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A

# A COMPARISON OF VISUAL FUNCTIONS BETWEEN COLOR-NORMAL AND COLOR-DEFICIENT OBSERVERS

by

HOWARD MARK DELMAN

A dissertation submitted to the Graduate Faculty in Psychology

in partial fulfillment of the requirements for the degree of

Doctor of Philosophy, The City University of New York

2001

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3/20/01
Date

Chair of Examining Committee

3/20/01
Date

Executive Officer

Dr. Israel Abramov

Dr. Sheila Chase

Dr. Louise Hainline

Dr. Vance Zemon

Supervisory Committee

THE CITY UNIVERSITY OF NEW YORK

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Abstract

A COMPARISON OF VISUAL FUNCTIONS

BETWEEN COLOR-NORMAL AND COLOR-DEFICIENT OBSERVERS

by

Howard Mark Delman

Adviser: Professor James Gordon

Human visual information processing is controlled by two largely separate parallel pathways: the magnocellular and the parvocellular. One approach to ascertaining pathway function is through the study of color-deficient individuals, one of several populations in which one of the pathways is naturally altered. Based on reduced ability to make certain chromatic discriminations and on different color perceptions compared to color-normals, color-deficients are presumed to have an altered parvocellular pathway. In order to determine which visual pathway serves which visual function, color-normal and color-deficient observers were run on an extensive battery of visual tests including color detection, color discrimination, hue- and saturation-scaling, spatio-temporal contrast sensitivity, binocular vision, stereopsis, monocular hyperacuity, and motion sensitivity. Results show two principal findings. First, genetic color-anomalous observers, as a group, have equal or greater contrast sensitivity to nearly all spatio-temporal frequency combinations, with the greatest effects at higher spatial frequency stimuli bordering on "supersensitivity." These individuals also displayed better stereoacuity and stereopsis, superior vernier acuity thresholds, and smaller minimum motion threshold values compared to the color-normals. The enhanced sensitivities found among these visual functions are discussed in terms of a common mechanism resulting from a reduction of

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chromatic antagonistic signals in the retinas of this subpopulation of observers, and the resulting gain in detection of luminance differences at high spatial frequency. Several possible versions of neural rewiring are considered and tracked from the level of the cone mosaic to higher cortical levels and advanced stages of information processing. Second, separate standard color-screening tests giving different diagnoses indicates a greater degree of overlap in characteristic traits exhibited by each subtype, indicating that color deficiencies are a more complex phenomenon than has been generally thought, with dimensions not clearly conveyed by existing designations. Additional experiments and directions for future research in the delineation of pathway function and control using a more varied sample of observers are suggested. Some of the potential clinical implications of the findings from the current investigation are also explored.

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Myself: for following my dreams

and I:...am a Ph.D.!!!

---

An optimist is a person who sees a green light everywhere,
while a pessimist sees only the red stop light...
but the truly wise person is colorblind.

---Dr. Albert Schweitzer (1955)

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## Overview

Since they were first identified, the magnocellular (M) and parvocellular (P) pathways have been shown to play essential roles in common, everyday visual functions, as well as in complex information processing at higher cortical levels. Through neuroanatomical, neurophysiological, and psychophysical studies, it has become evident that, while these pathways are initially segregated, there is a greater degree of cross-talk and interaction between the two pathways than was first believed. The number of studies involving human participants remains small, due mostly to experimental procedures that are too invasive in nature. As a result, our understanding of the neural wiring of the M and P pathways in man is largely presumptive, being based primarily on certain species of primates whose visual systems are highly congruent with our own.

The goal of this experiment is to study a population of individuals who are color-deficient and who must necessarily have different neural connections compared to color-normals. By measuring specific visual functions known to be primarily controlled by either the M or P pathway, and comparing each population's results, we will be able to gain further insight into the neural mechanisms and roles of the magnocellular and parvocellular pathways in visual perception.

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Introduction

Human visual information processing is achieved through various sets of parallel pathways. One set that continues to receive considerable attention is comprised of the magnocellular (M) and parvocellular (P) pathways. These pathways, so named for the sizes of the cells found in each respective pathway (i.e., magno is derived from the Latin "magnus," meaning large; parvo is derived from the Latin "parvus," meaning small [Simpson, 1977]), have been found to serve important and fundamental visual functions, including perception of color, shape, and motion, as well as stereopsis.

To more fully understand the roles that the M and P pathways play in these and other functions, we must first look at how these pathways are organized in the brain, on both the anatomical and physiological levels. Then, through the use of psychophysical procedures, we may be better able to elucidate the contributions made by each pathway.

Before beginning, it should be mentioned that, to date, the majority of the data accumulated in each of these areas is based on studies using non-human mammalian species (largely the cat and monkey), although certain human neurologic populations (to be described later) allow for some identification of visual pathway function. The quantity of human studies is limited for a number of reasons, including the ethical constraints regarding invasive procedures, as well as biochemical alterations and partial deterioration of tissue samples in the immediate hours after death which yield imprecise measurements in preserved specimens (see, however, Hockfield, Tootell, & Zaremba, 1990). Fortunately, as will be described later, neuroimaging techniques (e.g., positron emission tomography, PET; functional magnetic resonance imaging, fMRI) are allowing for research in live humans to finally become a reality (Tootell, Dale, Sereno, & Malach,

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1996; Tootell, et al., 1995; Zeki, et al., 1991). However, the poor spatial and temporal resolution (at least in PET scans), combined with the difficulties of choosing optimal stimuli to activate only certain cortical areas, limits the precision of the determination of each area's functional specializations, and will inevitably make it a number of years before these methods will be abundantly used. As a result, we must focus on the aforementioned non-human studies.

Historically speaking, until quite recently the cat was more widely studied than the monkey, because it was felt by many that the understanding of the cat's visual system was most complete (Rodieck, 1979). However, with the advent and continuous refinements of existing electrophysiological and histological/autoradiographic tracer methods (see, e.g., De Valois, Smith, Kitai, & Karoly, 1958; R. C. Graham & Karnovsky, 1966; Horton & Hedley-Whyte, 1984; Hubel, 1957; Kennedy, et al., 1975; Kreutzberg, 1984; Nauta & Gygax, 1954; Renshaw, Forbes, & Morrison, 1940; Straus, 1957; Wong-Riley, 1979), the monkey has moved ahead of the cat as the species of choice. In addition, due largely to the seminal microspectrophotometric work by P. K. Brown and Wald (1964) on human retinas (see also Bowmaker & Dartnall, 1980, and Dartnall, Bowmaker, & Mollon, 1983, for recent data obtained with more refined instruments) and that of Marks, Dobelle, and MacNichol (1964) on Old World monkeys (see also Bowmaker, Mollon, & Jacobs, 1983; MacNichol, Levine, Mansfield, Lipetz, & Collins, 1983) which showed striking similarities between the respective species' color vision capabilities, these monkeys (Macaca) are excellent animals for visual system study (Jacobs, 1981) from which we may be able to derive information about our own visual systems. Thus, the studies highlighted in this review will, for the most part, concern the monkey; the cat will be

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considered only in historical context, and of course, when available, human studies will be referred to.

Neuroanatomy/Cortical architecture

The brain has been studied in various ways, shapes, and forms for centuries. The striate cortex, found to be the seat of vision, is no exception. The first descriptions of the striate cortex were made in 1776 by Francesco Gennari, although the coining of the term (based on the region's visible striations), as well as the discovery of its function, did not occur until many years later (reviewed by Glickstein & Rizzolatti, 1984). Seminal work in this area was provided by the team of David Hubel and Torsten Wiesel, beginning in the late 1950's with the cat (see, e.g., Hubel & Wiesel, 1961, 1962, 1965), and continuing in the mid-1960's with the monkey (see, e.g., Hubel & Wiesel, 1968, 1970, 1977; Wiesel & Hubel, 1966). It is of interest to note that in many of their papers, Hubel and Wiesel link together aspects of anatomy and physiology. This is important because anatomical findings did not necessarily predate physiological findings.

Hubel and Wiesel were not the only scientists who were actively pursuing this area of research. However, to spotlight each person's or team's individual contribution is a great task, one which is not warranted for the current review. In the early 1980's, Hubel teamed up with Margaret Livingstone and produced a comprehensive series of papers (Hubel & Livingstone, 1987, 1990; Livingstone & Hubel, 1984, 1987a,b, 1988) which provide a framework within which anatomical, physiological, and psychophysical data concerning the M and P pathways are considered. As a result, these papers will be often referred to.

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According to Van Essen (1979), there exist at least eight visual areas in the owl monkey, at least six in the macaque, with other "higher" mammalian species showing upwards of a dozen. For the present discussion, we will limit ourselves to six areas of the macaque monkey that have been definitively identified and studied to a considerable degree.

Visual information processing has been found to occur as early as the retina. Signals are received from retinal ganglion cells and projected to higher levels in the cortex. Given the proximity of the ganglion cells to the photoreceptors, it might be expected that the signals projected by the ganglion cells are no more complex than those of the photoreceptors (i.e., conveying the "equivalent" of a quantal catch). This, however, is not the case.

The various types of ganglion cells found in the retina have been shown to serve different functions, insofar as pooling the signals of various neurons based on the presence or absence of specific properties of a given stimulus (hence the early use of the term "detectors"). As we shall see, the parallel processing of these signals from lower to higher cortical areas is nearly identical to that which follows in the lateral geniculate nucleus (LGN; see, for example, De Valois, Abramov, & Jacobs, 1966), and beyond.

Original classification of retinal ganglion cells (RGCs) in the monkey had largely followed the existing taxonomy used in the cat by Enroth-Cugell and Robson (i.e., X and Y; actually termed "X-like" and "Y-like"; see Levine & Shefner, 1991, and below). Additional studies showed, however, that RGCs in the monkey retina were considerably less similar to those in the cat than was initially thought, and more than two types of RGCs existed in the monkey, indicating the need for a different classification system.

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Numerous classification systems exist, however, due to some laboratories basing their schema on the morphology of the RGCs, while others base theirs on the physiology of the RGCs.

A recent attempt to alleviate this (see Dowling, 1987) suggests accepting the ganglion cell's receptive field properties as the determinant of the cell's class label. (It should be noted that an even more recent proposal, focusing on classifying ganglion cells based on the transmitters employed at their site of termination, has been suggested [Karten, Keyser, & Brecha, 1990].) For example, a number of RGCs have concentric receptive fields (often termed "antagonistic concentric regions") in which a central circular region is flanked by an annulus. While the central region responds to a stimulus in one direction (either by becoming more excited and firing more frequently, or by becoming inhibited and firing less frequently or not at all) the surround, when stimulated, shows the opposite effect. This leads to the terms ON and OFF, and ON-center and OFF-center. Other RGCs increase their firing rates only upon initiation and cessation of a stimulus, but not during the period of time that the stimulus is present.

Other methods classify ganglion cells on the basis of dendritic arborization. For example, it was found that within the inner plexiform layer, which consists of five layers, certain ganglion cells arborize in only one lamina, while others branch to two or more (Karten, et al., 1990). In addition, physiological analysis reveal that those cells arborizing in the outer portion of the inner plexiform layer are OFF cells, while those arborizing in the inner portion are ON cells. As will be discussed later, the physiological component of RGCs lends considerably more information for classification purposes than do their anatomical features.

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Every photoreceptor sends outputs to both ON and OFF ganglion cells. According to Tessier-Lavigne (1991), the information required to detect objects is contained mainly in variations in the light intensity across the visual scene. The appearance of an object is also influenced by the contrast between the object and it's surroundings. Thus, the firing rate of the ganglion cell provides a measure of the difference in the intensities of light illuminating the center and surround. Both the M and P classes of ganglion cells (in primates, named for cell soma size, arborization, and receptive field size, as above) contain both ON-center and OFF-center cells.

RGCs are often described as being either broad-band or spectrally-opponent (Gouras, 1991; Schiller & Malpeli, 1977). In concentric broad-band cells, both the center and surround combine inputs from both middle wavelength sensitive (MWS) and long wavelength sensitive (LWS) cones and respond only to the difference in brightness of the center compared to the surround when illuminated. (However, Lee, Pokorny, Smith, Martin and Valberg, 1990, report that high-frequency chromatic modulation [> 10 Hz] is determined by the M pathway, due to a nonlinearity of cone summation, thus questioning the usefulness of such terminology.) Spectrally-opponent cells, on the other hand, show a clear preference to certain wavelengths. These classes and their further subdivisions will be covered in more detail in the next section.

While the account given thus far regarding RGCs may seem somewhat simplistic, it should be mentioned that De Monasterio and Gouras (1975) reported 15 different varieties of spectrally-opponent cells, six varieties of broad-band cells, and at least four varieties of non-concentric cells. Schiller and Malpeli (1977) also described a third type of cell (in addition to broad-band and spectrally-opponent) which they called "rarely

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encountered," and De Monasterio (1978) labeled RGCs with Roman numerals (i.e., Type I, II, etc.) based on whether center-surround responses were mediated by similar or different cone types. All these are for concentric receptive field cells. While the aforementioned varieties account for the majority of RGCs sampled to date, additional, as-of-yet unknown, types cannot be ruled out. As mentioned earlier, advanced histochemical techniques continue to reveal a considerable heterogeneity among RGCs, resulting in too simplistic a classification system using morphology alone (and, perhaps remaining limited when used in combination with electrophysiology).

An important finding by De Monasterio and Gouras (1975) was that more cells of the spectrally-opponent variety seemed to terminate in the P layers of the LGN, while more broad-band cells terminated in the M layers, even though both types exist side by side in the retina. Such segregation is a key trend that continues into higher cortical areas.

After the numerous fibers exit the eye via the optic nerve and decussate at the optic chiasm, these fibers eventually end up in one or the other of the lateral geniculate nuclei, a subsection of the thalamus. (It should be noted that a subset of fibers project to various other regions, including the superior colliculus. This pathway will be considered later.) It is here in the LGN that the first tangible signs of segregation are found. The LGN is comprised of six layers, the two most ventral layers being termed the magnocellular layers, the four most dorsal layers being termed the parvocellular layers, each receiving axons from anatomically distinct types of RGCs (De Yoe & Van Essen, 1988; Livingstone & Hubel, 1988).

The LGN, in turn, sends axons, stemming from each layer, to the primary visual cortex. There are various alternative anatomical schemes for subdividing the cortex on a

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functional basis, terms from which are often used interchangeably with one another. The primary visual cortex goes by many names, including "striate cortex," "visual area I," and "V1." Primary visual cortex (V1) corresponds to Brodmann's area 17, and is located in the cortex (cuneate and lingual gyri) surrounding the calcarine sulcus of the occipital lobe (Montemuro & Bruni, 1988). V1 is also divided into six layers, and continues to maintain segregation as in the LGN. The magnocellular layers project to layer 4Cα, and then to layer 4B, while the parvocellular layers project to layer 4Cβ, and then to layers 2 and 3 (see Figure 1). Within layers 2 and 3, the P pathway diverges. The P cells here are further segregated into regions called blobs (so called because of their appearance after cytochrome oxidase staining) and interblobs (so called due to the relative absence of blobs; Livingstone & Hubel, 1988; see Figure 1).

The area which each of the pathways projects to next is the second visual area, V2. This area corresponds to Brodmann's area 18, and is one of two visual association areas. It is located on each side of the calcarine sulcus adjacent to area 17 in the cuneate gyrus above and the lingual gyrus below (Montemuro & Bruni, 1988). When sliced and stained, V2 appears as a series of parallel, repeating stripes. The stripes come in three varieties: pale, thick, and thin. Based on projections from each pathway, segregation is maintained at this level as well. The M pathway projects from layer 4B of V1 exclusively to the thick stripes of V2. On the other hand, the P pathway projects from layers 2 and 3 of V1 to both the pale and thin stripes of V2. It is, in fact, the blobs that project to the thin stripes and the interblob region that projects to the pale stripes (De Yoe & Van Essen, 1988; Hubel and Livingstone, 1987; Livingstone & Hubel, 1987a, 1988; see Figure 1).

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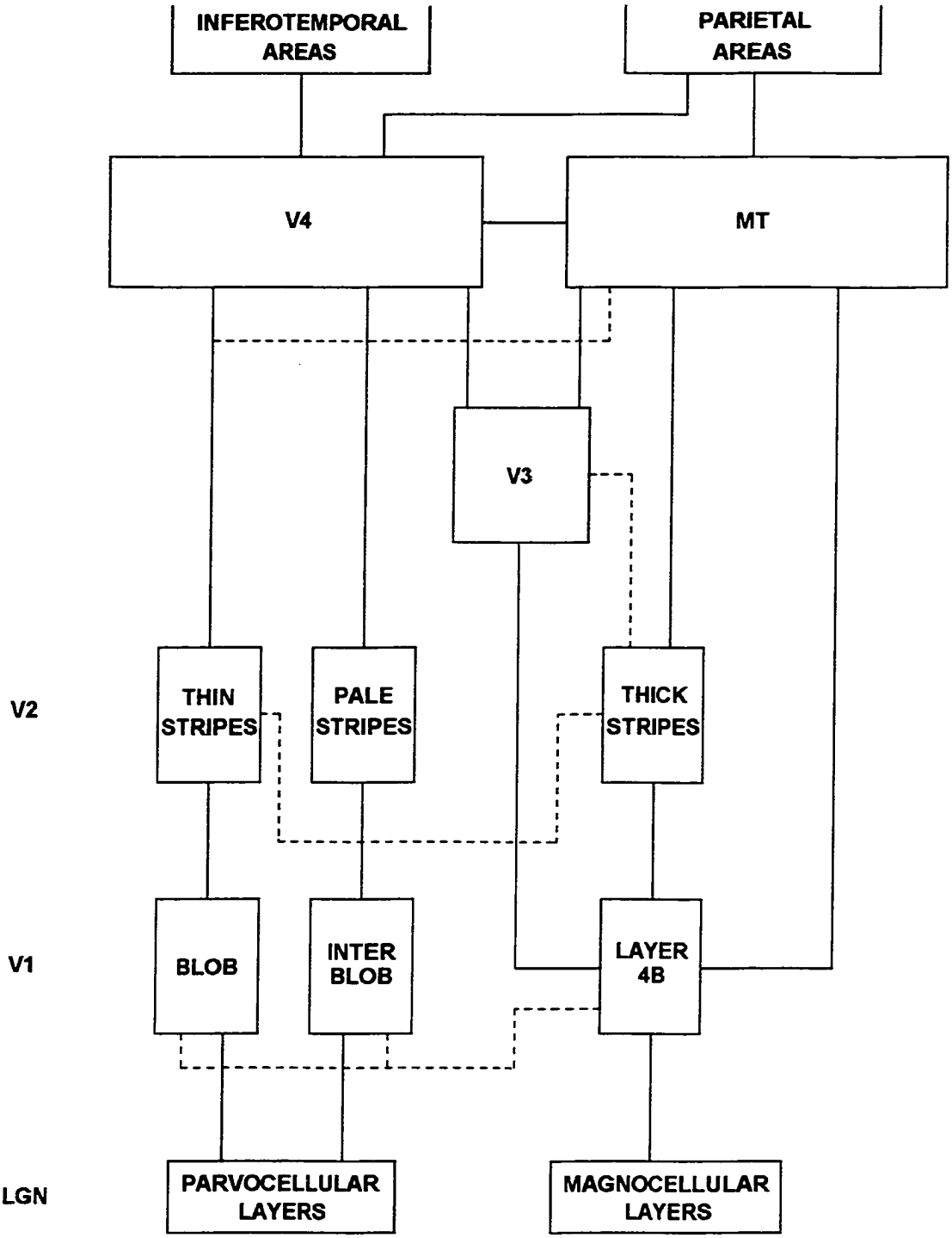


Figure 1. Schematic diagram depicting connections and projections of the geniculocortical visual system of primates. Adapted from De Yoe and Van Essen (1988) and Livingstone and Hubel (1987b). Solid lines represent strong connections; dashed lines represent weak connections.

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Continuing in an orderly fashion, visual area three (V3) is the next higher cortical processing area. It is also located in Brodmann's area 18. This area receives a direct projection from layer 4B of V1 via the M pathway (De Yoe & Van Essen, 1988; Zeki, 1975) and a weak projection from the thick stripes of V2 (De Yoe & Van Essen, 1988; see Figure 1). There is a separate extension of V3, called V3a, found in Brodmann's area 19 (Kandel, 1991) located adjacent to area 18 (Montemuro & Bruni, 1988); however, its antecedent projections have not been fully established.

Area V4 is also part of Brodmann's area 19 (Kandel, 1991). It receives projections from both the pale (De Yoe & Van Essen, 1988; Livingstone & Hubel, 1988) and thin stripes (De Yoe & Van Essen, 1988) of V2 via the P pathway (parvocellular-interblob and parvocellular-blob, respectively), from V3 (De Yoe & Van Essen, 1988; Zeki, 1975), which, as mentioned above, receives its input from the M pathway, as well as directly from V1 (Zeki, 1975; see Figure 1).

Finally there is area V5, more commonly called the middle temporal area (area MT). This area, also part of Brodmann's area 19, lies on the posterior bank of the superior temporal sulcus (Kandel, 1991), at the confluence of temporal, parietal, and occipital cortices situated laterally on the cortical convexity, approximately at the junction of Brodmann's areas 19 and 37 (Zeki, et al., 1991). This area receives direct connections from layer 4B of V1 (M pathway; De Yoe & Van Essen, 1988; Livingstone & Hubel, 1988), from the thick stripes of V2 (M pathway; De Yoe & Van Essen, 1988; Livingstone & Hubel, 1988), from V3 (De Yoe & Van Essen, 1988), and weakly from V4 (De Yoe & Van Essen, 1988; see Figure 1).

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Depending upon the sources one consults, a number of other visual areas may be said to exist, including V5a, MST (for medial superior temporal area), and 7a (Kandel, 1991). The reader is cautioned, however, to be certain of the type of taxonomy being used, as some areas could be duplications of ones previously mentioned. Nonetheless, as described earlier, while there are bound to be additional areas in both the macaque and human, those outlined above, to date, are the most thoroughly studied, and are clearly involved in the flow and processing of visual information.

In summary, the M pathway originates in the ventral layers of the LGN, projects to layer 4Cα of area V1, then to layer 4B, followed by independent, simultaneous projections to the thick stripes of area V2, area V3, and area MT. V2 and V3 each project to MT, and V3 also projects to V4. From area MT, projections are made to the upper superior temporal and intraparietal sulci (Mishkin, Ungerleider, & Macko, 1983; see Figure 1). The P pathway originates in the dorsal layers of the LGN, projects first to layer 4Cβ of V1, followed by layers 2 and 3, at which point they segregate into the blob and interblob regions, and then each region independently projects to V2, the blobs to the thin stripes and the interblobs to the pale stripes. Both the thin and pale stripes project to V4, which in turn projects weakly to MT, and strongly to the inferior temporal cortex (Mishkin, Ungerleider, & Macko, 1983; see Figure 1). From this, it appears that the M and P pathways remain, for the most part, largely separate.

Neurophysiology/Functional architecture

Neurophysiological studies have centered on the stimulation of isolated neurons and recording their outputs using microelectrodes. The stimuli that are used are selected

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based on particular characteristics which will evoke responses from cells found to be tuned to only certain visual capacities. This enables one to selectively measure a limited number of visual capacities at a given time. All neurons have a baseline spontaneous firing rate in the absence of any stimulation. This spontaneous firing rate varies considerably from higher rates in subcortical areas (including the ganglion cells and LGN) to lower rates in cortical areas. A stimulus eliciting an increase in firing rate in a given cell would allow that cell to be described as being more or less selective for that particular characteristic (e.g., selective for orientation, direction, etc.). Cells whose firing rate decreases upon stimulation also can be termed selective for that particular stimulus characteristic. A cell whose response does not change from baseline when presented with a stimulus is not selective for that stimulus property. (In order to ascertain if a cell is truly non-selective, several different stimuli often need to be tried, as a property of a given stimulus, such as wavelength of light, may conflict with that of the cell, leading to a signal cancellation on this basis alone.) A large number of cells from each visual area previously discussed have been recorded from, and a list of both general and specific properties common to these groups of cells has been compiled. We will now look at each of these areas in more detail, taking each one in turn, as was done in the previous section.

As described in the previous section, the RGCs project their fibers to the LGN. Early studies done on the cat by Enroth-Cugell and Robson (1966) revealed two types, X and Y, based on their responses to sinusoidal gratings. The X cell was found to average the amount of light falling on it's receptive field, responding only when the grating produced a change in the total effective amount of light falling on the receptive field (linear spatial summation), while the Y cell also takes into consideration the spatial

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configuration of the light, acknowledging that it is not merely diffuse illumination (nonlinear spatial summation). A similar division was found to exist in monkey ganglion cells, but the differentiation was not as simple. For example, Kaplan and Shapley (1982) found that while 99% of cells sampled in the parvocellular layers of the LGN were X-like, magnocellular layer cells were found in two varieties: 75% were X-like and 25% were Y-like (see also Lennie, 1980). But response to sinusoidal gratings is not the only difference between M and P cells (or X and Y cells, for that matter), and to base ganglion cell classification solely on this property would be inappropriate.

It was mentioned in the last section that RGCs can be grouped in many ways. Ignoring the physiological responses of RGCs to stimuli with other defining characteristics can lead to misclassification. According to Dowling (1987), RGC responses strongly reflect the properties of the input neurons (viz., bipolar, amacrine, and horizontal cells). Bipolar cells, for instance, give more sustained responses, whereas amacrine cells are more transient. Thus, the contrast-sensitive ganglion cells found to be largely fed by bipolar cells show evidence of spatial processing of visual information; those found to be fed by amacrine cells show more evidence of temporal processing of visual information, in that the RGCs are highly movement-sensitive and many show direction-sensitive responses. Hence, there is a further subdivision from that presented earlier, which includes direction-sensitive ON-OFF ganglion cells.

The M cells, as a whole, are found to be transient, to respond to large objects, and to analyze gross features and movement. The P cells are more sustained, wavelength-selective, and are implicated for color vision and the analysis of fine detail (Tessier-Lavigne, 1991).

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In the section on neuroanatomy and cortical architecture, the terms "broad-band" and "spectrally-opponent" were introduced. Electrophysiological studies have revealed a further subdivision of the spectrally-opponent cells. This class is divided into concentric single- (consisting of an antagonistic center-surround receptive field with one type of cone's inputs occupying the center and another's the surround) and double-opponent cells (consisting of an antagonistic center-surround receptive field with two or more cone's inputs occupying each part of the receptive field, with each cone type having actions opposite to each other and to adjacent parts of the receptive field). The former transmit information about both color (when stimulated with monochromatic lights of appropriate wavelengths) and achromatic brightness contrast due to MWS and LWS cones absorbing white or yellow light to similar degrees (Gouras, 1991). The latter have been found only in monkey cortex, and will not be considered further. Examples of opponent cells include those excited by long wavelengths (termed "red-ON, green-OFF" or "+R-G" and "yellow-ON, blue-OFF" or "+Y-B" by De Valois, et al., 1958, 1966, to signify the specific wavelength's excitation and complementary wavelength's inhibition), as well as those excited by short wavelengths ("+G-R" and "+B-Y"). A subset of single-opponent cells called "co-extensive" have a uniform receptive field in which short wavelength sensitive (SWS) cones antagonize the combined inputs of MWS and LWS cones (Gouras, 1991).

Schiller and Malpeli (1977) have found that spectrally-opponent cells have a more sustained response than do broad-band cells, respond less vigorously to low frequency sinusoidal gratings moved across the receptive field, and show a higher spontaneous firing rate. M and P ganglion cells project directly to the M and P layers of the LGN, respectively, carrying such characteristic properties to higher pre-cortical (and cortical)

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levels. Broad-band cells, as described earlier, signal information pertaining to brightness differences (i.e., achromatic contrast) across the receptive field. These can be either of the M- or P-type, whereas single-opponent cells (signaling chromatic information) are exclusively of the P-type. The M pathway is, thus, dealing exclusively with achromatic information, while the P pathway is processing both achromatic and chromatic information (Gouras, 1991).

Livingstone and Hubel (1987b, 1988) summarize the differences between M and P cells at the level of the LGN as follows. P cells are spatially linear, have receptive fields which are small in size, produce sustained responses, have slow signal conduction, have poor contrast sensitivity, have excellent spectral sensitivity, and have high spatial resolution. M cells, on the other hand, are either spatially linear or nonlinear, have larger receptive fields (2 to 3 times that of P cells, on average), produce transient responses, have fast signal conduction, have good contrast sensitivity (although cells saturate at levels above 10-15%), have poor spectral sensitivity, and have lower spatial resolution.

As we continue up to higher cortical levels, we will see that there is a logical progression from the properties found at the earliest levels and the visual functions that each pathway subserves.

According to Mason and Kandel (1991), the primary visual cortex has at least three major functions. First, it deconstructs the visual world into short line segments of various orientations, perhaps an early stage in the process thought to be necessary for discrimination of form and movement (however, see De Valois, Albrecht, & Thorell, 1978, and Albrecht, De Valois, & Thorell, 1980, for data showing that cortical cells are better suited to be referred to as spatial frequency filters, rather than bar or edge

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detectors); second, it segregates information about color from that concerned with form and movement; and third, it combines the input from the two eyes, a step in a sequence necessary for depth perception.

The neurophysiological proof can be found in describing the properties of the cells in each pathway at this level. The cells in layer 4Cα (directly projecting from the M layers of the LGN) are selective for orientation, are spectrally broad-band (i.e., while there is center-surround opponency found in these cells, there is no antagonism between the different cone types within the cell, and responses are made to changes in luminance), have a relatively high contrast sensitivity, have coarse spatial resolution, and have fast, transient responses (Livingstone & Hubel, 1987b). These characteristics carry over to layer 4B, where elements of orientation selectivity, direction selectivity, binocularity, broad-band receptive fields, high contrast sensitivity, coarse spatial resolution, and selectivity for moving stimuli are reported (De Yoe & Van Essen, 1988; Livingstone & Hubel, 1987b, 1988). The cells in layer 4Cβ (P projections) have very small, concentric receptive fields with center-surround antagonism (see above), some spectral coding (i.e., the cells have spectrally-opponent centers and antagonistic spectrally-opponent surrounds), lower contrast sensitivity, finer spatial resolution, and slower temporal properties (Livingstone & Hubel, 1987b). At layers 2 and 3, where the P system branches into the blob and interblob regions, the blobs are predominantly involved in spectral analysis, as evidenced by roughly half of these cells in macaque being selective for certain wavelengths (De Yoe & Van Essen, 1988; Dow & Vautin, 1987; Livingstone & Hubel, 1987a,b, 1988; Ts'o & Gilbert, 1988), although blob cells with M properties have been reported (Edwards, Purpura, & Kaplan, 1995; Livingstone & Hubel, 1987b, 1988). Blobs

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additionally have coarse spatial resolution (although not as coarse as M cells), are relatively selective for stationary stimuli, and show no selectivity for direction or orientation (Livingstone & Hubel, 1987b, 1988; but see Ts'o & Gilbert, 1988).

The interblob region appears to be well tuned for stimulus orientation, selective for moving stimuli (though not for direction), show a questionable degree of spectral sensitivity (not color-opponent like blobs), show greater contrast sensitivity than the blob region (though less than M cells), and have fine spatial resolution (De Yoe & Van Essen, 1988; Livingstone & Hubel, 1987a,b, 1988). Evidence of binocularity has also been reported (De Yoe & Van Essen, 1988).

In general, it must be pointed out that spectral sensitivity, for any cell at any level of the visual pathway, is not simply present or not present. The properties of brightness and spectral sensitivity are not completely independent when evoking responses from neurons. De Valois, Snodderly, Yund, and Hepler (1977), in studying responses of LGN cells to luminance and wavelength, found not only that changes in luminance also affect the responses of most spectrally-opponent cells, but also that wavelength information is processed spatially in a different way from luminance information by the same cells. Furthermore, a cell has, in effect, two types of receptive fields: one that responds to changes in luminance, and a second which responds to changes in wavelength. This inevitably has some bearing on the determination of properties of cells in higher cortical areas, and poses the question of whether the methods being used to record the cell's responses are the most adequate for producing unbiased measurements.

The cells of area V2 are found to remain segregated, as far as specific properties, based on the particular set of stripes being recorded from. For example, cells in the thick

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stripes, being projected to from layer 4B (M cells), are found to be particularly selective for stereoscopic depth (with approximately two-thirds precisely tuned for binocular disparity), to respond only to moving stimuli, and not to be direction-selective (Hubel & Livingstone, 1987; Livingstone & Hubel, 1987a,b, 1988; Zeki, 1975, 1978). De Yoe and Van Essen (1985, 1988) and Zeki (1978) report a large degree of orientation selectivity. Interestingly, the former papers also claim a greater than 20% incidence of directional selectivity, as well.

The thin stripes of V2 follow, in suit, like their predecessors (blob area of V1), in that they, for the most part, are lacking in orientation selectivity, but reportedly show a high degree of spectral opponency (De Yoe & Van Essen, 1985, 1988; Livingstone & Hubel, 1987b). This is in contrast to Zeki (1978) who reports that only 8% of cells in all of V2 are spectrally-opponent in nature.

The pale stripes (projected to from the interblob region) are found to be similar to the interblobs (i.e., they are binocularly- and orientationally-selective, and have some spectral sensitivity; De Yoe & Van Essen, 1988). A number of the cells contained within these stripes are end-stopped. They respond to short, but not long, bars (Hubel & Livingstone, 1987; Livingstone & Hubel, 1987b).

Area V3, again, is projected to solely by the M pathway, for the most part from layer 4B of V1. Thus, it is not surprising that the properties of the cells found in area V3 closely resemble those found in layer 4B. Felleman and Van Essen (1987) found 76% of the neurons to be strongly orientation-selective (of which 40% were strongly direction-selective), 45% to be selective for binocular disparity, and approximately 20% were wavelength-selective. In their studies, wavelength selectivity was assessed by examining

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the relative responses of an individual neuron to a range of monochromatic and white stimuli, all matched for their photopic luminance. This included comparing the magnitude of response to the least effective wavelength with the most effective wavelength among all monochromatic stimuli tested, and the neuron's response to white light. A color index was mathematically defined, and using a criterion cutoff of 0.7 for the classification of a cell as being wavelength-selective, 20% of the cells tested met such criteria. The finding of wavelength selectivity in V3 questions a purely M pathway input. The authors suggest that there may be specific V2 inputs (especially from interstripe regions) or there may exist a subpopulation of wavelength-selective cells in layer 4B of V1. Aside from the spectral component, these results agree with other studies (De Yoe & Van Essen, 1988; Zeki, 1978), making V3 well suited for the analysis of several aspects of stimulus motion, and some aspects of form analysis, particularly at low contrast levels (Felleman & Van Essen, 1987).

Area V4 has come to be known as an area which may be highly specialized in the analysis of spectral information for the visual system, since it is here that the blob and interblob regions converge. This area has been studied widely in the monkey by Zeki (1973, 1975, 1978; Zeki et al., 1991), who found close to 56% of all cells to be spectrally-coded to some extent (a striking drop from his report of 80% three years earlier, though [Zeki, 1975]), less than half to be selective for orientation, and very few (< 15%) to be directionally-selective (Zeki, 1978).

It should be noted that one possible reason as to why Zeki (and others) have had such variable results as far as the percentage of spectrally-coded cells found in V4 was provided by Schein and Desimone (1990). These authors point to the defining criteria of

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what constitutes a cell as being "spectrally-selective" as somewhat problematic. In a sample of 129 cells, few cells showed outright spectral opponency to spectrally-opponent wavelengths, some 30% of the cells were found to have spectral-response curves with two peaks, most responded fairly well to white light (giving a response on the order of 60% of the response made to the best chromatic light), and most gave at least a small response to nearly all the different broad-band chromatic lights presented. The interpretation of such findings can range from these cells not being spectrally-selective due to their responding to all stimuli presented, to the cells being spectrally-selective based on a greater response to a stimulus of optimal wavelength compared to a white stimulus of equal luminance. Similar discrepancies over the incidence and nature of wavelength selectivity has also been found in other visual areas (including V1 and V2), and the need to describe the full range of spectral properties of cells quantitatively seems more appropriate than existing qualitative classification systems (Schein & Desimone, 1990).

On a more positive note, through neuroimaging (Lueck, et al., 1989; Zeki, et al., 1991), an area homologous to V4 has been mapped in the human (see below). The addition of binocularity, and the above results agree well with other findings (De Yoe & Van Essen, 1988).

Last, we have area MT, commonly called the motion area, due largely to a high prevalence of directionally-selective cells. In addition, a large degree of binocularity and orientation-selectivity is found, as would be expected based on the characteristic properties of the cells in the areas from which MT's projections stem. Like other M pathway areas, MT's contrast sensitivity is high, but spatial resolution is quite low,

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making this area very able to detect movement, but not to clearly discern the identity of the object in motion (De Yoe & Van Essen, 1985, 1988; Livingstone & Hubel, 1987b, 1988; Zeki, 1978; Zeki, et al., 1991).

To summarize this section, let us take the major findings and describe them by pathway, according to Livingstone and Hubel (1987b). The M pathway is clearly selective for movement and binocular disparity, suggesting that it plays a major role in depth and motion perception. Some slight form perception may come in, as well, since the cells are also largely orientation-selective. The system, overall, has high temporal resolution, high contrast sensitivity, relatively low spatial resolution, and is probably colorblind.

The P-interblob system has high spatial resolution, orientation-selectivity, and end-stopping, making its role of high-resolution form perception quite probable. The P-blob pathway is basically concerned with color. Low resolution and lack of orientation-selectivity suggests that it is not important in form perception (Livingstone & Hubel, 1987b).

As discussed above, work by other laboratories have shown that Livingstone and Hubel's conclusions are by no means absolute and must be taken with caution. While the M and P pathways appear to be remaining largely functionally segregated, contrary evidence is beginning to uproot the foundation built by Livingstone and Hubel (for a review, see Merigan & Maunsell, 1993). Psychophysical experimentation, however, may allow us to verify, in a tangible way, whether the aforementioned characteristics of each pathway are accurate, while at the same time fill in any missing pieces to the puzzle that neurophysiological recordings were not able to take care of. Therefore, it is this topic to which we turn next.

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Lesion studies/Psychophysics

Introduction. The term "psychophysics," when loosely applied, can have various meanings. For our purposes, a working definition of psychophysics is the "study of the capacity of an organism to detect, quantify, or identify a stimulus" (Levine & Shefner, 1991, p. 671). Here, we are looking at an organism's visual capacities. As far as the detection, quantification, or identification of the stimulus, there are numerous ways in which this can be measured. Verbal responses are limited to human observers. Animals can, however, be taught to respond to stimuli by pressing buttons or levers, turning knobs and the like, through being rewarded for a correct response. Yet, in these instances, we still really have no way of knowing what the animal truly "sees" the stimulus as, and whether the set of responses is just all part of a complex conditioning process that the animal has associated with a means of reward, and is consequently using for this purpose. One of the ways that researchers have tried to alleviate this problem, is to combine certain methods, at least one of which, in the end, requires the scientist's own interpretation of the animal's response record. This way the scientist can be certain that the responses are based on the stimuli presented without confounds. One such widely used paradigm that is employed is lesion studies, which combines elements of both neurophysiology and psychophysics.

Lesion studies. As mentioned earlier, in order to determine the properties of the individual pathways, it is essential that the stimulus used be one whose characteristics can specifically isolate one pathway or the other, such that any response elicited will then necessarily be from the pathway in question. The task of choosing such a stimulus is not easy. What lesion studies have done, rather than find the perfect stimulus, is to present a

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stimulus, measure the response, and then to selectively lesion an area that has previously been shown to belong to one of the pathways, and then measure the response again. After careful lesioning, a decrease in (or abolishment of) the response rate suggests that that area (and pathway) may play an important role in the processing of visual information concerned with the property the stimulus represents. No change in the response rate indicates that the lesioned area (and pathway at that cortical level) does not fundamentally aid the organism in that visual capacity.

Peter Schiller and colleagues (Schiller & Logothetis, 1990; Schiller, Logothetis, & Charles, 1990, 1991) extensively studied the effects of M and P pathway lesions on various visual capacities. Lesions to the M pathway at the level of the LGN were found to have no effect on the processing of spectral information or form vision (including pattern, texture, and shape). Responses to intensity were not impaired, and only minor deficits (if any) were found in contrast sensitivity, despite the high sensitivity found in spectrally broad-band cells. No deficits were found to occur with stereopsis. Lesions to the M pathway did, however, produce marked deficits with regards to both flicker and motion sensitivity, especially at low contrasts, with monochromatic flicker sensitivity being reduced from an average of 22 Hz to 6 Hz, and heterochromatic flicker (under near isoluminance conditions) was reduced from 11 Hz to less than 5 Hz. Motion was affected in both the areas of detection and discrimination. Similarly, Merigan, Katz, and Maunsell (1991) found no reduction in visual acuity nor any reduction in luminance and chromatic contrast sensitivity to stationary gratings, but did find a reduction in luminance contrast sensitivity to modulated gratings. This finding reemphasizes that the M pathway is important for detection at lower spatial and higher temporal frequencies.

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Lesions to the P pathway severely impaired wavelength discrimination, severely compromised form vision (though less so at low spatial frequencies), caused pronounced deficits in contrast sensitivity at high spatial frequencies, and yielded major deficits in fine static stereopsis (Schiller & Logothetis, 1990; Schiller, et al., 1990, 1991). Merigan, et al. (1991) recorded a 3- to 4-fold reduction in visual acuity (noting that the larger the lesion, the larger the reduction, beginning beyond 3° eccentricity) and a 4-fold loss in chromatic and luminance contrast sensitivity to both static and modulated gratings. The authors conclude that the P pathway dominates chromatic vision, acuity, and contrast detection at low temporal and high spatial frequencies.

Schiller's group found that properties spared by P pathway lesions include the ability to discriminate stimuli on the basis of wavelength differences at isoluminance, brightness, contrast sensitivity at low spatial frequencies, and coarse stereopsis. Shape perception was only impaired in discriminating squares from circles (Schiller & Logothetis, 1990; Schiller, et al., 1990).

They conclude that the spectrally-opponent (P) system is crucial for wavelength discrimination and is essential for form and depth perception at high, but not low, spatial frequencies. The broad-band (M) system is important for motion and flicker perception, but apparently the spectrally-opponent system can process these capacities at high contrasts and low temporal rates. Both systems are able to process brightness, shape, and stereoscopic information at low spatial frequencies (Schiller & Logothetis, 1990; Schiller, et al., 1990, 1991).

Lesion studies were also carried out in areas V4 and MT. In the case of area V4, lesions resulted in mild deficits in texture perception, fine shape perception, and fine

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contrast sensitivity, while color vision and pattern perception were only moderately impaired. For area MT, there were mild deficits found for contrast sensitivity, moderate impairments in motion perception, and pronounced deficits in the instance of flicker perception. The authors state, contrary to the findings of others (e.g., Zeki, 1973, 1975, 1978; Zeki et al., 1991), that these areas are not unique in processing color, form, motion, or stereopsis, nor are they the sole gateways to the temporal and the parietal lobes for the spectrally-opponent and broad-band systems (Schiller & Logothetis, 1990).

The question of accuracy of lesion studies cannot be overlooked. Merigan and Maunsell (1993) note that lesioning intended for a particular area may not necessarily be successful. Lesions may be imperfect, and damage made to other areas in the process leads to difficulty in the interpretation of the effects of the lesions. In addition, a number of lesion studies attempt to measure the visual capacities of a particular pathway at one point along that pathway and compare it to a lesion at a different point along the other pathway. In doing so, researchers often use different visual capacities and/or behavioral measures (appropriate for the particular pathway), which makes any attempt at comparisons futile.

From the above data and our knowledge of the anatomy and physiology of the M and P pathways, one may start to question the accuracy of the statement that the two pathways are, indeed, segregated (for a review, see Merigan & Maunsell, 1993). However, it must be pointed out that the finding of similar functions between the pathways is not necessarily indicative of cross connections. It is quite possible that this information is carried in each pathway because it undergoes different processing in each

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case, or, if analyzed in similar respects, it is done so for cross-checking purposes when the separate pathways pool their information together at higher cortical levels.

Still, others may feel undeterred with the findings, simply because the methods used were imprecise insofar as not clearly delimiting the individual areas and pathways lesioned (a disadvantage of the technique; see above). Perhaps clarification on some of these issues can be achieved through psychophysical experimentation on human participants.

Psychophysics. Livingstone and Hubel (1987b) published a lengthy paper on results of psychophysical experiments they carried out employing stimuli believed to sufficiently evoke responses of cells tuned to specific visual capacities. In this paper, Livingstone and Hubel not only reported new data, but also importantly compiled various sets of old data, and retested certain elements in light of their recent neurophysiological findings (e.g., see Hubel & Livingstone, 1987, 1990; Livingstone & Hubel, 1984, 1987a).

The lack of chromatic processing by the M pathway played a very fundamental role in much of the psychophysical measurements made. The stimuli chosen were presented at isoluminance (as determined via color flicker photometry), and therefore any ability that breaks down under such conditions is probably dependent on a broad-band system (i.e., M). Flicker-photometry equiluminance was determined by changing the intensity of a red-green rectangle modulated at 15 cps until no visible flicker could be seen. The ratio was found to vary both between observers, as well as a function of eccentricity (with less green being needed; Livingstone & Hubel, 1987b).

While all the experiments that Livingstone and Hubel ran are too numerous to mention, a few select ones will be highlighted and the results summarized. In the realm of

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movement, a function repeatedly shown to be predominantly a property of the M system, it was shown that at isoluminance, many motion phenomena disappear (including illusions and illusory contours), while some components of form and movement are analyzed independently. For example, horizontal red-green sine-wave gratings, with red and green components 180° out of phase, moving downwards at a rate of 1 cps appeared to markedly slow (and even stop) over a range of 5% above and below isoluminance when relative brightness was varied. Illusions, such as the Necker cube and the MacKay illusion (in which an aftereffect of movement in the direction opposite to that of the original stimulus is perceived), were found to be virtually non-existent at equiluminance. With the lower spatial resolution of the M pathway, tests used to determine the independence of form and movement have focused on using stimuli of various degrees of fineness. Higher spatial-frequency dot patterns, when slowly rotated, were perceived to rotate considerably slower than lower spatial-frequency patterns (at times even appearing to be completely stationary) just like at equiluminance. The authors interpreted these results as motion being purely M pathway controlled, and that any convergence of the pathways for color and movement must occur at advanced stages of visual processing (Livingstone & Hubel, 1987b).

Stereopsis was found to resemble movement perception in being colorblind, being operative at low luminance contrasts, and in having lower spatial resolution than form perception. Random-dot stereograms (see below) composed of two equiluminant colors (instead of the traditional black and white) caused depth perception to be quite minimal or completely absent. However, random-dot stereograms consisting of light and dark dots with a difference in luminance contrast of only 5% were still able to give a clear

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indication of depth. High resolution form perception was lost at this level insofar as the observer was unable to clearly delineate individual dots. Hence, stereopsis, too, depends on the M pathway, and not on the P (Livingstone & Hubel, 1987b).

Form perception, on the other hand, was found to consist of contributions from both the M and P systems. This was determined by the range of luminance contrasts over which form perception is possible, as well as the lack of saturation at higher contrasts compared to movement and stereopsis. Specifically, those aspects pertaining to the M system include integration of whole images, while those concerning the P system focus on finer elements of the objects, including shape and surface properties. For example, shape discrimination is possible at both low luminance contrast and at equiluminance, pointing to both systems having some ability to discriminate form. High-resolution form discrimination appears to be solely a function of the P system. This puts the M system in a position to organize a given scene into three dimensions, taking into consideration the perception of motion and depth, and determining where the objects are (Livingstone & Hubel, 1987b).

The functions of the M and P pathways can best be summarized as follows. The M pathway is responsible for motion detection, including sensitivity to orientation and direction, spatial relationships among objects, detection of low contrast stimuli of various sizes, integration of whole images, depth cues (including stereopsis, binocular disparity, depth from motion, parallax, and contours), and figure-ground segregation. It seems to be able to integrate information from various parts of the visual field, correlating and linking spatially separate stimuli. The P pathway aids in the detection of form, shape discrimination, including sensitivity to orientation of edges, detection of chromatic

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borders of objects, detection of differences in wavelength, and the surface properties of objects. The interblob system seems more spatially restrictive. The pathway as a whole confers the ability to see well, or in finer detail, the static components of the environment (Livingstone & Hubel, 1987b, 1988).

The combination of the fast responses of the M pathway along with the P pathway's ability to analyze fine detail has led Ungerleider and Mishkin (1982; Haxby, et al., 1991; Mishkin, Ungerleider, & Macko, 1983) to coin the terms "where" and "what" for the M and P pathways, respectively. Goodale and Milner (1992) and Newcombe, Ratcliff, and Damasio (1987) found that lesions made to the posterior parietal cortex (projected to by area MT of the M pathway; see Figure 1) led to impaired spatial location of objects; lesions made to the inferotemporal cortex (projected to by area V4 of the P pathway; see Figure 1) led to impairment of visual pattern discrimination and recognition, reinforcing the concept of separate pathways for these purposes.

## Problems

A closer look at the evidence. Are the M and P pathways segregated? This question is posed because of a number of recent developments and other considerations that must be taken into account (for a review, see Merigan & Maunsell, 1993). First, the psychophysical experiments of Livingstone and Hubel (1987b) were carried out, for the most part, on themselves. In scientific research, we have come to appreciate the fact that small sample sizes are not very convincing. Levine and Shefner (1991) point out that we cannot be sure to what extent the effects seen in Livingstone and Hubel's results are due to what was actually seen, and to what extent they were what the authors expected to see.

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Second, it should also be mentioned that the lesion studies of Schiller and Logothetis (1990) and Schiller, et al. (1990) were done, in part, because of inherent problems imposed by isoluminance experiments. For instance, certain visual functions believed to be processed by the spectrally-opponent system, such as texture perception, are also compromised at isoluminance. Similarly, motion- and depth-perception, two functions ascribed to the broad-band pathway, were found to be compromised, but not abolished at isoluminance (see Logothetis, Schiller, Charles, & Hurlbert, 1990; Schiller, et al., 1991). In addition, there is incomplete elimination of broad-band system input under isoluminance at various levels of the visual cortex, including the retina, LGN, and area MT (Schiller & Logothetis, 1990; Schiller, et al., 1991).

At levels of isoluminance, there is an alteration of responses of the spectrally-opponent system to stimuli that the system would ordinarily make a response to based jointly on luminance and wavelength. This is due, in part, to up to 20% of P geniculate cells that lack sharply defined spectral-opponency becoming unresponsive at isoluminance (Schiller & Logothetis, 1990).

Certain laboratories (e.g., Cavanagh & Favreau, 1985) have shown that, in the absence of luminance, color can be used for the perception of form, motion, and depth. Also, certain conditions created at isoluminance, such as the marked slowing down of drifting red-green sine-wave gratings (see above), can be replicated under conditions of low achromatic contrast (Schiller & Logothetis, 1990). According to Kovacs and Julesz (1992), isoluminance is "a most unnatural situation" (p. 10390), and that at isoluminance "anything can happen" (p. 10390). Simply put, isoluminant stimuli are inappropriate for

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the psychophysical isolation of the M and P pathways (Logothetis, et al., 1990; Schiller, et al., 1991).

Furthermore, it was shown earlier, in comparing the papers by Livingstone and Hubel (1987b, 1988) to that of De Yoe and Van Essen (1988), that there is even disagreement among the various anatomical connections from lower to higher cortical levels between the two pathways. This, in effect, questions the validity of the very assumptions that the isoluminance studies mentioned above are based on.

Cross-talk between the systems is far from a new concept. Malpeli, Schiller, and Colby (1981), for example, a number of years before the work of Livingstone and Hubel, suggested a lack of complete segregation between the M and P pathways after finding that neither orientation nor direction depended exclusively on M or P inputs from the LGN. This has been followed by more recent reports including (a) the color-form pathway depends on both M and P input (Ferrera, Nealey, & Maunsell, 1992); (b) area V4 receives strong input from both subdivisions of the LGN (Ferrera, Nealey, & Maunsell, 1994; Nealey & Maunsell, 1994); (c) in a task involving face matching, dot location matching, and sensorimotor control tasks, the "where" and "what" pathways, previously found in monkeys, were also found in humans, but with an additional lateral occipital extrastriate region involved in both object and spatial vision (Haxby, et al., 1991); (d) there exist backward connections from such areas as MT to V1. These connections are found to be distributed more evenly across the cortex, and over a wider area of cortex, relating to all three major processing streams, and, thus, having the potential of mediating interactions between processing streams that may be critical in creating a unified perception of the world (Krubitzer & Kaas, 1989); (e) color and luminance share a common motion

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pathway (Cavanagh & Favreau, 1985; Gegenfurtner & Hawken, 1996; Hawken, Gegenfurtner, & Tang, 1994); and (f) most layer 4B neurons receive significant input from P-stream-recipient neurons in layer 4Cβ, indicating that parietal areas are probably influenced more by the P stream than was previously believed (and that information from both streams contributes importantly to higher-order visual processing in both parietal and temporal areas; Sawatari & Callaway, 1996).

Casagrande (1994; see also Hendry & Yoshioka, 1994) brought up the intriguing idea that, at least in primates, a third pathway, the koniocellular (K) pathway, may play a very important role in vision. This pathway, known for years to exist, contains small cells which lie within the interlaminar zones and within specialized layers, the superficial layers of simian primates (termed the "S-lamina," believed to be a displaced portion of layer 2 of the LGN, comprising three pairs of layers in the macaque) and the koniocellular layers of prosimians. K-cells closely resemble W-cells found in cats, both anatomically and physiologically, featuring large receptive fields, long response latencies, and heterogeneous functional properties. The K pathway has been found to have direct connections to the cytochrome oxidase blobs (relaying inputs from the SWS cones), pointing to its possible role in object recognition (as judged by where the blobs subsequently project to) and/or to visual resolution. Its role in color, however, is questionable due to the presence of cytochrome oxidase blobs in nocturnal primates and the cell's physiological characteristics mentioned above. Because collicular inputs are important to K cell function, perception during eye movements may also be a key function of this pathway, as the superior colliculus is concerned with motion in the visual field, visual attentiveness, and the identification of broad outlines of objects (Mason &

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Kandel, 1991). Despite no mention of humans having a similar third pathway, the finding of Haxby, et al. (1991; see above) does not eliminate this possibility. With this in mind, previous studies attempting to eliminate either the M or P pathway in an attempt to gain insight into the contributions of those pathways to vision need to be reviewed due to the possibility of K pathway input, especially with neurons from the K pathway extending to the extrastriate cortex, making certain visual behaviors likely to be sustained even in the absence of V1 (for a review, see Hendry & Reid, 2000).

Schiller and Logothetis (1990) note in their lesion studies that, except for wavelength discrimination, deficits for other visual capacities are graded, with no one system's responses becoming completely abolished. This was paralleled by similar findings by Logothetis, et al. (1990) in psychophysical experiments conducted under isoluminant conditions. Based on these findings, it was concluded that impairment of visual capacities at isoluminance cannot be uniquely attributed to either of the two systems, and that isoluminant stimuli may be inappropriate for the psychophysical isolation of these pathways (see above).

Zeki (1975) reported that in the monkey visual cortex, there exist both convergent inputs from one or from several antecedent areas, and divergent outputs to more central cortical areas, for each area (e.g., V4 receives inputs from V2 and V3, as well as from V1).

It was mentioned at the start of this essay that there are many sets of parallel pathways. The possibility exists that cross-talk between the M and P pathways may occur, in part, through an intermediary set of pathways. Schiller (1982) studied the connections between the ON and OFF pathways (so named by the resulting response, excitatory or

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inhibitory, upon stimulus onset and offset) in both the retina and the striate cortex of monkeys. As mentioned earlier, there are both M and P ON- and OFF-cells. In the LGN, Schiller found that ON responses were blocked by the drug APB (DL-2-amino-4-phosphonobutyric acid), while OFF-cells were spared. While it is noted that these results indicate that the surround inhibition observed in LGN cells does not appear to be a product of the interaction between the ON and OFF channels, it may be speculated that some interaction between the M and P cells may occur as early as this level. Since both M and P ON-cells are blocked, there may be fundamental properties that are identical among these cells. Cells similarly tuned for specific functions will most likely, at some point in the visual system, send and receive inputs from the same cortical areas that process the information related to that particular function. Considering the complexity of the visual system, coupled with the vast amount of information being processed at any given time, many such check points may exist, even at subsidiary locations, so as to avoid redundancy at higher cortical levels.

At the level of striate cortex, Schiller (1982) found evidence of convergence between the ON and OFF channels. The blocking of ON responses eliminated the light-edge response of cortical cells, and, in certain instances led to inhibitory interactions between the ON and OFF channels. Furthermore, orientation- and direction-specificity of cortical cells were unaffected by blocking the ON system, indicating that each channel has access to the mechanisms responsible for these attributes.

Given the abundance of new information detailed in the studies above, it appears that the system comprising the M and P pathways is more complex than the model put forth by Livingstone and Hubel. The question of whether or not the M and P pathways are

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truly separate remains unanswered. There may be partial segregation at certain levels of the cortex, but conflicting results reported from various laboratories have made the boundaries of functional specialization unclear. For example, Merigan and Maunsell (1993) note that the number of known connections between visual areas in the macaque cerebral cortex number over 300, and the complete set provides no clear impression of two parallel pathways. Furthermore, these connections differ in both their strength and their consistency.

As discussed earlier, problems exist in each of the main sectors of study (i.e., anatomical, neurophysiological, and psychophysical). In addition, there are no standards for carrying out such studies, and the methods employed under different conditions make the comparison of data sets difficult. As a result, making any sort of definitive conclusions is almost impossible.

Direct comparisons: Monkeys versus humans. The number of studies directly comparing macaque and human psychophysics is small. An early study by Weinstein and Grether (1940) compared visual acuity in rhesus monkeys and man. The authors report a similar minimum separable acuity of striated fields (discriminations made between striated and homogeneous fields; thresholds defined as smallest striae width at which correct discrimination was obtained in 75% of the trials) at three levels of brightness. This "similarity," however, is derived from the fact that, despite the monkeys' thresholds being higher than the humans' in all instances, in no case was the threshold visual angle for a human subject as much as 50% below the highest threshold for a monkey at the same brightness level. It seems more appropriate to call the acuities marginally identical,

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the authors stating that such a difference may be attributed, at least in part, to a shorter lens-retina distance in the monkey.

Sarmiento (1975) compared the stereoacuity of macaques to humans. Using the Howard-Dolman method, consisting of two vertical rods, one of which varies in depth, it was found that the monkeys' results corresponded well with the threshold disparity angle of humans. Yet, of five monkeys, only one successfully completed the experiment, making such a generalization questionably accurate.

De Valois and colleagues published a pair of papers (De Valois, Morgan, Polson, Mead, & Hull, 1974a; De Valois, Morgan, & Snodderly, 1974b) which directly compared a variety of psychophysical functions between macaques and humans. The main difference between these studies and others is that both monkeys and humans were tested using the same apparatus. Using a four-alternative forced-choice procedure, monkeys were required to signal which stimulus was different by pressing the corresponding window in which that stimulus appeared; humans were allowed to verbally make their decision. It was found that the flicker-frequency intensity function, Purkinje shift (coinciding with the photopic-scotopic system break point), scotopic sensitivity function, and photopic sensitivity function of macaques agreed considerably well with those of humans. Experiments dealing with color showed macaques to have trichromatic vision (based on the finding of no neutral points in their visible spectrum), Rayleigh matches (see below) comparable to humans', and that yellow is less saturated than red or blue (much like in normal human observers). There were, however, differences found, including absolute scotopic and photopic sensitivities, and overall poorer wavelength discrimination across the spectrum for the macaques. The authors (De Valois, et al.,

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1974a) also note that the interstimulus distance, in terms of angular subtense, was smaller for the human participants compared to the monkeys. A comparison of spatial luminance contrast sensitivity (De Valois, et al., 1974b) showed that macaque contrast sensitivity functions are very similar to those of humans, even as far as absolute thresholds (though humans are slightly more sensitive at the highest adaptation levels). A major limitation of the study (as pointed out by the authors) concerned the lack of control of pupil size, resulting in the inability to precisely quantify the effect of different adaptation levels on the contrast sensitivity function.

Interestingly, even more recent studies (e.g., E. L. Smith, Harweth, Levi, & Boltz, 1982) continue to use poorly designed methods and loosely controlled conditions (allowing for biased results), as well as comparing animal data to other laboratories' human data which were collected under different conditions.

Thus, despite the great similarities that exist between the visual systems of the monkey and human, there are differences, as well. As discussed earlier, the K pathway has, thus far, been found only in the monkey. In addition, Tootell, et al. (1995) have recently started to point out differences that are evident through neuroimaging. This raises the possibility that these differences, which may seem subtle at first, in fact, play a much greater role in visual information processing, making the monkey's visual system not as good a prototype of the human's as was once thought.

What is needed is a relatively stable population, preferably human, whose visual systems can be tested with various stimuli, where the need for the grouping of visual functions subserved by each of the individual systems can be easily obtained. Such a population exists, and as the following section explains, with a well thought out set of

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experiments, answers may be found to help in settling some of the aforementioned points of dispute.

## A Solution

Introduction. Let us assume that, when fully enabled, an element of complementarity exists, such that there is some overlap between the M and P pathways, and, in certain cases, one system picks up where the other system leaves off. The following questions may then be posed. First, if a deficit (i.e., deficiency or incompleteness, for example) exists in one pathway, how is the other pathway and it's functions affected? Second, how is the deficient pathway's other functions affected? To date, no study has systematically investigated these questions to any great length. Such deficits are common in the large population of observers who are color-deficient.

A color vision deficit necessarily implies a rewiring of the neural pathways, since one photoreceptor is different from those found in normal trichromats. Due to the variable nature and degree of rewiring between color-deficient observers, the possibility of the isolation of certain visual mechanisms under investigation exists. For example, the responses of color-deficient observers to chromatic stimuli differ from those of color-normals. But what about non-chromatic stimuli, such as a sinusoidal grating modulated at a certain frequency? If such stimuli also yield different responses compared to color-normals, the visual capacities evoked by the stimuli may share a common pathway. By comparing differences within the color-deficient population (anomalous trichromats versus dichromats versus monochromats), we may be further able to deduce the organization of the rewired systems.

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Trichromacy and color deficiencies. It should be pointed out that in this discussion the term "color-deficient" is used, as opposed to the more common, yet incorrect, label "color-blind," because the latter term implies that the affected individual is unable to perceive any color at all, and studies have shown that the proportion of individuals who fit this description (achromacy) is rather small (1 in 100,000 males; Gouras, 1991). The majority of colorblind individuals, thus, have at least some residual color perception, and, as will be shown, the degree of color perception lies along a continuum, ranging from very poor to nearly as good as non-color-deficient (color-normal) observers.

Normal trichromacy means the condition of having three color systems (i.e., three receptor types), and requiring three primaries to match any light (Levine & Shefner, 1991). Color matching is performed using a split-field display, in which one half is shown the light to be matched, and in the other is the sum of the three primaries to be used to make a match. Primaries can be any light or combination of lights, provided that no one primary can be matched by a mixture of the other two. The observer adjusts the portions of each of the primaries until a perfect match is made. Given that the spectra of the two half-fields are different, the match is only a perceptual one (a metameric match). In certain instances, a satisfactory match may be achieved only through the addition of a primary to the field to be matched to, the equivalent of adding a negative amount to the other two primaries.

Anything less than trichromacy falls under the heading "color-deficient." This includes not only dichromacy (where the individual possesses two color systems and requires only two primaries to match any light) and monochromacy (where only one color system is present), but also the group termed "anomalous trichromats," who maintain a

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three-parameter system, but within this, there exists one or more visual pigments with abnormal spectral absorbance characteristics (Levine & Shefner, 1991). In fact, of the approximately 8% of all males with a color deficiency, close to 70% are of the anomalous varieties (Nathans, Piantanida, Eddy, Shows, & Hogness, 1986a), with some estimates as high as 80% (Gouras, 1991).

The validation of the trichromatic theory of color vision has its roots dating back to the latter part of the nineteenth century. Among the different theories postulated, Hering's opponent-process theory deserves further attention. This theory proposes that the three fundamental color processes represent tradeoffs in red/green, blue/yellow, and black/white systems (Levine & Shefner, 1991). This theory was later quantified by Hurvich and Jameson (1957), in which they experimentally showed that observers were able to directly cancel out a hue sensation by adding a certain amount of the opponent hue (i.e., a stimulus initially perceived as red was "neutralized" by the addition of a certain amount of green, such that the stimulus no longer looked red, nor looked green). The quantities of opponent hues that were added could then be plotted as distributions of relative magnitudes of hue response evoked by each test wavelength (see also Romeskie, 1978).

Abramov and Gordon (1994), in discussing color appearance, have proposed a simple linear model of how the responses of the different cones may be combined to yield the hue mechanisms, with responses corresponding directly to sensations. It is important to note the cone weightings that produce these sensations. Spectral opponency is met through a subtraction of the different cones' responses, a characteristic found to belong to the P system (Livingstone & Hubel, 1988). Based on the above models, it appears as if

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the opponent-process theory has merit and can be used as a foundation upon which a number of psychophysical tests can be based.

Color-deficient observers, having missing or altered neural components, will have distinctly different mechanisms from those found in color-normals. Furthermore, the possibility of isolating the functions of the M pathway exists, because, depending upon the degree of color deficiency, the P pathway responses may, in some cases, be either highly, or even completely, suppressed (e.g., in monochromia). Then, in comparing responses both within the color deficient population, as well as between color-deficients and color-normals, the various roles and the degree to which each pathway is responsible for particular visual functions will be revealed.

An overview of colorblindness. Colorblindness was first reported in 1684 by Turberville (1684/1809), who had described the condition as merely "color peculiarities" (Judd, 1943; Parsons, 1914), but which may have been a psychiatric disorder (Emery, 1988). Boyle, in 1688, described, in a woman examined five years earlier, the symptoms of acquired achromatopsia (Mollon, 1989). Others (Edridge-Green, 1891; Geldard, 1972) cite that 1777 was the key year when Huddart recorded the instance of a certain Mr. Harris, a shoe-maker in Cumberland, England, who at age 4 found a child's stocking in the street, and upon inquiring the owner, a neighbor called it a "red stocking," though Harris did not understand why (Huddart, 1777).

The condition colorblindness was originally called "Daltonism" after the famous English chemist John Dalton who, in 1798, declared that while people in general distinguish six kinds of color, he saw two or three: yellow and blue, or yellow, blue,

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and purple (Dalton, 1798; Geldard, 1972; Dalton's yellow comprised the red, orange, yellow, and green of others, while his blue and purple coincided with theirs [Judd, 1943] as determined by Dalton after comparing the responses of various individuals' naming of colored ribbons). Dalton's discovery of this had reportedly occurred some four years earlier while viewing a geranium flower (Edridge-Green, 1891). The French still use the term "daltonisme" to describe colorblindness (Geldard, 1972). Thomas Young, whose name, along with Helmholtz', is synonymous with the trichromatic theory, had given Dalton the distinction of the first recorded protanope (Emery, 1988), but Hunt, Dulai, Bowmaker, and Mollon (1995) have recently shown that Dalton was, in modern classification, a deuteranope.

The taxonomic scheme that is most commonly used today was introduced by von Kries around 1890. Anomalous trichromia, differentiated into protanomaly (abnormal red function) and deuteranomaly (abnormal green function), was based on the finding by Lord Rayleigh (1881) of color normals needing different amounts of red and green in color mixtures. Dichromia consists of red-green blindness, taking the form of either protanopia (replacing the earlier designation "red blindness") or deuteranopia (replacing "green blindness"), and blue-yellow blindness, which is known as tritanopia. Monochromia is total colorblindness (Geldard, 1972). It should be noted that there are two types of monochromia, blue cone monochromia, and complete colorblindness, for which the term "achromia" is more suitable, since monochromia implies the existence of one type of cone, when, in fact, achromats have none. In addition, a second type of

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yellow-blue blindness has been found and termed "tetartanopia." In this rare deficiency, the affected individual has two neutral points (at ~470 nm and ~580 nm) as opposed to only one in the case of tritanopia (Hurvich & Jameson, 1955; Judd, 1943).

Color deficiencies can arise in one of two ways: congenitally and acquired. For the most part, the majority of cases of red-green color deficiencies are congenital (i.e., inherited), while most cases of yellow-blue deficiencies are acquired, often due to diseases of the outer retinal layer, although, in general, acquired color deficiencies can be caused by diseases of the central retina, optic nerve tract, optic tract or optic cortex, multiple sclerosis, blood dyscrasias, vitamin deficiencies, overexposure to toxins, and injuries (Judd, 1943).

Nathans and colleagues (Nathans, et al., 1986a; Nathans, Thomas, & Hogness, 1986b) have shown that the loci for the genes of the LWS and MWS cones are located on the distal part of the q-arm of the X-chromosome. Red-green colorblindness is a sex-linked recessive trait, which is why there are very few cases of females with such deficiencies. Only in instances where both the father is color-deficient and the mother is at least a carrier for the deficiency, will the deficit be passed on to female offspring (see Birch, 1993, for a discussion as to why the incidence of defective color vision in women is lower than the predicted value). The SWS cone gene is found on chromosome 7, and yellow-blue colorblindness is an autosomal dominant trait. Inherited variations are caused by alterations in the genes that encode each of the pigments.

Using the technique of Southern blot hybridization, Nathans, et al. (1986a,b) found that the observed genotypes appear to result from unequal recombination, or gene conversion, or both. The total number of genes found in color normals is highly

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variable to begin with, the number of MWS cone-opsin genes ranging from one to three (though there are reports of as many as seven [M. Neitz & J. Neitz, 1995]). If the genes are arranged in a tandem array, from head to tail, with the LWS cone gene(s) preceding the MWS cone gene(s), recombination can lead to the generation of a hybrid gene, the loss of a gene, or duplication of a gene. It was found that observers with anomalous trichromacy have at least one hybrid gene, the spectral sensitivity of which is determined by the exact point of crossing-over within the hybrid gene. The longer the fraction of the hybrid derived from one pigment as compared to the other, the more spectrally similar the encoded pigment will be to that pigment.

A test battery. Due to the uncertainty of M pathway contributions to visual capacities previously believed to be predominantly regulated by the P pathway (and vice versa), a large battery of visual tests has been compiled, which will measure a number of general visual functions within the chromatic, spatial, and temporal dimensions. These functions are visual acuity, color detection (perception), color discrimination, hue- and saturation-scaling, contrast sensitivity, binocular fusion, stereopsis, stereoacuity, vernier acuity, and motion sensitivity.

Since we are interested in discerning the neural wirings for each of these functions, it is important to first understand their neural bases. In the following sections, each function will be defined and discussed in terms of what is known insofar as the underlying neural components, what pathway(s) the Livingstone and Hubel model predicts the function should be subserved by, and what a comparison of this function between color-normal and color-deficient observers might reveal. The neural models

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discussed for each function are merely a representative sample, and should not be taken as an exhaustive search of the available literature.

Visual acuity

Visual acuity is defined as the ability to resolve finest detail, and its underlying neural basis is most easily shown by way of the decrease in acuity found as one stimulates more eccentric areas of the visual field. This is due largely to the combination of the organization of the receptor mosaic of the retina and the functions of the rods and cones (scotopic and photopic vision, respectively). As one goes more eccentric, in either direction from the fovea, the number of cones drops drastically. In addition, foveal cones, in general, have smaller receptive fields, whose centers often connect to separate ganglion cells. In comparison, rods and extrafoveal cones have larger receptive fields (with the center increasing in size with increasing eccentricity) with many receptors converging onto one ganglion cell. This convergence has been shown to be a factor in limiting resolution in this region (Goldstein, 1996; see also Croner & Kaplan, 1995).

A reduction in visual acuity must be interpreted very carefully. According to Spear (1993), there are many possible retinal/neural changes that could produce such a reduction. Within the retina, these include a loss of photoreceptors, bipolar cells, or ganglion cells, or changes in the connections among these cells. Anatomical or functional changes in geniculo-striate pathway, such as selective damage to the P pathway (but not the M pathway), have been shown to produce visual acuity deficits (see also Merigan, Katz, & Maunsell, 1991).

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But is visual acuity solely a function of the P pathway? It was described earlier how well received the notion is of the P system being called the "what" pathway and the M system being called the "where" pathway. Reasons for this include the differences in receptive field sizes of the P and M cells (especially the center, for which there is some overlap), the speed of each type of cell, and the deemed functions of each pathway in form perception (i.e., analysis of surface features of objects, in the case of the P pathway, versus whole object integration/location for the M pathway). However, the conditions under which the object is viewed must also be considered. Dim light may still be sufficient to yield a relatively fine degree of acuity. Whether there is some residual P input at these levels of illumination remains to be determined.

The possibility exists that there is a link between pigment genes and the mechanism underlying the packing-density of cones. Thus, in the case of a pigment gene not being expressed (i.e., in color-deficient observers), the packing of cones in these individuals would be different from that in color-normals, as would acuity. All battery tests will be conducted under conditions of "best corrected vision" (best optical correction) so as to eliminate potential biases that would otherwise not permit true measures. Such biases include various types of optical aberrations and retinal blur, found in such common vision disorders as astigmatism, hyperopia, myopia, and the like, as well as interobserver differences at optical infinity.

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Color detection/Color discrimination

According to Levine and Shefner (1991), "color attracts attention, enhances contrast between objects of similar lightness, and even appeals to our aesthetic senses" (p.387). It has been reported (e.g., Mollon, 1989) that, from an evolutionary standpoint, color vision evolved for the detection of fruit in the forest. Industrialization has taken us to the point where there are many things we take for granted, such as the consistent ordering of lights in a traffic signal. But if such cues were eliminated, and we were forced to discriminate on the basis of the color of the light, a number of individuals would have a difficult time on the road. Thus, some color-deficient observers have to make use of other cues for purposes of navigation (for a discussion, see Verriest, 1979). It is quite possible that these cues become heightened due to the great reliance placed upon them, much like a particular sense becomes heightened in the absence of another. As a result, in testing for color deficits, it is essential that the stimuli not contain any extraneous cues.

The methods used to diagnose color vision deficiencies, including colorblindness, are large in number and highly varied. Many tests were developed in an effort to better existing tests by correcting their flaws, only to later fall by the wayside themselves, in anticipation of a test that was both uniquely new and different (see Birch, et al., 1979, and references therein). Some researchers believe that the anomaloscope (see below) is the only accurate method, for it is not only able to diagnose the presence of a deficit, but it also is able to give the degree of deficiency. This is in contrast to other tests, such as pseudoisochromatic plates, which are often able to give only one measure, and often inconsistently, at that. If this is so, then why the varied number of different anomaloscopes produced over the years (e.g., Nagel I, Nagel II, Pickford, Pickford-

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Nicolson, etc.; see Birch, et al., 1979, for a review of the various types of anomaloscopes; Lakowski, 1972; Schmidt, 1955)?

The anomaloscope is an instrument designed, primarily, to evaluate specialized color matches and diagnose color defects in a large population. Making use of the Rayleigh equation, in which a special color match requires only two primaries (red and green) to match a test light (yellow), the anomaloscope's viewing aperture contains a bipartite field, and the observer's task is to vary the ratio of primaries to match the test light, such that the field appears uniform. Separate knobs control the radiant flux for each side of the field to attain a "perfect" match. The matching ranges for normal trichromats are narrow, whereas those of red-green dichromats are considerably larger, due to the primary lights lying on or close to the protanopic and deuteranopic confusion lines (Birch, et al., 1979).

The simple protanomalous trichromat (often abbreviated PA) makes a unique match containing higher proportions of red primary compared to normal trichromats. Their matching range also tends to be somewhat wider than normal trichromats'. Extreme protanomalous trichromats (EPA) have a very wide matching range, which includes the matches of both PA and normal trichromats, and may extend to one of the primaries. The protanope is able to match the test light to both primaries, as well as to all possible mixtures of the primaries. Simple deuteranomalous trichromats (DA) make a unique match that contains a higher proportion of green primary compared to normal trichromats. Their matching range also tends to be somewhat wider than that of normal trichromats. The extreme deuteranomalous trichromat (EDA) has a very wide matching range, which includes the matches of both DA and normal trichromats, and may extend to one of the

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primaries. The deuteranope matches the test light to both primaries and to all possible mixtures of the primaries. The main difference between the protanope and the deuteranope is that the protanope also experiences a luminosity loss for the red primary by a factor of about 10 (Pokorny, Smith, & Verriest, 1979; Rayleigh, 1881; Rushton, 1975).

While population norms for matching ranges and anomalous quotients (a statistic derived from the ratios of the primaries after a match has been obtained) exist for many of the commercially-made anomaloscopes, there are currently no models being manufactured. As a result, even devices built using previous models as guides are subject to reduced precision and cannot be stringently compared to the established norms.

W. D. Wright, a pioneer in the field of color vision, wrote, in one paper, that a combination of pseudoisochromatic plates, the Farnsworth-Munsell 100-Hue Test, and anomaloscope together are powerful, while no one test by itself is sufficient (Wright, 1972).

Pseudoisochromatic plates are designed to diagnose color-deficient observers by requiring individuals to identify a figure (usually a letter or number) embedded in a field of dots of varying chroma, size, and contrasts. When the elements comprising the figure and background are of equal brightness and consist of colors which are characteristically confused by observers with a particular type of deficiency, the figure is invisible (i.e., pseudoisochromatic) to the affected individuals. Some plates contain two figures, one of which, for example, is invisible to protans, the other invisible to deutans, while other plates can be read only by color-deficient observers (and are invisible to normal trichromats; Birch, et al., 1979).

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Arrangement tests (of which the Farnsworh-Munsell 100-Hue Test is one of several available) consist of having observers organize a set of color samples in a specified order, typically by hue or saturation, resulting in a measure of chromatic discriminative ability. The 100-Hue Test, for example, contains 85 samples divided into four separate boxes, and is designed to test hue discrimination and chromatic discrimination loss in color-deficient observers. Errors in certain regions of the hue circle are diagnostic of the type of color deficit, with total error score moderately indicative of the degree of severity. Other tests include the Farnsworth Dichotomous Test (Panel D-15), consisting of a subset of 15 color samples from the 100-Hue test, which selects observers with moderate to severe chromatic discrimination loss (as the full hue circle is represented in one box), and the Lanthony Desaturated 15 Hue Test. The Desaturated Panel D-15 is similar to the standard Panel D-15, but the color samples are paler and lighter (i.e., are less saturated), allowing for the diagnosis of milder cases of color deficiency (including individuals passing the standard Panel D-15 and acquired deficits), and resulting in a better classification of the severity of discrimination loss (Birch, et al., 1979; Lanthony, 1978).

Heeding Wright's words of advice, a wide series of color tests will be used in the visual battery. These are, for color detection, the Dvorine pseudoisochromatic plates, the Farnsworth-Munsell Dichotomous Test (D-15), and the Lanthony Desaturated 15 Hue Test, and, for color discrimination, the Farnsworth-Munsell 100-Hue Test, the anomaloscope, and a novel test concerning hue- and saturation-scaling (which examines both color detection and color discrimination).

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One of the main goals of this investigation is to further elucidate the underlying neural components of color vision, especially in the color-deficient population. The organization of the visual systems of color-normals will serve as a foundation.

The neural basis underlying the opponent-process color mechanisms was put forth by Hurvich and Jameson (1957, 1974). As discussed earlier, the opponent-process pairs can be thought of as being mutually antagonistic to one another, as illustrated by assigning opposite signs, indicative not only of response mode, but also to express the property that the ultimate net neural response is determined by the algebraic sum of the signed arousal inputs from the excited receptors to which each neural system is functionally related (Hurvich & Jameson, 1974).

This also parallels the linear model of Abramov and Gordon (1994; derived from a model by De Valois & De Valois, 1993) introduced earlier. This model was, in fact, based on the psychophysical results of hue- and saturation-scaling. The technique of hue- and saturation-scaling is simple. Given that there are four elemental or unique hue sensations that are paired in opponent-fashion to form the orthogonal hue dimensions red-green (RG) and yellow-blue (YB), observers are asked to state the percentages of their hue sensations, using any combination of the four unique terms, for a total of 100%. They also describe apparent saturation as the percentage of their entire sensation that was chromatic. J. Gordon, Abramov, and Chan (1994) discuss the excellent validity and reliability of this method, and the repeated findings that R and G form a mutually exclusive pairing of sensations (i.e., no "reddish-green" is reported), as do Y and B, lend further support to the underlying neural mechanisms of the opponent-process theory, upon which the method is based. While color-deficient observers have previously been

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tested on hue-scaling (see, e.g., Pokorny & Smith, 1977; V. C. Smith, Pokorny, & Swartley, 1973), a large scale population has not, to date, been thoroughly tested in this manner, nor tested in conjunction with the other functions we propose to study. With color-deficients' presumably altered P systems, the linear model representing their hue sensations will also necessarily be altered to some degree.

(Spatio-temporal) Contrast sensitivity

As shown previously, a major difference between the M and P pathways concerns their roles in contrast sensitivity (Kaplan & Shapley, 1986). Livingstone and Hubel (1987b, 1988) noted that the M cells have excellent contrast sensitivity at values below 10-15%, whereas the P cells are generally poor up until these values. This most certainly has some bearing on the later properties found in the respective pathways, as a whole, making this topic worthy of further discussion and study.

Contrast sensitivity is the inverse of the contrast required for the detection of a pattern (Levine & Shefner, 1991). The pattern often used is that of a sinusoidal grating, featuring alternating bands of light and dark, the contrast sensitivity being the reciprocal of the minimum contrast required for the detection of alternating bands. The spatial frequency of the sinusoid is also varied, thus giving rise to contrast sensitivities over a wide range of stimuli, from which a contrast sensitivity function can be plotted. In some cases, the sinusoids are presented as flickering, and in addition to finding the minimal contrast necessary to detect alternating bands, the observer is asked to reduce the amount of flicker so that it is just barely detectable. This gives an indication of the individual's

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temporal frequency sensitivity. Temporal frequency sensitivity might be thought to be largely dependent on the M pathway, considering the fast conduction and transient responses of the M cells, compared to the P cells. However, while this is found to be the case for high temporal frequencies, there is evidence that the P system contributes at lower frequencies (Livingstone & Hubel, 1987b, 1988).

A number of researchers have explored the neural mechanisms underlying contrast sensitivity. For example, Sekuler, Wilson, and Owsley (1984) used contrast sensitivity data, based on gratings of 0.5-16 cycles per degree (cpd), to try and determine the number of mechanisms needed to account for the properties of human spatial vision, and the characteristics that comprise these mechanisms. They found a three filter model to work best, with two of the three correlating with age. These results agree with the long-held notion that a number of spatially-tuned channels exist in human vision (see N. V. S. Graham, 1989, for a review; see also Billock & Harding, 1996; Peterzell, Werner, & Kaplan, 1993; Watson & Robson, 1981).

Schiller, Sandell, and Maunsell (1986), following the initial studies of Schiller (1982; discussed above) found that by pharmacologically blocking the ON channel in the visual systems of monkeys, contrast sensitivity was severely impaired.

Wilson, Mets, Nagy, and Kressel (1988), in studying differences between normal spatial vision and that found in albinos (a topic that will be considered in greater detail later), concluded that albino spatial vision parallels infant spatial vision at ten months of age (see Peterzell, et al., 1993). This, they believe, is largely due to the relative lack of extensive neural connections; cone spacing is 3-4 times greater than in normal foveas, and the length of their central cones' outer segments are reduced.

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Shapley (1990) discusses that response as a function of contrast grows much more steeply (approximately eight times more) for M than for P neurons, especially at low contrasts, and notes that this is found early in the visual pathway at the level of the RGCs. It is at this level that the neural innervation controlling contrast sensitivity originates. Contributing factors to this difference of growth rate include receptive field size and antagonistic cone interactions in some P cells.

Based on the known properties of the M and P pathways, it may be hypothesized that color-deficient observers will show contrast sensitivity functions quite similar to normal observers for low to middle spatial frequencies (largely a function of the M pathway), but reduced sensitivities to higher spatial frequencies (fine discrimination, dependent on P pathway), despite using achromatic gratings. Because we are dealing with various rewirings in these observers, the reverse ordering may also occur. Since temporal frequency is controlled by both pathways, it will be of interest to see if any compensatory mechanism exists, or if all contrast sensitivities at low temporal frequencies are poorer than those at high frequencies (paralleling the sustained and transient properties of the P and M pathways).

In the course of testing color-deficients, their range of responses may vary considerably as a result of the degree of color anomaly, as well as the uncertainty of a common rewiring scheme within groups of individuals with similar color discrimination abilities; however, assuming non-acquired color-deficiency is a result of genetic variation (Nathans, et al., 1986a,b), there should be considerably less variation among color-normal observers. Even though a number of color-normal males express more than three cone types (J. Neitz, M. Neitz, & Jacobs, 1993), the effect on the variation of normal color

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vision is both subtle and predictable (J. Neitz, et al., 1993; M. Neitz & J. Neitz, 1997). Furthermore, despite the existing variability in the number of pigment genes and cone types expressed, the number of possible combinations is finite. On the other hand, the degree of color anomaly lies on a continuum, ranging from very mild to severe, making for a much more heterogeneous group of conditions.

Because some comparisons will be made between color-deficient and color-normal observers, it is necessary to have certain guidelines with which we can assess the accuracy of our test method. While even small procedural differences can lead to variable results across studies, these results are sufficiently similar enough to provide us with a measure for determining whether we are producing effective data.

In attempting to find reasonable studies upon which to make such judgments, it should be mentioned that with functions such as the contrast sensitivity of sinusoidal gratings, animals may not be best for comparison purposes, especially when trying to set "norms." The particular environment of the organism has been shown to alter visual functions, from an evolutionary standpoint, to better serve the needs of the species. For example, the cat, which may have been useful when considering the anatomy and neurophysiology of the visual pathways, has a peak contrast sensitivity of about 0.5 cpd, well below that of humans (Blake, 1988; Ulrich, Essock, & Lehmkuhle, 1981). As a result, we will limit ourselves to studies using only human participants.

Historically, the first to measure human spatial contrast sensitivity was Schade (1956). The range of spatial frequencies that humans are sensitive to range from 0.4-60 cpd, with peak sensitivity occurring usually in the range of 4-6 cpd (Banks & Salapatek, 1981; Blake, 1988; Blakemore & Campbell, 1969; Campbell & Maffei, 1974; Campbell

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& Robson, 1968; Kelly, 1972; Kulikowski, 1971), although these values are found to decrease with age (Owsley, Sekuler, & Siemsen, 1983).

When factoring in temporal frequencies, Robson (1966) reported that lower temporal frequencies generally yielded higher contrast sensitivities across the range of spatial frequencies discussed above. A 1 Hz flicker, however, was lower than a 6 Hz flicker for spatial frequencies less than 2 cpd. Furthermore, fall-off in sensitivity at high spatial frequencies was found to be independent of the temporal frequency (and vice versa), and a fall-off in sensitivity at low spatial frequencies occurs only when the temporal frequency is also low (and vice versa).

The general findings of Robson have been reported subsequently by Kulikowski (1971), Kelly (1972), Tulunay-Keesey (1972), and Burbeck (1981). Kelly (1979a,b) has subsequently shown that image stabilization elevates the observer's contrast threshold (by a factor close to 20 in the case of a 3 cpd stationary grating), and that eye movements have important effects on the thresholds for drifting and flickering gratings. The role of eye movements in maintaining the unstabilized contrast sensitivity is best simulated by a constant, low velocity, compared to any other form of temporal variation (Kelly, 1979b).

Binocular fusion

A slight disparity exists between the two eyes insofar as the retinal images that form. One way to observe this is to hold a thumb about six inches out in front of you. With one eye closed, fixate on some object farther away, positioned to one side of your thumb, and alternate opening and closing each eye while viewing the thumb. There is a noticeable difference in the position of the fixated object relative to the thumb. The object

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appears to jump, back and forth, from one side to the other. Yet, the visual system is able to take this disparity, and, in many instances, incorporate the differences between retinal images into a single cyclopean percept. Thus, the resulting image (of the fixated object, in the above example), when viewed binocularly, is a single, clear one.

The different images are largely due to the approximately 6 cm distance between our pupils. For objects that are less than 100 ft away, the view of the object from each eye is slightly different. We are able to adapt to this disparity by positioning the eyes so that the left and right images of the object fall on corresponding points on the two retinas (Kandel, 1991; see Figure 2).

While we were able to correct for the disparity in the above situation by binocularly viewing the object, limitations become apparent in situations where more than one object is viewed, such as the complex scenes we experience everyday. At any given time there is only one point of fixation. The crisp clarity of the object being fixated upon, as mentioned above, is due to corresponding retinal points. The horopter is an imaginary circle that passes through the point of fixation, as well as all other points in the visual scene with zero disparity, at a given fixation distance. All of the points on the horopter will be perceived as being at the same distance from the observer as the fixation point (Levine & Shefner, 1991; see Figure 2).

There are, however, objects in the overall visual scene which are seen as both single, but in depth. Panum's fusional area is the region of space in front of, and in back of the horopter in which the disparity is small enough, such that binocular fusion is still

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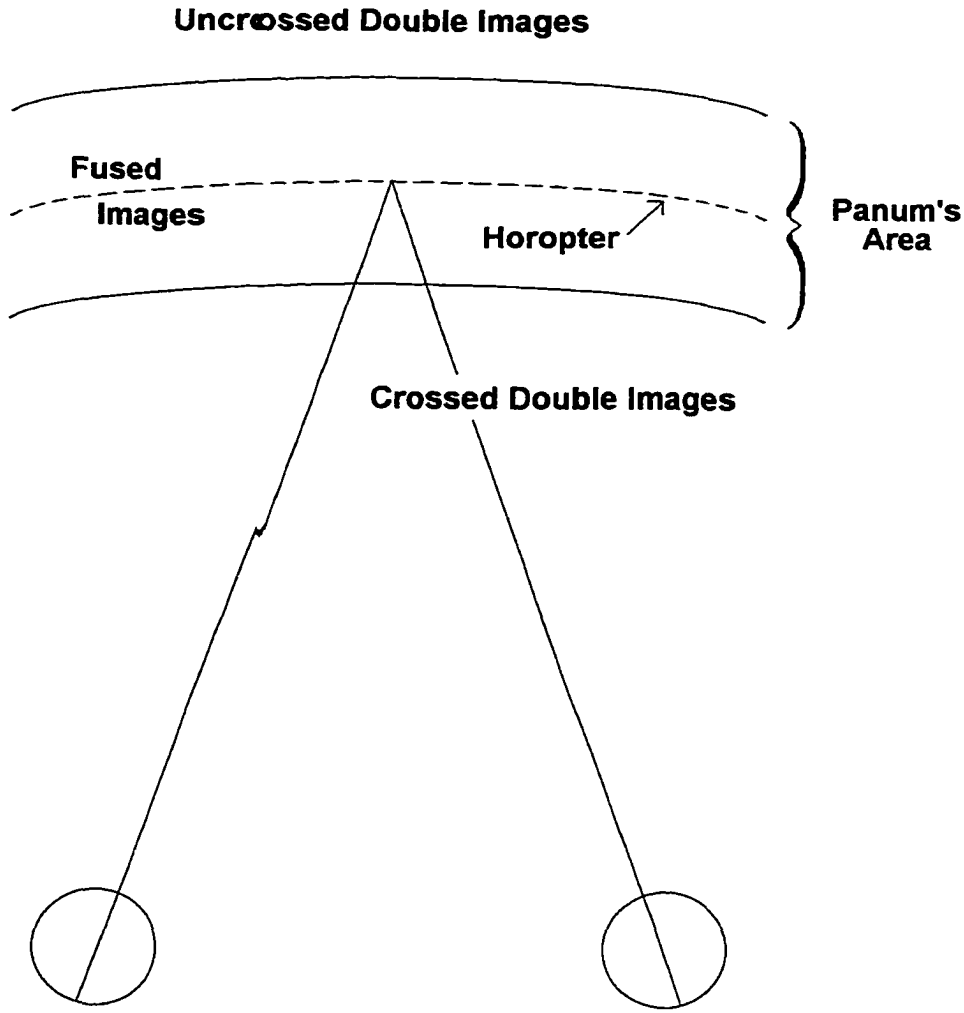


Figure 2. Schematic diagram depicting the horopter and Panum's fusion area.

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possible. This allows one to determine which of two objects is closer to the observer, based on the angle of disparity. When objects are located in front of the horopter, their images move out to the sides of the retinas, creating a crossed disparity; objects located beyond the horopter create images that move inward on the retinas, yielding uncrossed disparity (Goldstein, 1996). Objects lying outside the range for binocular fusion (i.e., outside Panum's area) create a diplopic (double) image, of which we still have a residual ability to categorize as being nearer or farther away than the point of fixation (see Figure 2).

There are many ways of measuring binocular fusion. One of the most common ways is through the testing of stereopsis. Stereopsis is our ability to see an image in depth, when viewed binocularly. As discussed below, while binocular fusion is necessary for stereopsis to occur, it is not sufficient for it. Both disparity and a disparity-sensitive mechanism are necessary.

While the construction of the first stereoscope by Wheatstone in the 1830's places the outset of the study of stereopsis at more than 150 years ago, it is has only seriously become an intense area of research in the last 40 years.

Early studies of stereopsis, which attempted to measure observers' ability to see an image in depth, turned out to be hampered by the stimuli used. Many researchers found that monocular cues were confounding the results. It was the formulation of the random dot stereogram (RDS) by Julesz (1971) that avoided this problem. The RDS is a pair of images, each consisting of a pattern of black and white dots, which, when viewed monocularly, appear to be highly random in spacing. However, when viewed binocularly and fused, the two images create an in-depth image. The reason this occurs is that the two

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separate images are actually related, in the sense that the pattern of dots are identical for the most part, with the exception of a portion of the dots, which, in one image, is shifted with respect to the other image. This is based on the naturally occurring horizontal shift between corresponding points in the two retinal images (i.e., retinal disparity) as mentioned above. According to Julesz (1971), the idea behind the RDS is that it deprives the subject of all familiarity cues, and, as a result, the stimuli, when presented individually, do not contain any global information on the retinae.

