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VISUAL SENSITIVITY AND PARALLEL RETINOCORTICAL CHANNELS

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Visual Sensitivity and Neural Mechanisms

There has been some excitement lately in relating psychophysical properties of visual sensitivity to neural mechanisms in the retina and in cerebral cortex. Parallel processing of visual information by the P and M retinocortical pathways has been a major focus of this interest. Visual psychophysicists and neuroscientists have devoted enthusiastic attention to each other's results. In this review I summarize the major psychophysical and neurophysiological findings on the role of P and M pathways that may allow a unified explanation for visual sensitivity, and also analyze several proposed hypotheses.

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The focus of interest is the degree to which color vision and achromatic vision may be thought of as parallel and independent sensory analyses of the visual scene. Theories of color vision have traditionally considered responses to black and white as the result of a neural mechanism different from those (the color-opponent neurons) that can discriminate among wavelengths or wavelength distributions (see, for example, Hurvich & Jameson 1957). This dualistic approach was reinforced by the neurophysiological work of De Valois and of Gouras and their colleagues in an earlier era of visual neurophysiology (reviewed in De Valois & De Valois 1975; and in Gouras 1984). The idea arose of a separate set of color-blind retinal ganglion cells that were "broad band" (i.e. sensitive to a broad band of the visible spectrum) and responsible for the visibility of black and white patterns. The numerous color-opponent ganglion cells were supposed to be the sole means by which signals about color traveled from eye to brain. Then opinion's pendulum swung the other way and hypotheses were formulated about how all of vision, both achromatic and chromatic, could be derived from the response characteristics of the color-opponent type of neuron (see e.g. DeValois & DeValois 1975; Ingling & Martinez-Uriegas 1983; Kelly 1983; Derrington et al 1984; Rohaly & Buchsbaum 1988, 1989). More recently, some neurophysiologists have returned to the dual-channel point of view (Shapley & Perry 1986; Livingstone & Hubel 1987, 1988; Lee et al 1988; Kaplan et al 1990).

As an advocate for a version of chromatic/achromatic dualism and parallelism, I here review the evidence for both sides in this ongoing debate. However, while trying to do justice to the single achromatic/chromatic channel hypothesis, I show why the idea of separate parallel neural channels is more appealing. The channels probably do not correspond exactly with the achromatic and chromatic channels of psychophysics, and they probably interact more than some theories predict. Nevertheless, there is good reason to believe there are two separate pathways carrying different kinds of signals about the appearance of the outside world. Much of the evidence is neurophysiological, but there are also compelling results from studies of motion, contour perception, and the visual consequences of diseases of the retina and optic nerve. For a somewhat different point of view, the reader should consult the chapter by Lennie et al (1989).

In the literature discussed in this review, authors frequently apply a neurophysiological result from the study of monkeys to human perception, and vice versa. This requires the strong assumption that the visual pathways in humans and monkeys function in a very similar way. Support for this assumption comes mainly from the work of R. L. DeValois and his colleagues (DeValois et al 1974a,b). They showed that for Old-World monkeys, such as the rhesus or cynomolgus monkeys generally used in neurophysiological experiments on vision, detailed behavioral measurements of the spectral

sensitivity function, wavelength discrimination function, and contrast sensitivity function resemble those in humans. The neuroanatomy of the human retinocortical pathway is similar to that of Old-World monkeys. More recent evidence on similarities in detailed structure and layout of the retina in human and macaque monkeys strengthens the argument for functional similarity (Rodieck 1988). Moreover, while cone photoreceptors are only the beginning of the pathway, evidence on the detailed quantitative similiarity of the spectral sensitivity curves of these receptors in humans and macaque monkeys (Baylor et al 1987; Schnapf et al 1987) reinforces the idea of cross-species similarities in visual function. The evidence for similarity of visual function concerns Old-World monkeys (e.g. the different macaque species) and does not apply to New World monkeys (e.g. squirrel monkeys). The direct relevance of the elegant work on the neuroanatomy and neurophysiology of the squirrel monkey visual system to human vision is at present problematical.

P (Parvocellular) and M (Magnocellular) Pathways

The story about parallel channels for color and brightness really begins in the layering of the lateral geniculate nucleus (LGN). For many years there was a mystery about the multilayered structure of the LGN of Old World primates, including humans (Walls 1942). In the main body of the Old World primate's LGN there are six clearly segregated layers of cells. The four more dorsal layers are composed of small cells and are named the parvocellular layers. The two more ventral layers, composed of larger neurons, are called magnocellular layers. Recent work on functional connectivity and the visual function of single neurons has revealed that the different types of cell layers in the LGN receive afferent input from different types of retinal ganglion cells. The evidence on functional connectivity of retina to LGN comes from Leventhal et al (1981) and Perry et al (1984), who labeled axon terminals in specific LGN layers of the macaque monkey with horseradish peroxidase (HRP) and looked back in the retina to see which ganglion cells were labeled retrogradely. Direct electrophysiological evidence about retina-to-LGN connectivity comes from Kaplan & Shapley (1986), who recorded excitatory synaptic potentials (from retinal ganglion cells) extracellularly in different LGN layers and found that different types of retinal ganglion cell drove different LGN layers. For example, LGN cells that are excited by deep blue (short-wavelength) light are only found in the parvocellular layers. These "blue-excitatory" LGN cells receive excitatory synaptic input from "blue-excitatory" ganglion cells; "blue-excitatory" ganglion cells provide direct excitatory input only to parvocellular LGN neurons of the "blue-excitatory" type. The specificity of ganglion cell types exactly matches that of their LGN targets. Our direct evidence about this issue confirmed the earlier correlative

results of DeValois et al (1966) and Wiesel & Hubel (1966) in the LGN, and Gouras (1968), DeMonasterio & Gouras (1975), and Schiller & Malpeli (1977) on retinal ganglion cells.

As discussed below in more detail, parvocellular neurons are color opponent. This means that their responses, to stimuli that fill their entire receptive fields, change sign from excitatory to inhibitory contingent on the wavelength of the stimulating light (DeValois et al 1966). The property of color-opponency is conferred on them by their ganglion cell inputs (Gouras 1968; Schiller & Malpeli 1977; Kaplan & Shapley 1986), from the class of ganglion cells called P cells by Shapley & Perry (1986). From the neuroanatomical work, one may infer that P cells are very numerous and densely packed, with small cell bodies and dendritic trees.

