

Lecture 29 - Color Opponent Theory, Color Anomalies

REVIEW FROM LECTURE 28

Relative saturation – minimum at 570 yellow

Wavelength discrimination – W-shaped curve, best for wavelengths slightly < than 500 & 600 nm

Bezold-Brücke phenomenon – Understand Schwartz Fig. 5-15.

Below ~500 nm, monochromatic hues become more blue (~480 nm) with increasing intensity.

Above ~500 nm, monochromatic hues become more yellow (~580 nm) with increasing intensity.

Abney's phenomenon – changes in hue increasing saturation.

Color constancy - even slight changes in lighting and wavelength, color look the same.

Simultaneous contrast and **simultaneous color contrast** - A Google search for "simultaneous color contrast" brought up the following web sites.

<http://www.webexhibits.org/colorart/contrast.html>

http://colorusage.arc.nasa.gov/Simult_and_succ_cont.php

COLOR OPPONENT THEORY

The trichromatic theory of color vision is based on three cone types, each of which is sensitive to a different range of wavelengths. This explains how we can discriminate different wavelengths, and the trichromatic theory is supported by color matching experiments and neurophysiological studies.

The neural processing of color becomes more complex beyond the photoreceptors. There is evidence that after the cones, neural signals are processed through **color opponent** channels. Opponent processing begins within the retina at the level of the horizontal cells. The theory states that signals from the S, M and L cones are combined and processed by three neural channels:

- **red-green opponent channel**
- **blue-yellow opponent channel**
- **brightness non-opponent channels.**

Neurons that respond with opposite signal polarity (excitation / inhibition; + / -) depending on the stimulus wavelength are known as **color opponents cells**. This illustrated by Figure 1, below.

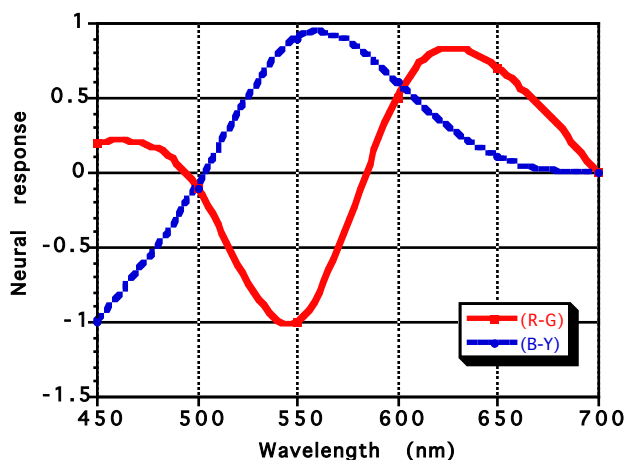


Figure 1. Opponent channels

This kind of signal processing enables the visual system to relay wavelength-dependent color information. Scientific studies have shown that our visual system includes color opponent neurons as well as non-color opponent neurons. Non-opponent cells respond with the same polarity for all wavelengths, though the magnitude of the response varies depending on wavelength.

Evidence of this kind of neural processing:

- Generally red and green are conceived to be opposite responses of a neural system that can signal either red or green but not both at the same time. Although we can conceive of a color such as blue-green, we cannot conceive of a color that is red-green.
- Similarly, with blue and yellow, most people cannot conceive of a color that is blue-yellow.
- Research by Hurvich and Jameson using the hue-cancellation method (See Schwartz p. 113-114) support color opponent theory. They proposed that the response of certain neuronal channels in the visual system varies in according to wavelength as shown in Figure 1 above. (Schwartz Fig. 5-16)
- Neurophysiological recordings from monkey LGN cells showed that certain neurons respond with opposite polarity depending on the wavelength (Schwartz Fig. 5-18A). For example, if the animal is exposed to a short wavelength, the neuron shows a reduced rate of firing; that is, inhibition. However, when exposed to long wavelengths, the neuron responds with excitation.
- Color afterimages. If you stare at a green image, then view a white field, you see a red afterimage. Likewise, if you stare at a blue image, then view a white field, you see a yellow afterimage. For examples, visit the IllusionWorks web site, (http://psylux.psych.tu-dresden.de/i1/kaw/diverses/Material/www.illusionworks.com/html/color_aftereffect.html) or see Figure 2 below. If you stare at the American flag, which is made with complementary colors, then look at a white field, you should see a normally colored American flag.

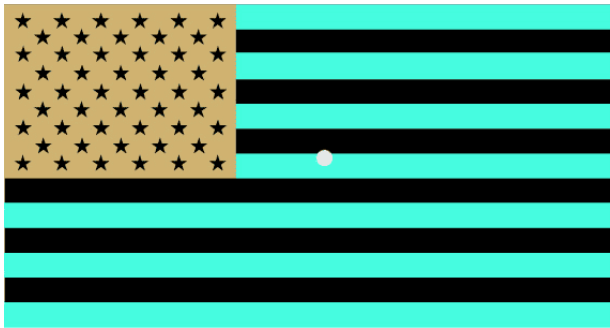


Figure 2. Color after-image example.

Over 100 years ago, Hering proposed that human color perception is based on red-green and blue-yellow color opponent channels, but scientists of that time thought opponent theory contradicted the already-accepted trichromatic theory. Hering's opponent theory of color vision was therefore not accepted.

Vision scientists now believe that both the trichromatic and opponent systems work together. At the receptor level, our visual system is trichromatic, but at higher levels, perhaps beginning with the horizontal cells, the visual system appears to use color opponent mechanisms. This combined model of color vision is called a **zone model** (Figure 3).

