

Dispatches

Colour Vision: The Wonder of Hue

Understanding the brain requires a kind of thinking outside the main tradition of natural science: the biology has to be linked to something intangible, a private experience. Physiologists have recently recorded from neurons that promise to help make the link in the case of colour experience.

Jay Neitz¹ and Maureen Neitz²

The restaurant hostess seats our children with paper colouring placemats and gives each a pack of crayons — red, green, yellow and blue, recognized by children everywhere who intuitively understand that there is something ‘unique’ about these *four* colours. In this issue of *Current Biology*, Stoughton and Conway [1] relate the fundamental nature of these colours to individual neurons in the brain.

According to Albert Einstein, great advances in our understanding of nature have originated from an “intuitive grasp of the essentials of a large complex of facts [which] leads the scientist to the postulation of a hypothetical basic law or laws. From these laws he draws conclusions”. This formula that has proved useful in illuminating the cosmos could be fruitful in fathoming the complex workings of the brain. Neuroscientists have yet to agree, however, on which hypothetical laws should be adopted. One grand postulate that has guided attempts to understand the brain is the ‘law of specific nerve energies’ or Müller’s law, after Johannes Müller (1801–1858): “Each type of sensory nerve ending, however stimulated (electrically, mechanically, etc.), gives rise to its own specific sensation; moreover, each type of sensation depends not upon any special character of the different nerves but upon the part of the brain in which their fibres terminate.”

The postulate of an individual neuron at some location in the brain giving rise to a specific sensation provides the link between the firing of a neuron and perceptual experience. In the case of colour vision, a goal is to discover the mechanisms that establish the relationship between the wavelength composition of light, the physical stimulus, and colour — the perception.

We can imagine that for each discriminable point in the retinal image there are a set of receptors that transform light absorption into electrical signals. As these signals are transmitted to higher centers, characteristics of each tiny part of the image are extracted to form the basis of percepts.

The brain’s representation of a small segment in a visual scene must be somewhat, but not perfectly, analogous to a pixel in a video display. For each pixel, the colour and brightness of light are represented as three numbers that indicate intensities of red, green and blue. In our visual brain, the characteristics of each small subdivision of a scene are experienced as some combination of fundamental colour sensations, the ‘unique hues’, red, green, blue, yellow, plus black and white, explaining why no fewer than four crayons added to the black and white page of a colouring book will satisfy our children as representing the real world. Stoughton and Conway [1] have now discovered a brain region, the posterior inferior temporal cortex, where the tuning of chromatic sensitivities of neurons cluster around the unique hues. The significance of this discovery can be understood from a historical perspective. The major question has been: how do *three* types of cone photoreceptor ultimately relate to the *six* fundamental colour percepts, black and white plus the *four* unique hues, red, green, blue, and yellow?

Thomas Young (1773–1829) recognized that representing the wavelength of light, a continuous variable, would require a set of receptors that encode the relative amount of light in discrete spectral bands. Because the visual system has to analyse the wavelength content of each point of an image, the constraints of biology would require a limit on the number of detectors with different spectral sensitivities, “as it is

impossible to conceive each point [in the retina] to contain an infinite number of particles..it becomes necessary to suppose the number limited...each sensitive filament of the nerve may consist of three portions, one for each principle colour”. Hermann von Helmholtz (1821–1894) championed Young’s idea and, being a student of Müller, he extended it to link the proposed receptors to human perceptions, saying “Young’s hypothesis is only a special case of the law of specific sense energies” accounting for the sensations of red, green, and violet [2].

Ever since Helmholtz’s statement, the quest for a theory of colour perception can be understood in terms of attempts to match up the properties of neurons to our perceptions. Ewald Hering (1834–1918) argued that the *three* receptors postulated by Young and Helmholtz did not correspond to the number of unique hues we experience. Figure 1A shows how an equal energy spectrum might be perceived if three photoreceptors accounted directly for hue perception. Hering pointed out that there are not *three* but *four* colours, blue, green and red plus one more that did not seem to be explained by trichromatic theory — yellow. These four seemed to have a simplicity that other colours do not and although colours may be described as tinted with one or two of the four psychologically simple colours, for example, a small patch of colour can be depicted as blue, green or blue-green but colours are never described as being simultaneously red and green or yellow and blue.

