Complete sparing of high-contrast color input to motion perception in cortical color blindness

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It is widely held that color and motion are processed by separate parallel pathways in the visual system, but this view is difficult to reconcile with the fact that motion can be detected in equiluminant stimuli that are defined by color alone. To examine the relationship between color and motion, we tested three patients who had lost their color vision following cortical damage (central achromatopsia). Despite their profound loss in the subjective experience of color and their inability to detect the motion of faint colors, all three subjects showed surprisingly strong responses to high-contrast, moving color stimuli — equal in all respects to the performance of subjects with normal color vision. The pathway from opponent-color detectors in the retina to the motion analysis areas must therefore be independent of the damaged color centers in the occipitotemporal area. It is probably also independent of the motion analysis area MT/V5, because the contribution of color to motion detection in these patients is much stronger than the color response of monkey area MT.

In central achromatopsia, patients lose the sensation of color as a result of damage to the cortex and often report that the world appears solely in shades of gray, as in a black-and-white movie[1–4]. Despite this almost total loss of color sensation, there are several reports that central achromats paradoxically preserve other contributions of color to visual performance. Some of these preserved functions may indicate that the damage to the color analysis areas was less than total. For example, tests of sensitivity to color increments and color contrast in several central achromats[5–9] and in one patient with partial achromatopsia[10] have, up to now, revealed performance quite similar to that of subjects with normal color vision. In contrast, the achromats in our experiments all show profound losses on these tests, indicating more extensive damage to color processing areas. On the other hand, it seems that all central achromats, including those in our experiments, can see color-defined shapes due to the preserved visibility of borders between colors they no longer distinguish[8,11–14]. A likely candidate for this border response is the magnocellular, or non-opponent pathway, which responds to color transitions[15,16].

Both the perception of motion for equiluminous color stimuli[11] and a color-specific motion aftereffect[5] have been demonstrated in one central achromat. However, neither study established the relative strength of the preserved motion response. If motion responses to color stimuli were only weakly preserved, we could attribute this to the partial sparing of some color-analysis areas adjacent to the cortical damage. Any particular spared response could mediate other responses to color stimuli. For example, to an observer with normal color vision, a saturated color appears brighter than its luminance would predict[17]. If an achromat lost the experience of color but retained this supplementary brightness response[8,11], equiluminant color bars would have a residual brightness difference. In this case, the visibility of the bars and their motion could be reduced but never eliminated. Conversely, if motion responses to color are totally preserved in central achromatopsia, it would argue against a residual, spared mechanism and suggest that color's contribution to motion takes a route that is anatomically remote from the damaged areas.

We now resolve this question with a population of central achromats who show little or no preserved opponent-color processing in spectral sensitivity, detection or motion tasks at threshold. At suprathreshold levels, however, these same achromats surprisingly demonstrate normal levels of motion responses to moving colored gratings.

To evaluate this performance, we included two control groups: normal subjects and congenitally red/green-deficient observers (where the site of loss is in the retina). These individuals are commonly described as color blind; however, they suffer only a partial loss of color experience limited to the red/green dimension, a loss much less extensive than that experienced by the central achromats. Because these observers have little chromatic response to red/green stimuli, however, we can use their performance to evaluate the level of residual