Magnocellular neurons are generally thought to give the same sign of response to all wavelengths of light; this property is referred to as broad-band spectral sensitivity (Gouras 1968; Schiller & Malpeli 1977). However, only some (about half) of the magnocellular cells are truly broad band; the other magnocellular neurons are color opponent by the above definition. These are the cells Wiesel & Hubel (1966) called Type IV. They have an excitatory receptive-field center mechanism that is broad band, and an antagonistic inhibitory surround mechanism that is selectively sensitive to long-wavelength red light. The properties of the magnocellular neurons, both broad-band and Type IV, are determined almost completely by their retinal ganglion cell inputs (Kaplan & Shapley 1986). The HRP experiments of Leventhal et al (1981) and Perry et al (1984) showed that magnocellular cells receive input from a class of retinal ganglion cells somewhat larger in cell body size and dendritic extent than P cells. This group of ganglion cells was labeled M cells by Shapley & Perry (1986).

Contrast Gain in M and P Pathways

Besides their spectral sensitivities, the other property that distinguishes parvocellular from magnocellular neurons is contrast gain. In vision research contrast denotes the variation in the amount of light in a stimulus, normalized by the mean amount of light. For example, in a periodic grating pattern in which the peak amount of light is P and the least amount of light is T (for trough), then contrast is defined as, $C = (P - T)/(P + T)$. This definition goes back to Rayleigh (1889) and Michelson (1927). Contrast is the stimulus variable that the retina responds to under photopic conditions (Robson 1975; and many others reviewed in Shapley & Enroth-Cugell 1984). It is thought that such response-dependence on contrast evolved because the contrasts of reflecting objects are invariant with changes in illumination occasioned by shadows, weather, or the passage of the sun. The retina thus sends signals to

the brain that are more closely linked to surface properties of reflecting objects than to variations in illumination.

Contrast gain is defined as the change in response of the neuron per unit change in contrast, in the limit as the contrast goes to zero. Contrast gain is thus the differential responsiveness of the neuron to contrast around the operating point of the mean illumination. The different contrast gains of parvocellular and magnocellular LGN neurons are illustrated in Figure 1 (Shapley & Kaplan, unpublished; compare with retinal ganglion cells in Kaplan & Shapley 1986). As can be seen from the figure, the response as a function of contrast grows much more steeply for the magnocellular neuron than for the parvocellular, especially at low contrast near the behavioral detection threshold. This is a general finding. The ratio of the average contrast gains of the population of magnocellular neurons to the population average of parvocellular neurons is approximately eight under mid-photopic conditions (Kaplan & Shapley 1982; Hicks et al 1983; Derrington & Lennie 1984). Subsequently, Ehud Kaplan and I showed that this contrast gain difference in LGN neurons is already set up in the retina. The retinal ganglion cells that innervated magnocellular neurons had eight times the contrast gain of ganglion cells that provided the excitatory drive for parvocellular LGN neurons (Kaplan & Shapley 1986).

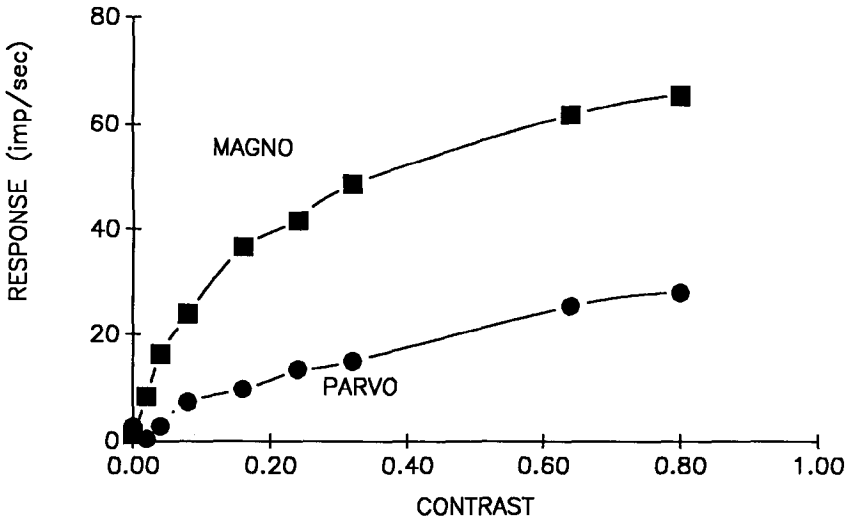

Figure 1 Responses of macaque LGN neurons as a function of contrast. One on-center magnocellular neuron and one off-center (+g−r) parvocellular neuron are shown. Mean luminance was 60 cd/m^2. Responses were calculated as the best-fitting Fourier component at 4 Hz, the temporal frequency of the drift.

We still do not know the mechanistic reason for the substantial differences in contrast gain for cells in the two pathways. Various factors may contribute. The receptive field centers of P cells are smaller than those of M cells, and if the local contrast gains from points in each field are equal, then the larger summing area of the M cells would lead to a higher contrast gain for an optimal sine grating pattern (see Enroth-Cugell & Robson 1966). Though this factor must contribute something, it does not seem to account for all the differences between M and P. In P cells, but not M cells, antagonistic interactions may occur between cone types within the receptive field center. Though this may be the case in many neurons, it is possible to find P cells in which the center is driven predominantly by one cone type only. Both these hypotheses are considered in the review by Kaplan et al (1990). Neither is sufficient to account for all the difference between M and P contrast gains. This is a problem that needs more research. Whatever the complete explanation is, it must involve retinal mechanisms, since the M and P differences in contrast gain begin in the retina.

Next, we must consider in more detail the responses of P and M neurons to chromatic stimuli. This discussion requires a prior analysis of the three cone photoreceptors in the Old World primate retina, and the effect of the properties of the cones on chromatic responses.

Three Photoreceptors and Spectral Sensitivity

Discussion of the spectral sensitivities of the photoreceptors must precede consideration of the chromatic properties of P and M pathways and the chromatic sensitivity of the human observer.