Although the subject of a great deal of argument during the century from the 1870s to the 1970s, in modern textbooks this problem is often explained as being resolved by a two-stage model of colour processing proposed by Hurvich and Jameson [3], in which the outputs of the three types of cone (the first stage) are combined by neural circuitry (the second stage) that compares the quantal catches of cones to form four circuits for hue percepts that exist as

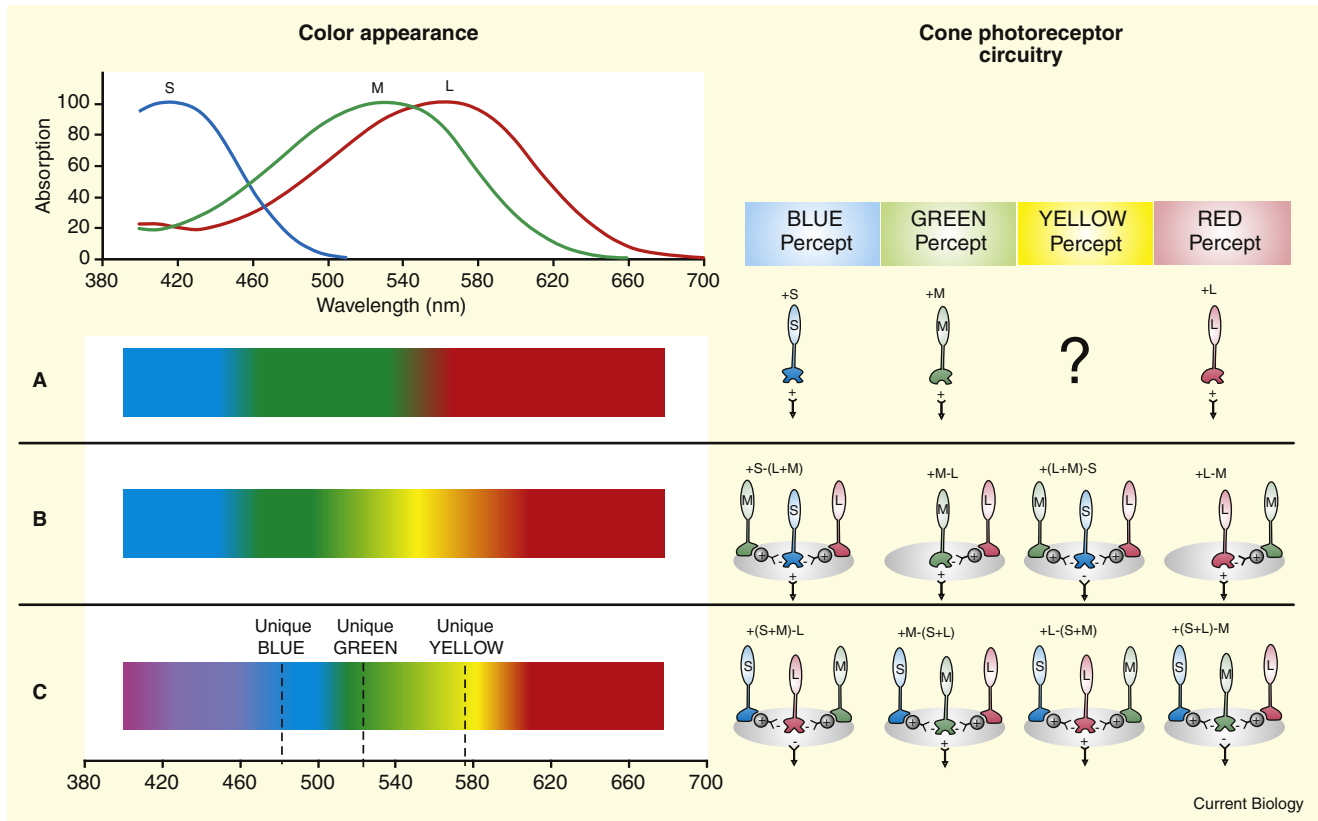


Figure 1. Color processing.

The law of specific nerve energies postulates that, at some level of neural processing, individual neurons produce specific sensations. Absorption spectra of the three types of cones, short (S), middle (M) and long (L) wavelength sensitive are shown at the top left. (A) If the individual cone photoreceptors (illustrated on the right) were directly responsible for hue sensations, an equal-energy spectrum would be expected to consist of only three unique hue sensations (left); the spectrum would, for example, be missing the colour yellow. (B) Neurons in the LGN carry signals from circuits that compare outputs from different cone types. The neural ‘wiring’ of four types of LGN cells is shown (right) but the cone inputs to these most frequent LGN neurons would be predicted to produce chromatic responses (spectrum at left) very unlike human color perception. (C) The way an equal energy spectrum appears to most normal observers (left) requires neural circuitry in which neurons responsible for the percepts of blue, green, yellow and red each get input from all three cones but in each case the cone signals are pooled using different combinations of positive and negative signs (right).

two opponent pairs, red–green and blue–yellow. The opponent character of the second processing stage explains why mixtures of all visible wavelengths of light yield a percept of white (the absence of hue) [4] and the observation of Hering that no colour is seen as bluish-yellow or reddish-green.

