily be related, a posteriori, to the known anatomy and physiology of P and M pathway cells. However, we believe that these lesion results could not be fully predicted from current anatomical and

physiological knowledge, given the large variety of predictions that would be consistent with this knowledge.

EFFECTS OF M PATHWAY LESIONS M pathway lesions cause a large decrease in luminance contrast sensitivity for stimuli of higher temporal frequency and lower spatial frequency (Merigan et al 1991a). Figure 2*B*, which shows sensitivity to temporal frequency measured with a low spatial frequency, illustrates this loss. Loss is restricted to stimuli that include both high temporal and low spatial frequencies. There is no loss of temporal frequency sensitivity at high spatial frequencies, nor loss of sensitivity to high spatial frequency at low temporal frequencies. (This explains why no loss in low spatial frequency sensitivity is seen in Figure 2*A*, which plots sensitivities measured with a low temporal frequency). The reduction of sensitivity to high temporal and low spatial frequencies results in reduced visibility of rapidly moving or rapidly flickering stimuli. This result is consistent with the greater sensitivity of M pathway neurons at high temporal and low spatial frequencies described above. M lesions cause almost no change in flicker resolution for high contrast stimuli (Merigan

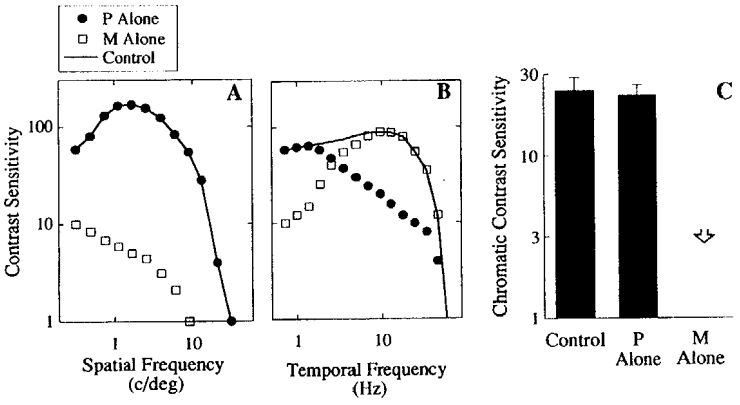

Figure 2 Contrast sensitivity of the P and M pathways determined from lesion studies. (*A*) Spatial contrast sensitivity for stationary gratings (zero temporal frequency). Contrast sensitivity is the inverse of the lowest stimulus contrast that can be detected. The solid line shows sensitivity of the intact monkey; filled circles show the contribution of the P pathway (after M lesions); and open squares the contribution of the M pathway (after P lesions). (*B*) Temporal contrast sensitivity measured at a low spatial frequency. Conventions are the same as in *A*. Labels in panels *A* and *B* should be taken as only relative, because these functions vary in spatial and temporal frequency with eccentricity. (*C*) Color contrast sensitivity in control, as well as after M or P pathway lesions. The arrow indicates the highest chromatic contrast that could be measured. Animals with P pathway lesions could not detect the target as this level. Adapted from Merigan et al (1991a,b).

& Maunsell 1990), although Schiller et al (1990a,b) have reported that such lesions reduce flicker resolution thresholds to about 9 Hz. The difference may be due to lower effctive contrast of the test stimuli in the experiments of Schiller et al (1990a,b). This explanation is illustrated in Figure 2*B*: The P and M pathways differ little in temporal resolution (highest temporal frequency that can be seen) at high contrasts (small y values), but differ greatly at lower contrasts (large y values). Additional measures indicate that M lesions cause no changes in either visual acuity or color contrast sensitivity (Merigan et al 1991a,b), which suggests little M pathway involvement in these functions. Effects of M pathway lesions on stereo vision and motion perception (Merigan et al 1991a; Schiller et al 1990a) are discussed below in the analysis of possible relationships of P and M to the cortical pathways.

EFFECTS OF P PATHWAY LESIONS P pathway lesions cause complementary effects to those of M lesions, thus reducing luminance contrast sensitivity for stimuli of higher spatial and lower temporal frequency content (Merigan et al 1991a,b; Merigan & Eskin 1986). Figure 2*A*, which plots sensitivities measured with a low temporal frequency, shows this effect. Thus, although physiological recordings show that individual P cells do not respond well to contrasts of less than 10% (Shapley et al 1981), these lesion studies indicate that the P pathway can mediate the detection of contrasts as low as 0.5% at low temporal frequencies (Merigan & Eskin 1986), which suggests a large contribution of spatial and probability summation to this behavioral response (Watson 1992). The role of the P pathway in detecting high spatial frequencies is also reflected in the approximately fourfold decrease in visual acuity that follows P lesions (Merigan et al 1991b). These behavioral findings match the superior low temporal frequency response of P cells (Purpura et al 1990), but are clearly not determined by the physiological spatial resolution of P and M cells, which are nearly identical (see above). The superior acuity mediated by the P pathway appears to be caused by the greater sampling density of retinal P ganglion cells (Merigan & Katz 1990), which follows from the approximately 8:1 ratio of P to M retinal ganglion cells (Perry et al 1984; Silveira & Perry 1991).

Perhaps the most dramatic effect of P pathway lesions is an apparently complete loss of color vision (Figure 2*C*) (Merigan 1989; Merigan et al 1991b; Schiller et al 1990a). This result is consistent with the color opponency of P cells and suggests that the residual frequency-doubled response of M cells at isoluminance (Lee et al 1989; Schiller & Colby 1983) is not sufficient to mediate even the most primitive aspects of color vision, such as detection of chromatic gratings. It is worth noting, in light of the higher luminance contrast sensitivity of the P pathway than of the

physiological response of individual P cells, that the color sensitivity mediated by the P pathway is also substantially higher than that of individual P pathway neurons (Derrington & Lennie 1984; Merigan 1989). The effects of P pathway lesions on pattern, texture, and stereoscopic vision (Schiller et al 1990a,b), and on form discrimination (Lynch et al 1992) are discussed later in the context of the relationships of the subcortical and cortical pathways.

