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Color Perception

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5 Introduction

6 *Color* is the name we assign to the experience elicited by an attribute of a surface, namely, its spectral reflectance. Color sensations have a reliable, though complex, relationship to the spectral composition of light received by the eyes. The visual system tackles a series of computational problems in the course of processing wavelength. Variation in the wavelength of light is isolated from variation in its intensity. The spectral reflectance properties of surfaces are isolated from the effects of the spectral composition of light illuminating them (matching surfaces with the same reflectance properties in different parts of the visual scene or under different illuminants are the two problems of color constancy). Finally, the resulting continuous color space is partitioned into discrete color categories. In addition, it will become clear that wavelength signals can be used in the course of perceiving form or motion, independent of their role in the subjective experience of color.

21 Wavelength-Dependent Differences

22 Within the Visual System

23 Color percepts derive from light that varies in both wavelength and intensity. A single type of photoreceptor in the eye responds with differing efficiency to light over a wide range of wavelengths. Consequently, a visual system in which there is only a single type of photoreceptor inevitably confounds wavelength and intensity. A visual system containing photoreceptors that differ in their spectral response can, in principle, disambiguate wavelength and intensity by comparing the responses of different types of receptors.

31 Receptors

32 Wavelength-selective processing can be traced from differentially wavelength-sensitive cone types in the retina to the lateral geniculate nucleus (LGN) and then on to striate cortex and extrastriate areas beyond it. There are three cone types in the human retina, with peak sensitivities at 560 nm, 530 nm, and 430 nm and referred to as L, M, and S (long-, medium- and short-wavelength-sensitive) cones, respectively. In some people one or more of these cone types are missing; hence, color sensations that would normally be perceived as distinct are confused, and the individuals are “color-blind.” The functions relating the sensitivities of these cone types to the wavelength of stimulating light can be inferred by comparing the wavelength sensitivities of color-blind and normal observers or by examining the effects of adaptation to light of one wavelength on sensitivity to light of other wavelengths. Figure 1 shows the relative absorption efficiencies of the three cone types and the typical pattern of behavioral sensitivity to intensity modulation.

48 The output of cones provides information about an object’s state, for example, allowing ripe and unripe fruit to be discriminated. The peak sensitivities of photoreceptors appear exquisitely matched to maximize the discriminability of the foliage or fruits that form the diets of a number of species of primates (Sumner and Mollon, 2000). Studies of the genetic coding of cone pigments indicate that human trichromacy evolved from ancestral dichromacy through the division of a single long-wavelength-sensitive pigment into distinct L and M pigments (Bowmaker, 1998).

57 *The Combination of Receptor Signals in the M, P, and K Channels*

59 Three anatomically distinct cell types in the retina combine cone signals in distinct ways (Dacey, 2000). In all cases, the response has a “center-surround” organization. A set of cones from one part

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62 of the visual field influences the cell in one way, while a set of
63 cones from the surrounding area influences it in a different way.
64 Parasol cells receive input from L and M, but not S, cones. Inputs
65 from L and M cones are summed in both the center and surround
66 fields of parasol cells (Figure 2A). Parasol cells cannot convey in-
67 formation about wavelength independent of intensity. They project
68 to the magnocellular layer of the LGN, which in turn projects to
69 layers 4C α and 4B of primary visual cortex (V1). This pathway,
70 and its onward projections, is known as the M-channel. The M-
71 channel contributes to the perception of luminance and motion but
72 does not convey wavelength-coded signals.

73 Midget ganglion cells have color-opponent receptive fields. This
74 center-surround organization sharpens the effective wavelength se-
75 lectivity of the ganglion cell, helping to unconfound wavelength
76 and intensity variation. Consider first a nonopponent cell, sensitive
77 to medium wavelength light. This cell will produce the same re-
78 sponse to a given intensity of medium wavelength light, or a
79 stronger intensity of longer wavelength light. Although its peak
80 sensitivity is to medium rather than longer wavelength light, be-
81 cause sensitivity only reduces gradually as wavelength deviates
82 from the peak, the longer wavelength light still produces a re-
83 sponse. Now consider the responses of an opponent cell excited by
84 medium wavelength light in the center of its field and inhibited by
85 long wavelength light in the surround to different intensities and
86 wavelengths of light falling on its entire receptive field. Medium
87 wavelength light produces excitation in the center and no inhibition
88 in the surround; there is a net increase in the cell's firing rate.
89 Higher intensities of medium wavelength light elicit stronger net
90 responses. A slightly longer wavelength produces some excitation
91 in the center field of the ganglion cell but also a small inhibitory
92 response in the surround. These roughly balance, and so the firing
93 rate of the cell is largely unaffected by the stimulus. The same
94 situation applies to a high-intensity stimulus; again, central exci-
95 tation is balanced by surround inhibition. This ganglion cell is
96 therefore capable of conveying information solely about the inten-
97 sity of medium wavelength light.