Starting in the 1950s, advances in electrophysiology made it possible to record chromatic response properties of neurons. Most exciting for biologists was that features of the two stage model seemed to be confirmed by recordings from spectrally opponent neurons in the lateral geniculate nucleus (LGN) of the thalamus, a target for axons from the retina carrying information to the cortex. But while there did appear to be two classes of blue (B) – yellow (Y) LGN neuron, +B – Y and +Y – B, which would be expected to

correspond to blue and yellow, and two types of red (R) – green (G) neuron, +R – G and +G – R, as needed for red and green, respectively, the ‘spectral signatures’ of the neurons, illustrated in Figure 1B, do not match human colour perceptions (Figure 1C). Discrepancies were noted at the time of discovery of opponent cells (for example [5]), but these have been largely ignored in textbook accounts.

In the last few decades, the absorption spectra of the three cone pigments have been characterized with great precision (top left of Figure 1). This has made it possible to explicitly describe the difference between the textbook LGN cells and perceptual, opponent hue mechanisms in terms of the way specific cones contribute to each [6–9]. The chromatic inputs differ substantially between the two

[10–12]. Textbook LGN ‘red–green’ opponent cells have no short wavelength sensitive (S) cone input and long wavelength sensitive (L) cone signals are opposed by middle wavelength sensitive (M) cones. In contrast, our red–green perceptions are based on circuitry in which signals from both S and L cones are responsible for red perception, such that the sensation of redness at the long-wavelength end of the spectrum is mediated by L cones, while the redness at the violet end is mediated by S cones. The S and L inputs are both opposed to signals from M-cones responsible for greenness.

Similarly, the best characterized ‘blue–yellow’ LGN cells have input from S cones which is opposed to the sum of L and M cones, but the spectral locations of unique hues require

blue–yellow colour vision to be based on (S+M)–L circuitry in which blueness above 460 nm is mostly produced by M cones [13], and blueness below 460 is mediated by S cones. Figure 1B illustrates how most typical LGN cells would predict an absence of redness in the short wavelength part of the spectrum, an absence of much blueness above 460 nm and very different locations of the unique hues than observed by humans (Figure 1C).

For years, progress in understanding colour vision in terms of biology has been stalled by the inability to resolve the lack of correspondence between phenomenological colour experience and the properties of LGN neurons, the only cells offered as candidates for mediating hue experience. This has led some vision scientists to question whether it is possible, or even sensible, to reconcile the domains of neurophysiology and phenomenology [12]. The new discovery of cells whose ‘spectral signatures’ match our hue perceptions, however, opens the way to ultimately solving the circuit that transforms cone signals into colour vision. In the past, the greatest challenge to resolving the discrepancy between the characteristics of colour opponent cells and hue perception has been the difficulty in finding a hypothetical solution that seems logical. The simplest idea is an extension of the two-stage model in which additional processing stages in the cortex would further transform LGN opponent signals, with the wrong spectral signatures into ones that match perception; however, even the most well thought out versions of this idea (for example [6]), raise more questions than they answer. It is not clear how, and even more puzzling why, the cortex would recombine the cone signals.

In contrast to the multistage idea with processing subsequent to the LGN in the hierarchy recombining cone signals to produce colour tuning that matches perception, Calkins [14] has offered the alternative that the most frequently recorded parvocellular LGN spectrally opponent cells are not a substrate for colour vision at all. He points out that, in addition to responding to wavelength, most spectrally opponent neurons are highly responsive to spatial contrast. He says that it is possible that only a small subset of opponent LGN cells, ones that do have the appropriate spectral

signatures all along, mediate hue perceptions. Calkins’ idea may turn out to be prophetic. This year, in their recordings from macaque LGN, Tailby *et al.* [15] specifically focused on neurons with substantial input from S cones and found small populations of cells with cone inputs that match the circuitry required for human perception of hue. It is possible that our brain exploits the majority of LGN cells for spatial vision by extracting their robust responses to luminance contrast and filtering out the spectral responses. If so, signals from the much smaller population of cells discovered by Tailby *et al.* [15] that already have the correct spectral signatures at the level of the LGN could be used for colour. In any case, it is very welcome news that, at long last, two discoveries have been made in one year of neurons that have the correct spectral properties to mediate phenomenological colour experience, one population in the LGN and the other in posterior inferior temporal cortex. These may represent two levels of a colour processing pathway that begins in the retina and ends in hue perception.

References

1. Stoughton, C.M., and Conway, B. (2008). Neural basis for unique hues. *Curr. Biol.* 18, R698–R699.
2. von Helmholtz, H. (1896). *Handbuch der Physiologischen Optik*, 2nd Edition (Hamburg/Leipzig: Voss).
3. Hurvich, L.M., and Jameson, D. (1957). An opponent process theory of color vision. *Psychol. Rev.* 64, 384–404.
4. Neitz, J., Carroll, J., Yamauchi, Y., Neitz, M., and Williams, D.R. (2002). Color perception in

mediated by a plastic neural mechanism that is adjustable in adults. *Neuron* 35, 783–792.