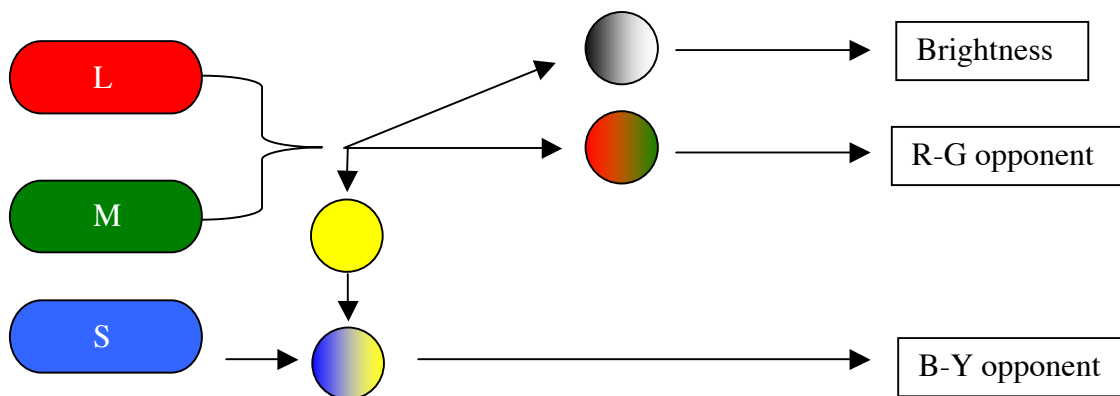


Figure 3. One example of a zone model

INTRODUCTION TO ANOMALIES OF COLOR VISION (Schwartz Chapter 6)

Anomalies of color vision affect about 4.5% of the general population, or nearly 1/20 people. You will therefore certainly see many patients who have some degree of anomalous color vision. Color anomalies may be broadly classified by etiology as either hereditary or acquired color defects. Characteristics of these two groups are distinct and are summarized below.

Hereditary anomalies

- More common
- Non-progressive, stable over time
- Usually don't confuse colors in real life
- Not sight threatening
- May affect job performance when color perception is critical to certain tasks
- Mostly affects males (96%)
- Binocular, or symmetric between eyes
- Diagnosis and classification straight-forward
- 99% of these are red-green (L or M cone) defects

Acquired color anomalies

- Rare
- Associated with ocular disease, therefore potentially sight threatening
- Recent onset, progressive, variable
- The patients more likely to confuse colors
- Affects males and females
- Monocular or asymmetric
- Diagnosis and classification more difficult
- Red-green (L-M) as well as blue-yellow (S) defects

TYPES OF COLOR ANOMALIES

The defects can be classified according to which cone photopigment is either missing or anomalous.

- **Protan-**_____ refers to an anomaly with erythrolabe (red deficient; L cone)
- **Deutan-**_____ refers to an anomaly with chlorolabe (green deficient; M cone)
- **Tritan-**_____ refers to an anomaly with cyanolabe (blue deficient; S cone)

The prefixes, proto, deuto and trito respectively mean, primary, secondary and tertiary. To help you remember which prefix refers to which anomaly, number the L, M and S cones in order starting from the long wavelengths; that is number them respectively 1, 2, 3 for the L, M, S cones defects (proto, deuto, trito).

Classification by magnitude

Color defects may also be classified according to the magnitude of the defect. Absolute color blindness of some kind ends with the suffix **-opia**. Normal vision is trichromatic, but people who are completely missing one of the cone types are known as **dichromats**.

- Absence of one of the three cones photopigments
- A more absolute form of "color blindness"
- Three types depending on which pigment is missing: **protanopia, deuteranopia, tritanopia**

Other people may have all three cone types, but there may be a weakness or anomaly in one of the photopigment. These partial color anomalies use the suffix, **-anomaly**. These people are known as **anomalous trichromats**.

- All three photopigments present (erythrolabe; L, chlorolabe; M, cyanolabe; S)
- One has an abnormal absorption spectrum that is shifted toward one of the other pigment's spectra.
- Protanomalous trichromat - erythrolabe spectrum shifted toward shorter wavelengths, making it closer to chlorolabe.
- Deuteranomalous trichromat - chlorolabe spectrum shifted toward longer wavelength, making it more similar to erythrolabe.
- See Schwartz Fig. 6-1.

Protans and deutans are sometimes referred to as **red-green** anomalous. These defects are usually hereditary, and are more common. The color response of patients with red-green anomalies are well defined.

Tritans are referred to as **blue-yellow** anomalous. Hereditary blue-yellow defects are rare. If detected, you should presume that it is an acquired defect caused by an ocular disease, until proved otherwise. You may see references to another color anomaly called, **tetartanomaly**. It is a rare subtype of tritanomaly.

Table 1. Summary of classifications

	red (protan)	green (deutan)	blue (tritan)
Dichromat	<i>protanopia</i> no erythrolabe (Patient is a protanope)	<i>deutanopia</i> no chlorable (Patient is a deuteranope)	<i>tritanopia</i> no cyanolabe (Patient is a tritanope)
Anomalous trichromat (Schwartz Fig. 6-1)	<i>Protanomaly</i> abnormal erythrolabe (normal peak @ 565nm) absorption spectrum shifted to shorter λ	<i>deuteranomaly</i> abnormal chlorable (normal peak @ 535 nm) absorption spectrum shifted to longer λ	<i>tritanomaly</i> abnormal cyanolabe (normal peak @ 430 nm)
Etiology usually..	heredity	heredity	acquired (rare)