It has been found that approximately 2% of humans are stereoblind (Julesz, 1971, 1986; Richards, 1970). These individuals are unable to see images in depth due to a problem involving the disparity mechanism not functioning properly. Causes of such a condition include childhood monocular deprivation and uncorrected visual refractive errors in one eye (Levine & Shefner, 1991). The stereoblind group includes persons with strabismus or some other known functional inability of one eye or of eye-coordination. An additional 15% have a stereopsis deficiency, often diagnosed by the ability to see simple RDSs, but difficulty in seeing more complex ones. This group includes individuals having strong astigmatism which has not been corrected (Julesz, 1971).

For those who possess stereopsis, additional measures can be made regarding the limits of stereoscopic vision. Such measures include the determination of the limits of Panum's area, the greatest distance at which an object can be just detected as nearer than an object at infinity, as well as the minimum detectable disparity. The latter case is termed "stereoacuity," and the fundamental question being asked is "What is the smallest degree of disparity that is permissible which still allows for the ability to see the object in depth?"

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Livingstone and Hubel (1987b, 1988) found stereopsis to be entirely an attribute of the M pathway, although this is questioned by De Yoe and Van Essen (1988). Livingstone and Hubel reach their conclusion largely on the fact that under conditions of equiluminance, depth was not evident in RDSs. (The problematic nature of isoluminance was discussed earlier.) De Yoe and Van Essen, however, stress that the physiological properties of the parvocellular-interblob stream overlap with those of the M stream, including disparity. Coupling this together with the possible notion of interactions among the pathways as early as in V1, does not permit a definitive conclusion of color-deficient versus non-color-deficient properties of stereopsis. Along the lines of Livingstone and Hubel, in the population of color-deficient observers who possess stereopsis, identical results to color-normals, also possessing stereopsis, would be expected, since an altered P pathway would cause no alterations in this function. A difference in stereoacuity, however, need not necessarily imply that there is P cell involvement. Stereopsis could still be a function largely controlled by the M pathway, but because M cells receive input from LWS and MWS cones, the M pathway may be altered as a result.

The neural basis of stereopsis has an interesting history. Barlow, Blakemore, and Pettigrew (1967) found neurons in the cat primary visual cortex that were more highly selective for binocularly positioned stimuli than monocularly positioned stimuli, and that disparity in the retinal images of such stimuli yielded much smaller responses. Furthermore, there are neurons that optimally responded to objects with different disparities lying at different distances. Julesz (1986) follows this by noting that, in man, pools of disparity neurons tuned to different binocular disparities must interact to result in the various examples of stereoscopic plasticity (e.g., the ability to fuse two RDSs varying

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in linear size without a problem). This is precisely what Richards (1971) described. He hypothesized the existence of three such pools, in order to account for the different types of stereoscopic anomalies observed during psychophysical experimentation (see Discussion).

Poggio and Poggio (1984) consider two types of cortical neurons involved in stereopsis: tuned (excitatory and inhibitory versions) and reciprocal (near and far versions). Tilt was added as an additional neural mechanism by Blakemore, Fiorentini, and Maffei (1972; see also Pettigrew, 1972).

Blake and Wilson (1991) have proposed three classes of models to attempt to explain the underlying neural mechanisms. The first class, employing cooperative interactions, eliminate what are termed "false targets" (ambiguities arising if the brain were to mismatch features contained in the left eye's image and the right eye's image) through excitatory and inhibitory interactions. Another class involves spatial frequency filters, each limited to processing a restricted range of disparities, serially going from coarse to fine. The third class involves feed-forward models, whereby local, non-interacting computations are performed, in parallel, for purposes of maintaining speed. While no model is completely flawless, the authors conclude that neural cooperativity and interactions must necessarily occur for such an efficient mode of operation as that of human stereopsis to take place.

Schiller et al. (1986), upon blocking the ON pathway, found there to be mild impairment of the perception of stereoimages. With both M and P ON cells, the possibility of P cell involvement in stereopsis cannot be discounted (also, see above).

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The eye is a very imprecise organ. No two people have identical visual functions. Thus, among individuals possessing stereopsis, there will be a range as to their limits of stereoacuity. Frey (1953, as cited in Gulick & Lawson, 1976), in fact, found there to be a positive correlation between stereoscopic acuity and visual acuity.

Various methods have yielded ranges of stereoscopic acuity thresholds. Howard (1919, as cited in Gulick & Lawson, 1976), in determining observers' least depth difference between viewing position and two rods that could be discriminated 75% of the time, found thresholds as low as 2 s arc in very sensitive observers. This was duplicated by Woodburne (1934, as cited in Gulick & Lawson, 1976), even when keeping retinal size constant. Matsubayashi (1937, 1938a,b, as cited in Gulick & Lawson, 1976) studied various factors that influence stereoscopic acuity (including target size, angular separation between stationary and moveable rods, distance of observation, and monocular acuity). Thresholds varied from 1.5 to 6 s arc. Foley (1978) cites studies reporting that, under optimal conditions, disparities as small as 5 s arc can be discriminated. Julesz (1971) reports an average of 2 s arc can be discriminated between two lines breaking in depth, though the longer the line, the smaller the break that can be detected.

McKee (1983) has determined that the finest stereoacuity (less than 5 s arc) is found with isolated vertical target lines 10-15 min arc in length. Increasing the length beyond 20 min arc produces little improvement due to the decrease in stereoacuity at small eccentricities. Presenting the target as little as 5 min arc in front of or behind the fixation plane elevates the threshold, as well. Unlike most other studies which concentrate primarily on determining minimum disparity, this study recognizes that there

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also exists a maximum disparity at which depth perception is lost (the upper limit of Panum's area in the region of uncrossed disparity). Studies on this are rather sparse.

Monocular hyperacuities

The size of a foveal photoreceptor is approximately 0.5 min arc in diameter. Yet, stereoacuity and other spatial acuity tasks yield thresholds considerably smaller than this. These types of tasks are subsumed under the name "hyperacuity" tasks (Geisler, 1984). One of the most often used hyperacuity tasks is vernier acuity. In its simplest form, vernier acuity consists of two abutted lines, slightly displaced from one another. The objective is to determine the minimum amount of displacement necessary in order to just detect that a break occurs. The observer may also be asked to describe the direction of the displacement of one line with respect to the other.

An excellent example of the underlying neural basis of hyperacuities comes from a study investigating the vernier acuities of an unconventional group of human observers. Freeman and Bradley (1980) found that, compared to a group of normal observers, a group of individuals who had been monocularly deprived for a period of time showed a lower vernier acuity threshold (i.e., were more sensitive and had a higher acuity) in their non-deprived eye. This parallels earlier work involving monocular deprivation in kittens. These early studies showed a shift in ocular dominance and an absence of areas of unresponsive cortex, pointing to neural reconnection and neural plasticity. Freeman and Bradley found that the ratio of means of vernier offset of binocular subjects compared to monocular subjects was on the order of $\sqrt{2}$. This led the authors to hypothesize that if vernier offsets were detected by noisy neural arrays, and, via developmental recruitment,

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a second channel was made available to process signals through one eye, the improvement in sensitivity of that eye, over one connected in a normal binocular pattern, would be $\sqrt{2}$. The authors, thus, attribute this "supernormality" in the monocularly deprived observers as possibly due to active neural recruitment as the result of disuse of one eye.

Studies intended to specify the contributory role that each pathway plays in hyperacuity have produced mixed results and interpretations. Westheimer and Pettet (1990), for example, in discussing the neural basis of vernier and stereoscopic acuities, point to the neuronal ensemble processing involved in the retrieval of positional information. The positional difference in an alignment task is in the same direction in the two eyes, and in opposite directions when disparity is introduced, yielding threshold differences due to differences in the subsequent pathways. In considering the limitations of hyperacuities, the authors consider this visual capacity to be a function of both pathways. The P pathway would be responsible for hyperacuity thresholds, since it fails for conditions of low contrast and short duration. This would necessarily require the M pathway as being capable of some positional, but not disparity, localization in order to account for vernier alignment at low contrast or short exposure durations. The ability of the P pathway to respond to displacements of hyperacuity proportions is backed by Shapley and Victor (1986), who found this property in X-cells in cats at a magnitude comparable to that found in human vision. (This would translate into the existence of cells with similar characteristics in both monkeys and humans.)

Lee and co-workers (Lee, Wehrhahn, Westheimer, & Kremers, 1993, 1995; Lee, Westheimer, Wehrhahn, & Kremers, 1992), on the other hand, have found that detection

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of displacement of an achromatic edge led to much more vigorous responses from macaque ganglion cells of the M pathway, compared to those of the P pathway, indicating that phasic ganglion cells of the M pathway provide the significant retinal signal for the cortical mechanism responsible for vernier detection (especially at contrasts up to 20%). Livingstone and Hubel (1994) also consider hyperacuities a function of M mechanisms.

The ranges of reported vernier thresholds vary somewhat. Burr (1979) found, for line segments presented stroboscopically and seeming to apparently move at a rate of 1.6°/s, two observers' acuities for 75% correct identification of spatial offset were 6.6 and 10.3 s arc. Levi, Klein, and Aitsebaomo (1985) measured four observers' vernier thresholds by asking them to judge the location of one of two abutted horizontal lines relative to the other. The pair of lines, subtending 1.5° of visual angle, yielded thresholds of approximately 5-7 s arc. Westheimer and Hauske (1975) found, for two lines with zero onset asynchrony presented for one s, thresholds on the order of about 5 s arc. Westheimer and McKee (1977b) found that lowest thresholds occurred when line lengths were approximately 3-4 min arc, with gap separations of 2-5 min arc between vertical lines. In another paper (Westheimer & McKee, 1977a), it was shown that vernier acuity thresholds were better for moving targets (at a rate of 1.8°/s) than for stationary targets. This leads one to consider the M pathway as having a key role due to the motion aspect.

Motion sensitivity

Motion sensitivity is largely due to the M pathway, as evidenced by such properties of the M cells as their fast, transient responses, and the selective properties found in area MT in the M pathway (Livingstone & Hubel, 1987b, 1988).

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Of the many ways to measure motion sensitivity, one way, which avoids confounds from additional cues not relevant to motion itself, is based on the RDS. It is called a random dot kinematogram (RDK), which is very similar to the flip movie books found in cereal boxes. At least two RDSs are presented in succession. The objective is to determine the minimum amount of displacement of dots necessary for the perception of motion to take place. It is often required that the observer state the direction of motion, as well. There also exists a maximal displacement threshold, at which point the direction of movement is no longer clear. It seems as if the separate arrays of dots have no commonalities, and, thus, appear to be unrelated. In between these two extremes lies a continuum, spanning areas which are perceived as smooth motion, as well as areas in which dots appear to literally jump from one spot to the next, although direction of motion is still readily discernible.

Early concepts of the neural bases of motion were supplied by Beverley and Regan (1973, 1975). They investigated visual sensitivity to movement in depth, measured as a function of the relative distances through which the left and right retinal images moved. They found that irregularly spaced dots, when binocularly fused and sinusoidally oscillated from side to side, yielded an appearance of oscillation in depth. Furthermore, altering the amplitudes of oscillation (left to right) changed the direction of the line of apparent oscillation in space.

Adaptation to movement directed to the left of a point located midway between the eyes depressed visual sensitivity for test targets which moved to the left of this midway point, but did not depress visual sensitivity for test targets which moved more than 1-2° to the right of this midpoint. A reverse effect was found for rightward

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movement. As a result, the authors hypothesized the existence of two groups of monocular detectors of retinal image position sensitive to either right-to-left movement or left-to-right movement. Either left or right monocular input to a given disparity detector can be group 'a' or group 'b,' but not both, yielding four possible groups of disparity detectors. Each of these is further subdivided into those selectively sensitive to motion directed to the left of center and those to right of center. Four groups of disparity detectors are tuned to four different directions of motion towards the head and four additional groups are tuned to the same four directions away from the head (Beverley & Regan, 1973). The directional selectivity of binocular motion detectors are due to the combination of left-to-right and right-to-left monocular signals being segregated, and contralateral detectors responding only when the contralateral eye's retinal image is moving faster than the ipsilateral eye's image (Beverley & Regan, 1973, 1975).

Srinivasan (1985) describes visual subsystems which perform directionally-selective movement detection as being colorblind. He states that movement detection and direction discrimination require a comparison or correlation of visual signals crossing from at least two receptors or visual interneurons that view neighboring regions of visual space. Fluctuation in time and advancement in time of one channel versus another indicates movement and direction of movement, respectively. Directional selectivity exists, as well, based on excitatory and inhibitory neural interactions. It is reasoned that if the two channels in question possess the same spectral properties, then the movement detector is, itself, colorblind. It is only in such a case that movement detection is at its optimum. The input channels need not be derived from a single spectral class of receptor. A given species' input channel's properties for maximally sensitive motion detection

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takes many factors into consideration, including visual habitat. For primates and man, there is evidence that signals from the three cone types (but see Lennie, Pokorny, & Smith, 1993, for evidence that SWS cones do not contribute) are pooled to create "luminance" signals prior to detecting movement. The spatial properties of the channels should also be identical in terms of their receptive fields.

Schiller et al. (1986) found only a mild impairment of the perception of movement upon pharmacologically blocking the ON channel.

Ramachandran and Gregory (1978), using RDKs, found that color and motion are handled separately by the human visual system, with color providing only a weak cue, at best, to movement perception. Livingstone and Hubel (1988) agree, pointing out that movement perception exhibits colorblindness, quickness, high contrast sensitivity, and low acuity, as evidenced by impairment for patterns made up of equiluminant colors at high spatial frequencies; these patterns, however, are vividly perceived with very rapidly alternating or very low contrast images. This gives further support for motion as an M pathway function.

If motion is, in fact, dependent on the M pathway, one might be tempted to hypothesize that color-deficient observers should not have any problems in any motion sensitivity tasks, and there should be no difference between the motion sensitivities of color-deficient and color-normal observers. However, one aspect of neural rewiring that cannot be overlooked is the effect of receptoral changes on the M pathway. We are currently uncertain as to whether neural rewiring is dependent on the severity of color-deficiency, and whether this rewiring scheme is consistent among individuals with similar

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anomalies. Such differences may result in different M pathway responses, and, consequently, different motion sensitivities between persons.

J. F. Brown (1931) recorded the threshold for movement of three observers for objects with different dimensions. Means of 0.2-0.3 cm/s led to the conclusion that factors affecting the velocity (e.g., homogeneity of the surrounding field, size of object, path of motion) also affect the threshold by a comparable amount.

R. H. Brown and Conklin (1954) identified the lower threshold of visible movement, for a sample of four observers and a stimulus exposure time of 0.5 s, as 17.68 min of visual angle/s. Leibowitz (1955) reported a lowest threshold of 0.25 min arc/s.

An early version of RDKs was tested by Oliver Braddick (1974) in an attempt to find the limits of coherent motion as a function of displacement. The study, however, focused on the perception of movement of the entire square in which the dots were located. That is, the perceptual segregation of the object began to deteriorate at a displacement of about 5 min arc, and was complete for displacements of about 20 min arc. It was concluded that it is over this range of displacements that coherent motion is lost.

This was followed by a pair of papers by Chang and Julesz (1983a,b) in which they tried to determine the maximal displacement ($d_{max}$) for motion-direction. It was found that $d_{max}$ increases approximately as the square root of the display area. At a viewing distance of 100 cm, an 8 x 8 dot target (where 1 dot equals 3.478 min of visual angle) yielded a $d_{max}$ of approximately 4 min arc; for a 16 x 16 dot target, the threshold was about 7 min arc; for a 32 x 32 dot target, $d_{max}$ averaged 13.7 min arc; for a 64 x 64 dot target, $d_{max}$ was about 23 min arc. In each case, the same target viewed at 50 cm gave

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a higher threshold. Furthermore, it was shown that thresholds were unaffected by retinal eccentricities up to 4° (Chang & Julesz, 1983a). When the same measurements were made with band-pass filtered stimuli (Chang & Julesz, 1983b), grain size was found to be the limiting factor of $d_{max}$. This led to the conclusion that apparent movement perception is a global process (i.e., no unique local features can be detected in the two successive arrays, resulting in only a global, correlation-like operation available to eliminate false matches). The sample size was limited to three observers in each of these papers, making deviations from the reported average results, upon replication, not unlikely.

G. Johansson (1978) cautions that motion thresholds studied are valid only for the stimulus context in which they are recorded, due largely to their variabilities as a function of their spatial and temporal parameters. This may well be the case for other stimuli and functions also.

The purpose of the present investigation is to study visual functions, including acuity, color perception, spatio-temporal contrast sensitivity, binocular fusion, hyperacuity, and motion sensitivity, of both color-normal and color-deficient individuals. In comparing the results, both within and between these two populations, we will be able to gain further insight into the neural mechanisms and roles of the magnocellular and parvocellular pathways in visual perception.

## Method

### Participants

The participants used in this study consisted of 40 female and male adults of various racial and ethnic groups between the ages of 17 and 69 ($\underline{M}$ = 27.05, $\underline{SEM}$ = 1.68,

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Mdn = 24.50), 80% (32/40) of whom were Caucasian, 12.5% (5/40) of whom were African-American, and 7.5% (3/40) of whom were Hispanic, Asian, Indian, Pacific Islander, et cetera (hereafter referred to as "Other"). Demographically, there were 22 females, ages 17 to 53 (M = 25.95, SEM = 1.99, Mdn = 22.50), and 18 males, ages 17 to 69 (M = 28.39, SEM = 2.85, Mdn = 25.50). Of the females, 81.8% (18/22) were Caucasian, while 9.1% (2/22) each were African-American and Other, respectively. The group of males was comprised of 77.78% (14/18) Caucasians, 16.67% (3/18) African-Americans, and 5.56% (1/18) Other.

All participants were judged to be healthy (both mentally and physically) throughout the period of testing, except that some individuals were confirmed as having color vision deficits. Participants were, thus, further subdivided into three groups depending on their type of color vision: "color-normal" (with no known or measurable color vision deficit), "acquired color-deficient" (with a known or measurable color vision deficit, more often tritan-like in nature [see, e.g., Verriest, 1963], often confined to one eye, of late onset, and no known familial history of color vision problems), and "genetic color-deficient" (with a known or measurable color vision deficit, affecting both eyes to approximately the same degree, and a known familial history of color vision problems).

The color-normal group consisted of 19 females and 9 males, ranging in age from 17 to 53 (M = 25.54, SEM = 1.60, Mdn = 23.00). 75% (21/28) were Caucasian, 14.29% (4/28) were African-American, and 10.71% (3/28) were Other. The females ranged in age from 17 to 53 (M = 26.53, SEM = 2.26, Mdn = 23.00), 78.95% (15/19) were Caucasian, 10.53% (2/19) were African-American, and 10.53% (2/19) were Other. The males ranged in age from 20 to 29 (M = 23.44, SEM = 1.32, Mdn = 23.00), with 66.67% (6/9) being

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Caucasian, 22.22% (2/9) being African-American, and 11.11% (1/9) being Other. The acquired color-deficient group included 3 females and 4 males, aged 18 to 69 ($\underline{M}$ = 31.57, $\underline{SEM}$ = 6.46, $\underline{Mdn}$ = 27.00). The females were between 18 and 27 years of age ($\underline{M}$ = 22.33, $\underline{SEM}$ = 2.60, $\underline{Mdn}$ = 22.00), all of whom were Caucasian. The males ranged in age from 25 to 69 ($\underline{M}$ = 38.50, $\underline{SEM}$ = 10.27, $\underline{Mdn}$ = 30.00), of whom 75% (3/4) were Caucasian, and 25% (1/4) were African-American. The genetic color-deficient group was exclusively male, the 5 members ranging in age between 21 and 48 ($\underline{M}$ = 29.20, $\underline{SEM}$ = 4.81, $\underline{Mdn}$ = 26.00), all Caucasian.

The majority of participants were Brooklyn College students and Hunter College students who wished to participate in the study. Recruitment was made via notices posted on bulletin boards near the Psychology Department offices at Brooklyn College and Hunter College, through classes in which the principal investigator taught/assisted, through fellow students, and through friends. Recruitment fliers containing computer-scanned images of a few of the Hardy-Rand-Rittler pseudoisochromatic plates (Richmond Products, Boca Raton, FL) were made in order to help in identifying suitable color-deficient candidates. Written informed consent, approved by the Human Subjects Committee of the Institutional Review Boards of both Brooklyn College and Hunter College, was obtained from each participant at the time of the initial meeting, prior to the commencement of testing. A complete copy of the consent form appears in Appendix A. All testing took place at the Applied Vision Institute and Visual Research Laboratory in the Department of Psychology at Brooklyn College, CUNY.

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Apparatus

Acuity. The optics of each observer's eyes were measured using a number of different available techniques. Participants underwent full refraction with the Ultramatic Rx Master Phoroptor (Reichert Ophthalmic Instruments, Buffalo, NY, model 11625), used in conjunction with the B-VAT II Video Acuity Tester (Mentor O & O, Norwell, MA), in order to determine the individual's best optical correction, according to an acuity criterion. The Topcon KR-7000 Auto Kerato-refractometer (Topcon America, Paramus, NJ) which directly measures the optics (i.e., sphere, cylinder, and axis) of each eye, was, in certain instances, used as a cross-check for the results from the phoroptor. Any observer who usually wore glasses also had their existing prescription checked with a lensometer (American Optical Corporation, Buffalo, NY, model 12603). Any differences between prescriptions was noted by the experimenter, and the observer was informed that they needed to have an eye exam done and/or was given a written prescription for new glasses to be made (see below). Individuals requiring slight corrections (and those unable to fill their new prescriptions in a timely manner) were supplied with trial frames of the appropriate strengths to use during testing.

Color perception. Color vision was measured using a number of standard tests for color vision deficiencies. These were the Dvorine pseudoisochromatic plates (2nd ed.; Harcourt, Brace, and World, New York, NY), the Farnsworth Dichotomous Test (Panel D-15; Psychological Corporation, New York, NY), and Lanthony's Desaturated 15 Hue Test (Luneau Ophtalmologie, Paris).

For viewing the Dvorine plates, the Farnsworth D-15 and the Lanthony Desaturated 15 Hue Test, the illumination chamber depicted in Figure 3 was used.

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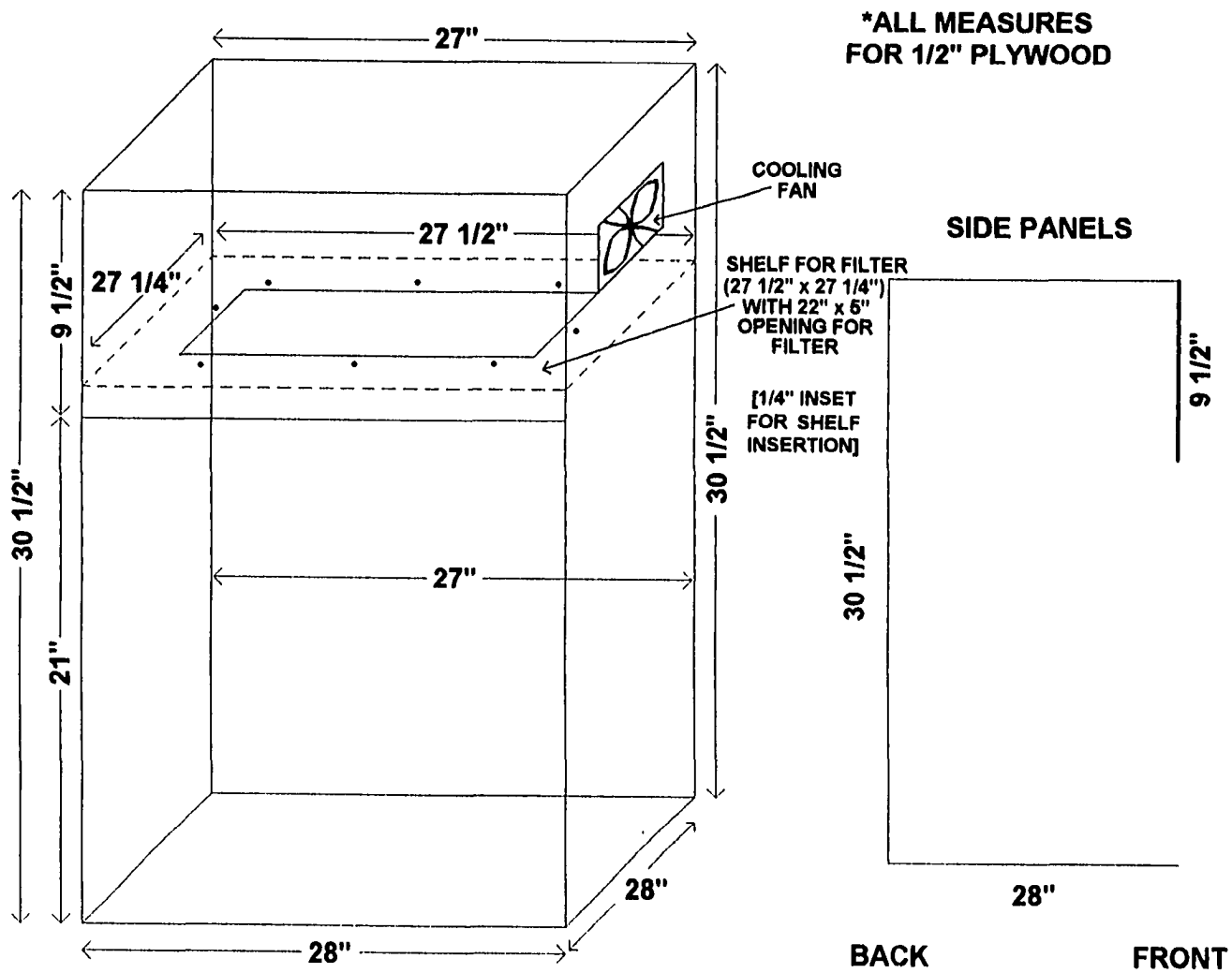


Figure 3. Dimensions of illumination chamber used to run the Dvorine Color Plates, Farnsworth D-15, Lanthony Desaturated 15 Hue Test, and Farnsworth-Munsell 100-Hue Test.

Briefly, this is an enclosed chamber, illuminated by a set of four 20-Watt Tru-aim halogen light bulbs (Sylvania, Winchester, KY) affixed to WAC lighting lamps (WAC, College Point, NY, model HHT-802). The lights are positioned 6.5" apart on a lighting track, which is bolted to the underside of the top of the chamber. The lights hang 24" above the floor of the illumination chamber. Twenty-one inches above the floor sits a shelf, which serves as a holder for colored filters used to control the color temperature and illuminance of the chamber. The set of filters used for these tests consists of Rosco Cinegel filter numbers 3202, 3204, and 3216 sandwiched between two pieces of Plexiglas. This produces a color temperature of approximately 6700 K and a luminance of 95 cd/m$^2$ for a white reflectance standard placed at the testing point, as measured by a Photoresearch PR-703A spot spectrascan fast spectral scanner. The lamps are cooled with a 115 V cooling fan (ETRI, model 125XR), and the entire viewing area is painted a flat white.

Color discrimination. Color discrimination was measured by the Farnsworth-Munsell 100-Hue Test (Macbeth Corporation, Baltimore, MD), also using the illumination chamber for viewing, and by the technique of hue- and saturation-scaling.

Hue- and saturation-scaling. The apparatus used for hue- and saturation-scaling was adapted and modified from Abramov and Gordon (1977) and Abramov, Gordon, and Chan (1991). Stimuli were presented in Newtonian view by an optical system in which light from a tungsten-halogen bulb was collected with a lens and filtered with an infrared heat-absorbing glass filter. The lamp was powered by a variable transformer (variac), which was used to vary the lamp's intensity. The lamp filament was focused on the entrance slit of a grating monochromator (Bausch & Lomb, Rochester, NY, high

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intensity, model 33-86-76); the monochromator's slits were set to give a half-power bandpass of 6.4 nm (entrance = 1.8 mm, exit = 1.0 mm).

For each trial, the variable transformer was adjusted, via a stepping motor, to yield a previously determined output from a calibration table. After passing through an electromagnetic shutter (Uniblitz, model 214L), the light was focused onto a piece of ground glass which served as a rear-projection screen (measuring 1.59 cm in diameter) that appeared uniformly illuminated, and subtended a 1° visual angle at a viewing distance of 86 cm. Calibration values were made using a photocell, which was hung directly in front of the rear-projection screen.

The apparatus was controlled through Alpha-Bus (Alpha Products, Woodhaven, NY). The monochromator's wavelength drive was connected to the stepping motor, which was manually set to a wavelength of 430 nm at the start of each session. Look-up tables controlled for non-linearity in the wavelength drive. The variac was also motor-controlled, and was manually set to its zero point at the start of the session. The look-up tables specified values for each wavelength based on spectrophotometric (Photoresearch PR-703A spot spectrascan fast spectral scanner) calibrations. Separate calibration files for young (< 30 years) and standard observers were used. The young observer file consisted of a blue-luminance correction to account for young observers' enhanced sensitivities to short wavelength light, relative to that of older observers whose sensitivity is decreased due to factors such as the aging of the macula and the yellowing of the crystalline lens. All stimuli were equated for effective luminance to yield an estimated retinal illuminance of 27 trolands (td; derived from Table 15 in Le Grand, 1957).

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Anomaloscopy. A second optical system served to produce heterochromatic flicker to obtain spectral sensitivity functions, as an anomaloscope for purposes of obtaining Rayleigh matches, as a source of adaptation to test for multiple LWS and/or MWS cones, and to measure SWS cone sensitivity. The schematic is shown in Figure 4.

The anomaloscope was housed in a wooden box, constructed using a Dexion cage-like frame, complete with a trap door located on top for easy access to the optics and light-emitting diodes (LEDs). A peephole, measuring 1" in diameter, bored in the front panel, allowed for monocular viewing of the test field. The test field, when viewed at the appropriate distance (see below), subtended slightly less than 2° visual angle.

The anomaloscope, itself, consisted of six independent, computer-controlled, beams of light (blue, green, red, red adapting, yellow, and yellow adapting) emanating from one of five separate integrating spheres (made from plastic Ping-Pong balls and baseballs spray painted white to increase opacity and minimize light loss; the green and red beams came from the same ball, either singly or in combination). The sources of the light were high intensity LEDs (ranging in number between 2 and 6 per beam: blue = 6, green = 2, red = 4, red adapt = 6, yellow = 6, yellow adapt = 6) of the appropriate color (blue: Ledtech, model LT18B3-81-SB, green: Ledtronics, model BP280CWAG6K-3.5VF-050T, red: Ledtronics, model L200CWR3KF, and yellow: AND, model 157HYP; T-1 3/4 size; 9 mm in length). Various beam-splitters, lenses, filters, and computer-controlled stepping motor-driven field-stops and mirror were used to create a large array of different full- and half-field stimuli to be used in the anomaloscope test procedure (see Figure 4 for full layout details). "Waffled" masonite, painted a flat black, isolated the beam(s) emitted from each sphere, while absorbing much of the light scatter created by

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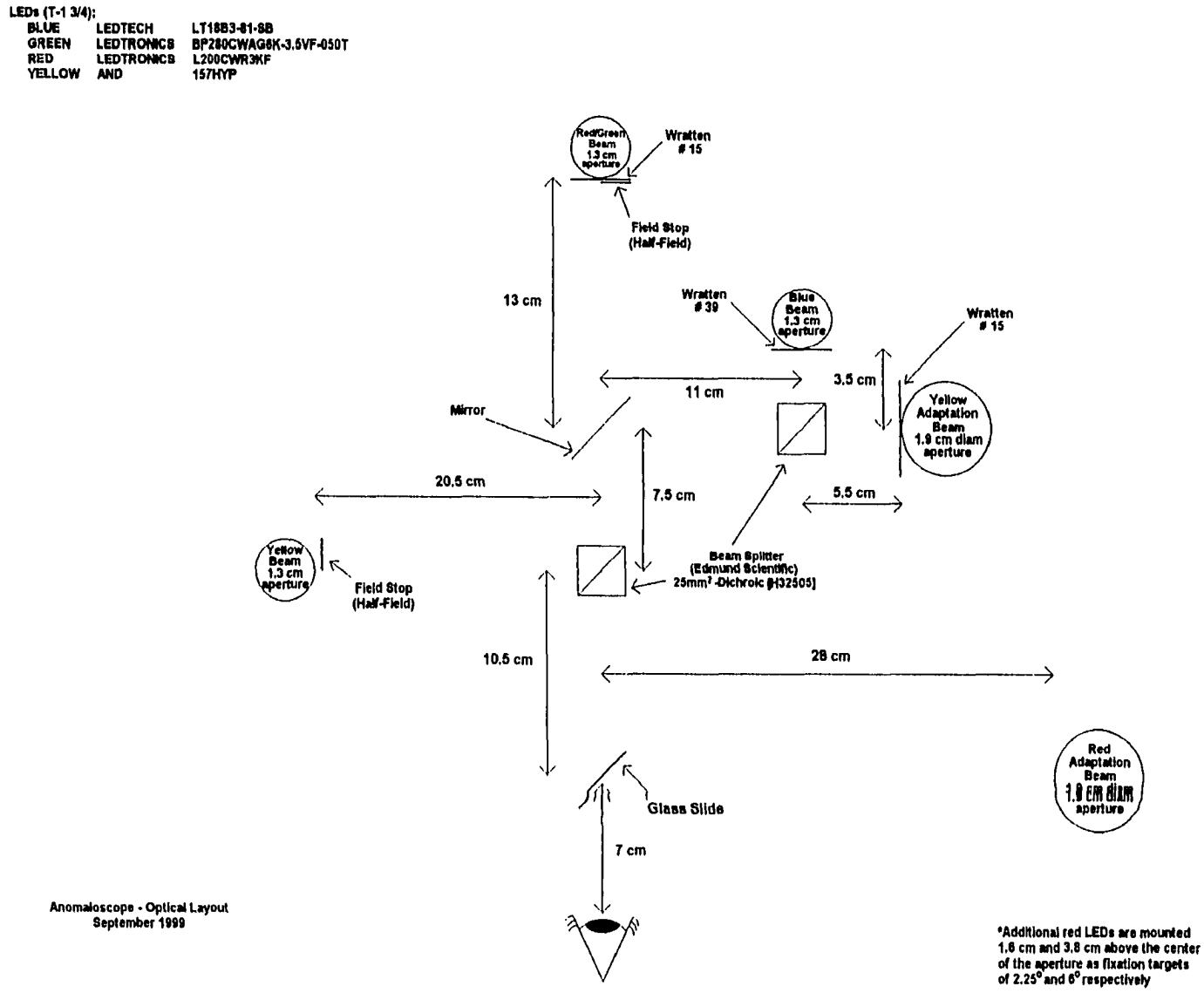


Figure 4. Optical system design and layout for anomaloscopy, HFP, and rod and SWS cone sensitivities.

the lights bouncing between the optics.

The LEDs comprising the test beams (blue, green, red, and yellow) were arranged in the Ping-Pong balls, with circular apertures of 1.3 cm in diameter. Those comprising the adaptation beams (red adapt and yellow adapt) were arranged in the baseballs, with circular apertures measuring 1.9 cm in diameter. The green and red test beams, as well as the yellow adaptation beam, were blocked by Wratten No. 15 filters (Eastman Kodak, Rochester, NY), effectively limiting the spectra to wavelengths longer than 520 nm. The blue beam was blocked by a Wratten No. 39 glass filter, limiting its spectrum to wavelengths shorter than 500 nm.

For the testing of heterochromatic flicker photometry (HFP), green (mean wavelength = 534 nm) and red (mean wavelength = 662 nm) beams were separately combined with yellow (mean wavelength = 596 nm), through a beam splitter (25 $mm^2$, dichroic cube; Edmund Scientific, Barrington, NJ, model H32-505), at one of three possible temporal frequencies (15, 20, or 25 Hz). This allowed for the observer to perceive a solid, flickering field of some hue or combination of hues (see Figure 5, upper left panel).

In order to create a bipartite field used for Rayleigh matches, the same beam splitter mentioned above was employed. The center of the beam splitter was 20.5 cm from the apertures of the green/red and yellow test integrating spheres. Half-field field stops with razor-blade edges, located directly in front of these spheres' apertures, allowed for only a portion of the beam to pass. This was maintained at the beam splitter, where the unblocked portions of each beam (i.e., the combined green/red and the yellow) converged, presenting to the observer a circular bipartite field, consisting of green-red and

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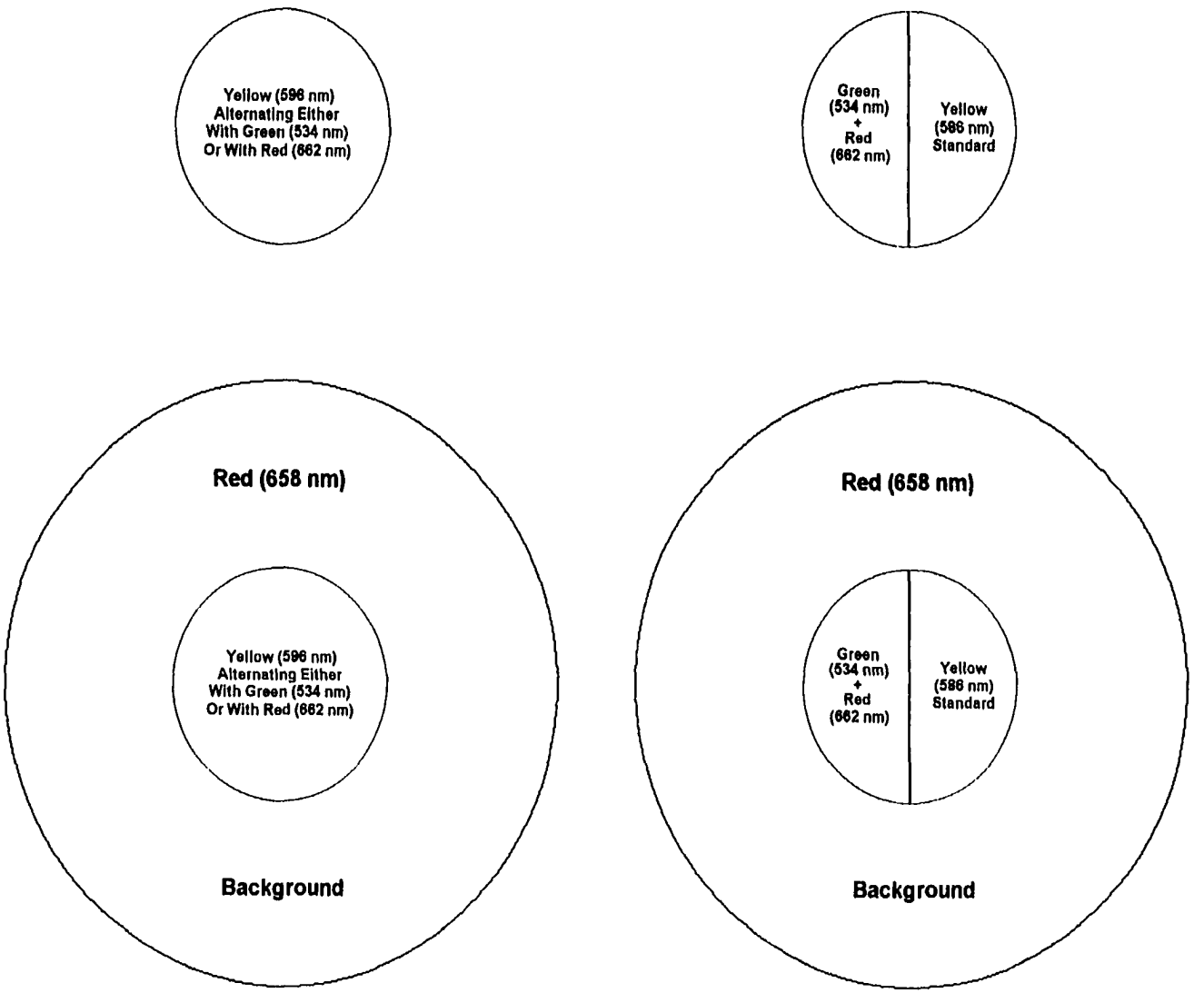


Figure 5. Anomaloscope session stimuli. Upper left panel: HFP; upper right panel: Rayleigh matching; lower left panel: HFP with chromatic adaptation; lower right panel: Rayleigh matching with chromatic adaptation. See text for further details.

yellow half-fields, the partition being formed by the edges of the half-field field stops. When viewed at a distance of 38 cm from the integrating spheres, the bipartite field subtended a visual angle of just under 2° (see Figures 4 and 5, upper right panel).

To test for the presence of multiple LWS and/or MWS cones, an additional red beam (mean wavelength = 658 nm) was combined with the existing beams from HFP and the Rayleigh match. This beam, when projected through a glass slide, situated at a 45° angle directly behind the peephole, created a large field (background) within which sat a smaller field, that of either the HFP or Rayleigh bipartite field (see Figures 4 and 5, lower panels).

We measured rod contribution to correct for SWS cone sensitivity (described below). Absolute limens were found using pulsing stimuli, separately, from the blue (mean wavelength = 425 nm) and green beams at two retinal eccentricities (see Figure 6, left panel). Two additional red LEDs were mounted above the center of the red adaptation integration sphere, one at 1.6 cm, the other at 3.8 cm, to give the observer fixation targets of 2.25° and 6° eccentricity, respectively. An inconel 3.0 log unit neutral density filter was used to optimize the range of potential thresholds, while minimizing the change in luminance from one digital to analog (D to A) step (see below) to the next.

SWS cone sensitivity was tested by measuring increment thresholds for a short-wave test light (mean wavelength = 425 nm) on a yellow adapting background (mean wavelength = 596 nm). The background suppressed LWS and MWS cone signals, thereby isolating the SWS cones. For this, a second beam splitter, of the type described above, was used. This beam splitter combined the blue and yellow adapting beams of light, projected them on to a mirror (situated at a 45° angle to the observer) which then cast the

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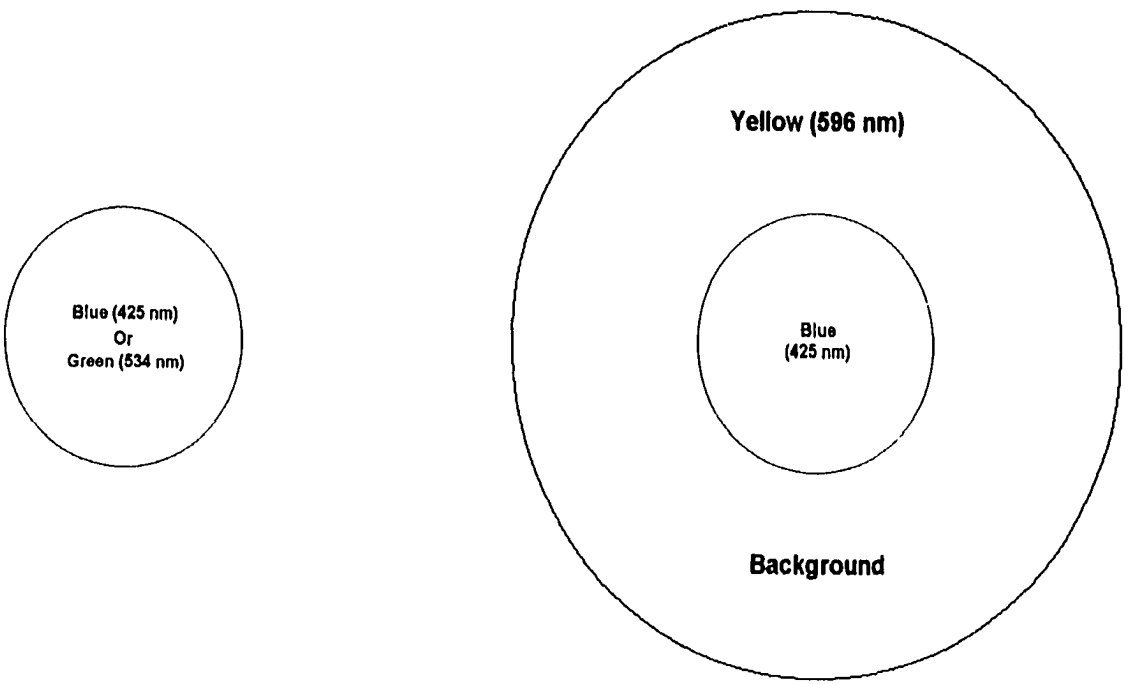


Figure 6. Rod and SWS cone sensitivity session stimuli. Left panel: rod sensitivity; right panel: SWS cone sensitivity. See text for further details.

beams through the other beam splitter, then through the glass slide, and finally through the peephole to the observer. The total length of this path was 41.5 cm from the yellow adaptation integrating sphere, and 39.5 cm from the blue, again giving an angular subtense of just under 2° (see Figures 4 and 6, right panel).

For each of the beams, the circuitry used to run the LEDs was adapted from Nygaard and Frumkes (1982; see their Figure 2), as modified by Alden (1983).

Observers used a numeric keypad to indicate whether they needed more or less light from any given beam. These inputs were used by the computer to control the output of D to A channels that, in turn, controlled linear operational amplifiers that gated high current transistors driving each set of LEDs. D to A values were determined from look-up calibration tables. These tables were based on luminance values obtained using a Photoresearch PR-703A spot spectrascan fast spectral scanner, for each of the 256 D to A steps.

Spatio-temporal contrast sensitivity. The stimuli used to test for contrast sensitivity and temporal frequency sensitivity were computer-generated sinusoidal gratings. The program used to generate these stimuli was VisionWorks (version 2.26; Vision Research Graphics Inc., Durham, NH). The computer used was a 90 MHz pentium processor (PC Warehouse). Stimuli were presented on a monochrome monitor (Image Systems Corporation, Hopkins, MN, model M21L-H5S01-117A) which was occluded, except for a central circular aperture, measuring 8.5" in diameter (subtending a visual angle of 3.4° at 3.6 m), constructed from plywood painted a flat white. The plywood also served as a surround. The aperture, whose center was located 46" above the floor, as well as the surround, was further illuminated by six 25 W picture lamps, whose intensities

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were controlled by a variac set to 70% of 115 V. The mean luminance of the monitor screen with the surround lights on was 55 cd/m$^2$, while that of the surround was 25 cd/m$^2$. The 1931 (x, y) CIE coordinates of the screen were (.443, .533), while those of the surround were (.523, .427).

The spatial frequencies used for the contrast sensitivity experiment were 0.57, 1, 2, 5, 12, and 24.38 cpd; temporal frequencies were 1, 4, 8, 15, and 24 Hz. Contrast sensitivity thresholds were measured by using the QUEST paradigm of Watson and Pelli (1983), as described in the Procedure section.

Binocular vision testing. Eye alignment was tested using the white maddox rod filter along with the rotary prisms built into the phoroptor, with the B-VAT's Snellen letters (white on dark background) as a target; letters were set close to the acuity limit to ensure accommodation to the target distance (6 m).

A pupillometer (Essilor Instruments, Creteil, France) was used to measure observers' interpupillary distances.

Ocular rivalry was tested using a standard hand-held stereo viewer (Keystone, model No. 50, Home Training Stereoscope, Meadville, PA), set to its "far" position setting.

Stereopsis. Stereopsis was tested three ways. For purposes of detecting the ability of the observer to see in stereoscopic depth, the TNO plates (5$^{th}$ ed.; Lameris Instrumenten, Utrecht) were used along with the accompanying spectacles. This test was conducted under the illumination of a 14 W fluorescent bulb. The two other screening tests, used in a similar fashion, were the Randot Stereotest (Stereo Optical Company, Chicago, IL) and the Titmus Fly test (Stereo Optical Company).

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To measure the limits of Panum's fusional zone, computer generated RDSs (through VisionWorks), measuring 2.5" square and subtending a visual angle of 2° at a distance of 1.8 m, were used. The RDS was presented against a dynamic surround. There were three black dots (fixation spots; 5 mm in diameter) spread evenly across the central 7" of the diameter of the surround's aperture, with the middle dot located directly in the center of the aperture (and RDS), and the others located approximately 8 cm to the left and right. In addition, the RDS was flanked, centrally, by two semi-transparent vertical bars (nonius lines; 5 mm x 75 mm; see Figure 7). Of the three fixation spots, only the central dot was seen by both eyes; the others were each seen by only one eye. As a result, the central fixation point appeared as single only when both eyes properly converged at the distance of the screen. Similarly, each bar was seen by only one eye (the upper bar by the left eye, the lower bar by the right eye); both lines were visible when the eyes were properly aligned and converging on the central fixation spot. RDSs consisted of random dots of 1.3 min arc pixels.

Stereo glasses (Newport Corporation, Irvine, CA) were used to present separate images to the left and right eyes. The images were alternated at the frame rate of the display (approximately 120 Hz) and were demultiplexed by the ferro-electric shutters. The shutter glasses allowed each eye to view only the proper display frames. A single stereo frame consisted of two frames (one for each eye), and took approximately 1/60 s. (The stereo frame rate was approximately 60 frames/s.) When an image was presented to one eye, the other shutter was closed off, rendering no visible image. With the aid of the stereo glasses, observers could see the image in depth, and, furthermore (in conjunction with the keyboard), could manipulate the amount of depth, relative to the plane of the

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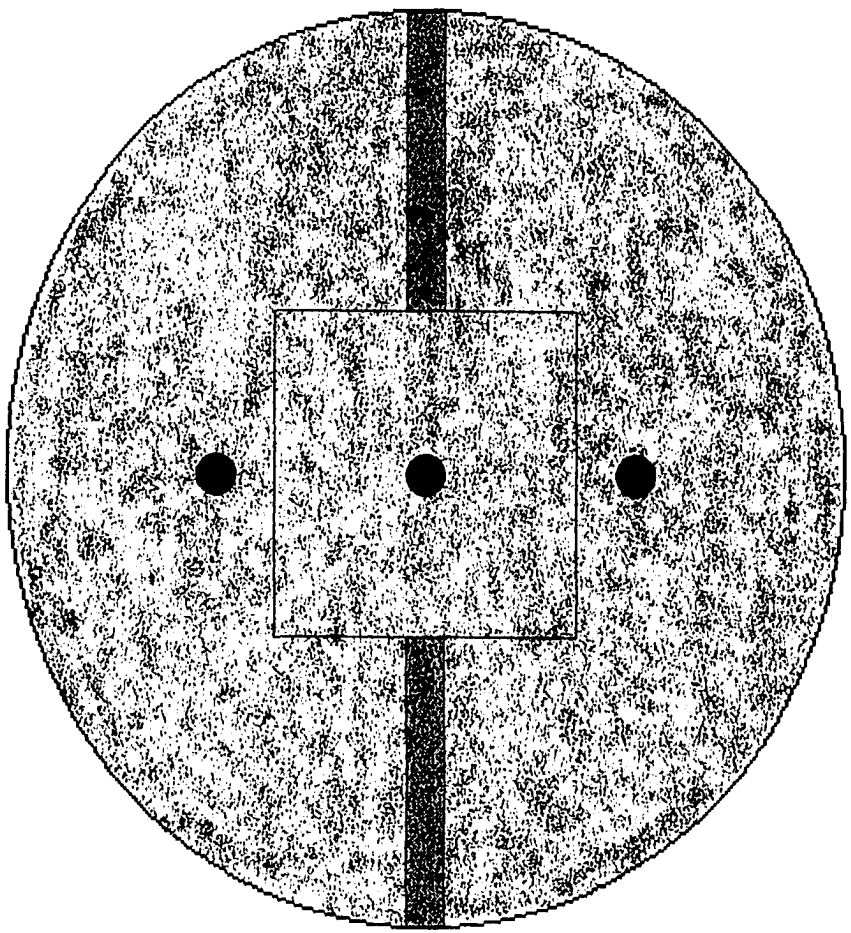

Figure 7. Stereopsis session stimulus. The central square was randomly presented as either popping out or popping in. Surrounding dots were in constant motion.

screen. Such manipulations were used to determine minimal and maximal amounts of displacement still yielding a sense of depth, insofar as the observer being able to determine the direction of depth relative to the plane of the screen. Using the QUEST paradigm, as described below, the minimum and maximum displacements, both in front of and in back of the plane of the screen, were determined.

Monocular hyperacuities. Vernier acuity was measured from computer-generated (VisionWorks) vertical black bars, abutted, but slightly displaced relative to one another. The bars, which together measured 2.5" x .06" (length x width) and subtended a visual angle of 2° x .05° at a viewing distance of 1.8 m, were located in the center of the surround's aperture. The upper bar was always fixed; the lower bar was slightly displaced, either to the left or right, relative to the upper bar (see Figure 8). Using a forced-choice procedure, thresholds were found with the QUEST paradigm.

Motion sensitivity. Motion sensitivity was tested using pseudo-RDKs. The difference from standard RDKs was that no stereoscopic fusion was required, even though the density of dots was the same as that found in the RDSs (and, consequently, in standard RDKs). A computer-generated (VisionWorks) random dot square, measuring 7.5" square and subtending a visual angle of 6° at a distance of 1.8 m, was centered in the middle of the surround's aperture. Successive presentations of this pattern (100 ms each), with areas of dots displaced either up or down, to some extent yielded the perception of motion (i.e., apparent motion; see Figure 9). Using the QUEST paradigm and a forced-choice procedure, both the minimum and maximum amounts of displacement, still rendering the correct identification of direction of motion, were determined.

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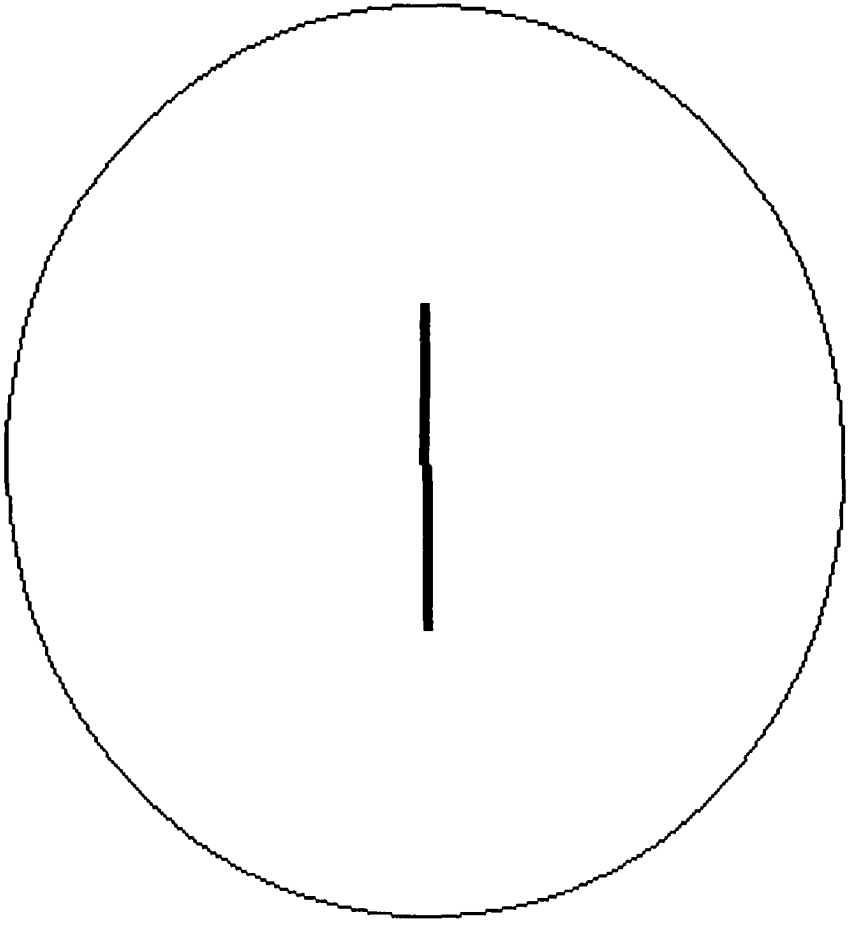

Figure 8. Vernier acuity session stimulus. The upper bar always remained fixed, while the lower bar was randomly displaced either left or right.

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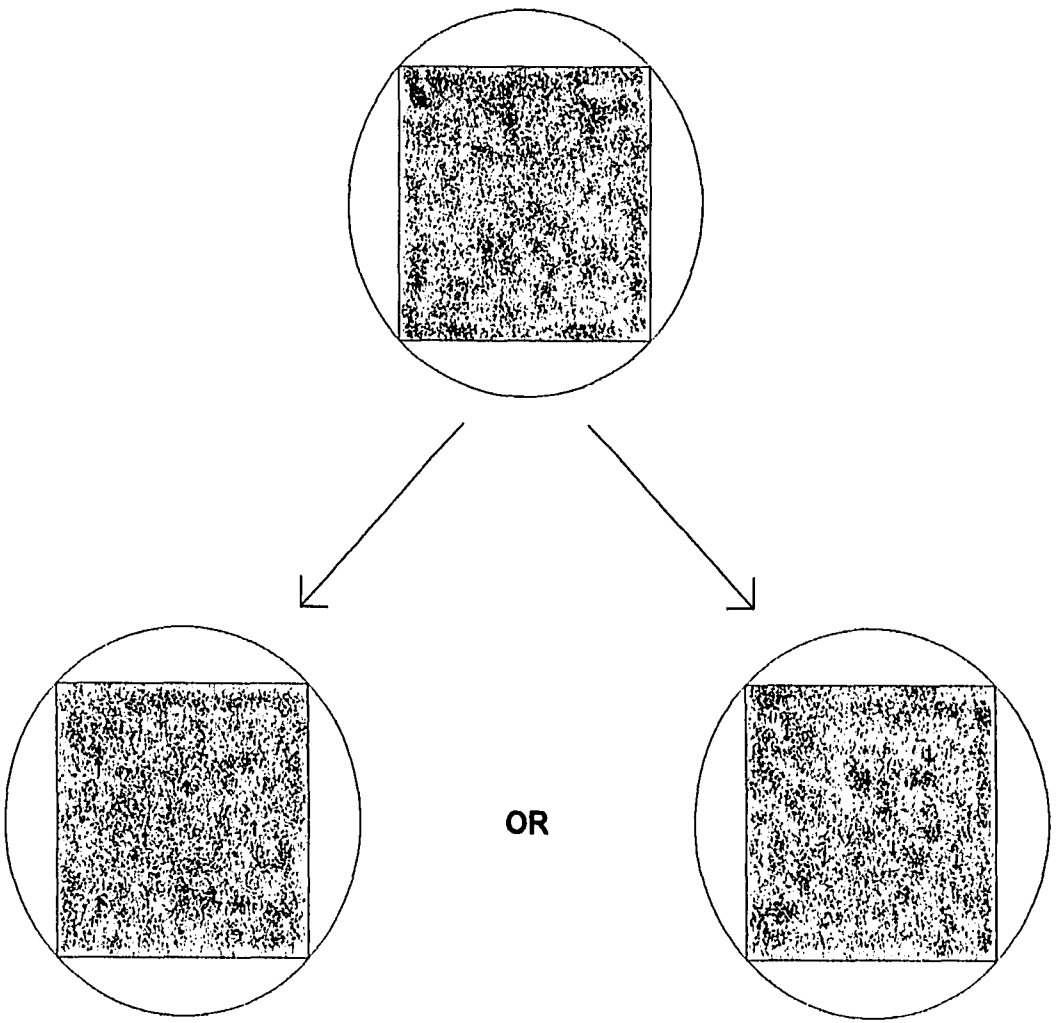


Figure 9. Motion sensitivity session stimuli. Following presentation of the initial stimulus (top), subsequent frames randomly displaced the dots either up (bottom left panel) or down (bottom right panel).

Ocular and dermal spectral reflectivity. With the possibility that pigments other than visual photopigments might affect, or correlate with, visual performance, each observer's ocular and dermal spectral reflectivity was measured. Using a Photoresearch PR-703A spot spectrascan fast spectral scanner, spectral radiances were taken of both the iris and a patch of skin from the inside of the participant's forearm.

Procedure

While it was our original goal to run each of the battery sessions in a random order, the need to administer certain tests in a fixed sequence was determined quite early in order to progress logically from one aspect of visual testing to the next. In all instances, the first session concerned participant intake. This was usually followed by optical testing, to insure that all tests were run with optimal (and best) correction, especially those requiring distance viewing. Color-screening (specifically color perception) needed to be conducted early, as it served not only to aid in an early diagnosis of color-deficiency (i.e., having some sort of color deficit versus not having any), but informed the experimenter that the observer was to be run monocularly in all subsequent tests (a requisite for all color-deficient observers for purposes of gaining maximal clarity and precision in diagnosing and assessing such a person's color vision).

The anomaloscope was the last piece of apparatus to be finished being built. This resulted in this test being non-accessible by certain participants (i.e., they were never run on it), and in it being one of the final sessions run in the battery by others. Individuals' time constraints regarding scheduling and availability were also taken into consideration, thus, making the battery sessions pseudo-random in orientation. For current purposes, where applicable, the order in which each test will be discussed will follow that as

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presented in the Introduction and Apparatus sections. All experiments were run in a clear, careful, and consistent manner as set forth in the laboratory's "Battery Manual," appropriate excerpts from which can be found in Appendix A.

Intake. At the first meeting, important information regarding the observer was compiled on the intake data sheets and spreadsheets (see Appendix A). After signing the informed consent, a thorough intake procedure was begun. The intake is a comprehensive assessment of the participant's personal and familial visual, physical, and mental health histories. Questionnaires covering such aspects as the cardiovascular, neurological, and allergy/immunological (including illnesses and medications taken for each) realms of health and fitness were used to get as precise an estimate of different factors that may influence the various measures obtained in the battery, either directly, or through the experimenters' interpretations. A familial history of color vision deficiency, for example, in conjunction with the observer not consciously perceived by him- or herself to be afflicted, would be an indication for the experimenter to carefully observe for signs of manifestation in this participant while running through the battery. The format of the questions allowed for the coding of degree of certainty of response (e.g., "unknown"), and, in all instances, the participant was free to abstain from answering any question that was felt to be of too personal a nature to be discussed with the research staff (despite assurance of strict confidentiality). Prevalence of color deficiencies in various racial and ethnic populations has, in the literature, been found to not necessarily coincide with the often cited "standard norms." As a result, we further queried the observers as to their genealogy (i.e., family's geographical origins), their language capabilities, and their eye, hair, and skin pigmentation. In addition, at this time, the participant was given a unique

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identification code, which was used for all subsequent data records and for maintaining anonymity when interacting with non-senior laboratory personnel.

Optical testing. Optics and acuity testing was performed by either a board-certified, licensed optometrist, or by an optometry student of advanced standing, attending the State University of New York's College of Optometry. All testing occurred in the laboratory setting. Exactly which tests were performed, and in what particular order, varied from one participant to the next. Minimally, standard refraction took place, but other tests, deemed as both appropriate and necessary for determining the observer's clinical diagnosis, may have also been run. This includes, but is not limited to, standard ophthalmological eye examination procedures such as determining the prescription of the participant's existing glasses, determining the prescription for glasses and/or corrective frames, pupillometry, fundus retinoscopy, choroidal examination, et cetera. Prescriptive values obtained for the observer for reading and distance vision were used for "best optical correction" throughout the duration of battery testing. (Principles and guidelines for becoming proficient in the art of refraction can be found in various sources, including standard ophthalmology textbooks; see, e.g., Brodie, 1994; Vaughan, Asbury, & Cook, 1971.)

Measuring the prescription of glasses with the lensometer consists of first focusing the reticle in the eyepiece, and then placing one lens of the glasses over the optical path. With the focusing dial on the plus side, the experimenter rotates the dial towards the minus side until double black lines come into focus. The display is then rotated until the black lines are continuous. Noting the preliminary dioptric value for spherical correction, three bright bars are focused at right angles to the black lines. If the

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dioptric value is the same for the black lines, there is no cylinder, and the spherical value stands. If there is a difference, the cylinder is found as follows. If the value for the three bright lines is smaller (more negative) than the value for the two black lines, the axis of the cylinder is read from the rotating dial, the value being the difference between the two readings. If the value for the three bright lines is more positive than that for the two black lines, the display is rotated 90° and the sphere, cylinder, and axis are measured and recorded. The same procedure is used for the remaining lens.

The Topcon autorefractor can also be used for refracting purposes. The participant is asked to look through the viewing aperture into the machine. The experimenter focuses the target eye and places a luminous point in the center of a collimation ring, visible on the TV monitor screen. When the luminous point is minimal and sharply focused, and the eye is properly aligned, automatic measurement occurs. Three measurements of each eye are made, and the results are printed out automatically.

For purposes of testing visual acuity, the B-VAT video acuity tester was employed. At a distance of 20 ft, the participant is asked to read lines of Snellen letters of various sizes. Initially there are three lines presented with acuities of 20/25 on top, to 20/20 in the middle, to 20/15 on the bottom. If the participant is unable to read the top line, the size of the letters is increased. A criterion of no more than one mistake is used to determine best acuity. That is, if a subject makes no more than one mistake in reading a line of letters of a given size, they are permitted to move on to the next smallest size character line. Two or more mistakes on a given line indicates that the person's acuity level is that of the previous line (assuming the above criteria have already been met). Randomly generated lines are possible at the push of a button, making the B-VAT

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superior to the standard eye chart, which has no means of preventing letter sequence memorization by the participant. This machine produces a letter size no smaller than 20/15. As a result, individuals whose acuity may be better than this will not be sufficiently determined using this procedure.

Color perception. Color detection tests, which consisted of the Dvorine plates, the Farnsworth D-15, and the Lanthony Desaturated 15 Hue Test, were administered (at a distance of 50 cm) according to the instructions provided (Dvorine, 1963; Farnsworth, 1947; Lanthony, 1974). In the cases of the D-15 and Lanthony, the colored caps were put in random order by the experimenter prior to the start of the test session to facilitate the overall time factor. The protocols are provided in Appendix A. Briefly, as mentioned earlier, the tests were run monocularly, so one eye was patched according to a predetermined randomized sequence. Observers were told to place their chin in a chin rest (Bausch & Lomb) and to arrange the colored caps in order according to color. The purpose of each test was made clear to the participant at the start of the session, and any questions were answered to the best of the experimenter's ability.

Color discrimination. For color discrimination, the Farnsworth-Munsell 100-Hue Test was run in a similar manner (Farnsworth, 1957; see Appendix A). This test was run monocularly (with eye-order randomized) only if substantially different results had been found for each eye in the previous tests of color detection (i.e., a tentative diagnosis of color-deficiency was made). The order in which the trays were presented was randomly determined in advance. The observer was made aware of the purpose of the test, and the instructions were read (see Appendix A).

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Hue- and saturation-scaling. For hue- and saturation-scaling, the participant was seated and allowed to become fully dark-adapted prior to beginning the session. The observer's head was placed in a chin rest, along with accompanying forehead rest, in order to limit any head turning during the test session.

Each trial block consisted of flashes of light taken inclusively from the twenty-four 10-nm incremental wavelengths between 430 and 660 nm. Each test flash was selected randomly, once per block. A total of four experimental blocks were run, for a total of 96 trials, with a practice block of 24 trials preceding the experimental trials.

A 250 ms beep signaled the observer to prepare for the upcoming test flash. There was a 500 ms pause preceding the stimulus presentation. The computer-controlled shutter was then opened for 500 ms, presenting the stimulus. A minimum intertrial interval of 15 s occurred between adjacent trials.

Upon presentation of the stimulus, the observer divided his sensation into hue and saturation components: hue, consisting of proportions of blue, green, red, and yellow (which must sum to 100%), and saturation, consisting of the degree of chromatic content of the stimulus (described as a percentage from 0-100%; instructions modified from J. Gordon, Abramov, and Chan, 1994; see Appendix A).

Anyone tested as positively or questionably deficient on the color screening measures ran this monocularly (with eye-order randomized), all others binocularly.

Anomaloscopy. Testing for heterochromatic flicker photometry, Rayleigh matches, and SWS cone sensitivity was run as follows (see Appendix A for complete instructions).

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HFP. Prior to beginning the test session, the optimum flicker rate at which the stimuli were to be presented to the observer was determined. Following foveal dark-adaptation, each participant was shown a stimulus, consisting of alternating green and yellow lights (square-wave function), flickered at a rate of 25 Hz. If the observer did not perceive this stimulus as flickering, the same stimulus was presented at a rate of 20 Hz. If this was not perceived as flickering, the flicker rate was dropped down to 15 Hz. The flicker rate used in the HFP experiment was the fastest rate the observer perceived as flickering (i.e., non-stable). Even if a rate of 15 Hz was reported as non-flickering, this rate was still used.

For the test session, with the yellow test LED as the standard fixed at a fraction of its maximum luminance (13.84 cd/$m^2$), the observer's task (ignoring any glare, double and/or mirror images, and aberrations) was to minimize a "red" alternating with "yellow" flicker by adjusting the luminance of the red test LEDs and a "green" alternating with "yellow" flicker (identical to the stimulus used to determine optimal flicker rate) by adjusting the luminance of the green test LEDs (see Figure 5, upper left panel). This was performed four times for each condition in a counterbalanced fashion, ABBAABBA, where A is red-yellow and B is green-yellow. The starting luminance values for the red and green test LEDs varied from slightly below that of the yellow (approximately 12 cd/$m^2$), to greatly below that of the yellow (approximately 5 cd/$m^2$), to greatly above that of the yellow (approximately 25 cd/$m^2$), to slightly above that of the yellow (approximately 18 cd/$m^2$). The resulting luminances of the red and green LEDs were stored when the observer was satisfied that the flicker was at a minimum.

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Rayleigh matching. For Rayleigh matches, the computer generated a randomized sequence of 11 numbers to be used as starting ratios of red to green light (0-100% in increments of 10%). In phase 1, the observer's task was to match this red-green color mixture to a fixed yellow. Each of these were constrained to one half of a bipartite field, with the red-green mixture half-field appearing on the left side, and the yellow half-field appearing on the right side (see Figure 5, upper right panel). The luminances of the red-green ratios were calculated using the observer's own mean HFP luminances for the red and green LEDs determined above. The luminance of the yellow test field was identical to that value used during the HFP phase of the experiment (i.e., 13.84 cd/$m^2$). The luminance of the red test LEDs was not permitted to exceed the maximum luminance value of the green LEDs (48.7 cd/$m^2$ at D to A step 255). As the observer adjusted the luminances, for any increase in percent luminance of red, there was an equal decrease in percent luminance of green (and vice versa), such that the sum of percent luminance of red and percent luminance of green always equaled 100% (unit luminance). Upon the participant finding as close a match as possible, phase 2 allowed the observer to adjust the brightness of the red-green mixture (where both red luminance and green luminance increased and decreased together, thus maintaining the red-green ratio) to more closely match that of the yellow test field. If, upon matching intensity, the hue match was disrupted, the observer had the opportunity to readjust the red-green ratio (followed by intensity, again, if necessary). The resulting luminances were stored. This was designed to determine the range of suitable matches for the participant, leading to a diagnostic of both type and degree of color vision perception.

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Chromatic adaptation. The law of additivity (Grassmann's third law) states that if two lights match in color appearance (e.g., the Rayleigh match), the addition of chromatic adapting lights will not change the equality of quantal absorptions for each photopigment, and the match should hold. J. Neitz, M. Neitz, and Jacobs (1991, 1993) found that adaptation to red light produced a small, but significant, shift in some males with normal color vision from matches obtained in the unadapted conditions. The findings in color-normal humans of polymorphism in the LWS and MWS cones, as well as polymorphism in the genes expressing these cone opsins (see, e.g., Merbs & Nathans, 1992; Nathans, Merbs, Sung, Weitz, & Wang, 1992), has led to the postulation that a number of these individuals may well have more than three spectrally different cone photopigments.

In order to assess whether certain individuals have more than three types of cones, a red adaptation beam (658 nm) at 20 cd/$m^2$ was turned on, and the observer was allowed to red adapt for approximately 2-3 minutes. Initially, it is assumed, as above, that the change in cone sensitivity which occurs as a result of this added light, will affect the LWS and MWS cones in approximately equal quantities. Thus, an individual's average red-green ratio needed to make a match should not deviate greatly from that value previously determined. Any such deviation would be indicative of at least a fourth cone type present.

HFP was done, in a manner identical to that described above, with the red adapting light on (see Figure 5, lower left panel). Next, an abbreviated form of the Rayleigh matching procedure was performed. In this instance, a randomized sequence of six numbers (representing red-green ratios from 0-100% in increments of 20%) was generated by the computer, and the matching procedure, as outlined above, was repeated (see Figure 5, lower right panel). Mean match settings that differed between non-

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adaptation and adaptation conditions indicated that at least one additional cone type was present.

(Rod and) SWS cone sensitivity. Color vision deficiencies involving the SWS cones are not sufficiently tested by either of the above methods. Therefore, a third procedure was implemented to measure SWS cone sensitivity.

Recent studies have shown macular pigment density to be a protective factor against age-related SWS cone sensitivity loss, and to be associated with clinical risk factors leading to age-related macular degeneration and other retinal diseases. Through early detection and appropriate corrective actions (such as dietary intake of carotenoid-rich foods) especially in highly vulnerable populations (including smokers, females, and people with blue irises), it may be possible to slow sensitivity loss and keep the SWS cone pathway fully functional across the lifespan (Hammond, Fuld, & Snodderly, 1996; Hammond, Wooten, & Snodderly, 1997, 1998).

In order to get an accurate measure of SWS cone sensitivity, we needed to consider other factors, such as rod intrusion, preretinal absorption, and density of macular pigment, which will absorb some of the light as it passes through to the cones. Thus, we first found absolute limens of blue (425 nm) and green (534 nm) lights against a dark background (see Figure 6, left panel) at retinal eccentricities of 2° (where macular pigment is present) and 6° (where macular pigment is mostly absent). These values were subsequently used to "correct" for SWS cone sensitivity (described below).

Following total dark adaptation, observers were instructed to fixate on a dot located either 2° or 6° above the test field. Blue (425 nm) and green (534 nm) lights were pulsed (200 ms on, 800 ms off) continuously as the observer attempted to find threshold.

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Initial luminance values, when observed through a 3.0 log unit neutral density filter, were below threshold for all observers. Adjustments were made in an ascending manner (to avoid possible adaptation). Each stimulus was presented four times in a counterbalanced fashion (ABBAABBA, where A is blue, and B is green) for a total of eight trials at each eccentricity (2° always preceded 6°), and a total of 16 trials. A practice trial preceded the first block of eight, allowing the observer to orient to the task and explore a range of possible luminances (followed by readaptation). A trial before the second block was used only to show the location of the new fixation point; no adjustment was permitted (see Appendix A for complete instructions). The luminances for each of the trials were stored by the computer.

SWS cone sensitivity consisted of measuring SWS cone increment thresholds by desensitizing the other receptor types. As mentioned earlier, this consisted of flashing a small blue monochromatic light upon a continuously presented yellow background (see Figure 6, right panel). A suitable choice of background wavelengths will depress the sensitivity of two mechanisms (LWS and MWS cones) so that the sensitivity of a third mechanism (SWS cones), less sensitive than the first two, may be revealed (Kinnear, Marre, Pokorny, Smith, & Verriest, 1979). With the continued finding of acquired blue-yellow deficits associated with various diseases (e.g., Alzheimer's; see, e.g., Cronin-Golomb, Sugiura, Corkin, & Growdon, 1993) both within and outside of the realm of vision, this type of test is an important component of the battery, considering the potentially diverse population of participants.

SWS cone sensitivity was measured following a 2-3 minute period of adaptation to a yellowish (596 nm) background. A blue (425 nm) light was flashed centrally within

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the yellow field, as above (see Figure 6, right panel). The starting luminance for the blue light was 0.001 cd/$m^2$, and observers were instructed to increase the luminance until they just detected the presence of a flash or pulse against the bright background (see Appendix A). This was repeated four times, and the luminances were stored.

For the anomaloscope sessions (HFP/Rayleigh and SWS cone), participants could only be run monocularly. Those who were color-deficient were tested on each eye, while color-normals were tested using the observer's dominant eye.

Spatio-temporal contrast sensitivity. For the contrast sensitivity experiment, participants were seated 3.6 m from the computer screen, their heads stabilized by a chin rest. Initial pairings of all combinations of spatial and temporal frequencies were presented, and the observers were required to reduce the degree of flicker, using the labeled keys on the keyboard, to the point at which they were just able to make out the orientation of the gratings (horizontal or vertical). After this adjustment phase, two separate QUEST sessions (stimuli presented in 3 interleaved blocks of 5 interleaved sequences) were run in separate sessions (to test for practice/order effects; see Appendix A) to more precisely measure contrast sensitivity thresholds. Data obtained during the adjustment phase were used as initial settings in the QUEST session.

According to Watson and Pelli (1983), individual trials are statistically independent. Prior knowledge derived from previous experiments, hunches, et cetera, which, when combined with the set of data currently being collected for the observer, forms the posterior probability density function which allows threshold estimation and placement of the next trial. Thus, for every combination of spatial and temporal

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frequency, results of previous trials are used to continuously estimate thresholds and guide further testing.

Using a two-alternative forced-choice procedure, a grating was presented on the screen for 2 s (ramp on 0.5 s, on 1 s, ramp off 0.5 s), and the task of the observer was to identify the orientation of the grating pattern (i.e., are the bars horizontal or vertical?). Feedback was immediate: a correct answer was followed by a single beep and then the next trial, whereas an incorrect response was signaled by three successive short beeps and then the next trial. The QUEST session continued until the 99% confidence interval had been reached for each spatio-temporal combination, the computer storing each threshold in turn.

For this experiment, color-deficient observers were tested monocularly (as well as binocularly, in some cases), while color-normal observers were run binocularly.

Binocular vision testing. The maddox rod test consisted of the reestimation of the participant's level of acuity (binocular alignment through horizontal and vertical fusion) via the reading of Snellen letters projected horizontally or vertically on the B-VAT monitor, through the phoroptor (with appropriate lenses dialed in), at a distance of 6 m. The white maddox rod on the phoroptor was dialed into place and the observer continued to read sets of Snellen letters, every so often being told to look away and report the location of the white horizontal (or vertical) streak of light with respect to the Snellen letters. The experimenter would turn the rotary prisms (see Appendix A), adding or subtracting prism diopters as needed, and the task was repeated. This procedure continued until the observer reported that the streak of light and the line (or column) of Snellen letters was aligned on top of one another. The amount of rotation of the prisms (i.e.,

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number of diopters) gave a direct measure of eye alignment (i.e., base in versus base out, base up versus base down).

Ocular dominance was assessed by having the participant walk up to a simulated peephole (a fixed metal tube), look inside, and read what was seen. The experimenter recorded the eye chosen by the observer for this task.

Interpupillary distance (IPD) was measured using a hand-held pupillometer. The observer fixated on a target at optical infinity, while the experimenter adjusted reference lines to cross through the pupillary corneal reflections made by the target in each eye (see Appendix A). The experimenter recorded the IPD, as calculated by the instrument.

Ocular rivalry was determined by having the observer view a pair of squares, consisting of oblique cross-hatched lines going in opposite directions, through a hand-held stereo viewer. The observer was told to report in which direction the lines appeared to slope most of the time when the squares were fused (see Appendix A). The experimenter recorded the eye corresponding to the response (in some cases it was "mixed" as the observer noted equal alternations between the two directions) as dominant, along with whether the rivalry was strong or weak.

Stereopsis. The diagnostic tests for stereopsis (i.e., TNO plates, Randot, and Titmus Fly) were administered according to the instructions provided (see Appendix A for examples) at a distance of 40 cm.

The test for stereoacuity consisted of an adjustment session immediately followed by a QUEST session. At a distance of 1.8 m, the observer, initially, was presented with four separate dynamic random dot stereograms. The first one, when viewed with stereo glasses, should have appeared to be in front of the plane of the computer screen. The

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observer's task was to move the square away from himself, using the labeled keys on the keyboard, until it was just barely in front of the plane of the screen (and still perceived as such). The second stimulus initially appeared behind the plane of the screen, and the task of the observer was to move the stimulus closer, until it was just barely behind the plane of the screen. The third stimulus, like the first, was in front of the plane of the screen, but now the observer was instructed to continue to move the square forward until he could barely tell it was in front (i.e., before it began to blur and the sensation of depth was lost). Similarly, the fourth stimulus, like the second, was located behind the plane of the screen; this time, however, the observer continued to move the stimulus further away until it was just barely discernible that the square was in back of the plane of the screen (see Appendix A). The adjustment data were used as initial settings for the QUEST session.

The QUEST session consisted of the computer presenting the observer with stimuli at various "depths" with respect to the plane of the screen (actually various degrees of displacement between the images presented to the separate eyes which, when fused, yielded the sensation of depth; see Figure 7). Using a two-alternative forced-choice paradigm, the observer was required to determine whether the stimulus was in front of or behind the plane of the screen. The stimulus remained on the screen until a response was made. A correct response was signaled by a single beep, followed by the next trial; an incorrect response was signaled by three successive beeps, followed by the next trial. Thresholds for minimum and maximum displacements for stimuli both in front of and behind the plane of the screen was determined through the QUEST procedure, terminating when the criterion (i.e., 99%) confidence intervals had been met (complete

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instructions are provided in Appendix A). The computer stored the resulting thresholds, giving the upper and lower limits of Panum's fusional zone and those of stereoacuity.

The very aspect which defines stereopsis (i.e., the ability to fuse two slightly disparate images, one presented to each eye) necessarily required that all participants, irrespective of group, be tested binocularly.

Monocular hyperacuities. Vernier acuity was measured at a distance of 1.8 m. The observer was presented with two abutting black vertical bars in the center of the computer screen (see Figure 8). The upper bar always remained fixed, while the lower bar was randomly displaced either to the left or to the right with respect to the upper bar. Each stimulus remained on the screen for 1 s. Using a two-alternative forced-choice paradigm, the observer's task was to decide whether the lower bar was left of the upper bar, or whether it was right of the upper bar. A correct response was indicated by a single beep, followed by the next trial, while an incorrect response was followed by three short beeps before the next trial was presented. The QUEST procedure found the minimum amount of displacement needed for the observer to still reliably judge the direction of displacement (see Appendix A). The initial trial presented a disparity of 0.2 min arc, after which subsequent trials' parameters were set based on previous responses by the participant. This initial value was selected arbitrarily, and is based on both numerous studies cited in the literature, as well as the accumulation of pilot data, which shows it to be an appropriate setting. If necessary, this initial value could be changed. The QUEST procedure terminated upon reaching the 99% confidence interval.

Hyperacuity tasks are quite prone to practice effects (see, e.g., McKee, Welch, Taylor, & Bowne, 1990; Westheimer & Hauske, 1975; Westheimer & Mckee, 1977a). As

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a result, all participants were run through this test twice, in separate sessions. The values obtained from the second run were used for subsequent analyses. All observers were run binocularly in this experiment.

Motion sensitivity. The experiment for motion detection was conducted at a distance of 1.8 m. Consisting only of a QUEST session, observers were presented with a square stimulus containing an array of random dots. Over a period of 200 ms, the array of dots were displaced, either up or down, by some amount, often giving rise to the perception of motion (see Figure 9). In a two-alternative forced-choice paradigm, the observer's task was to indicate the direction of movement (displacement) of the dots. A single beep followed by the next trial indicated that a correct response had been made; three short beeps followed by the next trial indicated that an incorrect response had been made. The QUEST procedure calculated both the minimum ($d_{min}$) and maximum ($d_{max}$) amount of displacement whereby the observer could still reliably discern the direction of movement to a 99% confidence interval (see Appendix A). The initial value for the first trial was set at 4.00 min arc for minimum displacement, and 10.00 min arc for maximum displacement. The parameters for subsequent trials were based on the participant's responses to previous trials. These initial values were arbitrarily chosen based on the literature, but have been extensively piloted in order to ascertain that they are, in fact, appropriate starting points.

As described above, in order to account for practice effects, all participants were run through this test on two separate occasions. Only those thresholds obtained during the second session were used for analysis. All observers were run binocularly in this experiment.

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In each of the above computer-oriented tasks, to assist the observer with identifying the keys utilized for each task, a plastic keyboard cover (Ring King Visibles, Inc., Muscatine, IA), with holes drilled out to reveal only those keys relevant to the task at hand, was employed.

Data analysis

The raw data obtained from several of the battery tests required transformations for purposes of additional data analysis.

As described in the Method section, all participants were classified into one of three groups (color-normal, acquired color-deficient, or genetic color-deficient). A tentative classification was possible, in some instances, as early as at the time of intake (though not necessarily of type and/or degree of severity), but a definitive classification was made following the completion of color perception testing.

Color perception. Scoring of the Dvorine, Farnsworth D-15, and Lanthony Desaturated 15 Hue Test was performed according to the respective manuals, the participant receiving a separate diagnosis from each. The interpretation of the graphs plotted for the Farnsworth D-15 and Lanthony Desaturated 15 Hue Test was not always straightforward. Choosing a diagnosis of "unknown" for cases showing evidence of a real deficit, but no clear axis (see Appendix A), was often a last resort. Only after several independent assessors viewed the results, and, collectively it was decided that no certain diagnosis could be made, was this classification used. Such collaborative assessment strategies were employed in other instances where, for example, an individual traversed from the axis of one type of deficit to that of another.

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While the results of these tests determined whether the participant would be run on subsequent tests binocularly or monocularly, an individual's final categorical type was based from the sum results of performances on the Dvorine, Farnsworth D-15, Lanthony Desaturated 15 Hue Test, Farnsworth-Munsell 100-Hue Test, and hue- and saturation-scaling. Often the latter two tests were used to render decisions for "questionable" observers (e.g., some participants showing only minor reversals on the arrangement tests, and whom would be considered color-normal, made these errors along a tritanopic axis, which, when confirmed by the 100-Hue test, led to a classification of acquired color-deficient).

Color discrimination. Results from the Farnsworth-Munsell 100-Hue Test were scored according to the manual (although here, too, interpretation of type and severity of deficit, may have, in some instances, been subjective). In addition to the regular scoring, a discrete Fourier transform of the error pattern was performed to assess for asymmetries. As suggested by Victor (1988), the amplitudes and phases of the first five harmonics were compared with the probabilities of getting such indices from random swaps per tray for a given total error score at the 0.95 and 0.99 levels of significance (also see Appendix A). A significant first harmonic is not indicative of a color deficiency. This harmonic reveals overall asymmetries ("bulges") which may or may not be bipolar (i.e., defining an axis). Color deficiencies are more often evident in significant higher-order harmonics. For any significant harmonic, the cap number representing the midpoint of the bulge was determined. This aids in classifying the type of deficit present, as various (and/or multiple) axes may reveal a more complex deficiency than does the traditional scoring method alone.

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A color screening categorical classification was made for each observer, based on the results of the above tests (and hue- and saturation-scaling, if necessary). Descriptors were normal, protan-like (with further subclassification regarding degree of severity [possible, moderate, or severe] and eye [left, right, both]), deutan-like (with further subclassification regarding degree of severity [possible, moderate, or severe] and eye [left, right, both]), tritan-like (with further subclassification regarding degree of severity [possible, moderate, or severe] and eye [left, right, both]), and poor discrimination (no particular category; not indicative of color-deficiency).

Hue- and saturation-scaling. Various analytic programs were used in deciphering the results of hue- and saturation-scaling. Mean hue- and saturation-scaling functions were compiled and expressed both tabularly and graphically. This included using the saturation values reported at each wavelength to reapportion the hue values, such that the sum of the hues equaled the saturation, while still preserving the ratios among hues (see J. Gordon & Abramov, 1988; see Figure 10, upper panel). The creation of a uniform appearance diagram (UAD), a color diagram with orthogonal and bipolar axes representing the dimensions of blue-yellow and red-green used to collapse the set of five curves (blue, green, red, yellow, and saturation) down to one, allowed us to extract a participant's unique hues (i.e., the wavelengths at which, for example, 100% green was observed; see Abramov & Gordon, 1994; Abramov, Gordon, & Chan, 1990; see Appendix A; see Figure 10, lower panel).

In addition, the determination of an individual's wavelength discrimination abilities helped us to spot potential color-deficient observers. Individuals requiring a large delta-lambda (to produce a just-noticeable-difference) in a particular region of the

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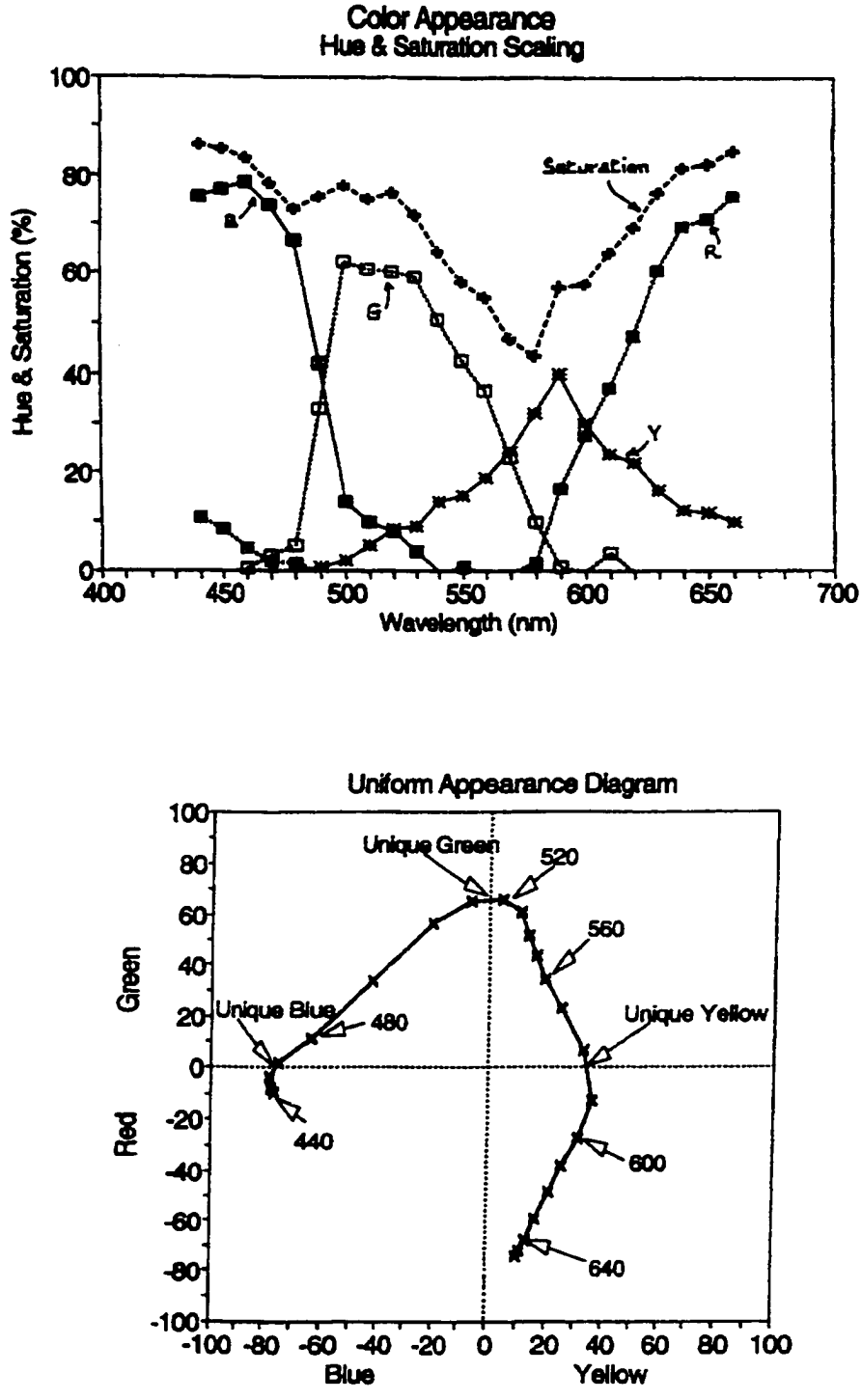


Figure 10. Typical hue- and saturation-scaling functions given by color-normal observers (upper panel), and the corresponding UAD onto which the functions translate (lower panel). Adapted from Abramov & Gordon (1994).

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spectrum to notice a change in hue indicated problem areas along the color circle. For example, a high delta-lambda value (e.g., 10) in the region of 450-460 nm is interpreted as a definite "blue weakness" and possible tritan-like deficiency. Saturation integrals, defined as the mean percentage of the summed saturations, were also calculated.

A hue- and saturation-scaling categorical label was applied to each participant, based on both the hue ratio curves and UAD. The labels (two or more of which could be concatenated, if appropriate) included no violet (no emergence of red hue in short wavelengths), tritanopic blue (blue function extending well beyond 500 nm), red delta (inflection in red curve anywhere between 610-640 nm), normal (normal scaling functions and UAD), and abnormal (abnormal scaling functions and UAD, other than those previously mentioned). A labeling other than "normal" does not necessarily imply that the observer is color-deficient.

Anomaloscopy. Transformation of the anomaloscope data included determining the mean HFP settings for green and red lights, finding each green/red luminance ratio required for an observer's Rayleigh matches, finding the range of acceptable ratios (indicative of degree of severity in color-deficient observers), finding the midpoint of the range (indicative of the type of color deficiency in anomalous trichromats), and finding the mean ratio needed for the Rayleigh match (and the variance), all under both chromatic and non-chromatic adaptation conditions, both linearly and logarithmically.

Upon finding the mean ratio needed for a Rayleigh match, an anomalous quotient (AQ) was calculated for each observer. The quotient was calculated according to Schmidt (1955), $(G_o/R_o)/(G_n/R_n)$, where $G_o/R_o$ is the (mean) luminance ratio of green to red needed by the observer to make a match, and $G_n/R_n$ is the (mean) luminance ratio of

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green to red needed by a typical normal observer to make a match. Since different laboratories use different equipment and test under different conditions, using another laboratory's set of norms for calculations is not permissible. Thus, for our purposes, the mean luminance ratio of red to green needed by a typical normal observer to make a match was the average of the mean luminances of red to green ratios for a set of 24 eyes from a group of observers classified as color-normal according to the criteria used in this study. This included a number of members of the color-normal group described earlier. The anomalous quotient is meaningful only for anomalous trichromats, for only in these cases will the mean ratio of green to red differ substantially from 1.00.