5. DeValois, R.L., Abramov, I., and Jacobs, G.H. (1966). Analysis of response patterns of LGN cells. *J. Opt. Soc. Am.* 56, 966–977.
6. DeValois, R.L., and DeValois, K.K. (1993). A multi-stage color model. *Vision Res.* 33, 1053–1065.
7. Abramov, I., and Gordon, J. (1994). Color appearance: on seeing red–or yellow, or green, or blue. *Annu. Rev. Psychol.* 45, 451–485.
8. Knoblauch, K., and Shevell, S.K. (2001). Relating cone signals to color appearance: failure of monotonicity in yellow/blue. *Visual Neurosci.* 18, 901–906.
9. Hofer, H., Singer, B., and Williams, D.R. (2005). Different sensations from cones with the same photopigment. *J. Vision* 5, 444–454.
10. Webster, M.A., Miyahara, E., Malkoc, G., and Raker, V.E. (2000). Variations in normal color vision II. Unique hues. *J. Opt. Soc. Am. A.* 17, 1545–1555.
11. Valberg, A. (2001). Unique hues: an old problem for a new generation. *Vision Res.* 41, 1645–1657.
12. Mollon, J.D. (2003). *The origins of modern color science*. In *The Science of Color*, 2nd Edition. Optical Society of America, S.K. Shevell, ed. (Oxford, UK: Elsevier).
13. Drum, B.A. (1989). Hue signals from short- and middle-wavelength sensitive cones. *J. Opt. Soc. Am. A.* 6, 153–156.
14. Calkins, D.J. (2004). Linking retinal circuits to color opponency. In *The Visual Neurosciences*, Volume 2, L.M. Chalupa and J.S. Werner, eds. (Cambridge, MA: MIT Press).
15. Tailby, C., Solomon, S.G., and Lennie, P. (2008). Functional asymmetries in visual pathways carrying S-cone signals in macaque. *J. Neurosci.* 28, 4078–4087.

¹R. D. and Linda Peters Professor in Ophthalmology, Medical College of Wisconsin, The Eye Institute, 925 North 87th Street, Milwaukee, Wisconsin 53185, USA.
²Richard O. Shultz & Ruth A. Works Endowed Professor of Ophthalmology, Medical College of Wisconsin, The Eye Institute, 925 North 87th Street, Milwaukee, Wisconsin 53185, USA.
E-mail: jneitz@mcw.edu; mneitz@mcw.edu

DOI: 10.1016/j.cub.2008.06.062

Centrosomes: Keeping Tumors in Check

Centrosomal abnormalities have been observed in a wide range of tumors, but it is not clear whether these abnormalities alone can induce cancer formation or whether they are a consequence of cancer progression. Recent work in *Drosophila* suggests that centrosome defects in asymmetrically dividing cells can induce tumors at a higher frequency than other conditions known to cause genomic instability.

Laurence Pelletier

Centrosomes are the major microtubule-organizing centers (MTOCs) in animal cells. They are composed of a centriole pair embedded in a proteinaceous scaffold called the pericentriolar material

(PCM). In a nutshell, while the number of centriole pairs defines the number of centrosomes present in the cell, the PCM controls the microtubule-nucleation capacity of centrosomes. To build a robust bipolar spindle capable of accurately segregating duplicated chromosomes

$p = 0.38$, respectively). Moreover, analyses of only high confidence states yielded similar results as when high- and low-confidence states were pooled (see Supplemental Data). Together with the numerically large and robust difference between expected and unexpected percepts in the test phase, these observations speak to a true perceptual bias rather than a mere response bias.

Our work shows that experimentally manipulated expectations not only affect the perception of pain [1,6] or emotion, but can have a more general influence on how we experience the world, as evidenced by a striking effect of expectations on the contents of visual awareness. This opens the door for studies of how perception and belief systems are biased by expectation in general and in pathological states such as delusions.

Supplemental data

Supplemental data are available at <http://www.current-biology.com/cgi/content/full/18/16/R697/DC1>

Acknowledgments

We thank Elliot Freeman for help with stimulus programming. Supported by the Wellcome Trust, the Osher Foundation, the Swedish Research Council, and the German Research Foundation.