The results of these lesion studies show clear differences in the contributions of the P and M pathways to vision. For the most part, these distinctions correspond well with the response properties of their neurons. For example, color vision appears to be subserved by the P pathway. However, these results also point to the risks of predicting behavioral contributions based on physiology. Although M pathway neurons have much higher contrast sensitivity than P cells, M lesions do not affect behavioral contrast sensitivity at low temporal frequencies (Figure 2*A*). The relatively poor contrast sensitivity of the P pathway appears to be overcome by the greater number of P cells. Likewise, the higher spatial resolution of the P pathway appears to reflect the higher sampling density of the more numerous P cells (Merigan et al 1991b), as P and M pathway cells do not differ in their physiological spatial resolution.

In summary, P and M pathways are anatomically and functionally distinct, but their basic specializations appear to be for low-level properties, such as spatial and temporal frequency. In the next section, we consider specialization of the cortical pathways and subsequently ask whether there are special links between the subcortical and cortical pathways.

THE SEGREGATION OF THE PARIETAL AND TEMPORAL PATHWAYS

Lesions of the Parietal or Temporal Pathways

Although functionally independent visual pathways have been discussed for many years in the context of parallel relationships between cortical and subcortical structures (Schneider 1967; Trevarthen 1968), Ungerleider & Mishkin (1982) proposed that distinct pathways exist within visual cortex itself. The behavioral data relevant to this proposal depended on differences in lesion-induced deficits that follow temporal and parietal lesions. A long-standing clinical literature had shown that lesions in human parietal cortex can cause extreme hemifield neglect and a disruption of visuomotor orientation. When the site of the lesion is temporal cortex, patients frequently have difficulty with form discrimination (agnosia) and problems with visual memory. These syndromes were so distinct that

neurologists concluded that the affected regions of the brain were specialized for spatial representation and object recognition, respectively (see Grüsser & Landis 1991).

The behavioral data from monkeys depended on testing animals with selective lesions of temporal or parietal cortex. Pohl (1973) reported one of the very few studies that involved a direct comparison of the effects of parietal and temporal lesion. He tested parietal and temporal lesions by using two tasks in which the animal had to locate which of two food wells contained a reward. In the "landmark" task, an object was placed next to the loaded food well. In the "object discrimination" task, an object was placed near each well, and the animal learned which one marked the loaded well. Groups of monkeys with temporal or parietal lesions were tested on both tasks. Animals with either lesion learned to do both tasks. They differed only in the rate at which they learned the tasks or, in some cases, the rate at which they relearned the tasks after the landmark was switched to the empty well, or the reward-contingent object was switched in the object discrimination task. With the landmark task, there was no difference between parietal and temporal lesions in initial learning. However, monkeys with parietal cortex lesions were slower at learning reversals than were those with temporal lesions. On the object discrimination task, monkeys with temporal lobe lesions made more errors during initial learning, and this difference persisted over several reversals. In a separate group of animals, Pohl tested post-lesion relearning, rather than initial learning, with temporal or parietal lesions after pretraining on a more difficult landmark task. On this task, there was also a clear difference between groups, with the parietal lesioned monkeys showing many more errors.

Other studies that have compared parietal and temporal lesions have not replicated these effects. Ungerleider & Brody (1977) tested acquisition of the landmark task after parietal or temporal lobe lesions and found a greater disruption during reversal learning in monkeys with temporal lesions, a finding at odds with Pohl (1973). Other groups found no deficit in landmark performance after posterior parietal lesions (Petrides & Iversen 1979; Ridley & Ettlinger 1975). These discrepancies might be explained by differences in the methods of testing or in precise placement of the lesions, but they indicate some vagaries of the behavioral observations.

Numerous studies have subsequently tested temporal and parietal lesions with a landmark task (often testing relearning) or an object discrimination task (often testing initial learning). Most of these studies have focused on questions specific to one or the other pathway, such as which regions of parietal cortex are most critical for landmark task performance (e.g. Mishkin et al 1982) and have not attempted further dissociation of temporal and parietal lesion effects. Collectively, the results of these studies

give the impression of dissociable effects of parietal and temporal lesions (see Mishkin & Ungerleider 1982); however, the different methods of measurement and analysis make comparisons between studies difficult to interpret.

An additional cause for concern in evaluating comparisons of cortical pathway lesions is that posterior parietal lesions often damage the optic radiations, as indicated by degeneration in the LGN. The visual field defects resulting from such collateral damage complicate the interpretation of these extrastriate lesions. Note that visual field scotomas following medial striate cortex lesions can produce larger effects on the landmark than on the object discrimination task (Mishkin 1966; Ungerleider & Mishkin 1982). Thus, a comparison of parietal and temporal lesions on these two tasks without the added complexity of visual field loss would be most helpful.

Overall, there are few studies that have attempted a double dissociation of the effects of temporal and parietal cortex lesions. Because questions remain regarding reliability of differential effects, the available studies provide only weak support for the segregation of parietal and temporal pathways. Other experiments have studied parietal or temporal lesions separately by using behaviors specifically chosen for the pathway being studied. These studies were generally not designed to distinguish cortical pathways and they provide little evidence to support or refute the notion of segregated pathways, because they typically have not compared the effects of different lesions on the same behaviors.

Included in this category are studies that have examined motion perception and eye movements following parietal pathway lesions made in areas MT, MST, or parietal cortex. Lesions of MT produce severe deficits in motion perception (Newsome & Paré 1988) and eye movements (Dürsteler & Wurtz 1988; Newsome et al 1985). However, if the lesions are not large, these results are transitory, with virtually complete recovery within days. Larger lesions, involving portions of MT and MST (Newsone & Paré 1988; Yamasaki & Wurtz 1991), produce more permanent disruptions of motion perception and both pursuit and saccadic eye movements. Complete, bilateral MT-MST lesions (Pasternak et al 1991) cause persistent disruptions of several aspects of motion perception, including speed thresholds, direction thresholds, and directionally noisy global motion.

Lesions higher in the parietal pathway, in posterior parietal cortex, cause a diffuse syndrome, which is more severe the larger the lesion (Lynch 1980). Unlike humans with parietal lesions, who show profound neglect of the field contralateral to the lesion, monkeys show a relative neglect, termed extinction, when two stimuli are presented simultaneously in ipsilateral and contralateral fields. Eye movements are affected by unilateral

lesions, with reduced slow phase OKN and increases in saccadic latency. Profound effects on pursuit eye movements are seen after bilateral lesions. Finally, there is some evidence of spatial disorientation, including disrupted maze performance and difficulty navigating in a familiar room (Sugishita et al 1978).