For SWS cone sensitivity (SWS cone increment threshold) data, mean absolute limens were determined for the blue (425 nm) and green (534 nm) lights at both 2° and 6° retinal eccentricities. Also, the mean SWS cone increment threshold was calculated. As mentioned earlier, the SWS cone increment threshold, as measured, was in its "uncorrected" form. This mean increment was adjusted for preretinal absorption and ocular density of the macular pigment as follows. First, relative radiances were calculated for each observer's mean absolute limens by taking the ratio of the measured mean luminances and dividing by the corresponding interpolated value on the photopic spectral sensitivity curve (V-lambda; see Wyszecki and Stiles, 1982, their Table 1[4.3.2]) for wavelengths of 425 and 534 nm at each eccentricity. From this, we derived log relative sensitivities for each wavelength. The difference in log relative sensitivities between 425 nm and 534 nm at 2° eccentricity, as measured psychophysically, when compared to the predicted difference from the absorption spectrum of human rhodopsin, yielded the difference in log density due to preretinal absorption including macular pigmentation. For

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the rhodopsin pigment values, we used an absorption spectrum for human rhodopsin from Wald and Brown (1958; their Figure 2, curve A) normalized to an optical density of 0.3. The data from Wald and Brown (1958) were chosen after an extensive review of the literature. Their measurements were made from human retinas, and the number of measurements over this spectral range was larger than in other studies. The normalized curve, when weighted with a 0.3 optical density, most closely fit that of V'-lambda (scotopic luminous efficiency function), thus giving the best estimate of preretinal and macular factors. A similar comparison, when made using the difference in log relative sensitivities between blue and green at 6° eccentricity gave the difference in log density due to preretinal absorption without macular pigmentation. Each observer's log mean relative radiance needed for SWS cone increment threshold was calculated in a manner identical to that used for absolute limens (again using the interpolated V-lambda value for 425 nm; see above). The resulting value was then "corrected" by subtracting from it the previously determined difference in log relative sensitivities at 2° eccentricity, as it is assumed that there are only negligible differences in absorption at this eccentricity and foveally, where the SWS cone thresholds were measured.

Spatio-temporal contrast sensitivity. CSF data were analyzed using the PSI-Plot curve-fitting program (Poly Software International, Sandy, UT), which allowed us to plot a three-dimensional graph representing an observer's entire spatio-temporal surface based on the mean sensitivities for each spatial and temporal frequency pair. Estimates of performance were based on the observer's sensitivity at the lowest spatial frequency (i.e., 0.6 cpd) for each temporal frequency, the sensitivity at the peak of the contrast sensitivity curve for each temporal frequency, and the spatial frequency at which the peak occurred.

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In addition, mean contrast sensitivity function curves were computed for comparison across groups and across observers, as were ratios of low-to-peak sensitivities (i.e., the contrast sensitivity at 0.6 cpd/maximum contrast sensitivity regardless of spatial frequency) for each temporal frequency.

Binocular vision testing. Binocular vision was categorized for each participant based on measures of fusion and stereo-screening. Observers were rated as having poor or problematic fusion if the value obtained for horizontal fusion exceeded five prism diopters. Stereopsis was decidedly present (i.e., in at least an elementary form) provided that the observer was able pass the most basic of screening plates among the TNO, Randot, and Titmus Fly tests. Thus, the following designations were possible: good fusion with stereopsis, good fusion without stereopsis, poor fusion with stereopsis, and poor fusion without stereopsis.

Ocular and dermal spectral reflectivity. Spectral scans obtained for each participant's iris and skin reflectance were converted from individual point measures of spectral radiance, to summed reflectances across the spectrum.

Stereopsis, monocular hyperacuities, and motion sensitivity. Analyses of the data from the stereopsis, hyperacuities, and motion sensitivity tests did not require any further manipulations beyond that which was already done by the VisionWorks QUEST procedure (e.g., calculation of measures of central tendency, variability, etc.).

All statistical analyses were performed using SPSS (version 9; SPSS, Inc., Chicago, IL), in conjunction with analytic spreadsheets (Microsoft Excel, Office 97 version, Microsoft Corporation, Redmond, WA; Quattro-Pro, version 4, Novell, Inc., San Jose, CA).

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## Results

General overview. Statistically speaking, it is difficult to analyze data from unequal samples. Given that certain individuals in our study had produced twice as much data as others (i.e., color-deficient observers who were run monocularly, giving two sets of data for a number of tests), we faced a dilemma. On the one hand, the data were quite valuable, as potential differences between an observer's two eyes could exist (e.g., in acquired color-deficients; see above). However, to include two sets of data from a given participant would confound the statistical results, as not all data would be from independent sources, and to place, for example, a non-color-deficient eye in the same grouping as a color-deficient eye on any measure could potentially end up skewing the group data. This may lead to suppression of a real difference between groups, or, likewise, present a difference that is actually false.

Since one of the main goals of this study was, in fact, to determine whether such differences between groups existed (within the visual functions addressed), it was decided that, for monocularly run observers, the data obtained from the poorer eye would be used in the analysis. The labeling of poorer eye was based, collectively, on the results of the color-screening measures (especially the Farnsworth-Munsell 100-Hue error score) and those from hue- and saturation-scaling. This was felt to be appropriate given that, in individuals with a color-deficiency restricted to one eye, they are inevitably affected by the differences in the two eyes (to some extent) in their daily environment and must somehow compensate. The chance of major differences between the two eyes in genetic color-deficient observers is slight. The above line of reasoning would similarly apply if this were not the case.

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If binocular data were obtained for all participants (in addition to the monocular data for acquired and genetic color–deficient observers), as was the case, for example, in contrast sensitivity testing, the binocular data were analyzed in an attempt to better equate the groups.

It should be mentioned that, post hoc, we found that two participants who had initially been classified as acquired color-deficient (and run monocularly) were, in fact, color-normal. The same criteria, as described above, were applied to these observers' data sets. One of these individuals, due to time constraints, was unable to be tested binocularly in the contrast sensitivity experiment. His data are, therefore, not included in any of the analyses run for this test.

With the intention of determining whether differences existed between groups among the functions measured (and possibly, if subserved by the same visual pathways, which functions clustered together), we initially performed a cluster analysis. However, based on preliminary results from this cluster analysis and correlation matrices, the overall sample size was deemed too small to yield any meaningful values.

Considering that this study is more of an exploratory investigation, with little in the way of preconceived notions or hypotheses set forth, very few data points were excluded from analysis, even if, by inspection, they appeared to be extreme values or outliers. Such differences may be real among individuals as well as groups. (Data points which were omitted included dummy values generated by VisionWorks for unstable thresholds, resulting in empty cells in the data set for those individuals, as well as other decidedly "impossible" values.) With this in mind, group medians were calculated for each measure, in addition to the standard means and measures of variability.

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Throughout this section, for the purpose of maintaining clarity, results, where applicable, are presented tabularly in full and graphically only in terms of group means for each of the battery tests. Graphs depicting group medians, as well as more detailed descriptions of inter- and intragroup variability on each measure, may be found in Appendix B.

Color perception. To reiterate, our sample of 40 participants used in this study was further subdivided into three color categories, based on the performance of individual observers on the various color-screening tests. Using these criteria, 28 were classified as color-normal (comprising what, hereafter, will be referred to as the "normal" group), 7 were termed acquired color-deficient ("acquired" group), and 5 were diagnosed as being genetic color-deficient ("genetic" group). All five members of the genetic group, during the intake, indicated some sort of familial history of color vision problems. The mode of acquisition of color deficiencies in the acquired group could be one of many, though purely speculative in most instances. An exception is one observer who was confirmed as having previously had Leber's idiopathic stellate neuroretinitis in one eye.

Results on the Dvorine test showed all normals as passing, with no more than two minor errors, none of which were made on plates 6 or 7 (diagnostic plates; see Appendix A). Acquireds fared just as well, with only one observer requiring a diagnosis other than "normal," that being of a borderline, unknown variety, in one of his two eyes. No genetic color-deficient passed this test with either eye. One observer was rated as being a severe deuteranope in both eyes, one a severe deuteranope in one eye and severe unknown in the other, and all others as moderate (n = 1, both eyes) or severe (n = 2, both eyes) unknown.

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The Farnsworth D-15 produced one or more minor reversals in four of the color-normals. However, the number of reversals was in no case found to be excessive, nor were any reversals found to be parallel to a deficiency axis. Five of the 7 acquired observers passed the test with each eye, while two showed errors along a tritanopic axis in one of their two eyes. The genetic group diverged in diagnosis, with two members being classified as deutan-like in each eye (in one case, confirming the diagnosis of deuteranope from the Dvorine), while the others showed signs of protan-like deficiencies in both of their eyes.

The data from the Lanthony Desaturated 15 Hue Test, also, showed, at most, only several minor reversals for each of the participants in the normal group. Previous diagnostic classes from the D-15 were retained for each of the acquireds. However, in addition to confirming the existence of tritan-like deficiencies in one eye in each of two members, one of these observers was diagnosed as tritan-like in his other eye as well. The two observers in the genetic group who had been classified as deuteranopes by the D-15 were not by the Lanthony test. One was termed as unknown (no clear axis, both eyes), and the other presented as protan-like (both eyes, although "unknown" may be more appropriate, especially in the right eye). The three protanopic individuals maintained their distinction. (Note that in three of the four color-normals showing reversals, one or more reversals were found which questionably parallel the tritanopic axis. The decision to pursue such observers' color vision in greater depth [i.e., run them monocularly and classify them as "acquired"] occurred after these individuals had completed the battery. Thus, the potential to have had fewer "normal" observers [and more "acquired"] does

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exist. Similarly, one or more acquired color-deficient observers could have been included in the color-normal group had their testing been done earlier.)

Color discrimination. Further clarification that we are dealing with three distinct groups of individuals comes from the comparison of errors and total error scores made on the Farnsworth-Munsell 100-Hue Test. The error scores of the genetic group are considerably higher compared to the other groups, as is seen in Table 1 and displayed graphically in Figure 11. (A box-and-whisker plot [representing values between the 25$^{th}$ and 75$^{th}$ percentiles, as well as high and low values, and the median] is presented in Figure B1 in Appendix B.)

Table 1. Color Discrimination: Farnsworth-Munsell 100-Hue Test Error Scores

| Group | Normal | Acquired | Genetic |
|---|---|---|---|
| Mean Farnsworth-Munsell 100-Hue Error Score | 21.29 | 95.86 | 152.60 |
| SEM | 2.71 | 45.71 | 17.91 |
| Median | 20.00 | 55.00 | 144.00 |

The Fourier analysis revealed two normal observers as having a significant first harmonic index. Two others had small errors evenly distributed around the color space, possibly indicating generally poor color discrimination. Four observers in the acquired group had significant harmonic indices: two were each significant on the first harmonic in one eye; one had significant first, second, and third harmonics in his tritanomalous eye, and a significant second harmonic in his non-tritanomalous eye; and one had significant

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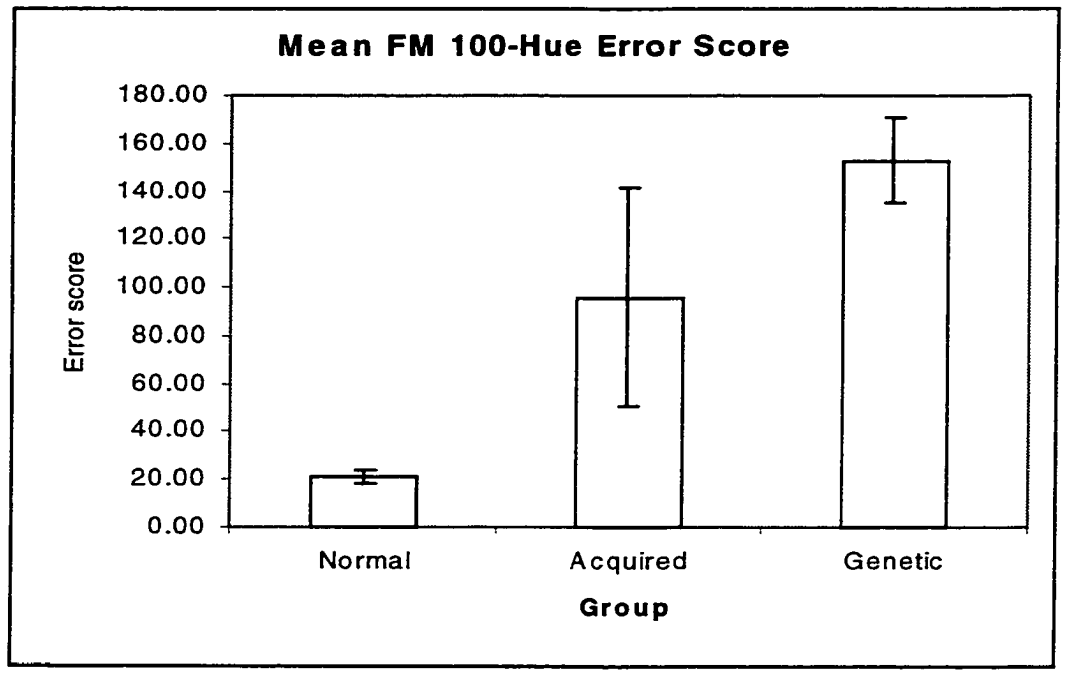


Figure 11. Group mean FM 100-Hue error score. Error bars equal +/- 1 SEM.

first and second harmonics in his tritanomalous eye, and a significant third harmonic in the other (questionably tritanomalous) eye. The data revealed tritanopic axes (one severe, one borderline) in the two members diagnosed as such by the D-15 and Lanthony test (though the second eye of the individual classified as tritan-like only by the Lanthony showed no clear axis in this test). Another two observers could be called generally poor discriminators based on the arrangement of errors. In the genetic group, each member had at least one significant harmonic in at least one of his eyes. Three observers had significant indices in both eyes: one on harmonics 2 and 4 in one eye, harmonic 2 in the other; one was significant on harmonics 2, 3, and 4 in one eye, harmonic 2 in the other; and one was significant in harmonics 2 and 4 in one eye, harmonic 4 in the other. The two other participants were significant in only one eye, at harmonic 2 and harmonics 2 and 3,

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respectively. Diagnostically, one observer, who was classified as either deuteranopic or unknown using the other color-screening tests, was protan-like in both eyes (one severe, one moderate). Another, who had been given contrasting labels by the D-15 and Lanthony tests, was diagnosed as deutan-like in one eye and unknown in the other (moderate in both cases). One member, previously classified as protanopic on each of the above tests, was typed as normal in one eye and moderately protan-like in the other. The remaining individuals retained protanopic status, with three of the four eyes showing moderate signs, the fourth was more severe.

A general color categorization was made for each observer based on the collective results from the above tests. Of the 28 color-normal observers, 27 were normal with regards to their color screening, and one was classified as having poor discrimination of no particular category. Each of the seven acquired color-deficient individuals were (in some cases with the aid of hue-scaling; see below) found to be tritan-like in at least one eye. In five instances, this diagnosis was "possible" (i.e., falling into the questionable to mild in degree range), in one case moderate, and in the last, severe. The genetic color-deficient group's classification revealed three observers showing deutan-like patterns and two showing protan-like results, manifested in both eyes, and each to a moderate degree.

Hue- and saturation-scaling. Representative samples of hue functions and UADs from each group are shown in Figures 12-14. (Refer back to Figure 10 for the interpretation of graphs and identification of unique hue points.) Color-normals' and acquired color-deficients' scaling functions show strong, independent hue functions across the spectrum, with no overlap between red and green, nor between yellow and blue (see Figures 12 and 13). The only differences are seen in a number of acquireds lacking

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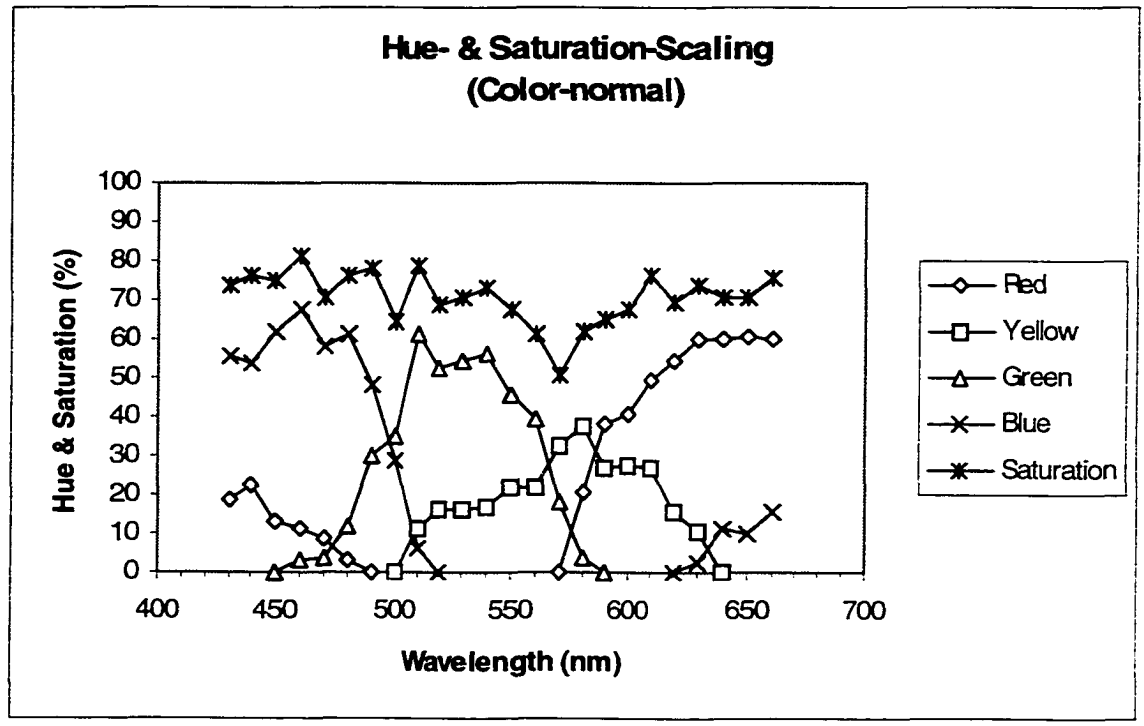


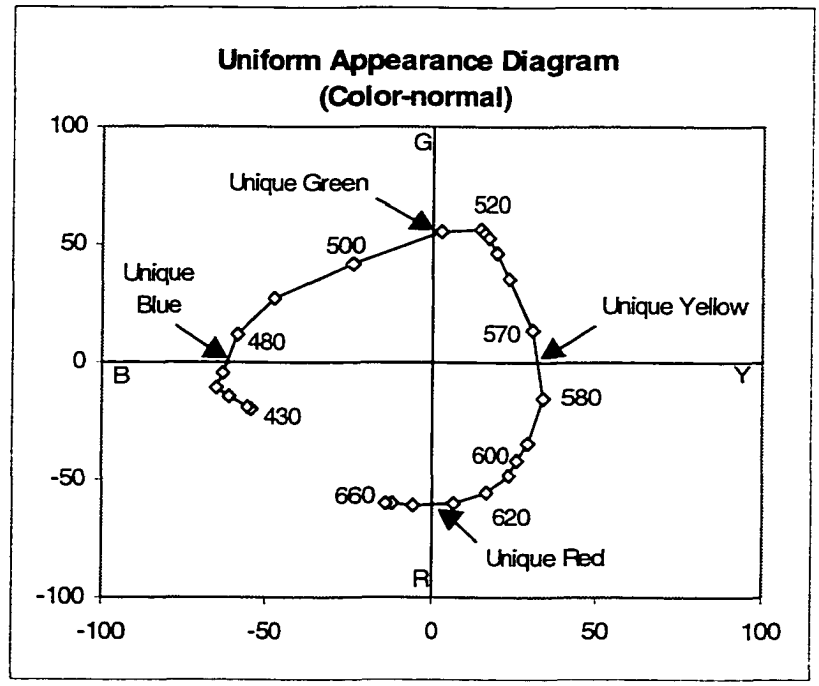


Figure 12. Mean hue- and saturation-scaling functions (upper panel) and UAD (lower panel) from a color-normal observer. This individual's scaling functions are entirely normal. Unique hues occur at 472 (blue), 509 (green), 574 (yellow), and 635 (red) nm.

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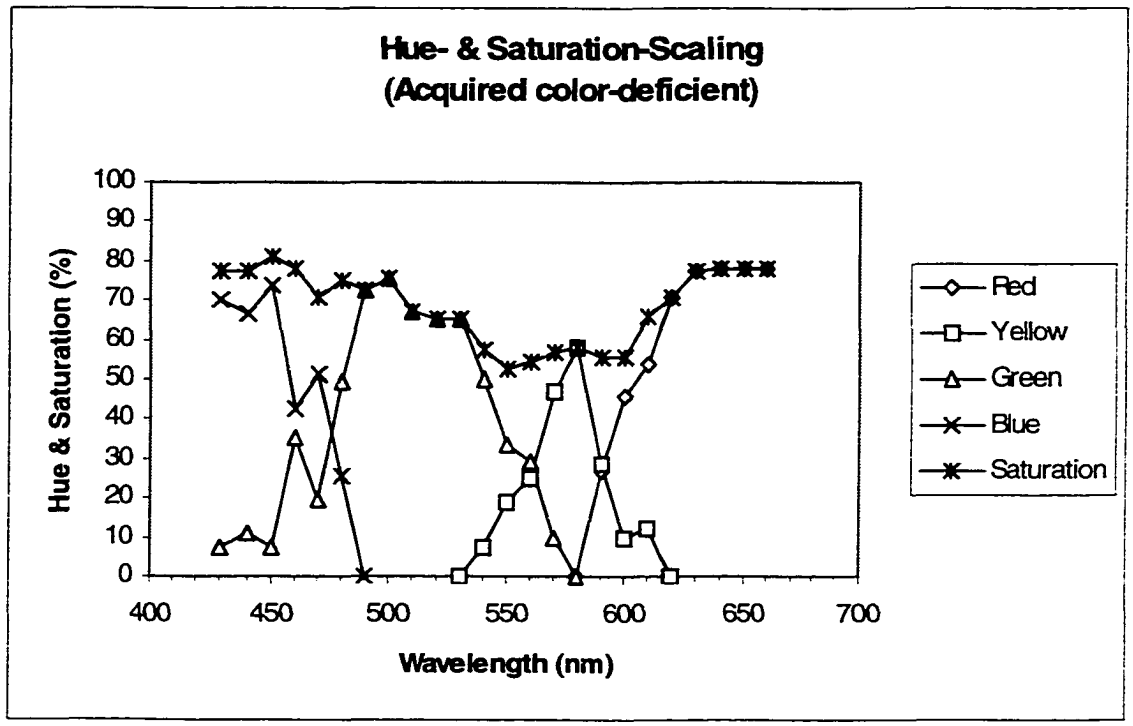


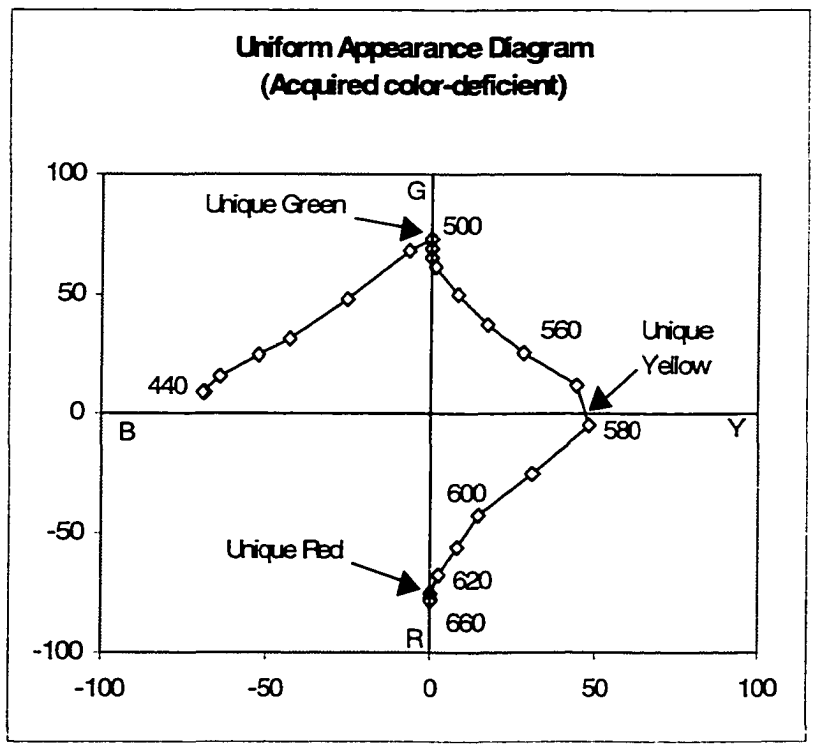


Figure 13. Mean hue- and saturation-scaling functions (upper panel) and UAD (lower panel) from an acquired color-deficient observer. This individual's hue-scaling functions show no violet, a definite red delta shift, and, while not apparent from this test, tritanopic blue. These differences, alone, do not constitute color-deficient vision. Unique hues occur at 500 (green), 578 (yellow), and 630 (red) nm. There is no unique blue.

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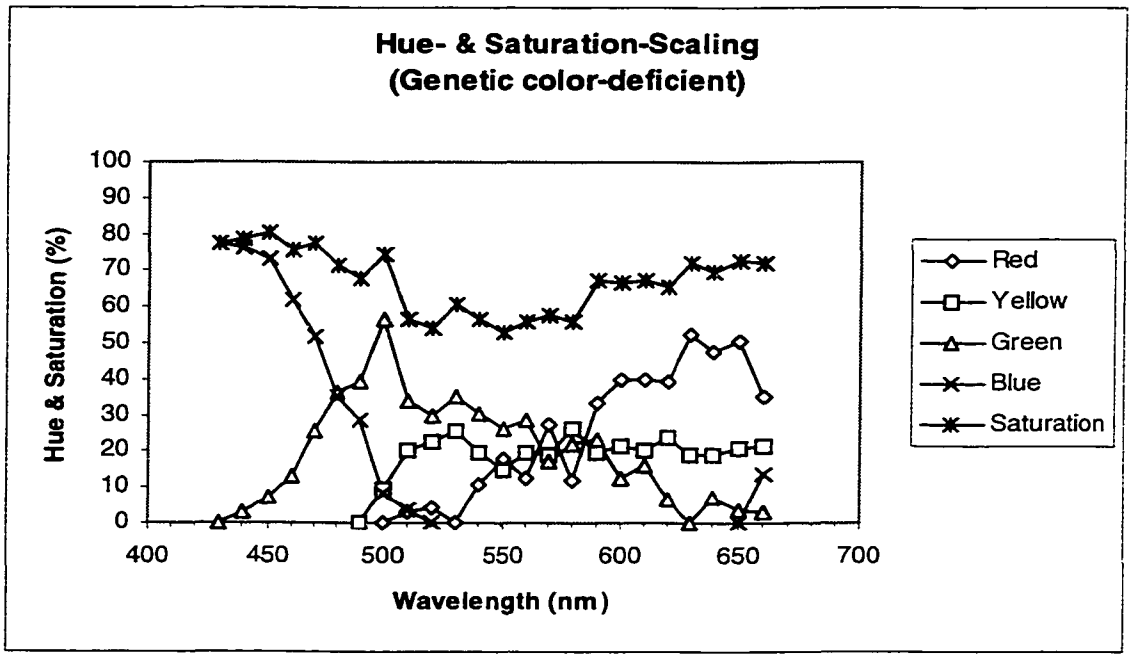


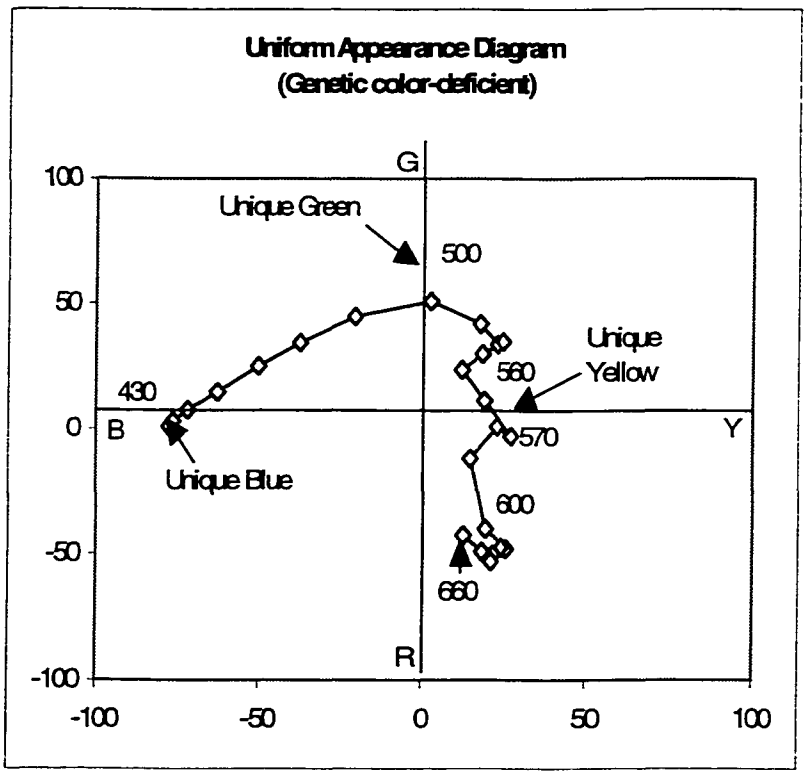


Figure 14. Mean hue- and saturation-scaling functions (upper panel) and UAD (lower panel) from a genetic color-deficient observer. This individual's hue-scaling functions are abnormal. Unique hues occur at 430 (blue), 499 (green), and 566 (yellow) nm. There is no unique red.

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red in the short wavelength region, yielding no sensation of violet. Genetic color-deficients, on the other hand, show abnormal hue functions with considerable overlap, especially among the green and red functions (i.e., generally for wavelengths greater than about 500 nm; see Figure 14). The observer's weakness in red-green color vision is quite apparent, as revealed by a highly contorted UAD in the middle to long wavelengths.

The subtle differences between normals and acquireds among their hue-scaling functions become greater when viewed in conjunction with mean delta-lambdas for the region of the spectrum between 450-460 nm (see Table 2 and Figure 15; median values are displayed in Figure B2 in Appendix B). The lack of violet (mentioned above), for example, in part translates into acquired color-deficient observers, on average, needing a much larger step size in order to discriminate hues in the tritanopic zone compared to color-normals and genetic color-deficients. The latter two groups were almost identical in mean delta-lambdas, the genetics being, perhaps, a bit more precise (see Figure 15). The combination of the above measures thus allows for the identification of three largely separate groups of observers.

From the hue- and satuaration-scaling data, we were also able to derive mean unique hues for each group. While differences between groups were found, there was no gain in our ability to diagnose or classify individuals based on these measures, which are fully described in Appendix B (see Table B1 and Figures B3-B10). Similarly, little difference was found among group mean saturation integrals; the reader is referred to Appendix B (see Table B1 and Figures B11 and B12) for these values as well.

Inspection of each observer's hue ratios and UAD led to a subjective hue-scaling categorization. This could aid in clarifying or confirming an individual's color perception

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Table 2. Hue- and Saturation-Scaling: Delta-lambda (in nm)

| Group | Normal | Acquired | Genetic |
|---|---|---|---|
| Mean delta-lambda 450-460 nm | 5.55 | 8.89 | 5.75 |
| SEM | 0.66 | 1.49 | 1.79 |
| Median | 4.13 | 9.75 | 4.00 |

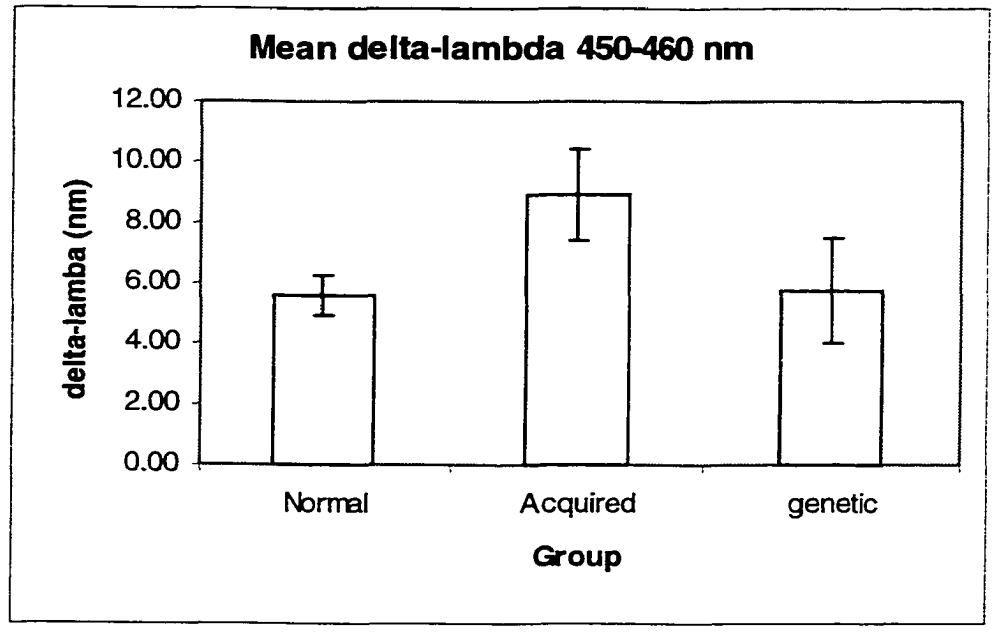


Figure 15. Group mean delta-lambda in the spectral region from 450-460 nm. Error bars equal +/- 1 SEM.

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and discrimination abilities determined from the plate and arrangement tests. In the color-normal group, 7 observers were found to have entirely normal scaling functions and UADs, 2 were found to be solely lacking violet, 7 showed only a tritanopic blue problem, 5 deviated only by red delta, 6 showed a combined tritanopic blue and red delta shift, and 1 presented both no violet and red delta. The acquired group had 1 individual with normal functions, 2 with simply no violet, 1 with red delta alone, 1 with tritanopic blue plus red delta, 1 with no violet and red delta, and 1 with no violet, tritanopic blue, and red delta. The genetics all showed abnormal scaling functions and UADs.

Anomaloscopy: HFP. The group data for heterochromatic flicker photometry are shown in Table 3 and in Figures 16 and 17 (see Figures B13 and B14 in Appendix B for medians). Only 22 of the 28 observers in the color-normal group were run through this portion of the experiment.

Table 3. Anomaloscopy: HFP Luminance Ratios

| Group | Normal | Acquired | Genetic |
|---|---|---|---|
| Mean Y/R luminance ratio for HFP | 0.68 | 0.69 | 0.56 |
| SEM | 0.05 | 0.04 | 0.17 |
| Median | 0.63 | 0.72 | 0.30 |
| Mean Y/G luminance ratio for HFP | 0.86 | 0.73 | 0.97 |
| SEM | 0.05 | 0.01 | 0.15 |
| Median | 0.81 | 0.72 | 1.05 |

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The mean luminance ratio of yellow (peak wavelength = 596 nm) to red (peak wavelength = 662 nm) light needed to minimize flicker was greatest for acquired color-deficients, followed closely by color-normals, and was least for genetic color-deficients (see Figure 16). This trend reversed when measuring the mean luminance ratio of yellow to green (peak wavelength = 534 nm) light needed to minimize flicker. Here, the genetics were lowest, followed (distantly) by normals and acquireds (see Figure 17). Among genetic observers, there was a clear bimodality not evident among members in either the color-normal or acquired color-deficient groups.

Among the genetic observers, three individuals needed considerably more red light than did members of both the normal and acquired groups. The mean Y/R luminance ratio of 0.29 points to these participants as having reduced sensitivity to this part of the spectrum, characteristic of protanopic deficiencies. The other two genetics needed nearly equal quantities of yellow and red (mean ratio = 0.96), which was less red than the average color-normal or acquired color-deficient required, yet higher individual mean ratios were recorded among some color-normals. These same two who needed less red, needed more green light, as evidenced by a mean Y/G luminance ratio of 0.62. This is characteristic of a "green weakness," much like that found in deuteranopic deficiencies. Those showing the red weakness described above needed less green than the aforementioned group members, as well as color-normal and acquired color-deficient observers (mean ratio = 1.21), with one color-normal surpassing them all.

HFP data represent points along an observer's photopic sensitivity function. Sensitivity loss in certain regions of the spectrum in some individuals has led to this technique being an additional diagnostic measure of color vision deficiencies. Figure 18

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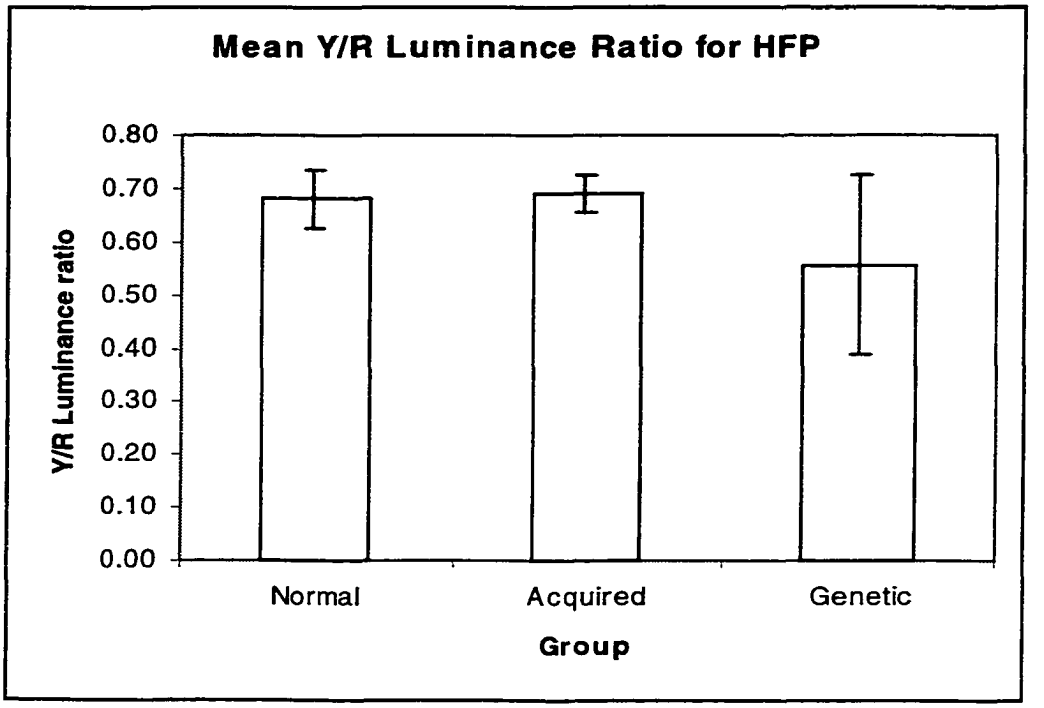


Figure 16. Group mean yellow to red luminance ratio needed to minimize a yellow alternating with red flicker. Error bars equal +/- 1 SEM.

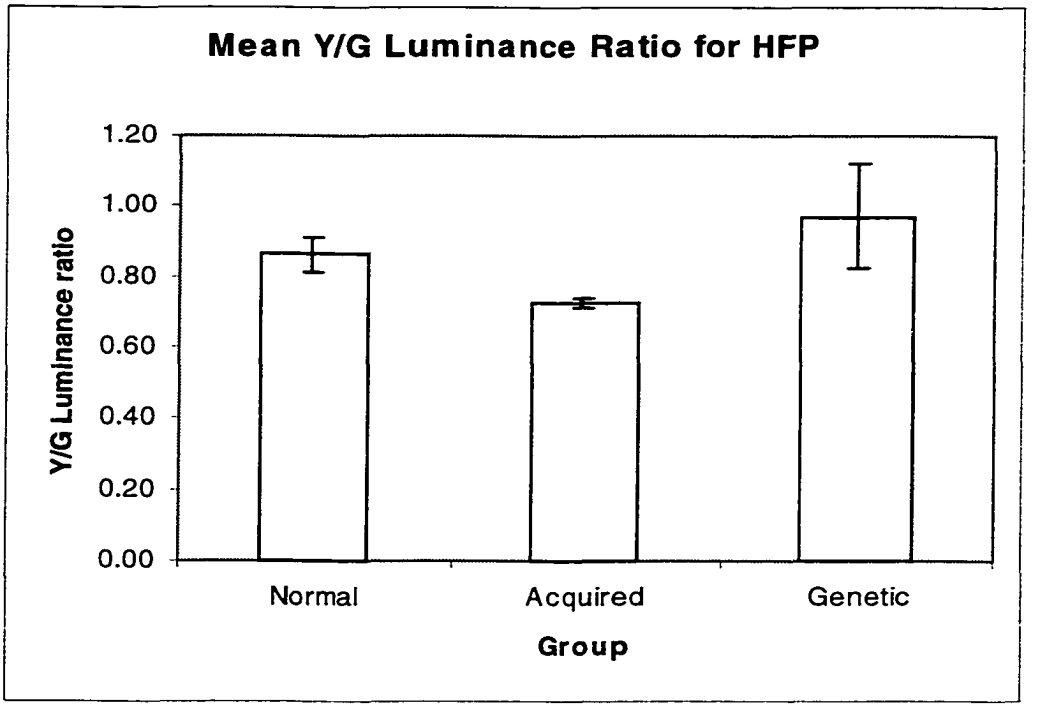


Figure 17. Group mean yellow to green luminance ratio needed to minimize a yellow alternating with green flicker. Error bars equal +/- 1 SEM.

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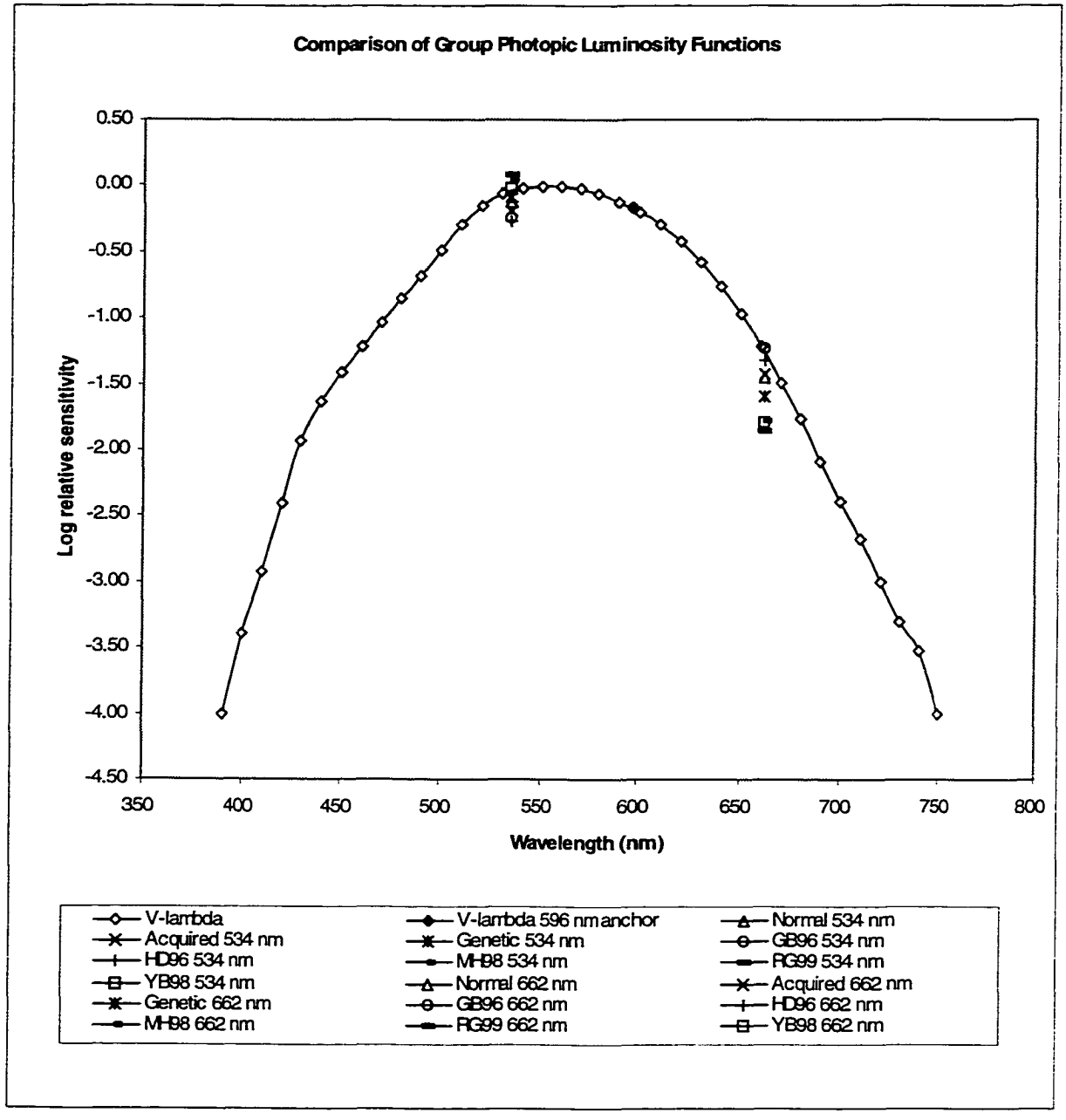


Figure 18. Comparison of group photopic luminosity functions. Group means have been adjusted to allow for the luminance of the yellow test LED to coincide with V-lambda at 596 nm. Genetic observers are also plotted separately to show regional sensitivity losses.

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reproduces the group mean HFP luminance ratios plotted along with V-lambda (photopic luminous efficiency function). Genetic color-deficients are also plotted individually for reasons mentioned above. From the graph, it is clear that observers GB96 and HD96 experience sensitivity losses in the short to middle wavelength region of the spectrum (paralleling that seen in deuteranopes), while observers MH98, RG99, and YB98 experience sensitivity losses in the middle to long wavelength region of the spectrum (paralleling that seen in protanopes). This greatly affects the genetic group means at each point. (The cross-over point [not shown] occurs at approximately 585 nm for both the deutan and protan types of deficiencies.)

Anomaloscopy: Rayleigh matching. The average mean luminance ratio of green to red light needed to match a standard yellow half-field (Rayleigh match) is presented in Table 4, and is shown in Figure 19 (medians are plotted in Figure B15 in Appendix B). The same 22 color-normal observers who participated in HFP completed this portion of the experiment.

Color-normal and acquired color-deficient observers, on average, required 3 parts green to 4 parts red to match the standard yellow. Genetics, however, needed almost 2 parts green to 1 part red. This difference between groups is entirely attributable to one genetic color-deficient observer needing a 9 to 1 green to red ratio. In fact, each of the other members of the group needed considerably more red than green compared to normals and acquireds, including the second genetic who was found to be green-weak in the HFP session (see above). This can be seen clearly in Figure B15.

The average range of acceptable green to red match ratios, sometimes indicative of the severity of the deficit in question (with a wider range denoting a more severe

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Table 4. Anomaloscopy: Raleigh Matching

| Group | Normal | Acquired | Genetic |
|---|---|---|---|
| Average mean G/R luminance ratio for Rayleigh matching | 0.75 | 0.78 | 1.96 |
| SEM | 0.03 | 0.10 | 1.77 |
| Median | 0.74 | 0.73 | 0.19 |
| Mean range of acceptable ratios for Rayleigh matching | 0.30 | 0.20 | 3.66 |
| SEM | 0.04 | 0.03 | 3.07 |
| Median | 0.23 | 0.17 | 0.47 |
| Mean range midpoint | 0.77 | 0.80 | 2.41 |
| SEM | 0.03 | 0.10 | 2.14 |
| Median | 0.76 | 0.70 | 0.27 |
| Mean Anomalous Quotient | 1.00 | 1.03 | 2.59 |
| SEM | 0.04 | 0.14 | 2.34 |
| Median | 0.98 | 0.96 | 0.25 |

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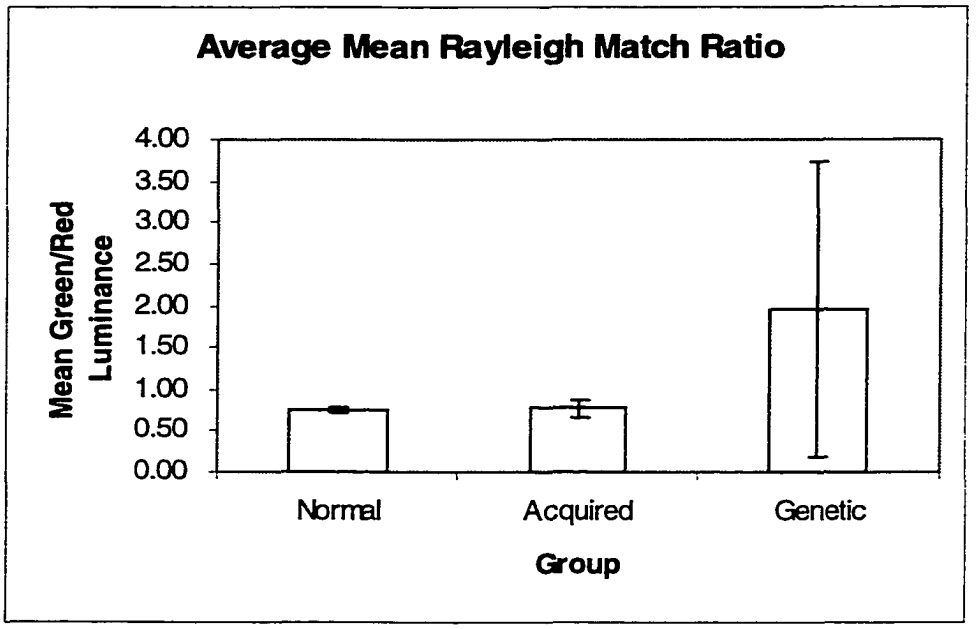


Figure 19. Average mean green to red luminance ratio required by each group to make a Rayleigh match (match to a standard yellow). Error bars equal +/- 1 SEM.

deficiency), was much broader for the genetic color-deficient group compared to the color-normal and acquired color-deficient groups (see Figure 20; medians are shown in Figure B16 in Appendix B). A one-way ANOVA run on this variable was found to be significant, $F(2, 31) = 4.01$, $p < 0.05$.

The mean of the midpoints for the range of acceptable matches, which when coupled with the range yields a comprehensive diagnosis (i.e., both type and severity of deficiency), was considerably higher among genetics than among normals and acquireds (see Figure 21; medians are presented in Figure B17 in Appendix B). This is due to the genetic observer, mentioned above, needing the large green to red ratio in order to make a satisfactory match.

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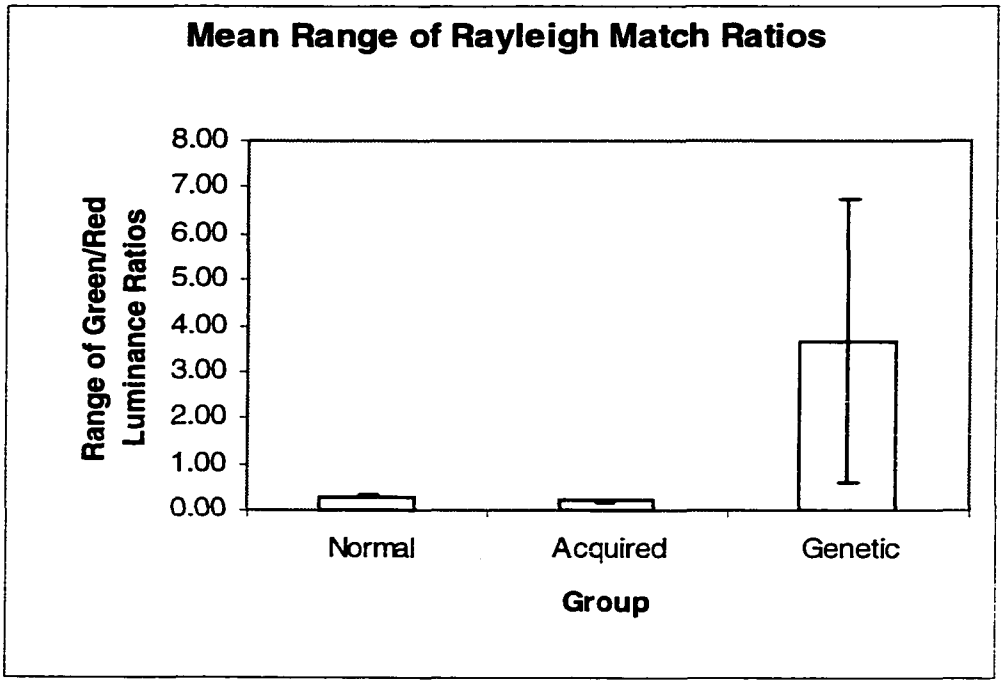


Figure 20. Mean range of green to red luminance ratios accepted as Rayleigh matches for each group. Error bars equal +/- 1 SEM.

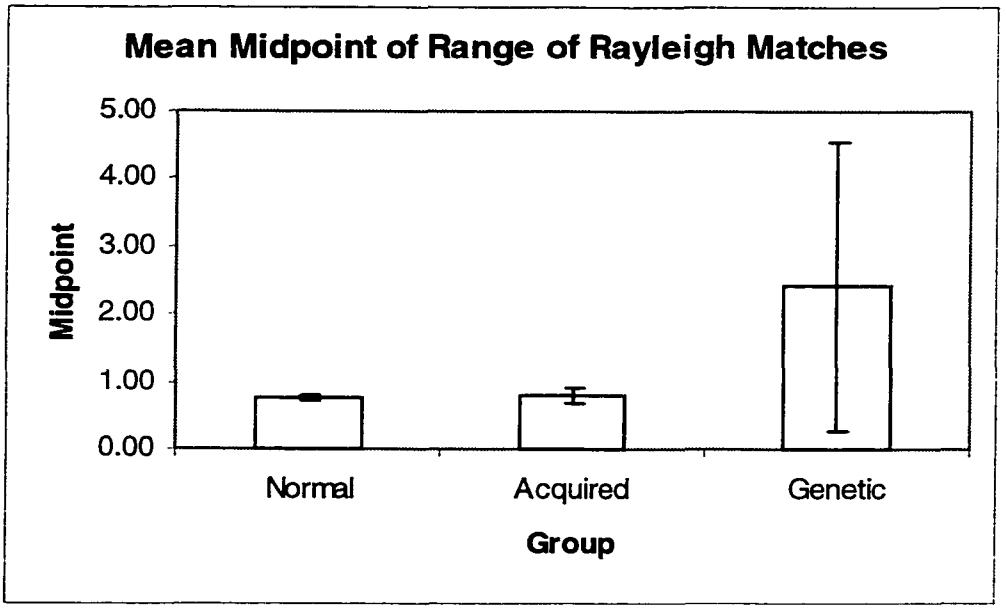


Figure 21. Mean midpoint of green to red luminance ratios accepted as Rayleigh matches for each group. Error bars equal +/- 1 SEM.

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The mean anomalous quotients computed for the groups of color-normals and acquired color-deficients were right around 1.00, while, for the genetic color-deficients, the mean was much higher (see Table 4 and Figure 22; see Figure B18 in Appendix B for medians). Based on the Rayleigh match ranges and midpoints, and the anomalous quotients, one member of the genetic color-deficient group was diagnosed as deuteranopic (average G/R ratio = 9.01, G/R range midpoint = 10.95, AQ = 11.94), and the other four were protanopic (mean average G/R ratio = 0.19, mean G/R range midpoint = 0.28, mean AQ = 0.26 [with three of the four individual AQs ≤ 0.25]), at least one severely so (see Discussion).

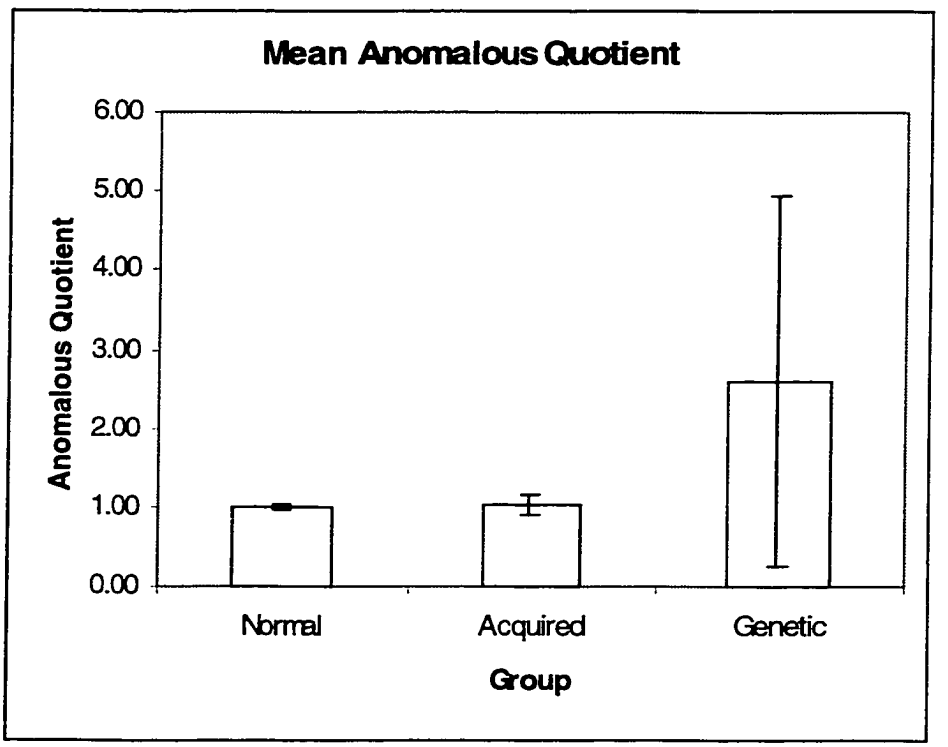


Figure 22. Group mean anomalous quotient. Error bars equal +/- 1 SEM.

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Similarly, each of the aforementioned variables was determined for the chromatic adaptation condition. The results are presented in Tables 5 and 6, and in Figures 22-28 (medians in Figures B19-B24 in Appendix B). These values represent data from 21 of the color-normals, all 7 of the acquired color-deficients, and 3 genetic color-deficients (whom were all protanopic using the above diagnoses). A computer malfunction, occurring during one of the Rayleigh match trials, required that the data from one of the color-normal observers be calculated from 5 (instead of 6) matches. In addition, one of the genetic group members, while having been successfully tested, had, in one instance, matched pure green to the yellow standard, thus giving a green to red ratio of infinity on that trial. As a result, no averages or ranges could be computed for this individual, and his data were excluded from the analysis.

Table 5. Anomaloscopy: Chromatic Adaptation: HFP Luminance Ratios

| Group | Normal | Acquired | Genetic |
|---|---|---|---|
| Mean Y/R luminance ratio for Red Adapted HFP | 0.66 | 0.86 | 0.59 |
| SEM | 0.05 | 0.12 | 0.23 |
| Median | 0.68 | 1.00 | 0.39 |
| Mean Y/G luminance ratio for Red Adapted HFP | 1.33 | 1.14 | 1.50 |
| SEM | 0.14 | 0.07 | 0.33 |
| Median | 1.18 | 1.14 | 1.38 |

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The mean luminance ratio of yellow to red light needed to minimize flicker in the presence of a red (peak wavelength = 658 nm) flooding beam was greatest for the acquired color-deficient group, and least for the genetic color-deficient group (see Table 5 and Figure 23). The mean luminance ratio of yellow to green light needed to minimize flicker under this condition was highest for the genetics and lowest for the acquireds, although the normals and acquireds performed nearly identically in this task (see Table 5 and Figure 24).

Rayleigh match ratios under chromatic adaptation produced an average green to red luminance ratio that was largest in acquireds and smallest among normals (see Table 6). The great difference between the acquired and genetic groups could be traced to one acquired observer requiring a more than 100-fold greater amount of green to red (see Figure 25). The range of acceptable matches was also greatest for the acquired color-deficient group by virtue of the same reasons as above. Genetic color-deficients had a mean range one-third this size, and color-normals were quite consistent from trial to trial (see Figure 26). The mean of the midpoints for these match ranges followed this same general pattern (see Figure 27).

For uniformity, anomalous quotients were calculated for the adaptation condition, as well. The mean AQ for color-normals was, again, found to be 1.00. This time, though, acquired color-deficients were highest, due largely to the extraneous value from the observer mentioned above. (The acquired group's mean AQ without this observer was 1.08.) The genetic color-deficient group was also found to have a high mean as a result of a similar bimodality (i.e., an individual diagnosed as protanopic in the unadapted condition was showing deuteranopic tendencies in this task; see Table 6 and Figure 28).

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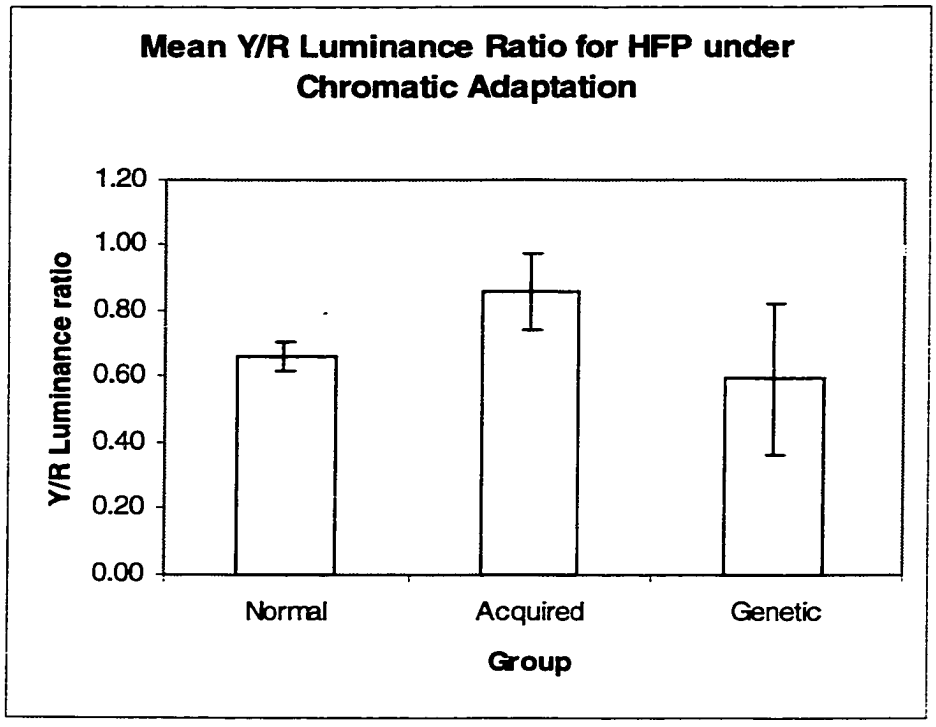


Figure 23. Group mean yellow to red luminance ratio needed to minimize a yellow alternating with red flicker under chromatic adaptation. Error bars equal +/- 1 SEM.

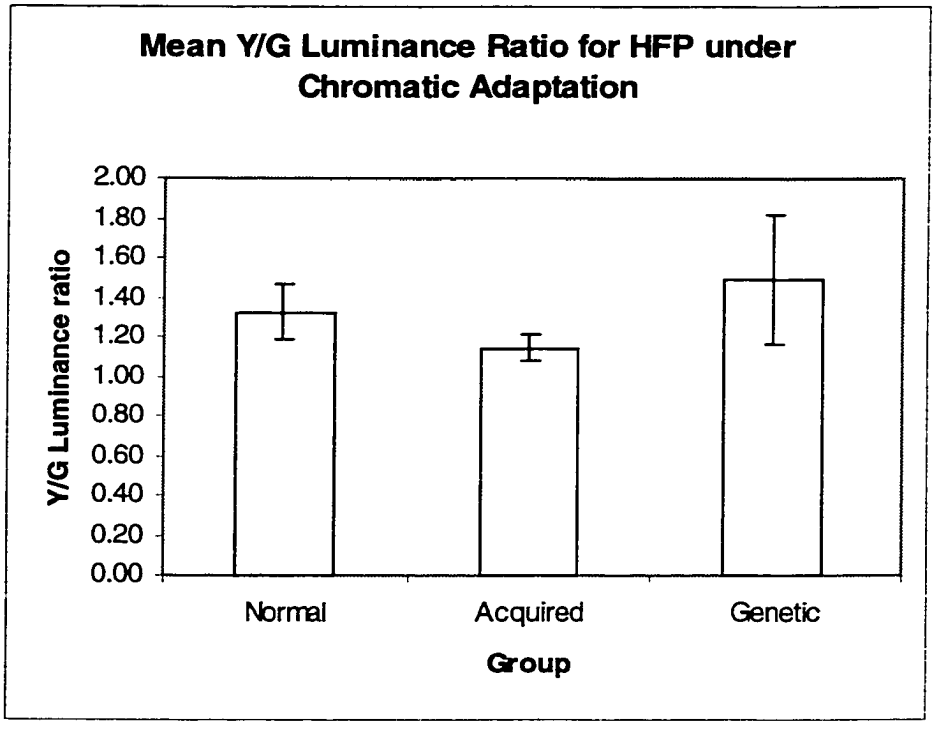


Figure 24. Group mean yellow to green luminance ratio needed to minimize a yellow alternating with green flicker under chromatic adaptation. Error bars equal +/- 1 SEM.

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Table 6. Anomaloscopy: Chromatic Adaptation: Rayleigh Matching

| Group | Normal | Acquired | Genetic |
|---|---|---|---|
| Mean G/R luminance ratio for Red Adapt Rayleigh matching | 1.43 | 17.78 | 6.25 |
| SEM | 0.09 | 16.24 | 1.60 |
| Median | 1.27 | 1.60 | 0.36 |
| Mean range of acceptable ratios for Red Adapt Rayleigh matching | 0.56 | 96.73 | 33.06 |
| SEM | 0.09 | 96.32 | 32.74 |
| Median | 0.41 | 0.35 | 0.36 |
| Mean Red Adapt range midpoint | 1.46 | 49.85 | 16.63 |
| SEM | 0.09 | 48.30 | 16.32 |
| Median | 1.29 | 1.62 | 0.36 |
| Mean Red Adapt Anomalous Quotient | 1.00 | 12.41 | 4.36 |
| SEM | 0.06 | 11.34 | 4.15 |
| Median | 0.89 | 1.12 | 0.25 |

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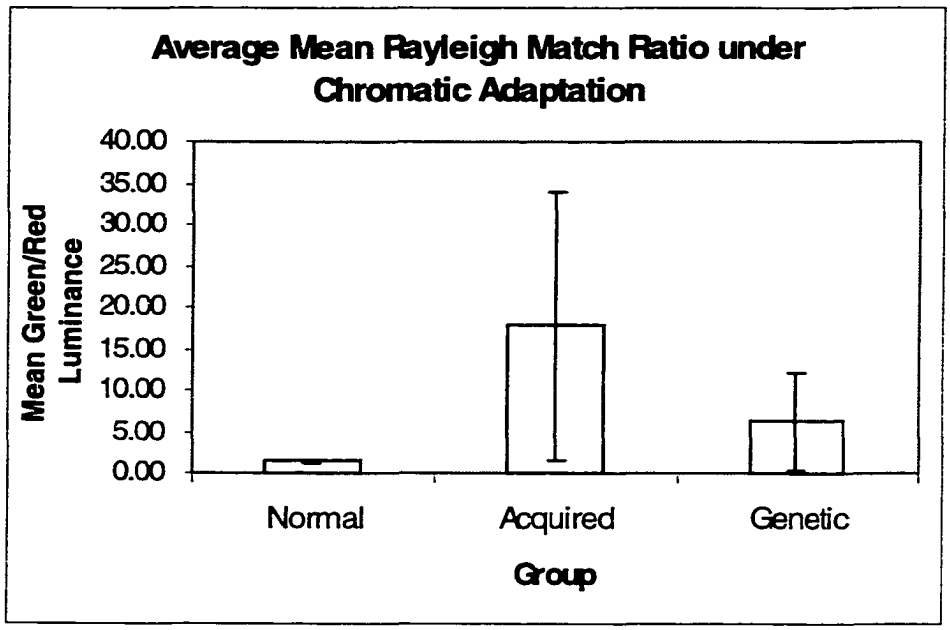


Figure 25. Average mean green to red luminance ratio required by each group to make a Rayleigh match (match to a standard yellow) under chromatic adaptation. Error bars equal +/- 1 SEM.

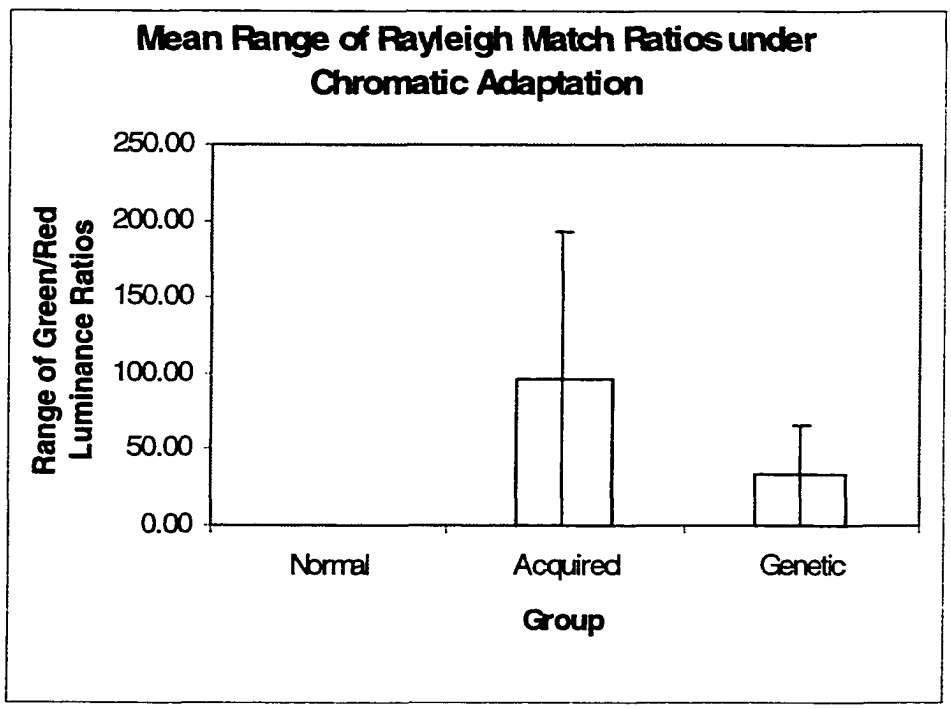


Figure 26. Mean range of green to red luminance ratios accepted as Rayleigh matches for each group under chromatic adaptation. Error bars equal +/- 1 SEM.

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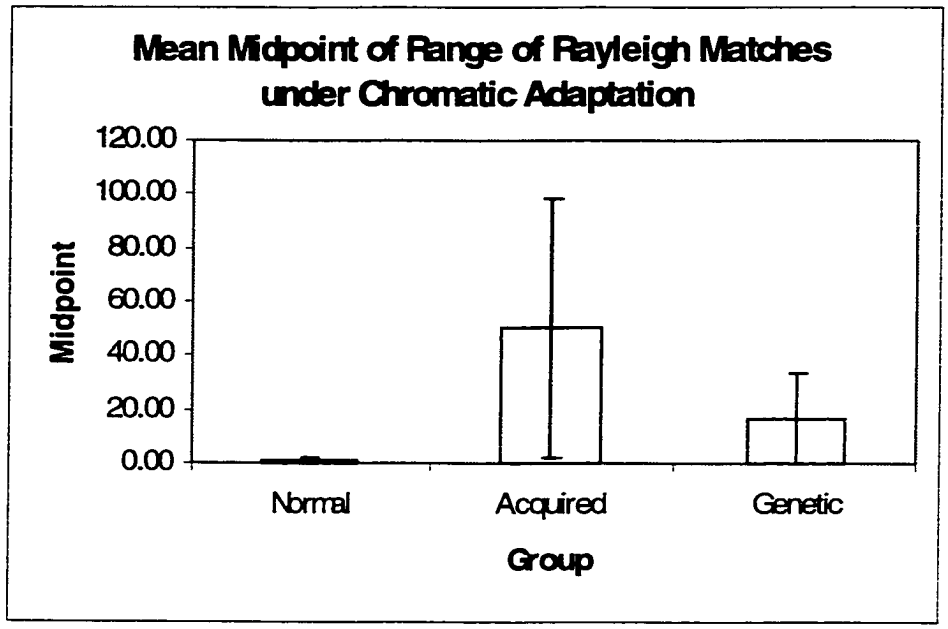


Figure 27. Mean midpoint of green to red luminance ratios accepted as Rayleigh matches for each group under chromatic adaptation. Error bars equal +/- 1 SEM.

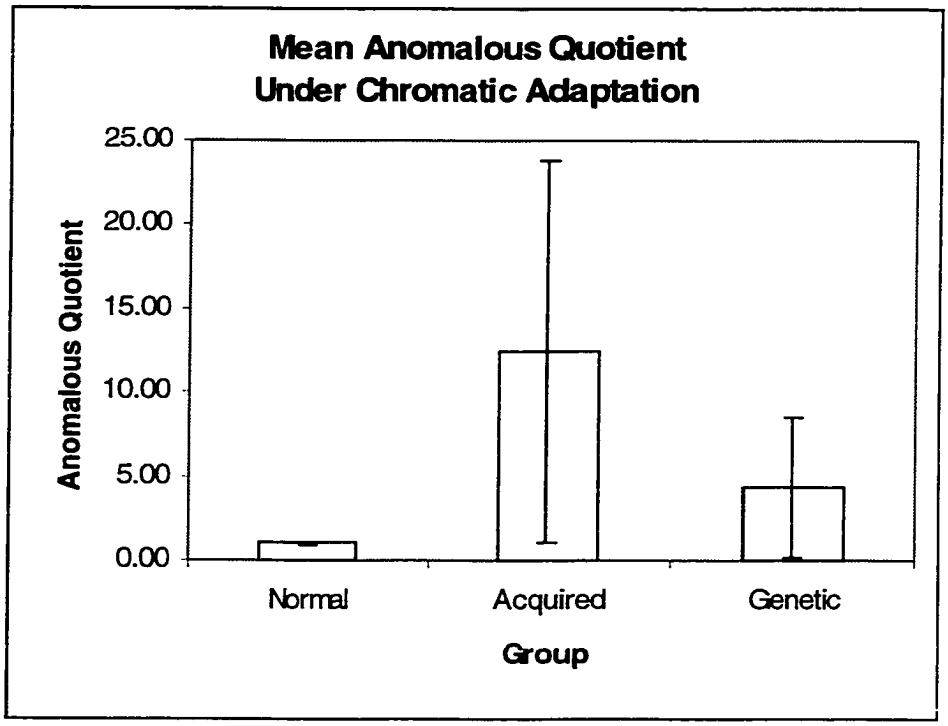


Figure 28. Anomalous quotient for each group under chromatic adaptation. Error bars equal +/- 1 SEM.

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To help facilitate the comparison of group performance on anomaloscope measures under the conditions of chromatic and non-chromatic adaptation, Figures 29-34 display the means (and Figures B25-B30, in Appendix B, the medians) for each condition side by side. For HFP, there was a general trend toward higher yellow to red and yellow to green luminance ratios across all observers (indicating a need for less red and green light) under chromatically adapted conditions (see Figures 29 and 30). Average mean green to red luminance ratios required for Rayleigh matching increased tremendously for the acquired group, less so for the genetics, and almost not at all for color-normals under chromatic adaptation (see Figure 31). The mean range of ratios was also higher across all groups (most markedly in the acquireds) when chromatically adapted. The genetic group was actually found to be more consistent in matching when red adapted, although this is most likely attributable to the reduced sample size run through this condition (see Figure 32). Similarly, the mean range midpoint was higher across all groups, shifted toward needing more green for an acceptable match. Again, the difference between conditions was most drastic in the acquired group (see Figure 33). Group mean anomalous quotients increased in each of the color-deficient groups under chromatic adaptation (see Figure 34).

Again, the main reason for running the anomaloscope sessions under chromatic adaptation was to see if there was a "breakdown" of any observers' Rayleigh matches (thus violating Grassmann's Law, and, presumably, indicating the presence of more than two cone types sensitive to longer wavelengths). The occurrence of such a breakdown would be most obvious upon the examination of differences in average mean green to red luminance ratios (i.e., Rayleigh match ratios) between the two adaptation conditions. To

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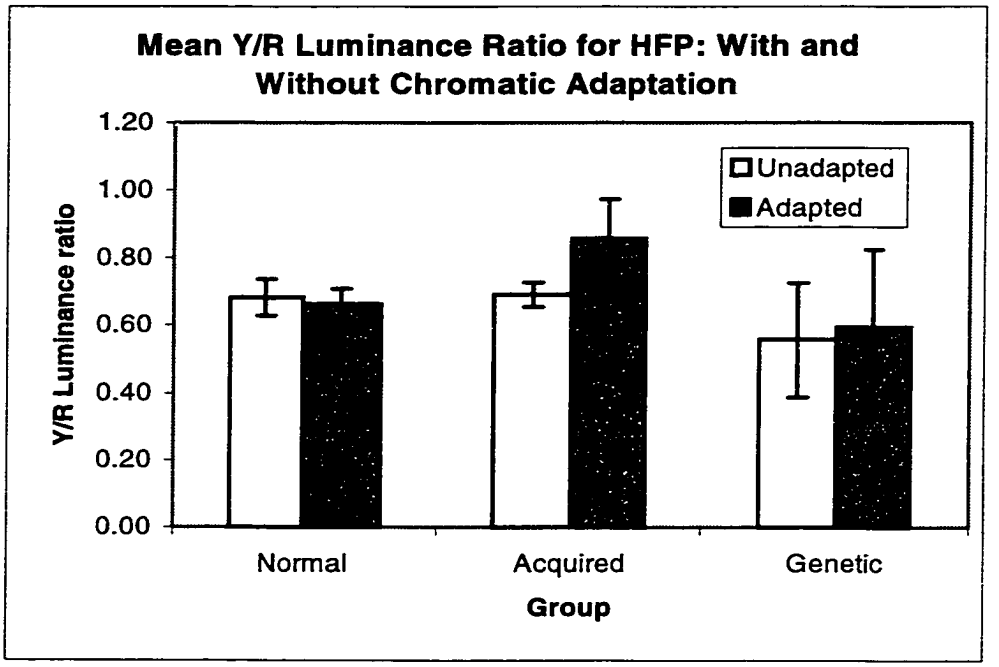


Figure 29. Mean yellow to red luminance ratio needed to minimize a yellow alternating with red flicker for each group with and without chromatic adaptation. Error bars equal +/- 1 SEM.

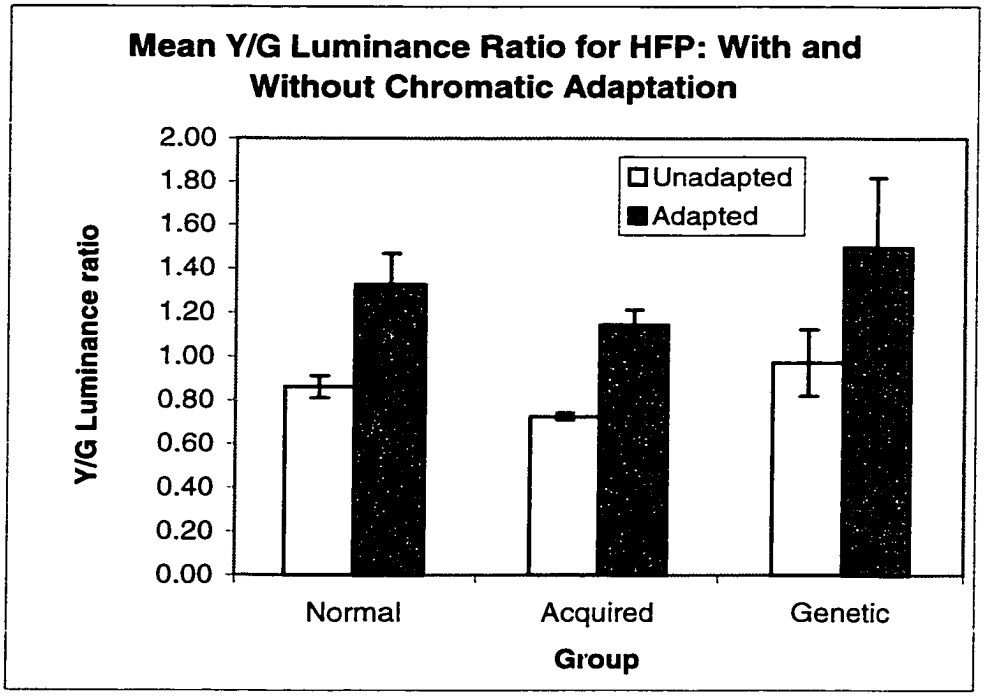


Figure 30. Mean yellow to green luminance ratio needed to minimize a yellow alternating with green flicker for each group with and without chromatic adaptation. Error bars equal +/- 1 SEM.

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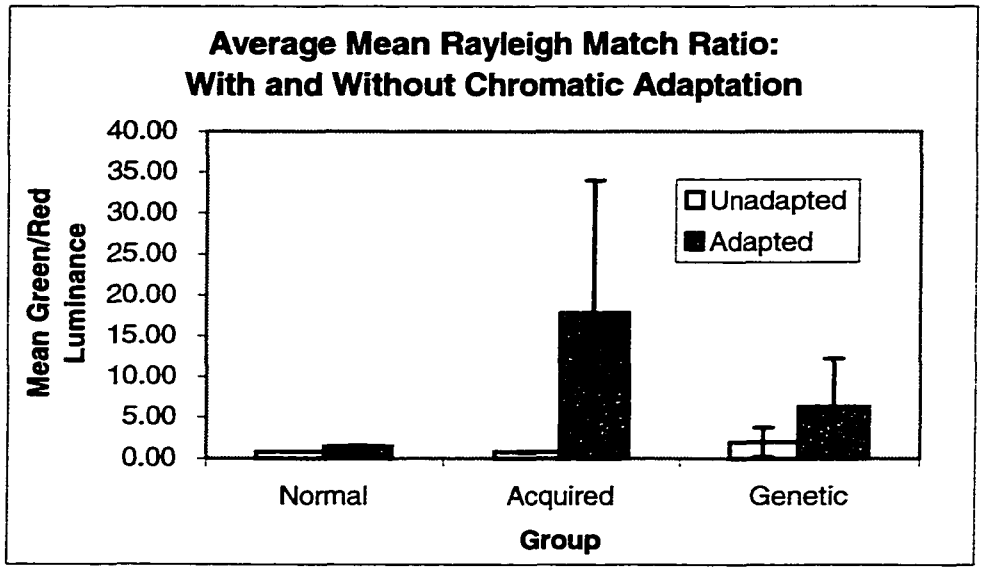


Figure 31. Average mean green to red luminance ratio required by each group to make a Rayleigh match (match to a standard yellow) with and without chromatic adaptation. Error bars equal +/- 1 SEM.

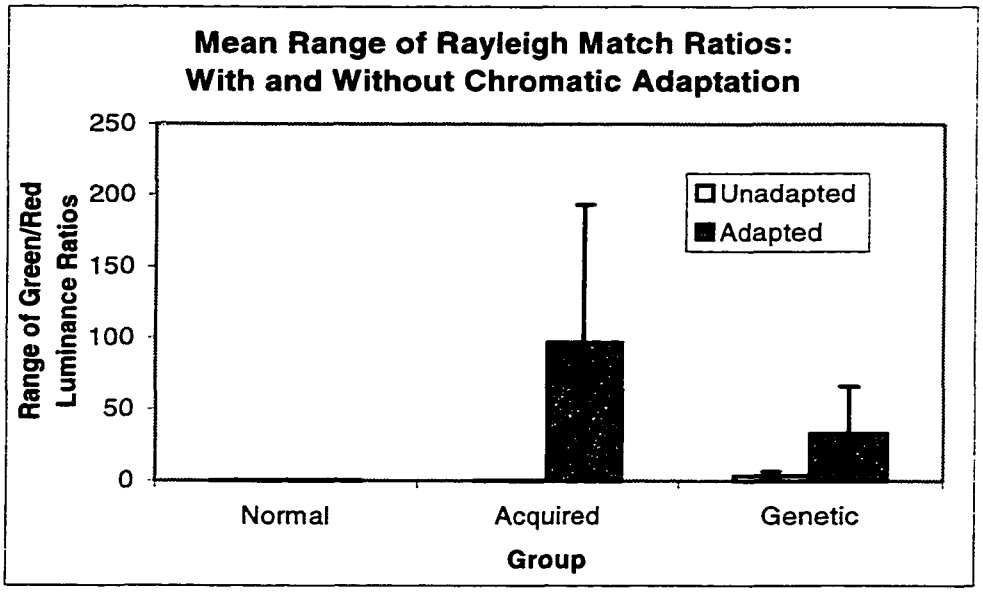


Figure 32. Mean range of green to red luminance ratios accepted as Rayleigh matches for each group with and without chromatic adaptation. Error bars equal +/- 1 SEM.

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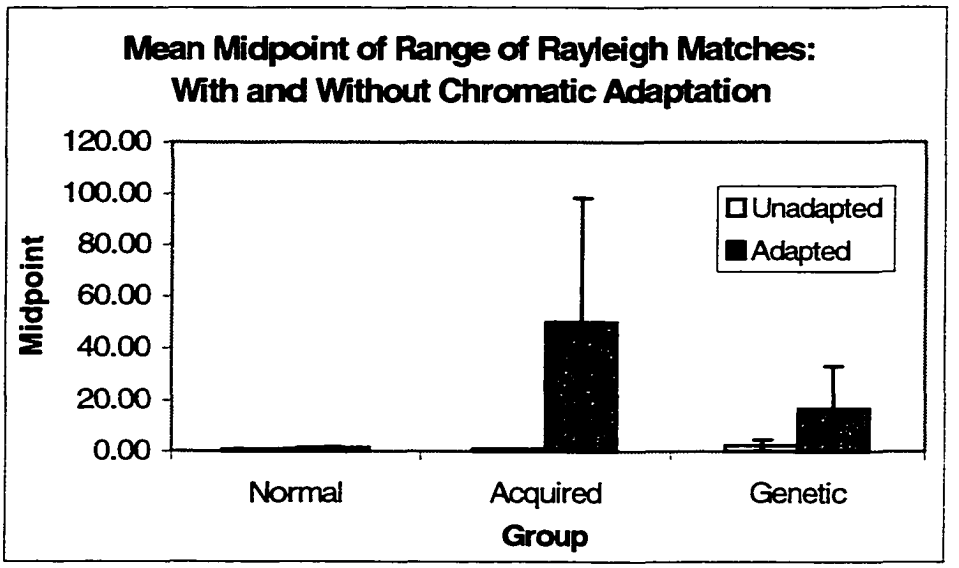


Figure 33. Mean midpoint of green to red luminance ratios accepted as Rayleigh matches for each group with and without chromatic adaptation. Error bars equal +/- 1 SEM.

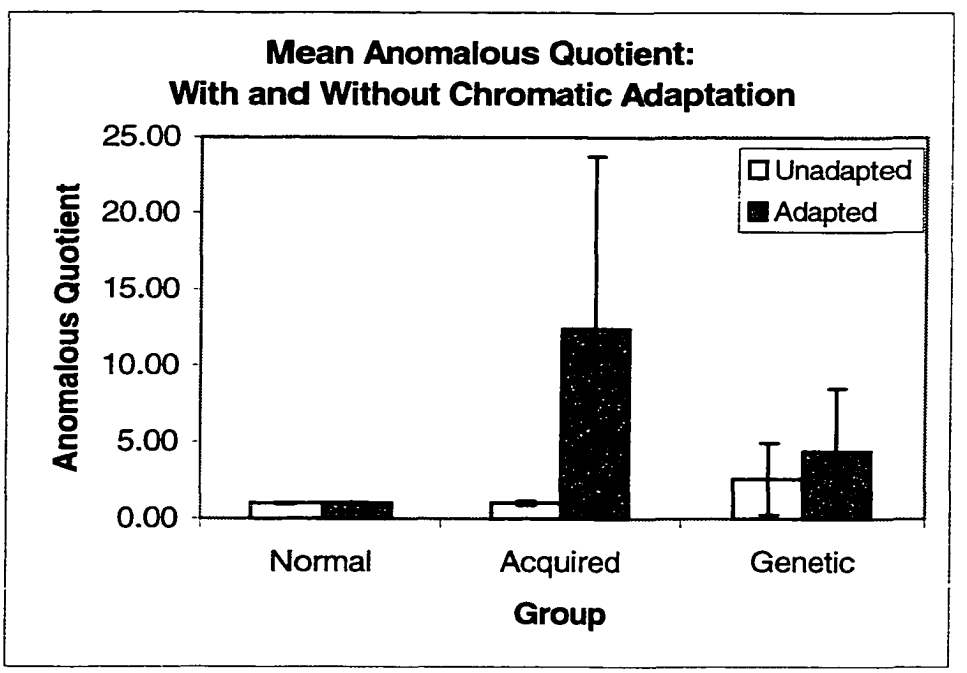


Figure 34. Group mean anomalous quotient with and without chromatic adaptation. Error bars equal +/- 1 SEM.

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allow for a closer look at this relationship, Table 7 presents each group's mean green to red luminance ratio difference (as well as SEM and median) when compared between conditions. These are also displayed, graphically, in Figures 35 (below) and B31 (in Appendix B). Again, one member each from the normal and genetic groups, who were counted in their groups' unadapted means, was not included in the means for chromatic adaptation.

As mentioned earlier, all three groups, on average, required a greater amount of green light (compared to red light) in order to match a standard yellow when chromatically adapted. The differences between the mean Rayleigh ratios in each condition readily show this. Chromatic adaptation affected acquired color-deficient observers more so than either genetic color-deficients or color-normals. However, there was a greater degree of variability in both of the color-deficient groups compared to the normal group due to extreme values recorded by two observers (see above). Regardless of group, though, every participant run through both conditions required, to some degree, a larger amount of green than red to satisfactorily make a match when adapted to a long wavelength light.

Rod and SWS cone sensitivity. Rod and SWS cone sensitivity was measured in 22 color-normals, 7 acquired color-deficients, and 5 genetic color-deficients. The values displayed in Table B2 (see Appendix B) and Figures 36-47 (and Figures B32-B42 in Appendix B) are based on this sample.

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Table 7. Anomaloscopy: Differences in Mean Rayleigh Ratio Between Adaptation Conditions

| Group | Normal | Acquired | Genetic |
|---|---|---|---|
| Mean adapted ratio - mean unadapted ratio | 0.70 | 17.01 | 6.06 |
| SEM | 0.09 | 16.26 | 5.76 |
| Median | 0.55 | 0.86 | 0.34 |

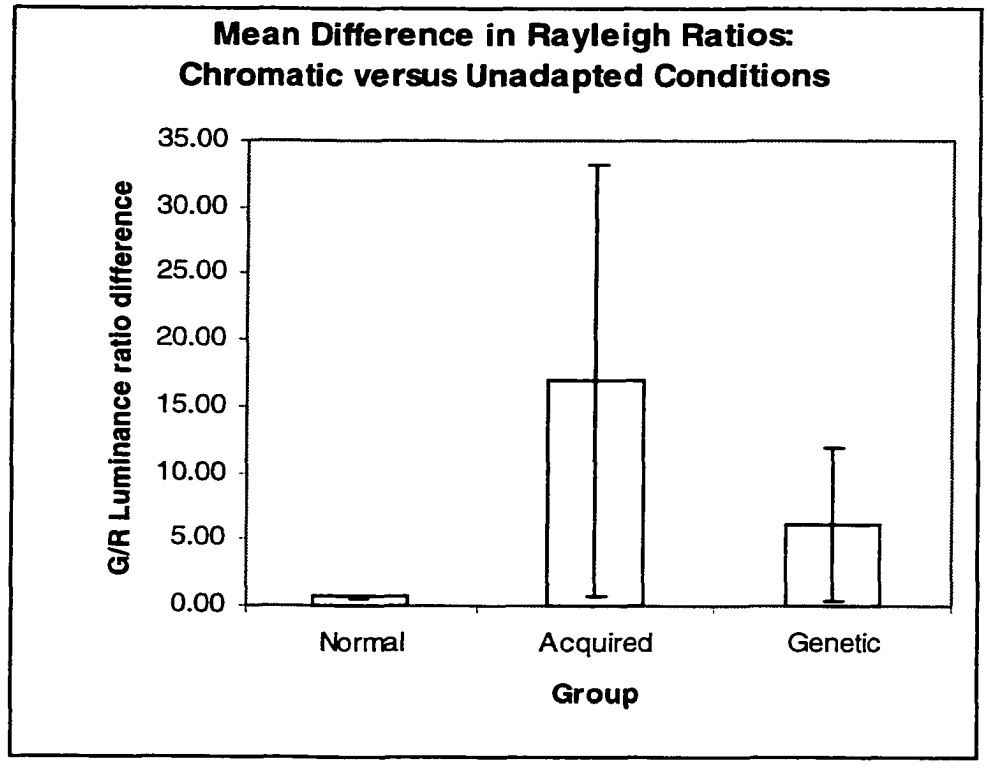


Figure 35. Mean difference of mean adapted and mean unadapted Rayleigh ratios for each group. Error bars equal +/- 1 SEM.

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Rod sensitivity was measured at retinal eccentricities of 2° and 6° for wavelengths of 534 nm and 425 nm. Obtained values were adjusted to fit a V-lambda curve that had been normalized to have a peak of 1.0. The mean relative radiance of a 534 nm light needed to just detect a flash at threshold (absolute limen) at an eccentricity of 2° was, for the normal group, more than twice what was required by the genetic group. The acquired group necessitated an intermediate amount (see Figure 36). At an eccentricity of 6°, these relative radiances decreased, with the highest mean belonging to the group of acquired color-deficients, and the lowest to the genetics (see Figure 37).