There are three cone photoreceptor types in human and macaque retinas. The spectral sensitivities of these photoreceptors have been determined for macaque retina by Baylor et al (1987) and for human retina by Schnapf et al (1987), using suction electrodes to measure cone photocurrent directly. These direct measurements of photoreceptor spectral sensitivities are in generally good agreement with microspectrophotometric measurements of cone absorption spectra (Bowmaker & Dartnall 1980; Bowmaker et al 1980). The photocurrent measurements agree even more closely with estimates of cone spectral sensitivity based on human psychophysics (Smith & Pokorny 1975). The Smith & Pokorny fundamentals (estimated cone spectral sensitivities as measured at the retina after the light has been prefiltered by the lens) are three smooth functions of wavelength peaking at 440 nm (b cones), 530 nm (g cones), and 560 nm (r cones).

The human sensitivity to light across the visible spectrum under photopic, daylight conditions is called the *photopic luminosity function,* denoted V_λ. It might be thought that the easiest, and certainly the most straightforward, way to determine V_λ would be to measure psychophysically the sensitivity for

increments of light of different wavelength on a photopic background. However, the photopic luminosity function is not measured in this way, mainly because such measurements are variable between and within observers because of the complexity of the visual system (Sperling & Harwerth 1971; King-Smith & Carden 1976). Rather, the procedure known as heterochromatic flicker photometry has been employed. Monochromatic light of a given wavelength is flickered against a white light at a frequency of 20 Hz or above, and the radiance of the monochromatic light is adjusted until the perception of flicker disappears or is minimized (Coblentz & Emerson 1917). This technique exploits the fact that neural mechanisms that can respond to the color of the monochromatic light are not able to follow fast flicker. The photopic luminosity function has been measured more recently using contour distinctness (Wagner & Boynton 1972) and minimal motion (Cavanagh et al 1987) as response criteria. These measurements agree remarkably well with the luminosity function determined by flicker in the same subjects.

The *luminance* of a light source is its effectiveness in stimulating the visual neural mechanism that has as its spectral sensitivity the photopic luminosity function. Thus, the luminance of any light may be computed by multiplying its spectral radiance distribution, wavelength by wavelength, by the photopic luminosity function, and summing the products.

The spectral sensitivities of the r and g cones and the photopic luminosity function are graphed in Figure 2. The purpose of this graph is to show the degree of overlap of the two longer-wavelength cones with the photopic

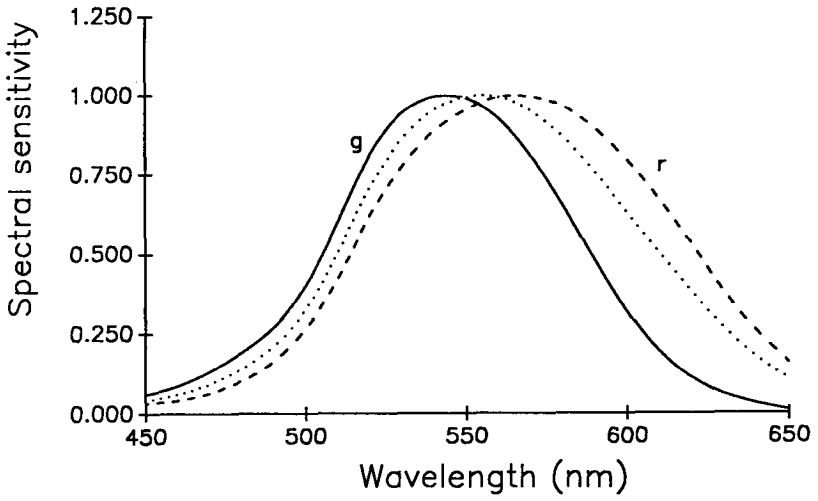

Figure 2 Spectral sensitivity functions of the r and g cones, and the photopic luminosity function (dotted line). Data are redrawn from Smith & Pokorny (1975).

luminosity function, and also to demonstrate the closeness of the luminosity function to the r cone sensitivity especially at longer wavelengths. This becomes significant in the consideration of cone contrasts in chromatic, isoluminant stimuli. The photopic luminosity curve graphed in Figure 2 is an average of curves from many subjects. There is substantial variation in the normal population in the peak wavelength and particularly in the long-wavelength limb of the V_λ curve (Coblentz & Emerson 1917; Crone 1959). For example, some people who have normal color vision can have half a log unit less relative sensitivity to 620 nm light than the average observer (Coblentz & Emerson 1917). There is variance also in the reported spectral sensitivity of cones (Baylor et al 1987) and in the pigments' spectral absorption (Bowmaker et al 1980).

Color Exchange and Isoluminance

Color exchange, or silent substitution (Estevez & Spekreijse (1974, 1982), is a technique for identifying contributions from particular photoreceptors or spectral response mechanisms. For any spectral sensitivity function, and any two lights with different spectral distributions within the band of the sensitivity function, one can perform a color-exchange experiment that will provide a characteristic color balance for that particular spectral sensitivity. For example, if one chooses two monochromatic lights with wavelengths such that they are equally effective at stimulating the r cone, then temporal alternation between these two lights at equal quantum flux should cause no variation in the response of the r cone. The same argument works for the photopic luminosity function which presumably is the spectral sensitivity of a neural mechanism that receives additive inputs from r and g cones. Two lights that, when exchanged, produce no response from the luminance mechanism are called *isoluminant*.

The results of a simulated color-exchange experiment on cones and a broad-band cell with a V_λ spectral sensitivity are illustrated in Figure 3. The calculations are based on the spectral sensitivities of the r and g cones and the photopic luminosity function as graphed in Figure 2. The spectral distributions of the light sources were those of the red and green phosphors on standard color television sets, designated P22 phosphors. The red phosphor is narrow-band centered around 630 nm. The green phosphor is more broad band centered around 530 nm. Such colored lights have been used in many of the experiments reviewed here (Derrington et al 1984; DeValois & Switkes 1983; Kaplan et al 1988; Livingstone & Hubel 1987; Tootell et al 1988b). The experiment simulated is color exchange between the red (R) and green (G) phosphors. The G/R ratio is the ratio of the luminances of the green and red phosphors. In this simulated experiment, the luminance of the red phosphor (R) was held fixed and the luminance of G was varied. When the luminance of