References

1. Colloca, L., and Benedetti, F. (2005). Placebos and painkillers: is mind as real as matter? *Nat. Rev. Neurosci.* 6, 545–552.
2. Petrovic, P., Dietrich, T., Fransson, P., Andersson, J., Carlsson, K., and Ingvar, M. (2005). Placebo in emotional processing--induced expectations of anxiety relief activate a generalized modulatory network. *Neuron* 46, 957–969.
3. Voudouris, N.J., Peck, C.L., and Coleman, G. (1990). The role of conditioning and verbal expectancy in the placebo response. *Pain* 43, 121–128.
4. Price, D.D., Milling, L.S., Kirsch, I., Duff, A., Montgomery, G.H., and Nicholls, S.S. (1999). An analysis of factors that contribute to the magnitude of placebo analgesia in an experimental paradigm. *Pain* 83, 147–156.
5. Nawrot, M., and Blake, R. (1989). Neural integration of information specifying structure from stereopsis and motion. *Science* 244, 716–718.
6. Petrovic, P., Kalso, E., Petersson, K.M., and Ingvar, M. (2002). Placebo and opioid analgesia - Imagine a shared neuronal network. *Science* 295, 1737–1740.

¹Wellcome Trust Centre for Neuroimaging, University College London, UK. ²Department of Psychiatry, Charité University Hospital, Berlin, Germany. ³Niels Bohr Project “Interacting Minds”, CFIN, University of Aarhus, Denmark. ⁴Department of Clinical Neuroscience, Karolinska Institute, Stockholm, Sweden.
E-mail: philipp.sterzer@charite.de

Neural basis for unique hues

Cleo M. Stoughton
and Bevil R. Conway

All colors can be described in terms of four non-reducible ‘unique’ hues: red, green, yellow, and blue [1]. These four hues are also the most common ‘focal’ colors — the best examples of color terms in language [2]. The significance of the unique hues has been recognized since at least the 14th century [3] and is universal [4,5], although there is some individual variation [6,7]. Psychophysical linking hypotheses predict an explicit neural representation of unique hues at some stage of the visual system, but no such representation has been described [8]. The special status of the unique hues “remains one of the central mysteries of color science” [9]. Here we report that a population of recently identified cells in posterior inferior temporal cortex of macaque monkey contains an explicit representation of unique hues.

Color in humans and macaque monkeys depends on the differential responses of the three cone types — L, M and S — an operation typified by parvocellular neurons of the lateral geniculate nucleus of the thalamus (LGN). LGN cells can be categorized according to color preference, but these categories do not correspond to unique hues [6,10,11]. Instead, multi-stage models have been developed, locating the essential color calculation to brain regions subsequent to the LGN in the visual processing hierarchy [12,13]. Such models describe a recombination of the cone signals to produce color tuning that corresponds to perception, but it is also plausible that the LGN output is simply filtered so that only that minority of LGN cells with appropriate color tuning is routed to color-processing regions of cortex. In either case, neurons downstream of the LGN at the first cortical stages of vision (V1 and V2), are, however, unlikely to encode unique colors [14–17]: like neurons in the LGN, color-opponent neurons in V1 are tuned to colors lying close to the cardinal color axes defined by cone opponency: L –M (bluish-red);

–L+M (cyan), S –(L+M) (lavender), and –S+(L+M) (lime) [15,16,18]. As in the LGN, the overwhelming majority of color-opponent neurons in V1 are tuned along the red-cyan axis [15,16].

Color-tuned neurons have recently been found in posterior inferior temporal cortex of the macaque monkey, clustered within millimeter-sized modules dubbed globs, downstream from V1 and V2 [19,20]. We determined the color tuning of the population of glob cells described in that study (Figure 1). Although neurons tuned to all directions in color space were found [20], the population distribution was not uniform, and is markedly different from that obtained in LGN or V1. The population distribution contains three prominent peaks. The largest peak aligns with red; the second largest, with green; and the third, with blue. The distribution also includes a bulge that peaks in the yellow. These peaks are roughly consistent with unique colors identified by human subjects (symbols, Figure 1). The three prominent peaks also correspond to the three most saturated colors in the stimulus set (see Figure S1 in the Supplemental data available on-line with this issue); and the size of each of the peaks corresponds to the relative saturations of the hues, suggesting that both hue and saturation are represented by relative number of glob cells. The relative size of each of the peaks also corresponds to the frequency with which these color terms is adopted by language: red is adopted first, then yellow or green, followed by blue [4]. These results extend those of Zeki [21] and Komatsu *et al.* [22] and are, to our knowledge, the closest explicit neural representation of unique colors in the primate brain.