Mean absolute limens for a 425 nm light at an eccentricity of 2° were greatest among color-normal observers and smallest among acquired color-deficients, although the means of the color-deficient groups were quite similar (see Figure 38). At 6° eccentricity, group means were nearly identical and each group showed an improvement in sensitivity (see Figure 39).

Sensitivity loss for short wavelength light may be the result of absorption defects. While the absolute thresholds measured above are not a direct indication of SWS cone deficiencies, they can serve to help establish whether the measures of observers' spectral sensitivity thresholds might be mired by, for example, rod input, or, in individuals with reduced visual acuity, the preferred fixation point and retinal area stimulated. Macular pigment density and other preretinal factors, such as the lens, are major contributors to absorption. The macular pigment is present in foveal and nearby extrafoveal areas (including retinal eccentricities of 2°), becoming less dense with increasing eccentricity. At 6° eccentricity, the macular pigment is mostly absent. Figure 40 plots the group mean absolute limens (in relative sensitivity) in relation to the scotopic spectral luminous

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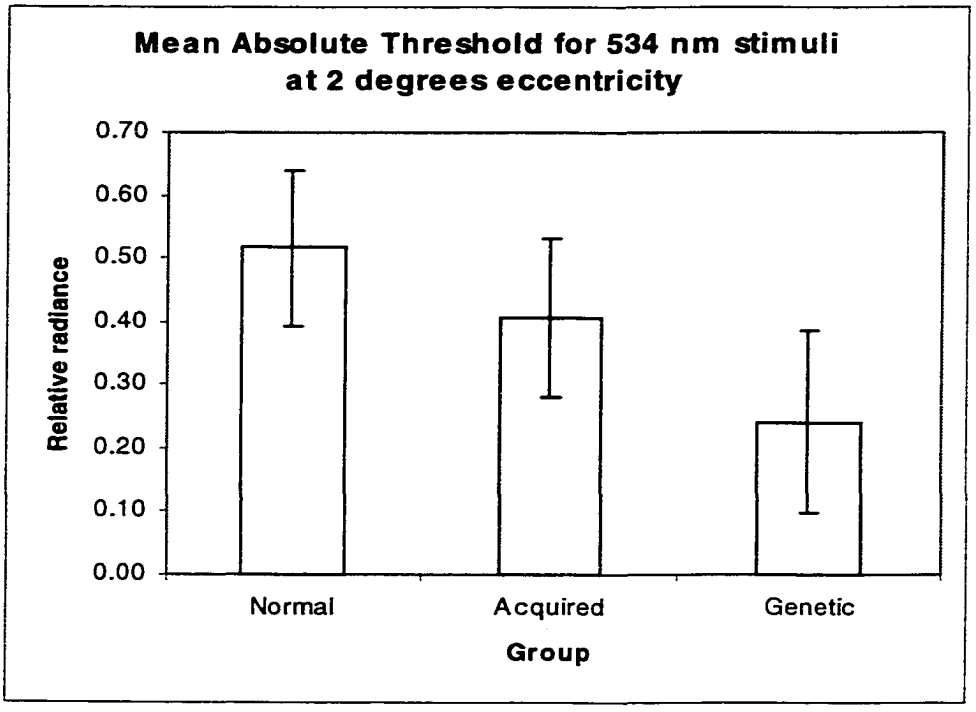


Figure 36. Mean relative radiance necessary for just noticeable detection of a flash (absolute limen) of 534 nm light at a retinal eccentricity of 2°. Error bars equal +/- 1 SEM.

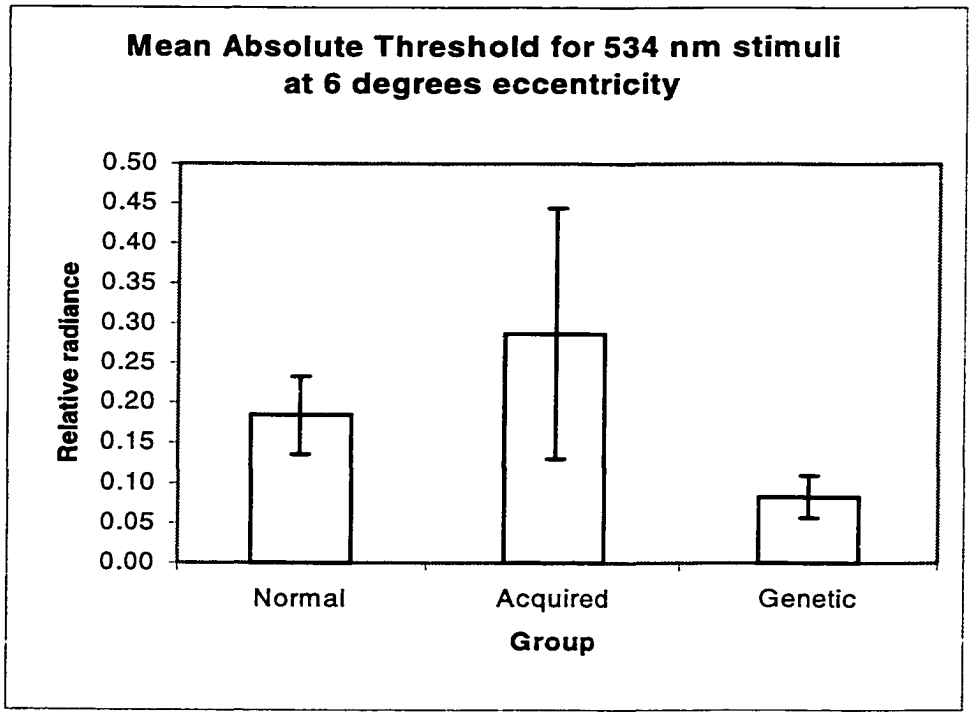


Figure 37. Mean relative radiance necessary for just noticeable detection of a flash (absolute limen) of 534 nm light at a retinal eccentricity of 6°. Error bars equal +/- 1 SEM.

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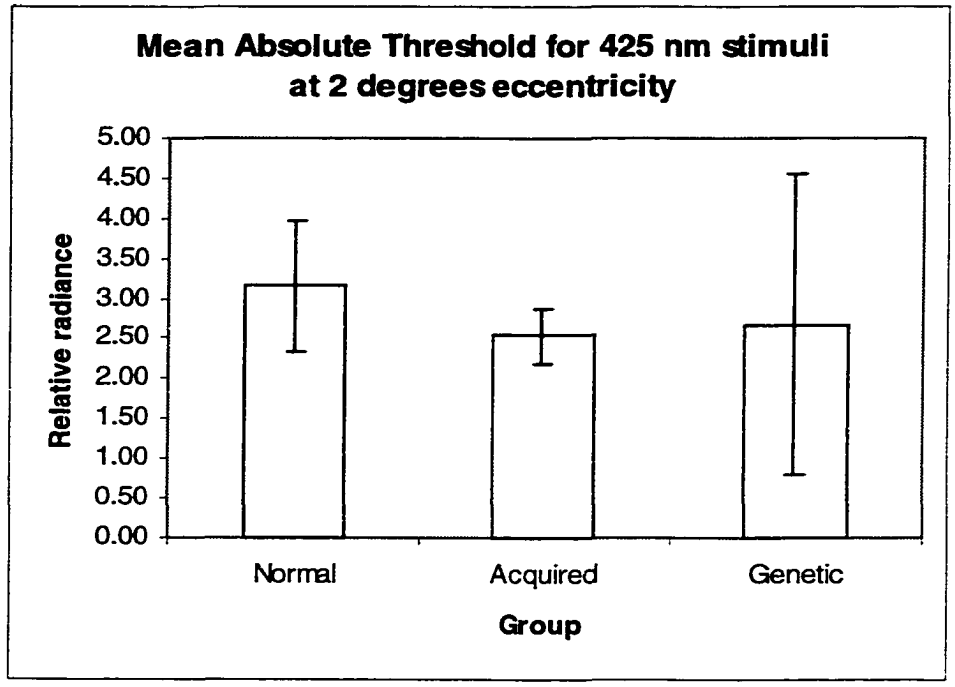


Figure 38. Mean relative radiance necessary for just noticeable detection of a flash (absolute limen) of 425 nm light at a retinal eccentricity of 2°. Error bars equal +/- 1 SEM.

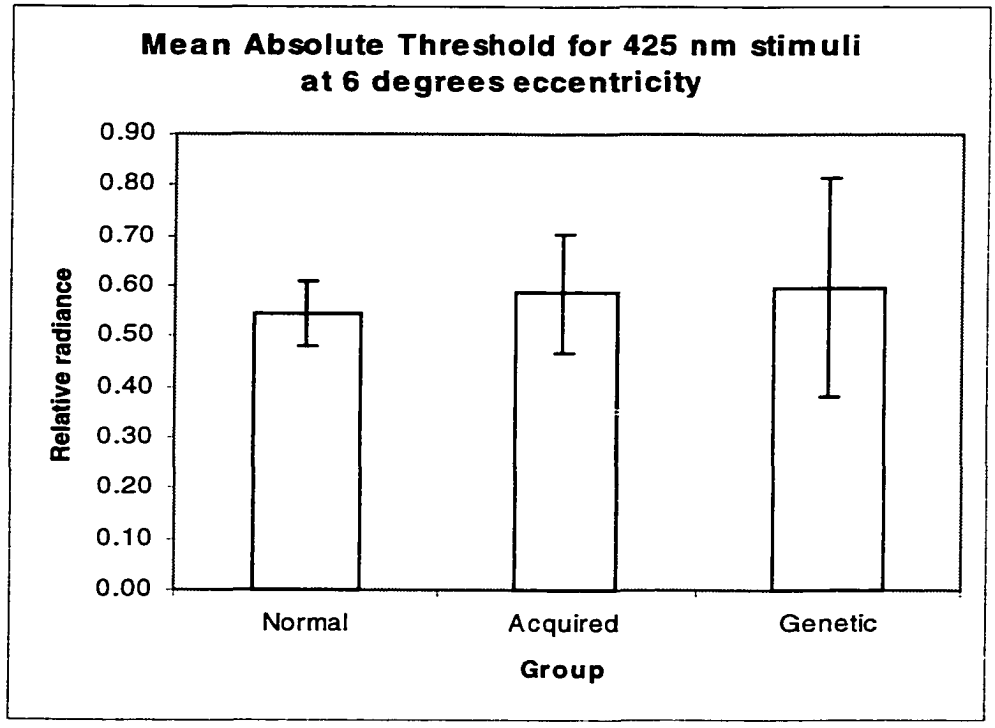


Figure 39. Mean relative radiance necessary for just noticeable detection of a flash (absolute limen) of 425 nm light at a retinal eccentricity of 6°. Error bars equal +/- 1 SEM.

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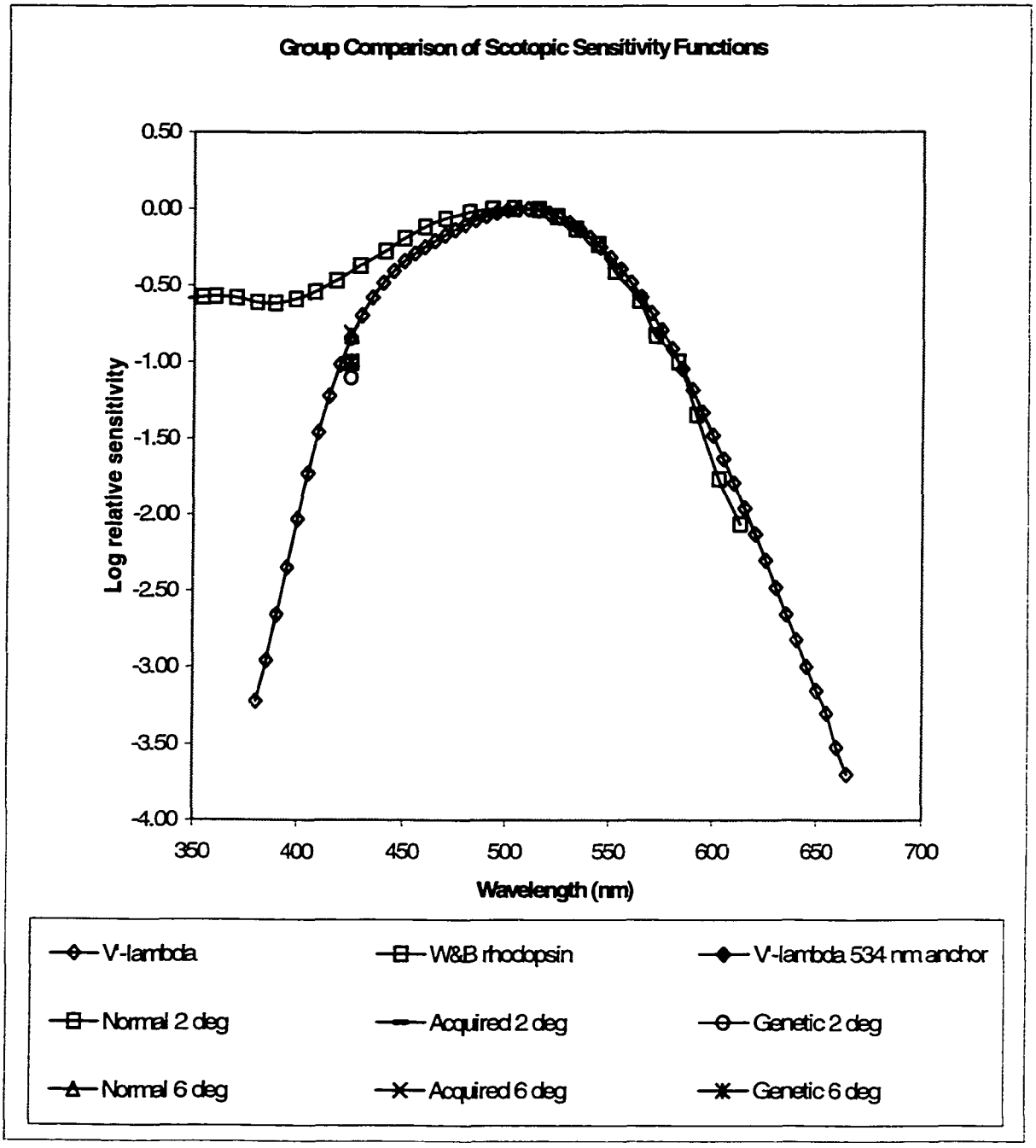


Figure 40. Comparison of group scotopic luminosity functions. Group means have been adjusted to eliminate the absolute sensitivity difference and coincide with V'-lambda at 534 nm. The normalized human rhodopsin curve (adapted from Wald & Brown, 1958), with an optical density of 0.3, is also plotted for reference.

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efficiency function (V'-lambda). The human rhodopsin curve is also shown. (Differences between V'-lambda and the rhodopsin curve are due to preretinal factors, as the rhodopsin curve is based on measurements from aphakic eyes.) At 534 nm there are no sensitivity losses resulting from macular pigment density or preretinal factors, as these filters have only negligible amounts of absorption in this region of the spectrum. With macular pigment having a maximal density (~ 0.35-0.5 log units) for wavelengths of about 460 nm, we plotted each group's mean absolute limens for 425 nm at each of the two eccentricities, adjusting the values (for absolute sensitivity differences) with respect to V'-lambda at 534 nm (anchor point in Figure 40). As seen in the figure, the normal and acquired groups' thresholds for 2° eccentricity are nearly identical, with genetics showing less sensitivity by a factor of 0.1 log units, on average. Thresholds for color-normals and acquired color-deficients at 6° lie almost directly on the V'-lambda curve, while that of the genetic group differs by about 0.2 log units (again in the less sensitive direction). The differences in sensitivity between the two eccentricities, for the normal and acquired groups, show these observers to be more sensitive at the more peripheral eccentricity (by 0.2 log units), this difference being attributable to (and equaling the density of) the macular pigment at 425 nm. The genetic group, while also showing increased sensitivity at 6°, had a net gain of only 0.1 log units. Thus, these data show that the genetics differ from the normals and acquireds in two ways. First, the smaller difference in sensitivities between eccentricities points to these individuals, on average, as having a reduced macular pigment density compared to members of the other groups. Second, the lower sensitivities at both the 2° and 6° eccentricities indicate that the lens of the genetic color-deficient is somehow different from that of color-normals and acquired color-deficients.

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That is, there is more preretinal absorption taking place in these observers. These differences are real, given the agreement of the normals' and acquireds' 6° thresholds with that of V'-lambda (which is based on measurements obtained from young observers at an eccentricity of about 5°; see Wyszecki & Stiles, 1982), and the conventional changes in sensitivity exhibited by all groups when shifting from a retinal eccentricity of 2° to that of 6°.

The mean relative radiance ratio of 534 nm to 425 nm light at absolute threshold at a 2° retinal eccentricity was determined to be highest for acquireds and lowest for genetics, although the difference between acquireds and normals was rather negligible (see Figure 41). This ratio at an eccentricity of 6° was found to exhibit a similar trend at the level of group means (though here there existed a greater difference between the acquired color-deficient and color-normal groups; see Figure 42).

The mean density of optical media at a 2° eccentricity was somewhat comparable across groups. (The method by which these values were calculated can be found in the Data analysis section, beginning on page 114.) The genetics tended to be slightly higher than the others at the group mean (see Figure 43). (We had earlier found this to be due to a difference in preretinal factors; see Figure 40 and above.) The mean optical density at 6° eccentric due to preretinal absorption was more dissimilar between groups: the genetic group exceeded the other groups by a factor of about 1.6 (as seen in Figure 40; see Figure 44). The log optical density of macular pigment at a retinal eccentricity of 2° was found, on average, to be highest among acquired color-deficient observers, followed closely by color-normals, and lowest in genetics (see Figure 45). (This can also be seen in Figure 40 by observing the dissimilarity in the differences in sensitivities between the

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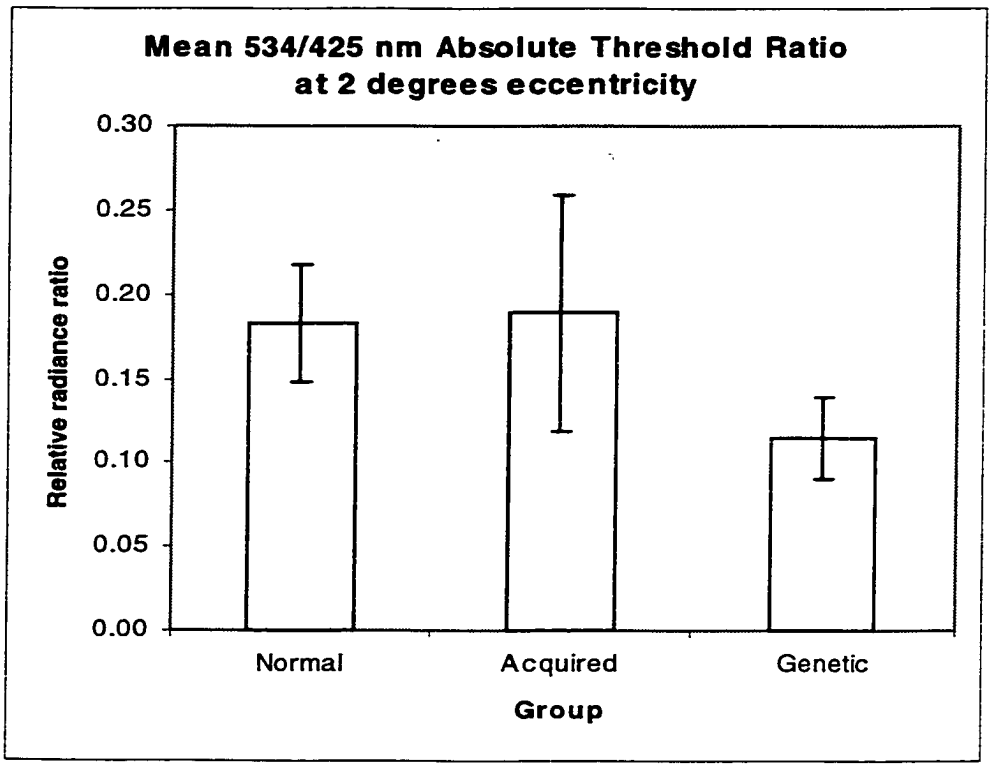


Figure 41. Mean 534 to 425 nm relative radiance ratio at absolute limen for each group at a retinal eccentricity of 2°. Error bars equal +/- 1 SEM.

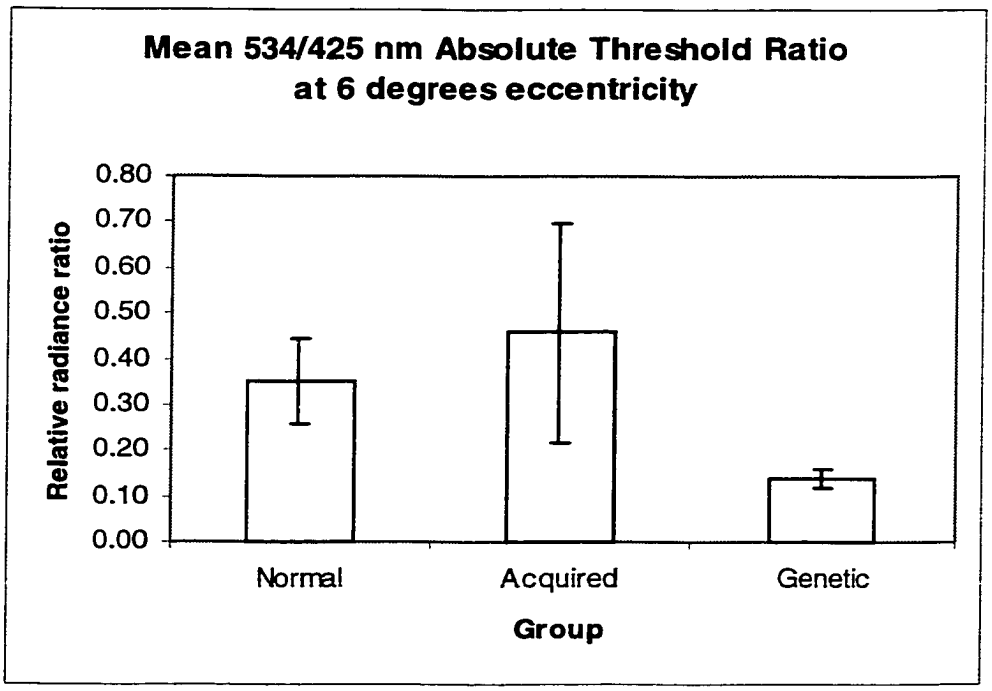


Figure 42. Mean 534 to 425 nm relative radiance ratio at absolute limen for each group at a retinal eccentricity of 6°. Error bars equal +/- 1 SEM.

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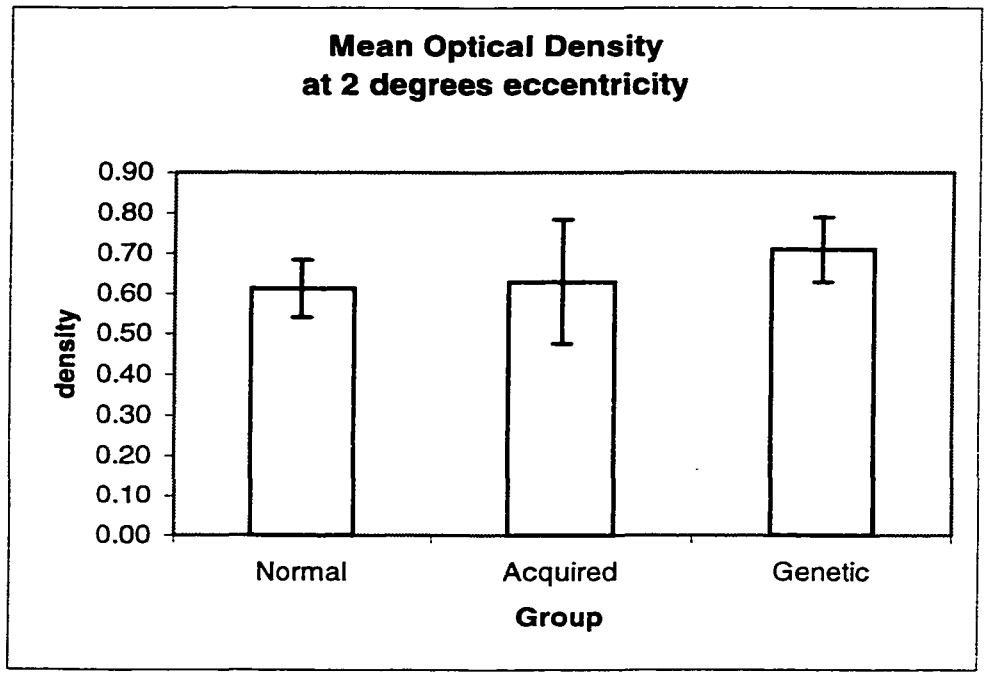


Figure 43. Mean optical density for each group at a retinal eccentricity of 2°. Error bars equal +/- 1 SEM.

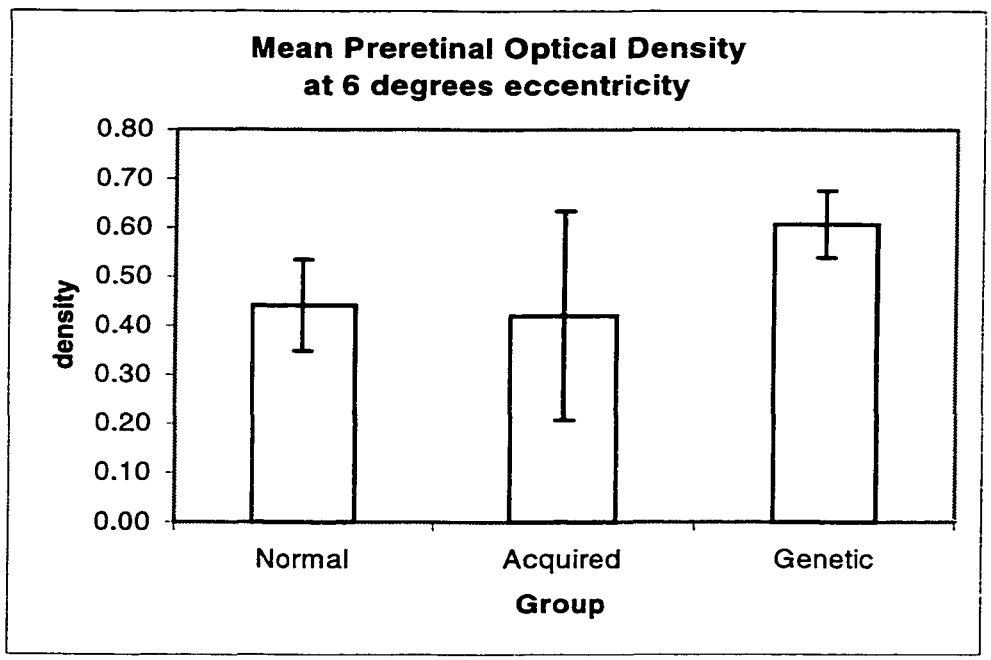


Figure 44. Mean optical density due to preretinal absorption for each group at a retinal eccentricity of 6°. Error bars equal +/- 1 SEM.

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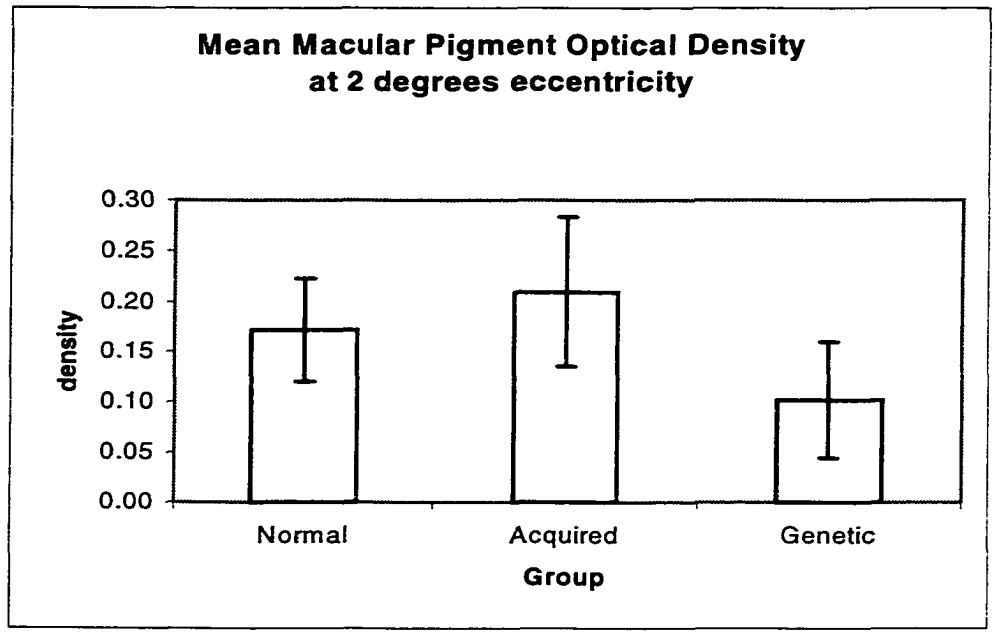


Figure 45. Mean optical density due to macular pigment for each group at a retinal eccentricity of 2°. Error bars equal +/- 1 SEM.

groups at the two eccentricities.)

The mean relative radiance of a blue (425 nm) light needed to be flashed against a yellow (596 nm) background (SWS cone increment threshold) was greatest for the normal group and least for the genetic group (see Figure 46). As is shown in Table B2 and in Figure 47, when corrected for the density of optical media, these mean SWS cone thresholds, measured in log relative radiance units, were found to be lowest for the genetic group and approximately equal (yet higher by a factor of about 1.6) in the normal and acquired groups.

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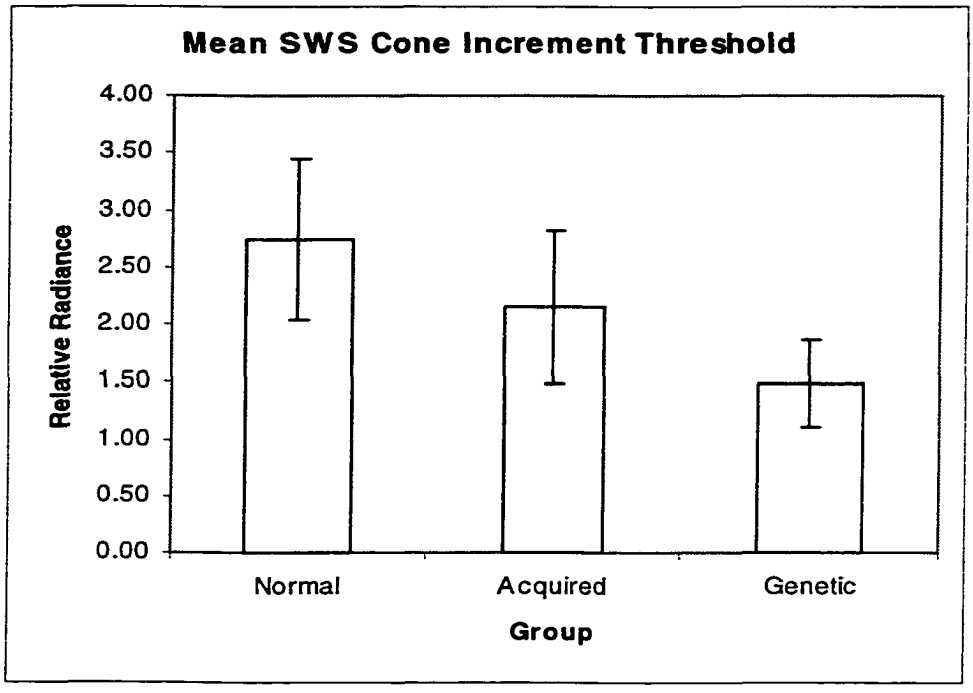


Figure 46. Mean relative radiance necessary for just noticeable detection of a flash (increment threshold) of 425 nm light on a bright 596 nm background for each group. Error bars equal +/- 1 SEM.

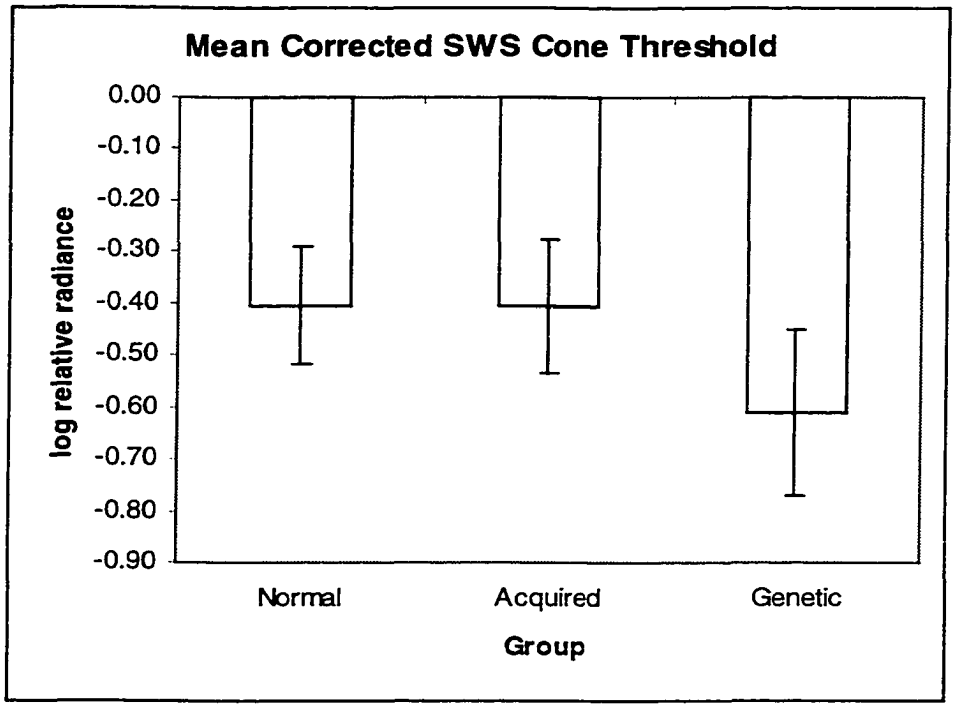


Figure 47. Mean relative radiance necessary for just noticeable detection of a flash (increment threshold) of 425 nm light on a bright 596 nm background for each group corrected for optical media density. Error bars equal +/- 1 SEM.

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Spatio-temporal contrast sensitivity. In comparing contrast sensitivities, regardless of spatial or temporal frequency, for nearly every spatio-temporal combination, the genetics had equal or better sensitivity than the normals (see Figures 48-52). (The values from which these figures are based can be found in Table B3 in Appendix B, accompanied by graphs displaying group medians in Figures B43-B47.) This can be seen most vividly in Figure 53, which plots the ratio of the genetic group's mean binocular contrast sensitivity to that of the normals. Genetics' superior performance is indicated by ratios greater than 1.0. (These and the following measures [described below] represent binocular data obtained from 27 of the 28 color-normal observers [see the General overview subsection, on page 118, regarding the exclusion of one participant], and the complete acquired color-deficient [n = 7] and genetic color-deficient [n = 5] groups.)

For each temporal frequency tested, we next obtained four values (whose definitions and methods of derivation can be found in the Data analysis section, beginning on page 115): the mean contrast sensitivity at the lowest spatial frequency (i.e., 0.6 cpd; the results of which can be found in Appendix B, Table B3), the mean maximum (peak) contrast sensitivity (results presented in Table B4), the ratio of sensitivities at lowest spatial frequency compared to peak contrast sensitivity ("low-to-peak"; results presented in Table B5), and the mean spatial frequency at which peak sensitivity occurred (results presented in Table B6). Few of these measures yielded any significant between-group differences. Those that did we are attributing to noise, since there is no consistency among the variables which did (and did not) lead to significant results at a certain

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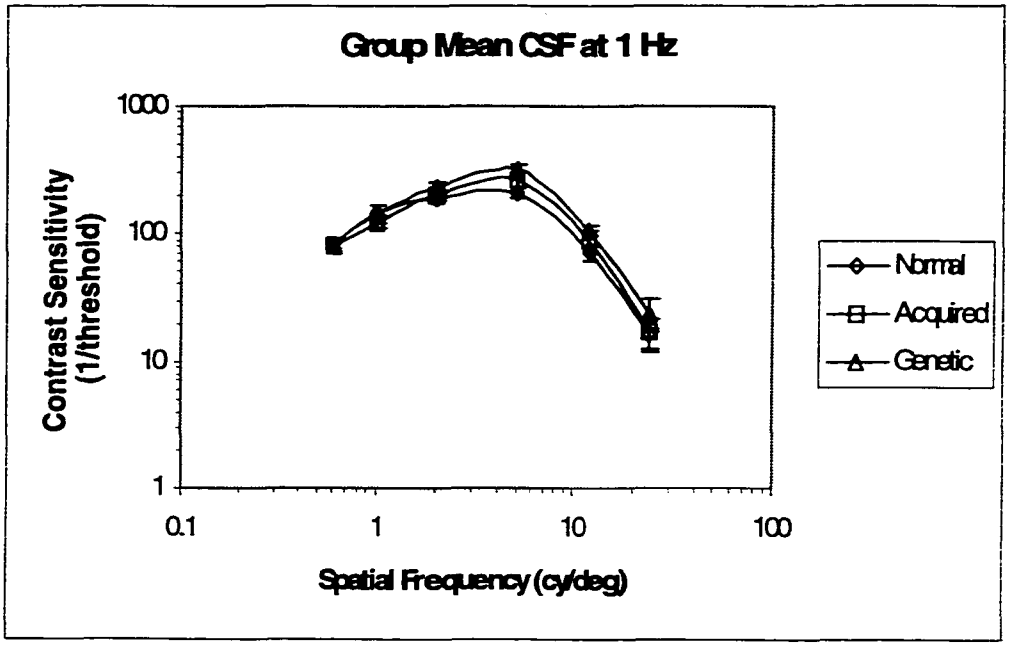


Figure 48. Mean contrast sensitivity function for each group at 1 Hz for the entire spatial domain tested. Error bars equal +/- 1 SEM.

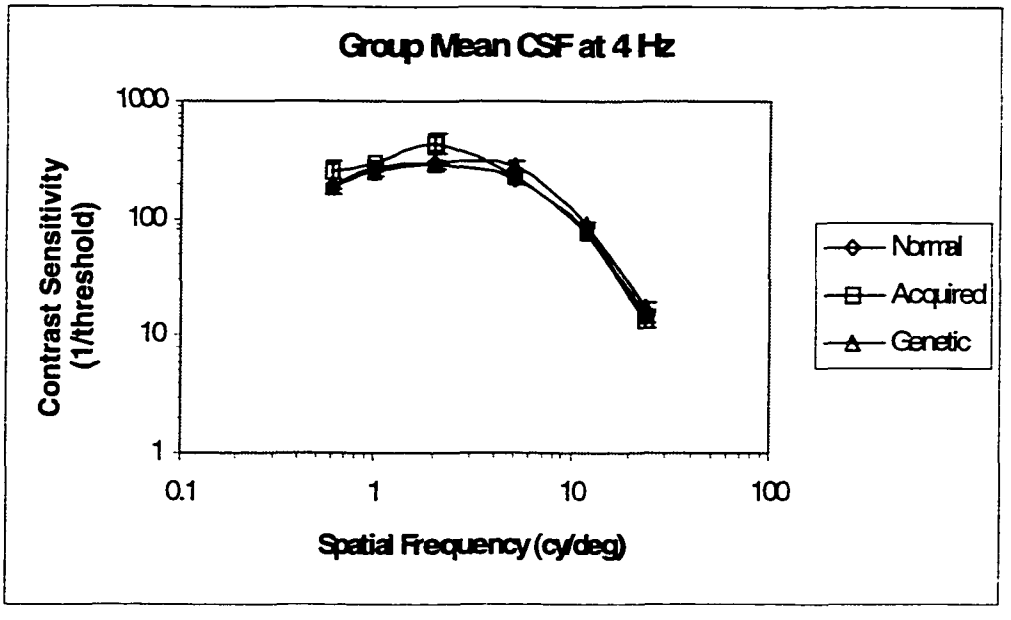


Figure 49. Mean contrast sensitivity function for each group at 4 Hz for the entire spatial domain tested. Error bars equal +/- 1 SEM.

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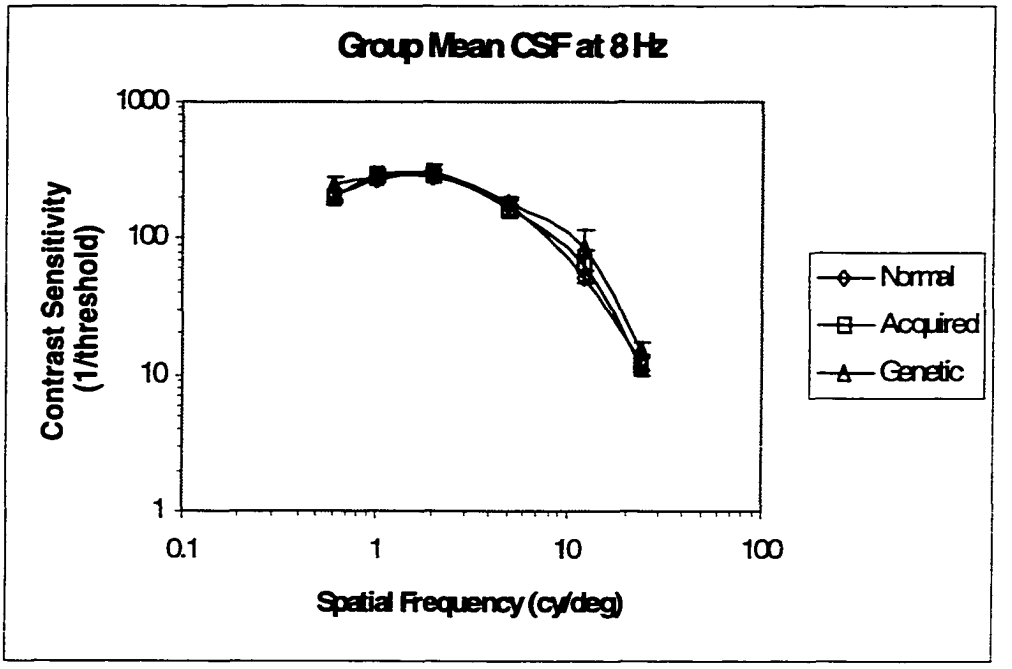


Figure 50. Mean contrast sensitivity function for each group at 8 Hz for the entire spatial domain tested. Error bars equal +/- 1 SEM.

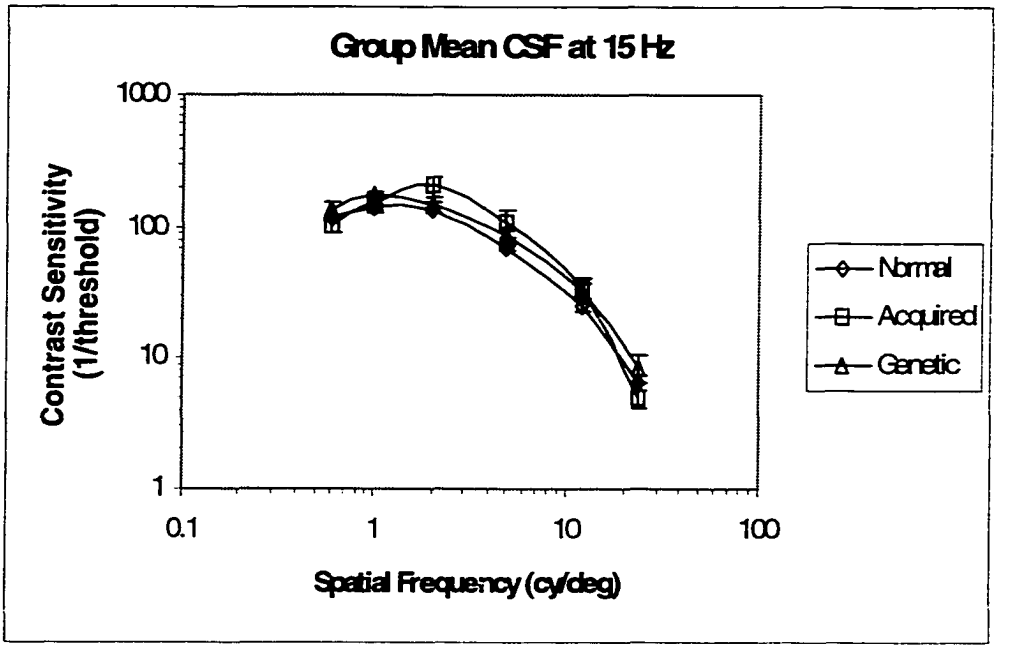


Figure 51. Mean contrast sensitivity function for each group at 15 Hz for the entire spatial domain tested. Error bars equal +/- 1 SEM.

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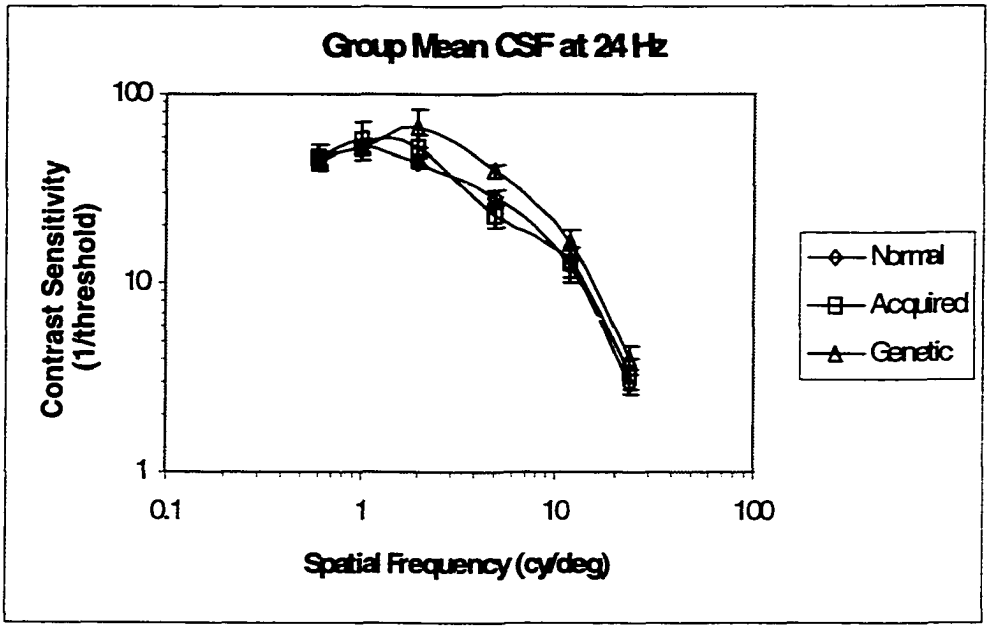


Figure 52. Mean contrast sensitivity function for each group at 24 Hz for the entire spatial domain tested. Error bars equal +/- 1 SEM.

frequency but not at others. Because of this, all information pertaining to these measures (graphs, descriptions of variability, statistical analyses, etc.) is presented in Appendix B.

Binocular vision testing. Tasks comprising the binocular vision testing component of the battery (i.e., measurement of eye alignment, interpupillary distance, ocular dominance, ocular rivalry, and stereo-screening) produced little in the way of significant between-group differences. (Those variables that did are subject to the question of adequate statistical power and, consequently, whether these differences are real.) Complete descriptions of the results for each of these measures, and the values obtained, can be found in Appendix B.

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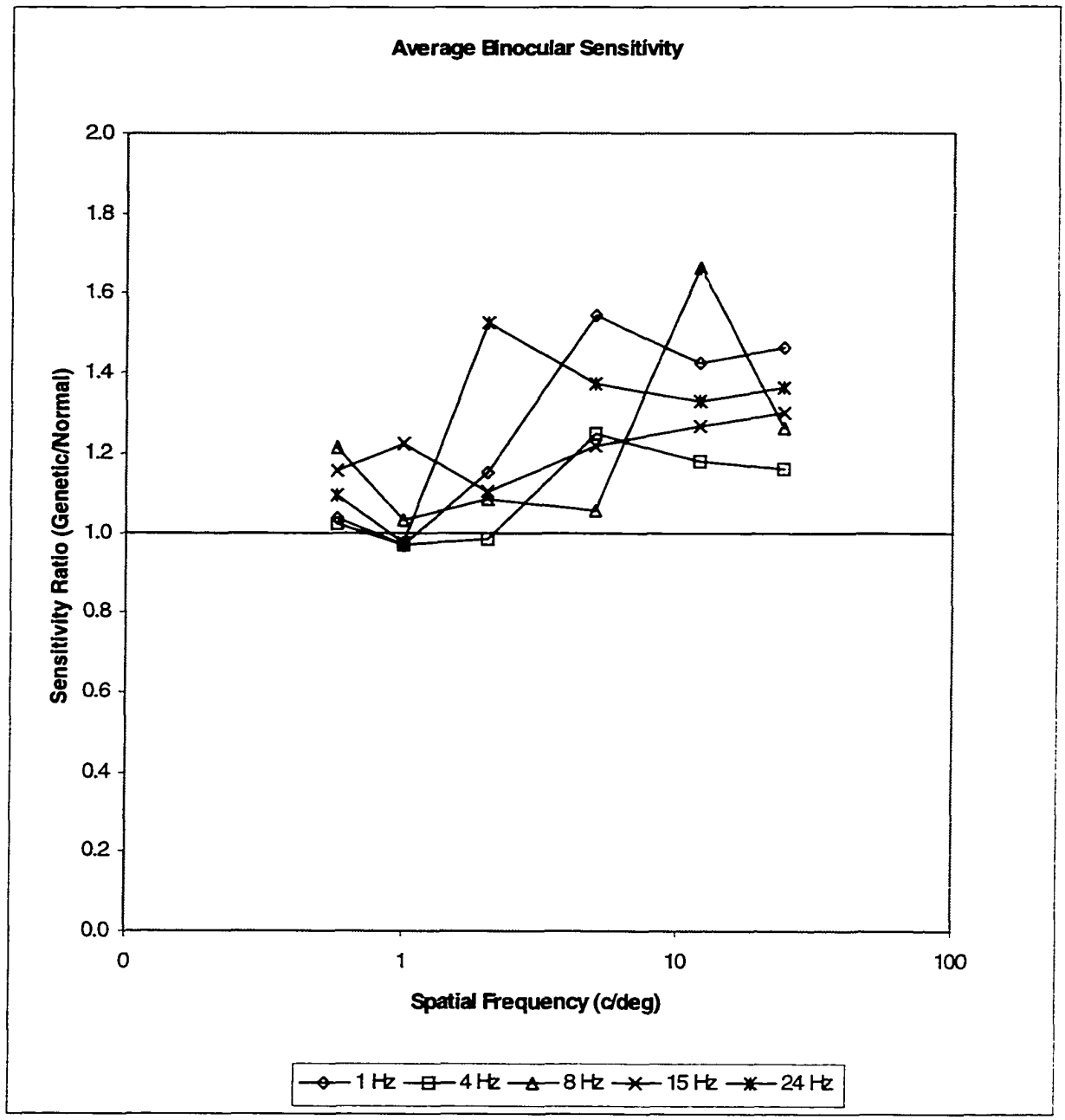


Figure 53. Comparison of genetic and normal group mean contrast sensitivities across the spatio-temporal domain. Ratios of sensitivities above 1.0 indicate that the color-deficient observers have a higher sensitivity at that spatio-temporal frequency pair.

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Stereopsis. Color-deficient observers were found to have higher sensitivities across all measures of stereopsis tested. These results are shown in Figures 54-57. (Values are presented, in full, in Table B8 in Appendix B, along with graphical displays of group medians in Figures B97-B100.) While each of the 40 study participants was run through this portion of the battery, not every observer gave reliable thresholds on each of the four measures.

The mean minimum amount of displacement needed by genetic color-deficients (n = 5) to detect an in-depth image popping out from the plane of the computer screen was more than twice as small as that required by color-normals (n = 27). The acquired color-deficient group (n = 6) needed the largest disparity (see Figure 54). The mean minimum amount of displacement needed for perceiving a pop-back image was, again, found to be smallest for the genetic group (by almost a factor of 2 compared to normals; see Figure 55). Differences between group means were due to several extreme scores among color-normals, and all 5 genetic group members having identical thresholds (rounded to two decimals). According to the results from both a one-way ANOVA and a Kruskal-Wallis test, differences between groups were not found to be significant on either of these measures ($p > 0.05$).

The mean maximum amount of disparity permitted by individuals prior to the breakdown and loss of depth in the forward direction was greatest for both the acquireds (n= 6) and genetics (n = 5), and smallest for the normals (n = 27; see Figure 56). In the backward direction, the largest mean disparity permitted was by the genetic color-deficient group (n = 5) followed behind by some 8 min arc by the acquired color-deficient (n = 5) and color-normal (n = 28) groups (these latter two groups differing by just over

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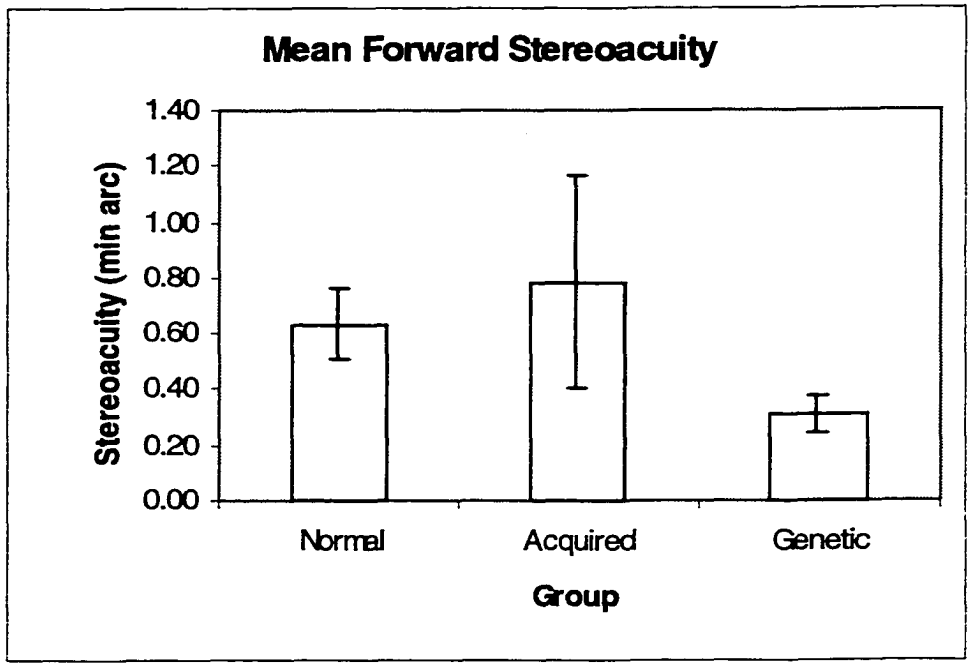


Figure 54. Mean forward stereoacuity for each group. Error bars equal +/- 1 SEM.

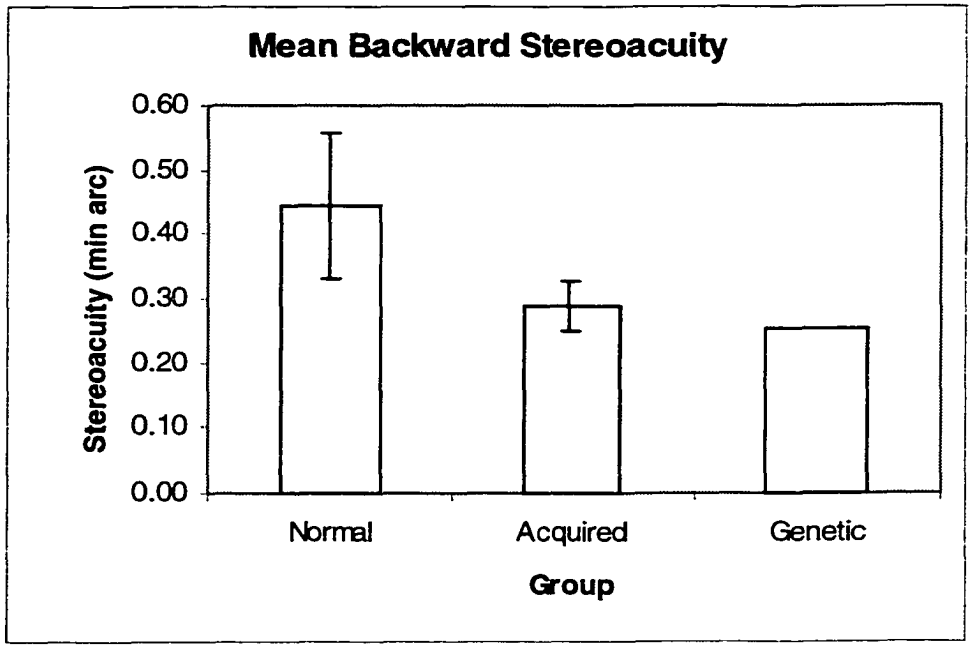


Figure 55. Mean backward stereoacuity for each group. Error bars equal +/- 1 SEM.

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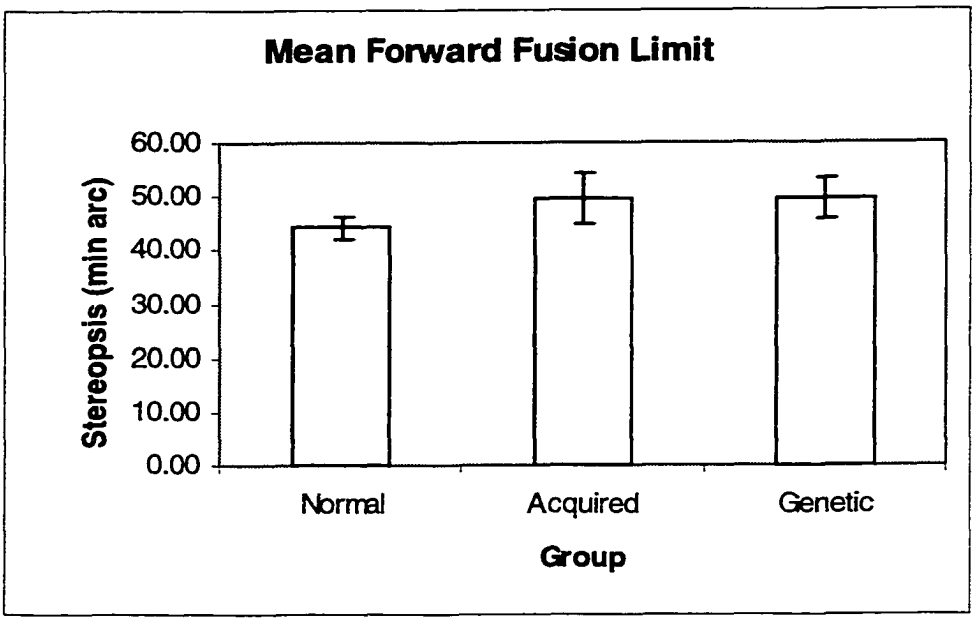


Figure 56. Mean forward fusion threshold for each group. Error bars equal +/- 1 SEM.

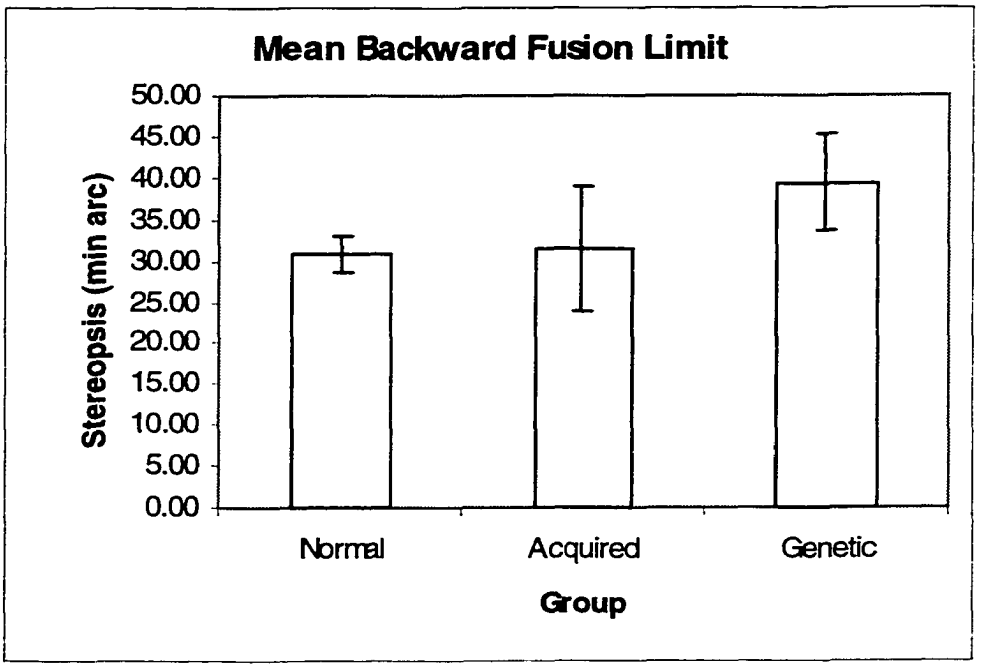


Figure 57. Mean backward fusion threshold for each group. Error bars equal +/- 1 SEM.

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0.5 min arc; see Figure 57). Neither the limits of fusion in the forward direction nor the limits of fusion in the backward direction were found to give significant differences between groups ($p > 0.05$) according to separate one-way ANOVAs and Kruskal-Wallis tests performed on the ranked data for each measure.

Monocular hyperacuities. Of the three groups of observers, the genetic color-deficients were the most sensitive on monocular hyperacuity tasks, having lower vernier acuity thresholds than both the color-normals and the acquired color-deficients. The mean amount of minimal vertical misalignment detectable between two bars was smallest for genetic color-deficient observers, and increased in nearly equal increments of about 0.08 min arc to the greatest disparities required by acquired color-deficients. A bar graph depicting these values is shown in Figure 58. (See Table B9 and Figure B101 in Appendix B for threshold data and a graph of the group medians, respectively.) These differences were not found to be significant at the 0.05 level when using either a one-way ANOVA or a Kruskal-Wallis test.

Motion sensitivity. The genetics displayed a mean minimum motion sensitivity which was superior to both the normals' and the acquireds', but were found to be no different among maximum motion sensitivities. The groups' results are compared in Figures 59 and 60 (see also Table B10 and Figures B102 and B103 in Appendix B). While each participant was run through this portion of the battery, reliable thresholds were not necessarily found on each of the measures for every observer.

The mean minimum amount of displacement of an array of random dots presented in succession needed to accurately describe the direction of motion was smallest for

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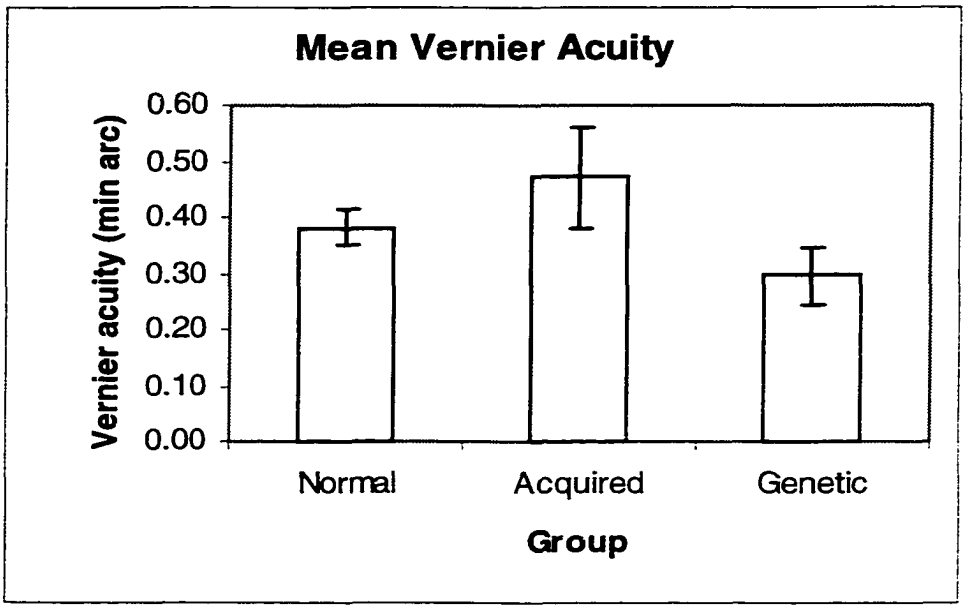


Figure 58. Mean vernier acuity for each group. Error bars equal +/- 1 SEM.

genetics (n = 5), greater (by a factor of about 3.5) for normals (n = 28), and largest (by a factor of about 5.3) for acquireds (n = 6; see Figure 59).

As depicted in Figure 60, the mean maximal displacement still allowing perceived motion and directional discrimination was nearly identical between the color-normal (n = 20) and genetic color-deficient (n = 5) groups, while that of the acquired color-deficient group (n = 5) was on the order of 4 min arc less. Neither of these measures was found to result in significant differences between groups, as shown by a one-way ANOVA, and as judged from the results of a Kruskal-Wallis test ($p > 0.05$).

Ocular and dermal spectral reflectivity. There were no statistically significant differences found between groups on measures of ocular and dermal spectral reflectivity. These results are more fully described in Appendix B.

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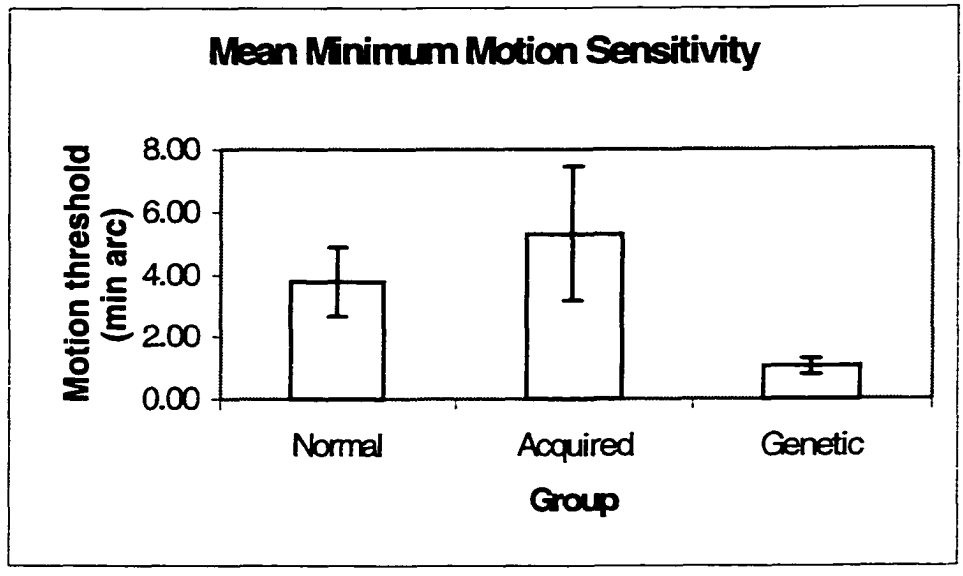


Figure 59. Mean minimum motion sensitivity threshold for each group. Error bars equal +/- 1 SEM.

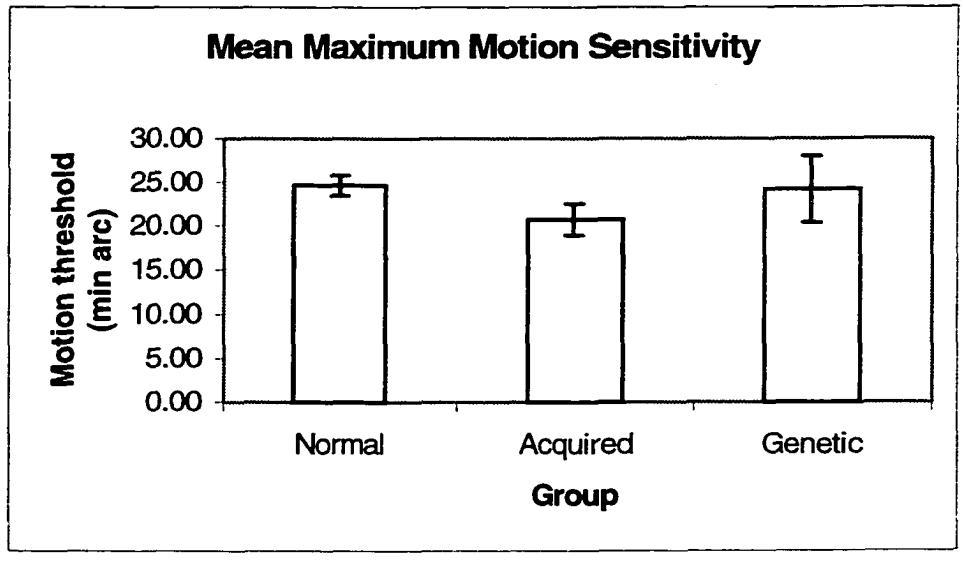


Figure 60. Mean maximum motion sensitivity threshold for each group. Error bars equal +/- 1 SEM.

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## Discussion

Our results provide a fresh, new outlook on several areas of focus in the field of color vision, as obtained from the exploration of color vision deficiencies. The goal of the current study was to compare color-normal and color-deficient observers across an array of very precisely measured general "achromatic" and chromatic visual functions. Such a goal has, to date, not previously been systematically attempted. Based on correlations and the clustering of functions showing differences between these populations, results from this investigation allow further insight not only into the complexities of color vision deficiencies, but also into the circuitry and wiring, functional specializations, and interactions of the visual pathways, thus leading to a better understanding of how we are able to process visual information overall. Despite having limited sample sizes, our study's findings reveal definite trends starting to emerge, which, we believe, not only warrant further explorations, but also, we hope, will pique the interests of others to pursue further, and, above all, keep this area of research fertile for many years to come.

The major findings of our study may be summarized as follows. For every visual function tested which required fine resolution, we found an enhanced sensitivity in genetic color-deficient observers. In this section, we begin with an in-depth discussion of the various intricacies associated with color vision deficiencies, their diagnosis and classification, and resulting implications. Following this, we offer several possible theories to explain genetics' enhanced sensitivities. These include models which address the observed between-group differences as a function of group membership (treating each member of a given group as possessing the same, identical features), as well as ones which focus on a more individual basis (members of a group still share common

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characteristics, but within-group differences may also prevail, perhaps as a function of type or degree of color deficiency). In the case of the former, enhanced sensitivities in color-deficient observers may result from a reduction of chromatic antagonism or from other compensatory mechanisms as a result of an altered P pathway. For the latter, we postulate that differences in the wirings of the visual pathways in color-deficients may lead to differences in the distribution of available resources, interactions between the two pathways, and their consequent effects on the processing of information. These models may occur either singly or in combination, as there are no clear boundaries regarding their influence on the visual system.

While our small samples prevent us from making conclusions regarding the general population as a whole, one important aspect that became quite clear during testing is the overall diversity of human vision. That is, while sample size may be suggested as a reason why differences between groups did (or did not) appear for a given measure or measures, the question of whether our samples are representative of their respective populations is equally as important. This, alone, is a justification for the continuation of study of the present topic.

A recent paper by Roorda and Williams (1999) shows how two seemingly equivalent normal trichromats on the surface can be quite different when viewed at the level of retinal circuitry. In this particular instance, one individual had an LWS to MWS cone ratio some three times greater than the other. In addition, it was revealed that retinal patches equal to or greater than five min arc containing only one of the two longer wavelength cones are quite common. Additional implications of these findings will be discussed later. For the moment, however, the need not only for larger samples is

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apparent (thus allowing for the full range of potential values on a given measure to be determined), but also the need for several measurements to be taken (preferably at different viewing points, to avoid an inadvertent sampling bias) seems clear. This may lead to the vexing problem of having to sacrifice quantity (i.e., the testing of large quantities of individuals in a short period of time) for quality (i.e., measurement precision and accuracy). With such diversity, however, both between and within individuals, the initial choice seems clear.

Before delving into a discussion of the results, a few comments are in order. First, we openly admit that it was probably a mistake to have lumped together all of the color-deficient observers into their respective groups (i.e., acquired and genetic). Given the diversity of type and severity of deficits, both in our subsamples and in general, we know quite well that the visual and perceptual experiences of these individuals are far from identical, and, consequently, should not be treated as such. However, considering the scope of the study (and, again, the limited sample sizes), we must necessarily restrict ourselves in the discussion of individual differences among these observers (even though such a discussion would be the right thing to do).

The assessment of acquired color-deficiencies in the overall scheme of things is problematic. While most of our reasoning as to why one group outperformed another for a given measure is going to be speculative (given the uniqueness of the current study in both approach and content), the interpretation of acquireds' performances among these is exceptionally difficult. The acquired group is probably more heterogeneous than either of the other groups. In some cases, it is pure speculation that an individual even has an acquired deficit (and, thus, belongs in this group), while in others, the degree (and

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severity) of the deficiency is unclear. Probably the most important aspect to consider, which was briefly discussed earlier, is compensatory mechanisms. The severity of a color vision deficiency is often determined by means of a standard test. Yet, an equally (if not more) compelling aspect needed for full interpretation of a given individual's color vision is how the deficit affects them personally. Is it a major impediment in their daily routine, or have they developed ways to cope with their problem to the point where they are no longer consciously aware of its presence? This is most relevant in the case of acquired deficiencies limited to one eye, because the unaffected eye should easily be able to compensate for the qualities and properties that the affected eye lacks.

The acquired group is far from uniform regarding the cause of an individual's deficiency (ranging from not knowing that they even had a deficit until the battery revealed this, to Leber's idiopathic stellate neuroretinitis), the age of onset, and (most likely) members' ability to cope with the affliction. It has been shown that neural plasticity exists in somatosensory systems for a variety of species, sometimes in great capacity depending on the location and extent of the injury (see, e.g., Harris, 1980; Merzenich, et al., 1984). Recent studies (e.g., Cicinelli, Traversa, & Rossini, 1997; B. B. Johansson, 2000; Nudo & Friel, 1999; Rossini, et al., 1998) have found this to occur in patients recovering from cerebrovascular accidents, and it is not difficult to conceive of a similar situation for our purposes, in some capacity, provided that critical periods have a minimal impact. As alluded to above, the variability of how each individual compensated on both the cellular and behavioral levels is important in determining whether they have learned to use their affected eye as often as, or in conjunction with, their unaffected eye, or whether they rely completely on their unaffected eye. In the latter case, by testing their

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affected eye in isolation, measures obtained on these monocular tests may well be different from the other eye or when such stimuli are viewed binocularly (i.e., in realistic settings). At the very least, given the little that we do know about each individual, and the acquired group as a whole, speculating on and discussing neural wiring for these observers is a moot point, as there are separate mechanisms operating at the sensory and perceptual levels, with one influencing the responses of the other to an unknown degree. With this in mind, treating genetic color-deficient observers as all having the same or similar wiring patterns may, similarly, be quite presumptive.

Color perception

While color-screening was predominantly intended for diagnostic purposes and the appropriate classification of study participants into their respective groups for comparisons of the other functions studied, some interesting and intriguing findings arose that deserve some attention, and from this, additional questions may be posed.

First, the need for multiple color-screening tools was confirmed and should be underscored. Simply put, several of our color-deficient observers, while diagnosed as having a deficit, were given different diagnoses by one or more of the tests. While the anomaloscope is the only "fool-proof" test (the results of which we used as our final diagnoses; see below for further discussion), the inherent problems of color vision testing are more far-reaching. Having a test such as the anomaloscope for breaking ties created from several different standard tests does not resolve these issues. The limited availability of anomaloscopes, coupled with the more extensive training needed for administration and the accurate interpretation of results compared to other color-screening tests, continue

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to be major drawbacks of this instrument in gaining a permanent position in most testing facilities. The problem of misclassification by standard tests affects all potential observers, color-deficient and color-normal, alike. Proper administration and the possible need for reevaluation of criterion cutoff points as to what does and does not constitute having a color vision deficiency (even in its mildest form) can have great consequences regarding the total number of individuals counted as such.

While a test such as the Dvorine plates or Farnsworth D-15 may seem quite easy to administer, score, and even interpret (thanks to the instruction manual being written in a non-technical fashion), proper lighting, viewing distance, and participant behaviors throughout the testing session must be emphasized. Based on Roorda and Williams's (1999) findings, it is not too difficult to envision how improper administration of this, or any other color-screening test, can have negative consequences. Given such patches of a single type of cone on the retina, stimulus field size and position have important implications regarding cone sampling. This is by no means a new concept. For years, researchers have been describing the existence of small-field tritanopia (e.g., Abramov & Gordon, 1977; J. Gordon & Abramov, 1977), how normal trichromats are comparatively "colorblind" (i.e., mimicking dichromacy) in certain regions of the color spectrum (e.g., Levine & Shefner, 1991), and how dichromats behave as trichromats at certain retinal eccentricities (e.g., Pokorny, et al., 1979). Thus, in the designing of new tests and test norms, the underestimation or overestimation of the percentage of individuals in the general population with color deficiencies can be greatly affected, especially with limited point sampling as a basis for diagnosis. In a similar vein, pass/fail criteria need to be investigated closely. Such boundaries need to be unambiguous, permit a high degree of

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certainty that a correct classification is made when properly used, and that these rules are strictly adhered to, so that individuals with a genuine deficit are not mistakenly diagnosed (and vice versa).

To take another example, consider the astounding persuasive capabilities that crayon manufacturers have on today's youth and society. Every crayon found in the box has a wrap-around label, which includes the (supposed) hue or color name. There are probably many individuals who are first exposed to a sampling of the wide range of names of the colors, hues, and shades that occur in nature in this very way. Such a person has no reason to believe that the name on the label is incorrect (i.e., mislabeled), because they do not have anything to compare it to. Children may, thus, be setting up exemplars and prototypes of color categories based on this. Great perceptual differences may exist from one person to another when looking at a "red" crayon, but by taking the color name at face value and calling whatever hue or shade (or even intensity) that this crayon looks as "red" (since everyone else does; i.e., demonstrating appropriate color-naming), many color-deficient individuals can go undetected.

Of course, the majority of undetected color-deficients are most likely only mildly impaired, and have gone so long without knowing of their affliction as a result of their daily activities not requiring very precise color discrimination tasks. According to Verriest (1979), occupations, even in business and industry, for which normal color vision is non-essential are wide-ranging. These include architecture, horticulture, landscaping, confectionery, and teaching. Others requiring normal color vision, but in which instrumentation or the assistance of colleagues can help out with color problems, include electronics, cartography, metallurgy, and medicine. Those which require unaided

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normal color vision include railway, marine, and aviation workers (from which accounts of late nineteenth century disasters caused by color-deficient employees dot the field's early literature), as well as a host of sorting of matters (e.g., fruits and vegetables). However, the sorting of certain materials is not problematic if the grading is based on a color or colors that the individual does not have a disadvantage with.

The underestimation of people with color deficiencies may stem from the fact that, from an early age, we are taught to listen and are inclined to believe that what we are told (especially by family members) is true. As a result, we have no need to question anything. Those of us who are fortunate enough to break out of this mold become scientists later in life, learning to accept nothing and to question everything!

Furthermore, considering the amount of variability among color-normals in color matching (see Lindsey, et al., 1992) and in hue naming, what really constitutes the perceptual boundary of what is normal and what is deficient? How many females (both in our sample and in general) fall under the classification of heterozygous carriers of color deficiencies? Are the group means (and comparisons between group means) in the present study biased from the inclusion of these observers under the color-normal category? Certainly the discovery of an underestimation of deficiencies in the general population will necessitate a need to follow up on this group of individuals. It is quite likely that this group is underestimated in size because, while color deficits in heterozygous individuals have been noted to span the entire range of severity, most fall in the very mild region. Since simple anomalous observers are less often aware of their deficiencies, carriers are less often aware of deficiencies in relatives (Nagy, MacLeod, Heyneman, & Eisner, 1981) and have no reason to believe their own color vision would

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be compromised. Methods of testing for purposes of identifying such observers will need to be sufficient for signs of subtle deficits while controlling for extraneous variables (e.g., rod signals, differences in retinal mosaicism; see Nagy, et al., 1981). Should this group be considered an entirely separate entity, or be integrated into existing color-deficient nomenclature (based on the typology most closely represented)? Will existing color-screening test scoring methods need to be reworked to allow for these observers' proper identification?

Along with the prevalence ratios of color deficiency in various regions and cultures (e.g. Birch, 1993; Jorgensen, Deeb, & Motulsky, 1990), similar statistics have been calculated for type and severity of deficit. While it was mentioned earlier that anomalous trichromacy occurs most often (compared to dichromacy and achromacy), Gouras (1991; see also Nathans, et al., 1986a) describes deuteranomaly as the most common defect in males, with a rate of incidence of 5%. Protanomaly and protanopia both occur at a rate of 1.3% in this population, followed closely by deuteranopia at 1.2%. Congenital tritanomaly and tritanopia both occur at the very infrequent rate of 0.001% in males. Given the somewhat marked differences in frequency, it would seem that finding a protanomalous individual would be hard to come by, and that any group of anomalous observers would be overwhelmingly of the deutan variety (considering that such a descriptor applies to some 70-80% of all deficient observers). As per the results of our Rayleigh matching experiment, we found the exact opposite of this prediction. Of our 5 genetic color-deficient observers, 4 (constituting 80% of the sample) were classified as protanomalous, and only 1 was deuteranomalous. As described above, the anomaloscope is the only "fool-proof" color test known to exist, in that it is most resistant to diagnosing

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false-positives and does not pass anyone with even the mildest of color deficiencies. The obvious question is whether the frequency statistics are incorrect and need updating, or whether our results were merely coincidental. Our previous argument regarding the general underestimation of people with color vision deficiencies, at the very least, lends credence to the possibility that our findings are not of pure coincidence. The use of several screening measures and the evaluation and inclusion of subtle to mildly affected individuals not presently accounted for could most certainly skew the distributions toward protanopic deficiencies being more common. A resolution to this will certainly come with the collection of additional data.

With an equal rate of occurrence, it is also interesting that all of the protanopic observers were anomalous trichromats, as opposed to all dichromats or a combination of the two. Some time ago it was postulated that dichromacy is not unique, but rather an extreme form of anomalous trichromacy (i.e., these individuals were found to possess a form of residual trichromacy; see Scheibner & Boynton, 1968). More recently, Frome, Piantanida, and Kelly (1982) showed, psychophysically, that dichromats possess more than one long wavelength sensitive receptor capable of detecting flicker of red and green light. Each of six dichromats was unable to completely eliminate perceived flicker from alternating red and green, flickering, sinusoidal counterphase lights, either by adjustment of amplitude of the lights, or by varying the phases of the lights once minimum flicker had been reached. The authors concluded that at least some dichromats have three cone types, anomalous cones, spectrally similar to those of the corresponding simple anomalous trichromat, but being relatively insensitive to both temporal and spectral variations compared to normal cones. Sanocki, Teller, and Deeb (1997) not only

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demonstrated that some dichromats were able to make chromatic discriminations using large test fields, but showed, also, that by having polymorphic versions of the same pigment in two cone types, a moderate degree of chromatic discrimination is possible provided there are differences in the pigments' optical densities.

Color discrimination

The arguments made for the brief color-screening tests similarly apply to the FM 100-Hue test. The only additional comments that need to be made center around its scoring and diagnostic efficiency. It was mentioned earlier that, for our study, in a number of instances, diagnostic classification was not straightforward, but rather quite subjective. Certain individuals' errors presented substantial spikes along one or more axes that did not parallel any of the standard (i.e., deutan, protan, tritan) deficiency axes. Furthermore, when considering the results of the Fourier analysis performed on these data, a wide array of different combinations of significant harmonics was found among the color-deficient observers, even between individuals with equivalent diagnoses. Is it a mistake to force an existing label on something that doesn't quite meet the necessary criteria (i.e., how close is "close")? By doing so, are we not masking what is apparently something more complex than the so-called standard, something that can only be revealed through further exploration? The current classification system may, thus, be too limited in allowing us to more fully understand color vision and its mechanisms.

Hue- and saturation-scaling

Hue-scaling is far from a novel concept, even when testing color-deficient observers. V. C. Smith, Pokorny, and Swartley (1973) investigated hue-scaling in

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deuteranomalous observers for wavelengths between 405 and 695 nm in 50-nm intervals at a luminance of 25 td. A unique blue point was recorded in only one of the six observers tested; unique green was found to occur in the range of 490-510 nm; and all observers showed a red response function (though it had a shallower slope and was displaced toward longer wavelengths compared to normal controls). Unique yellow is not mentioned, and examination of their figures shows this to be non-existent (although the use of the term "yellow" increased under conditions of increased luminance [250 and 1250 td]). Romeskie (1978) used a forced-choice, double randomly alternating staircase procedure to obtain wavelength estimates of unique blue, green, and yellow in a group of four protanomalous and two deuteranomalous observers. Unique blue (found for all observers) was generally at a slightly shorter wavelength than normals' for the protanomals (around 474 nm), while deuteranomals showed points at longer wavelengths (mean of 486 nm). Unique green followed this same pattern, although protanomalous observers were more variable, overlapping with the normals. Unique yellow was determined for two of the protanomals and only one deuteranomal, also maintaining the above trend.

In our sample, a unique blue point was determined in the one deuteranomalous observer, and in three of the four protanomalous observers (see Table B1 and Figures B2 and B3 in Appendix B). The unique blue point occurred at 455 nm for the deuteranomal, while the range for the protanomals spanned 72 nm, including 455 nm (see Figures B2 and B3). For diagnostic purposes, unique green was no more informative, the protanomalous observers giving variations of up to 110 nm compared to one another, including the deuteranomalous observer's 500 nm point (see Figures B4 and B5). Three

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of the four protanomals gave a unique yellow point below that of the deuteranomal (see Figures B6 and B7), while the deuteranomalous observer gave the highest unique red point of the four genetics from whom this value could be determined (see Figures B8 and B9). It would be difficult to classify an individual based on unique hues alone, given the large degree of interobserver variability (Table B1, SEMs). For example, one protanomalous observer's unique green, yellow, and red points occurred within a span of 10 nm, the latter two separated by just one nm. The unique red point, which for our genetic color-deficient sample was considerably lower in both mean and median compared to color-normals, again is biased not only by the weighting of the number of protan versus deutan observers (which shifted it toward shorter wavelengths due to the presence, in protans, of anomalous LWS cones more similar to their MWS cone), but also because of the lack of a measurable unique red point in many observers, in general (see Method section). V. C. Smith, et al. (1973) note that an individual's chromatic discrimination ability, use of luminance cues, and low apparent saturation of small brief stimuli must be considered in order to more fully understand these observer's hue-scaling functions. Romeskie (1978) indicates that her data were drawn from relatively few observations, and the reported data are only rough approximations. The fact that our genetic color-deficient subjects showed abnormal hue-scaling functions (e.g., extended and overlapping red and green functions; see Figure 14), when coupled with their performances on the other color-screening measures (the aforementioned limitations aside), supported our preliminary diagnostic group classification, even with such highly variable unique hues.

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Anomaloscopy

The building of an anomaloscope was a big undertaking, and an obvious question is whether our system worked up to specification. The initial impression is quite positive, although there is clearly a need for a greater number of observers to be run in order to verify the machine's validity and reliability. Locating a Nagel anomaloscope for use in testing some of the observers run on our system may prove helpful in comparing diagnostic impressions, discrepancies between which, once corrected for, may allow for a more versatile and powerful instrument, as well.

HFP. Expanding the number of points measured during HFP will give a better diagnostic indication of deficiency which can be cross-checked using the Rayleigh match, and also help in resolving a long standing debate regarding spectral luminous efficiency functions.

In 1958, Heath found that when comparing the luminosity curves (by way of critical flicker fusion frequencies; CFF) of normal and dichromatic observers, for wavelengths greater than 520 nm, protanopes showed a loss of luminosity, while deuteranopes showed supernormal luminosities (luminosity gain) at these same wavelengths. (CFF is the frequency at which a flickering light appears steady. This is measured psychophysically by having the observer increase or decrease the flicker rate in small steps while the test light remains at a fixed luminance. This is in contrast to HFP, where, as described earlier, two alternating lights are presented and the observer adjusts the luminance of one of the lights to minimize or eliminate the perceived flicker. HFP measures are dependent on both the spectral composition and the similarity of the lights used.) C. H. Graham and Hsia (1960) point out that the method employed by Heath may

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have provided conditions that gave rise to special wavelength effects. Using the same method, they found comparable heightened sensitivities in one protanomalous observer and in one deuteranomalous observer, but had previously reported losses in deuteranopes. The lack of uniqueness of sensitivity gains in deuteranopes and the presenting of evidence disconfirming the idea that all deuteranopes show this effect led C. H. Graham and Hsia to propose that Heath try correlating his data with that obtained by other methods. Heath (1960) pointed out that how one defines "luminosity" might underlie whether deuteranopes show losses or gains in luminosity. Furthermore, he built up the CFF technique as being more robust than other methods previously used. Heath's findings were confirmed in the early 1960's by Collins (see Pokorny & Smith, 1972). A study by Pokorny and Smith (1972), however, yielded results in favor of C. H. Graham and Hsia. Not only did they show how the method of CFF does, in fact, measure special wavelength effects, they also demonstrated that several other methods (viz., absolute threshold, HFP, and brightness matching) can provide the same estimates of luminosity. All deuteranopes showed sensitivity losses for wavelengths shorter than 570 nm using these latter methods, while, in addition, these individuals required less energy than normals or protanopes to reach a criterion CFF (i.e., they were more sensitive, requiring less luminance at a given flicker rate to reach threshold). Lennie (1984) found somewhat conflicting results with Pokorny and Smith. While the deuteranopes' results between studies were similar (as far as CFFs; e.g., maximum difference occurring at high illuminations was 58 vs. 53 Hz), protanopes and normal observers performed better in Lennie's (1984) experiment at both levels of illumination, with negligible differences compared to deuteranopes. Differences in apparatus, stimulus size, and LWS and MWS cones (i.e., elements focusing around

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potential foveal cone mosaicism variation, receptive field dimensions, and spatial frequency sensitivities) were discussed as factors needing to be accounted for in the comparison of these studies' results. Lutze, Smith, and Pokorny (1989), in acknowledging the differences in spatial structure of the stimulus field as a possible cause of differential sensitivity, altered surround condition and field size to see what effects these components might have on CFF. They found that, across all conditions, deuteranopes tended to show a higher asymptotic CFF than protanopes did (i.e., as in the earlier study, deuteranopes were more sensitive, showing higher CFFs at a given luminance level, irrespective of field size). Rather than spatial characteristics of the test stimuli, differences in cone mosaic densities was hypothesized as the underlying source of the main differences observed. The finding of the need of a cone density ratio of at least 9 to 1 for deuteranopes compared to protanopes to account for the observed differences, however, is not even close in value to the standard ratios given for color-normals. Furthermore, the resulting cone spacing for such a density ratio to occur necessarily implies a difference in visual acuities between protanopes and deuteranopes, which has not been reported (Lutze, et al., 1989). To this extent, the issue remains unresolved.

With Pokorny and Smith's (1972) finding that HFP produces comparable luminosity estimates, we are able to make a comparison of our results to the above studies. As was shown in Figure 18, we took measures at two points (534 and 662 nm) with one of the lights (596 nm) always at a fixed luminance. While the deutan in our sample (observer GB96) needed a considerably higher luminance for green compared to red to minimize the flicker (indicating a sensitivity loss for shorter wavelengths), and three of the four protans required the reverse (indicating a sensitivity loss for longer

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wavelengths), no general method could be sought for positive identification of color deficiency from this measure alone. The other protan (observer HD96, who was considered a deutan by the color-screening measures, but a protan by the anomaloscope) needed a higher intensity of green, as well, though not to the extent that the deutan did. This supports C. H. Graham and Hsia's (1960) finding that heightened sensitivities in protanomalous individuals are possible, as well. Pokorny, et al. (1979) note that short-wavelength luminosity loss in deuteranopes is not evident in all observers, that there is considerable interobserver variability, and that such sensitivity differences can probably be ascribed to interobserver variation in macular pigment. Based on our findings, perhaps this may also apply to protanopes. Interestingly, in our study, some normals also needed a higher intensity green light (in one instance, more intense than the deutan did). Measurements at additional wavelengths may allow us to gain further insight into resolving this issue.

Rayleigh matching. The Rayleigh match, in particular, the range, range midpoint, and derived anomalous quotient, provide for the main thrust of anomaloscopy. The combination permits for not only a diagnosis of color deficiency by type, but also by severity.