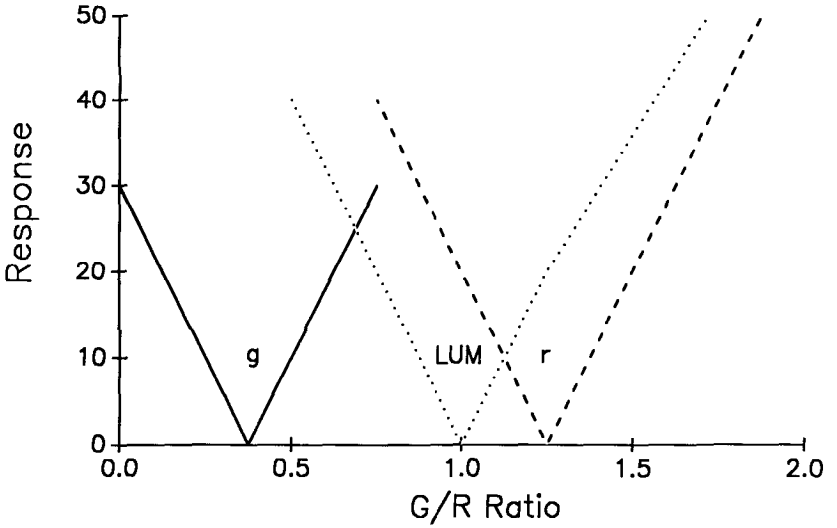

Figure 3 Color exchange response functions for r and g cones and luminance. The predicted response of the cones to different G/R ratios was calculated from the cross-product of the G and R phosphors with the spectral sensitivities of the g and r cones from Figure 2. In the calculation, contrast of the R phosphor was fixed at 0.8. Contrast of the G phosphor varied so as to change the G/R ratio.

the green phosphor is approximately 0.4 that of the red (G/R ratio 0.4), the response of the g cones is nulled. When the G/R ratio is about 1.2, the r cone response is nulled.

Notice that the shape of the response of each of these spectral mechanisms is similar; near the null the response vs G/R ratio forms a V. This is based on the assumption of small signal linearity, a good assumption in the case of macaque P and M pathways (Kaplan & Shapley 1982; Derrington et al 1984; Blakemore & Vital-Durand 1986). A spectral mechanism that sums the responses of g and r cones will have a null in a color exchange experiment at a G/R ratio between the nulls of the two cones. If the spectral sensitivity of the summing mechanism is Kr + g, where K is a number between zero and infinity, then when K approaches zero, the color-exchange null approaches the g cone from above. When K goes to infinity, the color-exchange null approaches the r cone null, from below. The null of the luminosity curve between the cone nulls in Figure 3 is a case in point. For that curve K is approximately 2. One must qualify the assertion to include the condition that the photoreceptor signals have the same time course, and that in the process of summation their time courses are unaffected. The existence of sharp Vs in color exchange experiments on M ganglion cells and magnocellular cells is

reasonably good evidence that r and g cones have similar time courses under the conditions of those experiments (Lee et al 1988; Kaplan et al 1988; Shapley & Kaplan 1989).

Next, we consider what happens in a color-exchange experiment on a color-opponent neuron. In such a cell, r and g cone signals are not summed but subtracted. The results of Figure 4 would ensue. The luminosity color-exchange results are included for comparison with three different possible color-opponent cells: one in which the strength of r and g signals is equal but the sign is opposite (g − r); one in which signals from g cones are twice as strong as those from r cones (2g − r); and one in which signals from r cones are twice as strong as those from g cones (2r − g). The curves would be unaffected if the signs of the cone inputs were reversed since only magnitude of response is plotted. What is striking about these simple calculations is that opponent neurons have no null response between the cone nulls along the G/R axis. The g − r response is perfectly constant. The 2g − r and 2r − g cells show response variation but no null. This result is general for any neural mechanism with a spectral sensitivity Kr − g, where K is a number greater than zero and less than infinity. As K goes to zero the null of the mechanism approaches the g cone null from below; as K goes to infinity, the null of the mechanism approaches the r cone null from above. As before, all these

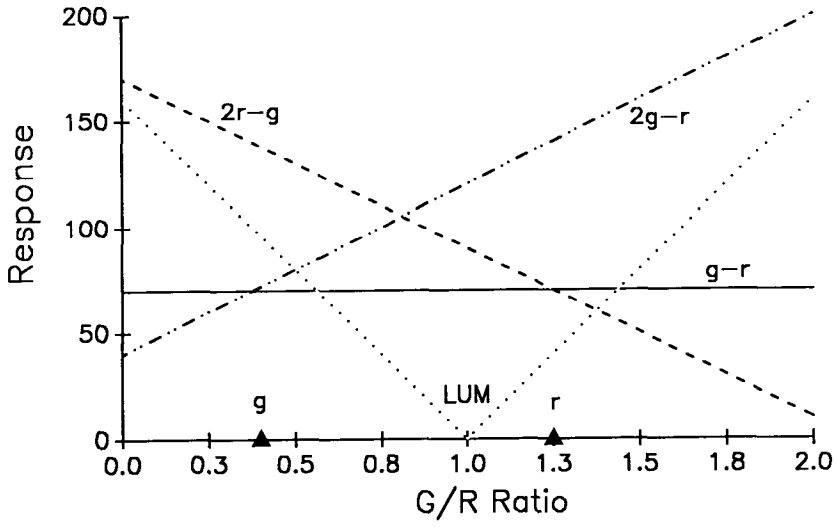

Figure 4 Color-exchange functions for opponent cells compared to luminance. Response magnitude as a function of G/R ratio is plotted for three different opponent neurons, with cone balances as labeled. The color-exchange response function for luminance is again shown (labeled LUM). As in Figure 3, calculations were done with fixed contrast on the R phosphor and varying contrast on the G phosphor.

statements hinge on small-signal linearity and identity of temporal response properties for g and r cones. Similarity of response time courses in r and g cones was found in parvocellular color-opponent neurons by Gielen et al (1982), who used color-exchange to isolate responses of the different cones. There have been several demonstrations of small-signal linearity in P and parvocellular neurons (Shapley et al 1981; Kaplan & Shapley 1982; Derrington & Lennie 1984; Derrington et al 1984).