The function of the temporal pathway has been tested with lesions placed either lower in the pathway, in cortical area V4, or in inferotemporal cortex. Numerous studies have examined the effects of inferotemporal cortex lesions and found that such lesions greatly increase the number of errors made during initial learning of visual discriminations, as well as in recall of previously learned discriminations. Such effects are found for tests along single dimensions (e.g. size, color, luminance), as well as for complex (e.g. shape) discriminations. However, these effects disappear with a variety of manipulations. None are seen in young animals, when easy discriminations are used, for previously overlearned discriminations, or when errors are punished with shock (Gross 1973). Lesions of area V4 show small to moderate deficits in rather complex functions. Desimone et al (1990) found modest deficits in orientation, direction, color, and texture discriminations. Schiller & Lee (1991) reported an exaggeration of the normal finding that stimuli are more detectable from background stimuli if they are distinguished by possessing, rather than lacking, a salient feature (Treisman 1988). Heywood & Cowey (1987) tested color and shape discrimination after making complete bilateral lesions of V4. The relearning of both types of discrimination was disrupted, and tests of color discrimination suggested a small, but permanent, deficit.

In general, it appears widely accepted among investigators that parietal and temporal lesions produce different deficits, although there is little direct evidence bearing on this question. Most studies have not directly compared temporal versus parietal pathways, so we are forced to rely heavily on results from studies that have examined one or the other pathway. This approach is problematic for the purposes of this review, because the effects of parietal or temporal lesions are frequently small and/or transitory, which suggests that the tests used were not well matched to the role of the pathway, that the visual system involves more distributed processing than could be detected with such local lesions (DeYoe & Van Essen 1988), or that other areas rapidly assume functions interrupted by lesions (Newsome & Paré 1988). There is not, at present, sufficient evidence to permit a choice among these alternatives.

Physiology of the Parietal and Temporal Pathways

The cortical visual areas that make up the parietal and temporal pathways are distinguished by the response properties of their neurons. Neurons in

the temporal pathway are relatively sensitive to color and form, whereas those in the parietal pathway are more sensitive to the movement of visual stimuli (see Desimone et al 1985; Desimone & Ungerleider 1989; DeYoe & Van Essen 1988; Maunsell 1987; Maunsell & Newsome 1987; Van Essen & Maunsell 1983). Differences in the emphasis on central versus peripheral parts of the visual field also exist (see Ungerleider & Mishkin 1982).

Some of the most striking response selectivities are seen in neurons in the highest levels of the parietal and temporal pathways, in inferior parietal and inferotemporal cortex. Many neurons in inferotemporal cortex are selective for colors, or complex patterns or shapes (occasionally faces or hands) (see Desimone 1991). Surveys of selectivity in inferotemporal cortex find that about one half to two thirds of visually responsive units show obvious selectivity of this sort (Desimone et al 1984; Tanaka et al 1991), although most of these units respond, to some extent, to any visual stimulus. In contrast, neurons in the later stages of the parietal pathway are selective for complex types of motion, such as expansion or rotation (Duffy & Wurtz 1991; Motter & Mountcastle 1981; Saito et al 1986; Sakata et al 1985; Snowden et al 1991; Tanaka et al 1986).

Unfortunately, it is difficult to compare the properties of inferotemporal and parietal neurons precisely based on the available data. As with the behavioral data, few studies have directly compared the response properties in the two regions. There has never been a detailed study of the selectivity of parietal neurons for stimulus form or color, nor have inferotemporal neurons been extensively tested for properties typically studied in parietal areas, such as direction selectivity. One neuronal property that has been examined in both regions is the effect of spatially directed attention in behaving animals, which usually has an opposite effect in the two regions. Spatial attention enhances responses of parietal neurons, but suppresses the responses of inferotemporal neurons (Bushnell et al 1981; Richmond et al 1983). The dearth of single-unit recordings that directly compare the higher levels of the parietal and temporal pathways can be taken as testimony to the confidence of physiologists about the differences between these regions of visual cortex. Nevertheless, the distinctions between parietal and temporal cortex remain documented primarily by incidental observations.

The direct comparisons that have been made between the parietal and temporal pathways mostly concern MT and V4. Figure 3 summarizes the selectivity of neurons in these areas for four of the best studied stimulus dimensions. There is a dramatic distinction in emphasis on color and direction. Numerous studies have shown that whereas MT lacks clear color selectivity (Maunsell & Van Essen 1983b; Movshon et al 1991; Saito et al 1989; Zeki 1974), many neurons in V4 are robustly color selective

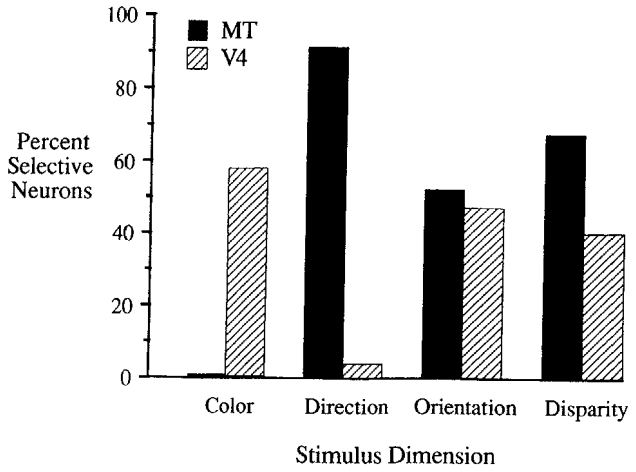

Figure 3 Stimulus selectivities in MT and V4. Proportions of neurons selective for four stimulus dimensions are plotted for both areas. The data are taken from numerous studies from several laboratories and include both quantitative and subjective assessments of selectivity. In particular, the differences between color and direction selectivity are based entirely on qualitative assessments. Some values are based on data from a single study. Adapted from Felleman & Van Essen (1987).