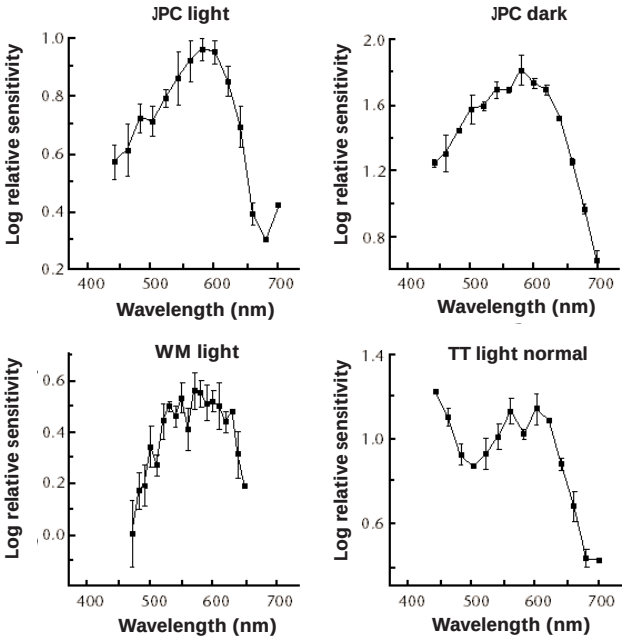

Fig. 1. Sensitivity for increment on light and dark fields as a function of wavelength for two central achromats, JPC and WM, and one normal, TT. The form of the function on a dark field was similar for both achromats and for the normal, and only one plot is shown (JPC, top right). Only the normal observer shows the typical three-peaked function on the light field. Both achromats show a single-peaked function on light and dark fields, indicating that there is little or no contribution of opponent-color mechanisms to these detection thresholds.

luminance signals in our red/green color stimuli.

Our tests reveal that motion responses to high-contrast color gratings are at normal levels in the three patients but absent in the congenitally red/green-deficient observers. These results suggest that the cortical damage that produces achromatopsia spares a particular cortical route for high-contrast color information, allowing it to contribute to motion perception without contributing to color sensations.

Results

Case histories

The three patients with central achromatopsia were similar in that they all had bilateral lesions, made several errors in reading the colored digits of the Ishihara text and, when asked to order several colored disks according to their hue (Farnsworth-Munsell test), they made virtually random orderings. All three also had visual agnosia and prosopagnosia (deficits in object and face recognition). Two had upper-field loss (WM, JPC) and one (JPN) had only one full quadrant of vision (lower right). Visual acuity and contrast sensitivity for achromatic tests in the preserved visual fields showed that at least coarse spatial resolution was retained for all three patients[2,12].

At the time of testing, JPC was 43, a former florist, and his lesions were the result of an assault that caused bilateral hemorrhage in the occipital-temporal regions. Magnetic resonance imaging (MRI) revealed lesions principally in both fusiform gyri but sparing the lingual gyri and the left posterior fusiform gyrus. WM was 74, a former electrician who suffered a severe reduction of vision following a brief loss of consciousness. (WM is described in more detail in ref. 18.) MRI and computer tomography subsequently revealed extensive occipitotemporal lesions bilaterally in the territory of both posterior cerebral arteries. JPN was 41 and had suffered bilateral strokes of the occipital region, producing a large lesion of the right occipital pole and a lesion of the left lingual and fusiform gyri.

JPN reported no conscious sensations of color and showed chance-level color naming in a forced-choice task. JPC noticed only reds (and some yellows) as being weakly 'tinted' but he did not name them as red because the tint differed from the red he remembered. WM reported no conscious sensations of color but when forced to guess, he demonstrated better-than-chance naming of red and yellow.

Spectral sensitivity

The increment threshold spectral sensitivity task is a measure of the degree of function of the opponent-color pathways[19,20]. A chromatic stimulus of varying intensity is presented briefly on a white background, and the observer reports whether anything has been presented. In the normal observer, the sensitivity peaks at three different wavelengths, which are characteristic of opponent-cone interactions[20]. If the tests are presented on a dark background, the luminance mechanism is more sensitive than any of the opponent mechanisms, and the sensitivity curve has a single peak. Both of these tests were performed on two of the patients, JPC and WM, and a normal observer, TT, one of the authors. The data for WM have been reported elsewhere[13].

Both patients showed single-peaked functions for thresholds on the light and dark backgrounds. The normal observer showed the expected three-peaked function with light backgrounds and single-peaked function with dark background. These results (Fig. 1) indicate that the two patients have significant losses in the opponent-color pathways, which mediate broad ranges of the typical three-peaked threshold function seen in normal subjects.

This experiment was very similar to previous experiments[7,8] on two other central achromats. Both of those patients showed the normal, three-peaked function. One explanation of these earlier results[8] was that some color-opponent response was spared and contributed to a brightness percept for the stimuli but not a color percept. We conclude that the previously tested patients had some sparing of high-level color centers, whereas WM and JPC have a more profound loss with no evidence of preserved color-opponent contribution to increment thresholds.