As the composition of our genetic color-deficient group indicates, one member was diagnosed as deuteranomalous, while the other four were classified as being protanomalous. Such diagnoses could be based, alone, on the mean green to red luminance ratios required by these individuals, as there was a definite bimodal distribution deviating from the normals' in both directions (i.e., shifts toward needing considerably more green or red to make a match; compare genetic group means and

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medians in Table 4; see Figures 19 and B15). The acquired group, again, was quite similar to the normals across each of these measures (Table 4 and Figures 19-22 and B15-B18), as would be expected if these observer's deficits were of a tritan nature, since LWS and MWS cones should be relatively unaffected in these individuals. Given the size of our genetic group, however, attempting to label the degree of severity for each observer was somewhat more difficult. With little in the way of comparison (only the 5 observers against whom we were trying to properly diagnose in the first place), we needed to contend with the problems of differentiating between what constitutes "simple" versus "extreme" anomalous trichromacy, where the boundaries between such subcategories lie, and how gradual or abrupt these transitions may be. It is important to note that not all of the previous anomaloscope-based studies differentiated between simple and extreme anomalous trichromats, and the definition of what constitutes extreme anomalous trichromacy leaves something to be desired. As defined by Franceschetti, the extreme anomalous trichromat, again, is an observer with a wide matching range that includes the normal match, the match of the corresponding simple anomalous trichromat, and perhaps one of the primaries (Pokorny, et al., 1979). Diagrams containing matching ranges (along with these descriptors) from large studies done by Pickford and by Lakowski (found in numerous sources; see, e.g., Hurvich, 1973; Pokorny, et al., 1979) do not aid in rectifying this ambiguity. (As was mentioned earlier, even to compare our ranges and AQs with those of another study would not be helpful, given the different apparatus, conditions, and populations from which the data were accumulated.) First, it goes without saying that it is quite nonsensical to define one ambiguous term in terms of another ambiguous term (in this case, "simple," and where such an observer's range is purportedly located). It does

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not seem correct to give a classification of "simple" to an individual whose matching range is much narrower than an "extreme" observer's, yet who has a midpoint and range more proximal to a primary (and which, in some cases, do not even overlap). In addition, how can one consider an observer who is able to make a match to one of the primaries as having anything less than a severe deficit, regardless of the size of his matching range? In our genetic color-deficient group, for example, one of the protanomalous trichromats was able to match the standard to the red primary. However, his match range did not extend to the normal match (or come even close to it, for that matter). It is hard to believe that a narrow range (and, hence, "simple" anomalous trichromacy) should be equated with only mild forms of deficiencies without considering its location with respect to the normal match. In using the existing paradigm, however, a different protanomalous observer from our sample would be considered "extreme," the others and the deuteranomal, "simple" anomalous trichromats.

Each genetic color-deficient observer was correctly described as being an anomalous trichromat (as opposed to a dichromat) based on having an anomalous quotient distinct enough from the normal group's mean and by failing to match to each primary alone. With larger group sizes, clearer normative samples will arise, allowing for relatively definitive limits to define color category group membership based on AQs.

It was rather surprising to find that under chromatic adaptation, every participant's Rayleigh match broke down! This would indicate that every observer run in our study has multiple cones that are sensitive to longer wavelengths. While the possibility exists, such a probability would be quite small. We decided to investigate this further by closely comparing our experimental design with that of J. Neitz, et al. (1991).

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Of immediate importance is the need to emphasize that J. Neitz, et al. (1991) found this shift in color matches to occur in males with normal color vision. Immediately we can reduce our sample size for direct comparison down to seven observers. This, alone, would lead to a greater probability of such an occurrence (7 vs. 40), but further information is needed. J. Neitz, et al. (1991) found that 680 nm adapting lights produced on the order of 0.05 log R/G shifts in the color matches of some males with normal color vision. The range of color shifts for the color-normal male observers in our study ranged from 0.13 to 0.4 log G/R units, all toward shorter wavelengths (i.e., more green and less red were needed for Rayleigh matches under adaptation). Known methodological and procedural differences between the two studies (with potential impact on the results ranging from trivial to possibly somewhat more substantial) include field size, viewing duration of each stimulus, retinal illuminance, and stimulus wavelength. Other, more questionable, differences include the type of optical system used, the exact procedure used to obtain color matches, and the overall sample size (from which we could derive the percentage of observers in their study exhibiting the color shifts). Unfortunately, J. Neitz, et al. (1991) was published in abstract form. We have no clear way of answering these questions or knowing what effects these elements may have on other factors or on the results, themselves.

While our levels of retinal illuminance were well below that of J. Neitz, et al. (1991), different degrees of adaptation (and bleaching) between our observers and theirs may have resulted from viewing duration and image stabilization (or lack thereof), among others. Was the choice of adapting wavelengths sufficient to suppress long wavelength cone signals? Given that a different match resulted in each of our observers, there clearly

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was an adaptation effect. Whether there remained some residual signaling of adequate magnitude able to contribute to the matching of certain individuals in the Neitz, et al. study, thus altering observers' matches, but not significantly so, is still a possibility. With several clusters of LWS cones with different peak wavelengths known to exist (perhaps a common one was found among our sample of observers), can we necessarily assume that an adapting wavelength will suppress these cones equally well between individuals? We might, then, restate the initial question as follows. Was the choice of adapting wavelengths sufficient to suppress all long wavelength cone signals equally well for all observers? This is all merely speculative at this point.

To summarize, various new advancements in DNA-hybridization techniques have given researchers further insight into the complexities of human vision. It has already been shown (e.g., Drummond-Borg, Deeb, & Motulsky, 1989; Nathans, 1987; Nathans, et al., 1986a,b; J. Neitz & Jacobs, 1986, 1990; J. Neitz, M. Neitz, & Jacobs, 1987, 1991, 1993; J. Neitz, M. Neitz, & Kainz, 1996; M. Neitz & J. Neitz, 1995; Piantanida, 1988; Winderickx, Battisti, Motulsky, & Deeb, 1992a; Winderickx, et al., 1992b) how highly variable the genetic code is for both normal and color-deficient observers. While this has helped in answering many questions, it has also led to the creation of others. Will the discovery of polymorphism in the longer wavelength sensitive cones hurt the diagnostic accuracy that we have come to expect from screening instruments such as the anomaloscope? For that matter, will it put an end to anomaloscopy as the outright choice method of diagnosing? While Nathans, et al. (1986a,b) first indicated the possibility that much of the nature of the color vision of anomalous observers could be uncovered on the molecular level, such as by knowing the location of the point of cross-over in hybrid

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genes on a given exon, J. Neitz, et al. (1996) have shown, by similar methods, that it is also possible to determine the severity of the defect simply from a drop of blood. In short, is DNA analysis the future of color vision deficiency research? Given that standard screening methods are insufficient in addressing deficiencies that are clearly more complex in nature (and which may be more common) than the textbook varieties these tests were designed for, perhaps it should be. In the end, only time will tell.

Spatio-temporal contrast sensitivity

To reiterate from the Introduction, Livingstone and Hubel (1987b, 1988), in comparing the properties of M and P cells, found M cells to have larger receptive fields, produce more transient responses, have faster signal conduction, have better contrast sensitivity, and have lower spatial resolution than P cells. At higher levels, these characteristics are maintained, such that the M pathway, in general, is found to have high temporal resolution, high contrast sensitivity, and relatively low spatial resolution, while the P pathway has low temporal resolution, lower contrast sensitivity (for contrasts less than 10-15%), and high spatial resolution. Hubel and Livingstone (1990), in further work with the macaque monkey, found that while contrast sensitivity led to the ability to clearly distinguish between M and P cells at the level of the LGN, this was not as simple a matter at the level of the cortex (especially in layers 2 and 3). Contrast sensitivities were found to be lower in layers 2 and 3 than in magnorecipient layers, but distinctly higher than in parvocellular geniculate cells. Possible interpretations of this finding, as postulated by these authors, include a magnocellular contribution or a result of intracortical processing,

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whereby convergence of geniculate inputs onto single cells might allow enough signal averaging to enhance contrast sensitivity (Hubel & Livingstone, 1990).

Based on the aforementioned tendencies of these cells' responses, we can hypothesize that in individuals with an altered parvocellular system, contrast sensitivity functions should show differences at high spatial and low temporal frequencies compared to other observers (see, e.g., Troscianko, et al., 1996).

Our results, as described earlier, show a somewhat different pattern than what was expected. For the entire spatio-temporal domain which was tested, we found that genetic color-deficient observers had higher contrast sensitivities (lower contrast thresholds) compared to color-normals, with only a few exceptions (see Figure 53). The color-normal observers fared slightly better when detecting 0.6 (group median only) and 1 cpd (group mean only) gratings modulated at 1 Hz, 1 and 2 cpd gratings modulated at 4 Hz (group means only), and 1 cpd gratings modulated at 24 Hz (group mean only). Somewhat larger discrepancies between these two groups occurred in thresholds obtained for 0.6 and 1 cpd gratings modulated at 4 Hz (group medians only). Collectively, it may be said that genetics performed more poorly in detecting gratings of low spatial frequencies at the lowest and highest temporal frequencies. This is remarkable since these spatial and temporal frequencies have traditionally been noted to be M pathway-controlled, and, thus, should be relatively unaffected in color-deficient observers. Furthermore, the results oppose the hypothesized between-group differences predicted for the frequencies often cited as P pathway-controlled (see above). Perhaps what was most unusual about our results was the finding that, of the spatio-temporal combinations in which genetic observers had better contrast sensitivities compared to normals, many were substantially

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better, almost to the point where we might suggest that these genetics exhibited a form of supersensitivity with respect to these stimuli. The same or similar mechanisms may be responsible for each of our exceptional findings.

There are several avenues that can be taken in trying to explain our results. As described in the introductory section on color, the possibility of genetic observers having a sort of sensorial augmentation on a local level as a result of other sensory loss, perhaps through compensatory mechanisms, is one way in which our findings can be accounted for. While such a proposal may seem to lack scientific credibility, recent research has espoused this idea. Roorda and Williams (1999), for example, explicitly state that as a result of having large retinal patches of a single cone type there is a necessary and inevitable tradeoff. This compromise concerns chromatic and achromatic functions, in that for high spatial frequency stimuli, what is lost regarding color variation (and discrimination) is gained in luminance variation, as a result of cortical neurons tuned to high spatial frequencies being more likely to be fed by contiguous cones of the same class. With anomalous observers having two longer wavelength sensitive cones that are more similar to one another than do color-normals, the lack of antagonistic chromatic signals from adjacent receptors (and patches of receptors) can conceivably lead to an even greater enhancement in sensitivity (through neural pooling) and spatial resolution limit (gain) at these frequencies.

Following this line of reasoning, Robson (1966) suggests that fall off in sensitivity at low spatial and temporal frequencies is the result of antagonism between signals from the center and surround of the cells' receptive fields, with a greater attenuation of signals from the surround (due to its size) at high spatial frequencies. From this, it is not difficult

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to envision that differences between our groups of observers may be the result of differences in the sizes of receptive field center and surround (i.e., degree of antagonism). Such differences can lead to properties and functions that are completely opposite to those of normals (e.g., less antagonism or more antagonism) depending on how differently the visual systems of anomalous observers are wired. Such differences may soon be able to be determined through the advancements in neuroimaging techniques discussed earlier.

The notion of visual processing channels, specifically those tuned and receptive to narrow intervals of spatial and temporal frequencies, has been around for many years and continues to receive attention, as various research teams are attempting to confirm these channels' existence and functional capabilities. Sekuler, et al. (1984; see also Billock & Harding, 1996) describe the human visual system as a small set of broadband neural filters with overlapping distributions of sensitivity. Based on our results, we may pose several questions regarding the underlying filter mechanisms and how they might apply toward our findings. For one, are the observed spatio-temporal differences between groups evident at this level of information processing?

The composition of our genetic group is quite diverse regarding type and severity of color deficit. It is intriguing, though, to consider that, despite these diagnostic differences and clear differences in individual contrast sensitivity functions between genetic observers (see Figures B48-B52), there is a definite clustering of sensitivities in this group. This clustering is indicative of low intragroup variability, and examination of the data shows that the increased sensitivity was not due to extreme values raising the group mean. Remarkably, this is also observed among the spatial and temporal

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frequencies in which anomalous observers were less sensitive compared to color-normals. Taken together, this suggests and supports the idea that a common mechanism exists among these color-deficient individuals.

Assuming that genetics have altered sensitivity filters compared to normals, are the differences in any way systematic with regard to diagnostic classification? As a first pass, we can say that there is nothing readily apparent. Our deuteranomalous observer, for example, was not found to be consistently more or less sensitive to any spatio-temporal frequency combination in comparison to our protanomalous observers. This is not to say that the rewiring scheme for the filters in color-deficients is identical from one person to the next. As depicted in Figures 48-52 and B43-B52, differences in contrast sensitivity functions exist both between and within groups. The question is whether or not this is (solely) due to noise in the system, and, if so, what the source of the noise is. Is it the result of a higher order mechanism (i.e., does it occur at a later stage in the stream of visual information processing), or is the noise introduced at the filter level itself? Perhaps the answer to the question of whether it is possible for there to be anatomical and/or physiological variations in frequency channels among the different members of the same group (e.g. especially genetics) will come from looking at the variations in normals. These observers displayed a greater range of sensitivity thresholds compared to the genetics. There may well be fewer alternative wiring possibilities in color-deficient observers than in color-normals. The observed differences in genetics may stem from slight gradations in an otherwise common mechanism found among these individuals.

The observed between group differences may not only be the result of different connections between the channels, but reflect differences in the filters themselves. Given

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that the color-deficient observers had poorer sensitivities compared to color-normals in several (not necessarily adjacent) places on the spatio-temporal surface tested, we might ask if the filters in anomalous observers show systematic deficits for only certain frequencies based on the rewiring scheme. Possible underlying causes range from filters that are damaged to those that are absent. At the same time, is it possible that two adjacent filters which in normal observers most likely share various properties as a result of their overlapping sensitivities, are not similarly receptive in color-deficient observers? This best addresses the observed degradation in sensitivities in genetics, as these individuals clearly were still able to see these frequency combinations. The degraded sensitivity may arise as a result of a wider span in the frequencies to which the adjacent receptors are maximally sensitive to, leading to a considerably reduced response signal to frequencies falling in between these peaks. Such a conception leads us to the following question. Could such supersensitivity as observed in our sample also result from the decreased signaling of proximal filters (either through damage, altered wiring, or complete absence), thus "requiring" intact filters (through a compensatory mechanism or reduced antagonism) to be better tuned toward their predestined frequencies? The wider space between adjacent filters' peak frequencies (i.e., less overlap) can allow for a greater response signal as a result of a given filter's resources not needing to be as largely apportioned. This may lead not only to enhanced sensitivities in the region of peak frequencies, but also to finer discrimination of frequencies in this area, as well. In the end, the direction and degree of correlations between filters are important in answering these questions.

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Similarly, could the type and/or degree of severity of a color vision deficiency alter the weightings of the various filters? If, for example, one particular filter's weighting is somehow forced to zero (or close to it), could adjacent spatially sensitive filters become more sensitive as a result of some (as of yet) unknown compensatory mechanism? Given the high variability of color vision deficits and the unknown wiring schemes, filters may not necessarily need to be adjacent to one another in order to be affected. This may be why we see differences not only in the contrast sensitivity curves, themselves, between genetic observers, but also the lack of relatively smooth functions across the spatio-temporal domain in certain individuals (i.e., perhaps not entirely due to noise). The weightings for a given filter or set of filters could also figure into whether a resultant contrast sensitivity curve for a given frequency is lowpass or bandpass. Such a proposition receives some backing from the work of Blakemore and Campbell (1969), if one considers the consequences of the different ways that the signals of narrow band detectors may be neurally pooled in the visual systems of color-deficient observers. Neural pooling is, of course, only one source of differentiation among observers' sensitivities. That is, neural pooling aside, differences between color-normal and genetic observers may be the result of differences in the sizes and/or handling capacities of these channel detectors in the latter group.

According to Peterzell, et al. (1993), there are several potential causes of individual differences in contrast sensitivity functions. First, there is measurement error. This, along with noise (from apparatus, method of measurement, random neural firing, etc.), may explain the slight differences that were observed between groups, but doubtfully the larger differences found between the genetic color-deficients and color-

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normals, for example. Paralleling our earlier discussion, these authors cite possible differences in the very spatial frequency-tuned mechanisms from which we are attempting to make measurements. Individual differences in the peak contrast sensitivity of a given spatial mechanism, for example, would necessarily be expected to cause some of the individual differences seen in contrast sensitivity functions.

One cannot exclude the possibility that our method of appraisal of contrast sensitivity has led, in part, to our unexpected results. Our stimulus field, for example, is quite large, and while observers are instructed to generally fixate in the center of the screen in addition to maintaining head stabilization, it is quite probable that foveal and peripheral receptors are being stimulated simultaneously in most individuals (see Peterzell, et al., 1993). Thus, the results may, for low to mid spatial frequencies, reflect a combination of the signals from both types of receptors. Depending upon whether or not these receptors are falling on channels that are similarly tuned, there may be more of a pronounced and enhanced sensitivity to a given frequency, or a deficit if this is not the case.

Owsley, et al. (1983; see also Hirsch & Curcio, 1989) stress the need to use techniques that bypass the optics of the eye, thus allowing for the measurement of truly neural influences. Can this be better controlled for in our existing system? Such modifications, while possible, would lead to only minimal differences compared to current measurements. For our purposes, having optically corrected each of our observers and subsequently running them on all sessions with best optical correction (a claim that no previous study can make), we believe that any potential for measurement error due to optical differences between individuals is sufficiently limited.

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Tulunay-Keesey (1972) cites an animal model in which the spatial contrast of a stimulus is found to be processed at the cross connections of the bipolar and horizontal cells, and the temporal qualities of the stimulus are processed at the level of the amacrine and ganglion cells. Restating a question posed earlier (in a slightly different context), are the differences that we observed between color-normals and genetic color-deficients similarly occurring at this level or at a post-receptoral stage of processing higher in the brain? While the possibility exists, at the present time we have no way of knowing this.

Burbeck (1981; see also Bowker & Tulunay-Keesey, 1983) discusses that thresholds for the detection of a stimulus, rather than both the detection and recognition of a stimulus (e.g., one requiring not only determining the presence of a grating, for example, but also some other property like the orientation of the grating), seem a simpler and more reasonable way of comparing spatial and temporal sensitivities of the visual system. Furthermore, detection takes a measurement at an earlier point in visual processing than does detection and recognition. Comparison of data between different studies must therefore take into consideration the stimulus and procedural method employed in order to determine if the same kinds of thresholds are being examined. Burbeck (1981) also notes that the spatio-temporal threshold surface for counterphase flickering gratings may be generated by responses of the pattern-detection mechanism alone.

Bowker and Tulunay-Keesey (1983), observing the effects of spatio-temporal adaptation on contrast sensitivity, found that sensitivity enhancements might result from local adaptation effects. Along the lines of Roorda and Williams's (1999) findings, we might then reason that if the retinas of color-deficient observers are substantially different

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than those of color-normals (i.e., containing much larger and more numerous patches of a single type of receptor), then even under non-adapted conditions, local retinal adaptation might be possible due to all cells being uniformly tuned (sensitive) to certain stimulus characteristics, thus yielding enhanced sensitivities.

Billock and Harding (1996) specify some of the types of interactions that can (and do) occur between spatio-temporal channels, including probability summation and cross-channel inhibition. Such interactions could be responsible for extreme values (maxima and minima) in color-deficient observers, which led to the observed differences between groups. Abramov, Gordon, Tannazzo, Delman, and Lawhorn (1999), using the battery apparatus and protocol, investigated the role of binocular summation in a subset of color-normal and color-anomalous observers. They report a clear binocular gain of sensitivity (by a factor of approximately two) across the entire spatio-temporal domain for all observers. In the present study, as described earlier and clearly shown in Figure 53, when analyzed on a group level, color-anomalous observers were found to have higher binocular spatial sensitivities for nearly all spatio-temporal frequency combinations compared to color-normals (on average by a factor of 1.2), resulting in the entire spatio-temporal surface being elevated in these individuals (see also Abramov, et al., 2000, for similar findings using a completely different test apparatus and method)!

Such non-trivial differences using the same system as that of the battery downplays the role of noise, and reinforces the idea that the observed differences between genetic color-deficient and color-normal observers are quite real. However, we are still uncertain as to what the role of each of the visual pathways is in this matter. At the level

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of the visual cortex, the parvocellular-interblob system has been suggested to play an intermediary role between the magnocellular and parvocellular-blob systems. If color-anomalous individuals are deficient in only their P-blob pathways, and not in their P-interblob pathways, the observed gain in spatial resolution may be due, in part, to resources, destined for the blob region, being rechanneled for use by the interblob region. Based on lesion studies and other methods used to isolate each of these systems, conflicting results point to the probable cross-talk between the M and P pathways at numerous points along the visual processing stream.

Binocular vision testing

Along the visual pathways, the earliest point at which binocularity is mentioned (exclusive of stereopsis) is in V1, specifically layer 4B. As mentioned earlier, Livingstone and Hubel (1987b, 1988) found cells in this layer to maintain the properties found in layer 4Cα, and make binocularity a characteristic predominantly of the M pathway. De Yoe and Van Essen (1988), however, also note the presence of binocularity in the parvocellular-interblob region as well. The thick stripes in area V2 (M pathway projections; see Figure 1) exhibit stereoscopic depth and binocular disparity, as do the pale stripes (parvocellular-interblob projections; see Figure 1). Areas V3 and MT (both magnocellularly fed; see Figure 1) also contain cells which display binocular sensitivity.

Given that binocularity is largely driven by the M pathway throughout the various visual regions of the cortex, provided that this pathway is not dissimilar in color-deficient observers compared to color-normals, we should not expect to find any major group differences among the binocular measures obtained.

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As described in the Results section, the number of prism diopters needed for horizontal fusion could be either positive (base out) or negative (base in). Given the large degree of overlap in the range of diopters needed (in each direction) across the three groups (see Table B7 and Figures B93 and B94), any differences in binocularity between the groups is most likely not due to dioptrics (or dioptrics alone) as demonstrated by a non-significant ANOVA. While we cannot ignore the fact that all six individuals classified as having poor or problematic fusion belong to the color-normal group, the subsequent determination of the presence of stereoptic ability in these observers permits us to preclude the possibility that this distinction would lead to any group differences.

IPD was found to be significantly different between groups, which might indicate differences in acuity as the reason for any additional differences found in binocular testing. Given the overlapping ranges across all three groups and the small variability among genetics (see Table B7 and Figures B95 and B96), larger sample sizes would most likely minimize this disparity, thus allowing us to confirm whether acuity can, in fact, be responsible for such differences. A significant correlation was, however, found between color-normals' IPDs and their measures of stereoacuity in the forward direction: $r(26) = -0.39$, $p < .05$. Those individuals with greater IPDs had lower thresholds (better stereoacuity) than those with smaller IPDs. While no other correlations between IPD and stereo measures were significant, either within or across groups, a positive trend was found among genetic observers: increasing forward fusion limits with greater IPDs.

Regarding ocular rivalry and ocular dominance, it was described earlier that several individuals, representing all three groups, exhibited strong rivalry. From this, one might gather that these observers, when performing binocular tasks, might end up

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favoring one eye over the other. This could lead to thresholds either above or below their actual binocular sensitivities depending upon the presence of monocular cues and how adept a given individual is at using them. Our stereopsis experiment (results from which are discussed in the next subsection) attempted to control for such cues by using, for example, a dynamic pattern surrounding the RDS square. Therefore, for our sample, we might expect to see reduced performance in some observers so affected. It is important to note, however, that the classification of a participant as having strong, weak, or mixed rivalry was based on a subjective interpretation on behalf of the experimenter of a self-reported description by the observer. Given several different experimenters, each with their own diagnostic criteria "rules," coupled with a moderate degree of inconsistency of eye dominance between the two tests used (see Results), it is questionable as to whether we would be able to separate out such observers with any sort of accuracy.

With the exception of the one acquired color-deficient observer who was found to possess no measurable form of stereopsis, all of the other observers in each of the three groups performed adequately enough on the various stereo-screening tests to allow us, for the time being, to dispel any immediate concerns regarding the above matter, and not pass judgement until after assessing the groups' measures on stereopsis.

Stereopsis

Following the same progressive sequence as binocularity, stereopsis (in the form of cells sensitive to and selective for binocular disparity) is first evident in layer 4B of area V1 (De Yoe & Van Essen, 1988; Livingstone & Hubel, 1987b, 1988). As alluded to earlier, while the existence of parvocellular blob cells with magnocellular characteristics

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have previously been reported (e.g., Edwards, et al., 1995; Livingstone & Hubel, 1987b, 1988), it is unclear if binocularity is one of these properties. Disparity-tuned cells were, however, definitely found in the parvocellular interblob regions (De Yoe & Van Essen, 1988).

Increased cell responses to stereoscopic depth cues have been recorded from the thick stripes of area V2 (Livingstone & Hubel, 1987b, 1988), the pale stripes also appearing to be relatively binocularly selective (De Yoe & Van Essen, 1988). Among the higher cortical regions, close to half of the cells in V3 (entirely projected to from M pathway ascendants; see Figure 1) are selective for binocular disparity (Felleman & Van Essen, 1987). In addition, De Yoe and Van Essen (1988) describe some binocularly sensitive cells in area V4. Area MT is also largely selective for binocular disparity (De Yoe & Van Essen, 1988; Livingstone & Hubel, 1987b, 1988).

Going back to the findings made by lesion studies on stereoptic ability, lesions made to the M pathway at the level of the LGN showed no deficits. Lesions made to the P pathway, however, led to deficits in fine static stereopsis (i.e., high spatial frequencies), while sparing coarse stereopsis (i.e., low spatial frequencies; spatial frequencies ranged from 2.2-4.5 cpd; Schiller & Logothetis, 1990; Schiller, et al., 1990). This would point to at least some P pathway input, and perhaps make stereopsis a function shared by the two pathways in some manner (e.g., at least at low spatial frequencies both channels are capable of processing stereoscopic depth; Schiller, et al, 1990). Psychophysically, stereopsis was found to be a M pathway function, as per studies performed at equiluminance (Livingstone & Hubel, 1987b, 1988; see, however, Stuart, Edwards, &

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Cook, 1992, for evidence indicating probable P pathway involvement, and a shared control of stereopsis between the two pathways).

With the exception of V4, each of the regions containing cells sensitive to binocular disparity are largely (if not entirely) M pathway controlled. As mentioned earlier, lesion studies are racked with methodological flaws, and results of equiluminance studies are difficult to generalize to situations outside of the laboratory. In addition, given that De Yoe and Van Essen's (1988) findings are based on data obtained from macaques which, as discussed earlier, may not be as similar to humans as was once believed, it is difficult to hypothesize what we should expect to see. It is possible that information pertaining to binocular disparity is processed at higher levels as well, and is not readily detectable by lesioning at the level of the LGN. Schiller, et al. (1990) raise the possibility that stereopsis may be able to be processed by either channel independently, as neither lesions made to V4 nor MT alone produced (what these authors consider) deleterious effects on stereoscopic depth perception. From this we would hypothesize that there would be no differences between groups, since deficits in one pathway could be corrected by the other. The results of the stereo-screening tests (i.e., the overlap across all three groups) support this assertion. On the other hand, an altered P pathway can lead to deficits or (similar to contrast sensitivity) enhancements in the region of high spatial frequencies (possibly via the M pathway, the P-interblob system, a combination of the two, or by another mechanism entirely).

We were somewhat surprised at the results of the stereopsis experiment, as the color-deficients were found to have higher sensitivities across all measures. On average, the genetic color-deficient group had stereoacuity thresholds that were more than twice as

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sensitive as that of color-normals in the forward direction (see Table B8 and Figures 54 and B97), and by nearly this factor in the backward direction (see Table B8 and Figures 55 and B98). Despite these differences becoming negligible when viewed at the level of group medians, the fact that the genetic group members' individual thresholds were so compact (especially in the minimum back condition), the possibility of some common underlying mechanism present in this group of observers cannot be ruled out. Similarly, in assessing the extent of Panum's fusional area, the genetic color-deficients showed larger thresholds (i.e., a larger area of fusion) in both directions on both the mean and median levels of comparison, although differences between groups were not as marked (see Table B8 and Figures 56 and 57 and B99 and B100).

Mirroring our discussion of the results of the contrast sensitivity experiment, there are various possible reasons as to why we found what we did in observers' performances on stereopsis. One interesting line of thought, that may actually serve as a corollary to discrepant findings in the above mentioned lesion studies, concerns the role of hemispheric specialization in stereopsis. While data are scant, several authors (e.g., Carmon & Bechtoldt, 1969; Julesz, 1971; cf. Richards, 1970) have suggested that the right hemisphere is involved with spatial perceptual tasks in man, including stereopsis, in the absence of monocular cues of form and depth. Compared to patients either with left hemispheric lesions or with no brain damage, patients with unilateral right hemispheric lesions exhibited marked deficits in performance (e.g., more errors and longer response times) in a task requiring the location of a form in depth (Carmon & Bechtoldt, 1969). Thus, a question that can be raised is whether there are any differences in the right hemispheres of the observers in our study. Are these differences restricted to the already

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designated groups, or is this a variable that would somehow (perhaps by some type of imaging) need to be accounted and controlled for prior to visual diagnostic classification? Furthermore, are our stimuli appropriate for such an assessment (i.e., tapping into both hemispheres equally)?

Problems involving stereoscopic deficits resulting from hemispheric asymmetries are not necessarily restricted to individuals with hemispheric lesions. Some theories postulate that these deficiencies may also occur in observers with abnormal binocular interactions in the visual cortex and/or the absence of proper ocular alignment. While these anatomical anomalies are often of childhood origin, persisting into adulthood only if uncorrected for (e.g., squinters; see Richards, 1970), are they and their perceptual drawbacks necessarily restricted to this time period? Given the broadness of neural plasticity (discussed earlier), might the reduced use of one eye over the other (which may occur, for example, in acquired color-deficient observers following incident) lead to some sort of cortical non-uniformities potentially resulting in altered states of perception? It is interesting to note that the acquired observer diagnosed with Leber's idiopathic stellate neuroretinitis had higher thresholds on a couple of measures compared to other group members. In addition, several observers from both the color-normal and acquired color-deficient groups did not have measurable thresholds on one or more of the stereo tasks. While the above conjectures may not necessarily reflect the current samples, it might be important with the accumulation of additional data. The possibility of gaining further information with extended testing of our acquired color-deficient observer possessing no stereoptic ability also exists.

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Given the possibility that stereopsis is a function that is shared by the M and P pathways, we may posit the following. Assuming, for the moment, that the P pathway is responsible for color and partially (or entirely, for that matter) responsible for stereopsis, in an observer who lacks one of these abilities, could the extra available resources present in this pathway normally devoted to the lacking function not be put toward the remaining function or functions, perhaps enhancing the observer's sensitivity in the process? This goes back to our discussion on contrast sensitivity, where we mentioned the tradeoff that comes with patches of a single type of cell (Roorda & Williams, 1999). In the color-deficient observer, the lack of diversity in the retinal mosaic was postulated as the source of sensitivity enhancement, due to the lack of chromatic opponency (antagonism) of the cells' receptive fields. With stereopsis lying along a continuum (with numerous gradations of coarseness), spatial frequency channels inevitably come into play. The underlying mechanisms leading to observed differences in stereopsis performance between groups (or individuals) can conceivably be any or all of those given above for contrast sensitivity. In fact, as discussed below, the spatial frequency channels used in contrast sensitivity may simultaneously be used for other purposes (including the processing of depth information), making such a theory quite plausible.

Julesz (1986), on the topic of the plasticity of stereopsis (i.e., the fusion of stimuli of different proportions), surmised that this is most likely due to pools of disparity neurons tuned to different binocular disparities interacting amongst one another. This parallels the idea of neural pooling among spatial frequency channels that are similarly tuned. Differences in measured stereopsis among individuals could stem from differences in type and/or number of neurons (or pools of neurons). For example, pools of neurons

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with peak disparities that are quite similar to each other could result in finer discriminability among similarly disparate stimuli. A sparsity of neurons in a given pool will necessarily sum signals from a smaller number of units which may yield a higher degree of imprecision when judging the amount or even the direction of the disparity.

Barlow, et al. (1967; see also Poggio & Poggio, 1984) remind us that fusion requires two elements of visual processing: the selection of the parts of the separate images that belong together, and the assessment of the small displacements in the relative positions of the paired parts resulting from binocular parallax. Different neurons require different disparities (i.e., for peak response signaling). Therefore, objects at different distances will excite different neurons. In cats, these authors found that one eye vetoes the response of the other eye when the disparity is incorrect (which, it appears, may be facilitated by monocular pattern recognition prior to binocular analysis; feature filtering), while in humans (citing Julesz), depth results from point by point comparisons of varying disparities or degrees of lateral shift of one of the two fields.

Poggio and Poggio (1984) reviewed what, at that time, were the current approaches to solving the correspondence problem (the first element of Barlow, et al. 1967). Based on computational algorithms, it seemed clear that the correspondence problem occurs early in visual processing, before object recognition. Likely ways the visual system could perform this includes matching to edges, or more interestingly, matching to intensity changes following a low pass spatial filtering of the image (i.e., elimination of high spatial frequency components). These filters are not tuned to orientation, but are, instead, physiologically similar to antagonistic receptive fields tuned to a restricted range of frequencies. Obviously differences in these filters, either in their

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composition or in their distribution, could lead to inaccurate or contrasting information that will have to be sorted through by higher levels of the brain. The disparity issue (second element) remains unresolved, although they postulate the possibility that some form of inhibition of cells with different types of disparities, in combination with other detectors, may lead to our ability to converge to three-dimensional images (and confirm that differences in these components give way to differences in stereopsis). Wiring differences may very well lead to differences in inhibition, and the resulting signals, when processed, will result in different percepts and sensations in different individuals. The authors state that disparities may be resolved by spatial frequency channels, themselves (coarse = large; fine = small; see above). It is most likely the case that many different channels (or several couplings) are involved in the matching process. Thus, channel interactions may be the source of miscues, especially among neuronal networks, which seem most apt to handle the disparity issue.

Stereoacuity may be based on a differencing mechanism measuring relative disparities. Exposure duration is vital to threshold measurements (and their comparisons). Stereoacuity may also use the same interpolating mechanism for input as monocular hyperacuity (Poggio & Poggio, 1984; see also Julesz & Spivack, 1967). Significant correlations found (in the unpartitioned sample) between stereoacuity (in both the forward and backward directions) and vernier acuity thresholds support this view.

Receptive field characteristics and the spatio-temporal configuration of the stimulus are believed to be key to neuronal response (Poggio & Poggio, 1984). Again, this points to common mechanisms among several visual functions, with differences in the mechanisms most likely leading to differences in threshold sensitivities. As

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mentioned earlier, reduced chromatic antagonism and a differing number of neurons are possible means by which enhanced stereopsis may occur. If such antagonism is reduced in one area, it is most likely carried over to another, given the need for the brain to process information as quickly and in as simplest a way as possible (which would not be the case with different degrees of antagonism). The question remains, however, if the same mechanisms are used for several functions, and reduced inhibition results in enhanced sensitivity for one function, will all other functions subserved by this mechanism be similarly enhanced? Or might this mechanism serve as an auxiliary component (occurring early on) to what otherwise are entirely different information processing sequences in higher cortical areas? This is especially poignant when considering the need for allocating a limited amount of resources to the processing of complex stimuli containing multiple perceptual characteristics and elements.

In discussing the neural mechanisms underlying stereopsis, Blake and Wilson (1991) speak of cooperative models, the facilitation and inhibition of neurons sensitive to a wide range of crossed and uncrossed disparities. A given cell inhibits all others tuned to different disparities at the same position in space, while exciting cells tuned to the same disparity at neighboring spatial locations. This could also occur via spatial disinhibition or spatial filtering with subsequent intensity matching. Errant signals may lead to miscommunication among neurons, eventually resulting in incorrect information regarding degree and/or direction of the disparity of the image. Depending on the severity, depth perception may be reduced or absent altogether. Models including coarse-to-fine spatial filtering by cortical receptive fields responsive to a limited range of spatial frequencies reinforce our comments made earlier regarding similar mechanisms involved

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in visual functions in which the available information is broken down into spatial components and analyzed as such. The suggestion that disparities may be summed across spatial scales to produce a net disparity estimate (from which the brain processes further) leads us to ask if some estimates are "better" than others. Do "poor estimators" necessarily have poor stereopsis, or could "training" somehow build up a compensatory mechanism that can make such an observer no different from "good estimators?" Could some alteration of these cortical cells (e.g., number, spacing, summation capacity and capability, neural innervation, etc.) result in a finer degree of estimation and a more enhanced sensitivity to subtle perceptual differences?

McKee (1983) mentions that stereoacuity may arise through summation of luminance, through probability summation, or through physiological summation. As a result of using line targets, summation of luminance was rejected since only the two end points of the line would be sources of information regarding disparity. However, might this be different for RDSs? The RDS is, itself, a random array of light and dark dots (and clumps of dots). Going back to Roorda and Williams's (1999) claim that one can gain in resolving high frequency luminance patterns by having large patches of a single type of receptor, could this not result in a greater sensitivity to such patterns? The possibility of probability summation among independent sensing units responsive only to point targets also has potential, especially if certain wiring schemes and reduced chromatic antagonism between units are considered. Physiological summation is a summation within a circumscribed area, the region which stimulates a particular unit. Variability in certain aspects of these units (e.g., number, type, placement) and the summation field (e.g., size, location) between individuals can conceivably lead to measurable differences between

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observers. By remarking that the degree of overlap of the receptive fields of disparity units is important in physiological summation, McKee (1983) gives support to the above notion, rendering it a realistic possibility, given the known diversity among individuals' visual apparatus. On the topic of fusion and disparity detection, the essence of stereopsis, the embattlement between parallel and cooperative interactions (leading to accurate depth judgement and vision) versus competition (resulting in an inaccurate assessment) are provided as competing processes.

McKee, Welch, Taylor, and Bowne (1990) report that hyperacuity thresholds (including stereoacuity) are limited by the spacing of cortical receptive fields. They reinforce the studies cited earlier by mentioning that the input signals for all hyperacuities pass through the same neural detectors, namely spatial and temporal filtering produced by the receptive fields of retinal, lateral geniculate, and simple cortical neurons. Such detectors may very well be different in some groups of observers.

Panum's area may be defined as the total range of disparities that can be fused about the fixation distance without the help of eye movements (Poggio & Poggio, 1984). Whereas other studies often employed tachistoscopic presentation of stimuli, our study permitted the observer to take as long a period of time as was necessary before responding to a given stimulus. This allowed for eye movements to occur during a potentially long period of viewing, and also led to an inevitable tradeoff. On the one hand, vergence movements (smooth movements, independent of conjunctive eye movements, which are controlled by disparity signals) aid in providing greater fusional capabilities, especially of largely disparate images, that are not possible when viewing flashed targets. At the same time, such fusings can continue to occur well outside the true

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range of Panum's area (hysteresis effect; see, e.g., Julesz, 1986). In addition, a lack of retinal image stabilization has also been shown to produce changes in retinal image disparities of 2-4 min arc due to ocular drifts and saccades. This presents the question of whether we were measuring the real limits of Panum's area. In addition, how comparable are these thresholds across observers and between groups if individuals' detecting methods and detection criteria (on both voluntary and involuntary levels) are different? It is important to stress that when dealing with small-increment scales, such as minutes and seconds of arc, there need not be vast differences in participants' approaches to a given task in order to make a large impact on the resulting measures (see Geisler, 1984). Our findings are, however, in agreement with those of other studies using dynamic RDSs, some of which used stabilized images, in which fusion limits of up to 50 min arc were reported (see, e.g., Julesz & Schumer, 1981).

McKee, et al. (1990) found that stereoacuity thresholds at large eccentricities were substantially higher than at the fovea. Problems similar to those discussed above for contrast sensitivity are just as probable for this function. For example, which area did we measure from in a given individual: fovea, periphery, or both? Given free-viewing, we assume that observers used the area most sensitive to them. Yet, how comparable are sets of data sampled from different retinal areas? Is peripheral viewing affected by the presence (or absence) of certain elements or properties compared to those found in the fovea?

Other factors considered by McKee, et al. (1990) to influence stereoacuity thresholds include occulomotor noise and issues pertaining to luminance. Provided that the input signals for all measures of hyperacuity pass through the same detectors, the

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authors feel that a common source of noise must occur early in visual processing, since the different dimensions that are tapped by the different hyperacuity tasks depend on different neural operations. While no new noise is being added in the later stages of processing, can we be certain that the levels of initial noise are comparable across all groups of observers? At the level of the cortical units, there may be signaling mistakes made between changes in contrast with apparent changes in disparity. Such miscues will result in this being processed incorrectly at higher cortical levels, with a potentially large range of differences among individuals. At the same time, reduced antagonism between adjacent cortical units, found in some observers, may actually result in less noise at the outset, making errors at the level of intercortical communication a relatively infrequent occurrence.

Richards (1970) found that a fair number of individuals (close to 3%) are stereo-weak. He posits that stereo-weak observers may, for example, have disparity detectors which are able to signal whether a figure is on or off the plane (i.e., the ability to see in depth), but lack disparity detectors which signal the direction of disparity. If stereo-weakness is the partial absence of the analyzing mechanisms required for the detection of binocular disparities, logically, then, stereoblindness is a lack of all disparity detectors (i.e., analyzing mechanisms).

In our sample, there were a total of seven observers who were not able to give reliable thresholds on at least one of the four measures. The one acquired color-deficient who had failed the stereo-screening test, gave no reliable thresholds on the stereopsis experiment, and can be classified as being stereoblind. For the other six (four color-normal, two acquired color-deficient), thresholds could not be obtained on no more than

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two of the measures, indicating that these individuals may well be stereo-weak. If these observers are, in fact, stereo-weak, the percentage of individuals so afflicted is, for our sample, 15%, five-fold greater than that reported by Richards (1970). What is puzzling, however, is that, of those believed to be stereo-weak, only one-third were consistent in showing deficits along both of the same-direction disparities. That is, if disparity detectors for uncrossed disparities (i.e., the equivalent of minimum back and maximum back in our experiment) are absent in an individual, then this person should show deficiencies for all measurements in this direction. This was not always the case in our subsample, and those who did produce one of the two same-direction disparity thresholds generally had sensitivities well within the range of their group for the measurable threshold. Are these observers not stereo-weak, but rather have some other anomalous mechanism or interaction resulting in the observed thresholds? Are these observers stereo-weak, but there are separate mechanisms responsible for stereoacuity and longer-range stereopsis, one of which is compensated for by some unknown means? It is also interesting to note that all genetic color-deficient observers gave reliable thresholds for all stereo measures. Whether this is unique to this group, or merely a function of current sample size, remains to be determined. Individuals lacking disparity detectors in not as simplistic a manner as previously described, however, could make mistakes most likely stemming from various miscues as suggested above, which might then be restricted to only certain dimensions of depth perception. Further research is clearly needed in this area.

According to Richards (1970), there are three types of disparities (and disparity detectors): crossed, uncrossed, and zero, each independently controlled. Results from our

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sample support this, as some observers had superb stereoacuity in one direction, and relatively poor stereoacuity in the other. (We did not assess zero disparity in our experiment.) The optimal disparity is said to lie near +/- 1°, presumably activating the greatest number of disparity detectors (Richards, 1971). This may not be so for our sample (or for the general population). There is no one-to-one relation between depth and disparity. Multiple detectors signal the same depth, even though they may respond optimally to entirely different regions of the disparity spectrum. These detectors then pool their responses, resulting in the magnitude of depth sensation (Richards, 1971). As discussed earlier, variations in the networking of these neurons (and the subsequent pooling) can lead to differences in the perceived magnitudes. The mechanism responsible for this pooling is presumably absent in stereoanomalous observers (Richards, 1971). Given the range of possible degrees of affliction (e.g., missing one or more types of disparity detectors and pools of detectors, and the extent to which the reduced neural circuitry presents problems to depth perception), the range of stereoscopic abilities most certainly takes the form of a continuum and is not simply all or none.

To summarize, we are uncertain as to why our results for this portion of the battery turned out as they did. We do not know if the higher sensitivities evident in genetic color-deficient observers on the mean group level are true differences or reflect a sampling error. Given the inclination to stereopsis employing the same or similar spatial frequency channels as are used for contrast sensitivity, we would opt for the former. From this, we may have expected even more pronounced differences between our groups of observers. Considering the numerous biological differences across observers, and the number of alternative approaches that can be taken toward a psychophysical task, it is no

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wonder why the comparison of measures of stereopsis is a difficult one. At the same time we must question whether our current approach to measuring the different thresholds is, in fact, best. Julesz and Schumer (1981) mention the possibility that large, spurious clusters may arise in RDSs, which may facilitate stereopsis (by way of low spatial frequency mechanisms). While we should probably not concern ourselves greatly because of this (since the combination of low probability of occurrence and the QUEST program's requirement of multiple trials to reach the 99% confidence interval would not affect the calculations of final thresholds), it does raise an even more provocative question. Are RDSs the most appropriate stimuli to use to measure stereopsis? In particular, how well do they approximate the natural settings upon which we are called to utilize depth perception?

Monocular hyperacuities

While vernier acuity can be measured monocularly (i.e., only one eye is necessary to detect a slight disparity between two lines, for example), on the cortical level this function shares many of the same cellular characteristics and properties as was found for stereopsis. Direct evidence of hyperacuity properties at the level of the LGN is somewhat questionable, and is most likely dependent on the type of hyperacuity task and stimuli used. For example, a vernier acuity task employing stimuli of very thin lines, and thus requiring fine spatial resolution for the discrimination of slight disparities, will implicate P pathway involvement if hyperacuity-level thresholds are to be reached. Again, Livingstone and Hubel (1987b, 1988) have found the high spatial resolution characteristic of P cells as early in the visual pathway as the LGN. This is not to exclude the role of the

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M pathway at this level. Wider bars and stimuli comprised of low spatial frequency components, along with factors pertaining to the contrasts of the stimuli and the backgrounds upon which they are presented, mark the presence of M cell-based properties, also present at the level of the LGN (see Livingstone & Hubel, 1987b, 1988; see also Shapley & Victor, 1986). Differentiating which pathway plays a greater role in hyperacuity tasks at such an early stage is difficult, since the properties mentioned thus far are components of many stimuli and are not unique to hyperacuity-based tasks. Furthermore, those properties which separate out hyperacuity tasks from others have not been introduced (or are not readily discernable) at this point.

In area V1, cells selective for elements such as orientation, direction, and binocularity (which may be important for threshold measurements in various capacities depending, again, on the type of task and stimuli) are found in layers 4Cα and 4B, both of which are M pathway projections (De Yoe & Van Essen, 1988; Livingstone & Hubel, 1987b, 1988; see Figure 1). We should not forget De Yoe and Van Essen's (1988) finding of cells responsive to orientation and binocularity also present in the parvocellular-interblob region, making for what, at this level, could be a function shared between the two pathways.

The thick stripes of V2 are highly sensitive to binocular disparity (Livingstone & Hubel, 1987b, 1988; Zeki, 1975, 1978), and are orientationally- and (moderately) directionally-selective (De Yoe & Van Essen, 1988; Zeki, 1978). The pale stripes are also selective for binocularity and orientation (De Yoe & Van Essen, 1988).

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The cells in V3 are strongly orientation-selective and relatively sensitive to direction and binocular disparity (Felleman & Van Essen, 1987). Zeki (1978) has reported the presence of orientation- and direction-selectivity in some of the cells in area V4. De Yoe and Van Essen (1988) have also identified binocular sensitivity in these cells. Area MT is also found to possess cells selective for direction, binocularity, and orientation (De Yoe & Van Essen, 1988; Livingstone & Hubel, 1987b, 1988; Zeki, 1978).

It is somewhat of a toss-up as to which pathway would be more adept at handling hyperacuity-based tasks. Are we dealing with form vision, where we need to discriminate stimuli as being separate shapes, such that a break is clear and a disparity is seen? Are we dealing with fine acuity which, as mentioned earlier, results from the high spatial frequency components comprising the stimuli? Or are we dealing with spatial relationships among the different objects that make up the stimuli? In the first two instances, there is a strong influence from the P pathway for controlling hyperacuity functions; in the latter case, the M pathway's role is quite certain. If we are dealing with all three of these elements, then it would seem that both pathways are involved, and depending on how versatile each pathway is, if the performance of one pathway is reduced for some reason, the other pathway may have sufficient resources to take over.

Interestingly, as seen from the results of our vernier acuity experiment, genetic color-deficient observers were found to be substantially (though not significantly) more sensitive (i.e., had lower thresholds) than color-normals (see Table B9 and Figures 58 and B101).

Vernier acuity and stereopsis are probably closely linked, as the stimuli used for each of these tasks can be virtually identical, except for the depth component which, in

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some cases, can be quite minimal (e.g., a break in depth; see Julesz & Spivack, 1967). Again, the finding of significant correlations between stereoacuity and vernier acuity in our sample attests to this. We have already reviewed some of the theories and results of experiments carried out by proponents of the belief that hyperacuities and stereopsis (and possibly contrast sensitivity) share many of the same neural mechanisms used in the detection and processing of visual information obtained. As a result, many of the same reasons given previously as to why the observed differences between our groups did (or did not) occur may be readily applied to the current discussion.

The neural basis for hyperacuities is not fully understood. Geisler (1984) determined that sensitivities in hyperacuity tasks are resistant to changes in the spatial configuration of the stimuli. His machine model for two spot stimuli assumes that photoreceptors are aligned in a hexagonal lattice, and that differences in acuity are based on the mean number of quanta absorbed. He felt that while this may be sufficient for the ideal observer, which the model is attempting to simulate, it does not measure up to real observers, whose receptoral array may or may not take on the same shape or spacing that might be assumed to be necessary for the recording of thresholds smaller than the width of a single receptor. Hirsch and Miller (1987) have subsequently found, in primate retina, that from the central fovea to about 1.5° eccentricity, the cone mosaic is, in fact, a high-quality hexagonal lattice. They found that human acuity measures, pooled from the results of other investigators, are congruent with the lattice model when rescaled to appropriate foveal resolutions. The cone mosaic in a human retina, obtained from a corneal transplant donor, also fit this model reasonably well (see Hirsch & Curcio, 1989).

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The best performance (i.e., lowest thresholds) occurs when the separation angle is on the order of 3-5 min arc, deteriorating at larger separations due to positional uncertainty or to the absence of detectors able to completely integrate information over large distances (Geisler, 1984; Westheimer & McKee, 1977b). While we can (confidently) say that the processing of information from hyperacuity tasks includes channels and neural pooling (which can, themselves, lead to measurable interobserver differences based on their anatomy and physiology), the properties of these channels have been difficult to fully elucidate. Luminance mechanisms have been suggested, based on such findings as decreased thresholds with increasing luminance levels (up to the mid-photopic range; see Westheimer & McKee, 1977b). The enhancement in sensitivity that is possible from such mechanisms (Roorda & Williams, 1999) was discussed earlier. Geisler (1984), however, questions whether or not high spatial frequency information is critical in such tasks.

Geisler (1984) postulated several factors to account for differences between real (results cited in the literature) and ideal (machine) detectors. The first is accommodation which is downplayed to having a quite minimal impact (and is probably so, provided experimental procedures are well designed and implemented). Next is internal noise, for which two types are mentioned: additive and multiplicative, with the effect largely dependent on the location of where the noise occurs in the visual pathway. Nonlinearities in the visual system may prove to be quite important. If significant noise were to arise following nonlinearity, the nonlinearity can strongly affect sensitivity. Given that no two individuals' vision is entirely the same, differences in nonlinearities among observers should be quite prevalent. Since the eye is such a sensitive organ, it is conceivable that

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even small differences in nonlinearities could result in large differences in measured thresholds (especially for hyperacuities, considering the scale units). The shape of the receptive fields may be a powerful variable. According to the model, multiple-lobed receptive fields are optimal for these tasks, and they may be found in the neurons early in the visual pathway. The immediate question is whether we all have these receptive fields in the same size, shape, and quantities, and what the resulting consequences are if this were not to be the case. Finally there is positional uncertainty, in which small eye movements and non-optimal fixation cues may cause the stimuli to fall on different receptors from one trial to the next. This produces uncertainty about stimulus position on the receptor lattice. This can pose problems of even greater proportions when one considers the need to integrate or pool the outputs of several units for determining detection and subsequent processing. If these receptors have different shapes, lengths, weights, or, in some cases are missing, signals may be processed incorrectly. Again, greater sensitivity is also possible if these cells and the channels they comprise operate by way of antagonistic signals (see above).

Even though the participants in Freeman and Bradley's (1980) experiment have little in common visually with any of our observers, some of the findings (and their implications) can be generalized to our study. The supersensitivity found among monocularly deprived observers was attributed to the result of neural recruitment, neural plasticity, and enhancement of sensitivity due to additional "practice" in having to overcome their handicaps. As we posited earlier, both pathways may be capable of, and contribute to, hyperacuities. On the one hand, if we assume a reduced contribution from the P pathway in color-deficient observers, it is possible that resources (e.g., cortical

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space) that were originally earmarked for the P pathway were given over to the M pathway. In addition to what the M pathway was already operating with, these additional means could result in a more enhanced sensitivity than would occur without them. The strengthening of this added neural network comes from its greater use (from lack of an alternative) in everyday situations (hence, the "practice" from above), and, thus, serves concomitantly as a compensatory mechanism. Alternatively, the altered P pathway may, once again, take on characteristics of supersensitivity as a result of the absence of chromatic antagonism. This may be further aided by the possible cross-talk between the two pathways, especially at higher level processing centers where signals and information processed separately are then integrated into a final judgement or solution, which then results in the observed percept.

In another study, Bradley and Freeman (1985) investigated both vernier acuity and contrast sensitivity in non-monocularly deprived amblyopes. Even though vernier thresholds were measured monocularly, a very interesting finding arose which may have some bearing on our own results. They found that maximum vernier acuities were obtained with spatial frequencies near or slightly above the peak of the contrast sensitivity function. This provides a mechanism for the potential superior performance of color-deficient observers in hyperacuity tasks based on the supersensitivity found for contrast sensitivity, and also gives support for a link between hyperacuity and contrast sensitivity tasks on a neural level (possibly by either a common processing mechanism or channel, or similar components in separate channels that may integrate at a later point in the visual pathway).

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Westheimer and Pettet (1990) point out that hyperacuities can best be explained by way of neuronal ensemble processing (see also Westheimer & McKee, 1975). This is akin to some sort of neural pooling, since the vast array of stimulus patterns which lead to hyperacuity thresholds cannot each result from neurons with special receptive field properties. They also believe that both the M and P pathways are involved, largely as a function of stimulus contrast and exposure duration.

The shared role by the M and P pathways in the control of mechanisms responsible for hyperacuities has recently gained strength from the work of Lee and colleagues. Lee, et al. (1995) found that when recording from macaque retinal ganglion cells, only the M pathway was able to support vernier acuity targets of less than about 20% contrast, whereas either pathway could provide an adequate signal for targets above this value. Further credibility was established when it was shown that the same effect resulted from psychophysical measurements obtained from regions in the human parafovea. The fact that the human psychophysical performance showed lower thresholds than those derived from simple cells indicates that vernier acuity is dependent on several receptive fields distributed along the edge of the stimulus (with subsequent summing and/or pooling of responses). The location of the vertical edge of the stimulus is postulated to be derived from an array of vertical orientation-selective mechanisms, perhaps via cells within vertical orientation columns. The authors note intercell variability, the central decision mechanism questionably having precisely the relative positions of ganglion cells in the retina, and an increase in response variance on the way to the central mechanism as possible reasons for degradation of psychophysical

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performance. In addition, stimuli (edges vs. lines) may have some influence on a particular pathway's response and role (based on the spatial frequency components).

Livingstone and Hubel (1994) believe that hyperacuities reflect cortical averaging mechanisms, and with the finding that hyperacuity can be obtained under dark-adapted conditions, this averaging mechanism can use either cone or rod inputs. This agrees with the idea of hyperacuities having both M and P pathway input.

Westheimer and McKee (1977a) echo several of the above findings. They discuss that the direction of vernier offset can be reported on the basis of the sum of signals available within a certain retinal distance and time. Local sign differences can be assigned on the basis of the pooled information from many individually presented patterns (Westheimer & McKee, 1977a,b). Even though these authors (Westheimer & McKee, 1977a) used moving targets, the neural processing (as well as the inherent implications of differences in wiring) is also applicable to stationary targets. They state that hyperacuity represents the lower limit of the relative localization of two feature components (e.g., two lines slightly disparate from each other) and is a spatial comparison process. This refutes previously widely held notions that hyperacuity is (simply) the averaging of local signs along lines or contours, and that detection is necessarily performed according to the criterion of explicit or implicit orientation (since stimuli to which hyperacuity thresholds apply need not contain these properties; Westheimer & McKee, 1977b).

We had already discussed the work of McKee, et al. (1990) above. This group looked at a common source of noise as setting the lower limit for all hyperacuity tasks. Several other issues are worth repeating and elaborating on. The authors indicate that the spacing of cortical receptive fields is a major limiting factor of hyperacuity thresholds,

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and also, in particular, the sensitivities and distribution of simple cortical units. With the idea that the input of signals from all types of hyperacuity tasks pass through the same neural detectors, contrary to Geisler (1984), channels whose components include spatial frequency mechanisms can now be included. In fact, McKee, et al. (1990) speak of spatial filtering models which posit that the precision of hyperacuity is limited by the size and contrast sensitivity of local spatial filters. They claim that a change in position will be detected if it produces a discriminable change in the activity of filters stimulated by the target. For line targets, in particular, thresholds depend on the relationship between the location of the target and the response function of optimally-sized filters, taking into consideration, of course, the weighting functions of the filters. Given that, in the fovea, there is a massive overlap of small receptive fields, it is assumed that the target must fall at the optimum position of some receptive field (thereby causing the cell to produce a greater response signal compared to adjacent cells whose receptive fields are not being stimulated as optimally). All local spatial filters responding to a given target are considered in determining whether a change is detected. We have already discussed, at length, the potential differences in thresholds that can occur as a result of neural miscommunication stemming from irregularly spaced and/or misshapen receptive fields, different weighting functions applied to filters, absent filters, altered pooling of neuronal responses based on variant wiring schemes, and the like.

The models cited by McKee, et al. (1990) are a montage of current theories regarding the underlying neural basis of hyperacuity. Pending tests of the validity of these models, we can easily speculate on possible reasons as to how interobserver variations in the obtained hyperacuity thresholds may have arisen. Again, provided that spatial

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frequency filters are used in the cortical assessment of disparity detection, we may list any or all of the hypotheses provided in our discussion of contrast sensitivity and stereopsis. By virtue of contrast sensitivity apparently playing a large role in our detection of hyperacuity stimuli, it would seem quite valid for future research to pursue these further. As claimed by Westheimer (1984), displacement is a spatial judgement.

Along similar lines of thinking, Mussap and Levi (1995) stress that positional acuity is determined on the basis of the distribution of activity of an array of positionally labeled detectors at the level of the cortex, where neurons receiving convergent inputs from several cells are compared. They further state that vernier acuity requires spatial filters that are both orientation- and spatial frequency-selective. Based on experiments employing dichoptic masking, the neurons involved in vernier offset detection are believed to receive binocular inputs and are postreceptoral in origin (i.e., subsequent to the neural site of binocular integration). The similarity in improvements of vernier acuity and contrast detection with binocular viewing, and the finding that such improvements are greater than predicted on the basis of simple monocular probability summation, suggest contributions from binocular cortical mechanisms. Such findings indicate not only which types of spatial filter alterations could lead to potential differences in vernier acuity thresholds between groups of observers, but also presents us with a possible location of these mechanisms.

In addressing the issue of whether we used the optimal stimuli and experimental procedures in order to measure vernier acuity thresholds (and the bearing this might have in comparing the observed differences between groups), we begin by returning to a point of focus alluded to earlier: conditions under which best hyperacuities have resulted.

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Geisler (1984) found this to occur when the separation angle was between 3 and 5 min arc. This is in close agreement with Westheimer and McKee (1977b) and McKee (1983) who recorded lowest thresholds with gap separations from 2-4 min arc.

Foveal fixation (specifically the foveola) produces the finest thresholds (McKee, 1983; Westheimer & McKee, 1977b), and many studies employ fixation markers in order to insure compliancy and certainty that thresholds are based on measures consistently taken from the same patch of retinal cells. We had told our observers to fixate in the center of the aperture, which, when combined with both head stabilization and (at most) a couple of trials for orientation, should have resulted in such consistency. However, given the small scale upon which this task is based, even minor variations across trials can weigh more heavily on the calculated thresholds. There is a very low probability that, in our study, the majority of one group was foveally fixated while the majority of another group was not (thus giving rise to the observed group differences). We are aware that our experimental technique may not have necessarily measured observers' finest thresholds. However, a design modification such as the use of eye-tracking equipment and artificial pupils, which may well be necessary to control for such confounds, can be quite cumbersome and may not necessarily add to our general finding that genetics are more sensitive, on average, than are normals. That is, this change in instrumentation might not lead to a greater difference in thresholds between groups, but rather would result only in a uniform shift toward higher sensitivities across groups.

Westheimer and McKee (1977b), perhaps confirming the idea that hyperacuity occurs from averaging mechanisms along the length of line targets, found that vernier acuity improves with line length up to five min arc. Interestingly, the gap separation

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mentioned above was found to be independent of line length, in that lines so short that they are perceptually seen as dots yield comparable thresholds, provided that there exists a gap separation of 2-4 min arc. As a further impetus to control for eye position, these authors point out that optimal performance is very dependent on retinal distances. They note that the retinal image spreads nearly one min arc beyond the geometrical image, losing demarcation if the lines comprising the stimuli are separated by less than one min arc. McKee (1983) states that even with the constraints of minimal gap separation and that longer line length is better, abutting lines are rather poor targets for measuring hyperacuity thresholds. Alternative stimulus configurations include whole lines with different lateral separations, set either singly or in pairs, whose disparities are judged relative to a standard, which is flashed moments before the test stimulus. Our stimuli, which consisted of abutting lines, were less than optimal as judged using the above guidelines. Yet, the obtained thresholds were well within hyperacuity range, and any degradation that may have resulted from the use of such stimuli was certainly not readily apparent. Furthermore, given that all observers were run under the same conditions, a change in stimulus component separation length, while perhaps resulting in better thresholds overall (i.e., greater sensitivity due to increased resolution), will not alter the trend found using our current parameters.

It is questionable as to whether we have drawn any closer to resolving the debate about how hyperacuities are processed from a neural perspective. Furthermore, it is still a matter of opinion regarding if and how each pathway may contribute. Given the numerous alternatives for stimuli, it is difficult to determine whether we are tapping into both the M and P pathways equally or are biased in one direction based on the presence or

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absence of certain components. Would the same group differences be observed if we were to simply reverse the contrast of the elements, thereby having bright bars on a dark background? How would our observers fare with a stimulus pattern that is less dependent on edges and contours? Even changes such as these will not necessarily give us a clear indication regarding the roles of the two pathways. Where the possibility exists that either pathway may fully control the function, nothing may be lost in the presence of an altered pathway, provided the fully functioning pathway is able to handle the extra responsibilities entirely. If, as proposed by several teams, either the same or similar neural mechanisms (i.e., filters, channels, etc.) are responsible in the detection and processing of information in vernier acuity as in stereopsis (based on common selectivity of disparity and orientation found in the cells), and the filters or channels are tuned to differences in spatial frequency, we may be more confident in asserting where our groups' differences may be originating from. (We have, however, seen some scientists advocating for a prereceptoral origin, while others support a postreceptoral theory, leaving this issue as unresolved at the present time.)

Motion sensitivity

As described in the Introduction, it seems that motion sensitivity is one of the clearest of visual functions regarding pathway control. The properties of movement- and direction-sensitivity are present at the earliest points of the visual pathway, at the amacrine cell-fed RGCs (Dowling, 1987). M cells, as a whole, are often characteristically referred to as being transient, and, as such, are perfect for analyzing movement (Tessier-Lavigne, 1991). Also at the level of the LGN, M cells are found to be transient, a property

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carried over to areas to which these layers project (i.e., layer 4Cα, along with which orientation-sensitivity is noted, and layer 4B, whose cells exhibit orientation-, direction-, and movement-selectivity; see Figure 1). The interblob region contains cells tuned for stimulus orientation and movement, but not for direction (De Yoe & Van Essen, 1988; Livingstone & Hubel, 1987b, 1988). At area V2, cells in the thick stripes are found to be highly selective for movement (Livingstone & Hubel, 1987b, 1988; Zeki, 1975, 1978) and orientation (De Yoe & Van Essen, 1988; Zeki, 1978), but are questionably direction-selective. Pale stripes are orientation-selective (De Yoe & Van Essen, 1988). Most cells in area V3 are strongly orientation-selective, and a fair number of these are selective for direction (Felleman & Van Essen, 1987). Area V4 has been shown to be slightly selective for orientation and direction (Zeki, 1978). Cells comprising area MT are very selective for both direction and orientation (De Yoe & Van Essen, 1988; Livingstone & Hubel, 1987b, 1988; Zeki, 1978). Furthermore, psychophysically, the M pathway has been determined to be responsible for motion detection and sensitivity to orientation and direction (Livingstone & Hubel, 1987b, 1988).

This suggests that motion sensitivity thresholds for color-deficient observers should be no different from those of color-normals. While this is what was essentially found for $d_{max}$, genetics performed considerably better on $d_{min}$ (see Table B10 and Figures 59 and 60 and B102 and B103). Again, it is important to note that the ranges of each group's thresholds overlapped, and different sample sizes were used in the calculations of the normal group's mean (and median) $d_{min}$ and $d_{max}$ values. Yet, the fact that $d_{min}$ represents a hyperacuity measure (see, e.g., McKee, et al., 1990) whereas $d_{max}$ does not, may put the color-deficients at an advantage in the former instance and not in

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the latter case. We had found earlier that genetics had superior thresholds for the vernier acuity and stereoacuity tasks compared to normals. Perhaps when dealing with motion perception there are different mechanisms at work when dealing with small displacements versus large displacements, and these groups are more equated when perceiving greater ranges. The investigation of the neural basis of motion perception will hopefully help in resolving this issue.

Beverley and Regan (1973, 1975) posit that each eye drives one pool of motion detectors which is sensitive to left-to-right motion, and another pool that is sensitive to right-to-left motion. The outputs of these four pools of monocular motion detectors are routed in such a way that there are four pools of binocular movement detectors that are sensitive to movements along four different directions in three dimensions. The outputs are compared at comparators so as to enhance the discrimination of direction. Different sets exist for motion directed towards and away from the head. Motion of intermediate direction excites two adjacent detectors to some extent, possibly being subtracted at a comparator, giving us the perception of fine angular discrimination.

The concept of multiples of multiple pools of detectors only further underscores the complexity of the processing of motion. How would motion perception be affected if, for example, one or more types of these detectors are missing or reduced in number? Can the other types of detectors be recruited to perform functions that are potentially "opposite" those that they are destined to perform (e.g., a preordained left-to-right motion detector switching to a right-to-left motion detector), or is that direction's sensitivity greatly impeded or, perhaps, even non-existent? The various rewirings that could result from such a situation would lead to differences in neural processing too numerous to

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mention, from miscues and subsequent misperceptions in direction of motion, to less than perfect weightings of mechanisms used for judging the signals from the detector pools (assuming such weightings come into play), to the potential enhancement of detection in some domain.

Srinivasan (1985) notes that directionally-selective motion detectors are maximally sensitive to movement only when they sacrifice color. (Any remnants of color in the M pathway would surely cancel any enhancements.) The neural pooling or integration between two or more receptors stimulated by a moving target must account for the time factor by which they are separated. For a pattern that is moving to the right, for example, if the receptor which is stimulated first is subjected to a neuronal time delay and is then multiplied and time-averaged with the adjacent receptor, the correlation would produce a substantial response signifying motion toward the right. The more richly colored the scene, the more important that the spectral properties of the input channels be matched. This would lead to the motion pathway being "colorblind" in the sense that the spectral sensitivity functions of the channels are identical and the only difference is the advance in time of one channel compared to the other (i.e., identical signals less the time factor), not that the input channels are necessarily derived from a single receptor class. However, given the spectral properties of color-anomalous observers to begin with, there might be some advantage in the visual systems of these individuals by not having to work as hard to overcome such limiting criteria.

In fact, Srinivasan (1985), based on studies of insect vision, conjectures that colorblind movement detectors will not respond to movement if the visual scene is a two-color pattern in which the intensities of the colors are such that the pattern provides no

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contrast to the input channels. This is a plus for color-deficients whose color vision is reduced to shades and intensity differences in certain regions of the spectrum, but such a pattern is felt to be a rare occurrence in nature (see Srinivasan, 1985). Srinivasan posits that if we regard the receptive field of each input channel as a spatial filter, then in order for the responses of the various channels to be maximally correlated, the channels must filter the scene in the same way (i.e., the receptive fields should all be identical). If this were so, and the contrast criterion mentioned above was also met, motion detection could be enhanced according to the method discussed by Roorda and Williams (1999; see above).

Braddick (1974), using RDKs, found that coherent motion was limited by the absolute spatial displacement of "partner" dots, and not by the number of pattern elements intervening between partner's positions. (As per Braddick, within the displaced region, each dot in one pattern has its shifted "partner" in the other pattern.) As described earlier, the observers in his study had found that the perceptual segregation of a moving target against the surround started to deteriorate around five min arc and was complete by about 20 min arc. This occurred with an interstimulus interval of 10 ms; results were reportedly even poorer with increasing intervals (see also Lappin & Bell, 1976). Braddick (1974) coined the terms "short-range" and "long-range" apparent motion. Short-range apparent motion (what his study was measuring) is explained by directionally-selective neurons, and is postulated to be a low-level motion detecting process with a very limited spatial range. Long-range apparent motion (i.e., perception of motion from the succession of two more widely separated stimuli) is explained by a higher level, more complicated process, which includes the interpretation of the stimulus configuration. Support for the

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direction-selective neuron hypothesis comes from motion aftereffects, in which perceived motion in the direction opposite that of a stimulus viewed for a prolonged period of time is attributed to neuronal fatigue. In addition, the upper limit of short-range apparent motion may, in some way, approximate the spacing of input channels to the motion detectors.

Chang and Julesz (1983a) mention certain limitations of Braddick's (1974) study, and note that the upper limit of pattern segregation (i.e., $d_{max}$) that was observed is not absolute. From this it is conceivable that if $d_{max}$ extends into a range that Braddick (1974) considered to be a long-range apparent motion process, and that if short-range and long-range apparent motion processes are handled in separate ways, the differences observed between color-deficient and color-normal observers regarding $d_{min}$ and the lack of such differences regarding $d_{max}$ may stem from the ways in which the information is processed.

Chang and Julesz (1983a) state that the long-range process responds to contour or form movement (characteristic properties found to be tapped at least partly by the P pathway; Livingstone & Hubel, 1987b, 1988), while the short-range process responds to element movement. Thus, the genetic group's loss of superiority to color-normals when shifting from $d_{min}$ to $d_{max}$ thresholds may reflect one of several possibilities. As alluded to above, it is quite conceivable that there are different mechanisms responsible for the processing of long-range and short-range motion. Provided it was the same mechanism employed in each case, we would expect the enhancement to remain independent of displacement distance. Alternatively, it may be that the same mechanisms are operating, but the mechanisms' responses might be more highly subject to stimulus characteristics and/or the interactions between the M and P pathways during information processing. It is

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possible that the observed enhancements occur only in the presence of high spatial frequency stimuli, and that the altered P pathway in genetics lead to deficits for other types of stimuli and stimulus properties. These deficits could be essentially masked by the M pathway taking over control of this function at lower spatial frequencies (making motion sensitivity a shared function regarding pathway control), or the P pathway may play only a minimal contributory role, perhaps at later stages of processing where final perceptual judgements need to be made. Support for these latter ideas comes from our finding that genetics' $d_{max}$ values were not any less sensitive than color-normals'.

Using two 60 ms random dot arrays with an interstimulus interval of zero, Chang and Julesz (1983a) selectively stimulated the short-range process of apparent motion, while simultaneously eliminating the effects of saccadic eye movements. They found that $d_{max}$ increased at a rate approximately equal to the square root of the area of the target (as did Lappin & Bell, 1976), attributable to the process of spatial summation. They further determined that, for asymmetrical stimuli (e.g., rectangles), moving targets are integrated only in the direction of movement. The authors conclude that the short-range process of apparent motion is dependent on the number of local elements stimulated, not solely on peripheral units having larger receptive fields (retinal eccentricities up to 4° did not affect $d_{max}$). We are uncertain, however, as to whether the receptive fields of color-normals and color-deficients are similar in shape and number foveally, such that the possibility of peripheral or peripheral-like receptive fields contributing to short-range motion, at least in some individuals, cannot be excluded. In a separate experiment, it was shown that differences in $d_{max}$ for motion direction and pattern detection exist, and that these are mediated separately by possibly two different levels of the same short-range process

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(Chang & Julesz, 1983a). It is intriguing to consider the existence of several other levels, one or more of which may help in contributing to observers' motion sensitivities under special circumstances, or if the mind cannot at times be tricked into using levels not normally used.

Chang and Julesz (1983b), using filtered RDKs, found that $d_{max}$ is limited by the bandpass spatial filtering of the stimulus and resulting grain size. By increasing the cluster size in the image, $d_{max}$ was also found to increase. These authors report that within a specific direction-detecting mechanism, there exist spatial frequency-tuned channels. High spatial frequency channels are believed to feed only small displacement detectors (based on the bandpass filtering of the stimulus). Furthermore, motion perception of the short-range process is processed by different size channels, and these channels exist at all eccentricities tested (0-4° arc). Differences in thresholds resulting from differences in the spatial frequency channels in color-deficient observers compared to color-normals is not a new topic, and the arguments stated above for the other visual functions to which spatial frequency channels pertain apply to the current discussion as well. The notion made by McKee, et al. (1990) that motion displacement is a hyperacuity (and is subject to the same sources of noise, spatial filtering, neural mechanisms, etc., as stereoacuity and vernier acuity) reinforces this.

Additional support comes from the analyses done by Julesz and Schumer (1981), who found that direction-selective motion mechanisms appear to operate best at low spatial frequencies and high temporal frequencies. These properties match those to which the M cells are tuned, and if we were to, again, envision the M pathway, by some compensatory mechanism, to have greater sensitivity as a result of a reduced or non-

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existent P pathway (as discussed above), we can explain differences in thresholds between groups of observers. Add to this the idea that motion mechanisms are also responsible for the analysis of the spatial form of the moving target, and we are, once again, presented with a paradoxical situation whereby sensitivities may either improve or become worse from a particular neural wiring scheme due to as yet unknown methods of information processing in the brain.

While it might seem that most of the studies cited thus far portray motion sensitivity as a function designed for the M pathway, recent evidence has turned up which undermines the idea of motion being controlled solely by this pathway. Aside from lesion studies which, again, showed that, lesions made to the M pathway at the LGN produced marked deficits in both detection and discrimination, the P pathway was found to have some motion processing capabilities at lower temporal rates (Schiller & Logothetis, 1990; Schiller, et al., 1990). Lesions made to area MT showed moderate impairments in motion perception, but the ability still remained. At equiluminance, psychophysical tests revealed that motion perception was compromised, but not abolished (see Livingstone & Hubel, 1987b). Color-specific motion detectors have been found to exist (Cavanaugh & Favreau, 1985; see also Tyler & Cavanaugh, 1991), and Shipp and Zeki (1989) found that the backward projection from area MT to area V2 extends not only to the thick stripes, but to the thin stripes and pale stripes, as well. They hypothesize that areas receiving a back projection from an area to which they do not project may be sites for the integration of signals relating to different attributes of vision (Shipp & Zeki, 1989).

Gegenfurtner and Hawken (1996), after reviewing the literature and performing several of their own experiments, speak of the presence of two motion pathways. The

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"slow" pathway is characterized as being high in sensitivity for color contrast, but unable to code for velocity in a contrast-invariant manner. The "fast" pathway, on the other hand, is high in sensitivity to luminance-defined stimuli. The authors state that in the fast pathway, color variations are processed like small luminance variations without actually signifying color itself, and that motion coding is contrast-invariant and veridical. They hypothesize that the neural substrate for the fast pathway is the M pathway, including area MT. That for the slow pathway is somewhat more elusive, but it is surmised that the pathway from area V3 to area V4 is a good candidate.

Separate motion pathways have been considered feasible for some time. Newsome, Mikami, and Wurtz (1986), recording from cells in the cortex of macaques, found that the maximum spatial intervals for which neurons from area MT were able to extract motion information corresponded to the maximum spatial intervals for which the short-range mechanism of apparent motion is operative. With minimal overlap between the psychophysical data and the physiological responses of neurons in the striate cortex, it was concluded that directionally-selective neurons in area MT were the likely substrate for psychophysical responses at maximum spatial separations. For separations that were less than the maximum, recordings from monkey MT cells revealed that at low speeds directional interaction of these cells were limited by the temporal interflash interval, while at high speeds it was a combination of spatial and temporal intervals. Cells in area V1 were limited both spatially and temporally over the entire range tested. Human psychophysical data, which measured the threshold spatial interval for which motion was perceived at five different apparent speeds, closely matched the physiological responses here as well. This indicated that the threshold for directional selectivity in cortical

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neurons is a correlate of the psychophysical threshold for perception of apparent motion. Based on these findings, the authors conclude that area MT is the likely neural substrate for the perception of motion at high speeds, while the requisite neuronal capabilities exist in both areas V1 and MT for the perception of motion at low speeds ($\leq$ 4-8°/s). This is in agreement with Thiele, Dobkins, and Albright (1999), who determined that the majority of cells in area MT of the macaque responded preferentially to stimuli with higher temporal frequency and lower spatial frequency components. While the pathway connections to area MT are almost entirely from M pathway-based predecessors (see Figure 1), area V1 is comprised of cortical regions from both the M and P pathways. Newsome, et al. (1986) give no indication as to whether they believe that all cortical regions in area V1 (and, thus, both pathways) form some sort of composite in the processing of slow speed motion stimuli. In fact, they refrain from using any terminology indicative of separate parallel visual pathways.

By using RDKs for our stimuli, we are subject to the same problems that we had encountered earlier with the use of RDSs for stereopsis. Random dot arrays are beneficial in the sense that cues, such as edges and contours, that would ordinarily facilitate detection and adjust one's thresholds accordingly are virtually nonexistent. At the same time, such stimuli lack elements such as orientation, a property common to many cells selective for direction of motion, thus questioning whether the cells are optimally stimulated. That is, stimuli containing elements of both direction and orientation may result in thresholds superior to those obtained from stimuli containing either element alone. It is important to remember, however, that the main goal of this study was to compare group differences on several visual functions. Obtained thresholds need not be

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perfect in order for this to be achieved, provided that the collected measurements are reasonable for the given task. Alternative stimuli do, of course, exist (e.g., slits and gratings), but not without their own sets of problems. The use of gratings, for example, leads to the difficulty of differentiating exactly what components of a stimulus a given cell is responding to. Less specialized cells (i.e., those responsive to multiple properties) may respond maximally to one element alone when several are simultaneously present, leading to thresholds that are enhanced or diminished relative to what would result had the property in question been sufficiently isolated. We are in agreement, however, with Chang and Julesz (1983a) who discuss, at length, reasons why RDKs really are ideal stimuli for this type of task. The only possible source of slight orientation cues in RDKs would arise, by chance, from a random cluster of dots. Such a cluster would then be subject to the criteria set forth by Chang and Julesz (1983b) in which motion detection is dependent on whether motion is parallel or perpendicular to the elongated direction of the "blob." They had found somewhat lower $d_{max}$ values for instances where motion was parallel to the elongated blob, a result possibly attributed to a reduction in the number of false targets. The rules governing the QUEST paradigm (e.g., 99% confidence intervals), along with a low probability for clusters occurring in consecutive trials, minimizes the influence such stimuli would have in biasing the obtained thresholds or the observed between-group differences.

Lappin and Bell (1976) note several key elements responsible for the accurate discrimination of the direction of apparent motion of successive random dot patterns: their separation in time, their separation in space, and the number of elements in the pattern. Specifically, the percentage of correct responses decreases with increasing

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interstimulus interval, spatial displacement, and number of dots. We need not be concerned with either the first or second of these, since our interstimulus interval was set to zero which, as mentioned earlier, was optimal, and we were attempting to find observer's thresholds, making spatial displacement one of the variables. Our choice of dot density (equivalent to that used in stereopsis), however, might need to be considered further. While Chang and Julesz (1983a) found $d_{max}$ to be invariant to distances between at least 50 and 100 cm for stimuli subtending 5.6° arc, such limits need to be determined for other angular subtenses and distances to allow provision of the most optimal of stimuli to be presented and the resultant thresholds to be obtained. Again, though, given that all of our study participants were subjected to the same stimuli under identical conditions, it is questionable as to whether such changes in parameter would procure anything more than minor variations of the existing differences currently found between the groups.

With dots uniform to both the fovea and periphery, it would intuitively seem that slight eye and head movements should not disrupt an accurate assessment of one's thresholds. This may not necessarily be the case if differences exist in the size, shape, and quantity of eccentric detectors located nasally compared to those located temporally, both within and between observers. This could lead to an undersampling of detectors in an individual who produces movements in a consistent manner and direction.

In addition, like we mentioned for stereopsis, random dots are a rather unnaturalistic prototype for what we see everyday and for which objects we will need to assess regarding motion. Thiele, et al. (1999), for example, show that neurons in area MT are responsive to heterochromatic stimuli, although they may not necessarily be the sole

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contributors of the perceptual experience elicited. This is an especially important finding since all of our color-deficient observers exhibit at least some color perceptual ability.

We have seen that, while a number of studies done over the years show strong support for the M pathway controlling motion perception, others are presenting data claiming otherwise. Due to the differences in method, including stimuli and observers used, there does not seem to be one apparent study whose results are able to conclusively allow for the resolution of this issue. Even the finding by Troscianko, et al. (1996) of a cerebral achromatopsic patient's motion perception being normal is lacking in power. While such a person is unable to see color, the function of the P pathway is entirely presumptive. Given that this individual suffered a bilateral stroke to the occipital cortex when in his mid-60's with little or no color vision problems prior to this, this observer cannot readily be compared to a genetic color-deficient on account of neural wiring. Similar to an acquired color-deficient observer, we have no indication with the achromatopsic as to whether there was active neural recruitment following the injury, if the observed compensation is due to practice, or if some additional factor or factors may be involved. Thus, in short, while such a finding points to motion perception being a function exclusively of the M pathway, it does not exclude the possibility that the M pathway might be able to take over P pathway responsibility (provided this pathway was even stimulated with a 1 cpd grating stimulus).

The cause of the observed differences between our groups of observers is still somewhat debatable. The notion brought up earlier regarding separate mechanisms for processing $d_{min}$ and $d_{max}$ is quite intriguing. Based on some of the ideas presented above, the question of why color-deficient observers would be at an advantage in one case, but

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not in the other, is one that can hopefully be solved with additional experimentation. The possibility that the observed differences are due to spatial frequency channels remains, although other factors probably play a role since the trend of enhanced sensitivity is not found for all degrees of displacement.

## Conclusions

In this study we have attempted to address several long-standing debates regarding the processing of visual information in human observers (including the functional separation of the parallel pathways) by way of a uniquely innovative comparison of color-normal and color-deficient individuals across a range of functions. The lack of power resulting from our use of small samples, coupled with the great diversity (and variability) of observers' visual functions, underscores the complexity of such an undertaking, reinforces the need for further research on the topic, and reemphasizes that to consider our findings conclusive as such would be premature.

While there were no significant differences between groups, we did find a tendency for genetic color-deficient observers to have considerably lower thresholds (on the order of being consonant with supersensitivity) on many of the dimensions measured, specifically those requiring fine spatial resolution/discrimination, compared to color-normals. The low variability among members of the genetic group on several of these tasks indicates that there may well be a mechanism common to all color-deficient observers (regardless of type and severity of deficit), but one that is different from, or absent in, at least some color-normals.

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Based on our results, it currently seems that the observed differences between groups may be a reflection of how the spatial frequency components of a stimulus or object are processed. Such elements, common in some capacity to the neural processing theories of all of the functions and tasks used, may be a link to some general, perhaps higher-order mechanism used in the processing of all visual information. We postulate this with some caution, for, as was discussed earlier, the type of stimuli used (i.e., high spatial frequency, low spatial frequency, mixed) is an essential factor needing to be considered in order to successfully confirm this. Additional support will come from the compiling of data from larger samples being run on the same battery of tests and apparatus as was our first sample, thereby avoiding the potential confounds invited through the comparison of data across different experimental conditions. We believe that our methodology and the stimuli utilized in this test battery are some of the best using currently available techniques, thus further increasing the power and credibility of our findings and speculative theories responsible for their occurrence.

To think that the main (and perhaps only) difference between color-normal and color-deficient observers is an altered (or "absent") P pathway in the latter group of individuals was too rudimentary on our part. Roorda and Williams (1999) have convincingly shown how differences in the retinal mosaic can lead to a loss of detection of color variations of high spatial frequency stimuli, with a resultant gain of luminance variation detection, in color-normals. From this we hypothesize that, given an even further reduction in antagonistic signals of color-related information from adjacent cells (or patches of cells) which occurs in color-anomalous observers as a result of having cones that are more congruent in spectral sensitivity, an enhancement in sensitivity of the

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sort seen in the genetic group is certainly conceivable. While it is possible that, anatomically, color-normal and color-deficient observers are no different in any other way, it far from precludes the possibility that there exists a vast array of alternative neural circuits and other resulting connections found within the various cortical layers of the color-deficients. That is, it is difficult to tell if any general color deficiency leads to the same neuronal reorganization, and the idea of spatial frequency channels as the only difference between color-normal and color-deficient observers may be a bit too simplistic.

Evidence for more widespread neuroanatomical differences leading to the observed neurophysiological and psychophysical differences come from studies that have been done using a limited number of neurologic populations. These studies, in conjunction with the present investigation, may be able to shed light on, and help answer questions that have previously remained inconclusive.

These neurologic populations alluded to above include persons suffering from the agnosias (see, e.g., Damasio, 1985), Alzheimer's disease (see, e.g., Cronin-Golomb, et al., 1993; Gilmore, Wenk, Naylor, & Koss, 1994), and albinism (see, e.g., Coleman & Sydnor, 1988; Coleman, Sydnor, Wolbarsht, & Bessler, 1979; Guillery, Okoro, & Witkop, 1975). In each of these instances, the M and/or P pathway is impaired, and the various functions regulated by the impaired pathway are found compromised to some extent.

Guillery, et al. (1975) found in post-mortem analysis of a human albino's brain that, in the LGN, cell groups that receive an abnormal (crossed) input tend to fuse with adjacent layers, which are also receiving a crossed input. Within the six-layer central part of the LGN, there were abnormal fusions between the P layers (possibly due to

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insufficient innervation), and only one distinct M layer was clearly recognizable. In addition, the bilaminar part of the nucleus was abnormally large, extending from rostral to caudal tip (probably due to fusion of layers at the borders of the four-layered segment, making the segment monocular); the nucleus had an abnormal orientation and had no swelling on the surface of the brain, as well as being partly rotated rostrolaterally. The authors concluded that the retinogeniculate pathways were abnormal, and that, due to many abnormal fusions, quite extensive parts of the retinogeniculate projections, representing both central and peripheral vision, must have been abnormal as well.

Color-deficient observers are apt to have considerably different anatomical abnormalities than those illustrated above in the albino. Yet, a common link shared by these two populations (i.e., different projections compared to color-normals), allows for further inquiry into additional similarities. If found, such likenesses will permit future studies to investigate human visual information processing through comparably paired reference groups.

While the dangers of generalizing post-mortem studies to populations that are alive and well was discussed earlier, and that such a probing procedure as was done in the above study would be deemed too invasive in nature to be performed on actual specimens, we are optimistic that the further advancements and refinements in neuroimaging procedures made in recent years will lead to a collaboration of several groups of scientists, and will limit the number of presumptions, thereby permitting a clearer view of the inner workings of the mind.

Neuroimaging has also helped in clarifying that the visual systems of human and non-human primates are not as similar as was initially thought (see, e.g., Tootell, et al.,

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1995), reinforcing the strength that would come from the comparison of different populations of humans.

Furthermore, we hope to gain additional insight into the connections between the various visual areas. How and where does cross-talk between the M and P pathways occur? Are the locations and amount of cross-talk fixed or somewhat variable? Could there be more cross-talk between the pathways in color-deficient observers because of the limited abilities of the P pathway? Are the observed enhancements in the genetic group the result of the interblob pathway serving as mediator to the parvocellular-blob and M pathways? Or are the differences between groups due to differences in the backward projections from the higher cortical areas to the lower cortical areas, and the information they provide?

While discussing neural networks in color-deficient observers, it might be worthwhile to mention that the current investigation may remedy the still-ongoing controversies surrounding the various models attempting to explain anomalous trichromacy (reviewed in Mollon, 1997; see also, Alpern, 1981; Alpern & Moeller, 1977; Alpern & Wake, 1977; Cicerone & Nerger, 1989; Jacobs, 1998; Jameson & Hurvich, 1956; MacLeod & Hayhoe, 1974; Pokorny & Smith, 1977). While not a central goal of this study, these models necessarily contribute to possible wiring schemes, neural recruitment, neural pooling, signal processing, et cetera, which are different from that found in color-normals, and which may result in some of the between-group differences that we found. This aspect should not be ignored, so that we may get the most complete picture of an individual's color vision.

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Finally, we need to address the issue of gender differences in visual perception. Given the large number of female color-normal observers used (compared to color-normal males) and the differences in sensitivities between the color-normal and color-deficient groups, some discussion is definitely warranted. Brabyn and McGuiness (1979) found that females differed from males in spatio-temporal contrast sensitivity, with females superior at low spatial and low temporal frequencies, and males superior at high frequencies. Such differences, however, may be largely explained as a function of the phase of the menstrual cycle that a given female observer is in at the time of testing. For reviews, see Guttridge (1994) and Parlee (1983). Studies have shown phase differences on several of the measures tested in the battery, including general visual perception (e.g., Barris, Dawson, & Theiss, 1978, 1980; Friedman & Meares, 1978; Scher, Pionk, & Purcell, 1981; Ward, Stone, & Sandman, 1978; cf. Jackson, 1965), spatial and temporal sensitivities (e.g., Asso, 1986; Becker, Creutzfeldt, Schwibbe, & Wuttke, 1980; De Marchi & Tong, 1972; Diamond, Diamond, & Mast, 1972; Dunn & Ross, 1985; Hampson, 1990; Johnson & Petersik, 1987; Wong & Tong, 1974; cf. H. W. Gordon & Lee, 1993), and acuity (e.g., Hampson & Kimura, 1988; Scher, Purcell, & Caputo, 1985). In addition, cyclic changes have been found to be associated with general cortical arousal (e.g., Braier & Asso, 1980), corneal sensitivity (e.g., Riss, Binder, Riss, & Kemeter, 1982), and the topography and thickness of the cornea (e.g., Kiely, Carney, & Smith, 1983), which may have a direct effect on any of the tasks used. With some studies reporting no differences (e.g., H. W. Gordon & Lee, 1993; Jackson, 1965), several questions remain, including whether the group of female observers used in our study is representative of the female population in general. Future research needs to control for

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menstrual cycle phase and, perhaps, use all-male groups in order to ascertain whether the observed group differences are real. The mere reduction of the color-normal group down to just male participants only replicates the lack of statistical power due to sample size currently found in the genetic color-deficient group.

In the end, the findings made while conducting the present study should, at best, be considered as preliminary. But the emerging trends should spark an interest for the pursuit of additional research on the topic of color vision deficiencies, bringing color vision research up to the next level, and redefining the very foundations upon which it is based. Irrespective of what the results might reveal, the implications of these findings will most certainly be widespread (for a review, see Bassi & Lehmkuhle, 1990).

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Appendix A

Protocols

**Approved use of this form will expire: 6/23/00**

## VISUAL TEST BATTERY -- INFORMED CONSENT

Applied Vision Institute and Visual Research Laboratory,
Department of Psychology, Brooklyn College of CUNY
Director: Israel Abramov, Ph.D., (Professor, Psychology)

Before you participate in our study, it is important that you understand the purpose and nature of this research study and what you will be asked to do if you participate.

### Nature of the Study

The brain extracts a wide variety of information from any visual image. However, there is considerable processing of the responses before anything is transmitted from the eye to the brain. There are several parallel systems of nerve cells, each carrying different sorts of visual information. But even within a system, each neuron responds to many aspects of the image. Ultimately, the brain must sort apart these streams of information and send the results to the different areas that deal with various visual capacities.

Our research is aimed at understanding which systems of nerve cells process different sorts of visual information. The strategy is as follows: for each observer, we will test many aspects of visual function. Once we have data from a large number of observers, we will ask how the results on each of the different measures relate to each other. Even though we expect considerable variation among observers, there should be high correlations among functions that depend on information transmitted by closely related neuronal pathways. Surprisingly, there do not exist published bodies of acceptable data from which such intercorrelations might be derived.

### Personnel

The study is being conducted in the Applied Vision Institute and Visual Research Laboratory. Israel Abramov, Ph.D. is the head of the laboratory. Collaborators include James Gordon, Ph.D., (Professor, Psychology, Hunter College), Nancy Oley, Ph.D., (Professor, Behavioral and Social Sciences, Medgar Evers College), Maria Pagano, Ph.D., (Assistant Professor, New York City Technical College/CUNY), Sharon Krinsky-McHale, Ph.D., (Postdoctoral Fellow, NYS Institute for Basic Research in Developmental Disabilities), Scott Brodie, M.D., (Professor, Ophthalmology, Mt. Sinai Medical School), Vance Zemon (Associate Professor, Albert Einstein College of Medicine). Senior personnel will be assisted in running the studies and in data collection and analysis by graduate and undergraduate research assistants.

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**Risks and Benefits**

There are no known risks associated with any of the tests. All of them measure your visual capacities from your external behavioral responses. There is no invasive procedure. Nothing will ever touch your eyes. The lights that we use are no more intense than in a normal interior environment. You will be asked to identify the presence, position, or orientation of stimuli, arrange stimuli in sequence, and describe the appearance of stimuli, etc.? Many of the tests will be conducted in a darkened room with only a small light or the test stimulus for illumination. You will also be given several questionnaires about your general medical and visual history and about other factors that might affect your vision.

You will not benefit directly from participation, but the results that are obtained may be of value someday to understanding vision in the general population. The results may also serve as a basis for comparison with vision in specific clinical sub-groups.

You are free to withdraw from this study at any time if you do not wish to participate. If there are specific questions you do not wish to answer, you do not have to answer them. If you have questions about your performance on any test, we would be happy to answer them.

**Confidentiality**

If you agree to participate, the first step will be to fill out several questionnaires. We will ask for your full name and address only so that we can get in touch with you to schedule appointments and in the unlikely event that we may need to contact you in the future. However, some of the questions we will ask about your general medical history may seem quite personal; these hand-written answers, together with your complete name and address will remain strictly confidential. You have the right to not answer a question if you do not want to. These questionnaires will be kept in a locked cabinet, accessible only to Dr. Abramov, the head of the laboratory. For all other tests, you will be assigned a coded ID and that is the only designation you need to give to any of the personnel who will run you through any of the subsequent tests. That is the only identifier that will appear on any computer records of your data.