(Schein & Desimone 1990; Van Essen & Zeki 1978; Zeki 1973). A prevalence of color selectivity persists to the highest levels of the temporal pathway (Desimone et al 1984; Gross et al 1972; Komatsu et al 1992). Direction selectivity, which is also often considered to distinguish neurons in the cortical pathways, is abundant in MT (Albright 1984; Maunsell & Van Essen 1983b; Zeki 1974) and conspicuous in areas in the posterior parietal cortex (see Andersen 1987; Wurtz et al 1990), but rarely found in V4 (Van Essen & Zeki 1978; Zeki 1978). Other stimulus selectivities, such as orientation and disparity sensitivity, do not clearly distinguish the parietal and temporal pathways, even at the level of MT and V4. Selectivity for orientation, speed, binocular disparity, and contrast sensitivity all appear to be comparable in both areas (Cheng et al 1991; see Felleman & Van Essen 1987).

Functional distinctions between the parietal and temporal pathways have recently been demonstrated in humans by using positron-emission tomography (PET). Differential activation of parietal and temporal cortex occurs when subjects view moving or color stimuli (Zeki et al 1991) and when they perform a face-matching or a spatial vision task (Haxby et al 1991). Corbetta and colleagues (1991) compared cortical activation when subjects were required to attend to different aspects of a single stimulus. Subjects viewing moving colored bars were directed to attend to the speed,

the shape, or the color of those bars. Attention to speed activated sites in the inferior parietal lobule and the superior temporal sulcus, whereas attention to color or shape activated sites that were closer to the inferior surface of the brain, although the separation of activity was not as clear as in the studies that used different stimuli.

In summary, the PET studies and single-unit recordings support the idea of a parallel division between the parietal and temporal pathways. These differences appear to fall along the lines of identification and recognition versus localization and spatial relationships, although our understanding of the division is far from complete. Also, the extent to which the neuronal properties of the two cortical pathways overlap remains to be established. Until more data become available, it will not be possible to describe physiological differences between the parietal and temporal pathways with precision and certainty.

Anatomy of the Parietal and Temporal Pathways

The distinction between the parietal and temporal pathways is also supported by segregated cortico-cortical connections. For example, the outputs of V4 are directed primarily to inferotemporal cortex, whereas those of MT lead for the most part to parietal cortex (Desimone et al 1980; Maunsell & Van Essen 1983a). The strongest anatomical evidence for parallel organization comes from studies of the extent of overlap in the areas projecting to parietal and temporal cortex that have been done in the same animal. Morel & Bullier (1990) and Baizer and colleagues (1991) injected different neuroanatomical tracers into inferior parietal (in and near LIP and VIP) and inferotemporal cortex (in CIT). Both studies found that although the injections labeled large portions of visual cortex, there was very little overlap in the distributions of the two labels.

This result may seem inconsistent with the numerous cross-connections that have been demonstrated between areas in the two cortical pathways. Some of these cross-connections can be seen in Figure 1, but many more exist. Figure 1, like almost all diagrams of the cortical pathways, shows a highly selected subset of areas and connections. The known connections between areas in macaque cerebral cortex now number over 300, and the complete set provides no clear impression of two parallel pathways (see Figure 4 in Felleman & Van Essen 1991). Indeed, far less segregation would probably be observed in the studies by Morel & Bullier and Baizer et al, had the injections been made at earlier or later levels in the pathways. For example, MT and V4 are reciprocally connected (Maunsell & Van Essen 1983a; Ungerleider & Desimone 1986). However, cross-connections at early and late levels do not preclude anatomical segregation over substantial portions of the parietal and temporal pathways.

The absence of obvious parallel pathways in the complete set of cortical connections is perhaps not surprising. Although quantitative estimates of the number of axons in projections are difficult to obtain, it is a common observation that connections differ greatly in their strength and consistency (e.g. Tanaka et al 1990; see Van Essen 1985). These anatomical differences are presumably reflected in their relative influence on response properties in the recipient areas. "Wiring diagrams" that give equal weight to all pathways may obscure functional relationships. The connections between the two pathways might be weaker than those within pathways, although this question has never been addressed experimentally. Also, some connections might be primarily modulatory and have little influence on stimulus selectivities. For example, there is little identifiable evidence of the connection between MT and V4 in the response properties of their neurons.

In summary, the evidence from behavioral, physiological, and anatomical experiments makes a clear case for the distinctiveness of the parietal and temporal pathways. However, the strength of the observations varies greatly, and the segregation between the cortical pathways is not as striking as that between the M and P pathways. Very few studies have directly compared the parietal and temporal pathways. The anatomical experiments that compared inputs to parietal and inferotemporal cortex are a notable exception and provide some of the best support for distinct cortical pathways. Corroborating evidence is provided by differences in neuronal reponse properties, although direct comparisons of the pathways are lacking in most of these studies. Studies that have compared parietal and temporal lesions show differential effects on spatial and recognition tasks, but these effects are, for the most part, disappointingly weak and primarily involve differences in rates of learning. Because large regions of visual cortex are relatively unexplored, this picture of two parallel cortical pathways may still change.

LINKS BETWEEN THE CORTICAL AND SUBCORTICAL PATHWAYS

The issue of visual subsystems concerns the degree to which the subcortical pathways map onto the cortical pathways in a one-to-one fashion. In this section, we consider first the anatomical and physiological data that address whether contributions from the M and P pathways remain segregated in visual cortex up to the level of MT and V4. We then consider data that compare the neuronal properties and behavioral contributions of the cortical and subcortical pathways.

The Pathways from V1 to MT and V4

ANATOMY One of the most important contributions to bridging the gap between the subcortical and cortical pathways was made in 1978 by Margaret Wong-Riley, who demonstrated cytochrome oxidase-rich blobs in the superficial layers of striate cortex (see Hendrickson 1985). The blobs and interblobs in V1 (Horton & Hubel 1981; Humphrey & Hendrickson 1980) and the thin stripes, thick stripes, and interstripes in V2 (Livingstone & Hubel 1982; Tootell et al 1983) create subdivisions that could support parallel segregation within these areas. Until the patterns of cytochrome oxidase staining were discovered in V1 and V2, their cytology had been thought to be uniform, and there was little evidence that they contained separate routes for different classes of visual information.