Contrast thresholds

These tests evaluated the color contrast that allow observers to detect a red/green sinusoidal grating or to determine its direction of motion. Both smoothly moving gratings and gratings moving in 90° steps were used. The 90° steps[11,21,22] effectively control for motion cues from the borders, which central achromats are known to detect well. With each step, the new positions of the red/green transitions fall exactly halfway between the previous positions, rendering the border cue ambiguous.

All three central achromats showed very high discrimination (Fig. 2) and detection thresholds. The thresholds for WM were exceptionally high. These thresholds suggest that WM, JPC and JPN have severe losses in their response to color. We did not find any evidence of a preserved brightness response

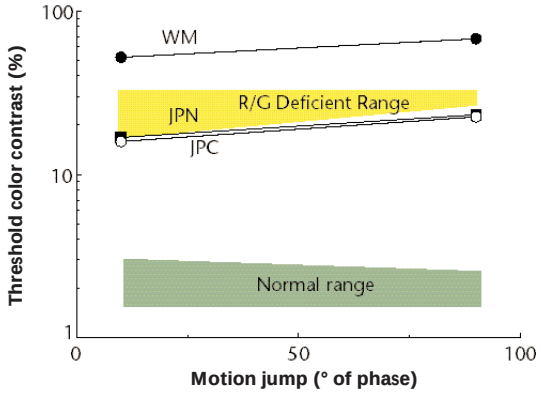

Fig. 2. Threshold color contrast for the three central achromats for direction discrimination of the moving color gratings. The range of values measured for two congenitally red/green-deficient observers is shown by a yellow band and that for two normals by a green band. Detection thresholds are not shown but are similar to the discrimination thresholds for all subjects, except for the normals, in which detection thresholds are lower than discrimination thresholds by a factor of two to three.

to color, which was proposed as the source of the preserved chromatic sensitivity in MS and other previously tested achromats[11].

There were no marked differences between the thresholds for smoothly moving or jumping stimuli, indicating that the response to the chromatic borders in these patients was not a significant factor in producing these threshold responses. Despite the elevated thresholds, the red/green equiluminance values for the achromats were in the normal range, indicating normal function of at least the red- and green-sensitive cone classes[21]. Of the two congenitally red/green-deficient observers, the deutan (with losses in the green-sensitive cone class) showed an equiluminance setting in the normal range, whereas the protan (with losses in the red-sensitive cone class) required significantly more luminance for red.

Motion nulling

Like a subject (MS) in a previous study[11], the three patients tested in the previous experiment could report the direction of a color grating (once it was above threshold). However, unlike MS, these three patients have severe losses in chromatic sensitivity and require very high contrasts for accurate judgements. A luminance mechanism, which is nominally but never perfectly insensitive to color, may start to respond to these very strong color stimuli. Therefore when the three patients tested here do respond to color at high contrasts, it may be due to a small response of the luminance pathway to color or it may be due to the response of a color-specific mechanism.

To resolve this question, we used a stimulus (Fig. 3) that dissociates the two components, color and luminance, at suprathreshold levels[21,23–25]. This gives us a measure of the strength of the color contribution to motion for the patients and for the normal observers. This test evaluates the low-level motion response to color as opposed to a high-level, tracking response[21,25]. To make the measurements, color and luminance gratings were superimposed and set in motion in opposite directions. At a particular balance of the contrast of the two gratings, their motions cancelled and ambiguous motion or flicker was seen. The contrast of the luminance grating that just cancelled the motion of the color grating was taken as the 'equivalent luminance contrast' of the color grating[21]. Moreover, by again using congenitally red/green-deficient subjects as a control group, we were able to estimate the baseline response of the luminance mechanisms to residual luminance components in the color stimulus.

Despite catastrophic loss in color sensitivity, when tested with these suprathreshold color gratings, the three achromatopsic patients have equivalent luminance contrasts that lie near or within the range of values found in normal subjects (Fig. 4). The responses for the 90° jump conditions can only be mediated by color-specific motion analysis because the red/green borders are producing ambiguous motion signals in this stimulus.