Responses of M and P Neurons to Isoluminant Stimuli

One particular color-exchange experiment has become crucial, namely measuring responses of P and M neurons to isoluminant color exchange. In their large paper on perceptual effects of parallel processing in the visual cortex, Livingstone & Hubel (1987) assumed that because magnocellular cells were broad band, their responses would be nulled at isoluminance. As the above discussion demonstrates, this is a non sequitur. To repeat, there could be a whole family of broad-band neurons in the visual pathway that summed signals from r and g cones with different weighting factors K_i, such that spectral sensitivity of the i-th mechanism was $K_i r+g$. Each mechanism would have a null at a different point on the G/R axis. The striking thing about M cells and magnocellular neurons is that, for stimuli that produce responses from the receptive-field center mechanism, the position of the null on the color-exchange axis is close to that predicted from the human photopic luminosity function, V_λ (Lee et al 1988; Shapley & Kaplan 1989; Kaplan et al 1990). There is no more variability in the position of the color-exchange null in the neurophysiological data than there is in psychophysical experiments on the luminosity function in humans (Crone 1959) or in behavioral experiments on macaques (DeValois et al 1974a). A crucial experiment would be to measure the variability of the isoluminant point within a large population of M cells from the same individual monkey, but this is so difficult it has not yet been done.

There are other experiments that indicate that, under stimulus conditions where the center of the receptive field is not the only response mechanism contributing to the response, M and magnocellular neurons do not have a color-exchange null at isoluminance. Lee et al (1988) reported that large disks that stimulate center and surround have nulls away from isoluminance. Shapley & Kaplan (1989) used heterochromatic sine gratings to study chromatic properties of receptive field mechanisms. Heterochromatic sine gratings are formed by producing a sine grating on, say, the red phosphor of a color monitor, and producing an identical sine grating on the green phosphor except for an exact 180° phase shift. Thus where the red phosphor has a bright red bar the green phosphor has a dark green bar, and vice versa. The sum of these two grating patterns in antiphase yields as a spatial pattern a red-green,

ergo heterochromatic, grating. Shapley & Kaplan (1989) reported that heterochromatic sine gratings of low spatial frequency may produce no color null in magnocellular neurons. Derrington et al (1984), using the technique of modulation in color space (discussed below), found that many magnocellular units exhibited properties expected of color-opponent cells. Undoubtedly, all these results are related to the earlier work of Wiesel & Hubel (1966), who found that many magnocellular neurons had a receptive-field surround that was more red sensitive than the receptive-field center. Such neurons could behave as color-opponent cells to stimulate that covered both center and surround if the spectral sensitivities of center and surround were different enough. Similar M ganglion cells were reported by DeMonasterio & Schein (1980). Thus, in psychophysical experiments, if the stimulus is designed to tap the receptive-field center of cells in the M pathway, it will elicit a spectral sensitivity function like V_λ. Such a stimulus will be nulled in a color-exchange experiment at isoluminance. However, should other stimuli be detected by the M-magnocellular pathway but not isolate the central receptive-field mechanism, one might discover a color-opponent mechanism driven by M cells.

There is another result that indicates a failure of nulling at isoluminance in magnocellular neurons. This is the second-harmonic distortion discovered by Schiller & Colby (1983). In color exchange experiments with large-area stimuli, these investigators often found strong frequency-doubled responses. Such results were not reported by Derrington et al (1984), who found frequency doubling rarely (20% of the time) in their experiments. Shapley & Kaplan (1989) reported that frequency doubling was dependent on spatial frequency of the pattern used for color exchange. Center-isolating stimuli elicited no frequency doubling; but it could be observed when spatial frequency was so low, less than 0.5 c/deg, that the receptive-field surround could contribute to the M cell's response. This also could contribute to failure to achieve sharp psychophysical isoluminance with stimuli of large area or low spatial frequency, even with stimuli that isolated a perceptual mechanism driven only by the M pathway.

Chromatic Opponency in P and M Cells

The basis for wavelength selectivity in the visual pathway is antagonistic (excitatory vs inhibitory) interactions between signals from different cone types. The simplest type of antagonism is subtraction. There is good evidence for subtractive interactions between r and g cones on P ganglion cells (DeMonasterio & Gouras 1975; Zrenner & Gouras 1983) and parvocellular neurons (DeValois et al 1966; Wiesel & Hubel 1966; Derrington et al 1984). The classical evidence is a change in sign of response with wavelength

(DeValois et al 1966). For example, many P cells that receive opponent inputs from r and g cones have a sign change at a wavelength near 570 nm. The "blue-excitatory" cells referred to above often have a change from excitation at short wavelengths to inhibition at long wavelengths at around 490 nm. These cells receive excitatory input from b cones and inhibitory input from some combination of r and g cones.

The precise mapping of cone types to receptive-field mechanisms is a problem not yet solved. Wiesel & Hubel (1966) postulated that color-opponent cells received excitatory (or inhibitory) input from one cone type in the receptive-field center and antagonistic inputs from a complementary cone type in the receptive-field surround. However, the detailed quantitative evidence that would be needed to support or to reject this hypothesis was not available then, and it is still not in hand today. One problem is spatially isolating center from surround: Receptive fields in the monkey's retina, and presumably in the human's too, are quite small. Though Wiesel & Hubel's (1966) proposal may be correct, there are a number of other possibilities. One alternative hypothesis is that there is mixed receptor input to the receptive-field surround, and only or predominantly one cone input to the center of the receptive field (see Kaplan et al 1990).

Some fascinating facts are known about the proportions of color-opponent P and parvocellular cells that have r cone centers and g cone centers. DeMonasterio & Gouras (1975) found that the majority of P ganglion cells in the central 5° of the visual field had g cone centers. The g cone input might be excitatory or inhibitory. The proportion of P cells with r cone centers increased with retinal eccentricity, as later confirmed by Zrenner & Gouras (1983). A similar finding about the high proportion of g cone centers among central parvocellular neurons in LGN was reported by DeValois et al (1977). This is worth dwelling on for a moment, especially because the finding of DeValois et al (1977) was apparently later misinterpreted by Ingling & Tsou (1988). DeValois et al (1977) stated that +g−r opponent cells had excitatory centers; thus the excitatory g cone input was to the center. They also wrote that +r−g neurons had inhibitory centers. This means again that the g cone input went to the center of the receptive field, as inhibition. Ingling & Tsou (1988) seemed to take this to mean that the neurons with r cone input to the center had inhibitory centers, a misinterpretation of the data. The three studies cited all concur that in central vision there is a preponderance of P cells, and parvocellular neurons, with g cone input to the center of their receptive fields. Ingling & Martinez-Uriegas (1983) had earlier used this fact to explain the hue shift towards green of a flickering yellow light.