98 The vast majority of foveal midget ganglion cells are driven by
99 L or by M cones in the center of their receptive field; these centers
100 can be either excitatory or inhibitory. Away from the fovea, midget
101 ganglion cells lose their spectral opponency, as more than one cone
102 type drives both the center and surround. There appears to be little
103 input from S cones to midget ganglion cells, just as there are very
104 few S cones in the retina. About 2%–3% of parafoveal midget
105 ganglion cells have S-OFF central receptive fields with an ON sur-
106 round driven by both L and M inputs (Figure 2B). There are no S-
107 ON center midget ganglion cells.

108 Small bistratified ganglion cells receive inputs from all three
109 cone types; however, their central field always appears to be driven
110 by an excitatory input from S cones, while their surround combines
111 inhibitory L and M inputs. The bipolar cells that convey signals
112 from cones to the central field receive inputs only from S cones
113 and are driven by multiple cells, unlike the bipolar cells that drive
114 the central fields of midget ganglion cells, which receive inputs
115 from single cones. The result is that, although these cells do show
116 clear spatial and spectral opponency, the size of the central field
117 (100 μm standard deviation) is much larger than that found in
118 midget ganglion cells (25 μm) (Figure 2C). The surround fields of
119 small bistratified cells are smaller than those of midget ganglion
120 cells (140 μm and 205 μm , respectively), so these cells show rela-
121 tively weak spatial opponency. One additional consequence of the
122 S-cone specificity of the small bistratified cells is that the S-ON
123 center, LM-OFF surround organization extends into the peripheral
124 visual field, whereas midget ganglion cells lose spectral opponency
125 beyond the parafovea. There are no S cones in the central 0.3 de-
126 grees of the visual field, so foveal vision is effectively color-blind
127 to color variation mediated by S cones.

128 Midget ganglion cells project to the parvocellular layer of the
129 LGN, and thence to layer 4C β of V1. This pathway, and its onward
130 projections, is known as the P-channel. The P-channel conveys
131 information about long and medium wavelengths and fine detail.
132 It has been suggested that small bistratified ganglion cells convey-
133 ing short wavelength information also contribute to the P-channel.
134 However, it is now widely believed that small bistratified cells

135 drive a distinct class of geniculate cells. The P-channel does con-
136 tribute to motion perception; however, its contribution is weaker
137 than that of the M-channel and nonveridical—the speed of per-
138 ceived motion depends on the chromatic contrast of the stimulus.

139 Small bistratified ganglion cells form the start of the K-channel
140 (Hendry and Reid, 2000). They project to koniocellular neurons in
141 the LGN, distinguished from magno- and parvocells on the basis
142 of their cell membrane chemistry. These cells mainly form layers
143 intercalated between the parvo- and magnocellular layers, but some
144 K-cells are also found in the parvocellular layer, with a smaller
145 number being found in the magnocellular layer. K-cells project not
146 only to layer 1 of V1, but also directly to V2. There is a particularly
147 rich innervation of V2 by K-cells with foveal receptive fields. K-
148 cells' receptive fields are large (at least as large as those of cells in
149 the magnocellular layer) and often have irregular shapes. K-cells
150 convey information contributing to color sensations, depending on
151 contrasts of the output of S cones to combinations of M and L cone
152 outputs; they may also contribute to motion perception.

153 The position summarized above remains controversial and has
154 been challenged on a number of counts. In particular, it has been
155 argued that the K-channel alone conveys chromatic signals (in-
156 cluding L versus M information), while the P-channel is dedicated
157 to fine spatial vision (Calkins and Sterling, 1999).