The congenitally red/green-deficient observers showed motion strengths in the 2% to 4% range, indicating that the sum of their weak color response and luminance artifacts from monitor and optical sources was less than 4%. The difference between these values and the strengths measured for the central achromats is therefore a lower bound on the actual strength of the color-specific input to motion for these patients. The high levels of equivalent luminance contrasts found for the three patients, as well as their normal equiluminance settings, again demonstrate that they have normal red- and green-sensitive cone function[21].

It is nevertheless possible that some residual cues might differentiate the red and the green of the gratings for the central achromats and allow them to track individual grating bars. It is unlikely, however, that these strategies (which the patients did not report) could produce equivalent luminance contrasts in the normal range seen here. On the other hand, JPC does report some residual color sensations for red stimuli, and so all the tests were repeated with purple/green gratings (differentially stimulating principally the blue-sensitive cones) for both WM and JPC. The patients again showed the same motion strength as normal subjects.

How can these patients have normal levels of equivalent luminance contrast but red/green-deficient levels of color-contrast threshold? How can the response to color be absent up to a significantly elevated threshold and then jump to robust, normal levels once above threshold? We tested one patient (JPC) for blindsight[26], to test the possibility that he might be able to report the direction of motion for stimuli he claims not to see. After instructions to guess, his reports of

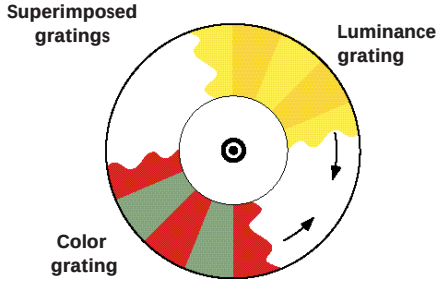

Fig. 3. A sinusoidal, equiluminous, color grating (red/green) and a sinusoidal luminance grating (light and dark yellow) were superimposed, moving in opposite directions. (The stimuli are depicted here as square-wave gratings for convenience.) At a particular contrast of the luminance grating, the two motions null each other, establishing the equivalent luminance contrast of the color grating.

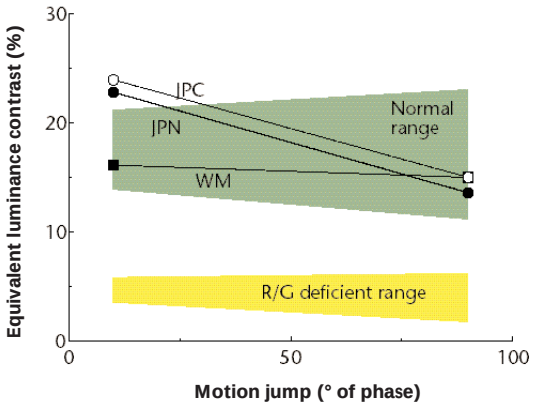

Fig. 4. Equivalent luminance contrasts for color gratings moving in smooth (10.8°) and 90° jumps. Data are shown individually for the three central achromats. The range of values measured for six congenitally red/green-deficient observers is shown as a yellow band and that for normals (five observers) as a green band.

motion direction for red/green gratings were random for stimuli in the 8% to 15% range of color contrast just below his detection threshold (but still about ten times higher than normal thresholds). It seems that the loss of performance at low color contrast was real and is independent of the performance at high color contrast.

We also tested his responses in the motion-nulling test (described above) at low contrasts. The color grating had no effect once it was below threshold; the motion of the opposing luminance grating was the only direction the patient reported. Above his color threshold, however, the contrast of the luminance grating required to null the motion of the color grating rose rapidly. Indeed, a normal subject (PC) showed the same, rapid rise in equivalent luminance contrast above 20% color contrast. However, the normal observer also had a weak residual equivalent luminance contrast (1% to 2%) for lower chromatic contrasts, whereas the achromat showed none. The patient seems to share with the normal subject a motion response to color that rapidly gains strength at high contrast. The patient has lost a more sensitive process that operates at low contrasts in normal subjects and apparently depends on the functioning of higher color centers.