None of your answers or identifiable data will be made available to anyone unless you specifically request that we do so. For example, you might want us to communicate some of our findings about your visual capacities with your physician. However, we will only do so if you specifically request this in writing. Also, should you want, we could provide you with names and addresses of other places to which you might go to follow up on any of our findings.

**Tests**

The full battery of tests requires a dozen sessions, each about 3/4 of an hour long; you may do more than one session per day with short breaks in between. Before each test you will be given a full description of what the test is about and exactly what your task will be during the test. Let me briefly describe all the tests you will take:

**(i) Optics & Acuity.** Your normal vision (acuity) in each eye will be evaluated by machine (autorefractor) and by asking you to look at letters from a distance on a computer screen. The procedure is similar to a standard optometric examination. If you

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need glasses to see clearly, or if you need a change in your prescription to see clearly, we will fit you with temporary frames and lenses which you will then use for the rest of the tests.

**(ii) Spatio-temporal Resolution.** In this test, you will look at alternating fuzzy-edged patterns of light and dark bars on a computer screen. These bars will be changing in width, contrast, orientation and rate of alternation. We will determine the smallest changes that you can detect (your thresholds) by asking you to press keys on a computer keyboard.

**(iii) Binocular Functions.** We will determine how well your two eyes are able to fuse two images into one using an optical system through which you will look at stimuli on a computer screen. To evaluate your ability to see things in depth (stereopsis) you will be asked to wear either polaroid or red-green goggles and to identify figures (e.g., butterfly, circle, square) that appear to pop out in depth from standard test plates.

Another aspect of this (stereoacuity) will be measured with a pattern of random dots on a computer screen. You will be asked to determine the location of the stereo target while you wear goggles with computer-controlled shutters to block your vision separately in each eye.

**(iv) Hyperacuities.** We will measure vernier and motion thresholds with stimuli presented on computer screens. For vernier thresholds you will have to decide if one segment of a line on a computer screen is displaced relative to the other segment. Minimum and maximum thresholds for perceiving motion will be measured from judgements of perceived displacement (up or down) of a random dot array. You will respond by pressing keys on a computer keyboard.

**(v) Color Vision.** The quality of your color vision will be assessed in many different ways. You will be tested with standard color-screening tests, including ones in which you rearrange small color chips in color sequence.

In another test you will observe small, circular, brief flashes of light against a dark background and will be asked to describe the color sensations evoked by each flash.

Finally, you will see circular, colored fields against a dark background and will be asked to adjust some aspect of one of them until it looks just like the other.

**(vi) Other Factors.** We will measure the amounts of light reflected by the iris of your eye and by a small patch of skin on your forearm -- these quantities are related to the amount of light that passes through the lens of your eye. We will also ask about demographic factors, such as the languages you speak -- these are relevant to interpretation of some of your data from the color vision tests.

**Consent form**

If you have any questions regarding this research, you can call Professor Abramov at 718-951-5454. If you have any questions concerning your rights as a participant in this study, you can call the Chairman of the Brooklyn College Institutional Review Board, Professor Glen Hass at 718-951-5601.

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I, (print your name)_____, have read this form. I understand that I may receive further information about any of the research procedures if questions occur to me now or in the future and I may withdraw from the research at any time with no penalty.

Signed:_____ Date:

I, _____, the designated researcher, have administered this form to the participant who signed above:

Signed:_____ Date:

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# CONFIDENTIAL PARTICIPANT INFORMATION
## (INTAKE1)

(Fill out form in PEN. CAPITAL letters only.)

| | |
|---|---|
| ID: | |
| LAST NAME: | |
| FIRST NAME: | |
| STREET ADDRESS: | |
| CITY: | |
| STATE: | |
| ZIPCODE: | |
| HOME PHONE: | |
| CONTACT PERSON: | |
| RELATIONSHIP: | |
| CONTACT PHONE: | |
| DATE: | |
| TESTER: | |
| COMMENTS: | |
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# GENERAL MEDICAL HISTORY FORM

**(INTAKE 2)**

**DATE: (MM/DD/YY)_____ EXPERIMENTER ID: (XXNN)_____ PARTICIPANT ID: (XXNN)_____**

*(Fill out in PEN. CAPITAL letters.)*

| # | SYSTEM | PROBLEMS WITH? | Y/N | ILLNESS, SURGERY, DAMAGE, OR CONDITION? | YR | FAMILY |
|---|---|---|---|---|---|---|
| 1 | Cardio-vascular | Heart/blood vessels | | | | |
| | | Blood pressure | | | | |
| 2 | Neurological | Hearing/touch smell/taste | | | | |
| | | Head or brain | | | | |
| | | Nerves | | | | |
| 3 | E-N-T | Nose/throat Smell/taste (sinusitis?) | | | | |
| 4 | Respiratory | Lungs | | | | |
| 5 | Gastro-intestinal | Liver | | | | |
| | | Stomach/ Intestines | | | | |
| 6 | Urogenital | Kidney | | | | |
| | | Urinary /Genital Tract | | | | |
| 7 | Musculo-skeletal | | | | | |

| # | SYSTEM | PROBLEMS WITH? | Y/N | ILLNESS, SURGERY, DAMAGE, OR CONDITION? | YR | FAMILY |
|---|---|---|---|---|---|---|
| | | Skin/breast | | | | |
| 8 | Endocrine | Hormones/ glands (thyroid, adrenal) | | | | |
| | | Metabolism/ Sugar regulation (diabetes) | | | | |
| 9 | Allergy/ Immunology | Immune system/ Allergies (sick or colds often?) | | | | |
| 10 | Hematology/ Lymph | Blood/ lymph | | | | |
| 11 | Constitutional | Weight (any problems with?) | | | | |
| 12 | Psych | Mood | | | | |
| | | Thinking | | | | |
| | | Memory | | | | |
| 13 | | Birth problems | | | | |
| | | Breast fed (formula can be a problem) | | | | |
| 14 | | Anything else? | | | | |



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# OCULAR MEDICAL HISTORY
## (INTAKE 3)

**VISUAL PROBLEMS** **GENDER_____AGE____PARTICIPANT ID(XXNN):_____**

*(Fill out in PEN. CAPITAL letters.)*

| PROB LEM | SELF | BIOLOGICAL MOTHER | MOTHER'S PARENTS (especially father) | MOTHER'S SIBLINGS (especially brothers) | MOTHER'S OTHER CHILDREN (specify) |
|---|---|---|---|---|---|
| COLOR VISION | | | | | |
| | | BIOLOGICAL FATHER | FATHER'S PARENTS | FATHER'S SIBLINGS | FATHER'S OTHER CHILDREN (specify) |
| | | | | | |

| | SPECIFIC | SELF | MOTHER | FATHER |
|---|---|---|---|---|
| OPTICS | MYOPIA (nearsighted) | age of onset: | age of onset: | age of onset: |
| | HYPEROPIA (farsighted) | age of onset: | age of onset: | age of onset: |
| | ASTIGMATISM (not same optic power in all meridians; some darker; lens cylinder) | age of onset: | age of onset: | age of onset: |
| ACUITY | AMBLYOPIA (uncorrectable acuity loss) | age of onset: | age of onset: | age of onset: |
| EYEMVT | NYSTAGMUS (horizontal) | | | |
| | STRABISMUS: EXO=walleye; ESO=crossed, nasal deviation. | | | |

| PROBLEM | SPECIFIC | SELF | MOTHER | FATHER |
|---|---|---|---|---|
| EYE MVT | HETEROTROPIA (misaligned up/down) | | | |
| | PHORIA (hidden misalignment; Maddox rod test; are they aware of it?) | | | |
| OCULAR DISEASE (eg.,glaucoma macular degeneration, cataract) | OD:<br>OS: | | | |
| OCULAR SURGERY (e.g.,cataract, eye alignment, glaucoma, retinal tear) | OD:<br>OS: | | | |
| OCULAR INJURY | OD:<br>OS: | | | |

| PRESCRIPTION & OVER-THE -COUNTER MEDICATIONS (recent or current) | PURPOSE | DOSAGE |
|---|---|---|
| e.g.; Eye Drops | | |
| | | |
| | | |
| | | |
| | | |



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# INTAKE

## DESCRIPTION OF PROCEDURE

The prospective participant is introduced to the battery of visual tests. The entire process is described to the participant, emphasizing the research goals for the battery as a whole and for many of the specific tests. The participant is told that additional information about each test will be given at run-time. Also, we clarify that the intent is not to "diagnose" an individual; the aim is research. However, if anything unusual is found, the participant will be told, any questions answered, and information made available to whomever the participant designates. After answering any questions, the participant is asked to read and sign the IRB-approved Informed-Consent Form.

Initial Intake instruments are only pencil-and-paper. They list information that is mostly confidential: participant's name, address, contact person, etc.; a detailed self-report about visual status and visual medical conditions; and general medical history with emphasis on situations that might impact on visual function. Each participant is assigned a coded ID, which is the only identifier used on all subsequent data forms/files.

Other demographic information is also gathered: age, gender, language(s), geographical origins of the mother's and father's sides of the family, and pigmentation (iris, hair, dermis). All these factors can impact on one or more of the visual tests.

## EXPERIMENTER'S INSTRUCTIONS (Intake)

### 1. Confidential Information (Intake)

All the intake forms are kept in a separate filing cabinet and are available only to senior lab personnel. These forms are administered only by senior lab personnel and are done one-on-one with the participant in a separate room.

*Note:* If the participant is female, the person doing the intake should also be female.

*Note:* All forms should be filled out in PEN and all names should be **printed in CAPITAL letters.**

Form INTAKE1 lists *confidential* information including name, address, etc., plus the *participant code* (**ID**) to be used in all subsequent data records for that participant. All data about each participant that will be used for subsequent analyses are identified only with the participant code given to each participant when INTAKE1 is filled out.

Form INTAKE2 lists information that might correlate with some aspect of vision. This includes general medical history, current health, medications, etc.

Form INTAKE3 lists information about the participant that is directly relevant to visual functions -- personal history of visual/ocular status and related medical events. Also, similar information about parents and siblings.

The final part of Intake is non-confidential and is entered directly into a computer – it asks about demographics of the participant.

Eventually, we will create a database and encode relevant portions of this information for use as co-variants in analyses of data from battery tests. This database will only use the ID as the identifier so that confidentiality is not breached.

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**2. Informed Consent.**

Read the instructions to the participant for informed consent. (These are exactly the same as on the form itself.) Ask the participant to sign two copies of the form. The experimenter should also sign both copies. The participant is given one copy, the other is placed with the other confidential information in the locked filing cabinet.

---

**PARTICIPANT'S INSTRUCTIONS: INFORMED CONSENT.**

(This is the same as the form the participant is asked to sign.)

**Approved use of this form will expire: 6/23/00**

**VISUAL TEST BATTERY -- INFORMED CONSENT**

Applied Vision Institute and Visual Research Laboratory,
Department of Psychology, Brooklyn College of CUNY
Director: Israel Abramov, Ph.D., (Professor, Psychology)

Before you participate in our study, it is important that you understand the purpose and nature of this research study and what you will be asked to do if you participate.

**Nature of the Study**

The brain extracts a wide variety of information from any visual image. However, there is considerable processing of the responses before anything is transmitted from the eye to the brain. There are several parallel systems of nerve cells, each carrying different sorts of visual information. But even within a system, each neuron responds to many aspects of the image. Ultimately, the brain must sort apart these streams of information and send the results to the different areas that deal with various visual capacities.

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You will not benefit directly from participation, but the results that are obtained may be of value someday to understanding vision in the general population. The results may also serve as a basis for comparison with vision in specific clinical sub-groups.

You are free to withdraw from this study at any time if you do not wish to participate. If there are specific questions you do not wish to answer, you do not have to answer them. If you have questions about your performance on any test, we would be happy to answer them.

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**Tests**

The full battery of tests requires a dozen sessions, each about 3/4 of an hour long; you may do more than one session per day with short breaks in between. Before each test you will be given a full description of what the test is about and exactly what your task will be during the test. Let me briefly describe all the tests you will take:

**(i) Optics & Acuity.** Your normal vision (acuity) in each eye will be evaluated by machine (autorefractor) and by asking you to look at letters from a distance on a computer screen. The procedure is similar to a standard optometric examination. If you need glasses to see clearly, or if you need a change in your prescription to see clearly, we

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will fit you with temporary frames and lenses which you will then use for the rest of the tests.

**(ii) Spatio-temporal Resolution.** In this test, you will look at alternating fuzzy-edged patterns of light and dark bars on a computer screen. These bars will be changing in width, contrast, orientation and rate of alternation. We will determine the smallest changes that you can detect (your thresholds) by asking you to press keys on a computer keyboard.

**(iii) Binocular Functions.** We will determine how well your two eyes are able to fuse two images into one using an optical system through which you will look at stimuli on a computer screen.

To evaluate your ability to see things in depth (stereopsis) you will be asked to wear either Polaroid or red-green goggles and to identify figures (e.g., butterfly, circle, square) that appear to pop out in depth from standard test plates. Another aspect of this (stereoacuity) will be measured with a pattern of random dots on a computer screen. You will be asked to determine the location of the stereo target while you wear goggles with computer-controlled shutters to block your vision separately in each eye.

**(iv) Hyperacuities.** We will measure vernier and motion thresholds with stimuli presented on computer screens. For vernier thresholds you will have to decide if one segment of a line on a computer screen is displaced relative to the other segment. Minimum and maximum thresholds for perceiving motion will be measured from judgements of perceived displacement (up or down) of a random dot array. You will respond by pressing keys on a computer keyboard.

**(v) Color Vision.** The quality of your color vision will be assessed in many different ways. You will be tested with standard color-screening tests, including ones in which you rearrange small color chips in color sequence.

In another test you will observe small, circular, brief flashes of light against a dark background and will be asked to describe the color sensations evoked by each flash.

Finally, you will see circular, colored fields against a dark background and will be asked to adjust some aspect of one of them until it looks just like the other.

**(vi) Other Factors.** We will measure the amounts of light reflected by the iris of your eye and by a small patch of skin on your forearm -- these quantities are related to the amount of light that passes through the lens of your eye. We will also ask about demographic factors, such as the languages you speak -- these are relevant to interpretation of some of your data from the color vision tests.

**Consent form**

If you have any questions regarding this research, you can call Professor Abramov at 718-951-5454. If you have any questions concerning your rights as a participant in this study, you can call the Chairman of the Brooklyn College Institutional Review Board, Professor Glen Hass at 718-951-5601.

I, (print your name)_____, have read this form. I understand that I may receive further information about any of the research procedures if questions occur to me now or in the future and I may withdraw from the research at any time with no penalty.

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Signed:_____ Date:

I, _____, the designated researcher, have administered this form to the participant who signed above:
Signed:_____ Date:

---

**3. Intake 1 (Confidential Participant Information).**

1. Read the instructions for Intake-1. *Note:* If the participant is female, the person doing this part should also be female.

---

**PARTICIPANT'S INSTRUCTIONS: INTAKE 1.**

"All the research information we will obtain from you will be identified only by a code designation. However, we should have your full name, telephone number, and address in case we have to get in touch with you. We also would like the name and telephone number of a contact person in case we cannot get in touch with you directly to make arrangements for additional test sessions.

"All this information will be kept strictly confidential. At this stage we will also assign you a coded ID that will identify all your other data. Please remember this ID because each assistant who administers a test to you will ask for it. Just to make sure, I will print your ID so that you can bring it with you to each test session."

---

2. Complete the form – Use a PEN! Write in CAPITALS!! Assign an ID and remind the participant to remember it.

2a. ID rule: **XXNN**, where XX = the participant's initials and NN = year participant began participating in the study -- if participation extends across two years, NN for ID does NOT change.

2b. Ask the participant if s/he has ever been a participant in a study at the Visual Research Lab at Brooklyn College. If yes, s/he may already have a lab ID and that is the one that must be used. In any case, check the list of all IDs to avoid any duplication. If the ID already exists in the list, use **XNNN**, where X = first initial, N = number from 1 to 9, NN = year.

2c. Print participant's ID on a small piece of paper so s/he does not have to remember it from session to session.

**4. Intake 2 (Confidential, General Medical History).**

1. Read the general instructions for this procedure (Intake-2) then follow the detailed script. *Note:* If the participant is female, the person doing this part should also be female.

---

**PARTICIPANT'S INSTRUCTIONS: INTAKE 2, General Medical History.**

"As you know, we are interested in your vision. I am going to ask you some questions about your past and current health. This is not a formal medical interview and

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you will not be asked to undergo a physical examination. I am one of the lab's senior personnel (doctoral student or Ph.D.), not a medical doctor, and therefore cannot answer medical questions. You may wonder what the various questions have to do with your vision. Although they may not seem to be related to vision, problems in one area (for example, high blood pressure or diabetes) can sometimes affect vision or lead to problems with vision. So please answer to the best of your ability. Your answers will help us to interpret your visual responses. Do you have any questions?

"Some of the questions may seem quite personal. Please be assured that your answers will remain confidential. Your name does not go on the answer sheet. Your answers will not be shared with any one except Dr. Abramov, the head of the laboratory. Once the results have been analyzed, the record will be kept in his locked safe. Your answers will not be given to any insurance company, medical person or any other authority. You have the right to not answer any of the questions. Do you have any questions about confidentiality?

"Now I'm going to ask you about a standard set of systems, from your head to your toes: Cardiovascular, Neurological, Ear/Nose/Throat, Respiratory, Gastrointestinal, Urogenital, Musculoskeletal, Endocrine, Allergy/Immunology, Hematology/Lymph, Constitutional, Psychological, and Birth experience. I will ask you if you have ever had problems with or taken medication for these systems. If you have, I will ask you the name of the illness, condition, injury, damage, or surgery you may have had related to each area. If you don't know the name, please try to describe the illness, etc. Are you ready to begin?"

---

2. Complete the form – Use a PEN! Write in CAPITALS!! Start with the PROBLEMS WITH? in column 1. Follow a set pattern for each item. *Note. Codes for Responses:* Y = yes, N = no; **DTA** = declined to answer; **DK** = don't know; **NA** = not applicable. Try to have a response for each item. Record comments, as needed, on back of sheet with asterisks and appropriate numbers. Print carefully. If the participant is having difficulty recalling, try asking generally if s/he ever had surgery, an accident or a major illness. Use the following script to go through the form. *Do not spend too much time on any one item.*

2a. Starting with *Cardio-vascular System*: **"Do you now have problems with or are you taking medication for your heart or blood vessels? Have you ever had problems with or have taken medication for your heart or blood vessels?"** Enter response. If Y, then go on to the ILLNESS, etc. column.

2b. **"Do you know the name of the problem you have now or have had in the past, or the diagnosis? What is it?"** (If the participant does not know the name, then try to get a very brief description of it: **"Can you describe the problem?"**).

2c. **"When did this problem start?"** Record year (col YR).

2d. FAMILY column: If answer to 2a was N, answer for family is NA. Otherwise: **"Is the problem you have/had with heart or blood vessels something that runs in your family?"** If yes, ask for some details but do not spend too much time on this question!

3. Repeat the pattern with all the other systems.

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3a. When you get to *Birth problems*, ask: **"Were there any complications or difficulties when you were born?"** (If yes, get explanation: **"What happened?"**)

3b. When you get to *Breast fed* ask: **"Were you breast fed as an infant?"** (If participant asks, we are interested in this because of a national controlled study of possible additives to baby formulas -- current formulas do not contain some of the fatty acids, e.g., DHA, in breast milk.)

4. **"That's it for general medical history. Now we have to go through some questions specifically about your visual system."**

**5. Intake 3 (Confidential, Ocular Medical History).**

1. Read the general instructions for this procedure (Intake-3) then follow the detailed script.

---

**PARTICIPANT'S INSTRUCTIONS: INTAKE 3, Visual History.**

"Now I am going to ask you some questions about problems you have had with your eyes and your vision. These questions will be about color vision, the optics of your eye, eye movements, eye diseases, eye surgery and eye injury. There will also be questions about your family's eye and vision problems. Your family is important because some visual problems are passed on from generation to generation, such as color blindness. These family questions apply to your biological parents only, not your adoptive parents if you were adopted or raised by others. If you do not know the name of the problem you have, please try to describe it. Are you ready?"

---

2. Complete the form – Use a PEN! Write in CAPITALS!! Start with COLOR VISION. **"Do you know if you have anything unusual about your color vision?"** The participant may not know of a diagnosed color deficit but ask for anything they might have noticed that is unusual about their color vision. Even if the participant does not report any unusual color vision, you must still probe about family history – the participant may be a *carrier* of an unusual gene even if she does not express it. *Note:* We are interested only in the participant's *biological* parents.

3. OPTICS. Ask about each of the categories by name (e.g., MYOPIA) -- most of our participants will know those words. If unsure, use the terms in parentheses (e.g., "nearsighted"). **"Now we will deal with any optical problems you might have. Do you have myopia -- that is, are you near sighted?"** If Y, **"Do you know your age when this was first diagnosed?"** -- enter under "age of onset". Repeat for parents.

4. Continue in similar fashion for other categories.

5. When you get to OCULAR DISEASE: **"Do you have, or have you had, any ocular disease -- for example, glaucoma, macular degeneration, cataract?"** If Y, check which eye (OD = right eye, OS = left eye).

6. Continue in similar fashion for other categories.

7. PRESCRIPTION, etc. **"Many medications can affect your visual performance. Are you currently taking, or have recently taken, any prescription drugs or over-the-counter medications?"** Probe for *any* medications -- not just those

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that the participant thinks might affect vision. Avoid descriptions like "the little pink pills" unless they can tell you what they were prescribed for or supposed to do -- only then is the lack of a name for the drug acceptable.

When finished with all the confidential Intake forms, staple the Intake-1 form (full name, etc.) to the lab's copy of the Informed Consent. Mark top right corner with the participant's ID. Bring **all** the confidential forms to Dr. Abramov so that they can be filed in his locked cabinet.

***Do NOT discuss any of the confidential information with other lab personnel!***

**6. Intake: Demographic Information**

1. Read the general instructions for this procedure, then follow the detailed script:-

---

**PARTICIPANT'S INSTRUCTIONS, Demographic Information.**

"We now want to gather some non-confidential demographic information about you and your origins. This will include things like your age and gender. Also, which languages you can speak -- this may help us interpret some of your answers in tests that require purely verbal responses. We also want to know about the geographical origins of your family, both on your mother's and on your father's side. We will give you specific instructions about each item as we get to it.

"Do you have any questions?"

---

WHO, TSTR --Standard participant or experimenter IDs (XXNN)
DATE--Enter as character string: "MM/DD/YY."
AGE-- Enter age in years in specified field *under* the AGE field of the database. Enter number of months over the age in years. Formula in database field converts entries to age with correct decimal fraction.
DOB--Date of birth using convention of DATE.
GEN-- Gender: male or female, M or F.

(Information about languages that the participant knows -- helps identify his/her ethnic origins. Also important for hue scaling, which uses linguistic categories for responses.)
**"Now we want to know which languages you can speak -- this will help us interpret some of your verbal responses in later tests."**
LANG1A, LANG1B – Participant's "first" SPOKEN language and CURRENT fluency of that language: **"What was the first language that you spoke?" "How fluent are you in that language *today*?"** Language entered as a string of 4 (must be four!) characters: 0NNX, or 00NX. First entries are *zeros* (not the letter O!), (N)N are from Catalog of language codes (*Catalog 9)* (e.g., 001 = English); add suffix for current fluency (E = excellent, G = good, F = fair, P = poor). If language does not exist in Catalog (*Catalog 9*), do NOT enter anything -- check with senior lab

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personnel (either immediately or after completing and saving this intake form) who will add to the catalog list.
If Participant indicates that two languages were equally "first," record the second one as LANG1B.

LANG2—Participant's CURRENT SPOKEN language (spoken most of the time, e.g., at home and with friends -- not just at work/school). **"What is the language that you currently use most of the time, especially at home and with friends?"** This can be the same as LANG1. Use LANG1 conventions.

LANG3—Participant's CURRENT SPOKEN language, OTHER than LANG1 or LANG2. **"Do you speak any other languages?"**

LANG4—Participant's CURRENT SPOKEN language, OTHER than LANG1, LANG2 or LANG3.

(*Geographic* origins of participant's family.) **"We want to know from which areas of the world your family comes."** (Note: we are only interested in origins of *biological* parents.)

MMORIGIN-- Origin of mother's mother's (maternal grandmother's) side of the family: **"First we will concentrate on your mother's side of the family, starting with your mother's mother -- your grandmother on your mother's side of the family. As far back as you know, where did that side of the family come from? Do you know the name of a town or city, a country, a geographical area?"** Type exactly as given by participant (you can enter up to 256 characters -- ignore fact that field may only display first part of you entry). This will be in spreadsheet only; later it will be converted to longitude and latitude for the database file.

MPORIGIN-- Origin of mother's father's (maternal grandfather's) side of the family: **"We continue with your mother's side of the family, but switch to her father's side -- your grandfather on your mother's side of the family. As far back as you know, where did that side of the family come from? Do you know the name of a town or city, a country, a geographical area?"** Use same conventions as above.

MMLONG--Longitude of MMORIGIN. Do NOT enter anything during intake. *Later* we will enter longitude and latitude of the places named by the participant. Sign conventions: + = North or West. If only a country is named, use values for capital.

MMLAT--Latitude of MMORIGIN.

MPLONG, MPLAT--Longitude and latitude of MPORIGIN.

PMORIGIN-- Origin of father's mother's (paternal grandmother's) side of the family: **"Now we will concentrate on your father's side of the family, starting with your father's mother -- your grandmother on your father's side of the family. As far back as you know, where did that side of the family come from? Do you know the name of a town or city, a country, a geographical area?"** Type exactly as given by participant.

PPORIGIN-- Origin of father's father's (paternal grandfather's) side of the family: **"We continue with your father's side of the family, but switch to his father's side -- your grandfather on your father's side of the family. As far back as you**

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**know, where did that side of the family come from? Do you know the name of a town or city, a country, a geographical area?"** Use same conventions as above.

PMLONG, PMLAT -- Longitude and latitude of PMORIGIN. Details as for MMLONG.
PPLONG, PPLAT --Longitude and latitude of PPORIGIN.

(Pigmentation. Spectral reflectivity of iris and skin, usually inner surface of forearm. These values should correlate with pre-retinal absorption.)

IRX, IRY -- Iris: CIE 1931 x, y chromaticity coordinates. Reset display format to Numeric, fixed, 3 decimals.

IRAL--Iris, albedo -- total reflectance. Reset display format to Numeric, fixed, 1 decimal.

DERX, DERY -- Forearm: CIE 1931 x, y chromaticity coordinates. Reset display format to Numeric, fixed, 3 decimals.

DERAL--Dermis (usually forearm), albedo -- total reflectance. Reset display format to Numeric, fixed, 1 decimal.

HAIR-- Natural, adolescent hair color (not dyed or aging color). **"We want to record the natural color of your hair when you were an adolescent or young adult. Your choices are brown, blonde, black, white, and red. You can modify your choice by adding light, medium, or dark."** Codes: BRN (brown), BLD (blonde), BLA (black), WHI (white), or RED (red), followed by L, M, or D for Light, Medium or Dark.

GRPCAT-- Group category, e.g., COLAB (color abnormal), DOWNS -- see Catalog. *(Catalog 8)* Leave blank for normal or unknown.

## COLOR1: COLOR SCREENING
## (Dvorine Plates, Farnsworth D-15, Lanthony D-15)

### DESCRIPTION OF TESTS

These tests are designed to detect variations in color vision from normal trichromacy. Variations from normal performance can be interpreted as evidence for genetic forms of dichromacy, anomalous trichromacy, and acquired (usually mixed form) deficits.

All consist of materials printed with broad-band pigments. These surfaces are fragile and can be damaged by excessive light exposure and direct touching. Choice of illuminant is critical in order to maintain the intended differences among the stimuli: tests are presented in a chamber whose illumination is controlled to provide illuminance of approximately 350 lux, with a color temperature of about 6800 K.

Since color deficits may only affect one eye, all tests are run monocularly.

**1. Dvorine Pseudo-Isochromatic Plates.**

(2nd edition, Harcourt, Brace & World, 1963)

There are 15 test plates in the book. Each plate consists of a pattern of dots, some of which may be seen to form numerals. The dots vary in size, brightness, and hue. Numerals and backgrounds have different hues, but the same ranges of dot size and brightness, so that perception of the numerals depends on distinguishing the hues of the dots. Diagnosis is based on failure to detect specific combinations of numeral and background hues. Useful mostly for identifying Protan and Deutan deficits.

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**2. Farnsworth Dichotomous Test for Color blindness, Panel D-15.**
(Psychological Corp., 1947)

Consists of 15 "caps" -- samples of Munsell chips in black plastic caps. Differ only in hue, chosen to go round the hue circle with approximately equal sensory spacing. All are moderately saturated. First cap is used as reference; participant's task is to place the remaining caps in color sequence. Major reversals in participant's sequence indicate color deficits; angle of reversal across the color circle is diagnostic for nature of deficit. Can indicate Protan, Deutan, Tritan, and Scotopic-only deficits.

**3. Lanthony's Desaturated 15 Hue Test.**
(Luneau Ophtalmologie, Paris)

A heavily desaturated version of the Farnsworth D-15. Even though much more difficult than the Farnsworth, normal trichromats produce sequences with only a few minor reversals. But any tendency to color deficit is magnified.

## EXPERIMENTER'S INSTRUCTIONS (COLOR SCREEN)

NOTE: For the battery all three tests are run in one session, in the order: Dvorine, Farnsworth, Lanthony. Follow the same procedure for each eye. Eye sequence will be given on the data spreadsheet.

**SETTING UP:**

1. Position the illumination chamber (EQUIP name: KELVBX) and lock the rear wheels.

2. Open trapdoor on top of the chamber *carefully*.

2a. *Check the Color-Temperature Filter!* The correct one is marked: "6800 K, 300 lux", and "Color Screening". If in doubt, remove the filter carefully, unclip the Plexiglas covers, and check the individual filters (avoid getting finger prints all over each sheet): Rosco Cinegel filter numbers 3202 [Full-B], 3204 [1/2-B], and 3216 [1/8-B]. NOTE: The filter numbers are printed on the **upper left corner** of each piece. Re-assemble and replace the filter pack carefully -- the Plexiglas cracks easily. Be sure that the filter is sitting evenly flat on the shelf! Close the trapdoor.

3. Switch on the power strip at the back of the chamber. This will also start the cooling fan. Adjust the Variac to 100% of its range (120VAC). Turn off the room lights.

4. Misc. steps: Have pencil and paper ready -- you may need to note unusual events. Check that clock with sweep second hand is working -- some steps must be timed. Turn on reading lights over computer keyboard and next to the light box. Have a floppy disk ready to transfer the data after you finish.

5. Prepare the test trays for Farnsworth and Lanthony tests -- see below.

**RUNNING THE EXPERIMENT:**

1. **Participant.**

1a. Position participant's head on chin rest and adjust its height for comfort.

1b. Have the participant place an eye patch over the eye not being tested; or use hand-held occluder.

1c. During each test, *watch the participant* carefully to note any unusual behaviors and to ensure that only one eye is being used!

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2. **Dvorine Plates.**

2a. Place the white book stand in the center of the illumination chamber; to ensure correct viewing distance (approx 50 cm), insert the spikes on the bottom of the stand into the holes in the floor of the chamber. Place the book on the stand. Open the test book to the demonstration plate. Make sure the transparent plastic overlay remains on top of the plate.

2b. Read the instructions (see Participant's Instructions, below) to the participant.

---

**PARTICIPANT'S INSTRUCTIONS:**
**Color Screening, Dvorine Pseudo-Isochromatic Plates.**

"Please sit down and make yourself comfortable.

"In this experiment we will attempt to measure your ability to detect different colors. In order to do this, you will be shown a series of cards. Each card is made up of many dots of different sizes and different colors. On each card, some of the dots form a number of one or two digits. All digits are the same size. For each card that you are shown, tell me what number you see. Some numbers may be difficult to see -- do your best.

"We will be testing each eye separately, which, as you may have already guessed, is why I have asked you to place an eye patch over one eye.

"Now, please place your chin in the chin rest. Tell me if the chin rest is too high or too low for you.

"Please try to be as accurate as possible. Finally, please do not touch the surfaces of the cards.

"Is everything clear?...Let's begin..."

---

2c. Ask the participant what number he sees. NOTE: It is virtually impossible for an individual not to see the number "48" on this plate. Any deviations probably indicate problems of a non-sensory nature.

2d. Present each of the remaining 14 plates for that eye. For each plate enter, in row marked "Resps", the participant's response -- you must enter the responses, even if they were correct. Allow *no more than* **5** *s* for the identification for each plate. Observe the participant for any hesitant or studied responses, as well as tilting and turning of the head; note the particular behavior and plate number on which it occurred on a separate sheet of paper.

2e. Turn back to the demonstration plate. Repeat for other eye.

3. **Farnsworth D-15.**

3a. The backside of each cap has a number corresponding to the correct sequence -- invert the tray and then open it to see the numbers. Caps are presented to the participant in their tray. Caps should be placed in random order *prior* to the start of the test session. (Invert closed tray, open it, place caps on table, mix them up, and replace them in that random order.)

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3b. Place the tray holder so that its locating rods are in the holes in the center of the illumination chamber. Place the tray on the holder with the fixed, starting (blue) cap to the participant's left. *Never* touch, or allow participant to touch, the colored surfaces of the test caps. If participant cannot deal with the caps properly have participant point to each cap and then you move the cap to the correct position.

3c. Read the instructions to the participant.

---

**PARTICIPANT'S INSTRUCTIONS: Color Screening, Farnsworth D-15.**

"In this experiment we will attempt to measure your ability to detect different colors. In order to do this, you will be presented with a set of 15 plastic caps or buttons, and you will be asked to arrange them according to color.

"We will be testing each eye separately, which, as you may have already guessed, is why I have asked you to place an eye patch over one eye.

"Please place your chin in the chin rest.

"The object of the test is to *arrange the buttons in order according to color.* Take the button from this panel (indicate) which looks most like this button (point to pilot cap) and place it here (point to space next to fixed pilot cap). Take the button which looks most like that, and place it here; and the button most like that and place it here. Continue doing this until all the buttons are arranged in order. In transferring the caps, please try *not* to touch the colored pigment in the middle of each cap. Begin."

[If participant does not comprehend, state the following:

"Take the button which looks most like that (point to fixed pilot button) and place it there (indicate next to pilot button)...and the button most like that (indicate previous one pointed to) and place it there (indicate next position), and so on".]

---

3d. Note the time at which the participant begins arranging the tray. This should not take more than about two minutes, but, if necessary, participants can take longer.

3e. When participant is finished, ask if satisfied with the sequence; if not, can re-arrange. When satisfied, remove tray and invert it so that cap numbers are visible. Enter that sequence in spreadsheet by overwriting the sequence shown.

3f. Repeat for other eye.

4. **Lanthony Desaturated D-15.**

Procedure is exactly the same as for the Farnsworth D-15.

---

**PARTICIPANT'S INSTRUCTIONS: Color Screening, Lanthony Desaturated D-15.**

"In this experiment we will again attempt to measure your ability to detect different colors. You will note that this test is very similar to the one which you have just completed. However, there are some important differences: for example, as you will see, the changes in color among the caps are more subtle. As a result, please pay close attention when arranging the colored caps or buttons.

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"We will be testing each eye separately, which, as you may have already guessed, is why I have again asked you to place an eye patch over one eye.

"Please place your chin in the chin rest.

"The object of the test is to *arrange the buttons in order according to color.* Take the button from this panel (indicate) which looks most like this button (point to pilot cap) and place it here (point to space next to fixed pilot cap). Take the button which looks most like that, and place it here; and the button most like that and place it here. Continue doing this until all the buttons are arranged in order. In transferring the caps, please try *not* to touch the colored pigment in the middle of each cap. Begin."

[If participant does not comprehend, state the following:

"Take the button which looks most like that (point to fixed pilot button) and place it there (indicate next to pilot button)...and the button most like that (indicate previous one pointed to) and place it there (indicate next position), and so on."]

---

5. **Saving the data.**

5a. Move cursor to beginning of spreadsheet and complete the entries for DVTYP, FATYP, and LATYP. These are the diagnostic categories for the three tests. (All other evaluations are done automatically by formulas in the spreadsheet.) If you are unsure about this step, you can carry on and save the data with these cells left blank -- they can be edited later. However, you *must* inform lab personnel that this has to be done.

5b. DVTYP: Check DVDEG for severity of deficit. Determine type of deficit from responses to plates 6, 7. Plate 6 ("95"); protan sees only 5, deutan only sees 9 (or gets only this digit correct). Plate 7 ("26"); protan sees only 6 (or gets only this digit correct), deutan sees only 2 (or gets only this digit correct). Enter diagnosis: <NORM = normal; PROT = protanoid; DEUT = deuteranoid; UNKN = unknown>.

5c. FATYP: Check graph of participant's sequences: click on Graph, Name, Display, specific graph. Enter diagnosis: <NORM1 = max two minor errors; NORM2 = several minor errors; PROT, DEUT, TRIT, SCOT = several major reversals parallel to corresponding axes; UNKN = real deficit, but no clear axis>.

5d. LATYP: Same procedure as for Farnsworth, except for normal category: <NORM = zero to several minor reversals>.

## COLOR2: COLOR DISCRIMINATION
## (Farnsworth-Munsell 100-Hue Test.)

### DESCRIPTION OF TEST

Measures variations in ability to discriminate among stimuli differing primarily in hue. Variations from normal performance can also be interpreted as evidence for genetic forms of dichromacy, anomalous trichromacy, and acquired (usually mixed form) deficits.

Consists of 85 "Caps" -- samples of Munsell chips in black plastic caps. Differ only in hue, chosen to go round the hue circle with approximately equal sensory spacing. All are moderately saturated. Divided into four trays, each sampling a quadrant of color space. In each tray, first and last caps are used as references; participant's task is to place the remaining caps in color sequence. Since the sensory distances between chips are

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small, errors indicate lowered discriminative capacity. Clustering of errors along certain portions of color space can indicate specific color deficits: Protan, Deutan, Tritan.

Materials printed with broad-band pigments. These surfaces are fragile and can be damaged by excessive light exposure and direct touching. Choice of illuminant is critical in order to maintain the intended differences among the stimuli: tests are presented in a chamber whose illumination is controlled to provide illuminance of approximately 350 lux, with a color temperature of about 6800 K.

Testing is binocular. However, if Color Screening (Color 1) indicates a difference between the eyes, each eye is tested separately. (Check previous data before running this test!)

## EXPERIMENTER'S INSTRUCTIONS (COLOR DISCRIM)

### SETTING UP:

1. Position the illumination chamber (EQUIP name: KELVBX) and lock the rear wheels.

2. Open trapdoor on top of the chamber *carefully*.

2a. *Check the Color-Temperature Filter!* The correct one is marked: "6800 K, 300 lux", and "Color Screening". If in doubt, remove the filter carefully, unclip the Plexiglas covers, and check the individual filters (avoid getting finger prints all over each sheet): Rosco Cinegel filter numbers 3202 [Full-B], 3204 [1/2-B], and 3216 [1/8-B]. NOTE: The filter numbers are printed on the **upper left corner** of each piece. Re-assemble and replace the filter pack carefully -- the Plexiglas cracks easily. Be sure that the filter is sitting evenly flat on the shelf! Close the trapdoor.

3. Switch on the power strip at the back of the chamber. This will also start the cooling fan. Adjust the Variac to 100% of its range (120VAC). Turn off the room lights and close the door.

4. Misc. steps: Have pencil and paper ready -- you may need to note unusual events. Check that clock with sweep second hand is working -- some steps must be timed.

5. Prepare the test trays -- see below. Place the tray holder so that its locator pins fit into the holes in the floor of the illumination chamber.

### RUNNING THE EXPERIMENT:

1. **Participant.**

1a. Position participant's head on chin rest and adjust its height for comfort.

1b. During each test, *watch the participant* carefully to note any unusual behaviors.

2. **F-M 100-Hue Test.**

2a. The backside of each cap has a number corresponding to the correct sequence -- invert the tray and then open it to see the numbers. Caps are presented to the participant in the lid of the tray. Caps should be placed in random order *prior* to the start of the test session. (Invert closed tray, open it, place caps on table, mix them up, and replace them in that random order. Replace them upside down -- when the tray is

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presented to the participant they will be right side up on the lid, while the two fixed reference caps are right side up on the bottom half of the tray.)

2b. Place the tray on the special holder in the center of the illumination chamber that keeps it inclined at the correct angle. *Never* touch, or allow participant to touch, the colored surfaces of the test caps. If participant cannot deal with the caps properly have participant point to each cap and then you move the cap to the correct position.

2c. Read the instructions to the participant.

---

**PARTICIPANT'S INSTRUCTIONS:**

**Color Discrimination: Farnsworth-Munsell 100-Hue Test.**

"Please sit down and make yourself comfortable. In this experiment we will attempt to measure how well you are able to discriminate between colors. In order to do this, you will be presented with a series of 4 trays, each consisting of 21 or 22 plastic caps, and you will be asked to arrange these caps according to color.

"At this time, please place your chin in the chin rest. Tell me if the chin rest is too high or too low for you.

"The object of the test is to arrange the caps in order according to color. Please transfer them from this panel (indicate) to this panel (indicate) and place them so they form a regular color series between these two caps (point to fixed pilot caps). In transferring the caps, please do NOT touch the colored pigment in the middle of each cap.

"It should take you about two minutes per panel. However, accuracy is more important than speed---so you will be told when the two minutes are up but the panel will not be taken away from you. Arrange them as best you can, but don't dawdle.

"Do you understand? Begin."

[If participant does not comprehend, state the following: "Take the button which looks most like that (point to left fixed pilot button) and place it there (indicate next to pilot button)...and the button most like that (indicate previous one pointed to) and place it there (indicate next position), and so on."]

---

2d. Note the time at which the participant begins arranging the tray. This should not take more than about two minutes. After that time quietly remind them that two minutes have passed, but then allow them to carry on at their own pace.

2e. When participant is finished, ask if satisfied with the sequence; if not, can re-arrange tray. When satisfied, remove tray and invert it so that cap numbers are visible. Enter that sequence in spreadsheet by overwriting the sequence shown -- while you are doing this, save time by starting the participant on the next tray. (If participant hears that sequence is not in numerical order and asks about this, say that caps come in all sorts of orders.)

2f. Repeat for the remaining trays according to the random sequence.

3. **Saving the data.**

FMTYPE: If the value in FMDX = SUPerior or AVG, enter N (for normal). If FMDX=LOW, examine the polar-coordinate graph of the errors for a bulge in

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one part of the circle. To do so, click on Graph, Name, Display, 100HUEPOLAR. Enter diagnosis, based on cap number at mid-point of bulge: protanoid, midpoint between 62 and 70; deuteranoid, midpoint between 5 and 7; tritanoid midpoint between 46 and 52. Possible entries: <N = norm; PB = prot/borderline; PM = prot/moderate; PS = prot/severe; DB, DM, DS = deut; TB, TM, TS = trit; XB, XM, XS = unknown.>

## HUE & SATURATION SCALING

### DESCRIPTION OF TEST

Several spectral color functions can be derived from magnitude estimation procedures: participants view a small rear-projection screen on which monochromatic light flashes are presented; stimulus wavelengths are spaced across the visible spectrum; the stimuli appear as circular, (1.0°) colored flashes against a "dark" background; all are made equal in luminance at a low photopic level (under these conditions they are all roughly equally bright). Different luminosity functions for younger and older participants. No fixation spots because target area is always just visible -- room is not totally dark. Brief buzzer cues participant 0.5 s before flash.

Stimuli range from 430 to 660 nm in 10 nm steps (24 total). Presented in blocks with random order; first block is "practice"; next four blocks are "experimental"; total 120 trials.

Participants use standard hue and saturation scaling methods (Gordon, Abramov, & Chan, 1994) to describe the color sensations evoked by each flash.

A participant's mean hue and saturation functions are processed to represent the stimuli in sensory coordinates on a Uniform Appearance Diagram (UAD). Spacing of stimuli on the UAD is used to derive: spectral loci of unique hues, wavelengths corresponding to other specific hue ratios, and wavelength discrimination.

Participants are run dark-adapted (5 minutes suffices -- stimuli are foveal); inter-trial interval has a minimum of 15 s to maintain adaptation state. Binocular viewing, unless there was a big difference between their eyes from Color Screening and Color Discrimination tests.

### EXPERIMENTER'S INSTRUCTIONS (Hue Scaling)

### SETTING UP & RUNNING THE EXPERIMENT:

1. Seat participant and switch off room lights -- this allows participant to dark-adapt while you continue setting up the computer.

2. When prompted, choose calibration file. YOUNGOBS = participants younger than 30 years; STANDOBS = participants older than 30.

3. Instructions for the participant will appear on screen. Read them. *Note*: If participant asks about number of color words that can be used, say "Try to use only one or two words to describe your sensation of hue."

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### PARTICIPANT'S INSTRUCTIONS (Hue Scaling)

(These instructions are displayed on the screen of run-time computer.)

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"Please sit down and place your chin on the chin rest. A beep is used to tell you to get ready. After each beep, a flash of light will be shown to you. As soon as you hear the beep, you MUST look at this screen <point>. After each flash, DESCRIBE your sensation of the color of the light. We want to know how each flash looked TO YOU, not how to mix paints to produce something like it.

"Divide YOUR sensation into TWO parts:

"1). The first consists of hue. You can describe this portion of your sensation using the words: RED, YELLOW, GREEN and BLUE. You can use single words or combinations of these words, for example Red and Yellow, or Yellow and Green, or Green and Blue, or Blue and Red. Your answers must be given in percentages and the hues MUST add up to 100 percent.

"2). For the second part of your sensation, you must state how saturated the stimulus appeared to you. SATURATION is the percentage of your entire sensation that was Chromatic. You may think of it as indicating the concentration of the hue. Therefore, if your sensation had NO hue, then the saturation was zero; if the entire sensation was only chromatic, that is, was ONLY made up of hue, saturation would be 100%.

"Please note that there are NO RIGHT or WRONG answers. Think CAREFULLY about your sensations and assign numbers accordingly.

"The first few trials will be practice trials to familiarize you with the procedures. Then, the experimental trials will follow. Is everything clear?"

---

4. Experimental RUN-TIME SCREEN will appear and program starts setting up monochromator.

Top left of screen indicates stimulus that will be shown first (on subsequent trails this will be the stimulus that had just been shown to the participant!); its wavelength is shown on a colored background -- this is simply a very rough indicator of what that stimulus might look like to a color-normal observer. Its purpose is to alert the experimenter to something drastically wrong in the participant's responses: e.g., if the wavelength is 530nm and the background is greenish, any response by the participant that is vaguely "green" (including blue-green and yellow-green) is acceptable; but if the response is "red", either the apparatus malfunctioned, or the participant did not understand the instructions. Do not say anything to the participant. Treat that response as a failure-to-see and the computer will automatically repeat the stimulus after resetting the apparatus. Use this option *very, very, very* sparingly.

5. PARTICIPANT'S RESPONSES.

5a. Responses are percentages of sensations of red (R), yellow (Y), green (G), blue (B), and saturation (S). They can be recorded in any order.

5b *Warn* participant that the next trial is about to begin -- if you took more than 15 s to record data, ENTER key will immediately trigger cueing buzzer followed by next flash; otherwise, inter-stimulus-interval timer will pause until 15 s are over before buzzing, etc. (Ignore any messages about aborting the inter-stimulus interval.)

5c. *VERY IMPORTANT! Participant who Misbehaves*! Some participants categorize hues -- use only 0 or 100%. Some participants insist on telling about paint mixing -- use B and Y together when they probably mean G. Pay attention to behavior

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during first block -- these trials are practice and not included in final analyses. If the participant misbehaves, pause after this block and remind the participant of some parts of the instructions: "Remember to use a full range of numbers to describe your sensations." and/or "Remember, we want you to tell us how each light *looked* to you; do not tell us how you would make it. Do you understand?" If participant still is puzzled, try adding: "For example, if it looks green, say that -- do not tell us that you could make it by mixing blue and yellow paints. But if it looks green, you can also tell us about the shade of green -- either greenish-yellow, or greenish blue, and so on."

## ANOMALOSCOPE

### DESCRIPTION OF TEST

**1. Introduction.**

We are using many standard color screening tests for the battery (see Color I, Color II) to obtain a good estimate of an observer's color vision. No one standard test alone is sufficient, since each test does not measure all aspects of color vision (or all aspects equally). Furthermore, investigators have noted that these tests are by no means "fool-proof" -- individuals who possess a deficit can pass a test, resulting in the deficit going undocumented, or the tests can yield conflicting diagnoses, or no specific diagnosis, except that "something is different".

To date, the best attempt at a fool-proof test is running the participant through anomaloscopy. This will clear up any controversial diagnoses that have arisen from the other tests, and also give us an indication regarding the degree of severity of any deficit.

For the battery, the anomaloscope tests involved in this procedure include heterochromatic flicker photometry (HFP) to estimate photopic spectral sensitivity, and the Rayleigh match to estimate the number and relative sensitivities of LWS- and MWS-cones. Also part of this apparatus, though not strictly part of anomaloscopy, is a test to estimate the sensitivity of SWS-cones.

(a) HFP: When a test light is alternated rapidly with a standard light of different hue, the two hues fuse and no hue flicker is seen (spectrally opponent P-cells have relatively low temporal resolution); however, if the lights are not equal in their ability to stimulate "intensity" responses (probably M-cells), the field will appear to flicker. The standard appears yellow; test lights appear red, or green.

A participant can adjust the intensity of the test light to minimize or even eliminate the flicker -- the two lights are now equally capable of stimulating the luminosity system and their relative intensities are measures of the participant's photopic sensitivity to these lights (the lights are viewed foveally and hence do not stimulate rods). Divergence from the standard observer's photopic sensitivity may be diagnostic: protanopes are much less sensitive at long wavelengths.

(b) Rayleigh Match: In additive color mixing, "primary" lights in one field can be mixed to match exactly the appearance of a test light in neighboring field. The ratios of the intensities of the primaries needed to produce a match (a visually equivalent stimulus, or metamer) are determined entirely by the spectral sensitivities of the visual system's fundamental spectral channels -- these could be the different cone types. For this specific match, all wavelengths are longer than about 520 nm; therefore, SWS-cones cannot

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participate effectively in any of the matches, which are determined only by LWS- and MWS-cones. A participant must adjust the relative intensities of additively mixed red- and green-appearing lights to match the appearance of a yellow field.

(c) Chromatic adaptation: Adaptation to a long-wavelength red (dominant wavelength = 658 nm) should differentially desensitize the cone most sensitive to that light. But, if only two cone types (LWS and MWS) are involved, this should not affect the Rayleigh match since the adaptation would affect both fields equally. However, if there are several types of, say, LWS-cone, which are then combined by the nervous system to act as a single chromatic channel, long wavelength adaptation might differentially change their sensitivities, thereby changing the spectral sensitivity of one of the channels involved in the Rayleigh match -- the two fields would no longer appear the same.

(d) SWS-cones: The Rayleigh match provides information only about LWS- and MWS-cones. For completeness, a measure of SWS-cone sensitivity is needed. Responses of these cones can be isolated by superimposing a test wavelength (violet-appearing) to which they are very sensitive on an adapting field (yellow-appearing) to which SWS-cones are completely insensitive, but which strongly adapts LWS- and MWS-cones. Increment thresholds measure SWS-cone sensitivity, but the values are based on intensity at the cornea; this will underestimate SWS-cone sensitivity since some of the short-wavelength test light will be absorbed by pre-retinal structures and never reach the cones. Pre-retinal absorption can be estimated by dark-adapting the participant and measuring rod sensitivity at short wavelengths: the discrepancy between measured thresholds measured at the cornea and the sensitivity of rods measured at the retina indicates how much light was lost in passage through the eye. These measurements just outside the fovea include losses due to lens pigmentation and macular pigment; the same measures several degrees away from the fovea exclude absorption due to the macular pigment. The pre-retinal measures can be used to correct the photopic SWS-cone increment-thresholds.

**2. Psychophysical Procedures.**

All thresholds are obtained from adjustment procedures -- forced-choice methods would be excessively time-consuming. All settings (participant's final values and sequences) stored for later analysis. Tests in following order:-

(a) HFP: Initial flicker rate is 25 Hz, which can be reduced to 20 or even 15 Hz, if participant cannot see this flicker. Yellow standard's luminance never varies; participant adjusts luminance of red or green to minimize flicker; participant can increase or decrease test luminance as much as necessary until satisfied with result; computer stores matching value. Order: RGGRRGGR -- total of 4 matches for each test light. Means of the matching luminances used as starting points for Rayleigh Match.

Luminance of yellow fixed at approximately 15 nits. Luminances of green and red lights will be able to be adjusted by the participant from the numeric keypad. Initial luminance values of the green and red lights at the start of each trial vary from slightly below that of yellow (approximately 12 nits), to greatly below that of the yellow (approximately 5 nits), to greatly above that of the yellow (approximately 25 nits), to slightly above that of the yellow (approximately 18 nits).

(b) Rayleigh Match: Bipartite field (2° vertically divided, no overlap of half-fields). Luminance of yellow standard half-field never varies. Matching half-field

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presented at each of 11 starting luminance ratios of R/G; ratios from 0%R to 100%R in 10% steps -- random order. For each one, participant can adjust ratio until additive field looks as similar as possible to standard -- free adjustment. Computer continuously calculates and holds the summed luminance of the R/G field constant and equal to luminance of yellow standard; luminance equation taken from HFP settings of given participant. When satisfied, participant can try to match fields exactly by adjusting overall luminance of R/G field while maintaining the final ratio. If result still not entirely satisfactory, participant can then readjust ratio of R/G, and so on, for up to 5 iterations.

(c) Chromatic adaptation: Participant adapts briefly (2 minutes) to red adapting field (20 nits). Entire HFP and Rayleigh match procedures repeated with all fields superimposed on red adapting field. All procedures the same, except that only 6 starting R/G ratios (20% steps) are used.

(d) SWS-cones: Participant must be dark-adapted. To reduce waiting time, this test usually done immediately after hue scaling. Instructions given to participant in darkness. Small, dim, red fixation light; stimuli presented first at 2.25° above fixation and then at 6° above fixation. Start by presenting green test slightly above expected absolute threshold; 200 ms on, 800 ms off, repeated continuously. Participant can practice adjusting intensity to absolute threshold while maintaining fixation; upper limit of adjust clamped to prevent excessive light adaptation. Followed by 2 minutes of dark-adaptation. For thresholds, stimuli started below threshold and participant increases luminance until just visible -- can adjust up/down if overshoots. Order: BGGBBGGB -- total of 4 matches for each test light. Complete measures close to fovea, then repeat at greater eccentricity. These values will be used to correct SWS-cone thresholds for pre-retinal absorption.

SWS-cone thresholds measured as increments on yellow adapt (31 nits); central fixation. Other details as for absolute threshold; 4 repeats.

## EXPERIMENTER'S INSTRUCTIONS (ANOMALOSCOPE)

There are two parts to this experiment: (a) HFP and Rayleigh matching, (b) SWS-cone thresholds. These are run as separate sessions. Ideally, the SWS-cone part should be tacked onto hue scaling -- SWS-cone requires complete dark adaptation, and after hue scaling a participant will be almost fully dark-adapted.

**SETTING UP (HFP and Rayleigh Match)**

1. You will be asked to align the field stops. *Note:* YOU the EXPERIMENTER must do this!!!

1a. Remove cover from the numeric keypad located below the anomaloscope; turn OFF the NumLock key. Replace the keypad cover (making sure that the #2, #4, #6, and #8 keys are exposed), and then, using the arrow keys, adjust the field stops to produce the bipartite field used for the Rayleigh match. The UP/DOWN arrows control one of the field stops, the LEFT/RIGHT arrows control the other. The optimum alignment is obtained when a thin black vertical line is observed through the middle of the field.

1b. If the participant to be run is present during alignment, before accepting this position, start lining up the chin rest. Seat the participant, and with the field still illuminated, move the chin rest so that the participant has an unobstructed view of the

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field using the eye to be tested; other eye covered with eye patch. Adjust height of chin rest so that eye is level with the center of the viewing aperture.

1c. Make certain that the position of the field stops has not changed, and then accept this position by pressing enter on the main computer keyboard.

2. Participant must be foveally dark adapted -- approximately 5 minutes. Turn off room lights, and begin reading the first part of the instructions to the participant; instructions will appear on computer screen.

---

**PARTICIPANT'S INSTRUCTIONS (Anomaloscope -- General)**

(These instructions are displayed on the computer screen.)

"Please sit down and make yourself comfortable. For this experiment, you must use the optical correction that you use for reading.

"In this session we will be measuring some further aspects of your color vision. By now your color vision has already been evaluated using a number of standard tests. However, there are certain subtle variations that the standard tests may not be able to detect. The tasks you will encounter in this session will range from adjusting intensities of flickering lights, to mixing colored lights to match another colored light. You will be looking directly at these lights through the peep-hole in front of the chin rest. You should concentrate only on the lighted field you are trying to adjust -- ignore any glare or reflections off of other surfaces inside the apparatus. You will be provided with an eye patch to wear over the eye that you are not using. Now, place your chin in the chin rest and keep it there throughout the experiment. If it is too high or too low please tell me. I will give you more specific instructions as the experiment continues. Are there any questions?"

---

*Note:* This test is always monocular -- color-anomalous participants MUST be run through the anomaloscope for each eye; color-normal participants should be run through once using their preferred eye; eye should be corrected for reading distance.

**RUNNING THE EXPERIMENT (HFP and Rayleigh Match)**

**1. Finding Optimal Flicker Frequency**

1. Read the instructions for Optimal Flicker to the participant.

---

**PARTICIPANT'S INSTRUCTIONS (Anomaloscope -- Flicker)**

"Individuals vary in their sensitivity to the rate at which a light is flickering. Before we get to the experimental trials, we need to find the best flicker rate for you. When asked, please indicate whether you see the field as flickering (i.e., non-stable). Are there any questions?"

---

2. Press SPACE to determine the rate of flicker at which participant will be run (either 25, 20, or 15 Hz).

2a. You will prompted to ask the participant "Is the field flickering? (i.e., non-stable)" The first stimulus is presented at 25 Hz. If the participant sees the field as

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flickering, type "Y" to continue with the rest of the experiment. If the participant does not see the 25 Hz field as flickering, type "N". This will present a stimulus at 20 Hz. Again, as prompted, ask the participant if the field is flickering, etc. *Note:* Even if participant reports not seeing 15 Hz as flickering, this rate will be used.

**2. Heterochromatic Flicker Photometry (HFP)**

A red or green light will be presented along with a yellow light, flickering in counterphase with one another at a rate of 15, 20, or 25 Hz (based on results of preliminary tests).

1. When the participant is sufficiently close to being foveally dark adapted, read the instructions for the HFP portion of the experiment to the participant.

---

**PARTICIPANT'S INSTRUCTIONS (Anomaloscope -- HFP)**

"In the first part of this experiment, you will be presented with a colored field that should appear to you as flickering. It is not important what color the field looks like, but rather that it is, in fact, flickering. Your task will be to change the field to the point where the flicker is either completely eliminated, or, at the very least, minimized. Do this by using the arrow keys located on the keypad in front of you. The up and down arrow keys constitute large step sizes (up increases, down decreases), while the left and right arrow keys constitute small step sizes (left decreases, right increases).

"When it appears that you have reached a point of minimum flicker, continue to go beyond that point, and then come back to it. If there seems to be a range over which the flicker is at a minimum, try and find the midpoint of that range.

"Do NOT stare fixedly at the light beam for any prolonged period of time. Blink frequently and even briefly look away from the field. Does the flicker remain at a minimum when you look back at the field? When you have found the point of minimum flicker, please inform me.

"You will be presented with 8 separate trials. Please do not touch any of the keys on the keypad until you are told. I will inform you when you may begin adjusting each field. Are there any questions?"

---

2. Press SPACE to initiate the first trial.

3. Ask the participant if the field as flickering (i.e., non-stable). If the participant is unsure, have the participant adjust the test luminance away from the initial starting point to "increase the apparent intensity of the flicker" and then adjust it back to minimize flicker.

3a. Occasionally remind the participant to concentrate on the *center* of the field and not a corner or edge.

3b. If the participant is unable to eliminate the flicker completely, remind him or her that the objective is to reduce the flicker to a minimum -- it may not be possible to eliminate flicker completely.

3c. Pay attention to the current luminance level displayed on the computer screen!!! If the participant reaches a maximum (approximately 48 nits) or a minimum luminance value (0 nits), tell her or him to go in the opposite direction.

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3d. Every so often (especially for the first few stimuli), when the participant has allegedly found the point of minimum flicker, ask if *all* of the arrow keys were used (i.e., not just the up/down large step size). Ask if he or she tried going beyond the matched point in both directions. Ask if the field looks the same after looking briefly away.

4. When the point of minimum flicker has been reached, press SPACE to record the current luminance setting, and to initiate the next trial.

5. Repeat until all 8 trials have been run.

**3. Rayleigh Match.**

1. Read the instructions for the Rayleigh match portion of the experiment.

---

**PARTICIPANT'S INSTRUCTIONS (Anomaloscope -- Rayleigh)**

"For this part of the experiment, you will be asked to make color matches. The field you were just looking at will now consist of two half-fields. On the left side will be a mixture of two colored lights and on the right will be a standard colored light. You will be adjusting the proportions of the two lights in the mixture field in order to make the mixture field look the same as the standard. You will do this by using the arrow keys on the key pad in front of you. The up/down arrows are for large steps (up increases, down decreases), and the left/right arrows are for small steps (left decreases, right increases).

"First, I will ask you to match the fields according to hue; then I will ask you to match the two fields according to brightness. We will be alternating between these two adjustments up to 5 times. You are urged to take your time, and use all the available keys.

"Remember: once you reach a point that appears to be a match, go back and forth to make certain. If there appears to be a range over which a match is possible, try to find the midpoint of that range. When the two fields are matched, we'll move on to the next trial.

"This may sound a bit confusing now, but I will prompt you with more specific instructions as we proceed. Remember not to stare at the half-fields for any prolonged period of time. Do blink, and occasionally look away briefly, especially after having reached a match point in order to assess whether the match holds. Do you have any questions?"

---

2. Press SPACE to initiate the first trial.

3. You will be prompted to ask the participant if the two fields match according to *hue.* If the participant answers "No" then type "N", and continue to step #4. If the response is "Yes" then type "Y", and go to step #5. (Note: A positive response to this question will not frequently occur, but this will test for true dichromacy).

4. Phase 1.

4a. Tell the participant "You may now begin to match the half-fields according to *hue*."

4b. Pay attention to the current luminance level, as displayed on your screen!!! If the participant reaches a maximum (approximately 48 nits) or a minimum (0 nits) luminance value, tell him or her to go in the opposite direction.

4c. Occasionally remind the participant not to stare at the stimulus.

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4d. Every so often (especially for the first few stimuli), when the participant has allegedly made a match, ask if *all* of the arrow keys were used (i.e., not just the up/down large step size). Ask if he or she tried going beyond the matched point in both directions. Ask if the field looks the same after looking briefly away.

4e. When the participant is finished, once again, ask if the two fields match in *hue*. If the participant is satisfied, press SPACE to record the luminance values of the matching R/G ratio.

5. You will then be prompted to ask if the two fields match according to *brightness.* If the participant answers "No" then type "N", and continue to step #6. If the response is "Yes" then type "Y", and go to step #7.

6. Phase 2.

6a. Tell the participant, "You may now begin to match the half-fields according to brightness or intensity."

6b. Pay attention to the current luminance level, as displayed on your screen!!! If the participant reaches a maximum (approximately 48 nits) or a minimum (0 nits) luminance value, tell him or her to go in the opposite direction.

6c. Occasionally remind the participant not to stare at the stimulus.

6d. Every so often (especially for the first few stimuli), when the participant has allegedly made a match, ask if *all* of the arrow keys were used (i.e., not just the up/down large step size). Ask if he or she tried going beyond the matched point in both directions. Ask if the field looks the same after looking briefly away.

6e. When the participant is finished, ask if the two fields match in *brightness*. If the participant is satisfied, press SPACE to record the luminance values.

7. When prompted, ask the participant if the two fields are identical. If the participant answers "No", then type "N" and go back to step # 4 to repeat phase 1 and (if necessary) phase 2. (*Note:* This repeat can be done up to 5 times.)

8. Repeat until all 11 trials have been run.

**4. Chromatic Adaptation: HFP.**

The format for this part of the experiment is identical to the earlier version, except for the adaptation light being on and all stimuli superimposed on it.

1. After the Rayleigh matching portion of the experiment, a red adapting field will be turned on. Have participant maintain fixation on this field in order to become adapted to this light. Read the instructions for this portion of the experiment to the participant.

---

**PARTICIPANT'S INSTRUCTIONS (Anomaloscope -- HFP+Red Adapt.)**

"Please keep looking through the peep hole. The remainder of this session contains no new procedures. Basically, we will be doing each of the previous procedures again, but this time in the presence of an additional colored beam of light chosen to chromatically adapt your visual system. We need to know whether any of your previous matches are changed by this adaptation.

"We begin, again, with reducing the amount of flicker you see in the test field. As before, your task will be to change the field to the point where the flicker is either completely eliminated, or, at the very least, minimized. Do this by using the arrow keys located on the keypad in front of you. The up and down arrow keys constitute large step sizes, while the left and right arrow keys constitute small step sizes.

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"When it appears that you have reached a point of minimum flicker, continue to go beyond that point, and then come back to it. If there seems to be a range over which the flicker is at a minimum, try and find the midpoint of that range.

"Do NOT stare fixedly at the light beam for any prolonged period of time. Blink frequently and even briefly look away from the field. Does the flicker remain at a minimum when you look back at the field? When you have found the point of minimum flicker, please inform me.

"You will be presented with 8 separate trials. Please do not touch any of the keys on the keypad until you are told. I will inform you when you may begin adjusting each field. Are there any questions?"

---

2. Press SPACE to initiate the first trial. All procedures the same as for previous HFP.

3. Ask the participant if the field as flickering (i.e., non-stable). If the participant is unsure, have the participant adjust the test luminance away from the initial starting point to "increase the apparent intensity of the flicker" and then adjust it back to minimize flicker.

3a. Occasionally remind the participant to concentrate on the *center* of the field and not a corner or edge.

3b. If the participant is unable to eliminate the flicker completely, remind him or her that the objective is to reduce the flicker to a minimum -- it may not be possible to eliminate flicker completely.

3c. Pay attention to the current luminance level displayed on the computer screen!!! If the participant reaches a maximum (approximately 48 nits) or a minimum luminance value (0 nits), tell her or him to go in the opposite direction.

3d. Every so often (especially for the first few stimuli), when the participant has allegedly found the point of minimum flicker, ask if *all* of the arrow keys were used (i.e., not just the up/down large step size). Ask if he or she tried going beyond the matched point in both directions. Ask if the field looks the same after looking briefly away.

4. When the point of minimum flicker has been reached, press SPACE to record the current luminance setting, and to initiate the next trial.

5. Repeat until all 8 trials have been run.

**5. Chromatic Adaptation: Rayleigh Match.**

The format for this part of the experiment is identical to the earlier version, except for the adaptation light being on and all stimuli superimposed on it; also, there will only be 6 starting R/G ratios instead of 11.

1. Read the instructions for the red-adapted Rayleigh match portion of the experiment.

---

**PARTICIPANT'S INSTRUCTIONS (Anomaloscope -- Rayleigh+Red Adapt)**

"Finally we will try to make color matches with this adapting light on. The routine will be the same: Do the two half-fields initially match? If not, then we'll enter phase 1. Phase 1 requires a hue match. Upon completing phase 1, you'll be asked if the two

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half-fields are identical in both hue and brightness? If they are, then we'll move on to the next trial; if not, then we'll enter phase 2, where intensity adjustment of the mixture half-field is made. Upon completing this, you'll be asked if the two half-fields are identical? If they are, then we'll go to the next trial; if not, we'll return to phase 1.

"Please note that you may NOT be able to make a perfect match! However, try to do as best you can in getting to as close a match as possible. Remember not to stare at the fields for any prolonged period of time. Do blink, and occasionally look away briefly, especially after having reached a match point in order to assess whether the match holds. When you return to looking at the fields after having looked away, WAIT a moment before answering if the match still holds.

"This time there are a total of 6 separate trials. Please do not touch any of the keys on the keypad until you are told. I will inform you when you may begin adjusting during each phase. Are there any questions?"

---

2. Press SPACE to initiate the first trial.

3. You will be prompted to ask the participant if the two fields match according to ***hue.*** If the participant answers "No" then type "N", and continue to step #4. If the response is "Yes" then type "Y", and go to step #5. (Note: A positive response to this question will not frequently occur, but this will test for true dichromacy).

4. Phase 1.

4a. Tell the participant "You may now begin to match the half-fields according to ***hue.***"

4b. Pay attention to the current luminance level, as displayed on your screen!!! If the participant reaches a maximum (approximately 48 nits) or a minimum (0 nits) luminance value, tell him or her to go in the opposite direction.

4c. Occasionally remind the participant not to stare at the stimulus.

4d. Every so often (especially for the first few stimuli), when the participant has allegedly made a match, ask if *all* of the arrow keys were used (i.e., not just the up/down large step size). Ask if he or she tried going beyond the matched point in both directions. Ask if the field looks the same after looking briefly away.

4e. When the participant is finished, once again, ask if the two fields match in ***hue***. If the participant is satisfied, press SPACE to record the luminance values of the matching R/G ratio.

5. You will then be prompted to ask if the two fields match according to ***brightness.*** If the participant answers "No" then type "N", and continue to step #6. If the response is "Yes" then type "Y", and go to step #7.

6. Phase 2.

6a. Tell the participant, "You may now begin to match the half-fields according to brightness or intensity."

6b. Pay attention to the current luminance level, as displayed on your screen!!! If the participant reaches a maximum (approximately 48 nits) or a minimum (0 nits) luminance value, tell him or her to go in the opposite direction.

6c. Occasionally remind the participant not to stare at the stimulus.

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6d. Every so often (especially for the first few stimuli), when the participant has allegedly made a match, ask if *all* of the arrow keys were used (i.e., not just the up/down large step size). Ask if he or she tried going beyond the matched point in both directions. Ask if the field looks the same after looking briefly away.

6e. When the participant is finished, ask if the two fields match in *brightness*. If the participant is satisfied, press SPACE to record the luminance values.

7. When prompted, ask the participant if the two fields are identical. If the participant answers "No", then type "N" and go back to step # 4 to repeat phase 1 and (if necessary) phase 2. (*Note:* This repeat can be done up to 5 times.)

8. Repeat until all 6 trials have been run.

**EXPERIMENTER'S INSTRUCTIONS (SWS-CONES)**

*Note:* Try to run this session immediately after hue scaling!!! This test needs a fully dark-adapted participant -- that is the state immediately after hue scaling, so do NOT switch on room lights if you plan to continue with this test.

*Note:* This test is always monocular -- color-anomalous participants MUST be run through the anomaloscope for each eye; color-normal participants should be run through once using their preferred eye; eye should be corrected for reading distance.

**SETTING UP (SWS-cones)**

1. You will be asked to align the field stops. *Note:* YOU the EXPERIMENTER must do this!!!

1a. Remove cover from the numeric keypad located below the anomaloscope; turn OFF the NumLock key. Replace the keypad cover (making sure that the #2, #4, #6, and #8 keys are exposed), and then, using the arrow keys, adjust the field stops to produce the bipartite field used for the Rayleigh match. The UP/DOWN arrows control one of the field stops, the LEFT/RIGHT arrows control the other. The optimum alignment is obtained when a thin black vertical line is observed through the middle of the field.

1b. If the participant to be run is present during alignment, before accepting this position, start lining up the chin rest. Seat the participant, and with the field still illuminated, move the chin rest so that the participant has an unobstructed view of the field using the eye to be tested; other eye covered with eye patch. Adjust height of chin rest so that eye is level with the center of the viewing aperture.

1c. Make certain that the position of the field stops has not changed, and then accept this position by pressing enter on the main computer keyboard. *Note:* While the bipartite field will not be used in this session, proper alignment is needed to insure that the field stops are completely *out* of the field of view.

2. Place inconel 3 log unit neutral density filter into holder directly behind cover glass near the exit point of the optical path. If it needs to be cleaned, use Windex and Kimwipes *only*!!!

3.Participant must be dark adapted -- at least 10 minutes. *Note:* If this session is run following Hue Scaling, participant is already sufficiently dark adapted! Turn off the room lights, close the door, and begin reading the first part (introduction) of the instructions to the participant; instructions will appear on the screen.

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**PARTICIPANT'S INSTRUCTIONS (SWS-cones -- General)**

(These instructions are displayed on the computer screen.)

"Please sit down and make yourself comfortable. For this experiment, you must use the optical correction that you use for reading.

"In this session we will be measuring some further aspects of your color vision.

"As we age, our vision changes; some of the ages are in the eye's optics, others include reductions in sensitivity to certain portions of the color spectrum. We are interested in this because some of the changes may be early indicators of possible damage to the retina or ophthalmic diseases. It has been suggested that in some of these cases it may be possible to take protective measures to slow, or even reverse, the changes.

"There are several tasks in this session, including measuring absolute threshold for various colored lights -- that is, the least amount of light your eye can detect; these lights will appear at different distances from the fixation target, so we can measure sensitivity at different places on the retina.

"We have tried to build this apparatus so that the test fields you will see are as clean as possible. However, it is impossible to eliminate all unwanted reflections and glare; try to ignore any reflections and extra images that are not part of the specific test fields that you will be instructed to adjust. To help you do this, you will be provided with an eye patch to wear over the eye that you are not using."

---

**RUNNING THE EXPERIMENT (SWS-cones)**

**1. SWS-cones: Blue and Green Absolute Limens.**

This portion of the session is used to find mean absolute thresholds for blue and green lights at two parafoveal eccentricities: 2.25° and 6°. Trials are run according to *ascending* adjustment procedures to insure that no adaptation occurs due to too high an initial luminance. However, in order to instruct the participant, a low luminance stimulus demonstration flash, along with the 2.25° eccentric fixation target, will be shown initially and the participant allowed to test the keypad they will be using to adjust intensity. This orientation is followed by a two-minute black-out to allow the participant to readapt to the dark and then thresholds are measured for this eccentricity.

Next, the 6° eccentric fixation target is lit along with a low luminance field. Since the task for the remaining trials is identical to those preceding, this time there is no opportunity for the participant to test the keypad. This trial is simply to inform the participant as to where to keep fixated during the series of trials. Following a two minute black-out period, the second series of test trials is run.

1. Read the instructions for Blue-Green Absolute Limen to the participant.

---

**PARTICIPANT'S INSTRUCTIONS (SWS-cones: Blue-Green Absolute Limen)**

"First, we will measure the light intensity at which you are just able to detect a flashing light against a dark background. The flashing light will not be directly in front, but will appear a little above a small, red fixation target. The fixation target is always on and you must keep looking directly at it -- you must concentrate on this and avoid looking directly at the place where the flashing test field appears.

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"Each trial will start below threshold -- that is, the intensity of the flashing light will be too low for you to see it. You will increase the intensity until you can *just* see it. Do this using the arrow keys on the keypad in front of you. The up/down keys are for large steps (up increases, down decreases), and the left/right keys are for small steps (left decreases, right increases).

"We'll start with a practice trial so you can practice using the arrow keys. For this practice series we will start with a flashing test light that is *above* threshold and is clearly visible. Remember: real trials will not start at as high an intensity!

"While you are adjusting intensity, you MUST keep fixating on the small, red fixation light -- DO NOT look directly at the flashing test light. However, you should also remember to blink a few times between adjustments.

"During this practice series and during the real trials, whenever the flashing field is clearly visible (is above threshold), you should decrease intensity so that it drops below threshold and then increase intensity in small steps until you are just able to detect it. You can repeat this up and down adjustment until you have found your threshold. Tell me when you are done.

"After practicing, there's a 2 minute delay. The 16 test trials follow. After the first 8, I'll show you a new fixation target, and then continue with the remaining trials. Please do not touch any of the keys until you are told. I'll inform you when you may begin adjusting. I'll also remind you of the instructions as we go on. Are there any questions?"

---

Press SPACE to initiate demonstration trial.

2. Ask participant if they see a flashing or pulsing field above the fixation target. Remind participant to keep fixating. Allow the participant to test all of the keys on the keypad. Do not permit this to go on longer than one minute.

2a. Tell participant to relax, but to continue looking in the general vicinity of the fixation point.

2b. Terminate the demonstration trial by pressing SPACE.

3. A two minute delay period follows allowing for the participant to re-dark adapt.

3a. Watch the screen for the interstimulus interval to be over.

3b. Press SPACE to begin the experimental trials.

4. Instruct participant to fixate on the fixation point, and maintain fixation while increasing the luminance to threshold.

4a. When they have reached a threshold, ask if they dropped back below threshold (i.e., test flash invisible) and increased again to the point of threshold (i.e., test flash just barely visible). Make certain that final adjustments were in small steps [right/left].

4b. When the participant is satisfied with their threshold, press SPACE to record value and to initiate next trial.

5. Repeat step #4 until each of the first 8 trials with the near fixation point have been run.

6. At conclusion of 8th test trail, the far fixation point will be lit for second demonstration trial.

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6a. Inform participant that this is the new fixation point. Tell participant that they may or may not see a demonstration flashing light. There is no practice adjustment. Inform participant that the remaining trials will be identical to those which had just taken place, but fixation must be maintained at this new position.

6b. Press SPACE to terminate demonstration trial.

7. A two-minute delay period follows allowing for the participant to re-dark adapt.

7a.Watch the screen for the interstimulus interval to be over.

7b. Press SPACE to begin the experimental trials.

8. Instruct participant to fixate on the fixation point, and maintain fixation while increasing the luminance to threshold.

8a. When they have reached a threshold, ask if they dropped back below threshold and increased again to threshold.

8b. When the participant is satisfied, press SPACE to record value and to initiate next trial.

9. Repeat step #8 until each of the remaining 8 trials with the far fixation point have been run.

**2. SWS-cones: Increment Threshold**.

This part of the session will measure thresholds of the SWS-cone pathway: SWS-cone responses will be isolated by superimposing blue test flashes on an intense yellow background designed to suppress LWS- and MWS-cones while leaving SWS-cones unaffected. Test trials start after a 2-minute adaptation to the background, which remains on throughout this portion of the session. Test trials are, again, run according to *ascending* adjustment procedures to insure that no SWS-cone adaptation occurs due to too high an initial test luminance.

1. Remove the inconel filter from the filter holder, and return it to its envelope.

2. Read the instructions for the Increment Threshold portion of the experiment to the participant.

---

**PARTICIPANT'S INSTRUCTIONS (SWS-cones: Increment Threshold)**

"The next part of the experiment will measure the sensitivity of your visual system to short-wavelength light. For this, the test flashes will be added onto a constant adapting field. As you look through the peephole, you should see a big, bright, yellow, adapting field. Test flashes will appear in the middle of that field.

"Your task will be similar to the last one, except you will be fixating on the center of the adapting field. Your task will be to locate the intensity at which you are just able to detect the presence of a flashing test light against this bright background. Again, the test flash will start at an intensity that is invisible and you must increase to threshold.

"Increase the intensity of the test flash light using the arrow keys located on the keypad in front of you. The up and down keys constitute large steps (up increases, down decreases), while the left and right keys constitute small steps (left decreases, right increases).

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"When you feel you've reached a threshold, it's important that you drop back down below threshold and increase again (in small steps, using the right arrow key), until you are just able to detect the presence of a flash.

"You should always be looking at the center of the yellow adapting field. However, do NOT stare fixedly at it! Blink frequently.

"When you're convinced that you've found your threshold, please inform me. There are 4 separate trials. I'll inform you when you may begin adjusting test flash intensity -- until I tell you to, please do NOT touch any of the keys on the keypad. I'll remind you of the instructions as we go on. Are there any questions?"

---

Press SPACE to initiate adaptation.

3. Tell participant to look directly at adapting field for a period of 2 minutes. *Note:* This is also the first trial! DO NOT allow participant to press any keys until the 2 minute period has elapsed!

4. Have participant begin to increase the test flash luminance.

4a. When they have reached a threshold, ask if they dropped back below threshold (i.e., test flash invisible against the bright background) and increased again to the point of threshold (i.e. just barely detecting the presence of a flash against the bright background). Make certain that final adjustments were in small steps [right/left].

4b.When the threshold is reached, remind participant to blink a few times in order to assess whether the stimulus remains visible.

4c. When the participant is satisfied, press SPACE to record value and to initiate next trial.

5. Repeat step #4 until all four trials have been run.

## SPATIO-TEMPORAL CONTRAST SENSITIVITY

### DESCRIPTION OF TEST (CSF)

This test measures the spatial and temporal resolution of the visual system to monochrome targets that vary in contrast, while keeping mean luminance constant. Stimuli are sinusoidal gratings whose Michaelson contrasts vary from near zero to near 100%. The spatial frequency of the gratings can be changed as can the rate at which the patterns are temporally modulated; temporal modulation is counterphase with a sinusoidal profile. The luminances of the test display and its surround are photopic. The resulting data should allow complete description of a photopic spatio-temporal solid.

Total of 30 stimuli: 6 spatial frequencies (0.6, 1, 2, 5, 12, 24.4 cpd) and 5 temporal frequencies (1, 4, 8, 15, 24 Hz). There is no static condition both for analytic reasons (logarithmic abscissa) and practical considerations -- eye movements prevent a true static condition. Temporal modulation is sinusoidal and counterphase -- this avoids the eye-tracking, which would thereby stabilize a drifting grating. Stimuli viewed in circular aperture to minimize edge artifacts.

Psychophysical procedures: Test assumes that when optics are corrected there is no significant difference between thresholds for horizontal and vertical gratings. Two sessions required. In first session participant views each stimulus (random order and

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randomly horizontal or vertical) and adjusts contrast to threshold (each stimulus presented once); criterion should be ability to just detect orientation. Stimulus present until participant ends trial.

Adjustment data used to set starting points for next measures using *adaptive, forced-choice* procedures. Random subset of 15 stimuli for first session; remainder in separate session well-separated from first (to check on possible practice/order effects). Use QUEST algorithm: on each trial participant must vote whether grating was horizontal or vertical; feedback provided; performance determines contrast on next trial. Session's 15 stimuli presented in 3 blocks of 5 interleaved sequences -- in general, each trial displays a different stimulus. Each QUEST proceeds until reach 99% confidence. Stimuli whose thresholds have been attained are still presented at varying contrasts until all QUEST sequences in a block are finished. Record threshold contrast plus standard deviation (slope of psychometric function at threshold) for each stimulus. (If QUEST cannot find a stable threshold, series will eventually terminate and a dummy value entered.)

QUEST trial is fixed duration (2s) and participant votes, either during or after trial to minimize transients, stimulus is "ramped" on and off for half s each (i.e., remains at full contrast for given trial for 1s).

Results should apply to central/foveal region -- even though participants not prevented from using peripheral retina, tendency is to look directly at stimulus. Range of stimuli is a compromise dictated by need to keep accommodation (i.e., viewing distance) and display size constant. At 0.6 and 1 cpd fewer than 6 cycles present on screen, which may artificially lower sensitivity as compared with larger display size -- but this applies equally to all participants and may also enhance differences among participants. Lowest temporal frequency (1 Hz) may approximate static condition because of limited trial duration.

Participants usually tested with binocular viewing, unless there is some major difference between the eyes. Can wear own corrective lenses, if appropriate, as found during Intake and Acuity tests; otherwise, they wear appropriate lenses in a trial frame.

## EXPERIMENTER'S INSTRUCTIONS (CSF)

### SETTING UP & RUNNING THE EXPERIMENT

1. Participants must have their "best" optical correction! If they have a prescription correction, check that it is acceptable for tests that require optimal correction -- if not, use the appropriate lenses in a trial-frame (see Acuity procedures).

2. The procedure is divided into 2 sessions: the first session consists of adjustment, usually followed immediately by QUEST; each procedure generates separate set of data files, but if procedures are run one after the other, the *same* Battery Session number is used -- if the first set of QUEST data obtained on a separate day from adjustment, assign a new session number. The second session uses only QUEST, and must be run on a separate day, preferably much later in the battery.

3. Place the display screen and table with chin rest in correct positions; measure the distance using the 3.6m rope.

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**FIRST SESSION (CSF)**
**(Adjustment)**

1. Turn the computer screen towards the wall so participant is not disturbed by it. Place the keyboard cover over the keyboard, and put the keyboard on table in front of participant. For the adjustment session, the participant uses only the keys marked "D" (decrease), "I" (increase), and Enter.

1a. Turn off the room lights and shut the door so that the participant can adapt to the display luminance.

1b. Read the instructions for the adjustment session to the participant.

---

**PARTICIPANT'S INSTRUCTIONS: ADJUSTMENT (CSF)**

<EXPERIMENTER: point to the various items as you mention them in the Instructions!>

"Please sit down and make yourself comfortable. For this experiment, it is important that you wear any optical correction you might need for seeing distant objects clearly. We are going to measure the capacity of your visual system to detect different patterns, ranging from coarse patterns to very fine patterns displayed on this screen at the far end of the room. The results will allow us to specify an attribute of vision that is known as the Contrast Sensitivity Function.

"Specifically, we are trying to find the least amount of contrast in a pattern of dark and light areas for you to barely detect that pattern. That contrast level is called your Contrast Threshold; it tells us something about the sensitivity of your visual system to different patterns. To find these thresholds, you will be shown several computer-generated patterns of alternating light and dark bars; all the bars have fuzzy edges. We refer to these patterns as Gratings.

"You will be asked to judge in which orientation each grating is presented. There are two possible orientations: vertical (in which the bars extend from the top to the bottom of the screen) and horizontal (in which the bars extend from the left to the right of the screen). In addition, the grating patterns will be flickering at different rates.

"The experiment will be divided into two sessions and will use different procedures. In this session we will begin with an Adjustment Procedure to get a rough estimate of your threshold; later, we will use those values as starting points for a procedure that will measure your threshold with greater precision. Since the Adjustment Procedure is used only to get an estimate of your threshold, please do not spend too much time on every stimulus.

"At this time, please place your chin in the chin rest and keep it there during your observations. Try to avoid tilting or turning your head too much. If the chin rest is too low or too high, please tell me. Now look at the screen and keep looking there throughout the experiment. However, please do NOT stare fixedly at any one point on the display.

"A BEEP is used to tell you to get ready for a stimulus presentation. Your task will be to adjust the visibility, or contrast, of the pattern using INCREASE (labeled "I") and DECREASE (labeled "D") keys located on the keyboard in front of you. You must continue doing these adjustments until you can barely detect the orientation of the stimulus. In other words, your task is to reduce visibility until you are just able to say what is the orientation of the grating and NOT whether you can see some sort of

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flickering pattern. When you are finished adjusting, press ENTER to record your threshold.

"To initiate the next trial, you must press ENTER again. A BEEP will sound, followed immediately by the pattern. On each trial you must make at least one adjustment before trying to record your data.

"Briefly: after the BEEP, use the I or D keys to increase or decrease contrast until you can barely tell which way the grating is oriented. Press ENTER to record your threshold. Press ENTER to start the next trial.

"Is everything clear? You may begin when ready..."

---

2. ENTER starts a trial; D and I change the contrast of the grating in small (linear steps); the participant adjusts until satisfied; ENTER stores the results, produces a BEEP, and starts next trial. Note: at least one D or I press must be registered for each trial! The experimenter *must watch* the computer screen: bottom (red) portion lists the stimulus number (1-30) -- this red screen disappears when a stimulus is on the display screen; upper right displays the contrast of the grating (scale, 0-1); if the participant increases the contrast to 1.0 and still cannot see the grating, tell the participant simply to ENTER.

3. A sequence of BEEPS announces the end of the adjustment procedure. (Experimenter should have warned the participant so that there is no attempt to add more thresholds.)

4. Remove the keyboard cover, and ignore Vision Works' request for participant data, etc.; press ESC, and choose the option to exit VisionWorks. If this is the first CSF session and the QUEST is to be run immediately, choose that option. The participant may relax for a brief interval before starting. If the first QUEST session is on a separate day, then simply start with the option to run a QUEST session.

**FIRST SESSION (CSF)**
**(QUEST)**

5. Read the instructions for the QUEST session to the participant.

---

**PARTICIPANT'S INSTRUCTIONS: QUEST (CSF)**

"Please sit down and make yourself comfortable. For this experiment, it is important that you wear any optical correction you might need for seeing distant objects clearly.

"In this session you will be shown the same kind of stimuli as in the previous session in which we started measuring your Contrast Sensitivity Function. However, this time we will be trying to measure your threshold with greater precision. For this, we will use a different procedure, known as the QUEST procedure.

"In this procedure your task will be to decide whether the grating pattern was vertical or horizontal in orientation. To record your decision, you must press either the response key marked "V" for Vertical, or the key marked "H" for Horizontal. These response keys are located on the keyboard in front of you.

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"The orientations of some grating patterns will be more obvious than others. In fact, in some instances you may not be able to see any pattern at all. But you must still make a choice. Even though this may be difficult, and you may think you are guessing, try to do your best on every trial. If you respond correctly, the next trial will start automatically. If, however, you respond incorrectly, you will hear 3 short beeps -- the next trial will follow immediately after those beeps. It is OK to be incorrect. Remember, we are attempting to find your contrast threshold; that threshold is the level at which you are as likely to be correct as incorrect -- this means that there will be many incorrect responses. Nonetheless, we still want you to try hard on each trial. Do not give up when it gets difficult.

"At this time, please place your chin in the chin rest and keep it there during your observations. Try to avoid tilting or turning your head too much. If the chin rest is too low or too high, please tell me. Now look at the screen and keep looking there throughout the experiment. However, please do NOT stare fixedly at any one point on the display.

"Trials will start automatically. A single BEEP signals the start of a trial. Immediately after the BEEP, a grating pattern will be presented on the screen for two seconds. This means that as soon as you hear the BEEP, you must pay careful attention to the screen. You do not have to wait until the stimulus disappears from the screen before responding. You may press either of the response keys at any time.

"If you believe that you've missed a stimulus, do *not* press a response key -- instead press ENTER to repeat that trial. Since you will not be able to see some of the patterns even if you are paying attention, please try to avoid this option as much as possible.

"Briefly: Pressing ENTER starts a trial. A trial starts with a BEEP. Immediately after the BEEP a grating pattern appears briefly on the screen. Press H or V to record your decision about the orientation of the grating.

"Is everything clear? When you are ready, you may begin by pressing ENTER..."

---

6. ENTER generates a BEEP and starts a trial (also aborts dummy trial at beginning). After each trial, the participant votes H or V. *Only* if the participant missed a trial can ENTER be used without first voting H or V -- now, ENTER simply repeats the trial. An inability to see the grating when looking at screen is *not* the same as missing a trial -- a vote must be made, even if it is a guess. A correct response is signaled by single BEEP, which also starts the next trial (note, the participant can pause simply by not entering a vote until ready for next trial). An incorrect vote is signaled by 3 rapid BEEPS, which also starts the next trial.

7. The QUEST session is divided into 3 blocks. A sequence of BEEPS announces the end of each block. The participant can pause between blocks, but they must remember to readapt to the screen before starting the next block!

8. When the procedure is finished, press ESC and chooseYes to exit VisionWorks.

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**SECOND SESSION (CSF)**
**(QUEST)**

1. Turn the computer screen away from the participant. Turn the room lights off and allow a few minutes for the participant to adapt to the display luminance.

1a. Read the instructions for the QUEST session to the participant.

---

**PARTICIPANT'S INSTRUCTIONS: QUEST (CSF)**

"Please sit down and make yourself comfortable. For this experiment, it is important that you wear any optical correction you might need for seeing distant objects clearly.

"In this session you will be shown the same kind of stimuli as in the previous session in which we started measuring your Contrast Sensitivity Function. However, this time we will be trying to measure your threshold with greater precision. For this, we will use a different procedure, known as the QUEST procedure.

"In this procedure your task will be to decide whether the grating pattern was vertical or horizontal in orientation. To record your decision, you must press either the response key marked "V" for Vertical, or the key marked "H" for Horizontal. These response keys are located on the keyboard in front of you.

"The orientations of some grating patterns will be more obvious than others. In fact, in some instances you may not be able to see any pattern at all. But you must still make a choice. Even though this may be difficult, and you may think you are guessing, try to do your best on every trial. If you respond correctly, the next trial will start automatically. If, however, you respond incorrectly, you will hear 3 short beeps -- the next trial will follow immediately after those beeps. It is OK to be incorrect. Remember, we are attempting to find your contrast threshold; that threshold is the level at which you are as likely to be correct as incorrect -- this means that there will be many incorrect responses. Nonetheless, we still want you to try hard on each trial. Do not give up when it gets difficult.

"At this time, please place your chin in the chin rest and keep it there during your observations. Try to avoid tilting or turning your head too much. If the chin rest is too low or too high, please tell me. Now look at the screen and keep looking there throughout the experiment. However, please do NOT stare fixedly at any one point on the display.

"Trials will start automatically. A single BEEP signals the start of a trial. Immediately after the BEEP, a grating pattern will be presented on the screen for two seconds. This means that as soon as you hear the BEEP, you must pay careful attention to the screen. You do not have to wait until the stimulus disappears from the screen before responding. You may press either of the response keys at any time.

"If you believe that you've missed a stimulus, do *not* press a response key -- instead press ENTER to repeat that trial. Since you will not be able to see some of the patterns even if you are paying attention, please try to avoid this option as much as possible.

"Briefly: Pressing ENTER starts a trial. A trial starts with a BEEP. Immediately after the BEEP a grating pattern appears briefly on the screen. Press H or V to record your decision about the orientation of the grating.

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"Is everything clear? When you are ready, you may begin by pressing ENTER..."