158 *Primary Visual Cortex*

159 The M, P, and K pathways project to groups of cells within V1
160 that can be distinguished on the basis of cytochrome oxidase reac-
161 tivity (Livingstone and Hubel, 1984). K and P, but not M, pathways
162 innervate cytochrome oxidase-stained regions known as blobs. P
163 and M, but not K, pathways innervate the remaining regions,
164 known as interblobs. There is recent evidence that cells show dif-
165 ferent specificities for wavelength processing in V1 (Conway,
166 2001; Johnson, Hawken, and Shapley, 2001). The cells discussed
167 earlier in this article had a “single-opponent” organization. They
168 can convey information about the intensities of light of particular
169 wavelengths while being relatively uninfluenced by other wave-
170 lengths. They cannot, however, convey information about wave-
171 length contrast. This requires “double-opponent” cells in which a
172 central receptive field excited by one wavelength and inhibited by
173 another is surrounded by a field in which the same two wavelengths
174 have the opposite actions (Figure 1D). Double-opponent organi-
175 zation allows a cell to convey a consistent response to the boundary
176 between two surfaces, regardless of the light illuminating them. If
177 the illuminant changes, for example lengthening in wavelength,
178 then longer wavelength light will be reflected from both sides of
179 the boundary. Consider a double-opponent cell whose central re-
180 ceptive field is excited by long wavelengths and inhibited by me-
181 dium wavelengths and whose surrounding field is inhibited by long
182 wavelengths and excited by medium wavelengths. Imagine that the
183 cell's receptive fields fall on a boundary between a pair of surfaces,
184 one of which is good and one poor at reflecting long wavelength
185 light, so that the good reflector falls in the cell's central field. The
186 net result will be excitation—that ratio of long to medium wave-
187 length light is high in the central field and low in the surround.
188 When the light illuminating both sides of the boundary lengthens
189 in wavelength, the L/M ratios in both the excitatory center and the
190 inhibitory surround will increase. The response of the cell is there-
191 fore largely unaffected by a change in illuminant. Obviously, such
192 cells perform the preliminary computation necessary for color con-
193 stancy. Of course, their responses only indicate spatially local
194 changes in surface reflectance. To recover absolute reflectances
195 throughout a scene, then, one also needs to estimate the response
196 likely to be elicited by some fixed “anchoring” color in that scene
197 and then to integrate local border contrasts from that anchoring
198 point (see, e.g., Gilchrist et al., 1999, for similar arguments with
199 respect to lightness perception). Until recently, evidence for the
200 existence of double-opponent cells was controversial; however, re-
201 cent findings indicate that such cells occur in V1 and, moreover,
202 are sensitive to the orientation of chromatic (wavelength-depen-
203 dent) borders as well as to the contrast of cone ratios across them.

204 *Extrastriate Cortex*

205 The clinical condition of cerebral achromatopsia, in which patients
206 lose the ability to perceive color not as a result of retinal abnor-
207 malities but rather as a consequence of brain damage, provides
208 strong evidence that brain areas specialized for color perception
209 exist beyond striate cortex. The identification of these areas is, how-
210 ever, wreathed in controversy. The damaged areas include extra-
211 striate cortex in the vicinity of the fusiform and lingual gyri. Neu-
212 roimaging studies have also shown increases in cerebral blood flow
213 (implying increased brain activity) in these areas when normal sub-
214 jects observed colored scenes. Zeki et al. (1991) therefore sug-
215 gested that there was a specific color center in human extrastriate
216 cortex. Early studies in which the responses from single neurons
217 in monkeys were recorded in response to visual stimuli suggested
218 that the color center might correspond to cortical area V4. A num-
219 ber of problems arose with this interpretation. The selectivity of
220 the response of neurons to particular characteristics of stimuli dif-
221 fers only in degree between brain areas. Some neurons in nearly
222 all visual areas respond selectively to wavelength; the proportion
223 in V4 is not comparatively large. In addition, damage to area V4
224 in monkeys did not cause deficits in discriminations based on wave-
225 length, although deficits were induced by damage to areas anterior
226 to V4 (Heywood and Cowey, 1998). These findings appeared con-
227 sistent with neuroimaging studies in humans identifying a color-
228 selective area anterior to V4, christened V8 (Hadjikhani et al.,
229 1998). Whether V8 really corresponds to the anterior areas that,
230 when damaged, caused deficits for wavelength discrimination in
231 monkeys remains controversial.

232 **Wavelength Information Contributes to More Than** 233 **Color Perception**

234 The fact that our visual system can disambiguate wavelength and
235 intensity makes it possible to ignore variations or sharp changes in
236 intensity caused by shadows. One role of a wavelength-selective
237 visual system is therefore segmentation of the visual scene on the
238 basis of chromatic boundaries. Often chromatic boundaries will
239 provide better cues for segmenting objects from their backgrounds
240 than brightness boundaries—for example, in the dappled sunlight
241 of a forest floor.

242 The residual abilities found in cerebral achromatopsia indicate
243 that wavelength is exploited in more than one way. Although ce-
244 rebral achromatopsics deny a phenomenal experience of color and
245 cannot discriminate between stimuli differing only in wavelength,
246 they can effortlessly perceive boundaries between areas differing
247 only in wavelength (Heywood, Kentridge, and Cowey 1998). Their
248 ability to use wavelength information to perceive form or motion,
249 but not to perceive color suggests, that these functions may have
250 distinct anatomical bases. Destruction of the putative color center,
251 be it V4 or V8, disrupts the perception and experience of color, but
252 not other functional uses of wavelength.

253 **Discussion**

254 Some of the earliest insights into the coding of color derived from
255 work on color mixing. Following his discovery of the composition
256 of white light, Newton developed the concept of the color circle,
257 an arrangement of light sources around the periphery of a circle in
258 which the mixture of any pair of diametrically opposite lights
259 would produce white. Despite the color circle showing a continuum
260 of light sources, Newton identified five primary colors (red, yellow,
261 green, blue, and a violet-purple). However, attempts were soon
262 made to discover how few colors were required in order to produce
263 all other colors by mixing. Although there was some disagreement
264 about which colors were primary, it was apparent to most investi-
265 gators that three were sufficient. This culminated in the Young-
266 Helmholtz trichromatic theory of color vision. Young believed that
267 the primaries were red, green, and violet.