Discussion

At high color contrasts, the strength of the color contribution to motion for the three central achromats was equivalent to that for normal subjects, demonstrating that there is a direct path for color information from the retina to cortical motion detectors that is spared in central achromatopsia. Motion responses mediated by this pathway were fully preserved, suggesting that the pathway was completely independent of the lesioned color centers of the ventral surface of the occipitotemporal region.

Previously tested central achromats have shown residual opponent-color processing as indicated by a three-peaked spectral sensitivity curve and normal detection thresholds[7–9,11]. In contrast, in our study, both patients who were tested for spectral sensitivity had only a single-peaked function, and all three had significantly elevated color-detection thresholds. The damage to the color centers in these three patients appears to be the most profound yet tested. The preserved motion responses to color were found despite this extreme loss of color processing.

To gauge the magnitude of color's contribution to motion in our patients, consider that the maximum red/green color contrast we used produced 14% and 35% of cone contrast for the red- and green-sensitive cones, respectively. This is the same order of magnitude as the luminance contrast required to null it, 12% to 23% in these patients (and the normal subjects). In other words, the contribution of a color stimulus to motion, where the cone responses are out of phase, seems to be quite similar to the contribution of a luminance stimulus, where the same cone contrasts are in phase.

This result seems at first at odds with the many reports of degraded motion perception for chromatic stimuli. However, recent studies have shown that thresholds for motion of gratings defined by color are actually lower than those for luminance-defined gratings[27,28]. The explanation is that the threshold for seeing a color-defined stimulus is lower still, the lowest of all stimuli[29], so that color gratings can be seen before their motion is noticed. The result is the phenomenon of 'stopped motion'[30,31], which led to the erroneous belief that color was a poor source of motion information. More recent studies demonstrate a strong[21] and independent[32] contribution of color to motion, and our results here show a contribution at a level that could not be mediated by any secondary, residual effect. The secondary, residual responses probably account for the eventual detection of the color stimuli at the very high thresholds found in the patients and the congenitally red/green-deficient observers. This suggest that the secondary cues have about one-tenth the power of the color signal.

One possible site for mediating the response to color is MT, an area specialized in motion analysis, which is located far from the areas damaged in achromatopsia. Physiological recordings in monkeys[33–35] and fMRI studies in humans[36] have shown responses to equiluminous chromatic gratings in area MT. Experiments with a 90° stimulus like that used here have shown that this response can be based on color information, not just on the color borders[33]. However, this response is very weak, with an equivalent luminance contrast of only 2.5%, within the range that congenitally red/green-deficient observers show and therefore attributable to distortions in the monitor, the eye or the non-opponent pathway. This level of response is far too low to explain the performance of normal subjects and patients whose equivalent luminance contrasts were in the range of 12 to 23%.

If MT is not the site of the robust, low-level motion response to chromatic stimuli that we report here, where could it be? The site of cortical damage in humans with achromatopsia invariably includes the ventral surface of occipitotemporal region, and although this deficit is often claimed to involve the human homologue of monkey V4, recent lesion studies in monkeys suggest that this may not be the case[37].

In humans, the most recent fMRI studies[38] suggest a more anterior site, V8, for color analysis in a location consistent with the damage in the achromatopsic patients. The human homologue to V4 on the ventral surface (V4v) is probably also damaged in these patients but it includes only a representation of the upper visual fields[39]. The matching area that represents the lower visual field has not been identified with certainty. If there is a dorsal equivalent to V4 with a map of the lower visual fields, we speculate that it might offer a pos-

sible site for the strong contribution of color to low-level motion in these patients and in normals. In monkeys, V4 does have a fair percentage of cells that are directionally selective[40]. Moreover, V4 in monkeys is directly involved in maintaining a representation of motions in a delayed match-to-sample task[41]. Although the site of the robust motion response to high-contrast color remains to be determined, our results demonstrate that the path from the retina to this area does not pass through the damaged ventral area of these patients. This suggests that color information does not flow as a single, hierarchically organized stream through the visual system but rather projects in parallel to several different centers, only one of which leads to the conscious experience of color.