Studies of the connections of the cytochrome oxidase compartments suggest that they make up parallel routes through early visual cortex and that these routes connect to the parietal and temporal pathways in a selective way (Figure 1). Retrograde tracer studies demonstrated that MT receives V2 projections mainly from the thick stripes, whereas projections to V4 arise from both the thin stripes and the interstripes (DeYoe & Van Essen 1985; Shipp & Zeki 1985). The thin stripes and interstripes in V2 in turn interconnect with the blobs and interblobs in V1 (Livingstone & Hubel 1984a). The thick stripes receive input from layer 4B (at least in squirrel monkey) (Livingstone & Hubel 1987), which also sends a direct projection to MT (Lund et al 1976; Maunsell & Van Essen 1983a). Further anatomical evidence for differences between the cytochrome oxidase compartments is provided by the antibody Cat-301, which selectively labels layer 4B, the V2 thick stripes and MT (DeYoe et al 1990).

However, the separation between the compartments is not complete. Although many of the connections of the cytochrome oxidase compartments in V1 and V2 appear to maintain their independence (Livingstone & Hubel 1984b), other relevant connections do not respect their borders. Local circuit neurons make extensive cross-connections within V1 (Yoshioka & Lund 1990). In V2, direct connections exist between the thin and thick stripes (Livingstone & Hubel 1984a), and both are labeled when the pulvinar is injected (Livingstone & Hubel 1982). In owl monkey, thick and thin stripes are also labeled following injection of the dorsomedial visual area (Krubitzer & Kaas 1990). All three stripes receive feedback projections from area V4 (Zeki & Shipp 1989). These cortical and subcortical connections (as well as others not yet identified) could thwart the apparently sharp segregation laid out in other connections of the cytochrome oxidase compartments.

The idea that the M and P pathways correspond to the parietal and

temporal pathways rests largely on the cytochrome oxidase compartments in V1 and V2 acting as conduits, thus making selective connections with the subcortical pathways in V1 and selective connections with the parietal and temporal pathways in MT and V4. Although the anatomical evidence for selective connections with MT and V4 is strong (DeYoe & Van Essen 1985; Shipp & Zeki 1985), the connections in V1 appear to maintain only a partial segregation between the contributions of the M and P pathways. Layer 4B receives direct input from 4Cα (M pathway), but not 4Cβ (P pathway) (Lund & Booth 1975; Lund et al 1979), consistent with M pathway dominance in the route that leads to MT and the parietal pathway. On the other hand, although 4Cβ projects to the blobs and interblobs in the superficial layers, the blobs in superficial layers also receive major inputs from the M pathway by way of layers 4B and 4Cα (Blasdel et al 1985; Fitzpatrick et al 1985; Lachica et al 1992; see also Lund 1988).

The anatomical data suggest a partial mixing of M and P pathway contributions in V1, but they are not conclusive about the degree of functional segregation that exists in the early stages of visual cortex. Collectively, interlaminar connections in V1 and the cross-connections between the cytochrome oxidase compartments in V1 and V2 could intermix M and P contributions completely before they reach the level of the MT and V4. Alternatively, they could have no appreciable effect, and connections that appear to mix the different pathways might provide only modulatory inputs. Other observations confirm the suggestion that both the M and P channels make substantial contributions to response properties in the superficial layers in V1 and to later stages in the temporal pathway.

PHYSIOLOGY Segregation between the cytochrome oxidase compartments and their relationships to the M and P pathways have been examined by comparing response properties in the different cytochrome oxidase compartments with each other and with those seen in the M and P pathways. Such comparisons have revealed clear differences between the cytochrome oxidase compartments, consistent with a segregation of streams of information. However, the physiological segregation is not complete, and its relationship to the M and P pathways is not clear.

Segregation Between the Cytochrome Oxidase Compartments Observations on the distribution of direction selectivity suggest segregation of function between the cytochrome oxidase compartments. Direction selectivity is concentrated in layer 4B in V1 (Dow 1974; Hawken et al 1988; Hubel & Livingstone 1990) and in the thick stripes in V2 (De Yoe & Van Essen 1985; Levitt et al 1990). Moreover, both structures project to MT, which is distinguished from V4 by abundant direction selectivity (Maunsell

& Van Essen 1983b; Zeki 1978). Layer 4B and the V2 thick stripes also appear to be relatively enriched in neurons that are sensitive to binocular disparity (DeYoe & Van Essen 1985; Hubel & Livingstone 1987; Poggio 1984; Ts'o et al 1991).

The blobs contain neurons that are selective for color and relatively unselective for orientation, whereas the converse holds in the interblobs (Blasdel 1992a,b; Landisman et al 1991; Livingstone & Hubel 1987; Tootell et al 1988b; Ts'o et al 1990; Ts'o & Gilbert 1988). Corresponding properties are found in the thin stripes and interstripes in V2 (Hubel & Livingstone 1987; Ts'o et al 1990). Color-sensitive neurons are far less common in layer 4B (Dow 1974; Dow & Gouras 1973; Hubel & Livingstone 1990; Poggio et al 1975; Tootell et al 1988b).

However, the segregation of response properties is not complete. The data of Livingstone & Hubel (1987) suggest that about two thirds of neurons in the blobs are color sensitive, compared with one third in the interblobs. And, Lennie and colleagues (1990) found little evidence for differences in color sensitivity between the blobs and interblobs. Conflicting results have been reported with respect to the segregation of response properties in V2. Hubel & Livingstone (1987) described a remarkably clear-cut division of neurons into three groups: disparity-tuned neurons in thick stripes, color-selective in thin stripes, and end-stopped in interstripes. Other studies have reported only a tendency for thin stripe neurons to be color selective and for thick stripe neurons to be disparity tuned, with neurons in all stripes showing similar selectivities for spatial and temporal frequency (DeYoe & Van Essen 1985; Levitt et al 1990; Ts'o et al 1991). These other studies also found appreciable numbers of color-selective neurons in the interstripes and no sharp division between color-selective and disparity-tuned cells. Some of the discrepancy may arise from difficulties in distinguishing thin and thick stripes anatomically (Crawford & Chodosh 1990). Because the analyses in the latter studies were more thorough and based on objective response measurements and computer-controlled stimuli, it is likely that they provide a more accurate picture and that the segregation of response properties in V2 is far from complete.