The reason that the proportion of P cells driven by g cones is significant is that the M cell centers are dominated by r cones, and the difference in cone connectivity to the different pathways may illuminate functional specializa-

tion. Referring back to Figures 2 and 3, we see that the V_λ function lies closer to the r cone spectral sensitivity. The V_λ isoluminant point is at a relative cone weight of 2:1 for r to g cones. From color-exchange experiments on macaque M cells we can infer that the cone weighting is about 2r for every g cone signal for the M cell center. This bias in favor of the r cones in the M pathway seems to be the opposite of the g cone bias in the centrally located P cells.

The diminution in relative numbers of those P cells with g cone receptive-field centers at increasing retinal eccentricity is associated with a decline in perceived saturation of colors of stimuli presented to the periphery of the visual field (Gordon & Abramov 1977). There is evidence against the idea that this shift to the r cones in P cells occurs because of an increasing proportion of r cone photoreceptors with eccentricity (reviewed in Shapley & Perry 1986). Rather, the r shift appears to be a result of eccentricity-dependent shifts in cone-to-P cell connectivity.

Modulation in Color Space

Chromatic opponency of LGN cells has been investigated using a technique very similar to color exchange, namely modulation in color space around a white point. This technique grew out of psychophysical investigations of chromatic opponent mechanisms.

The color space that is used is a re-mapping of cone excitation space. Any spectral distribution over the visible spectrum can be represented as a three-dimensional vector of cone excitations. The three coordinate axes in this vector space are b, r, and g cone excitation by the light. Based on earlier work of MacLeod & Boynton (1979), Krauskopf et al (1982) proposed a (linear) mapping of this space into another color space in which the axes were luminance modulation, b excitation (Constant R and G), and r and g modulation such that b cone excitation was constant (Constant B axis). The Constant B and Constant R and G axes formed a plane, the Isoluminant Plane. These axes would be preferred modulation directions for color-opponent mechanisms: Lights along the Constant B axis would stimulate cells that received $+r-g$ or $+g-r$ input, while the Constant R and G axis would isolate those lights that only excited cells that received excitation (or inhibition) from b cones. Krauskopf et al (1982) demonstrated that these three axes were preferred axes for habituation of the response to chromatic flicker. Krauskopf et al (1982) named these axes "cardinal directions of color space." It is important to note that the transformation from r,g,b space to cardinal direction (CD) space is a linear transformation but angles are not preserved. Thus, the cone vectors which are all orthogonal in r,g,b space are no longer orthogonal in CD space. The vectors for r and g cones are about 45 deg from the b cone vector in CD space (Derrington et al 1984). In CD space, the r and g cone vectors are

only 10–20° apart and are mapped close to the luminance axis (Derrington et al 1984)—i.e. almost orthogonal to the isoluminant plane.

Derrington et al (1984) used stimuli modulated along different vectors in this CD space to characterize macaque LGN neurons. Modulation in the isoluminant plane should have been ineffective in stimulating neurons with a spectral sensitivity like V_λ, the photopic luminosity function. Each neuron should have a null plane, like the isoluminant plane for luminance units, within which color modulation should be ineffective. The elevation of this null plane with respect to the isoluminant plane is a measure of the degree to which the neuron's response is determined by opponent mechanisms. The closer to zero the elevation, the more nearly the neuron's response is completely determined by luminance. Since the cone vectors are pointing so close to the luminance direction in CD space, neurons that are being driven by either the r or the g cone will have a null plane near the isoluminant plane, with a low elevation. Derrington et al (1984) used the position of the null planes in CD space for each neuron to calculate cone weighting factors for each neuron studied. They also measured the effects of spatial and temporal frequency on these derived cone weights. They found that virtually every parvocellular neuron was color opponent in that at least two cone weights were of opposite sign; that temporal frequency up to 16 Hz had little effect on the position of null planes and thus cone weights; that increasing spatial frequency had a marked effect in lowering elevation of null planes, thus reducing the strength of the cone weight from the receptive field surround; and that magnocellular responses to large-area stimuli were often color opponent, but their null planes were pushed down towards zero elevation by grating stimuli, as the V_λ spectral sensitivity of the receptive-field center was revealed.

Comparison of Achromatic and Chromatic Contrast Sensitivity

The spatial characteristics of vision have been studied for many years by measuring the contrast sensitivity function for sinusoidal gratings (e.g. Campbell & Robson 1968; DeValois et al 1974b, among many others). These have traditionally been achromatic measurements, and the contrast sensitivity has been taken to be the reciprocal of the luminance contrast at psychophysical threshold. More recently, luminance contrast sensitivity has been compared with the spatial frequency dependence of chromatic contrast sensitivity as measured with isoluminant heterochromatic grating patterns (van der Horst et al 1967; Kelly 1983; Mullen 1985). An example of the kind of results obtained is shown in Figure 5 from Mullen's (1985) paper. The luminance contrast sensitivity function is band-pass while the chromatic contrast sensitivity is low pass and cuts off at a fairly low spatial frequency compared with

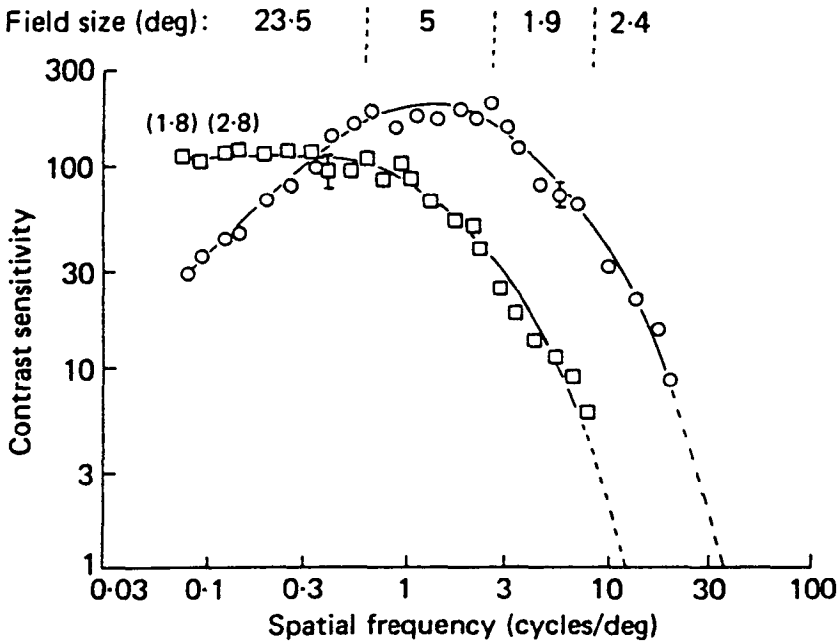

Figure 5 Contrast sensitivity functions for luminance and isoluminant color gratings. The luminance data are drawn as empty circles; the red-green grating data are drawn as empty squares. The luminance data were taken with a cathode ray tube (CRT) display filtered through a 526 nm narrow-band filter, while the red-green data are for an isoluminant sine grating where the red member of the antiphase pair was produced by a CRT filtered through a 602 nm narrow-band filter, while the green grating was filtered through the same 526 nm filter as for the luminance grating. The field size at the top of the graph indicates the size af the stimulus screen, in degrees of visual angle, for the various measurements. Reproduced with permission from Mullen (1985).