Methods

SPECTRAL SENSITIVITY. The observers were two central achromats, WM and JPC, and one normal subject, TT, one of the authors. Observers in this and following experiments gave informed written consent before the experiments, which were approved by the Ethisches Kommitee der neurologischen Klinik des Universitätsspital Zürich, by the Comité consultatif de protection des personnes dans la recherche biomédicale du centre Léon Bérard, Lyon and by the F.A.S. Human Subjects Committee, Harvard University.

The uniform light surround was 20 cd per m^2 and the dark surround was 0.3 cd per m^2. The test stimuli were presented for 0.5 s with a gap of 2.5 s between stimuli. The target was a slightly blurred rectangle subtending about 2.2° by 1.6°. Fixation was not monitored during the experiment. The chromatic tests were provided by a monochromator with a bandwidth of 10 nm.

After adapting to the light or dark background for several minutes, testing began at the shortest wavelength. Increment thresholds were determined using a method of limits, and the procedure was repeated three times, reversing the order of the wavelengths each time. The mean of the three values was used to calculate the sensitivity for each wavelength.

CONTRAST THRESHOLDS. The observers were the three central achromats, two congenitally red/green-deficient (one deutan and one protan) and two normal subjects. The red/green-deficient subjects each failed at least 18 of the 24 Ishihara plates, whereas neither normal subject made any errors.

The stimulus was a rotating wheel of eight cycles of a color grating, varying sinusoidally between red and green (Fig. 2). The wheel was set in rotation either 'smoothly' (because of the 66.7 Hz raster rate, the actual step was 10.8° on each frame but it appeared to move smoothly) or in 90° jumps. The rate of rotation was fixed at 2 Hz in all cases (0.25 revolutions per second). The outer diameter of the wheel was eight degrees of visual angle, and its inner radius was three degrees. Its mean luminance was approximately 45 cd per m^2, and the surround was dark. The control of luminance was linearized through gamma correction look-up tables.

The chromatic contrast of a grating was defined in terms of the percentage of the maximum chromatic modulation obtainable with the phosphors involved. 100% color contrast of the monitor produces, at equiluminance, 14% cone contrast for the red-sensitive cones and 35% cone contrast for the green-sensitive cones.

The colors were set to equiluminance[42] individually for each observer. Thresholds were measured at that value and often two adjacent values bracketing the equiluminance setting as well. In every instance tested, the highest thresholds occurred at the predetermined equiluminance setting. Observers were instructed to fixate the central bull's-eye, and the tests were presented on the display until a response was given. Observers reported whether a stimulus was absent or present in the detection conditions or moving clockwise versus counterclockwise in the direction-discrimination condition. Detection thresholds were taken as the chromatic contrast for which the subject reported 'present' on 50% of the trials. Discrimination thresholds were taken as the chromatic contrast for which the subject reported the direction correctly on 75% of the trials.

MOTION NULLING. The observers were the three central achromats, six congenitally red/green-deficient observers and five normal observers. The red/green-deficient subjects (two deutans, four protans) each failed at least 18 of the 24 Ishihara plates, whereas the normal subjects made no more than three errors.

The stimulus was a rotating wheel of eight cycles of a red/green sinewave as before but now (Fig. 4) superimposed additively on a similar grating with eight cycles of a light/dark yellow sinewave rotating in the opposite direction. The color contrast was set at the maximum available between the red and green phosphors with allowance for the superimposed luminance grating. The resulting color contrast was between about two (WM) and five (JPC, JPN) times threshold contrast. The stimulus was otherwise identical in all respects to that used in the previous experiment.

In a forced-choice procedure, while fixating the bull's-eye, the observers reported the direction of the global motion seen in the combined stimulus. The luminance contrast that produced a motion null (equal frequency of reports favoring luminance and color directions) was taken as the 'equivalent luminance contrast' of the color grating[21]. We also tested red/green luminance ratios bracketing the equiluminance setting and, in every instance, the minimum equivalent luminance contrast occurred at the predetermined setting.