The stimuli consisted of flashed (200 ms ON/200 ms OFF) optimally shaped bars surrounded by a neutral-adapting gray field. Color tuning was assessed by varying the color of the bar. Three sets of equiluminant colors were used: one set was equiluminant with the adapting-gray field; one set was higher luminance than the adapting field; and one set was lower luminance than it. The population tuning was consistent across stimulus sets, except for a subtle shift in the location of the

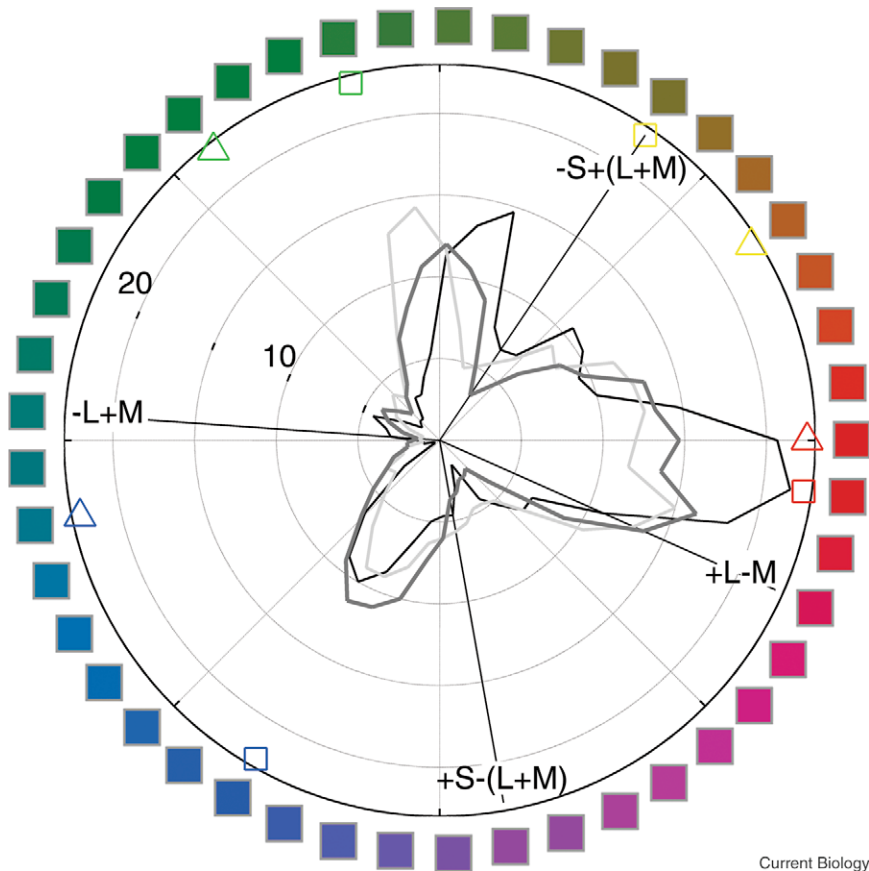


Figure 1. Histogram of optimal color tuning of glob cells recorded in alert macaque monkey shown as a polar plot.

Globs are regions of posterior inferior temporal cortex (including V4, PITd and posterior TEO) that show higher fMRI responses to equiluminant color than to black-and-white [19,20]. Single-unit responses were obtained from two monkeys using microelectrodes targeting globs (for all methods and detailed description of the stimuli see [20]). Number of cells tuned to each color is indicated by the radius (308 cells; smoothing: 1-bin-wide boxcar). Cells were tested with stimuli of optimal spatial configuration, varied only in color (Table S1 in the Supplemental data gives C.I.E. values; colors around the perimeter are approximate). Color tuning was assessed with three sets of equiluminant colors: one set was equiluminant with the adapting gray field (thick dark-gray line); one set was higher luminance than the adapting field (thick light-gray line); and one set was lower luminance than the adapting field (thin black line). The location of the cardinal color axes is shown, along with the average location of unique colors judged by human subjects from two studies (squares, [11]; triangles, [23]).

peaks, most pronounced for green (compare the three plots, Figure 1). These shifts were consistent with the Bezold-Brücke hue shift – at lower luminance, a green stimulus must contain more intensity at long wavelengths (yellow) to appear constant green – providing further evidence that this population of cells is encoding color experience.

The population of glob cells has a strong explicit representation of three of the four unique colors; yellow is weak. The stimuli were generated with a computer monitor, and were constrained to be equiluminant; thus all colors were limited by the maximum luminance of the dimmest

computer phosphor gun (blue). At this luminance, stimuli in the yellow region appear ochre, lacking the brilliance one associates with focal yellow. We interpret the weak yellow peak not to a lack of neurons tuned to yellow, but rather to a lack of focal yellow in the stimulus set.

Supplemental data

Supplemental data are available at <http://www.current-biology.com/cgi/content/full/18/16/R698/DC1>

Acknowledgments

We thank Doris Tsao for help throughout the project; and Ralph Pridmore, Michael

Webster and Thorsten Hansen for comments on the manuscript. The authors declare that they have no competing financial interests.