Association with the M and P Pathways The differential distribution of response properties among the subdivisions of V1 and V2 is consistent with the idea that they segregate classes of visual information. Some studies have directly compared response properties in the M and P pathways with those in the V1 and V2 subdivisions to assess the relationship between them. Although, in some cases, correlations have been put forth as evidence for maintained segregation, such comparisons prove difficult to interpret.

An example of the difficulties can be seen in the case of contrast sensi-

tivity. Neurons in V1 layer 4B and the V2 thick stripes have high contrast sensitivity (Blasdel & Fitzpatrick 1984; Hawken & Parker 1984; Hubel & Livingstone 1990; Tootell et al 1988a), which matches that in the M pathway. Yet, high contrast sensitivity may not require M pathway contributions. Although P pathway neurons have far less contrast sensitivity, they are not blind to low contrasts. Cortical processing that summed inputs from many relatively insensitive parvocellular neurons could produce a response as sensitive as any in the M pathway (Watson 1992). Summation of this sort has been demonstrated by the existence of cortical neurons that have greater contrast sensitivity than neurons in either subdivision of the LGN (Sclar et al 1990). This consideration leaves most observations about cortical response properties inconclusive regarding contributions from the M and P pathways, whether the observations suggest segregation or mixing. For example, contrast sensitivity comparable to that in the M pathway also exists in the V1 blobs and interblobs (Hawken et al 1988; Hubel & Livingstone 1990; Tootell et al 1988a). This sensitivity might reflect either M pathway contributions provided by the connections with layers 4B and 4Cα (Lachica et al 1992) or summation of P pathway inputs. Because the properties of the M and P pathways overlap so extensively, and summation of responses can increase sensitivities, it is difficult to reach firm conclusions about the presence of M and P pathway contributions from correlations of response properties.

One might hope that color sensitivity, which is perhaps the most distinguishing property of the P pathway, could provide a conclusive test of segregation. This approach is also of limited use. Demonstrating that a cortical neuron possesses a particular response property says little about segregation of the M and P pathways in cortex. Although the color sensitivity in the blobs clearly suggests input from the P pathway, it provides little insight into whether M pathway input is also present. Demonstrating segregation, which depends on showing an absence of one pathway's contribution, is more difficult than demonstrating a contribution. Many forms of processing can reduce sensitivities that exist at earlier levels, and such reductions might be expected as a consequence of cortical processing directed at elaborating more complex representations. Ultimately, the correspondence between response properties can be taken as corroborative, but not conclusive, evidence.

The contributions of the M and P pathways to cortical neurons can be assessed more directly by inactivating individual layers in the LGN (Malpeli & Schiller 1979). The presence or absence of changes in cortical visual responses following block of an LGN layer can be used to infer the contribution of the blocked pathway to the response. Malpeli et al (1981) used this approach to explore M and P contributions to cortical neurons.

They found that the P and M pathways mix within V1, with about 40% of neurons affected when either LGN subdivision was blocked. Subsequent work has shown that substantial M pathway contributions exist in both the blobs and interblobs in the superficial layers (Nealey et al 1991). This observation of M pathway contributions to both the blobs and interblobs differs from the anatomical data that show that layer 4Cα sends axons primarily to the blobs (Lachica et al 1992). The difference might depend on the influence of lateral connections within the superficial layers (Ts'o et al 1986). Also, the sharp distinction that is normally drawn between blobs and interblobs may be artificial (see below).

Although none of the anatomical or physiological data allow precise statements about the degree of segregation of contributions from the M and P pathways in the early stages of cortical processing, collectively they suggest that an incomplete segregation exists. The anatomical connections of layer 4B and the V2 thick stripes point to dominance by the M pathway. However, the existence of some color-sensitive neurons in layer 4B suggests some P pathway contribution. In the other cytochrome oxidase subdivisions, anatomical connections and prevalent color sensitivity suggest P pathway contributions, but selective LGN inactivations and anatomical connections with layers 4Cα and 4B all suggest that the M pathway makes a substantial, and possibly equal, contribution.

Correspondence Between the Cortical and Subcortical Pathways

If the apparent intermixing in V1 and V2 progresses at later stages of processing, the contributions of the M and P pathways might be completely mixed in the first few levels of cortical processing. Were this the case, physiological differences between the parietal and temporal pathways would reflect not differential M and P pathway contributions, but rather differences in the way that a combined signal was processed. It is, therefore, important to consider additional data that compare the subcortical and cortical pathways.

PHYSIOLOGICAL EVIDENCE Although correlations of response properties are inconclusive for the reasons stated above, the rough correspondence that exists between certain response properties in the M pathway and those in layer 4B and the V2 thick stripes persists in the parietal pathway. Such properties as transient responses and a lack of color specificity in parietal cortical areas led to suggestions of M pathway dominance before segregated pathways had been identified in V1 and V2 (Maunsell & Van Essen 1983b; Motter & Mountcastle 1981). Conversely, the presence of color-

selective neurons in the temporal pathways suggest contributions from the P pathways.

Unfortunately, correspondence between response properties in the cortical and subcortical pathways has rarely been demonstrated directly. Perhaps the best basis for a comparison between pathways exists for color. Neurons in the parietal pathway frequently lack color sensitivity (Maunsell & Van Essen 1983b; Robinson et al 1978; Zeki 1974, 1978). The few quantitative studies that have been performed for color sensitivity in the parietal pathway have focused on MT. Most neurons in MT are relatively unresponsive to isoluminant color borders, although many neurons demonstrate some capacity for distinguishing colors (Charles & Logothetis 1989; Dobkins & Albright 1990; Movshon et al 1991; Saito et al 1989). The decline in responsivity at isoluminance in most MT neurons is similar to the behavior of cells in the M pathway (Logothetis et al 1990; Schiller & Colby 1983). However, some neurons in MT do not show such a sharp decline. The residual color response of these cells almost certainly depends on contributions from the P pathway. As described above, poor color sensitivity could result from mixing P pathway inputs that had different color sensitivities.

Selective inactivation of the M or P pathway at the level of the LGN affects responses in the parietal and temporal pathways in ways that fulfill expectations, based on the pattern of partial segregation seen in V1 and V2. The responses of most neurons in V4 are reduced when either the M pathway or the P pathway is blocked (Ferrera et al 1991). In contrast, selective LGN inactivation suggests that the parietal pathway is largely dominated by the M pathway. Blocking the magnocellular layers of the LGN usually eliminates responses in MT and always reduces responses markedly (Maunsell et al 1990). Responses of some MT neurons are also reduced when parvocellular layers are inactivated, but the effects of P pathway block are weaker and less frequently observed.