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1. Damasio, A., Yamada, T., Damasio, H., Corbett, J. & McKee, J. Central achromatopsia: Behavioral, anatomic, and physiologic aspects. *Neurology* 30, 1064–1071 (1980).
2. Grüsser, O.-J. & Landis, T. in *Vision and Visual Dysfunction, Vol 12: Visual Agnosias*. (ed. Cronly-Dillon, J.) 394–400 (MacMillan, London, 1991).
3. Pearlman, A.L., Birch, J. & Meadows, J.C. Cerebral color blindness: An acquired defect in hue discrimination. *Ann. Neurol.* 5, 253–261 (1979).
4. Zeki, S.M. A century of cerebral achromatopsia. *Brain* 113, 1721–1777 (1990).
5. Mollon, J.D., Newcombe, F., Polden, P.G. & Ratcliff, G. in *Colour Vision Deficiencies* (ed. Verriest, G.) 130–135 (Hilger, Bristol, 1980).
6. Wooten, B.R. Partial cerebral achromatopsia with selective hue loss. *Doc. Ophthalmol. Proc. Series* 33, 139–144 (1982).
7. Jaeger, W., Krastel, H. & Braun, S. Cerebral achromatopsia (symptoms, course, differential diagnosis and examination strategy). II. [Article in German] *Klin. Monatsbl. Augenheilkd.* 194, 32–36 (1989).
8. Heywood, C.A., Cowey, A. & Newcombe, F. Chromatic discrimination in a cortically colour blind observer. *Eur. J. Neurosci.* 3, 802–812 (1991).
9. Heywood, C.A., Nicholas, J.J. & Cowey, A. Behavioural and electrophysiological chromatic and achromatic contrast sensitivity in an achromatopsic patient. *J. Neurol. Neurosurg. Psychiatry* 60, 638–643 (1996).
10. Victor, J.D., Maiese, K., Shapley, R., Sidtis, J. & Gazzaniga, M.S. Acquired central dyschromatopsia: Analysis of a case with preservation of color discrimination. *Clin. Vis. Sci.* 4, 183–196 (1989).
11. Heywood, C.A., Cowey, A. & Newcombe, F. On the role of parvocellular (P) and magnocellular (M) pathways in cerebral achromatopsia. *Brain* 117, 245–254 (1994).
12. Henaff, M.A. & Michel, F. A case of central achromatopsia: Possible implicit treatment of hue. *Invest. Ophthalmol. Vis. Sci.* 34, 745 (1993).
13. Troscianko, T. *et al.* Human colour discrimination based on a non-parvocellular pathway. *Curr. Biol.* 6, 200–210 (1996).
14. Barbur, J.L., Harlow, A.J. & Plant, G.T. Insights into the different exploits of colour in the visual cortex. *Proc. R. Soc. Lond. B* 258, 327–334 (1994).
15. Lee, B.B., Martin, P.R. & Valberg, A. The physiological basis of heterochromatic flicker photometry demonstrated in the ganglion cells of the macaque retina. *J. Physiol. (Lond.)* 404, 323–347 (1988).
16. Schiller, P.H. & Colby, C.L. The responses of single cells in the lateral geniculate nucleus of the rhesus monkey to color and luminance contrast. *Vision Res.* 23, 1631–1641 (1983).