268 The fact that we require three primary colors in order to produce
269 the full range of colored sensations reflects the fact that we have
270 photoreceptors sensitive to three distinct wavelength distributions.

271 The consequence is that any combination of lights that produces
 272 the same amount of activation in the three receptor types will pro-
 273 duce the same response in the visual system and the same percep-
 274 tion of color. There are, therefore, a large number of colors that are
 275 potential primaries.

276 Other features of color perception suggest an alternative to the
 277 trichromatic theory. In particular, there are limits to our abilities to
 278 see pairs of colors tinting one another. People perceive bluish reds
 279 and yellowish reds, but never greenish reds; they perceive reddish
 280 yellows and greenish yellows, but never bluish yellows. These op-
 281 ponent color pairings, red-green and blue-yellow, are also apparent
 282 in afterimages, color shadows, and color contrast. Observations
 283 such as these led Hering in 1905 to suggest a four-color opponent-
 284 process theory of color vision.

285 Both these theories assumed that the similarities between color
 286 sensations are completely determined by the outputs of the wave-
 287 length-dependent neurons in the visual system. For example, it is
 288 tempting to believe that opponent processes operating in V1 are
 289 the direct precursors of our space of colors. They have been taken
 290 as the sources of four primary colors (red, green, yellow, and blue)
 291 that are irreducible to other colors, and each contains one sensation
 292 that is pure (unique) in that it contains no trace of any other pri-
 293 mary. However, the outputs of the cells in V1 would not produce
 294 these unique colors even if there were agreement as to what they
 295 might be (Saunders and van Brakel, 1997; Webster et al., 2000).
 296 Nor would the categories of color arise from variation in discrim-
 297 ination across the visible spectrum. The wavelengths at which there
 298 are minima in threshold do not correspond to the boundaries be-
 299 tween primary colors. Controversially, it has been argued from
 300 cross-lingual evidence that color categories are determined by the
 301 speaker's color terms. The neurophysiology produces a given per-
 302 cept, but the assignment of that percept to a color category is a
 303 matter of agreement among observers.

304 **Roadmap:** Vision

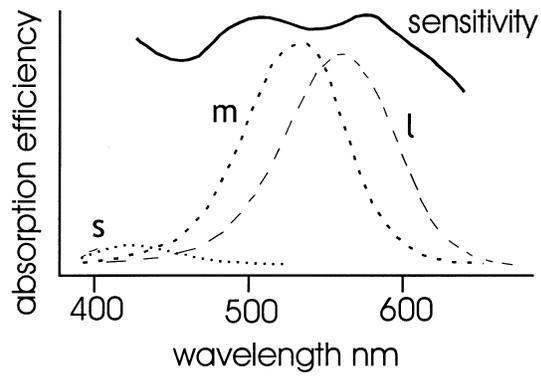
305 **Related Reading:** Contour and Surface Perception; Retina

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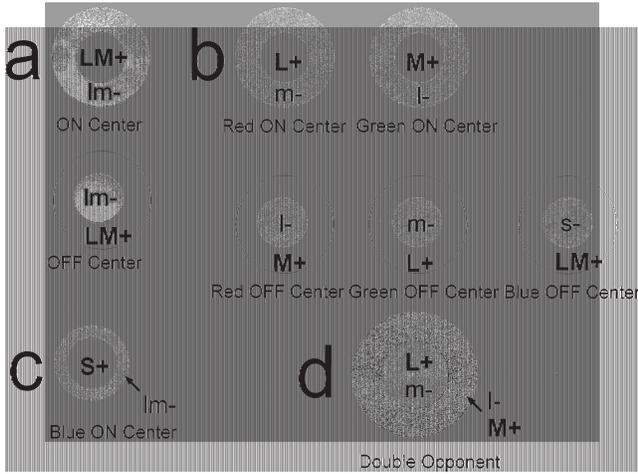
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354 **Figure 1.** The relative absorption efficiencies of short-, medium-, and long-
355 wavelength cone types, labeled s, m and l, are shown as dotted, short-
356 dashed, and long-dashed lines, respectively. The solid line shows sensitivity
357 to increments in luminance for lights of different wavelengths. The sensi-
358 tivity decreases falling between the peaks of the cone absorption spectra
360 are known as Sloan-Crawford notches.

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Figure 2. Schematic representations of receptive field organization of cells in (A) the M-channel, (B) the P-channel, and (C) the K-channel; (D) an example of receptive field organization of a cortical double-opponent cell.