luminance. Thus, at this mean luminance, the subject could resolve 30 c/deg with the luminance system but only about 10 c/deg with the chromatic system. Parvocellular neurons respond much better to isoluminant heterochromatic gratings of low spatial frequency because, under those conditions, the antagonistic center and surround become synergistic (DeValois & DeValois 1975). However, Type IV M cells also become more sensitive at low spatial frequencies of heterochromatic gratings because of their color opponency.

The responses to middle and high spatial frequencies are better when luminance than when isoluminant gratings are used as stimuli as in Figure 5. Thus, if the data were plotted as response vs G/R ratio, one should expect a dip in response near isoluminance. Such results were reported by Mullen (1985). It would be important to measure the isoluminant G/R ratio on the same subject with heterochromatic flicker photometry or minimal motion or

minimally distinct border to see whether the same or different spectral mechanisms are at work in detecting the heterochromatic gratings.

K. K. DeValois & Switkes (1983) and Switkes et al (1988) have demonstrated that heterochromatic grating patterns are detected by spatial frequency channels like those involved in achromatic grating detection (Campbell & Robson 1968; Graham 1980). Thus, elevation of threshold for detecting an isoluminant grating is produced by preexposure to an isoluminant grating of the same spatial frequency, and less elevation of threshold is produced by more distant spatial frequencies. Moreover, color gratings mask and adapt color and luminance gratings but, as discussed below, luminance gratings may facilitate detection of color gratings.

The work on spatial frequency channels in color throws a new light on receptive-field models that have sought to explain chromatic and luminance spatial contrast sensitivity functions in terms of single channel receptive field models (Kelly 1983; Rohaly & Buchsbaum 1988, 1989). The chromatic contrast sensitivity function is an envelope of chromatic spatial frequency channels, just as the luminance contrast sensitivity function is thought to be an envelope of the well-studied achromatic spatial frequency channels. Single-channel models, though they may be of heuristic value in summarizing a body of data, must be only a first approximation to a true mechanistic model of these multichannel systems.

One recent paper about the spatial properties of chromatic spatial channels may advance our understanding of the peculiar contribution of color to spatial vision (Troscianko & Harris 1988). These authors estimated the spatial phase sensitivity in compound sine gratings that were the sum of a fundamental component and its third harmonic, both set at twice detection threshold. Phase discrimination at isoluminance was worse than for all other color balances tested. The authors hypothesize that color information comes into the cortex with a great amount of positional uncertaintly and that this leads to losses in phase discrimination when only color is available as a stimulus.

Possible Neural Substrates for Contrast Sensitivity

The M and P pathways must be the conduits for signals about detection of contrast. The high-gain M system is well suited to handle detection of grating patterns with low to medium spatial frequencies (Shapley & Perry 1986; Kaplan et al 1990). The numerous P cells may be required to represent veridically the spatial waveform for grating patterns near the acuity limit (Lennie et al 1989).

Recent neurophysiological results by Purpura et al (1988) indicate that the P cells become visually unresponsive to grating patterns when the mean luminance drops below 0.1 cd/m^2, at the rod/cone break. M cells become less sensitive progressively as mean luminance is reduced, but they remain responsive into the scotopic range. We suggested that these results might mean

that spatial vision under scotopic conditions would be dependent on M cell signals. Wiesel & Hubel (1966) and Gielen et al (1982) reported rod-driven responses in parvocellular LGN cells under scotopic adapting conditions, an apparent contradiction to the results of Purpura et al (1988). However, both these sets of authors reported that a rod-driven parvocellular neuron was rarely encountered; moreover, they did not test for spatial vision under scotopic conditions. In the Purpura et al study, we did observe rod-driven responses in P cells but only with very low spatial frequency gratings or diffuse light as spatial stimuli.

Cortical Target Areas For P and M Signals

V1 There is indirect evidence that magnocellular and parvocellular signals are kept somewhat segregated within striate cortex, V1. Hawken & Parker (1984) and Hawken et al (1988) have shown that cortical neurons with high contrast gain, like magnocellular neurons, can be found in layer IVc α of V1. Color-opponent neurons are located in layer IVc β, and these are presumably the targets of the LGN afferents from parvocellular cells.

There are subdivisions within the upper layers of the cortex, layers II and III, that may be preferentially influenced by magnocellular signals. All of layers II and III receive inputs from layer IVc β, so, presumably receive parvocellular signals filtered through the cortical network. However, from experiments of labeling of active cells with 2-deoxyglucose, Tootell et al (1988a) found that there was weak but significant labeling of the cytochrome oxidase blobs in layers II and III of V1 cortex when low-contrast stimuli were used. The cytochrome oxidase blobs were shown by Livingstone & Hubel (1984) to contain cortical neurons broadly tuned for orientation. Tootell et al's (1988a) finding may mean that magnocellular and parvocellular signals converge onto blob neurons.