References

- Hurvich, L.M. (1981). *Color Vision* (Sutherland, MA: Sinauer Associates Inc.).
- Miyahara, E. (2003). Focal colors and unique hues. *Percept. Motor Skills* 97, 1038–1042.
- Pridmore, R. (2006). 14th century example of the four unique hues. *Color Res. Appln.* 31, 364–365.
- Berlin, B., and Kay, P. (1969). *Basic Color Terms: Their Universality and Evolution* (Berkeley, CA: University of California Press).
- Regier, T., Kay, P., and Cook, R.S. (2005). Focal colors are universal after all. *Proc. Natl. Acad. Sci. USA* 102, 8386–8391.
- Webster, M.A., Miyahara, E., Malkoc, G., and Raker, V.E. (2000). Variations in normal color vision. II. Unique hues. *J. Opt. Soc. Am. A Opt. Image Sci. Vis.* 17, 1545–1555.
- Hinks, D., Cardenas, L.M., Kuehni, R.G., and Shamey, R. (2007). Unique-hue stimulus selection using Munsell color chips. *J. Opt. Soc. Am. A Opt. Image Sci. Vis.* 24, 3371–3378.
- Valberg, A. (2001). Unique hues: an old problem for a new generation. *Vision Res.* 41, 1645–1657.
- Jordan, G., and Mollon, J.D. (1997). On the nature of unique hues. In *John Dalton's Colour Vision Legacy*, C. Dickenson, I. Murray, and D. Carden, eds. (Taylor and Francis: London), pp. 381–392.
- De Valois, R.L., Abramov, I., and Jacobs, G.H. (1966). Analysis of response patterns of LGN cells. *J. Opt. Soc. Am.* 56, 966–977.
- Wuerger, S.M., Atkinson, P., and Cropper, S. (2005). The cone inputs to the unique-hue mechanisms. *Vision Res.* 45, 3210–3223.
- Guth, S.L. (1991). Model for color vision and light adaptation. *J. Opt. Soc. Am. A* 8, 976–993.
- De Valois, R.L., and De Valois, K.K. (1993). A multi-stage color model. *Vision Res.* 33, 1053–1065.
- Lennie, P., Krauskopf, J., and Sclar, G. (1990). Chromatic mechanisms in striate cortex of macaque. *J. Neurosci.* 10, 649–669.
- Conway, B.R. (2001). Spatial structure of cone inputs to color cells in alert macaque primary visual cortex (V-1). *J. Neurosci.* 21, 2768–2783.
- Conway, B.R., and Livingstone, M.S. (2006). Spatial and temporal properties of cone signals in alert macaque primary visual cortex. *J. Neurosci.* 26, 10826–10846.
- Kiper, D.C., Fenstemaker, S.B., and Gegenfurtner, K.R. (1997). Chromatic properties of neurons in macaque area V2. *Vis. Neurosci.* 14, 1061–1072.
- Derrington, A.M., Krauskopf, J., and Lennie, P. (1984). Chromatic mechanisms in lateral geniculate nucleus of macaque. *J. Physiol.* 357, 241–265.
- Conway, B.R., and Tsao, D.Y. (2006). Color architecture in alert macaque cortex revealed by fMRI. *Cerebr. Cortex* 16, 1604–1613.
- Conway, B.R., Moeller, S., and Tsao, D.Y. (2007). Specialized color modules in macaque extrastriate cortex. *Neuron* 56, 560–573.
- Zeki, S. (1980). The representation of colours in the cerebral cortex. *Nature* 284, 412–418.
- Komatsu, H., Ideura, Y., Kaji, S., and Yamane, S. (1992). Color selectivity of neurons in the inferior temporal cortex of the awake macaque monkey. *J. Neurosci.* 12, 408–424.
- Pridmore, R.W. (1999). Unique and binary hues as functions of luminance and illuminant color temperature, and relations with invariant hues. *Vision Res.* 39, 3892–3908.

Neuroscience Program, Wellesley College, Wellesley, Massachusetts 02481, USA.
E-mail: bconway@wellesley.edu

Supplemental Data: Neural basis for unique hues

Cleo M. Stoughton and Bevil R. Conway

Table S1 CIE xyY values for the stimuli used to measure color tuning; color number begins with red (corresponding to 0 degrees in the polar plot, Figure 1), and proceeds counterclockwise with orange-red (8 degrees) etc. Each of the three color sets were equiluminant with each other; the “Dark colors” were lower luminance than the adapting gray background; the “Equiluminant colors” were equiluminant with the adapting background; and the “Bright colors” were higher luminance than the adapting background.