17. Wagner, G. & Boynton, R.M. A comparison of four methods of heterochromatic photometry. *J. Opt. Soc. Am.* 62, 1508–1515 (1972).
18. Scheidler, W., Landis, T., Rentschler, I., Regard, M. & Baumgartner, G. A pattern recognition approach to visual agnosia. *Clin. Vis. Sci.* 7, 175–193 (1992).
19. Harwerth, R.S. & Sperling, H.G. Effects of intense visible radiation on the increment-threshold spectral sensitivity of the rhesus monkey eye. *Vision Res.* 15, 1193–1204 (1975).
20. King-Smith, P.E. & Carden, D. Luminance and opponent-color contributions to visual detection and adaptation and to temporal and spatial integration. *J. Opt. Soc. Am.* 66, 709–717 (1976).
21. Cavanagh P. & Anstis S.M. The contribution of color to motion in normal and color-deficient observers. *Vision Res.* 31, 2109–2148 (1991).
22. Dobkins, K.R. & Albright, T.D. What happens if it changes color when it moves?: psychophysical experiments on the nature of chromatic input to motion detectors. *Vision Res.* 33, 1019–1036 (1993).
23. Chichilnisky, E.J., Heeger, D. & Wandell, B.A. Functional segregation of color and motion perception examined in motion nulling. *Vision Res.* 33, 2113–2125 (1993).
24. Teller, D.Y. & Palmer, J. Infant color vision: motion nulls for red/green vs luminance-modulated stimuli in infants and adults. *Vision Res.* 36, 955–974 (1996).
25. Cavanagh, P. Attention-based motion perception. *Science* 257, 1563–1565 (1992).
26. Weiskrantz, L. Blindsight revisited. *Curr. Opin. Neurobiol.* 6, 215–220 (1996).
27. Metha, A.B., Vingrys, A.J. & Badcock, D.R. Detection and discrimination of moving stimuli: the effects of color, luminance, and eccentricity. *J. Opt. Soc. Am.* 11, 1697–1709 (1994).
28. Stromeyer, C.F. III, Kronauer, R.E., Ryu, A., Chaparro, A. & Eskew, R.T. Jr Contributions of human long-wave and middle-wave cones to motion detection. *J. Physiol. (Lond.)* 485, 221–243 (1995).
29. Chaparro, A., Stromeyer, C.F. III, Huang, E.P., Kronauer, R.E. & Eskew, R.T. Jr Colour is what the eye sees best. *Nature* 361, 348–350 (1993).
30. Ramachandran, V.S. & Gregory, R. Does colour provide an input to human motion perception? *Nature* 275, 55–56 (1978).
31. Cavanagh, P., Tyler, C.W. & Favreau, O.E. Perceived velocity of moving chromatic gratings. *J. Opt. Soc. Am.* 1, 893–899 (1984).
32. Cropper, S.J. & Derrington, A.M. Rapid colour-specific detection of motion in human vision. *Nature* 379, 72–74 (1996).
33. Dobkins, K.R. & Albright, T.D. What happens if it changes color when it moves?: the nature of chromatic input to macaque visual area MT. *J. Neurosci.* 14, 4854–4870 (1994).
34. Saito, H., Tanaka, K., Isono, H. Yasuda, M. & Mikami, A. Directionally selective response of cells in the middle temporal area (MT) of the macaque monkey to the movement of equiluminous opponent color stimuli. *Exp. Brain Res.* 75, 1–14 (1989).
35. Gegenfurtner, K.R. *et al.* Chromatic properties of neurons in macaque MT. *Vis. Neurosci.* 11, 455–466 (1994).
36. Ffytche, D.H., Skidmore, B.D. & Zeki, S. Motion-from-hue activates area V5 of human visual cortex. *Proc. R. Soc. Lond. B* 260, 353–358 (1995).
37. Heywood, C.A., Gaffan, G. & Cowey, A. Cerebral achromatopsia in monkeys. *Eur. J. Neurosci.* 7, 1064–1073 (1995).
38. Hadjikhani, N.K., Liu, A.K., Cavanagh, P., Dale, A.M. & Tootell, R.B.H. Retinotopy and color sensitivity in human visual cortical area V8. *Nature Neurosci.*, 1, 235–241 (1998).
39. Tootell, R.B., Dale, A.M., Sereno, M.I. & Malach, R. New images from human visual cortex. *Trends Neurosci.* 19, 481–489 (1996).
40. Zeki, S.M. Uniformity and diversity of structure and function in rhesus monkey prestriate visual cortex. *J. Physiol. (Lond.)* 277, 273–290 (1978).
41. Ferrera, V.P., Rudolph, K.K. & Maunsell, J.H. Responses of neurons in the parietal and temporal visual pathways during a motion task. *J. Neurosci.* 14, 6171–6186 (1994).
42. Cavanagh, P., Anstis, S.M. & MacLeod, D.I.A. Equiluminance: Spatial and temporal factors and the contribution of blue-sensitive cones. *J. Opt. Soc. Am.* 4, 1428–1438 (1987).