The cytochrome oxidase blobs have been found to form a network throughout macaque V1 (Horton 1984; Livingstone & Hubel 1984), and it has been hypothesized that they form a separate system for the analysis of color (Livingstone & Hubel 1984, 1987). Many of the cells in the blobs are color selective. The real test of this idea is whether cells in the inter-blob regions of layers II and III of V1 are not color selective or are substantially less color selective than blob neurons. There are recent single-unit data on this question from Lennie et al (1989b), and the results indicate that color selectivity in blob cells is not different from that in inter-blob cells. Furthermore, Tootell et al (1988b) used isoluminant color gratings to label layer II-III cells with 2-deoxyglucose; labeled cells were found throughout the upper layers, though there was stronger labeling of the blobs with diffuse color patterns. These data are essentially consistent with the findings of Lennie et al (1989b).

v2 Using cytochrome oxidase as a marker, Tootell et al (1983) demonstrated stripe-like structures in secondary visual cortex V2 in macaque monkeys. Subsequently, Shipp & Zeki (1985) and DeYoe & Van Essen (1985) have shown that distinct anatomical regions within primary visual cortex make characteristic connections with regions in macaque V2. Neurons in the blobs of V1 are connected to one of the sets of darker stripes in V2; neurons in the inter-blob regions of layers II and III are connected to stripe-like regions of low cytochrome-oxidase staining in V2. Livingstone & Hubel (1987), from their measurements in squirrel monkeys, also propose that layer IVb, which receives magnocellular signals from layer IVc alpha, projects to the alternating dark cytochrome stripes in macaque V2. The functional consequence of this complex sequence of connection is that parallel functional pathways proceed from V1 to V2. Livingstone & Hubel (1987; 1988) have made a detailed psychophysical linking proposition based on this anatomy and the receptive-field properties of neurons in V2. They propose that blob cells, connecting to one set of V2 stripes, constitute a system for color vision. The putative magnocellular pathway from layer IVc α through layer IVb to the other set of dark V2 stripes is supposed to be important for responding to objects in depth. The interblob neurons in V1, connected to pale stripes in V2, are supposed to be important for form vision, mainly because neurons located in pale stripes in V2 were found to be end stopped—i.e. more strongly responsive to corners and the ends of lines than to long contours (Hubel & Livingstone 1987).

Among the psychophysical proposals discussed by Livingstone & Hubel (1987), one particularly attractive idea is that magnocellular signals form the basic excitatory drive of the motion pathway.

Motion

Motion perception is greatly disturbed at isoluminance. Heterochromatic color gratings appear to move more slowly (Cavanagh et al 1984; Livingstone & Hubel 1987). Apparent motion is greatly reduced or abolished (Ramachandran & Gregory 1978; Livingstone & Hubel 1987). However, Livingstone & Hubel (1987) state that they observed reduction in apparent motion at a G/R ratio that was 20% less than the G/R ratio for isoluminance determined with flicker photometry. This is significant because it may indicate that contrast in a cone mechanism, or some other neural mechanism than the specific V_λ mechanism, is being selected in these experiments. Many experiments on isoluminant vision have been designed with random dot kinematograms (Ramachandran & Gregory 1978) or random dot stereograms (Livingstone & Hubel 1987). These may all be subject to artifacts as a result of chromatic aberration (Flitcroft 1989). Chromatic aberration may affect spatial frequen-

cies as low as 4 c/deg; it certainly may affect experiments with random dot patterns, which will be broad band in spatial frequency.

Cavanagh et al (1987) used a minimum-motion technique to estimate the cone inputs to the motion mechanism as well as to determine spatial and temporal tuning of the motion pathway. One of their chief findings was that b cones provide little input to the motion pathway. Furthermore, minimum motion and flicker photometry give virtually the same isoluminant point for a given pair of colored lights. This is strong evidence for a single pathway with a single spectral tuning curve, as would be the case if M signals were the front end for the motion signal. However, there is a motion response to isoluminant stimuli; the motion system just signifies a lower velocity. Furthermore, evidence from motion aftereffects (Cavanagh & Favreau 1985; Mullen & Baker 1985) also indicates there may be some, albeit weaker, inputs from color-opponent signals to the motion pathway. There are many sites along the visual pathway at which interactions may occur (see below) and where a magnocellular signal might be modulated by parvocellular signals before it reached the site of motion perception. The evidence for parvocellular inputs involves suprathreshold motion. I have some preliminary evidence that, at motion threshold, isoluminant stimuli are particularly ineffective.

Interactions between M and P Pathways

The evidence reviewed so far has shown the remarkable independence of P and M pathways as they travel in parallel to cortex from the retina, and through visual cortex. However, several psychophysical experiments on facilitation of detection and on suppression of detection indicate substantial coupling between chromatic and achromatic signals. First, there are the results of Switkes et al (1988) on masking and facilitation of color by luminance, and luminance by color. To me the most interesting of many interesting results in this paper is the facilitation of detection of isoluminant color patterns by luminance patterns even if the latter are substantially suprathreshold. This suggests to me that one of the functions of the magnocellular pathway might be to gate parvocellular signals into the cortex. This concept would also make sense of Kelly's finding that isoluminant chromatic patterns suffer great losses in contrast sensitivity when stabilized on the retina (Kelly 1983). It is well known that parvocellular signals are sustained in time when the stimulus is a colored pattern (e.g. Schiller & Malpeli 1978). Yet, an image defined solely by color fades faster and more completely than a luminance pattern.

Other studies that suggest a role for luminance signals in facilitating or

gating chromatic signals are the investigations of the gap effect by Boynton et al (1977) and by Eskew (1989). These studies show that luminance steps near the border of a colored test object may facilitate chromatic discrimination. The effect is significant only for colored stimuli that are defined by b cone modulation. Yet the effect does indicate the possibility for interaction between M and P pathways.

While luminance facilitates color, color stimuli suppress the response to luminance variations. This is seen in the masking data of K. DeValois & Switkes (1983) and Switkes et al (1988). Such an effect is also evident in the chromatic suppression of flicker detection described by Stromeyer et al (1987). Another kind of evidence comes from the flash-on-flash paradigm of Finkelstein & Hood (1982), who showed that detection of a brief flash, while mediated by a V_λ mechanism, could be suppressed by superimposition of a flashed background. The spectral sensitivity of the flashed background was broad, like those seen by Sperling & Harwerth (1971) and King-Smith & Carden (1976), indicating suppression from opponent mechanisms. All of these phenomena, while elicited with different stimuli, have the common theme of color suppressing luminance.

Conclusions

In order to make some sense of the implications of possible roles of P and M pathways in visual processing, we had to consider optics, photoreceptors, the retina, the LGN, areas V1 and V2 in visual cortex, and psychophysics. Much was omitted. But I have attempted to examine the critical evidence on the roles these cell types might play in vision. It seems to me the weight of the evidence is that M cells are the luminance pathway, though they do not control the finest achromatic acuity. P cells must provide color signals, but it seems they may need cooperation from the M pathway for that signal to be interpreted by the brain. Cooperative and suppressive interactions, revealed mainly so far by psychophysical experiments, demonstrate that these pathways may start out in parallel but they converge.

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