color	Dark colors			Equiluminant colors			Bright colors		
	x	y	Luminance (cd/m ²)	x	y	Luminance (cd/m ²)	x	y	Luminance (cd/m ²)
0	0.616	0.354	0.6	0.616	0.355	3.11	0.615	0.354	7.8
8	0.61	0.375	0.63	0.61	0.357	3.04	0.611	0.355	7.73
16	0.586	0.359	0.67	0.594	0.369	3.05	0.595	0.368	7.73
24	0.559	0.396	0.68	0.561	0.396	3.06	0.56	0.395	7.77
32	0.528	0.434	0.58	0.515	0.426	3.14	0.518	0.427	7.77
40	0.487	0.473	0.61	0.475	0.458	2.96	0.475	0.459	7.95
48	0.44	0.481	0.62	0.438	0.489	3.07	0.437	0.49	7.76
56	0.391	0.51	0.64	0.404	0.508	3.09	0.406	0.513	7.69
64	0.337	0.528	0.58	0.37	0.541	3.08	0.381	0.532	7.86
72	0.363	0.544	0.58	0.348	0.555	3.01	0.355	0.553	7.85
80	0.292	0.607	0.6	0.324	0.576	3.04	0.336	0.565	7.79
88	0.299	0.606	0.64	0.309	0.585	3.06	0.314	0.583	7.69
96	0.31	0.578	0.64	0.303	0.593	3	0.298	0.596	7.79
104	0.325	0.564	0.64	0.292	0.599	3.05	0.297	0.597	7.74
112	0.299	0.605	0.64	0.295	0.593	3.05	0.293	0.601	7.84
120	0.288	0.612	0.62	0.29	0.601	3.04	0.291	0.599	7.85
128	0.189	0.26	0.6	0.257	0.477	3.01	0.274	0.546	7.75
136	0.206	0.254	0.6	0.26	0.476	3.03	0.275	0.546	7.74
144	0.206	0.255	0.6	0.254	0.48	3.03	0.261	0.496	7.86
152	0.2	0.28	0.67	0.257	0.478	3.03	0.248	0.447	7.79
160	0.207	0.262	0.6	0.232	0.387	3.04	0.237	0.41	7.72
168	0.197	0.258	0.6	0.216	0.321	3.06	0.222	0.347	7.8
176	0.208	0.278	0.67	0.203	0.276	3.09	0.204	0.286	7.85
184	0.207	0.257	0.6	0.191	0.237	3.03	0.196	0.256	7.77
192	0.172	0.182	0.7	0.182	0.194	3.18	0.184	0.212	7.75
200	0.176	0.161	0.61	0.17	0.159	3.07	0.173	0.169	7.88
208	0.158	0.113	0.61	0.16	0.127	3	0.162	0.131	7.74
216	0.152	0.086	0.61	0.152	0.096	3.11	0.154	0.1	7.8
224	0.148	0.074	0.5	0.147	0.077	3.11	0.148	0.078	7.81
232	0.145	0.068	0.59	0.144	0.067	3	0.145	0.066	7.75
240	0.141	0.066	0.57	0.144	0.064	3.06	0.144	0.064	7.74
248	0.145	0.066	0.7	0.144	0.064	3	0.145	0.064	7.75
256	0.147	0.066	0.7	0.147	0.066	2.98	0.149	0.067	7.87
264	0.154	0.072	0.64	0.157	0.072	3.05	0.159	0.073	7.74
272	0.17	0.081	0.58	0.171	0.081	3.06	0.175	0.083	7.88
280	0.18	0.087	0.63	0.191	0.093	2.96	0.195	0.095	7.82
288	0.21	0.105	0.6	0.211	0.105	3.08	0.22	0.111	7.77
296	0.207	0.104	0.59	0.242	0.124	3.06	0.247	0.128	7.84
304	0.259	0.138	0.56	0.271	0.143	3.12	0.281	0.148	7.8
312	0.279	0.146	0.64	0.305	0.164	3.08	0.318	0.17	7.76
320	0.36	0.192	0.54	0.358	0.194	3.09	0.371	0.203	7.68
328	0.372	0.212	0.61	0.431	0.242	3.08	0.433	0.242	7.8
336	0.394	0.221	0.7	0.481	0.273	3.03	0.498	0.282	7.78
344	0.379	0.208	0.62	0.542	0.311	3.09	0.555	0.317	7.84
352	0.379	0.208	0.62	0.544	0.31	3.09	0.584	0.335	7.85

Background: (x,y, luminance): 0.316, 0.314, 3.05 cd/m²; **white:** 0.274, 0.303, 77.4 cd/m²; **black:** 0.02 cd/m²

Figure S1 Color tuning of glob cells recorded in alert macaque monkey, shown as a bubble plot [22] on the CIE $u'v'$ diagram. Size of each bubble corresponds to the number of cells with peak tuning to that color; total number of cells, 308. The triangle represents the maximum gamut of the color monitor display. Stimuli were optimally configured bars that were equiluminant with each other and with the background gray (same data as shown by the thick dark-gray line in Figure 1; “Equiluminant colors”, Table 1).