Overall, the available anatomical and physiological data suggest that the relationship between the M and P pathways and the parietal and temporal pathways is asymmetric. The M pathway seems to dominate the parietal pathway, although some P pathway contributions are found. On the other hand, both the M and P pathways contribute appreciably to the temporal pathway. The segregation of the P and M pathways in extrastriate cortex appears to consist mainly of a partial exclusion of P contributions from the parietal pathway.

The evidence for substantial M pathway contributions to the temporal pathway raises the question of whether this input might be confined to one of the two routes that lead from V1 to V4. If so, one component of the temporal pathway might be dominated by the P pathway. The anatomical

routes stemming from the blobs and interblobs remain distinguishable at least to the level of V4, and probably beyond (DeYoe & Sisola 1991; Felleman & McClendon 1991; Zeki & Shipp 1989). They may, therefore, comprise two parallel divisions within the temporal pathway, perhaps serving different visual functions (DeYoe & Van Essen 1988). Because the V1 anatomy suggests that the M pathway contribution goes predominantly to the blobs (Lachica et al 1992), the interblobs might receive primarily P pathway contributions. However, the effects of selective M pathway inactivation show that M and P pathway contributions converge on individual neurons in V1 (Malpeli et al 1981), and that the M contributions are found in the interblobs (Nealey et al 1991). Furthermore, it remains possible that the blobs and interblobs are not components of independent pathways. They may, instead, belong to a single system in which properties vary continuously from blob-like to interblob-like, analogous to the way that neurons in the superficial layers of striate cortex vary between vertical-preferring and horizontal-preferring (see Silverman et al 1989). The borders of the blobs are diffuse and are not marked by interruptions of axonal or dendritic arborizations (Hübener & Bolz 1991; Malach 1991), and there is little compelling evidence for an abrupt physiological transition at the borders of blobs (Born & Tootell 1991). Whatever the actual relationship of the blobs and interblobs, at present there is little indication that either is strongly dominated by P pathway contributions, and the M and P pathways probably mix in the temporal pathway.

BEHAVIORAL EVIDENCE Two types of behavioral studies bear on the relationship between subcortical and cortical pathways. The first involves attempting to create stimuli that effectively isolate one subsystem. The second involves comparing the effects of lesions of P or M pathways with those following lesions of parietal or temporal cortex.

Selective stimuli Numerous studies have attempted to stimulate the P pathway alone by using isoluminant chromatic stimuli (Livingstone & Hubel 1987; Ramachandran & Gregory 1978), or random dot stereograms or texture defined stimuli (Cavanagh & Mathers 1989). These studies are not especially useful for exploring the contributions of the P and M pathways. Although the use of isoluminant chromatic stimuli only poorly stimulates neurons in the M pathway (see Cavanagh & Anstis 1991), the P pathway is not fully functional with isoluminance stimuli (Ingling & Grigsby 1990; Merigan et al 1991b). Any observed failures might be due to the lower spatial resolution of chromatic vision (Mullen 1985), the lower effective contrast delivered by chromatic stimuli (Smith & Pokorny 1975), or other limitations of chromatic mechanisms. Thus, the loss of many visual capabilities at isoluminance (Livingstone & Hubel 1987) suggests

only that the affected functions are normally mediated by the response of either M or P pathway neurons, or both, to luminance contrast. Likewise, the attempt to stimulate the P pathway selectively by using texture or random dot stimuli (Cavanagh & Mathers 1989) will succeed only if the perception of such stimuli requires the higher acuity of the P pathway (Merigan et al 1991b). This approach is also of questionable value, because there is currently no strong evidence that either chromatic or texture and stereo patterns can selectively stimulate the temporal pathway (Heywood & Cowey 1987; Movshon et al 1991).

Lesions Before considering lesion studies, it is important to stress that our conclusions are limited by shortcomings of the available data. The major deficiency is that, in most cases, the effects of temporal versus parietal pathway lesions have not been compared using the same visual capacities, although this is not the case for lesions of the M and P pathways. It is also difficult to compare M lesions with parietal pathway lesions or P lesions with temporal pathway lesions for the same reason. Thus, it is difficult to reach strong conclusions about the similarity, or lack thereof, of the effects of lesions of P and M or temporal and parietal pathways. Despite these limitations, the available evidence clearly suggests little relation of the subcortical to the cortical streams.

As described above, lesions of the P pathway disrupt color vision, acuity, and contrast sensitivity for stimuli of low temporal and high spatial frequencies. There is no indication that lesions of the temporal cortical pathway cause any similar effects, although these particular capabilities have not all been tested after temporal pathway lesions. The severe loss of color vision in some human patients with cortical lesions (e.g. Mollon et al 1980) appears as profound as that caused by P lesions, although it differs in that acuity is spared. However, lesions of cortical area V4 in the monkey produce only subtle disruptions of color discrimination (Heywood & Cowey 1987), which suggests that this portion of the temporal pathway is not critical to color vision. Visual acuity also appears not to be reliably reduced by V4 lesions (W. H. Merigan and J. Maunsell, unpublished), and thresholds for acuity, flicker, and orientation discrimination are not affected by IT lesions (Gross 1973).

The most characteristic effects of temporal pathway lesions are disruptions of shape discrimination, which have been seen after V4 (Heywood & Cowey 1987) or inferotemporal cortex lesions (Gross 1972), as well as alterations of visual memory that result from lesions of area TE of inferotemporal cortex (Phillips et al 1988). One might expect comparable effects following P lesions, if the only input to the temporal stream came from the P pathway. Those few studies that have examined shape discrim-

ination after P lesions (Lynch et al 1992; Schiller et al 1990a,b) have found no obvious impairment beyond that which would be expected from the loss of spatial resolution. No studies have examined effects of P lesions on visual memory.