---

2. ENTER generates a BEEP and starts a trial (also aborts dummy trial at beginning). After each trial, the participant votes H or V. *Only* if participant missed a trial can ENTER be used without first voting H or V -- now, ENTER simply repeats the trial. An inability to see the grating when looking at the screen is *not* the same as missing a trial -- a vote must be made, even if it is a guess. A correct response is signaled by single BEEP, which also starts the next trial (note, the participant can pause simply by not entering vote until ready for next trial). An incorrect vote is signaled by 3 rapid BEEPS, which also starts the next trial.

3. The QUEST session is divided into 3 blocks. A sequence of BEEPS announces the end of each block. The participant can pause between blocks, but must remember to readapt to the screen before starting the next block!

4. When the procedure is finished, press ESC and choose Yes to exit VisionWorks.

## BINOCULAR & STEREOSCOPIC FUNCTIONS

### A) BINOCULAR SCREENING TESTS.

**DESCRIPTION OF BINOCULAR SCREENING TESTS**

Series of tests to establish basic aspects of binocular functions.

(i) Inter-pupillary distance. When participant looks at target at optic infinity, measure separation of corneal reflections (1st Purkinje images) of targets.

(ii) Ocular dominance. To assess degree to which one eye is preferred/dominates. Use: sighting task (which eye used at a peephole?); asked to fuse pair of monocular images chosen so that some parts are not inherently fusible, resulting in binocular rivalry (which eye's segments predominate in percept?).

(iii) Binocular alignment. Measure residual heterophorias (*tendencies* for eyes not to be exactly aligned when looking at given target): esophoria (turn towards nose), exophoria (turn towards temple), hyperphoria (turn upwards), hypophoria (turn downwards). Use Maddox rod test: Maddox rod (very strong cylindrical lens) over one eye as participant looks at distant luminous target; fellow eye sees target, eye with rod sees target stretched into a line. These two images are not inherently fusible; if a phoria exists, line will not appear to pass through target. Add prisms to Maddox rod to move line until it passes through target; amount of prism that is needed is the measure of the phoria. Problem: the line appears to wander back and forth, either because the two images are not good stimuli to accommodation, or because their dissociation opens the loop normally controlling convergence. We use a modification: targets are luminous optotypes (white-on-black on CRT screen) close to the acuity limit and shown at distance (6m); participant asked about location of line *while* trying to read letters. Accommodation thereby held at distance, which also prevents proximal convergence due to percept of a closer target -- this helps control "wandering" of line.

(iv) Stereopsis. Depth perception exclusively from small displacements of images on each retina of object in front of or behind plane of fixation/convergence. Series of

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standard clinical screening tests using printed materials and goggles that separate the images for each eye; goggles either use polarizing filters (axes of right and left eyes at right angles) or red/green filters. Some tests are random-dot stereograms in which, in principle, no object can be seen with unfiltered binocular or monocular viewing -- object results from central processing of image-differences in the two eyes and object pops out in depth. Titmus tests (not random-dot, and visible monocularly): fly, to demonstrate stereopsis, plus various subtests with graded stereoseparations to estimate stereo-acuity. Randot tests: similar to Titmus, but also includes some random-dot stereograms. TNO plates: red/green anaglyphs, all presented as random-dot stereograms.

Stereopsis, especially in artificial situations, often requires practice. Therefore use a range of stereotests, including many random-dot stereograms. Essential practice for later measures of stereopsis.

**EXPERIMENTER'S INSTRUCTIONS (BINOCULAR SCREENING)**
**SETTING UP AND RUNNING (BINOCULAR SCREENING)**

**1. Binocular Screening -- General Set-up.**

These tests require several different pieces of equipment. All data are entered into a single spreadsheet. Prepare *all* the equipment ahead of time. Separate sub-tests, below, can be run in any convenient order, but given order is probably optimal.

1. The distance from the participant's eyes to the B-VAT screen, when reflected from the mirror, should be 6 m. Check this distance, using the 6m purple cord (note: the 6m point is marked by piece of tape about 10" from one of the ends); hold one end at the phoropter and the other against the B-VAT screen; when the center of the cord is pulled towards the mirror, it should almost touch it. (*Note:* Do not actually touch the mirror -- it is a first-surface mirror and can easily be damaged; do NOT wipe off any fingerprints or marks -- get one of senior lab personnel to do so!)

2. Turn on the light on the easel that will hold the stereo screening books (same easel for one of the ocular-dominance tests).

3. Read the general instructions for Binocular Screening.

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**PARTICIPANT'S INSTRUCTIONS (Binocular Screening -- General)**

"In this experiment we will measure several different ways in which you use the information you get from your two eyes. Both eyes work together to give a uniform image of the object being viewed. However, if you were to view an object with one eye, and then immediately view the object with the other eye, you'd notice that the images were not entirely the same. The particular object that you are viewing appears to have moved, or been "displaced" slightly. When your brain puts together these slightly different images from each eye, you perceive single objects, but ones that are at different distances from you.

"Several factors contribute to this cooperation between the eyes. In these tests we will obtain some essential baseline data and also use a few standard screening tests of binocular vision and depth perception. The tests include the following: (a) Measurement of how well you can align your two eyes when you are looking at a small target;

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(b) measurement of your depth perception when you use both eyes -- this is known as Stereopsis; (c) assessment of ocular dominance, that is, which eye do you favor.

"Since there are several tests, we will give more specific instructions as we go along. Do you have any general questions?"

---

4. The participant should be wearing appropriate correction for distance and for reading, depending on the specific test – this need not be their "best" correction!

**1. Inter-Pupillary Distance.**

1. Measure the inter-pupillary distance (IPD) when the participant is converged for a target at "infinity".

1a. Use the Digital C.R.P. (Essilor Instruments).

1b. Check that the distance dial is set at infinity -- this places the internal optical target at that distance.

1c. Use both hands to hold the instrument with its dials facing the floor; place your thumbs on the two sliding switches so you can make adjustments. Hold instrument against participant with forehead-bar above the eyelids. Instruct participant to **"Look at the small white circle in the center of the green donut. Keep looking directly at it."**

1d. Look in the experimenter's viewing port and locate the participant's eyes. You should see a vertical light line on each of the eyes, as well as a bright spot close to the center of each pupil (this spot is an image of the fixation target after reflection from the cornea).

1e. Move each sliding switch until the line it controls passes exactly through the center of the bright spot in the middle of the given eye -- do this for both eyes. Keep repeating the instructions as you verify your settings.

1f. Remove the instrument and note the settings (in mm) on the LCDs: the two distances add up (to the nearest 0.5 mm) to the total IPD on the central LCD.

**2. Binocular Screening -- Horizontal and Vertical Fusion.**

1. Apparatus is the phoropter plus the B-VAT. The participant should be wearing acceptable correction – this need not be their "best"-- or have acceptable acuity (6/10 is acceptable). (Otherwise, you may have to dial in a correction on phoropter.)

2. PHOROPTER (see Figure labeling the different parts): (Seat the participant in front of the phoropter.)

2a. Use the P.D. KNOB to set the participant's IPD (see P.D. scale). Position the phoropter in front of the participant's eyes -- if glasses get in the way, check if participant can see adequately at distance without them. Check SPIRIT LEVEL and use LEVELING KNOB to center the bubble exactly.

2b. The SPHERE POWER SCALE and the CYLINDER POWER SCALE (for both eyes) should read 0.00 -- if not, rotate the CYLINDER POWER KNOBs and WEAK SPHERE DIALs to achieve this.

2c. Turn off room lights.

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3. B-VAT. When activated it should display several rows of text, mirror-reversed, because it is intended to be viewed through a mirror at end of the lab bench.

3a. Choose a single line of text set for about 20/50

3b. Reverse the contrast so the letters are bright against dark background.

4. Horizontal Fusion. Measures the ability to fuse in the horizontal plane.

4a. Set the AUXILIARY LENS KNOB for *left* eye for a white, horizontal Maddox rod (WMH) -- this blurs images seen by this eye into vertical lines; set the Auxiliary Lens Knob for the *right* eye for a clear aperture (O). (If the aperture is partially blocked, rotate the STRONG SPHERE CONTROL until clear.)

4b. Move the *left* eye's ROTARY PRISM UNIT in front of that eye's aperture -- this allows the experimenter to use prisms to move, left and right, the vertical line that will be formed by the Maddox rod. Rotate the dial of this unit so that zero points UP. Set the prism power to zero -- use the small knurled knob on this unit to move the arrow in line with the zero. Units on this dial are in Prism Diopters.

4c. Check that the letters on B-VAT are in a vertical column.

4d. Specific instructions: **"Please place your head against the apparatus and look through it at the screen. You should see a column of letters and a bright vertical line. Do you see them? If not, try blinking or moving your head slightly."** Continue: **"Can you read the letters?"** Depending on participant's answer, increase or decrease the letter size (using 20/nn keys) until the participant can read them without too much trouble --the aim is not to measure acuity but to ensure that the participant is attending to the letters and is accommodated for the screen distance.

4e. Continue: **"Please read the letters to me. When you read the letters, was the line running through them, or was it to the left or to the right of them?"** Depending on the answer, use the small knurled knob that adjusts prism power to move the line towards the letters.

When the arrow points away from zero towards the participant's temple, the prism is BASE-OUT, which means the vertical line has been moved *to the right*. When the arrow points away from zero towards the participant's nose, the prism is BASE-IN, which means the vertical line has been moved *to the left*. (When you face the participant, rotating the knob towards your right moves the line to the right, rotating it towards your left moves the line to the left.)

Move a little. Stop. Change the letters (press CHANGE). Repeat the question. Keep doing this until the line is running through the letters. (*Note:* at that point, the line may partially obscure the letters, so expect additional errors -- all that matters is finding the prism power that puts the line directly through the letters while the participant is accommodated for them, as shown by his attempt to read them.)

4f. Record the prism power needed to put line through letters; +n.n = Base-Out, -n.n = Base-In.

5. Vertical Fusion. Measures the ability to fuse in the vertical plane.

Same general procedures as for Horizontal Fusion are used except for:

5a. Set the AUXILIARY LENS KNOB for *left* eye for a white, vertical Maddox rod (WMV) -- this blurs images seen by this eye into horizontal lines.

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5b. Rotate the dial of the *left* eye's ROTARY PRISM UNIT so that zero points directly at participant's nose. Set the prism power to zero -- use the small knurled knob on this unit to move the arrow in line with the zero.

5c. Make the row of letters on B-VAT into a horizontal row; press the key 20/50 to select that size; reduce the size until the participant's limit is approached.

5d. Specific instructions: **"Please place your head against the apparatus and look through it at the screen. You should see a row of letters and a bright horizontal line. Do you see them? If not, try blinking or moving your head slightly."** Continue: **"Can you read the letters?"** You probably do not need to change the size from the previous test.

5e. Continue: **"Please read the letters to me. When you read the letters, was the line running through them, or was it above or below them?"** Depending on the answer, use the small knurled knob that adjusts prism power to move the line towards the letters.

When the arrow points UP, away from zero, the prism is BASE-UP, which means the horizontal line has been moved *towards the bottom.* When the arrow points DOWN, the prism is BASE-DOWN, which means the horizontal line has been moved *to the top.*

Move a little. Stop. Etc.

5f. Record the prism power needed to put line through letters; +n.n = Base-Up, -n.n = Base-Down. (*Note:* In this particular case, it is very unusual to find a value greater than 1 or 2 diopters.)

6. Turn room lights ON for rest of tests.

**3. Binocular Screening -- Ocular Dominance.**

1. Sighting test of dominance. Apparatus is the tube attached to the top of the easel used to display stereo test books.

1a. Ask participant to stand a few feet away from the apparatus, directly in front of the tube. Instruct: **"Please walk up to this tube, look through it, and read the text you see through it."** Observe carefully which eye they use -- that is the dominant one for this test (what they read is irrelevant!). If they do not go right up to the tube, tell them to do so.

1b. Enter eye used; codes: L, R, M (mixed -- participant alternates eyes and cannot decide which to use).

2. Rivalry test of dominance. Apparatus is the hand-held stereo viewer.

2a. Set device for "FAR POS.". The stimulus card should be two squares with oblique cross-hatching -- opposite directions: on left square, sloping up to the right, and on right square, sloping up to the left.

2b. *Before* participant looks in, instruct: **"When you look into this device you will see a square with lines drawn across it. Tell me which way the lines are going. If they keep changing, tell me which way they seem to be going most of the time and which way they seem to cover most of the square. If you are unsure, blink and try again."**

2c. Record which square has the lines that are visible more of the time or are more obvious: L, R, M (mixed -- can't decide, alternate equally between the two directions), plus suffix, S (strong), W (weak).

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**4. Binocular Screening -- Stereopsis.**

1. Stereopsis, using Titmus Fly book

1a. Place open book on easel. Seat the participant so his head is against head rest and book is easily visible. The participant should wear appropriate correction for reading distance. On top of correction, he must wear *plastic polarizing glasses.*

1b. Open book panel "1" – this exposes the full-page image of the fly. Ask the participant: **"Do you see the fly? Does it appear solid? Does it stand out from the page? When you move your head back and forth a little, does the fly seem to move?"**

1c. Close panel "1" and open panel "2". For each of the rows labeled A, B, and C ask: **"Is one of the animals standing out from the page? Which one?"** If participant gets it wrong, offer verbal encouragement: **"Not everybody sees things this way. Try your best."** (Do NOT use the word "guess"!!) Note which was the *first* row with an error. If the participant failed on all rows, enter X.

1d. Close panel "2" and open "3". For each of the nine figures (going in numerical order) ask: **"Start with figure 1. Which of the four circles seems to be sticking out from the page? -- top, bottom, left, or right?"** (Again, offer verbal encouragement.) Note figure with *first* error. If the participant failed on all figures, enter 0.

2. Stereopsis, using Randot book. General procedure is the same as for Titmus Fly.

2a. Panel "1". Ask: **"Is one of the animals standing out from the page? Which one?"** Note which was the *first* row with an error. If the participant failed on all rows, enter X.

2b. Panel "2". For each of the ten figures (going in numerical order) ask: **"Start with figure 1. Which of the three circles seems to be sticking out from the page? -- left, middle, or right?"** (Again, offer verbal encouragement. Spreadsheet lists correct answers.) Note figure with *first* error. If the participant failed on all figures, enter 0.

2c. Panel "3". This panel is mostly to familiarize the participant with random-dot stereograms. Each panel contains a geometrical figure. Ask: **"Starting with the top-left panel -- is there a shape that sticks out from the page? What is it?"** Go through the panels in order: left to right and top to bottom. Count the number correct.

3. Stereopsis, using TNO plates

General procedures are the same as for the Titmus and the Randot, except that the participant wears, over their correction, red-green plastic goggles (check that red is over the left eye). The first few pages of this book are to familiarize the participant with random-dot stereograms and to check for stereopsis using these sorts of displays. For each page there is always a "correct" answer even if the participant has zero stereopsis -- what matters is whether the correct answer corresponds to the figure that is visible only with stereoscopic vision.

3a. Plate I. Ask: **"How many butterflies do you see?"** If the participant answers TWO: **"Point to them."** Stereo figure is on lower right. If the participant answers ONE: **"Look very carefully. There may be more. Look all over the page."**

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3b. Plate II. **"How many circles do you see? Point to the biggest one."** Stereo figure is biggest -- top right.

3c. Plate III. Point to the figures on left-hand page and ask: **"Which of these figures do you see on the right-hand page? Show me where each one is."** (circle on bottom left, diamond on lower right, triangle on upper right, square on upper left.)

If the participant was correct for most of these figures, enter Y, otherwise enter N. If entered N, do NOT go on to the rest of this test.

3d. Plate V. (Ignore other pages. This and subsequent pages measure stereoacuity -- stereo separation gets progressively smaller.) Point to the figures on the left-hand page and say: **"Here are some pictures of "Pac-man". In each picture, his mouth is pointing in a different direction. You may be able to see Pac-man in the panels on the right-hand page. Show me which Pac-man you see in each panel, starting with the top row."** Both figures in each row have the same stereo separation; the participant must get *both* correct for that row to count. If all of Plate V was correct, continue to Plate VI, and then, if all of VI was correct, to Plate VII (i.e., stop on first error). Enter coded value of last correct row -- 51 for top row on Plate V, etc.

## B) STEREOPSIS.

### DESCRIPTION OF TEST (STEREOPSIS)

Measures of stereo acuity and zone of maximal disparity that can be fused to give depth sensation (i.e., extent of Panum's area). Stimuli presented on Vision-Works Graphics screen at 1.8 m viewing distance -- far enough to avoid accomm/convergence problems, but near enough to give useable stereo. Illuminated surround.

Uses *dynamic random-dot stereograms*. Each frame of display has two vertical nonius lines above and below central square in which stereo target will appear; also three horizontally arrayed fixation spots, two flanking the square (2°) and one in middle. Any given frame seen only by one eye (computer-controlled ferrite electro-optical goggles synchronized with display frames at 120 Hz). Each eye sees only one of vertical lines, while both eyes see central fixation spot -- together these act as stimuli to accommodation and convergence -- participant told to keep lines aligned with each other and to "see" single fixation spot. For each *pair* of frames, all pixels (1.3 min arc) are re-randomized; central stereo zone offset by given amount to create crossed or uncrossed disparities. Continuous randomization gives a flickering display. This dynamic version used in order to avoid certain edge artifacts with static displays on this system.

Four interleaved QUEST procedures to measure 4 thresholds -- stereo acuity for minimal "popout" in front of convergence plane (horopter), behind plane, and maximal extent, front and rear of zone over which disparity can be fused to give stereoscopic depth. (If QUEST cannot find stable threshold, dummy value entered, either -9999 or negative threshold.) Forced-choice votes (front vs. back); when disparity too large, central square clearly seen but depth is not apparent. (QUEST parameters: alpha=.01, max trials=100, confidence interval=3 decades)

Start with adjustment of thresholds to get starting points for QUEST and to familiarize participant with display. Participant must wear appropriate optical correction.

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## EXPERIMENTER'S INSTRUCTIONS (STEREOPSIS)

### SETTING UP & RUNNING STEREOPSIS

1. Participants must have their appropriate optical correction! If over 50 years of age, you must add 0.5D sphere to each eye to correct for this test's viewing distance -- clip trial lenses directly into goggles frame. If younger, the participant can wear his own correction, provided he can see screen clearly; if usually tested with trial lens corrections, use the same but add 0.5 diopter sphere. (Keep track of sign when adding 0.5 diopter to the spherical correction -- e.g., with spherical correction of -1.5 diopters, adding 0.5 diopter yields -1.0 diopter sphere; with spherical correction of +1.5 diopters, adding 0.5 diopter yields +2.0 diopters.)

2. Move the chin rest to testing distance -- use 3.6 m rope folded in half.

3. Plug glasses either into the computer.

4. Read the *General Instructions* for stereopsis to participants, then fit the goggles and helmet to the participant (carefully -- delicate!). Angle the lenses until they are just in front of participant's eyes. Adjust laterally until participant is looking through the center of each -- have participant test by alternately closing each eye.

---

### PARTICIPANT'S INSTRUCTIONS (General Instructions, Stereopsis)

"Please sit down and make yourself comfortable. In this experiment we will attempt to measure your depth perception when you use both eyes -- this is known as Stereopsis. Both eyes work together to give a uniform image of the object being viewed. However, if you were to view an object with one eye, and then immediately view the object with the other eye, you'd notice that the images were not entirely the same. The particular object that you are viewing appears to have moved, or been "displaced" slightly. When your brain puts together these slightly different images from each eye, you perceive single objects, but ones that are at different distances from you. In this experiment, we are asking the questions "What are the minimum and the maximum amounts that the views of the two eyes can be displaced in order for you to still see a single object popping out in depth?"

"You will be shown computer-generated patterns on this screen. With the help of special goggles, that are also controlled by the computer, you should see some of these patterns as floating out from the rest of the screen. Part of the pattern will seem either to have popped in front of the screen or to have popped back from the rest of the screen. You will be asked to judge whether the square is closer to you compared to the rest of the pattern, or whether it is farther away from you than the rest of the pattern.

"Now we will fit you with the goggles, which should be worn over your usual optical correction -- if you need any additional correction, this will have been added to the goggles. Please place the goggles and their helmet on and, with the help of the experimenter, adjust the head bands so that they are comfortable. The lenses should be adjusted so that your vision is not blocked. The lenses are properly aligned when you see approximately the same image as you alternately open and close each eye.

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"This experiment will be divided into two phases. In the first phase, we will get a rough estimate of the range of pattern displacement your eyes can sufficiently fuse, while in the second phase, we will measure this with greater precision."

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5. Check the goggles. If have finger-prints, clean very gently only with KIMWIPES.

6. Check that computer is correctly driving the glasses: when wearing goggles and closing one eye, only one of the vertical bars should be seen; other eye sees the other bar.

7. Prepare to collect data. Turn computer screen away from participant; turn off room lights; place keyboard cover over keyboard and place keyboard in front of participant.

**a) Adjustment Phase (Stereopsis).**

1. Read the *Adjustment Instructions* for stereopsis. Note that there is a pause in the instructions while the participant tries the keys to see what they do. (*Important*: Throughout the Adjustment and QUEST tests, *keep reminding* the participant to fixate on the central fixation spot, which should keep the vertical lines aligned with each other. This is vital in order to keep the plane of the horopter constant!)

2. Complete the *Adjustment* instructions and collect the four thresholds for: MINIMUM-FRONT, MINIMUM-BACK, MAXIMUM-FRONT, MAXIMUM-BACK, in that order! (The full script for doing this is included in Participant's Instructions.)

2a. After reading the instructions for the first of these, wait until the participant has finished and entered the result. Then read the instructions for next, collect result, and so on.

2b. While the participant is making adjustments, watch the panel at the top right of the VisionWorks screen: If CMP value does not change after each key-press by participant, or if several changes occur when participant stops pressing, participant is probably pressing too rapidly -- slow down! Also, setting for a FRONT threshold must be positive, for a BACK threshold, negative; minima will usually be less than 10, maxima up to about 50.

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**PARTICIPANT'S INSTRUCTIONS:**
**Adjustment Instructions & Running Script (Stereopsis).**

"Please place your chin in the chin rest. If the chin rest is too low or too high, please tell me so that we can adjust it. When looking at the screen, make sure that the black vertical bars are lined up, and that only ONE fixation point is seen in the center. If this is not the case, please tell me.

"In the center of the display, you should see a square region which appears closer to you than the surrounding pattern -- it should appear to float in front of the rest of the pattern. In this first phase, you will pull the pattern out farther from the screen and closer to you; then you will push it back until it appears to be behind the rest of the screen. You will do this using the key on the UPPER LEFT of the keyboard in front of you: the one that is labeled "FRONT" pulls the central square closer to you; the other key, labeled

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"BACK", pushes it farther away from you. If, at any time, you have a problem seeing the square as closer or farther away, try blinking and refocusing on the fixation point in the center. Do not press the keys too quickly -- the computer may not be able to keep up. Now, try these keys and see that they work.

<LET THE PARTICIPANT PRACTICE; CHECK THAT KEY PRESSES ARE SLOW ENOUGH>

"Now you must adjust the setting of the central square so that we can collect four initial thresholds from you:

<1: MINIMUM-FRONT>:-

"For the first threshold, you will start with the square floating *in front* of the screen. Then push it gradually back until it is *just in front* of the screen -- i.e., you should see the display as an almost even surface, but with the central square just raised towards you. You can move it back and forth until you are satisfied. *Go slowly!*

"When you are satisfied, press ENTER to record your threshold.

<2: MINIMUM-BACK>:-

"When you are ready to begin with the second threshold, press ENTER to start. (You may have to wait a moment until the new stimulus appears.)

"Now the square should be floating *in back* of the screen -- i.e., it should appear farther away than the rest of the screen. Pull it gradually towards the rest of the pattern until it is *just behind* the screen -- i.e., you should see the display as an almost even surface, but with the central square just indented away from you.

"When you are satisfied, press ENTER to record your threshold.

<3: MAXIMUM-FRONT>:-

"When you are ready to begin with the third threshold, press ENTER to start. (You may have to wait a moment until the new stimulus appears.)

"Now the square should be floating *in front* of the screen. You will try to find the maximum displacement which still allows you to see the square as being in front of the rest of the display. Pull it towards you until the square is as far from the screen as possible.

"If you pull it too far, one of two things may happen: you will still see a square but will *not* be able to tell whether it is in front of or behind the screen; or, you may find it very difficult to continue seeing only ONE fixation spot in the center of the field. If you go too far, push it back a little until it again pops out at you.

"When you are satisfied, press ENTER to record your threshold.

<4: MAXIMUM-BACK>:-

"When you are ready to begin with the fourth threshold, press ENTER to start. (You may have to wait a moment until the new stimulus appears.)

"Now the square should be floating *in back* of the screen. You will try to find the maximum displacement which still allows you to see the square as being in back of the rest of the display. Push it away from you until the square is as far from the screen as possible.

"If you push it too far, one of two things may happen: you will still see a square but will *not* be able to tell whether it is in front of or behind the screen; or, you may find it very difficult to continue seeing only ONE fixation spot in the center of the field. If you go too far, pull it forward a little until it again pops back from the screen.

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"When you are satisfied, press ENTER to record your threshold."
<CHECK EXPERIMENTER'S INSTRUCTIONS>

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3. When the adjustment is finished, continue immediately to the QUEST phase of stereopsis.

**b) QUEST Phase (Stereopsis).**

1. Read the *QUEST Instructions* for stereopsis. ***Keep reminding*** the participant to fixate on the central fixation spot, which should keep the vertical lines aligned with each other – the participant should only see a single fixation spot in the center of the field.

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**PARTICIPANT'S INSTRUCTIONS: Quest (Stereopsis).**

"In this phase, your task will be to determine whether the central square appears closer to you than the surrounding pattern or farther away from you than the surrounding pattern. It will always be one or the other. Make your selection by pressing either the key labeled "FRONT" or the one labeled "BACK"; in this phase of the experiment, these keys are the ones located on the RIGHT side of the keyboard..

(<SHOW KEYS>)

"Use FRONT if the square appears closer to you -- i.e., it is popped out *in front* of the stimulus display. Use BACK if it appears to have popped away from you -- i.e., it appears *in back* of the rest of the display.

"You can look at the display as long as you like before making your choice. Sometimes it will be very difficult to tell. However, do not give up. Do your best on each trial.

"Each trial begins with a BEEP. If your vote about direction of the square (front or back) is correct, you will hear a single BEEP that signals the automatic start of the next trial. However, an incorrect response will be followed by 3 short beeps and then the next stimulus.

"It is OK to be incorrect. Remember, we are attempting to find your thresholds for displacements right next to the plane of the display and for displacements far from that plane. These thresholds are the values at which you respond correctly as often as you respond incorrectly. This means that you can expect to be wrong quite often.

"At this time, please place your chin back in the chin rest. Is everything clear?

"When you are ready, you may begin by pressing ENTER. Once again, make sure the black vertical bars are lined up, and that only ONE fixation point is seen in the center."

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## VERNIER ACUITY

**DESCRIPTION OF TEST (VERNIER)**

Vernier alignment is a hyperacuity. Measures the minimum detectable displacement between two vertical, abutting lines.

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Stimuli presented (1 s) on bright computer screen as two dark lines; lower one offset to left or right; forced-choice vote. Adaptive psychophysics using QUEST algorithm to find offset acuity threshold.

***Note:*** This test has a strong practice effect. Therefore, each participant must be run twice, preferably on separate occasions. Both sets of data will be added to the main database, but only the thresholds from the second (or most recent, if several were run) will be used for the analyses of the Battery project. Each session must have a separate session number.

***Note:*** For some of the participants tested in the early days of the battery, there may be no practice sessions in the database. However, everyone included in the database will have run this test at least twice.

## EXPERIMENTER'S INSTRUCTIONS (VERNIER)

### SETTING UP (Vernier)

1. *Optics*. Participants must have their "Best" optical correction! This test is run at 1.8 m; therefore, you may have to add correction for this distance: if under age 50, check if the participant can comfortably see the screen at the testing distance while wearing their usual correction; if over 50 years, you must add 0.5D sphere to each eye!

1b.Use trial frames for added lenses; you must re-measure inter-pupillary distance (PD) with the pupil-measuring device set for slightly under 200 cm – this should give PD 1-2mm less than the participant's PD for distance.

1c. If best correction requires other trial lenses, add 0.5 diopter to the spherical correction, keeping track of sign -- e.g., with spherical correction of -1.5 diopters, adding 0.5 diopter yields -1.0 diopter sphere; with spherical correction of +1.5 diopters, adding 0.5 diopter yields +2.0 diopters.

2. Place display screen and table with chin rest in correct positions; measuring distance using half of the 3.6m rope.

### RUNNING THE EXPERIMENT (Vernier)

1. During the reading of the instructions, the screen displays a static version of a vernier display with a very clear offset; use this to clarify the instructions.

2. Turn the computer screen towards the wall so the participant is not disturbed by it. Place the keyboard cover over the keyboard and put the keyboard on the table in front of the participant. The participant uses the arrow keys to record votes.

2a. Turn off the room lights and shut the door so that the participant can adapt to the display luminance.

2b. Read instructions to participant:-

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## PARTICIPANT'S INSTRUCTIONS (Vernier)

<EXPERIMENTER: point to the various items as you mention them in the Instructions!>

"Please sit down and make yourself comfortable. For this experiment, it is important that you wear your optical correction.

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"In this experiment we will attempt to measure your sensitivity to Vernier-Offset. Specifically, we are asking the question "What is the least amount that a vertical line can be offset from another vertical line in order for you to detect the offset?"

"The computer-generated stimulus patterns will be displayed on this screen in front of you. The two lines shown now are grossly misaligned. During a trial you will see this sort of pattern, but only for a very brief period. On each trial the top line will always be in the same position, but the lower one will be offset either to the left or to the right. You must judge the direction of the offset of the ***lower*** line -- i.e., is the lower line to the left or to the right of the upper line. To record your decision, you must press either the arrow key pointing to the LEFT, or the arrow key pointing to the RIGHT. These response keys are located on the keyboard in front of you.

"During this experiment, you must pay attention to the center of the screen where the two lines meet. On some trials it will be quite obvious in which direction the bottom line has been moved. However, on other trials you may not be able to detect any offset. But you still must make a choice. Even though this may be difficult, try to do your best on every trial.

"A trial starts with a BEEP. If you respond correctly, the next trial will start automatically. If, however, you respond incorrectly, you will hear 3 short beeps-- the next trial will follow immediately after those beeps. It is OK to be incorrect. Remember, we are attempting to find your threshold, which is defined as the level at which you are as likely to be correct as incorrect -- this means that there will be many incorrect responses. Nonetheless, we still want you to try hard on each trial. Do not give up when it gets difficult.

"At this time, please place your chin in the chin rest and keep it there during your observations. Try to avoid tilting or turning your head too much. If the chin rest is too low or too high, please tell me. Now look at the center of the display and keep looking there throughout the experiment. However, please do NOT stare fixedly at any one point on the display.

"Once we begin he experiment, trials will start automatically. A single BEEP signals the onset of a trial. During a trial, the stimulus pattern is on the screen for a brief period. This means that as soon as you hear the BEEP, you must pay careful attention to the screen. You may press either of the response keys at any time.

"If you believe that you've missed a stimulus, do ***not*** press the arrow keys -- instead press ENTER to repeat that trial. Please try to avoid using this option as much as possible. You should only use it if you happened to blink or look away during a trial.

"Briefly: Pressing ENTER starts a trial. A trial starts with a BEEP. Immediately after the BEEP a stimulus pattern appears briefly on the screen. Press LEFT or RIGHT arrow keys to record your decision about the direction of offset of the lower line.

"Is everything clear? When you are ready, begin by pressing ENTER."
[First ENTER removes pattern from screen. Must do it again to start trials.]

"Please press ENTER again."

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# MOTION

## DESCRIPTION OF TEST (MOTION)

Sensitivity to moving stimuli: measures both minimal and maximal displacements that give rise to sensation of movement (i.e., $D_{min}$ and $D_{max}$); latter estimates size of perceptive field for motion, former estimates size of subunits in that field.

Stimuli are presented on computer screen: bright field replaced by fields of random dots. Present two frames (100 ms each) of random dots with displacement of the dots either up or down between the two frames -- forced-choice vote on direction of displacement. Degree of coherence between fields = 100%. Uses QUEST algorithm, two interleaved series. (QUEST parameters: alpha = .01, max trials = 100, confidence interval = 3 decades)

***Note:*** This test has a strong practice effect. Therefore, each participant must be run twice, preferably on separate occasions. Both sets of data will be added to the main database, but only the thresholds from the second (or most recent, if several were run) will be used for the analyses of the Battery project.

***Note:*** For some of the participants tested in the early days of the battery, there may be no practice sessions in the database. However, everyone included in the database will have run this test at least twice.

## EXPERIMENTER'S INSTRUCTIONS (MOTION)

### SETTING UP

1. *Optics*. Participants must have their "best" optical correction! This test is run at 1.8 m; therefore, you may have to add correction for this distance: if under age 50, check if the participant can comfortably see the screen at the testing distance while wearing their usual correction; if over 50 years, you must add 0.5D sphere to each eye!

1a. Use trial frames for added lenses; you must re-measure inter-pupillary distance (PD) with the pupil-measuring device set for slightly under 200 cm – this should give a PD 1-2mm less than the participant's PD for distance.

1b. If best correction requires other trial lenses, add 0.5 diopter to the spherical correction, keeping track of sign -- e.g., with spherical correction of -1.5 diopters, adding 0.5 diopter yields -1.0 diopter sphere; with spherical correction of +1.5 diopters, adding 0.5 diopter yields +2.0 diopters.

2. Place the display screen and the table with the chin rest in correct positions; measuring the distance using half of the 3.6m rope.

### RUNNING THE EXPERIMENT (Motion)

1. During the reading of the instructions, the screen displays a static random-dot field; use this to clarify the instructions.

2. Turn the computer screen towards the wall so the participant is not disturbed by it. Place the keyboard cover over the keyboard and put the keyboard on the table in front of the participant. The participant uses the arrow keys to record votes.

2a. Turn off the room lights and shut the door so that the participant can adapt to the display luminance.

2b. Read the instructions to participant:-

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## PARTICIPANT'S INSTRUCTIONS (MOTION)

<EXPERIMENTER: point to the various items as you mention them in the Instructions!>

"Please sit down and make yourself comfortable. For this experiment, it is important that you wear your optical correction.

"In this experiment we will attempt to measure your sensitivity to Motion. Specifically, we are asking the question "What are the minimum and maximum amounts that a stimulus pattern can be moved (or displaced) in order for you to still be able to identify the direction of motion?"

"The computer-generated stimulus patterns will be displayed on this screen in front of you. During a trial you will see this large square filled with a random dot pattern like this one. However, during a trial, the pattern will only be visible for a very brief time. During that time, the dots will be displaced, and you will be asked to judge in which direction the dots moved. There are two possible directions: up (the dots appeared to move towards the top of the screen) and down (the dots appeared to move towards the bottom of the screen). To record your decision, you must press either the response key marked "UP", or the key marked "DOWN". These response keys are located on the keyboard in front of you.

"During this experiment, you must pay attention to the center of the screen -- ***do not*** concentrate on the edges of the pattern. On some trials it will be quite obvious in which direction the dots move. However, on other trials you may not be able to detect that any movement has taken place. But you still must make a choice. Even though this may be difficult, try to do your best on every trial.

"A trial starts with a BEEP. If you respond correctly, the next trial will start automatically. If, however, you respond incorrectly, you will hear 3 short beeps-- the next trial will follow immediately after those beeps. It is OK to be incorrect. Remember, we are attempting to find your threshold, which is defined as the level at which you are as likely to be correct as incorrect -- this means that there will be many incorrect responses. Nonetheless, we still want you to try hard on each trial. Do not give up when it gets difficult.

"At this time, please place your chin in the chin rest and keep it there during your observations. Try to avoid tilting or turning your head too much. If the chin rest is too low or too high, please tell me. Now look at the center of the display and keep looking there throughout the experiment. However, please do NOT stare fixedly at any one point on the display.

"Once we begin the experiment, trials will start automatically. A single BEEP signals the onset of a trial. During a trial, the stimulus pattern is on the screen for a brief period. This means that as soon as you hear the BEEP, you must pay careful attention to the screen. You may press either of the response keys at any time.

"If you believe that you've missed a stimulus, do ***not*** press the *up* or *down* keys -- instead press ENTER to repeat that trial. Please try to avoid using this option as much as possible. You should only use it if you happened to blink or look away during a trial.

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"Briefly: Pressing ENTER starts a trial. A trial starts with a BEEP. Immediately after the BEEP a stimulus pattern appears briefly on the screen. Press UP or DOWN to record your decision about the direction of motion.

"Is everything clear? When you are ready, begin by pressing ENTER."

---

## SPECTRAL REFLECTIVITY OF IRIS AND SKIN

### DESCRIPTION OF TEST

This test is part of INTAKE for participants in the Battery of visual tests. The results of this test are also included in the database that describes all relevant parameters for each participant. In this database all entries are coded only by the participant's ID. Actual identification of the participant (name, address, telephone, etc.) is *not* part of this file and is kept only on confidential handwritten records.

The aim of this test is to specify pigments, other than visual photopigments, that might affect, or correlate with, visual performance: pigmentation of iris and skin are both correlated with density of lens pigment and may be correlated with other functions -- e.g., across the lifespan, lighter irises may be associated with increased SWS-cone damage due to prolonged light exposure.

A scanning spectroradiometer (Photo Research; model 703A/PC) is used to measure reflectance of a representative area of iris (approx 20% of area) and of skin on inner surface of forearm (occasionally, of forehead just above nose) -- this area is chosen because it is less likely to be heavily suntanned. The instrument measures spectral radiance -- i.e., the energy, per wavelength, that reaches its detector; these values can be displayed graphically and converted to assorted standard measures of light. Radiances of these targets are measured under standardized conditions: chin rest + two fluorescent illuminators at either side of head – measures are taken of the area of the pupil which is free of corneal reflections. Also, we measure the radiance of a neutral white reflector under the same conditions; we use this to compute reflectances of participants' targets.

### EXPERIMENTER'S INSTRUCTIONS (IRIS)

**1. Setting up and measuring.**

1. Switch on the illuminator's fluorescents. Switch off room lights.

2. Measure the spectral radiance of the standard white card when it is held in place of the participant's face.

2a. To do so you must first check that the photometer's eyepiece is focused for your eyes: look in and check the black horizontal rectangle; if its edges are not sharp, turn the knurled ring on the eyepiece until the rectangle looks sharp. (This rectangle covers the area of any object that is measured by the photometer.)

2b. (i) Slide the photometer to its rear position on its sliding track; (ii) focus the front lens of the photometer on the plane at which you will hold the white card -- a page of text held in that position will make this easier; (iii) hold the white card in place and check that the small cross at the center of the card is sharply in focus; (iv) make sure that the area of card the photometer will measure does not include the cross -- the measuring area is the black rectangle you see in the viewfinder.

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3. *Read the instructions to the participant.*

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**PARTICIPANT'S INSTRUCTIONS (IRIS)**

"This test is intended to measure the amount of light absorbed by some of the body's pigments. We are interested in this because some of these measurements may be correlated with visual functions. For example, the lens in the eye contains a pigment that absorbs some parts of the visible spectrum more than others. Light that is absorbed by the lens does not reach the retina at the back of the eye and so cannot produce a visual response. The amount of light absorbed by the lens is correlated with the pigment in the iris (the colored portion of a person's eyes) and also the pigment in the skin. In order to interpret completely some of the values we will obtain for your visual functions, we need measures of these pigments.

"The procedure is very simple. We will measure the amount of light absorbed by the iris in your eye and by a patch of skin on the inside surface of your forearm. To do this, we illuminate your face or arm with these ordinary reading lamps <SHOW>. We measure the amount of light reflected from you with this instrument <SHOW>. This is *not* a camera; it is a scanning spectroradiometer that simply measures the amount of light reflected from you at each point in the spectrum.

"First we will measure your arm while you hold it on this chin rest. Then we will measure your iris while you sit on the chair and place your head on the chin rest -- for this last measurement, all you will have to do is look directly into the lens of the photometer and try not to blink too much. Each measurement takes only a few seconds.

"Do you have any questions?"

---

4. To measure *skin*: have the participant hold his arm just above the chin rest; make sure that the inner part is turned towards the photometer. With the photometer still in its rear position, check the focus and position of the measuring rectangle on the arm. Warn the participant to keep still.

5. To measure *iris*: Switch the photometer's shutter to OPEN -- this allows you to continue viewing during the measurement.

5a. Slide the photometer to its "front" position. Focus the photometer's camera lens on a page of text held in place of the participant's face.

5b. Seat the participant. Point the photometer at an eye (usually left) and check that the iris is sharply in focus; make sure that the measuring rectangle is wholly on the iris and does include any of the images of the light sources seen on the cornea.

5c. Because the photometer's rear shutter (in the eyepiece) is open you must block any stray light from entering through the eyepiece. By looking into the eyepiece throughout the time of measurement your head will block out stray light -- you will also be able to watch the participant's eye and check that the measuring rectangle stays in the right place and that the participant does not blink too much.

5d. Warn the participant to try to look directly at the camera lens and to try not to blink.

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Appendix B

Supplementary Results

Color discrimination. Figure B1 is a box-and-whisker plot (representing values between the 25th and 75th percentiles, as well as high and low values, and the median) of the results of the Farnsworth-Munsell 100-Hue Test.

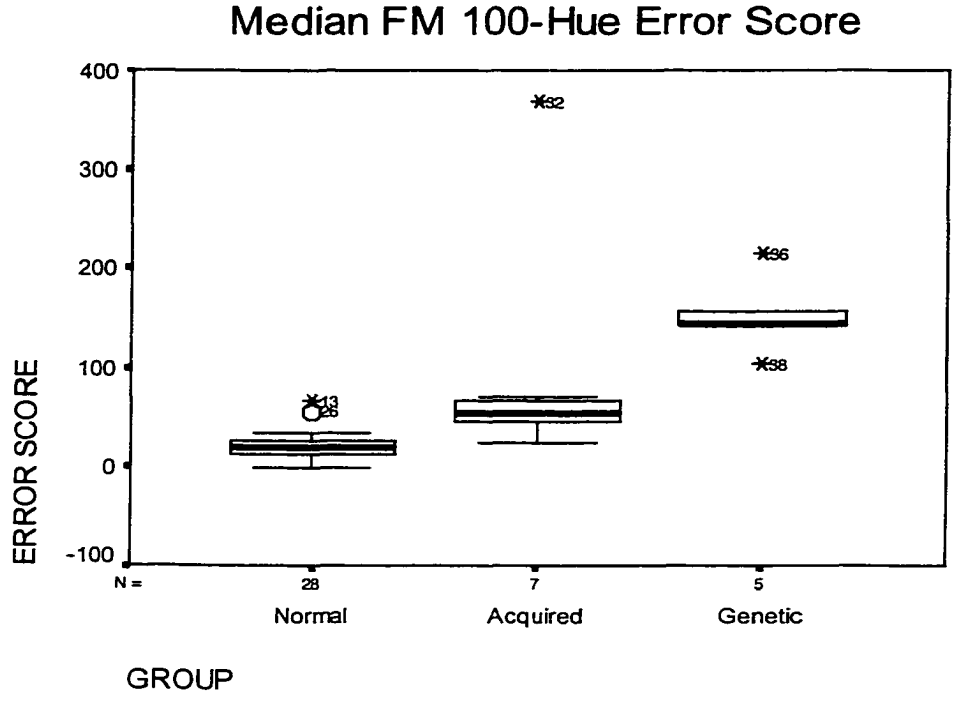


Figure B1. Box-and-whisker plot of group FM-100 Hue error score. Each box represents the middle 50% of scores in each group. Whiskers extend to high and low scores, exclusive of outliers or extreme values. The median is represented by the heavy black bar within the box. Outliers and extreme values are shown as single points.

As mentioned in the Results section, error scores of the genetic group members were found to be considerably higher than the other groups'. An exception is one acquired color-deficient individual whose error score exceeded that of all the genetics.

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This observer, mentioned earlier, recovered from Leber's idiopathic stellate neuroretinitis. Even with the exclusion of this participant's extreme measure, the acquired group, as a whole, still tended toward higher total error scores compared to the normal group, though some overlap in the range of scores can be noted (see Figure B1).

Hue- and saturation-scaling. Following the nearly identical mean delta-lambdas between the color-normal and genetic color-deficient groups, these groups' median values were also very similar to one another. To say that the genetics were more precise in discriminating among hues in this region of the spectrum, however, is questionably accurate, given the widespread variability and ranges of values across observers (see Figure B2).

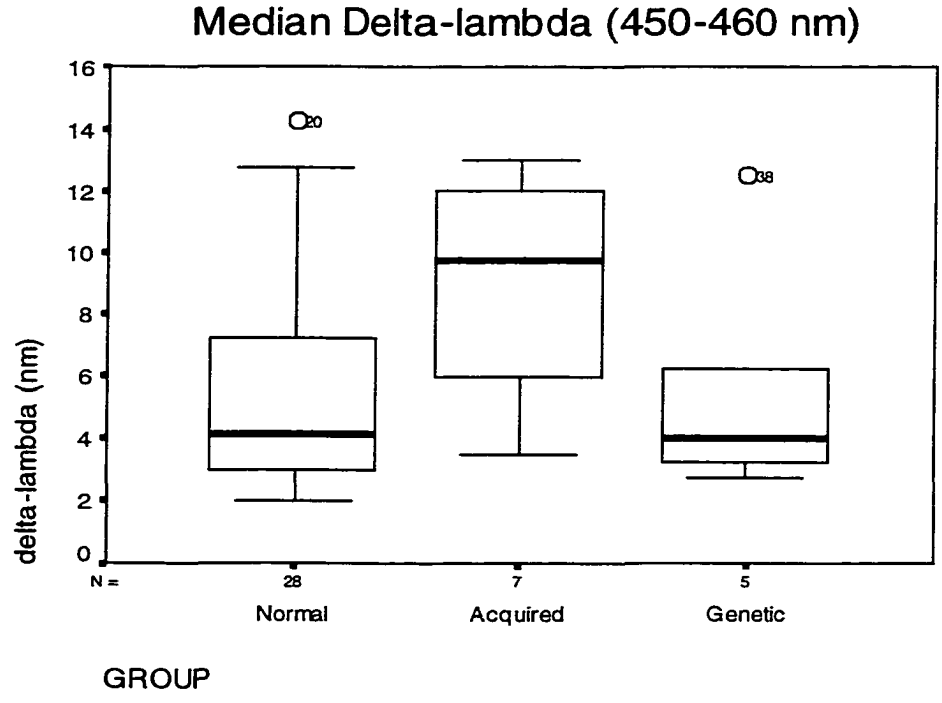


Figure B2. Box-and-whisker plot of group delta-lambda in the spectral region from 450-460 nm. As in Figure B1.

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Each group's mean and median unique hues from the hue- and saturation-scaling experiment are presented in Table B1 and are shown in Figures B3-B10. It is important to note that not every individual tested gave measurable results on each of the hue measures. Lack of a unique red point, for example, is a quite common occurrence, even in observers with superior color discrimination. This is due to a unique red point occurring extra-spectrally, not as a result of a receptoral or signaling problem. Unique blue points were not obtained in 3 of the acquired observers and in 1 genetic observer. Six color-normals, 2 acquired color-deficients, and 1 genetic color-deficient did not display a measurable unique red point. The values given below are, thus, based on the aforementioned reduced sample sizes. (The sample size for each group of observers with measurable unique hues can be found along the abscissa of each graph displaying group medians.)

The mean wavelength at which unique blue occurred in the normal group was slightly higher than those of the other two groups, the acquired group being lowest (see Figure B3). When measuring unique blue, there was a large amount of variability among the genetic color-deficient observers. When group medians are considered, a different pattern emerges than when comparing group means: a trend toward lower unique blues from normals to acquireds to genetics (see Figure B4). Unique green was found, on average, to be highest for the genetic color-deficient group and lowest for the color-normal group (see Figure B5). Like unique blue, a similar trend reversal occurred among group median unique green values, where the groups with the highest and lowest means had the lowest and highest medians. Variability, while present in each group, was much less in the genetic group compared to the normal group, with only one extreme point found. Values among color-normal and acquired color-deficient observers overlap

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Table B1. Hue- and Saturation-scaling: Unique Hues (in nm) and Saturation Integrals (in percent)

| Group | Normal | Acquired | Genetic |
|---|---|---|---|
| Mean Unique Blue Point | 464.93 | 460.25 | 462.25 |
| SEM | 1.27 | 4.09 | 14.92 |
| Median | 465.00 | 460.50 | 458.50 |
| Mean Unique Green Point | 513.75 | 514.00 | 518.60 |
| SEM | 2.20 | 8.41 | 20.48 |
| Median | 509.50 | 507.00 | 500.00 |
| Mean Unique Yellow Point | 577.82 | 581.00 | 563.80 |
| SEM | 0.80 | 1.25 | 18.40 |
| Median | 577.00 | 583.00 | 566.00 |
| Mean Unique Red Point | 649.91 | 644.00 | 597.25 |
| SEM | 1.79 | 5.10 | 31.29 |
| Median | 650.00 | 640.00 | 610.00 |
| Mean Saturation Integral | 76.28 | 69.38 | 76.73 |
| SEM | 1.85 | 6.34 | 2.85 |
| Median | 78.17 | 76.09 | 79.68 |

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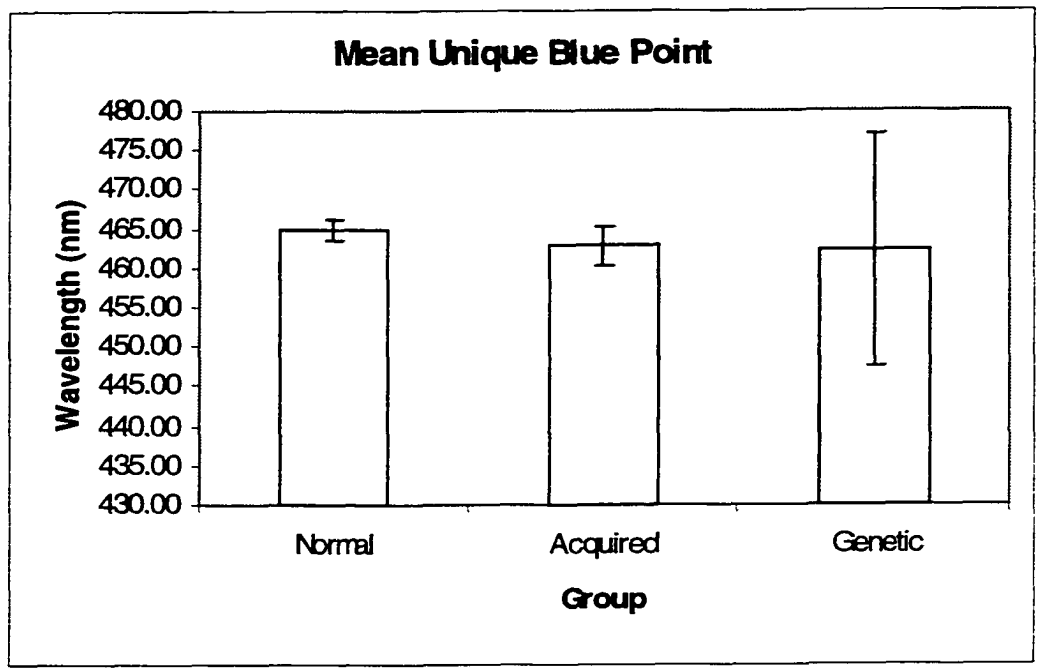


Figure B3. Group mean unique blue point. Error bars equal +/- 1 SEM.

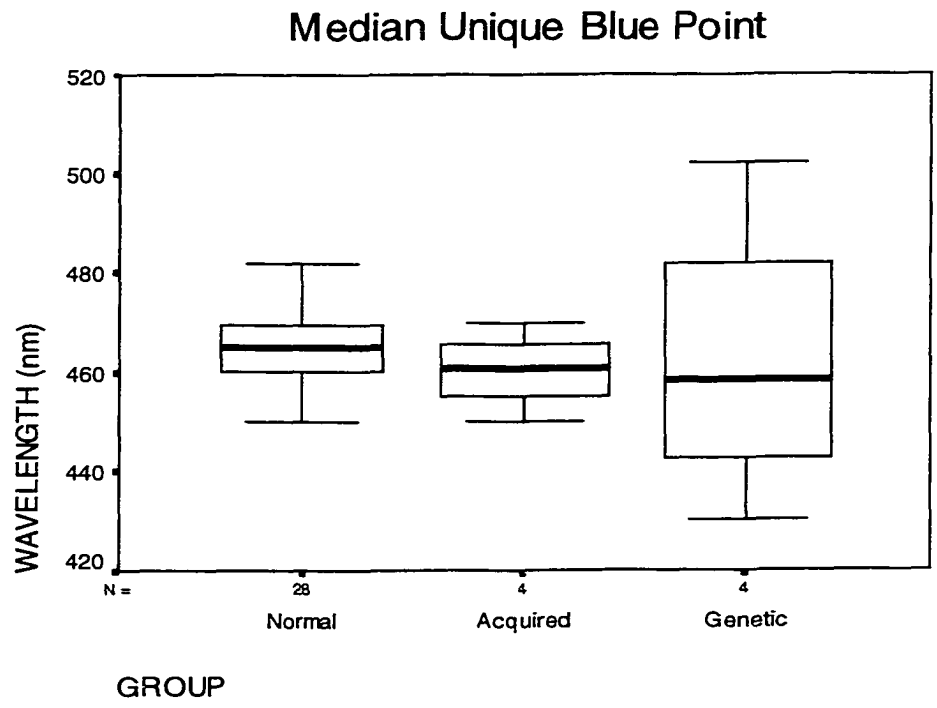


Figure B4. Box-and-whisker plot of group unique blue point. As in Figure B1.

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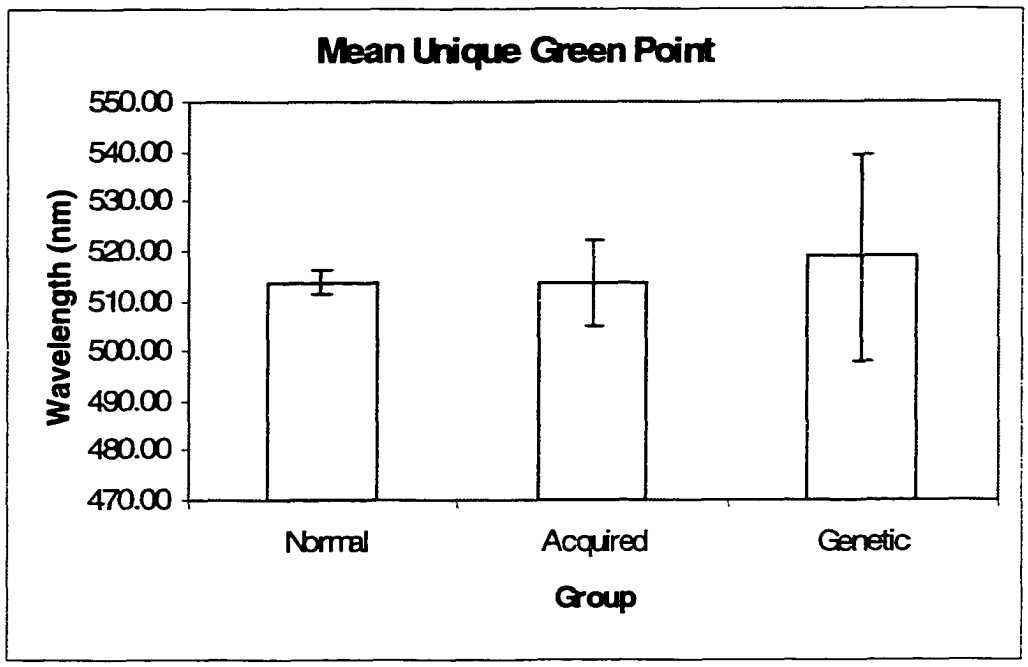


Figure B5. Group mean unique green point. Error bars equal +/- 1 SEM.

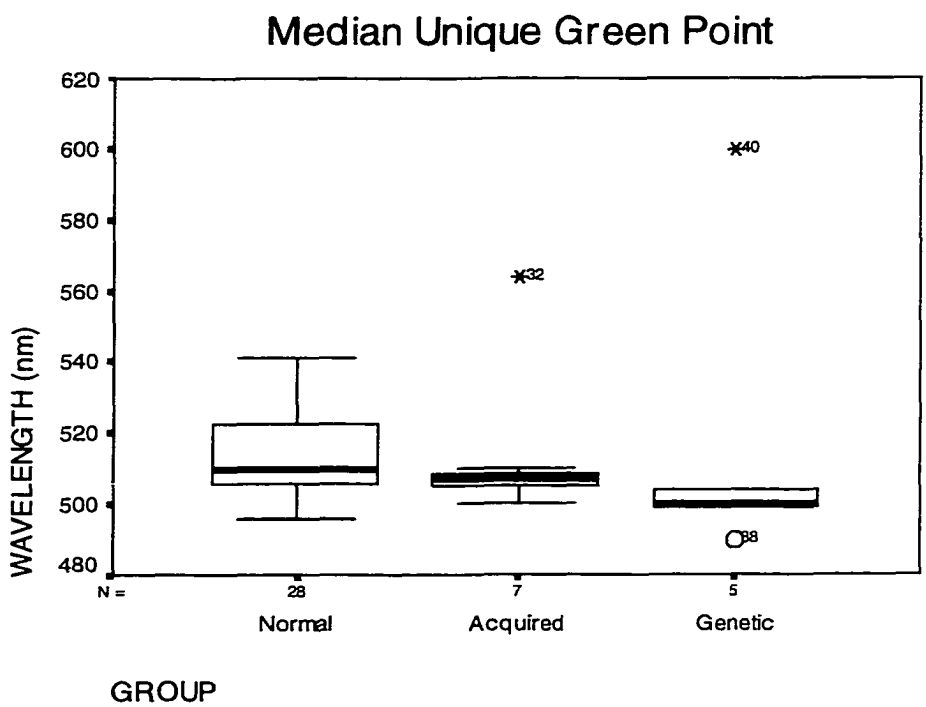


Figure B6. Box and-whisker plot of group unique green point. As in Figure B1.

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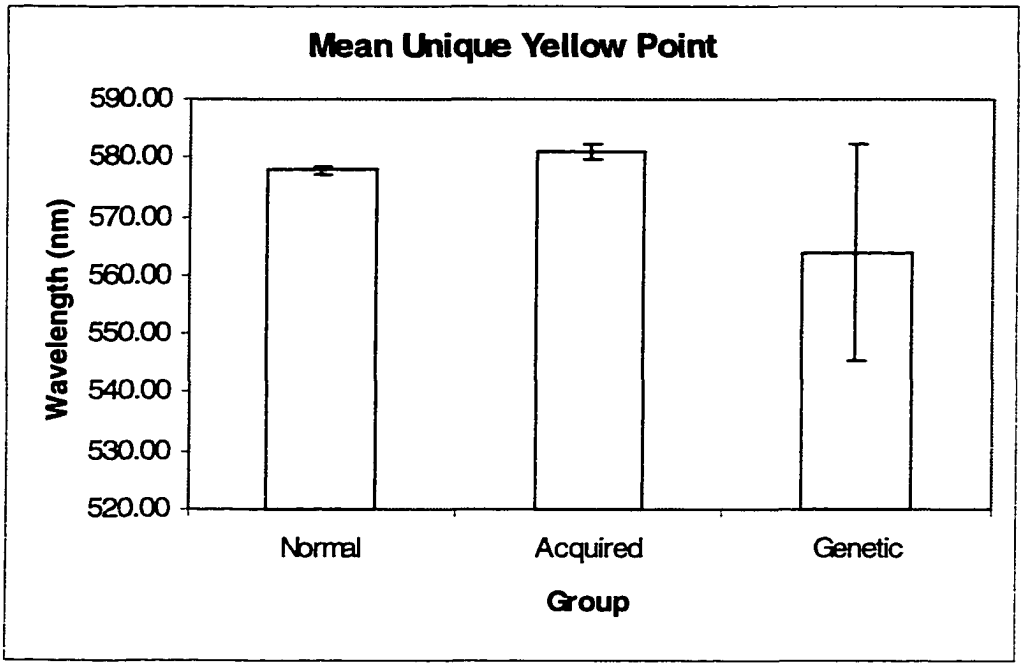


Figure B7. Group mean unique yellow point. Error bars equal +/- 1 SEM.

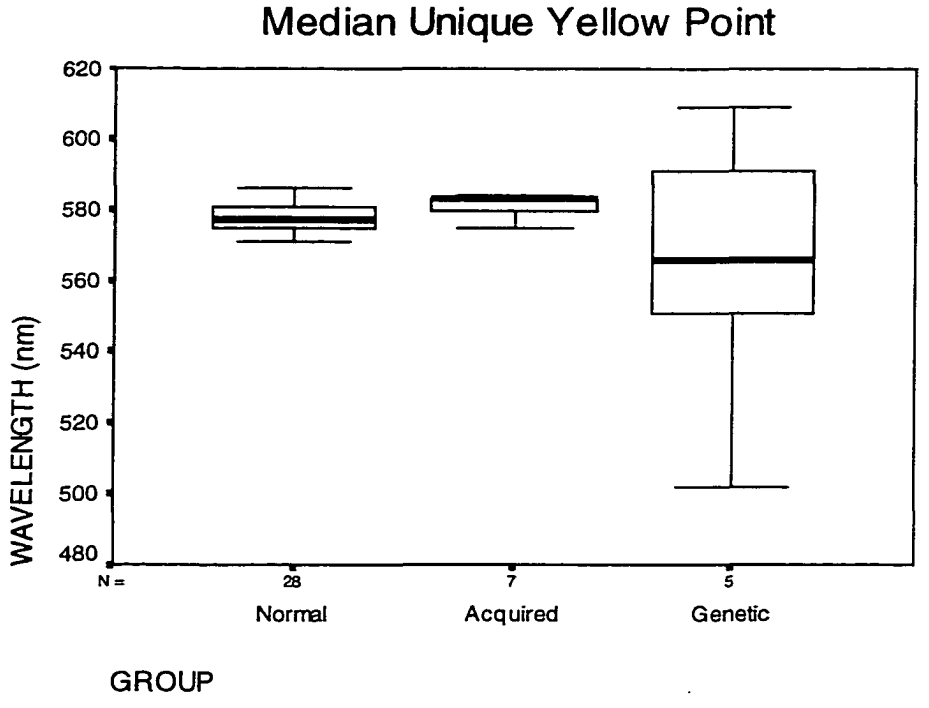


Figure B8. Box-and-whisker plot of group unique yellow point. As in Figure B1.

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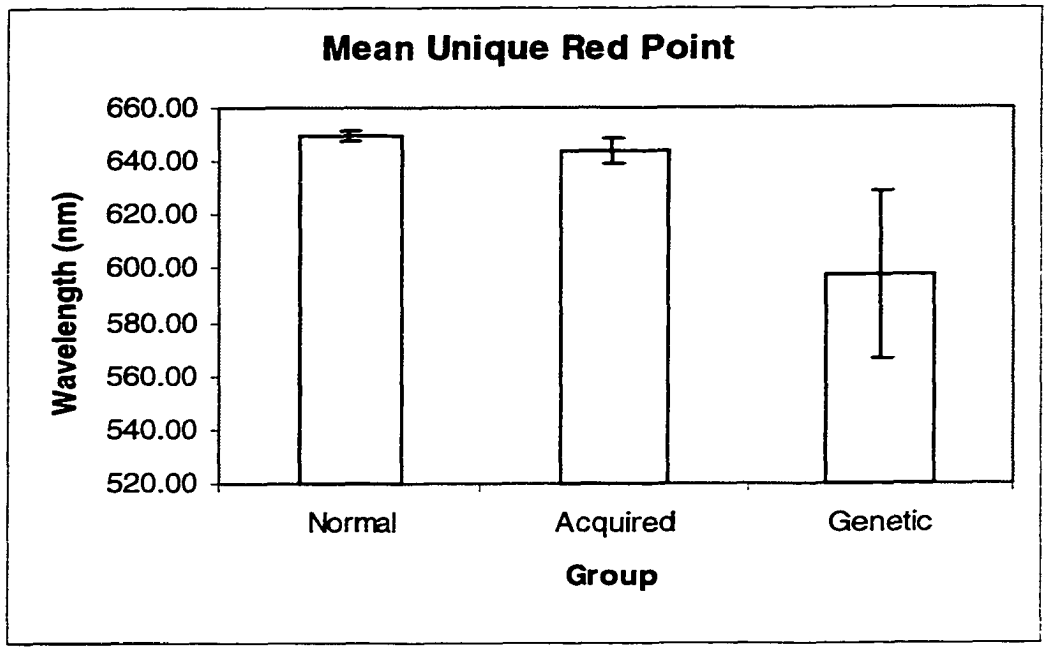


Figure B9. Group mean unique red point. Error bars equal +/- 1 SEM.

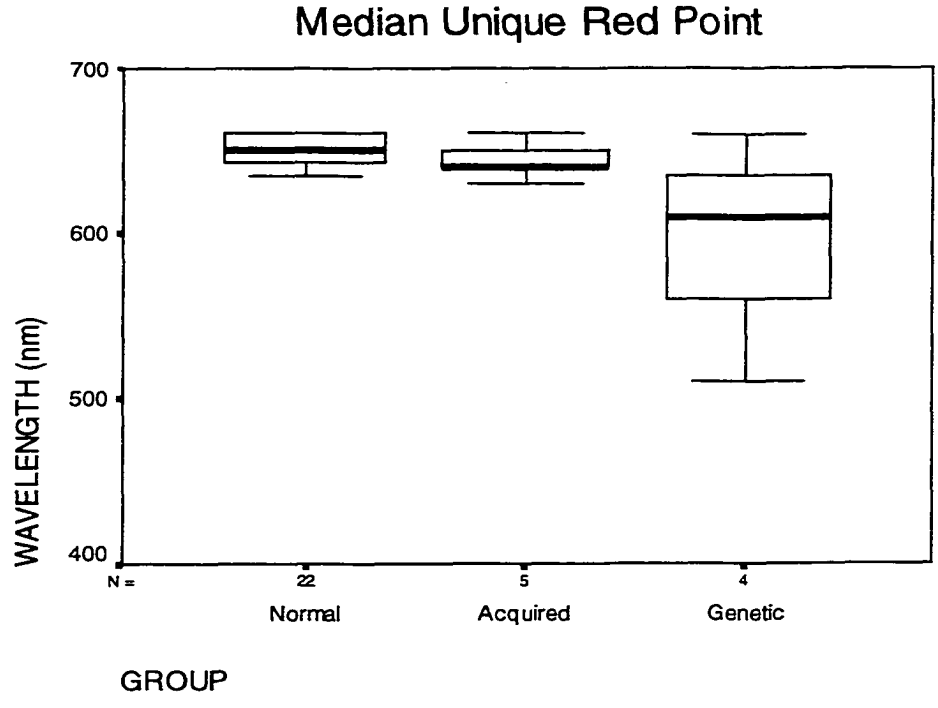


Figure B10. Box-and-whisker plot of group unique red point. As in Figure B1.

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much more so (see Figure B6). The acquired observers had the highest mean unique yellow, followed closely by the normals. The genetics, however, on average, needed a wavelength some 14 nm shorter to be acceptable (see Figure B7). For yellow unique hues, trends found among group medians paralleled those found among group means. Despite needing the shortest wavelength, on average, for unique yellow, the variability across genetic color-deficient observers was substantial, with a range of more than 100 nm (see Figure B8). Color-normals found unique red at a mean wavelength above that of both color-deficient groups, but this difference was much greater when compared to genetics (see Figure B9). In assessing median unique red points, while the range of individual values across observers overlapped among all groups, the majority of genetic color-deficients still needed a lower wavelength for their unique point (see Figure B10). Hence, the observed greater difference between the normal and genetic groups' means compared to the difference between the normal and acquired groups' means.

The mean saturation integral for the normals across the region of color space sampled nearly matched that for genetics. The acquireds yielded a slightly lower mean (see Table B1 and Figure B11). Variability among individuals' saturation integrals was highest within the acquired group, followed by the color-normals and genetic color-deficients, respectively; however, there was considerable overlap among the scores across all three groups (see Figure B12).

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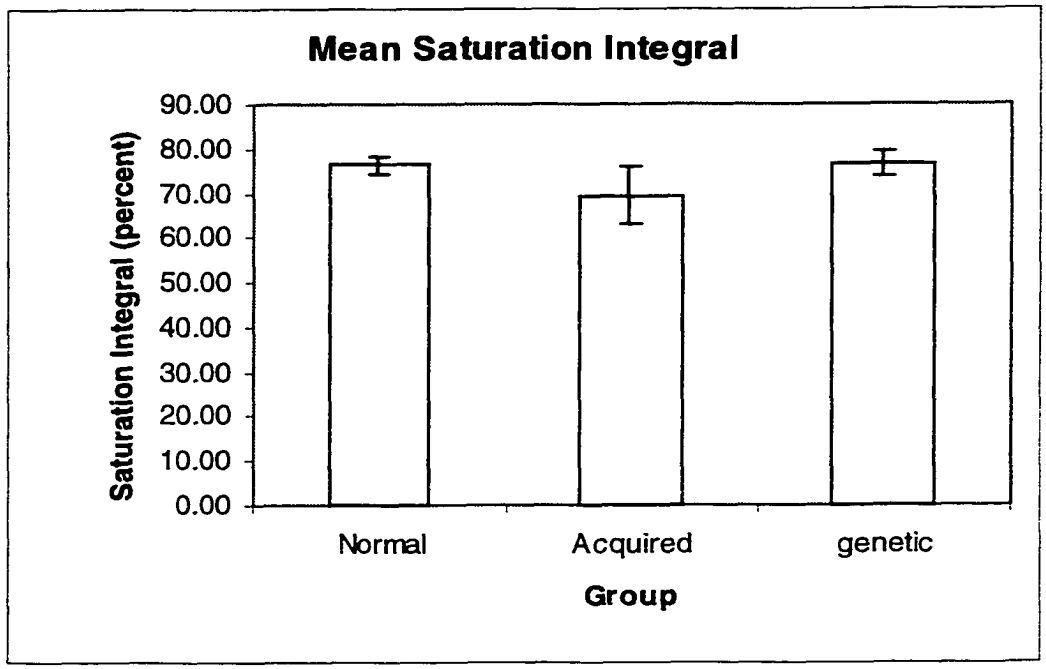


Figure B11. Group mean saturation integral. Error bars equal +/- 1 SEM.

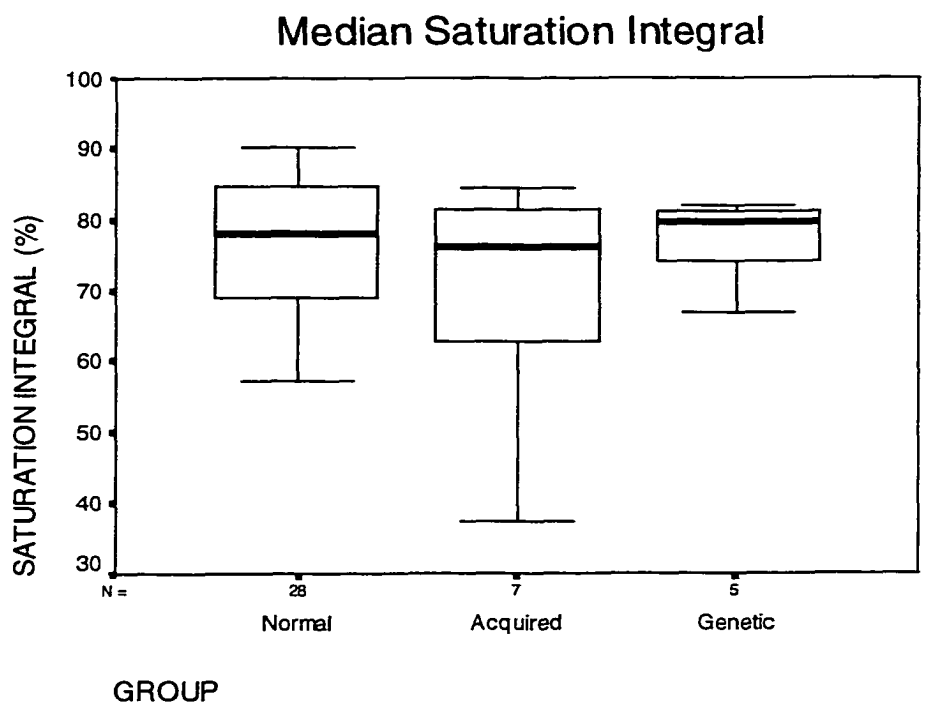


Figure B12. Box-and-whisker plot of group saturation integral. As in Figure B1.

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Anomaloscopy: HFP. Group differences in luminance ratios of yellow to red light are more apparent when viewing the median data (see Figure B13). The reversed trend found among mean luminance ratios of yellow to green light is maintained among the medians (see Figure B14). In both instances, the groups' data did overlap, but, as described in the Results section, there was a bimodal distribution of ratios in the genetic group.

Anomaloscopy: Rayleigh matching. The average median luminance ratio needed to make a Rayleigh match, unlike the mean, was larger for the normal and acquired color-deficient groups compared to the genetic group (see Figure B15). The "extreme" value plotted above the genetic group's box-and-whisker plot is the one observer who needed more green than red in his color matches.

The broader range of acceptable green to red match ratios in the genetic color-deficient group was maintained among group medians (see Figure B16), though it was not as drastic as it had been among group means. The trend for higher mean midpoints for the range of acceptable matches among genetics was found to reverse itself among group medians as, again, only one genetic color-deficient consistently needed much more green than red in his color matches (see Figure B17).

Median anomalous quotients for the color-normal and acquired color-deficient groups, like the means, were also close to 1.00; however, unlike the means, the genetic group's median AQ was much lower than the other groups', due, again, to a bimodal distribution among observers (see Figure B18).

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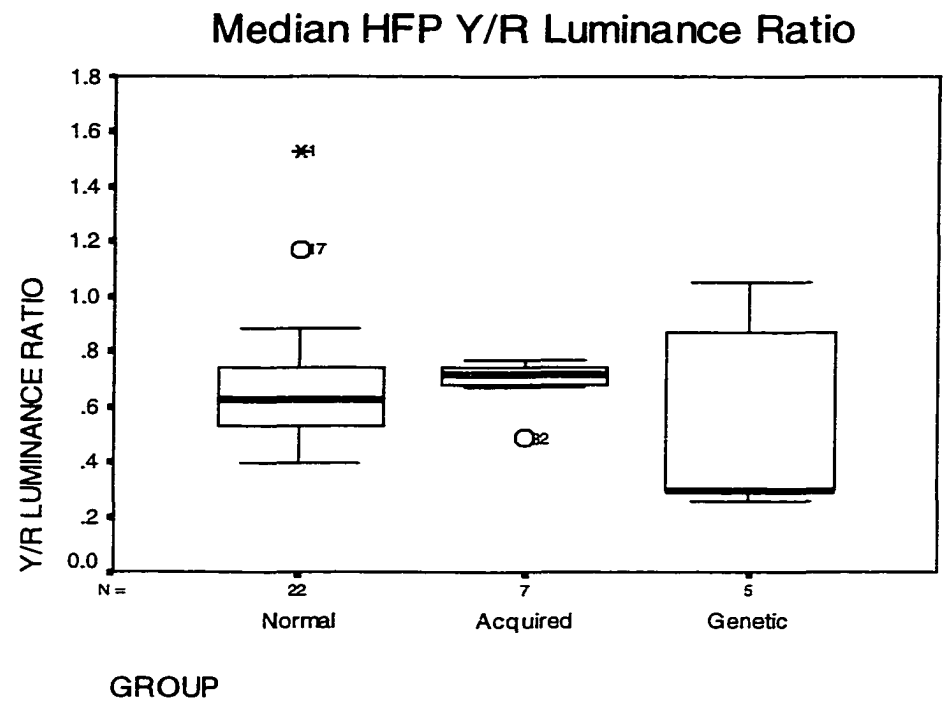


Figure B13. Box-and-whisker plot of group yellow to red luminance ratio needed to minimize a yellow alternating with red flicker. As in Figure B1.

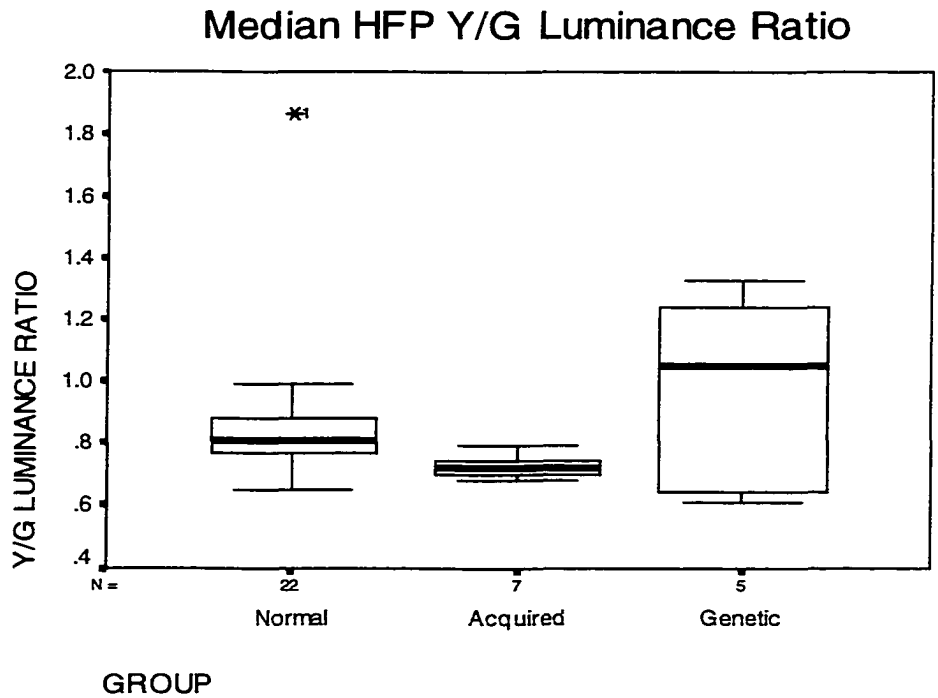


Figure B14. Box-and-whisker plot of group yellow to green luminance ratio needed to minimize a yellow alternating with green flicker. As in Figure B1.

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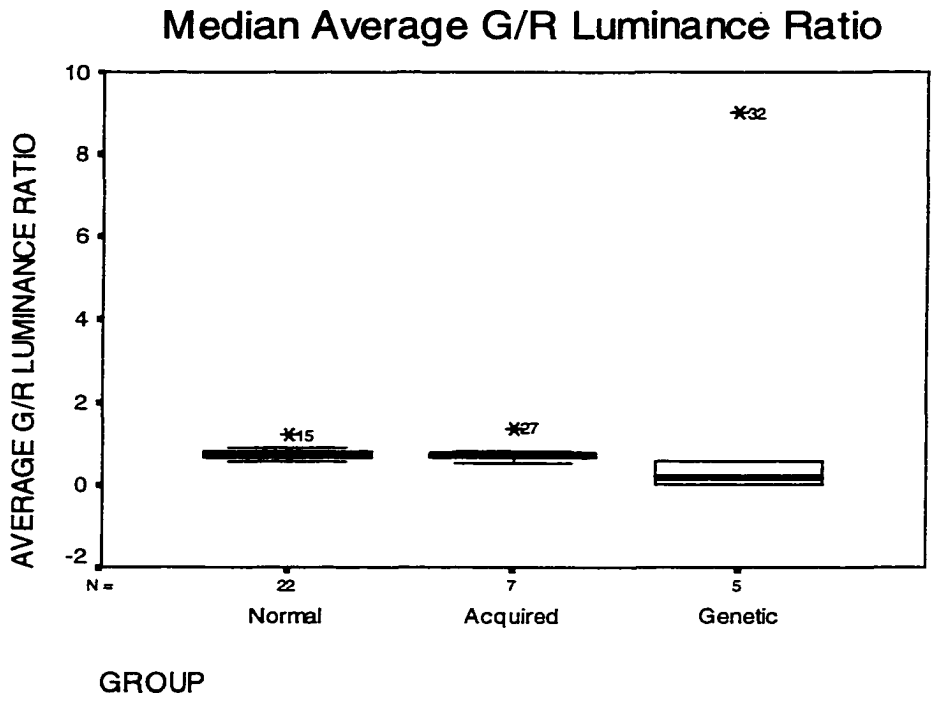


Figure B15. Box-and-whisker plot of mean green to red luminance ratio required by each group to make a Rayleigh match (match to a standard yellow). As in Figure B1.

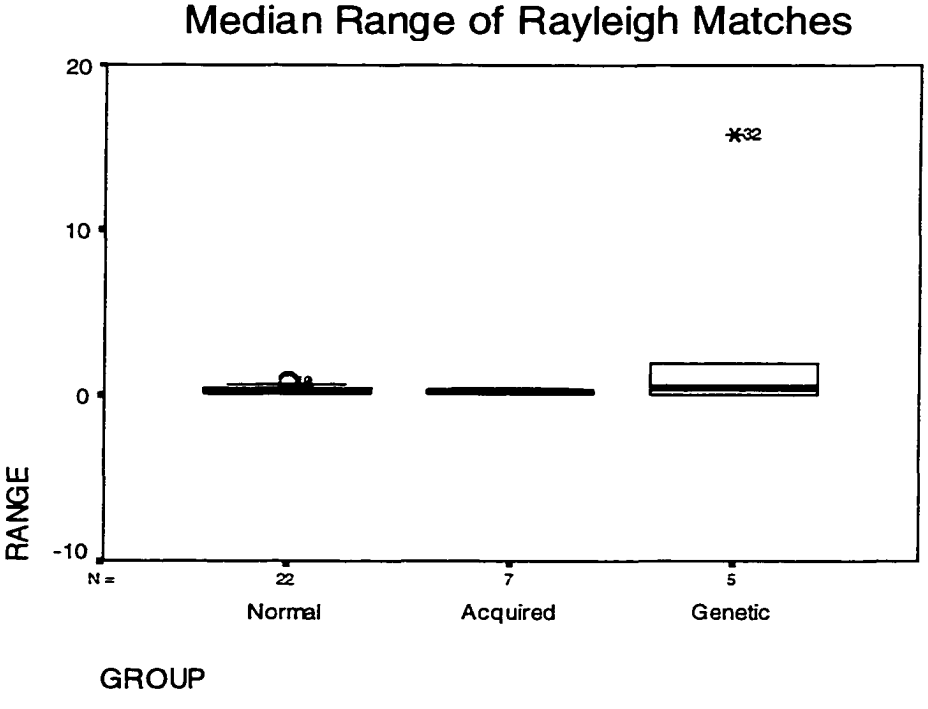


Figure B16. Box-and-whisker plot of range of green to red luminance ratios accepted as Rayleigh matches for each group. As in Figure B1.

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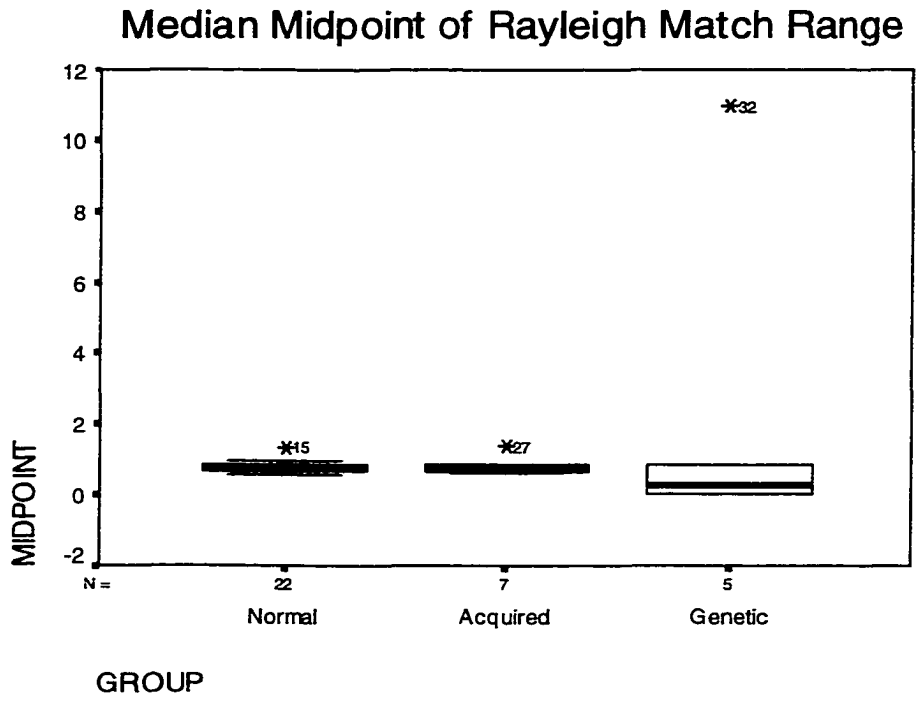


Figure B17. Box-and-whisker plot of midpoint of green to red luminance ratios accepted as Rayleigh matches for each group. As in Figure B1.

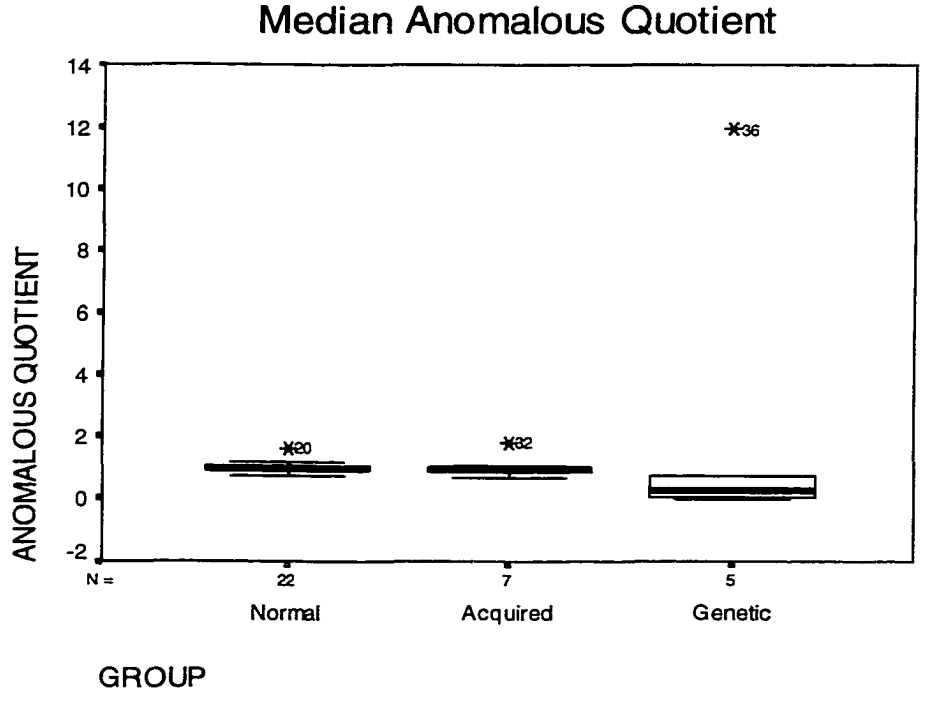


Figure B18. Box-and-whisker plot of group anomalous quotient. As in Figure B1.

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Under conditions of chromatic adaptation, the trend of acquired color-deficients displaying the highest luminance ratios of yellow to red light and genetics the lowest continued at the level of group medians, where the gaps between adjacent groups widened even further than was seen among means (see Figure B19). Ratios varied largely amongst individual observers within each group, and considerable overlap is seen between groups. Genetics needing a larger luminance ratio of yellow to green light was also maintained among group medians (with nearly identical performances by normals and acquireds). There was less variability within each group, though values still tended to overlap (see Figure B20).

It was mentioned in the Results section that, under chromatic adaptation, acquireds needed a substantially larger mean green to red luminance ratio in order to produce a match due to one extreme observer. This trend remains for group medians, but by a much narrower margin. With the exception of this one individual, variability is minimal within the normal and acquired groups (see Figure B21). As opposed to the group differences among mean range of acceptable Rayleigh matches, medians were nearly equal across all groups, with normals actually being slightly higher (see Figure B22). While the median midpoint was still highest for the acquired color-deficient group (like the mean), the genetic group's median, was well below that of each of the other groups' (see Figure B23). The marked differences found among mean AQs, resulting from an extreme value in each of the two color-deficient groups, were reduced at the level of group median, as the genetic group's AQ fell below that of the normal's and acquired's (see Figure B24).

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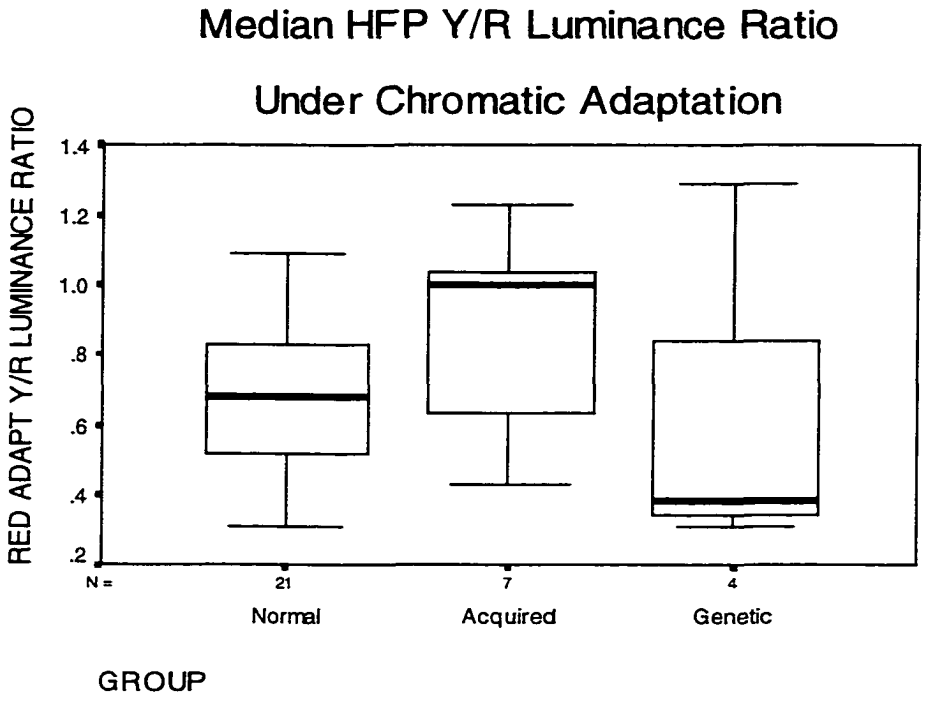


Figure B19. Box-and-whisker plot of group yellow to red luminance ratios needed to minimize a yellow alternating with red flicker under chromatic adaptation. As in Figure B1.

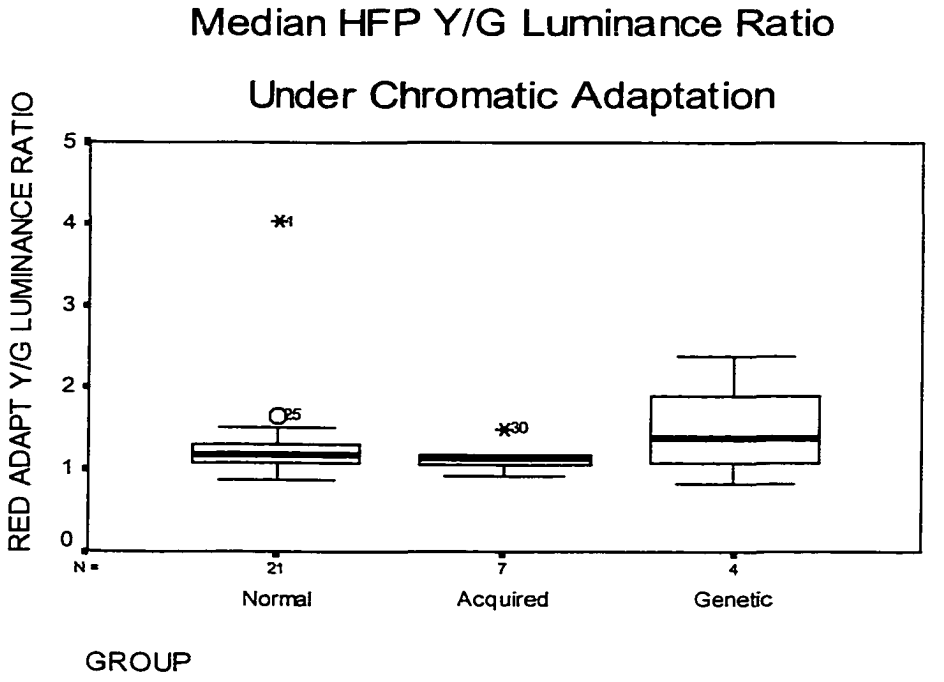


Figure B20. Box-and-whisker plot of group yellow to green luminance ratios needed to minimize a yellow alternating with green flicker under chromatic adaptation. As in Figure B1.