Lesions of the M pathway result in decreased contrast sensitivity for stimuli containing high temporal and low spatial frequencies (see above). No comparable findings have resulted from lesions in the parietal pathway. No alteration in contrast sensitivity for stationary gratings was found by Newsome & Paré (1988) after lesions of area MT (although the temporal frequency content of these stimuli were probably closer to those detected by the P than the M pathway). More recently, Merigan et al (1991c) found no effect of combined MT/MST lesions on the detection of gratings drifting at velocities that included the range preferentially detected by the M pathway (Merigan et al 1991a). Studies of lesions elsewhere in the parietal pathway, such as posterior parietal cortex (Andersen 1987), have not tested effects on contrast sensitivity.

The most characteristic permanent effects of lesions in the parietal pathway include disruption of some aspects of motion perception (Merigan et al 1991c) and subtle changes in eye movements (Dürsteler & Wurtz 1988; Yamasaki & Wurtz 1991) after lesions of areas MT and MST, as well as rather profound changes in eye movements, and evidence of spatial disorientation (Lynch 1980) after parietal lesions. The effects of M pathway lesions described above are very different from these findings.

Lesions of the subcortical M pathway have not borne out the expectation that stereopsis should be dominated by the M pathway (Livingstone & Hubel 1987). This expectation was based on performance at isoluminance and the prevalence of disparity selectivity in layer 4B of V1 and in the thick stripes of V2. Schiller and colleagues (1990a,b) found that stereopsis mediated by high spatial frequency dot patterns was disrupted by P lesions, an effect that may simply reflect reduced visual acuity. M pathway lesions did not disrupt stereopsis.

The question of whether M pathway lesions affect motion perception is more complicated. Formally, motion perception survives M lesions, because direction discrimination at threshold, as well as speed discrimination, is possible in the absence of the M pathway (Merigan et al 1991a). Thus, if a stimulus is visible to a monkey with an M lesion, it can discriminate its direction of motion. We believe that the recent report that M pathway lesions can disrupt motion perception (Schiller et al 1990a,b) may reflect the monkeys' failure to detect the test stimulus after M lesions. Thus, motion perception may be indirectly altered by M lesions, because fast-moving stimuli that may be important to motion perception are difficult to see after an M lesion. This analysis may provide some insight

into the impressive anatomical and physiological evidence (above) of a special relationship between the M and the parietal visual pathways. It suggests that the M pathway is not specialized for motion perception, but is specialized for the transmission of middle and high velocity stimuli that are important to some functions of the parietal visual stream. However, neither stereopsis nor motion perception findings support the idea of separate subsystems for specific visual behaviors.

In summary, there appears to be little relationship between the effects of P or M pathway lesions and lesions of temporal or parietal cortex. The lesion effects that have been reported suggest that the subcortical pathways are specialized for the transmission of low-level stimulus properties to the cortical pathways and that the cortical streams themselves may be specialized for more sophisticated visual analysis.

CONCLUDING COMMENTS

Collectively, the available data provide a strong basis for a division between the M and P pathways and between the parietal and temporal pathways, but they suggest that the original notion of parallel visual subsystems that extend from the retina to higher visual cortex must be extensively modified. The mapping between the subcortical and cortical pathways is not simply one to one. Many lines of evidence suggest that the parietal pathway in cortex depends largely on M pathway contributions, but not to the exclusion of contributions from the P pathway. Anatomical, physiological, and behavioral evidence all point to the temporal pathway receiving major contributions from both subcortical pathways. Thus, we are left with an asymmetric organization that is only partially consistent with parallel subsystems.

Why should a partial relationship exist between the cortical and subcortical pathways? We are far from a complete understanding, but insights can be gained by comparing the lines along which each pair of pathways segregates. The P and M pathways are specialized for low-level stimulus features. The P system provides greater spatial resolution, selectivity for color, and the ability to respond to slowly changing or slowly moving stimuli. The M pathway is not selective for color, but achieves much higher sensitivity to moderate or rapidly moving stimuli. Lesion studies suggest that the most fundamental specialization of these two pathways may be the ability to transmit different regions of the "window of visibility" (Watson & Ahumada 1985), i.e. the range of temporal and spatial frequencies that can be seen. In this respect, the P and M pathways resemble specialized detectors that sense different, but overlapping, portions of visible spatial and temporal frequencies. Color vision, which

also sharply distinguishes the pathways, may play a less fundamental role, given that similar divisions of spatial and temporal frequencies are seen in species that lack color vision (Stone 1983). Color sensitivity may be a more recently evolved property that has become associated with P pathway, because the spatial or temporal frequencies in that pathway are better suited to color analysis. This perspective on the differences between the P and M pathway sets constraints on the types of possible relationships between subcortical and cortical pathways. In particular, it may help account for the strikingly asymmetric dominance of the parietal pathway by M input. The functions of the parietal pathway (about which we still know little) may almost exclusively depend on the moderate to high velocities transmitted by the M pathway, whereas functions of temporal cortex may require more of the full range of visible spatio-temporal contrasts.

Many readers may wonder how our conclusions can differ so much from the view of parallel subsystems that has reached such prominence. The explanation lies partly in overly enthusiastic acceptance of the notion of parallel subsystems. Few ideas in neuroscience have achieved anything approaching the acceptance that this proposal has received in the six years since it first appeared in print. Two factors have fueled its rapid acceptance. First, it promised to simplify greatly the vast collection of data on visual system organization, thus providing a rationale for the physiological differences between the M and P pathways and between the parietal and temporal pathways. However, simple ideas are not always robust. A relevant example is that the simple and long-standing model of orientation hypercolumns in V1 required fundamental revision following the discovery of cytochrome oxidase blobs. Second, the proposal of parallel subsystems was consistent with an impressively large collection of observations. Although none of those observations is conclusive, their number would make a strong case if each supported parallel subsystems independently. Unfortunately, they do not. For subsystems to exist from the retina to the highest levels of visual cortex segregation must be maintained at every level in the system. If V1 completely intermixed P and M pathway contributions, subsystems of this sort would not exist, regardless of observations that suggest segregation in earlier and later stages. If more than a few of the observations supporting segregation are proven wrong, the case for parallel organization quickly disintegrates.

We expect that the question of parallel pathways will continue to generate intense interest, and it is likely that our understanding will be refined in coming years. Whatever consensus emerges in the future, clearly the simple description that has held sway in recent years is, at best, a rough approximation of the truth.

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