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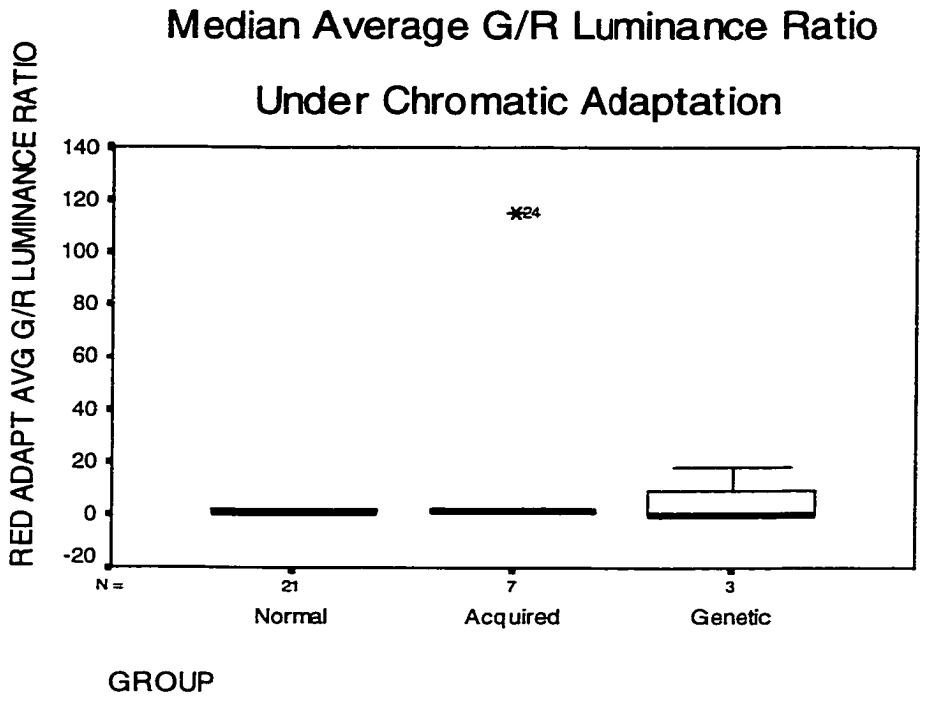


Figure B21. Box-and-whisker plot of group mean green to red luminance ratio required to make a Rayleigh match (match to a standard yellow) under chromatic adaptation. As in Figure B1.

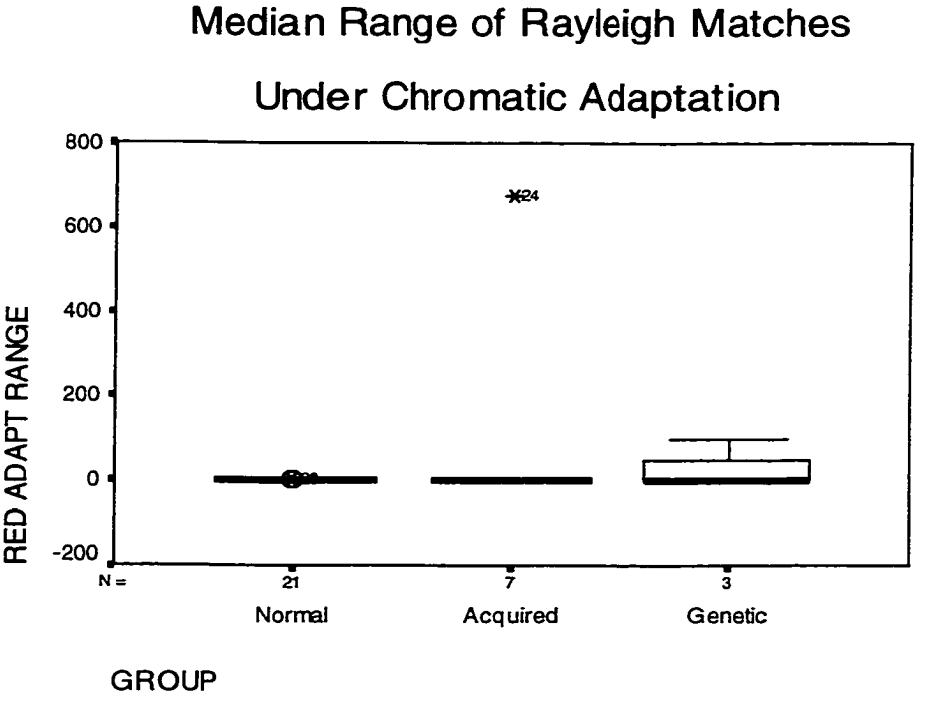


Figure B22. Box-and-whisker plot of range of green to red luminance ratios accepted as Rayleigh matches for each group under chromatic adaptation. As in Figure B1.

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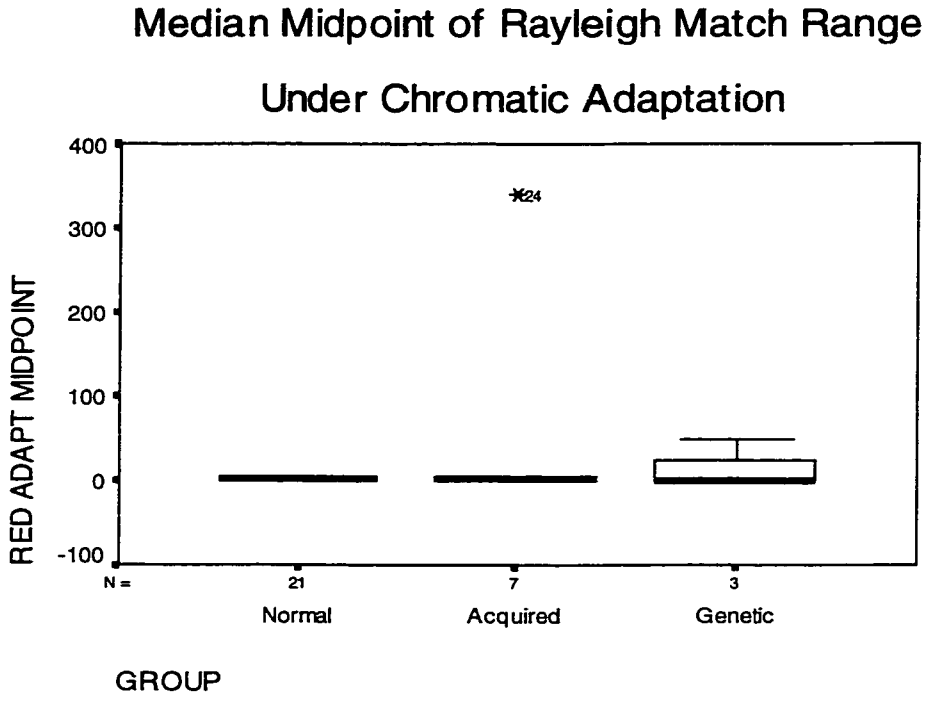


Figure B23. Box-and-whisker plot of midpoint of green to red luminance ratios accepted as Rayleigh matches for each group under chromatic adaptation. As in Figure B1.

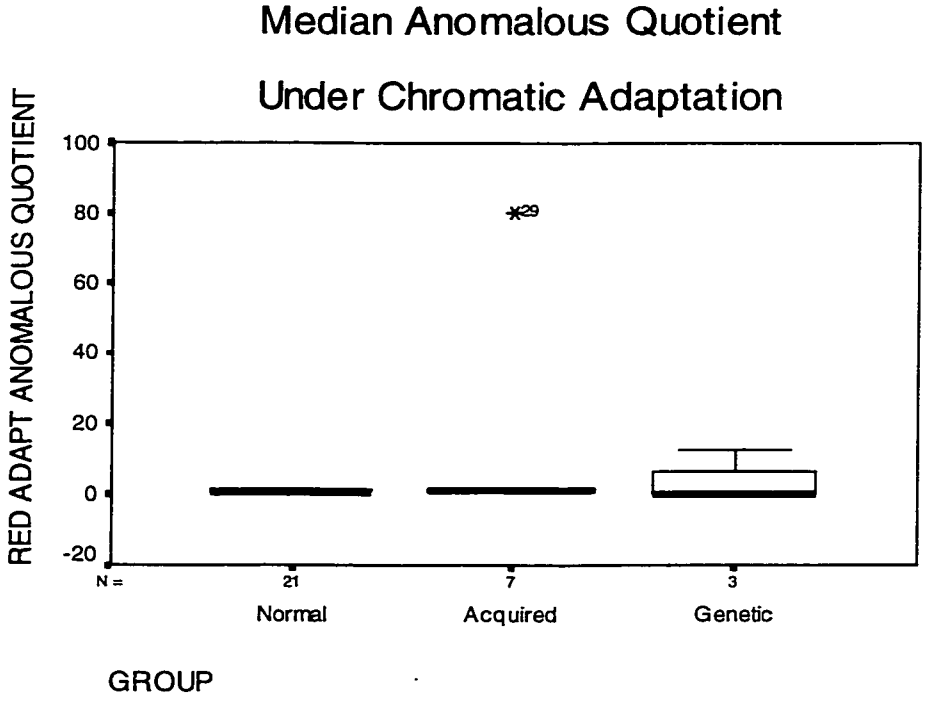


Figure B24. Box-and-whisker plot of anomalous quotient for each group under chromatic adaptation. As in Figure B1.

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Even though there was a general trend toward observers needing less red and green light for HFP when chromatically adapted (see Figures B25 and B26), individual differences were found to exist. When examining green to red luminance ratios needed to make a Rayleigh match on the level of group medians, acquireds were found to need more than twice as much green, and normals and genetics almost twice as much green as when they were not adapted (see Figure B27). The marked differences in the range of acceptable match ratios between adaptation conditions found among means was substantially less so among group medians (see Figure B28). For range midpoints, among group medians, substantial differences between the two conditions were found in the color-normal and acquired color-deficient groups. The genetic group's difference was small, still requiring more red than green light (see Figure B29). In assessing anomalous quotients across conditions, the trend for all color-deficients having higher AQs under chromatic adaptation remained in only the acquired group when medians were considered. The color-normal group's median anomalous quotient decreased slightly (indicating a general need for more red than green), while that of the genetic group remained virtually identical across conditions (see Figure B30).

Examining group median differences of Rayleigh ratios removes much of the variability found among the means, with acquireds still showing the greatest difference between conditions, followed by normals, and then genetics (see Figure B31). Color-normal and (for the most part) acquired color-deficient observers are situated closely among one another, the genetics showing really the largest range.

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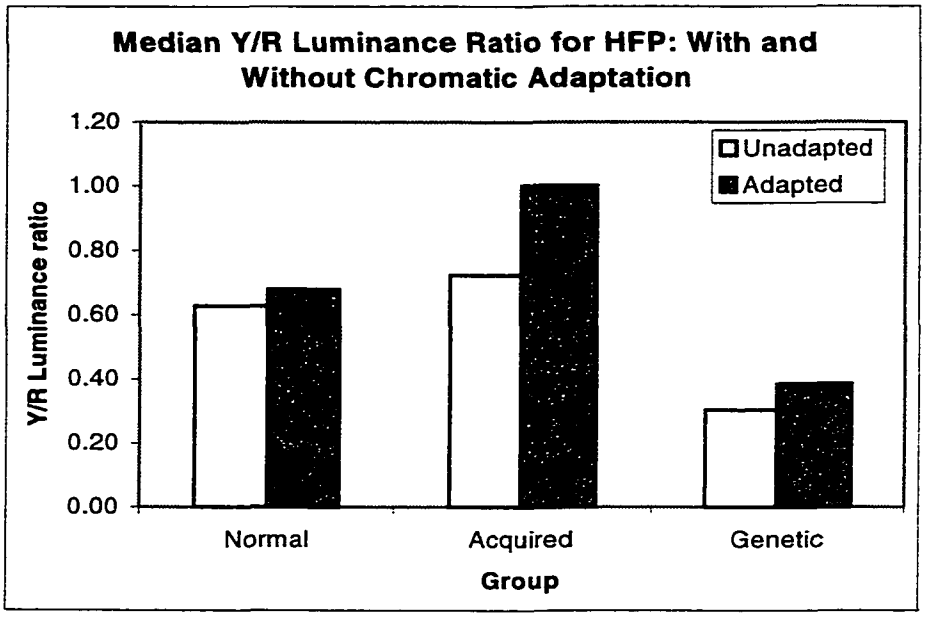


Figure B25. Median yellow to red luminance ratio needed to minimize a yellow alternating with red flicker for each group with and without chromatic adaptation.

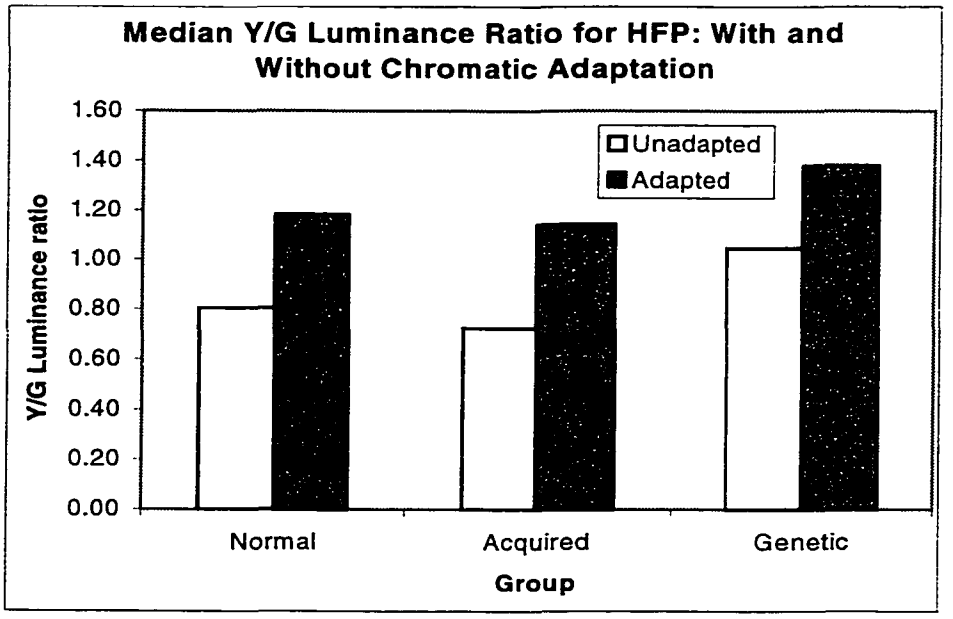


Figure B26. Median yellow to green luminance ratio needed to minimize a yellow alternating with red flicker for each group with and without chromatic adaptation.

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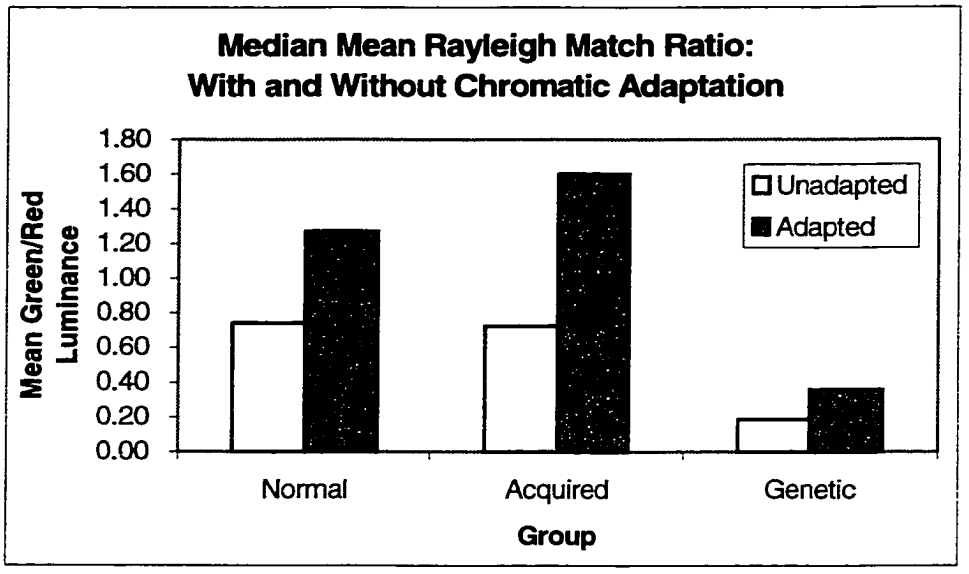


Figure B27. Average median green to red luminance ratio required by each group to make a Rayleigh match (match to a standard yellow) with and without chromatic adaptation.

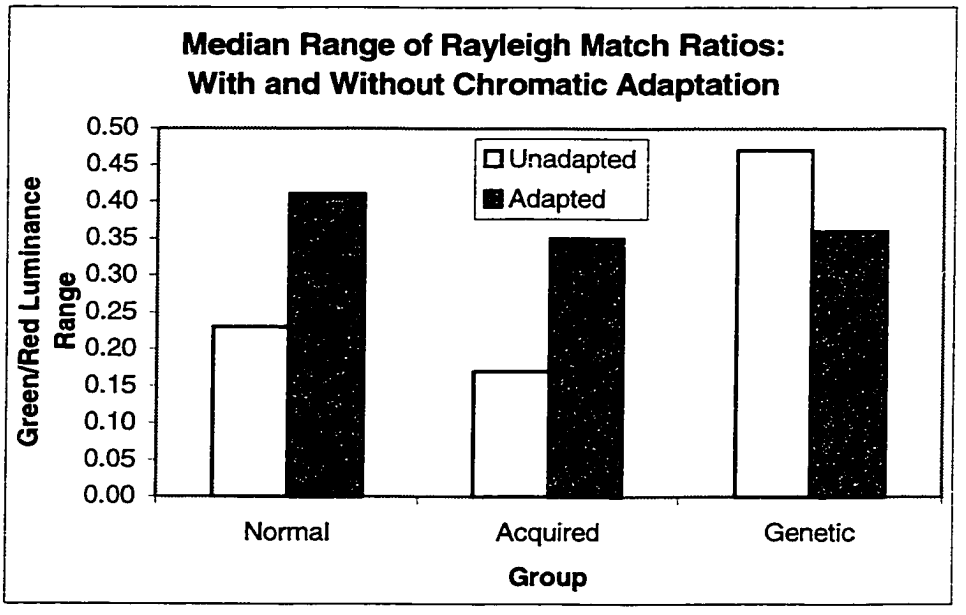


Figure B28. Median range of green to red luminance ratios accepted as Rayleigh matches for each group with and without chromatic adaptation.

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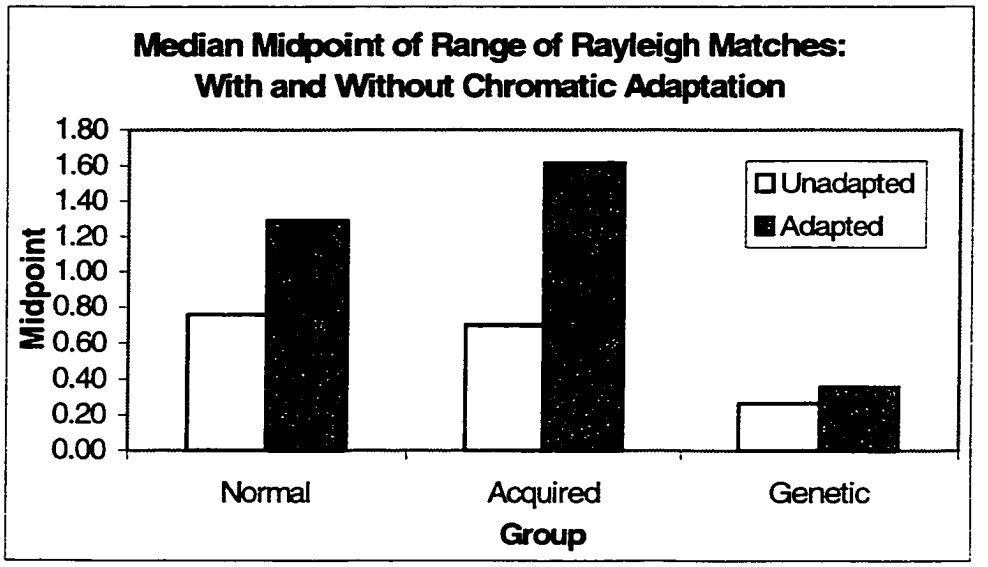


Figure B29. Median midpoint of green to red luminance ratios accepted as Rayleigh matches for each group with and without chromatic adaptation.

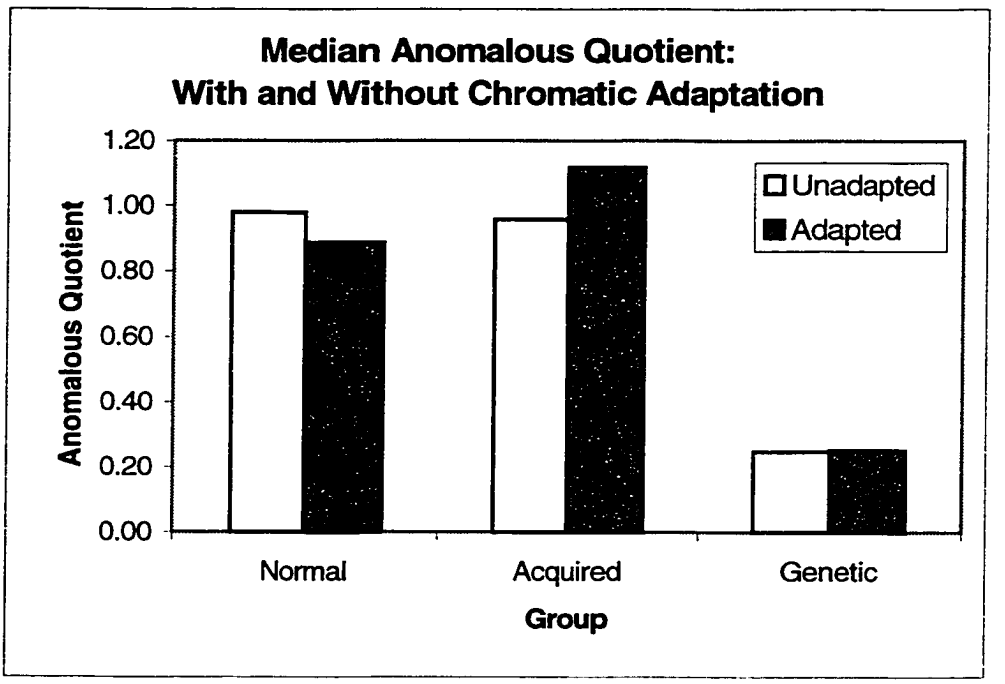


Figure B30. Group median anomalous quotient with and without chromatic adaptation.

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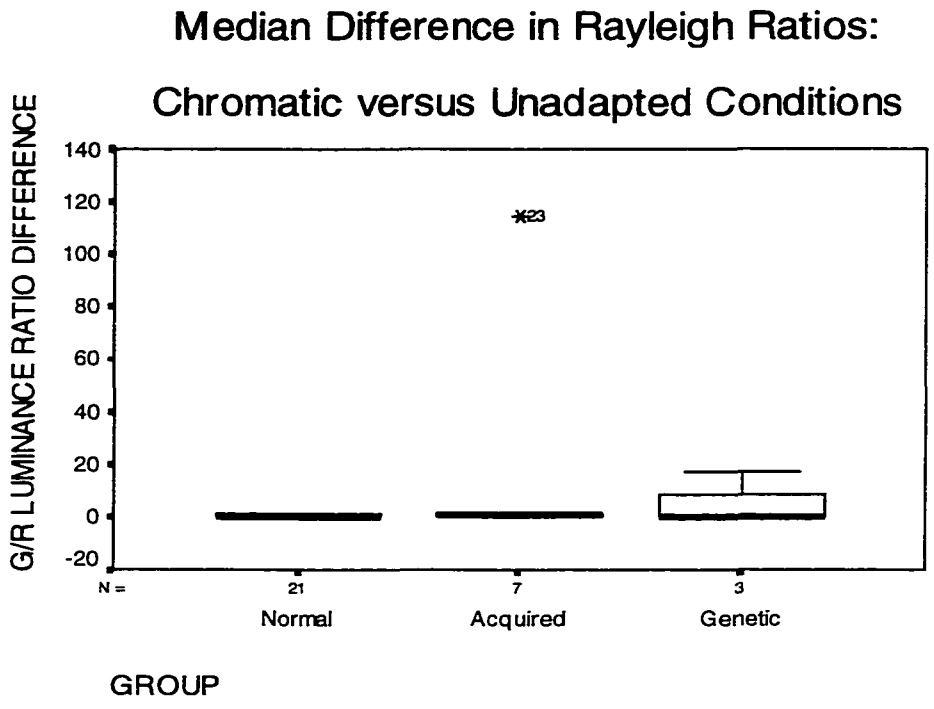


Figure B31. Box-and-whisker plot of median difference of mean adapted and mean unadapted Rayleigh ratios for each group. As in Figure B1.

Rod and SWS cone sensitivity. Table B2 displays the mean and median rod sensitivities, optical densities, SWS cone increment thresholds, and corrected SWS cone sensitivity thresholds for the three groups of observers.

For a 534 nm light flashed at an eccentricity of 2°, the median relative radiance of the acquired group, unlike that of the mean, was found to be above that of the normal group, with much variability across, and overlap between, these two groups (see Figure B32). At 6° eccentricity, the trends found among the means reversed among the medians. The color-normal and genetic color-deficient groups had identical medians, while that of the acquired color-deficients was slightly lower (see Figure B33). Additionally, values among the genetics were quite proximal to one another, whereas those of the other groups had larger ranges and a greater degree of overlap.

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Table B2. Rod and SWS Cone Sensitivity: Absolute Limens (in relative radiance), Optical Densities, and SWS Cone Increment (in relative radiance) and Sensitivity Thresholds (in log relative radiance)

| Group | Normal | Acquired | Genetic |
|---|---|---|---|
| Mean 534 nm Absolute Limen at 2° eccentricity | 0.52 | 0.41 | 0.24 |
| SEM | 0.12 | 0.13 | 0.14 |
| Median | 0.19 | 0.27 | 0.16 |
| Mean 534 nm Absolute Limen at 6° eccentricity | 0.18 | 0.29 | 0.08 |
| SEM | 0.05 | 0.16 | 0.03 |
| Median | 0.07 | 0.06 | 0.07 |
| Mean 425 nm Absolute Limen at 2° eccentricity | 3.16 | 2.53 | 2.67 |
| SEM | 0.81 | 0.33 | 1.89 |
| Median | 2.11 | 2.44 | 0.74 |
| Mean 425 nm Absolute Limen at 6° eccentricity | 0.55 | 0.59 | 0.60 |
| SEM | 0.06 | 0.12 | 0.22 |
| Median | 0.50 | 0.44 | 0.46 |
| Mean G/B Absolute Limen ratio at 2° eccentricity | 0.18 | 0.19 | 0.11 |
| SEM | 0.03 | 0.07 | 0.02 |
| Median | 0.11 | 0.11 | 0.09 |

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Table B2 continued

| | | | |
|---|---|---|---|
| Mean G/B Absolute Limen ratio at 6° eccentricity | 0.35 | 0.46 | 0.14 |
| SEM | 0.09 | 0.24 | 0.02 |
| Median | 0.14 | 0.12 | 0.15 |
| Mean optical density at 2° eccentricity | 0.61 | 0.63 | 0.71 |
| SEM | 0.07 | 0.15 | 0.08 |
| Median | 0.69 | 0.70 | 0.79 |
| Mean optical density at 6° eccentricity due to preretinal absorption | 0.44 | 0.42 | 0.61 |
| SEM | 0.09 | 0.21 | 0.07 |
| Median | 0.59 | 0.67 | 0.55 |
| Mean optical density at 2° eccentricity due to macular pigment | 0.17 | 0.21 | 0.10 |
| SEM | 0.05 | 0.07 | 0.06 |
| Median | 0.12 | 0.18 | 0.14 |
| Mean 425 nm relative radiance required for increment threshold | 2.75 | 2.15 | 1.48 |
| SEM | 0.71 | 0.66 | 0.39 |
| Median | 1.86 | 1.36 | 1.43 |

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Table B2 continued

| | | | |
|---|---|---|---|
| Mean log relative radiance SWS cone threshold | -0.40 | -0.41 | -0.61 |
| SEM | 0.11 | 0.13 | 0.16 |
| Median | -0.26 | -0.33 | -0.48 |

For a 425 nm light at 2° eccentricity, in terms of group medians, the normal group performed slightly better (i.e., had lower thresholds) than the acquired group, neither coming close to the high sensitivities displayed by the genetic group (see Figure B34). There was considerable overlap across all groups, with large ranges found in the normal and genetic groups. Similar to the means, at an eccentricity of 6°, the group medians for a 425 nm light were nearly identical, with each group showing less interobserver variability, and closely matching ranges of thresholds (see Figure B35).

The trend of acquireds having the highest relative radiance ratio of 534 nm to 425 nm light at absolute threshold at an eccentricity of 2° and genetics the lowest was maintained at the level of group medians with the disparity between groups, evident among means, substantially reduced. The range of ratios was found to be moderately compact among the genetics, relative to the normal and acquired groups. The latter two showed much variability and overlap across individual observers' measures (see Figure B36). At a retinal eccentricity of 6°, the pattern found among means (similar to that at 2° eccentricity) reversed itself with medians. Genetics presented a slightly higher ratio than normals, with acquireds placing third. Differences between groups, however, were, in this instance, quite minimal. Variability within groups was less extensive in all

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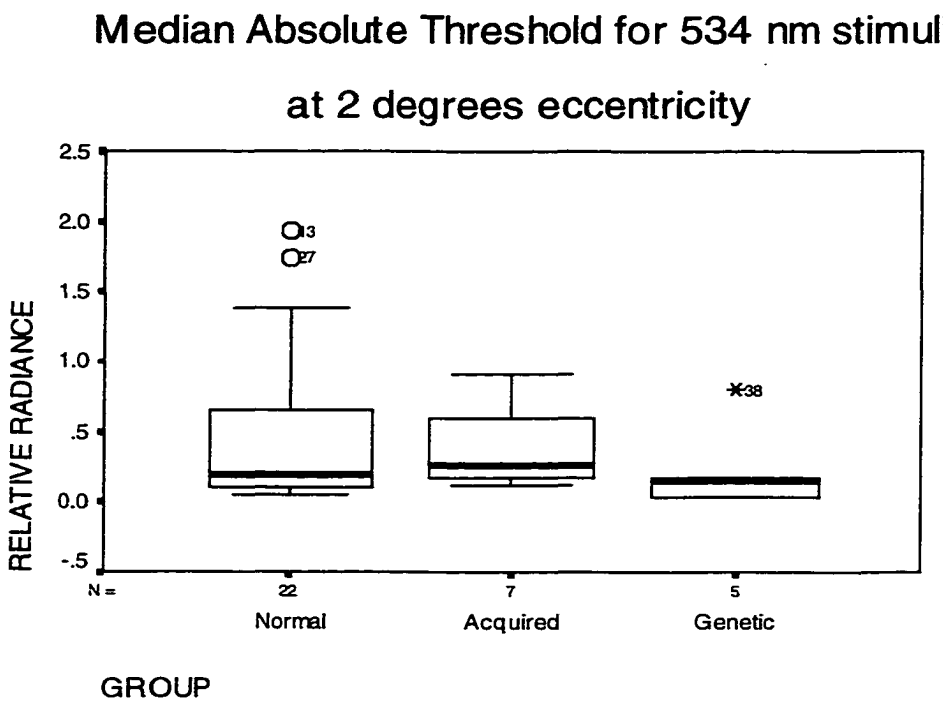


Figure B32. Box-and-whisker plot of relative radiance necessary for just noticeable detection of a flash (absolute limen) of 534 nm light at a retinal eccentricity of 2°. As in Figure B1.

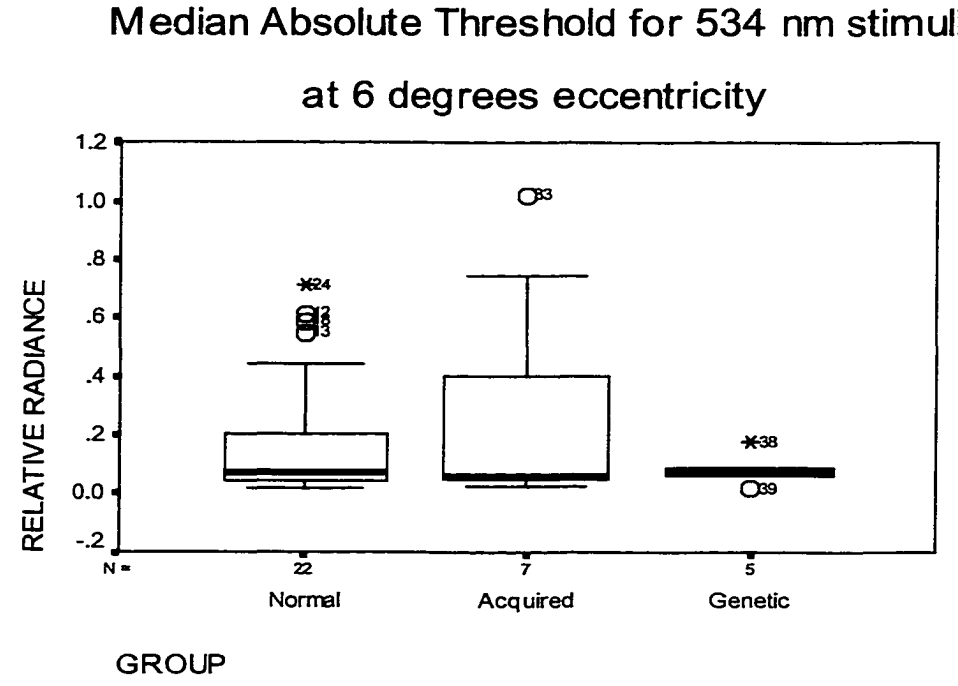


Figure B33. Box-and-whisker plot of relative radiance necessary for just noticeable detection of a flash (absolute limen) of 534 nm light at a retinal eccentricity of 6°. As in Figure B1.

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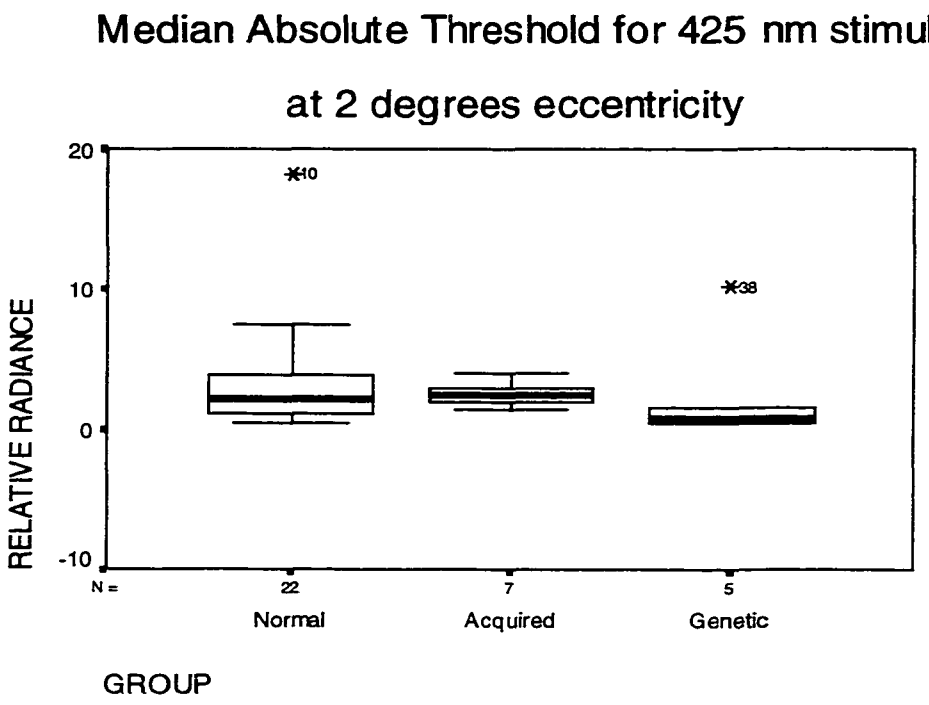


Figure B34. Box-and-whisker plot of relative radiance necessary for just noticeable detection of a flash (absolute limen) of 425 nm light at a retinal eccentricity of 2°. As in Figure B1.

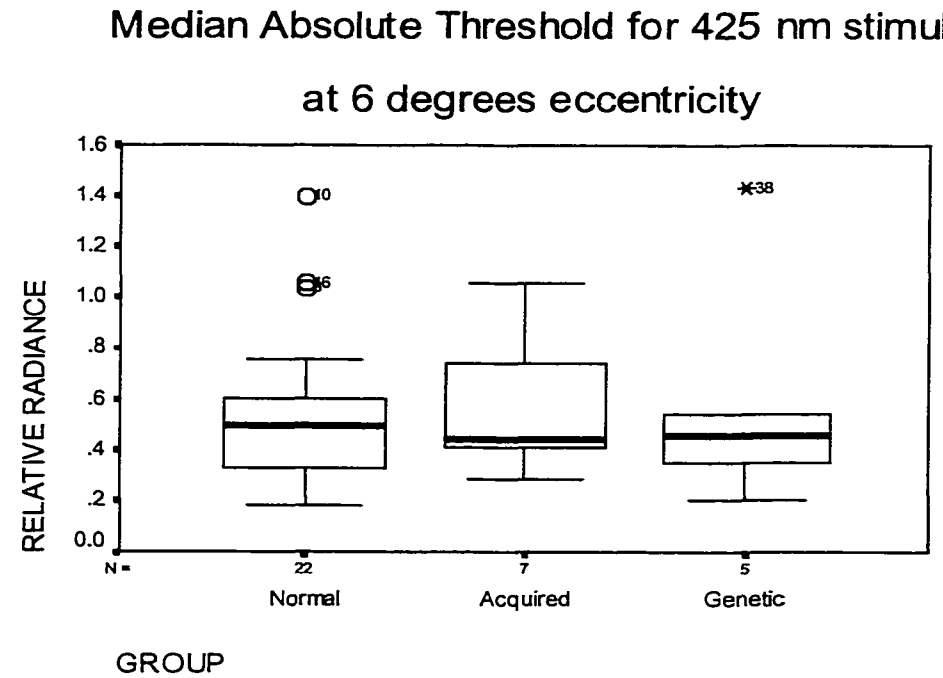


Figure B35. Box-and-whisker plot of relative radiance necessary for just noticeable detection of a flash (absolute limen) of 425 nm light at a retinal eccentricity of 6°. As in Figure B1.

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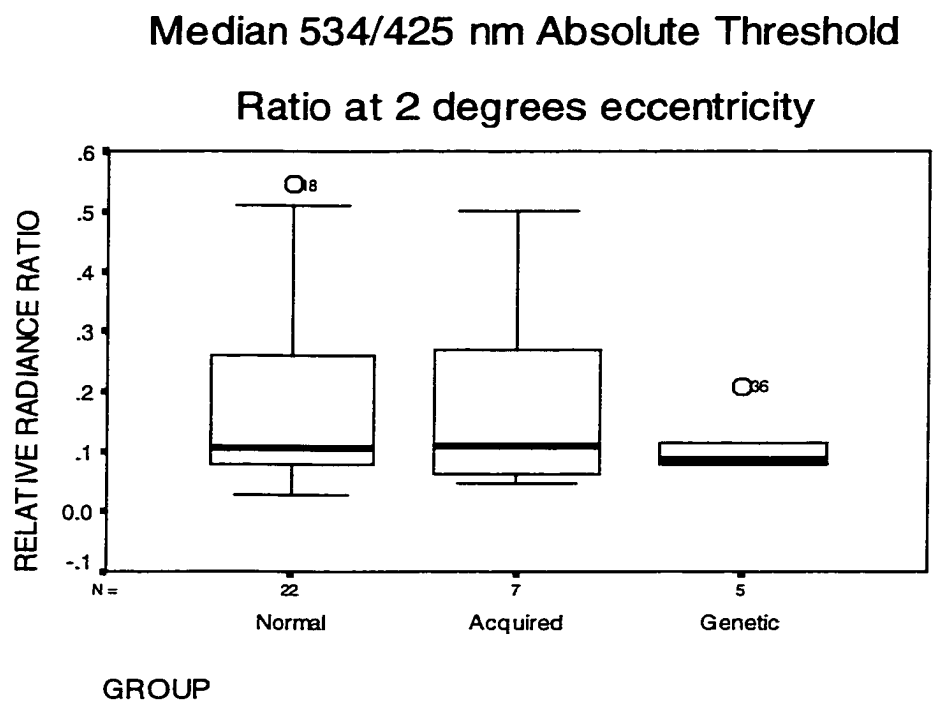


Figure B36. Box-and-whisker plot of 534 to 425 nm relative radiance ratio at absolute limen for each group at a retinal eccentricity of 2°. As in Figure B1.

cases at this eccentricity (see Figure B37). Paralleling the observed trend for genetics having a slightly higher mean density of optical media at 2° compared to normals and acquireds, so too was this group's median (see Figure B38). The degree of variability and overlap among individuals in the normal and acquired groups was quite extensive. At 6° eccentricity, the trend found among mean optical density due to preretinal absorption (with genetics greater by a factor of nearly 1.6) was reversed at the level of group medians. Here the acquireds had about a 1.6 greater density than the genetics, with the normals in between. Variability was found heavily in the acquired group, less so in the normal group, and least among the genetics (see Figure B39).

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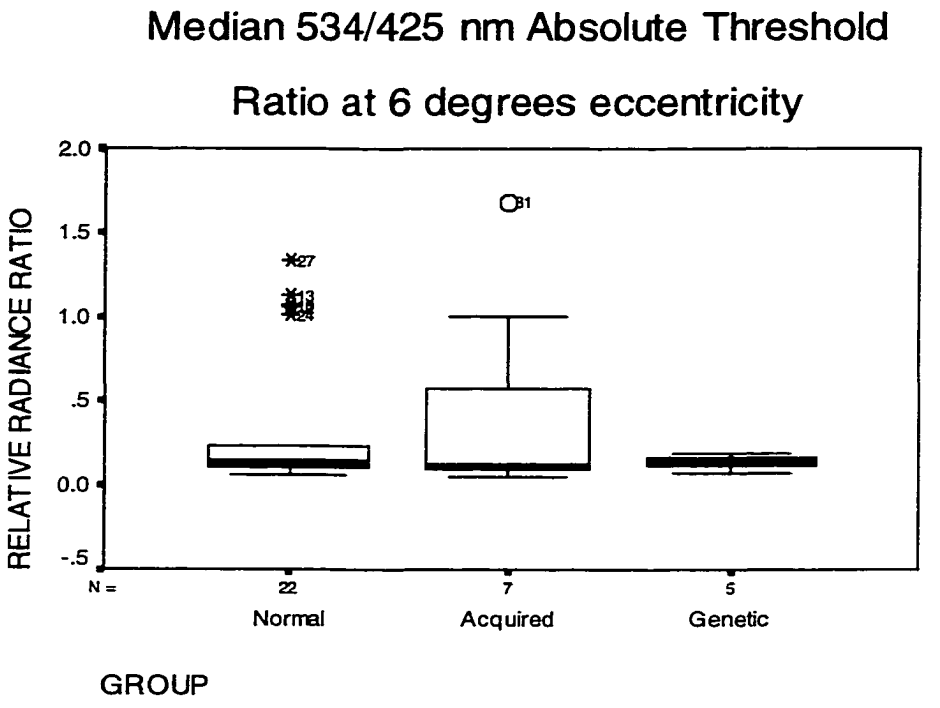


Figure B37. Box-and-whisker plot of 534 to 425 nm relative radiance ratio at absolute limen for each group at a retinal eccentricity of 6°. As in Figure B1.

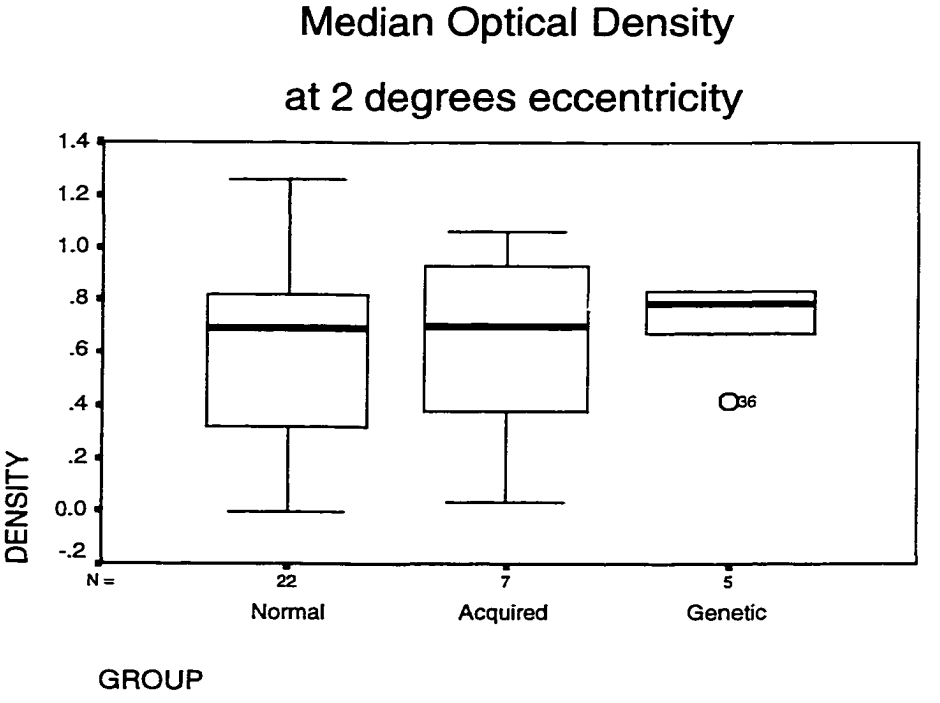


Figure B38. Box-and-whisker plot of optical density for each group at a retinal eccentricity of 2°. As in Figure B1.

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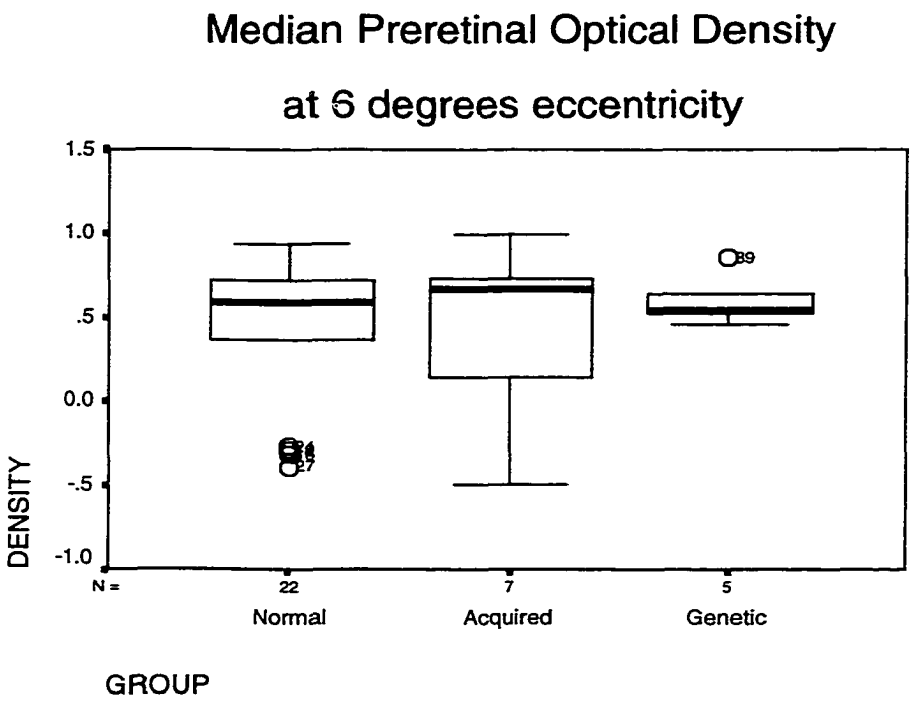


Figure B39. Box-and-whisker plot of optical density due to preretinal absorption for each group at a retinal eccentricity of 6°. As in Figure B1.

Differences found among mean optical density of macular pigment at a 2° eccentricity were even less exaggerated when comparing group medians, with genetic color-deficients now falling close to midway between the acquireds and normals (see Figure B40). Variability on this measure was greatest in the normal group and least among genetics.

In comparing group medians, the acquired group had a higher sensitivity (i.e., a lower threshold) than the genetics in the detection of a 425 nm light flashed against a 596 nm background when using increment threshold (see Figure B41). Variability between observer's thresholds was generally wide across all groups, but more so among color-normals, as this group possessed both the most and least sensitive individuals

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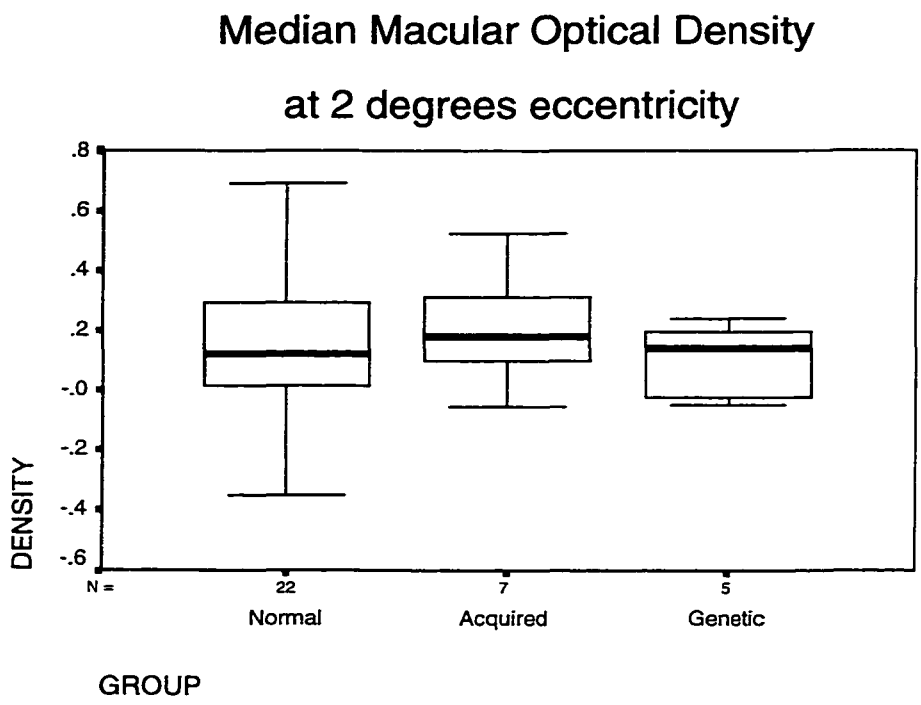


Figure B40. Box-and-whisker plot of optical density due to macular pigment for each group at a retinal eccentricity of 2°. As in Figure B1.

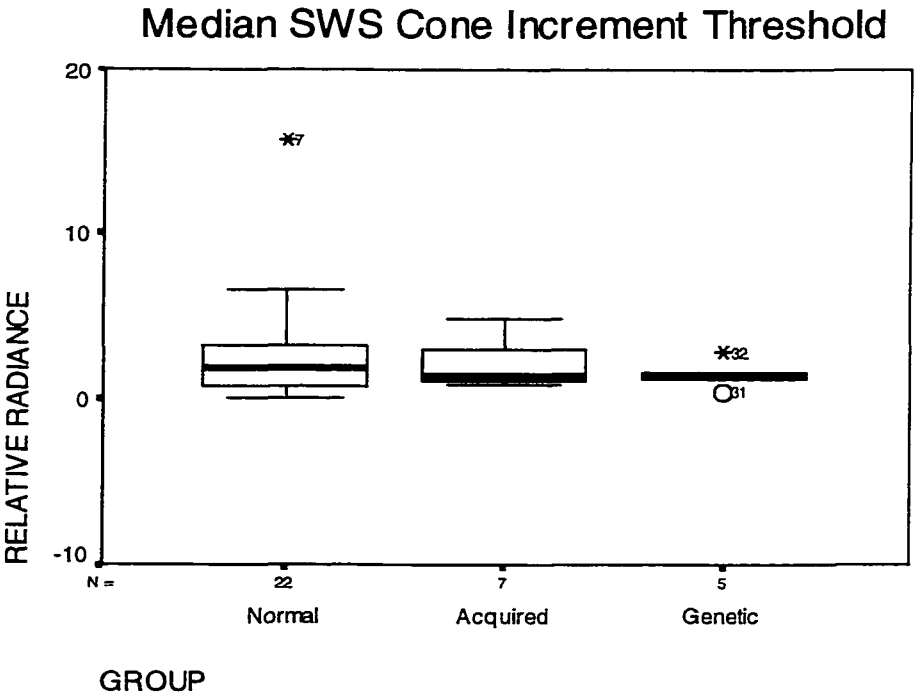


Figure B41. Box-and-whisker plot of relative radiance necessary for just noticeable detection of a flash (increment threshold) of 425 nm light on a bright 596 nm background for each group. As in Figure B1.

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across all observers combined. After correcting for the density of optical media, when examining group medians, the lower thresholds found among group means were maintained by the genetics (though less sensitive compared to the mean), while acquireds were found to be slightly more sensitive than the normals (see Figure B42). The degree of variability within groups followed this trend as well (i.e., greatest in normals, least in genetics), but the amount of overlap in the ranges of thresholds measured across the groups was rather large.

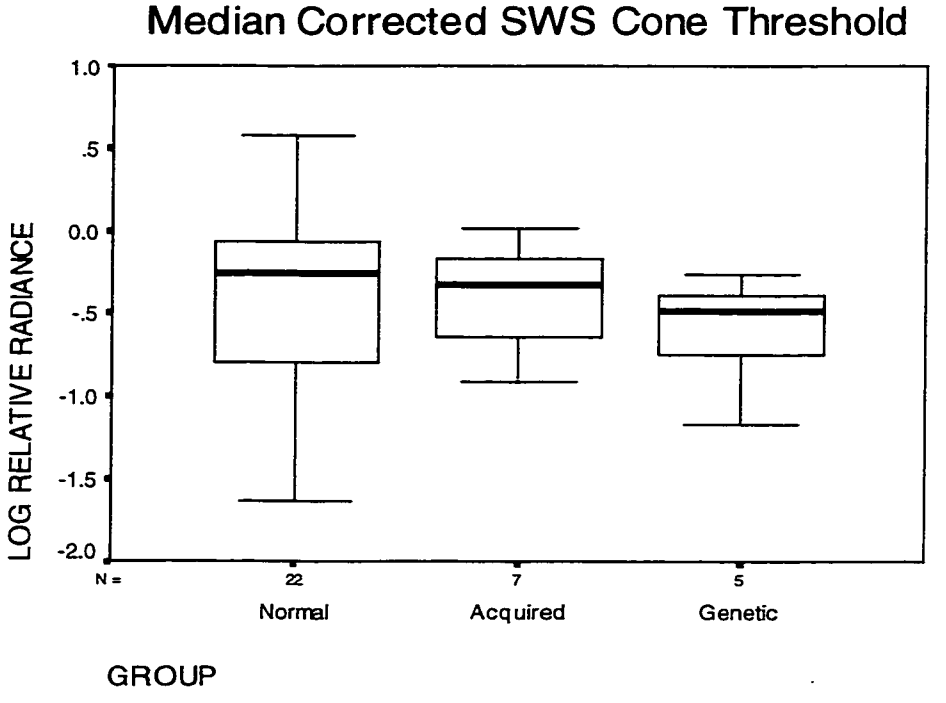


Figure B42. Box-and-whisker plot of relative radiance necessary for just noticeable detection of a flash (increment threshold) of 425 nm light on a bright 596 nm background for each group corrected for optical media density. As in Figure B1.

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The SWS cone mechanism is relatively insensitive to the wavelengths of light used in HFP and Rayleigh matching. The use of increment threshold to measure SWS cone sensitivity was intended to not only set apart those individuals with color vision impairments in this region of the spectrum, but also to verify and confirm the diagnostic impressions made earlier by our (limited) color-screening tools in this small number of participants (i.e., the acquired group). We were surprised to find that our acquired color-deficient observers had SWS cone sensitivities equal to, if not greater than, most of the color-normals.

There are several ways that this may have come about, as revealed by examining the task, the stimuli, and the procedure used. For the measurement of absolute limens, we had decided that stable thresholds would require an initial dark-adaptation period of at least 10 min. While such a time allotment is in agreement with Wald (1945; who assumed that 4 to 8 min was sufficient), it falls far short of that of Frumkes and colleagues (see, e.g., Frumkes, Sekuler, & Reiss, 1972; Frumkes & Temme, 1977; Temme & Frumkes, 1977) and Naarendorp, Rice, and Sieving (1996), who, in specifically studying rod-cone interactions, used dark-adaptations of 25-35 min. (Interestingly, the 35 min interval was used to study rod-SWS cone interactions.) As such, we may need to consider if a given threshold was the result of rods, of cones, or of an interaction between the two. At the very least, the scotopic system is not maximally sensitive until 20-30 min of dark adaptation (see, e.g., Levine & Shefner, 1991). This not only has a direct influence on absolute limens, but, consequently, will also affect the correction for SWS cone sensitivity (on which it is based).

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In order to orient the observer to the task, we presented a practice trial, which allowed the participant to test out the keypad and observe a large range of intensities available through adjustment. While we had set an upper limit for maximum intensity for the practice trial which was well below that for test trials, it could still have been sufficiently intense that, provided the practice trial was terminated at this level of intensity, the two minute period of re-dark-adaptation was too brief, thus affecting the first few thresholds. An examination of the raw data revealed that, in fact, for a number of individuals, intensities for at least the first absolute limen trial (in one or more sets) were found to be considerably higher than those required for subsequent trials. Several additional trials would permit us to reject the first one or two and hopefully alleviate this possible error of measurement, and again allow for maximal rod (scotopic) sensitivity.

With the potential for individual observers with low sensitivities, we needed to allow for a large range of intensities over which participants could adjust. Despite all trials starting at intensities below threshold, the lack of a buffer on the keypad permitted a rapid ascent to very high intensities, from which reduction in receptor sensitivity could have compromised actual thresholds from being found.

In measuring SWS cone sensitivity, we may inquire whether the intensity and wavelength of our adapting field used for increment threshold was adequate to suppress the LWS and MWS cone mechanisms, or if they "assisted" in detection. That is, by similar virtue of the argument posed for multiple long wavelength sensitive cones (see Discussion), if LWS and MWS cones are not sufficiently suppressed, we cannot be certain that what we are measuring is a purely SWS cone response. In such a case, individuals with normally functional longer wavelength sensitive cones, for example,

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may be able to give thresholds superior to those obtained when their SWS cones are fully isolated.

One pertinent inquiry is whether our assumed optical density of 0.3 for rhodopsin, used in computing the SWS cone correction, is optimal. In particular, should we necessarily assume that differences in densities from one individual to the next are minimal enough that a single value can be used for all observers? Our choice, which resulted in Wald and Brown's (1958) data most closely fitting V'-lambda, is slightly below (but in relative agreement with) that of Stiles's (1953) estimate (based on estimates obtained by different methods) of a suitable correction for a 425 nm light at a retinal eccentricity of 8° of 0.34. Other estimates have ranged from 0.23 (derived psychophysically; Hecht, Shlaer, & Pirenne, 1942) to 0.475 (obtained from microspectrophotometry; Bowmaker & Dartnall, 1980).

Perhaps the unanticipated results were not apparatus- or procedure-related, but rather simply due to individual differences between observers' visual systems. It is important to note that of all the acquired group members, only two can be called "tritanopic-like" with any objective degree of certainty. In addition, it is quite conceivable that the SWS cone sensitivities of these two individuals fall on the tail end of the distribution, overlapping with and approximating sensitivities of normal observers. Whether these two observers are, in fact, exceptions to the acquired norm can only be answered with a greater sample size. Stiles (1953), while referring in particular to foveal increment threshold data for what were most likely color-normal observers, described highly variable thresholds (in some cases of up to five log units depending on target and background wavelengths; e.g., 476 nm stimulus on 578 nm field) between subjects.

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Reminiscent of our earlier discussion regarding the findings of Roorda and Williams (1999), Stiles (1946) posited more than half a century earlier that some observers' foveas may contain rods, that their rod-free areas may be smaller than the test field or size of the test stimulus, and that while foveal fixation as normally understood may be maintained, their rod-free areas may be displaced. This may well lead to rod intrusion, especially at low intensities (Stiles, 1946; see also Frumkes, et al., 1972; Frumkes & Temme, 1977; Temme & Frumkes, 1977). Furthermore, we had not controlled for variables such as gender, tobacco use, and iris color, which are key factors, according to Hammond, et al. (1996, 1997, 1998), regarding macular pigment density, and could be important contributing factors to the observed differences in SWS cone sensitivity in a sample of our size and composition. Still, there may be some other difference, at some level, in our acquired observers, one that only a more sensitive test procedure can reveal.

Spatio-temporal contrast sensitivity. Each group's mean and median contrast sensitivities for each spatial frequency at each temporal frequency tested are presented in Table B3. Median contrast sensitivity functions for each group are presented separately, in Figures B43-B47, for each of the five temporal frequencies used in the battery. A comparison of group means versus group medians shows, in certain instances, reversals in the ordering of group sensitivities. These findings, coupled with individual differences between group members (examples of which are shown in Figures B48-B52 in the depiction of mean contrast sensitivity functions of the individual members of the genetic color-deficient group by temporal frequency), point to the possibility that some of these effects may be due to differences in sample sizes among the groups, and this should not be overlooked when reaching any general conclusions.

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Table B3. Spatio-temporal Contrast Sensitivity: Contrast Sensitivities Across the Entire Spatio-temporal Domain Tested

| Group | Normal | Acquired | Genetic |
|---|---|---|---|
| Mean Contrast Sensitivity for a 0.6 cpd grating at 1 Hz | 79.07 | 82.31 | 81.82 |
| SEM | 4.51 | 10.33 | 6.79 |
| Median | 79.40 | 80.00 | 76.30 |
| Mean Contrast Sensitivity for a 1 cpd grating at 1 Hz | 149.22 | 121.17 | 144.62 |
| SEM | 14.94 | 10.00 | 20.14 |
| Median | 120.50 | 114.90 | 156.30 |
| Mean Contrast Sensitivity for a 2 cpd grating at 1 Hz | 197.04 | 203.40 | 226.72 |
| SEM | 17.10 | 15.40 | 27.95 |
| Median | 188.70 | 192.30 | 243.90 |
| Mean Contrast Sensitivity for a 5 cpd grating at 1 Hz | 210.74 | 264.20 | 325.30 |
| SEM | 17.09 | 35.41 | 21.71 |
| Median | 175.40 | 312.50 | 344.80 |
| Mean Contrast Sensitivity for a 12 cpd grating at 1 Hz | 75.39 | 90.09 | 107.32 |
| SEM | 7.19 | 18.35 | 8.84 |
| Median | 75.20 | 92.60 | 104.20 |

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Table B3 continued

| | | | |
|---|---|---|---|
| Mean Contrast Sensitivity for a 24 cpd grating at 1 Hz | 16.26 | 16.71 | 23.76 |
| SEM | 1.99 | 4.24 | 6.47 |
| Median | 13.00 | 14.00 | 16.80 |
| Mean Contrast Sensitivity for a 0.6 cpd grating at 4 Hz | 183.27 | 249.39 | 187.60 |
| SEM | 8.83 | 49.79 | 23.84 |
| Median | 181.80 | 200.00 | 172.40 |
| Mean Contrast Sensitivity for a 1 cpd grating at 4 Hz | 266.19 | 292.27 | 257.34 |
| SEM | 16.47 | 20.59 | 30.97 |
| Median | 243.90 | 270.30 | 217.40 |
| Mean Contrast Sensitivity for 2 cpd grating at 4 Hz | 289.81 | 440.03 | 285.22 |
| SEM | 21.04 | 79.90 | 22.09 |
| Median | 243.90 | 344.80 | 285.70 |
| Mean Contrast Sensitivity for a 5 cpd grating at 4 Hz | 221.85 | 228.29 | 276.38 |
| SEM | 24.21 | 24.06 | 29.05 |
| Median | 204.10 | 243.90 | 270.30 |
| Mean Contrast Sensitivity for a 12 cpd grating at 4 Hz | 77.49 | 75.67 | 91.32 |
| SEM | 10.85 | 8.18 | 4.63 |
| Median | 64.10 | 81.30 | 90.10 |

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Table B3 continued

| | | | |
|---|---|---|---|
| Mean Contrast Sensitivity for a 24 cpd grating at 4 Hz | 15.00 | 13.76 | 17.36 |
| SEM | 1.82 | 2.01 | 2.39 |
| Median | 12.60 | 14.70 | 19.20 |
| Mean Contrast Sensitivity for a 0.6 cpd grating at 8 Hz | 205.53 | 198.34 | 249.50 |
| SEM | 13.60 | 16.75 | 31.55 |
| Median | 196.10 | 212.80 | 232.60 |
| Mean Contrast Sensitivity for a 1 cpd grating at 8 Hz | 272.40 | 291.71 | 281.16 |
| SEM | 14.93 | 26.36 | 21.01 |
| Median | 256.40 | 303.00 | 270.30 |
| Mean Contrast Sensitivity for a 2 cpd grating at 8 Hz | 285.10 | 288.59 | 308.36 |
| SEM | 25.81 | 36.24 | 33.75 |
| Median | 243.90 | 243.90 | 285.70 |
| Mean Contrast Sensitivity for a 5 cpd grating at 8 Hz | 172.82 | 170.09 | 182.30 |
| SEM | 25.71 | 30.13 | 8.69 |
| Median | 142.90 | 161.30 | 192.30 |
| Mean Contrast Sensitivity for a 12 cpd grating at 8 Hz | 52.24 | 64.14 | 87.00 |
| SEM | 5.38 | 17.72 | 29.29 |
| Median | 56.50 | 45.20 | 59.90 |

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Table B3 continued

| | | | |
|---|---|---|---|
| Mean Contrast Sensitivity for a 24 cpd grating at 8 Hz | 12.08 | 11.74 | 15.20 |
| SEM | 1.44 | 2.06 | 1.72 |
| Median | 11.30 | 11.90 | 14.90 |
| Mean Contrast Sensitivity for a 0.6 cpd grating at 15 Hz | 115.34 | 101.09 | 133.56 |
| SEM | 4.42 | 9.99 | 18.84 |
| Median | 113.60 | 101.00 | 128.20 |
| Mean Contrast Sensitivity for a 1 cpd grating at 15 Hz | 141.00 | 155.57 | 172.60 |
| SEM | 6.40 | 28.93 | 12.21 |
| Median | 135.10 | 156.30 | 181.80 |
| Mean Contrast Sensitivity for a 2 cpd grating at 15 Hz | 133.73 | 206.20 | 147.26 |
| SEM | 7.25 | 34.85 | 7.28 |
| Median | 133.30 | 212.80 | 153.80 |
| Mean Contrast Sensitivity for a 5 cpd grating at 15 Hz | 70.40 | 108.43 | 85.68 |
| SEM | 4.93 | 24.42 | 14.99 |
| Median | 68.00 | 80.6 | 74.6 |
| Mean Contrast Sensitivity for a 12 cpd grating at 15 Hz | 25.03 | 34.03 | 31.66 |
| SEM | 2.60 | 5.76 | 5.18 |
| Median | 21.50 | 34.00 | 33.60 |

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Table B3 continued

| | | | |
|---|---|---|---|
| Mean Contrast Sensitivity for a 24 cpd grating at 15 Hz | 6.56 | 5.01 | 8.54 |
| SEM | 0.83 | 0.80 | 1.98 |
| Median | 6.10 | 4.60 | 7.20 |
| Mean Contrast Sensitivity for a 0.6 cpd grating at 24 Hz | 44.03 | 46.76 | 48.10 |
| SEM | 2.48 | 7.18 | 5.39 |
| Median | 43.70 | 47.40 | 45.90 |
| Mean Contrast Sensitivity for a 1 cpd grating at 24 Hz | 53.11 | 57.76 | 51.78 |
| SEM | 4.45 | 12.71 | 3.33 |
| Median | 50.50 | 54.30 | 52.60 |
| Mean Contrast Sensitivity for a 2 cpd grating at 24 Hz | 44.12 | 52.21 | 67.32 |
| SEM | 2.57 | 9.53 | 14.80 |
| Median | 43.30 | 48.30 | 56.50 |
| Mean Contrast Sensitivity for a 5 cpd grating at 24 Hz | 28.80 | 23.16 | 39.56 |
| SEM | 2.69 | 3.50 | 3.07 |
| Median | 28.00 | 21.3 | 40.2 |
| Mean Contrast Sensitivity for a 15 cpd grating at 24 Hz | 12.26 | 12.69 | 16.28 |
| SEM | 1.49 | 2.84 | 2.61 |
| Median | 11.80 | 7.70 | 14.00 |

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Table B3 continued

| | | | |
|---|---|---|---|
| Mean Contrast Sensitivity for a 24 cpd grating at 24 Hz | 2.96 | 3.36 | 4.04 |
| SEM | 0.34 | 0.57 | 0.56 |
| Median | 2.80 | 3.00 | 3.80 |

Table B4 presents the mean maximum (peak) contrast sensitivity for each group of observers. The ratios of sensitivities at lowest spatial frequency compared to peak contrast sensitivity ("low-to-peak") are given in Table B5. The mean spatial frequency at which peak sensitivity occurred for each of the groups is shown in Table B6. (The measure of mean contrast sensitivity at the lowest spatial frequency, along with which the above variables' results are described, can be found in Table B3.)

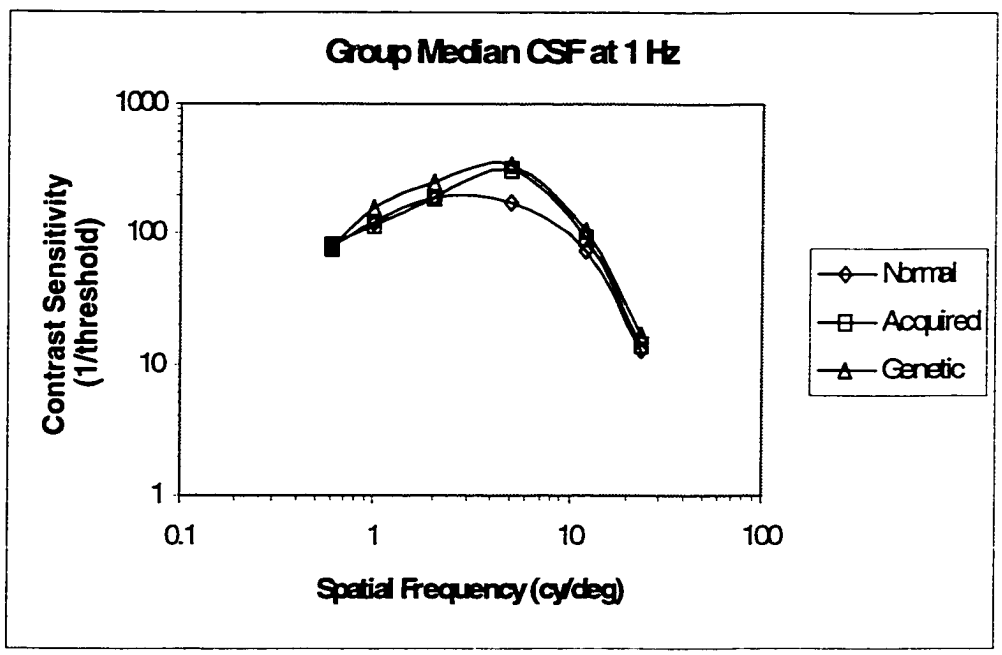


Figure B43. Median contrast sensitivity function for each group at 1 Hz for the entire spatial domain tested.

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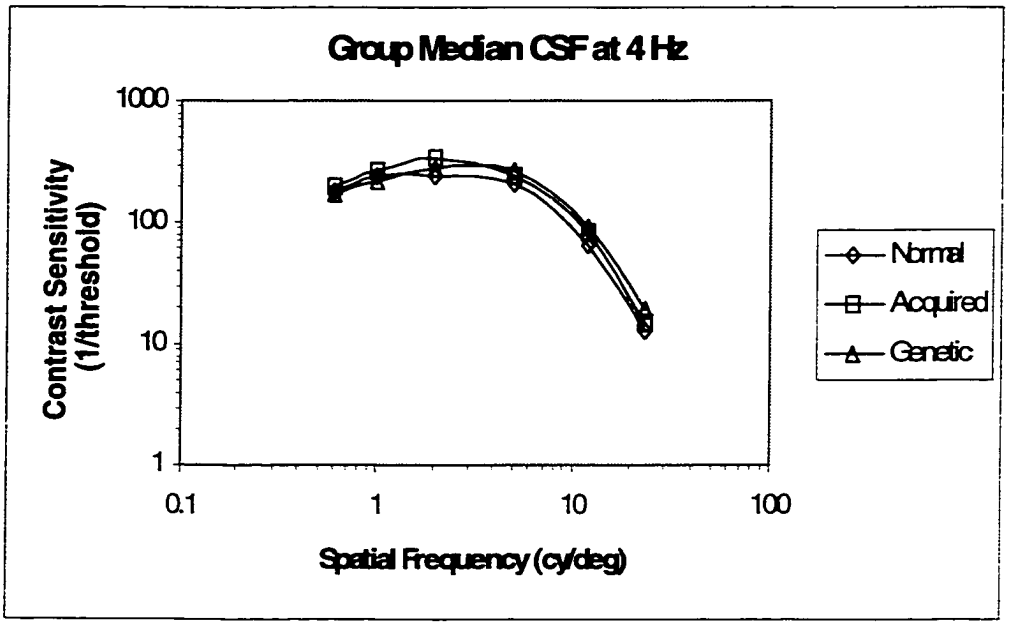


Figure B44. Median contrast sensitivity function for each group at 4 Hz for the entire spatial domain tested.

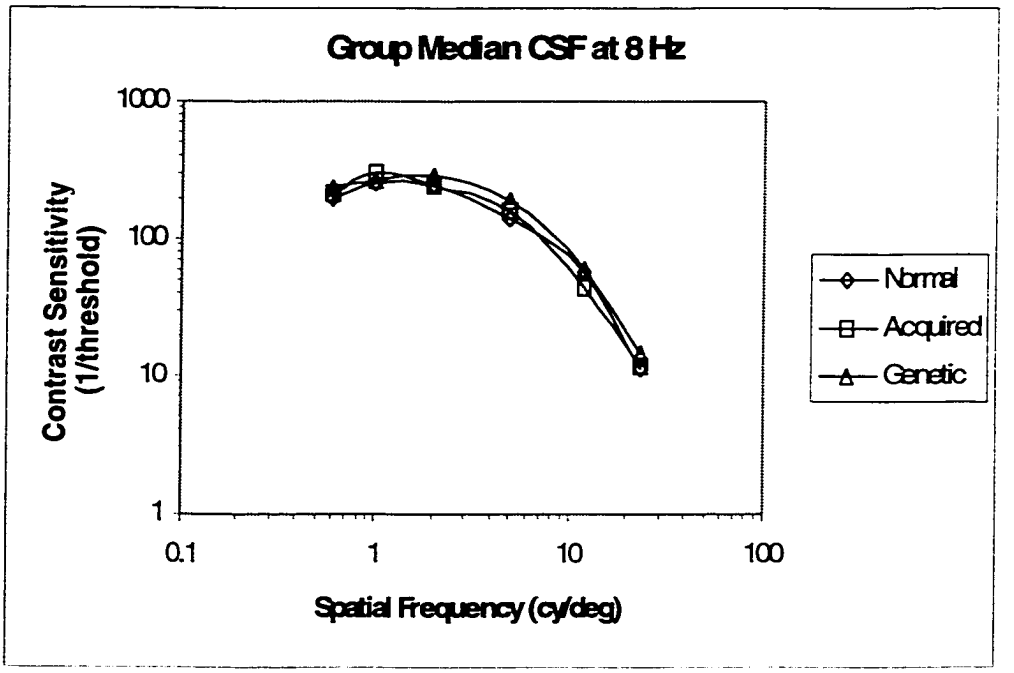


Figure B45. Median contrast sensitivity function for each group at 8 Hz for the entire spatial domain tested.

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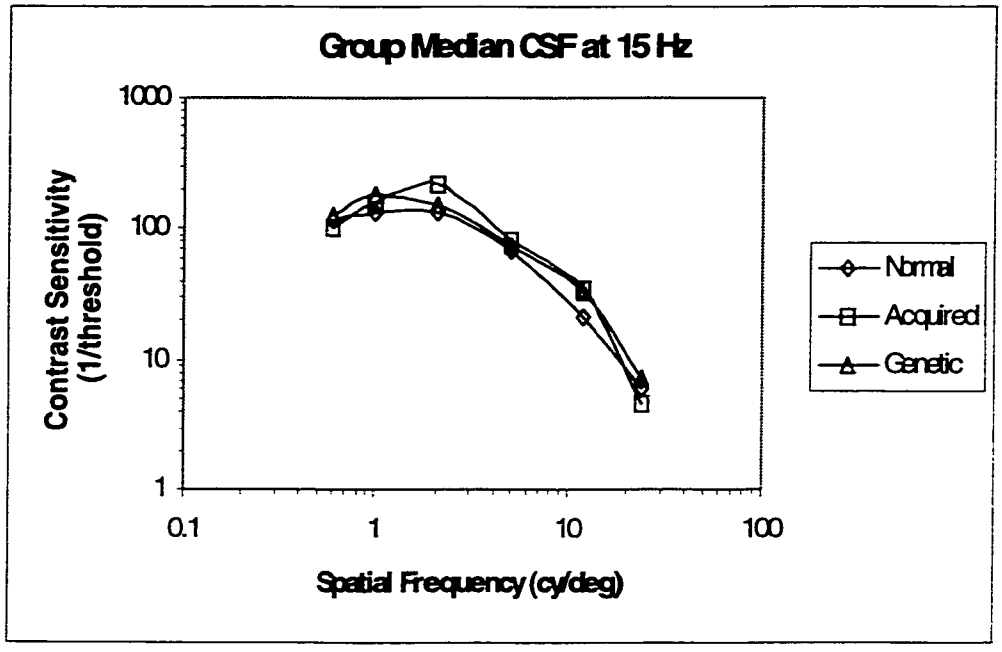


Figure B46. Median contrast sensitivity function for each group at 15 Hz for the entire spatial domain tested.

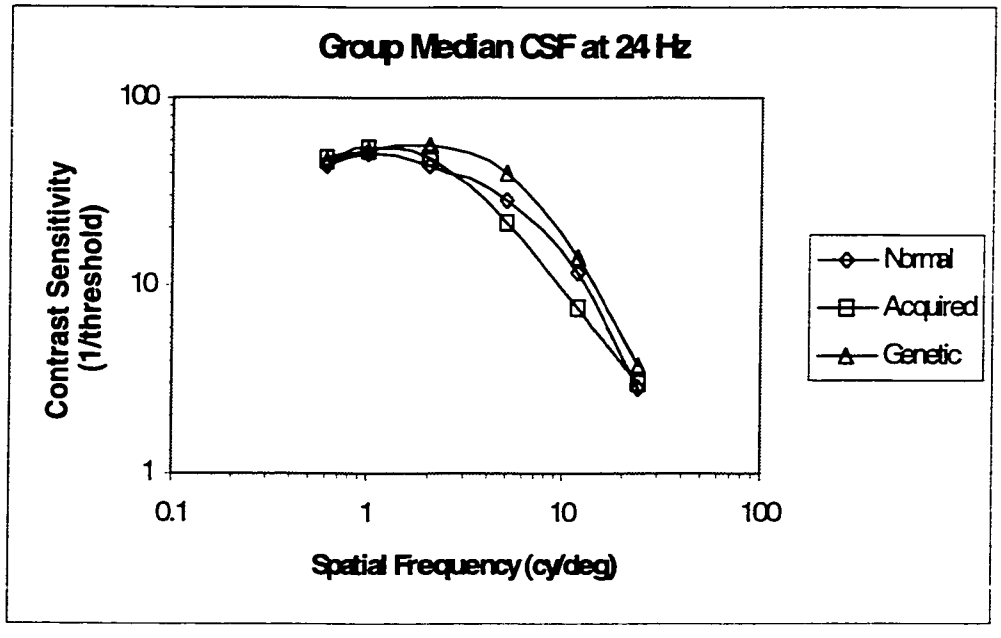


Figure B47. Median contrast sensitivity function for each group at 24 Hz for the entire spatial domain tested.

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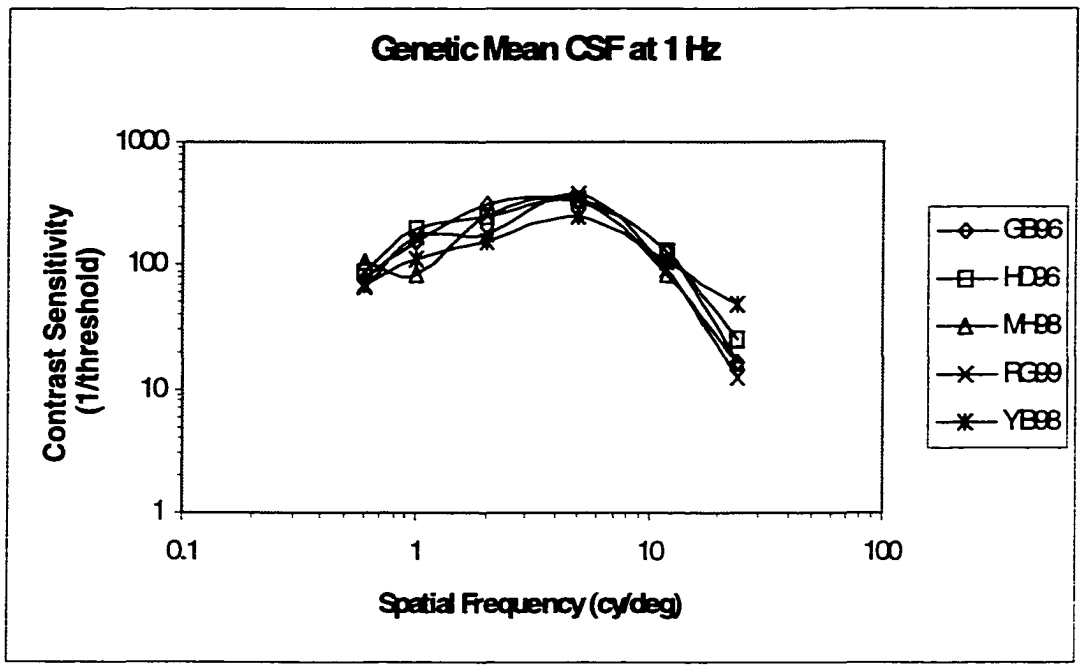


Figure B48. Mean contrast sensitivity function for each genetic color-deficient observer at 1 Hz for the entire spatial domain tested.

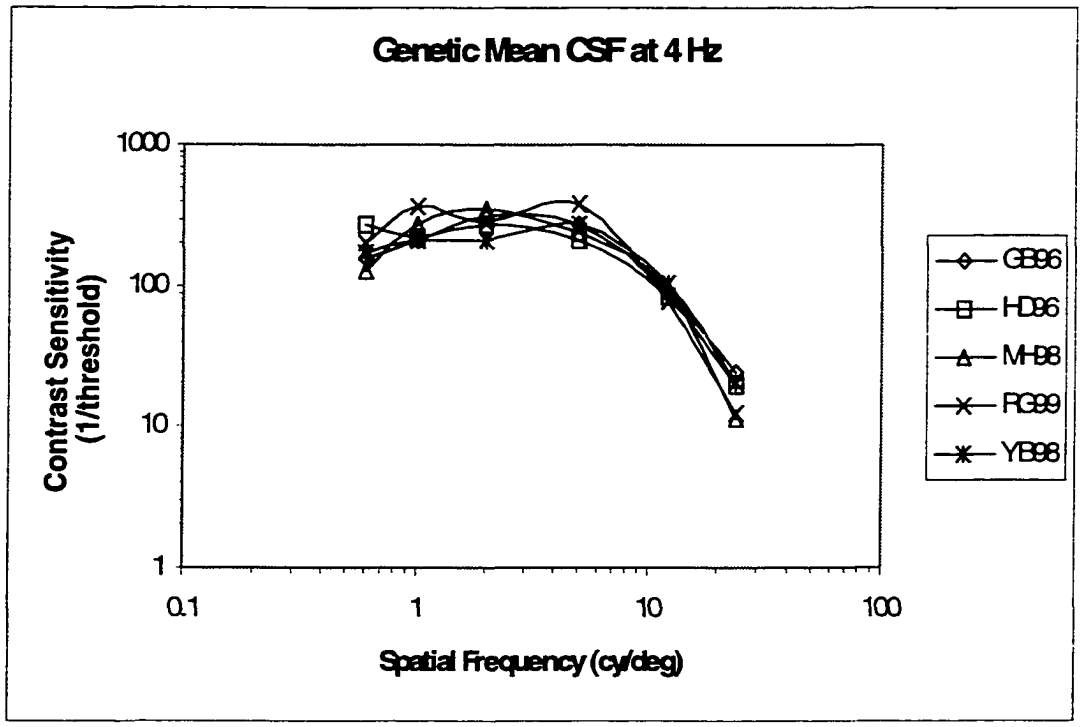


Figure B49. Mean contrast sensitivity function for each genetic color-deficient observer at 4 Hz for the entire spatial domain tested.

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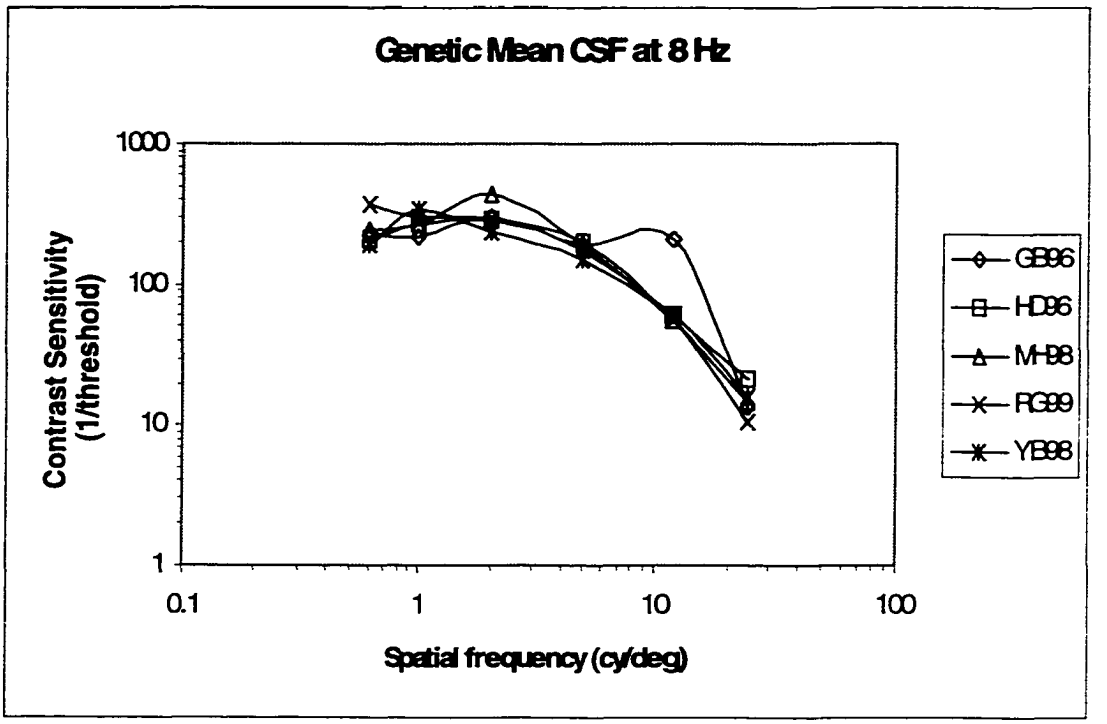


Figure B50. Mean contrast sensitivity function for each genetic color-deficient observer at 8 Hz for the entire spatial domain tested.

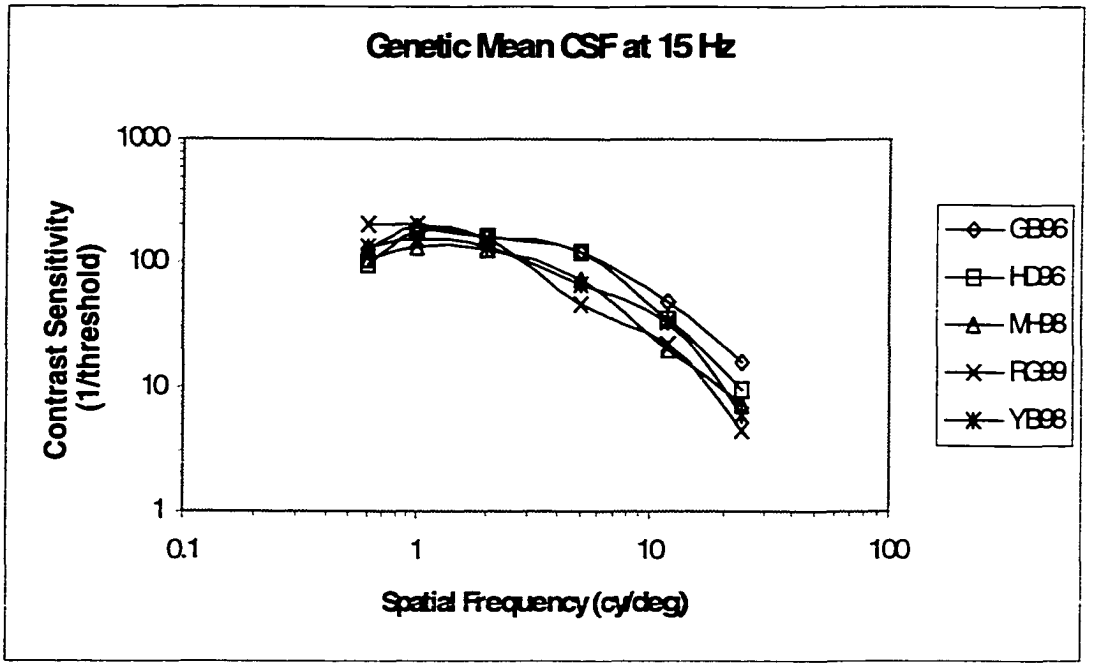


Figure B51. Mean contrast sensitivity function for each genetic color-deficient observer at 15 Hz for the entire spatial domain tested.

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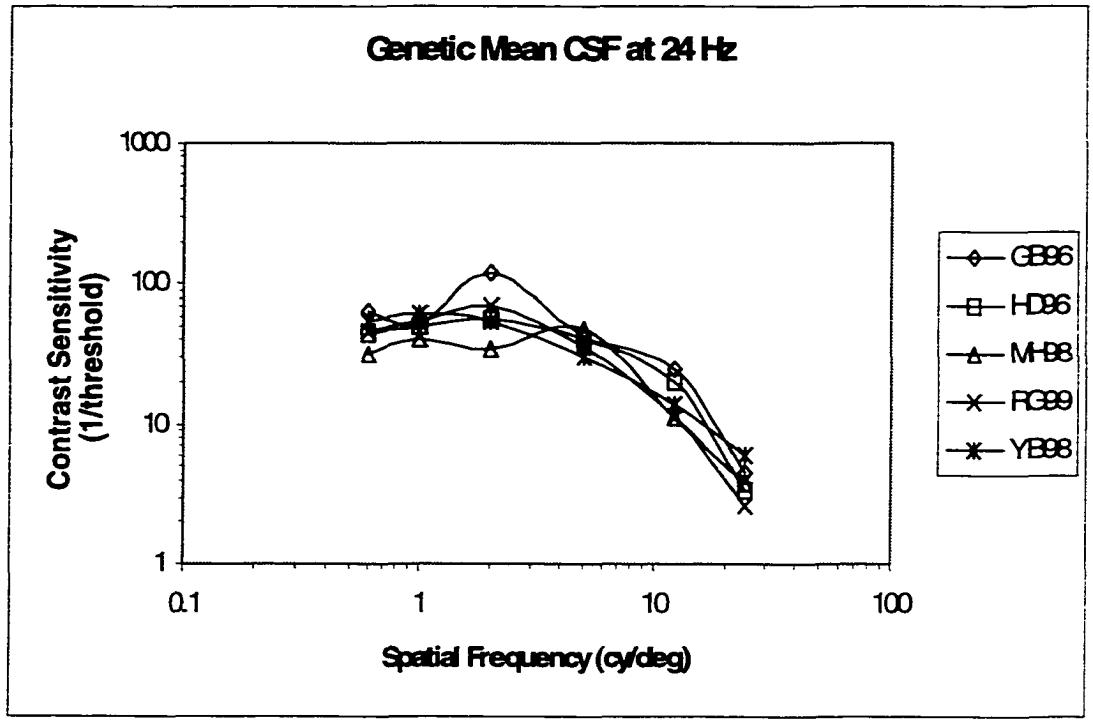


Figure B52. Mean contrast sensitivity function for each genetic color-deficient observer at 24 Hz for the entire spatial domain tested.

The mean contrast sensitivity occurring at a spatial frequency of 0.6 cpd for a sinusoidal grating modulated at 1 Hz was comparable across the three groups of observers, with acquireds ranking slightly above genetics, who, in turn, were somewhat higher than normals. The difference between color-normals and acquired color-deficients was virtually non-existent at the level of group medians, whereas genetic color-deficients were found to be a bit less sensitive in comparison (see Table B3 and Figures B53 and B54). Each group exhibited some variability in sensitivities among individual observers (especially the normal and acquired groups), and there was much overlap in the range of observed sensitivities across all groups. The mean maximum contrast sensitivity for a 1 Hz stimulus was greatest for the genetics, less for the acquireds, and least for the

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Table B4. Spatio-temporal Contrast Sensitivity: Maximum Contrast Sensitivities at each Temporal Frequency

| Group | Normal | Acquired | Genetic |
|---|---|---|---|
| Mean Peak Contrast Sensitivity at 1 Hz | 228.40 | 289.55 | 341.18 |
| SEM | 22.28 | 30.56 | 25.36 |
| Median | 230.56 | 317.19 | 356.83 |
| Mean Peak Contrast Sensitivity at 4 Hz | 328.49 | 456.60 | 324.41 |
| SEM | 25.12 | 74.47 | 23.23 |
| Median | 267.62 | 385.98 | 330.39 |
| Mean Peak Contrast Sensitivity at 8 Hz | 339.51 | 327.96 | 349.76 |
| SEM | 29.62 | 31.42 | 26.90 |
| Median | 270.70 | 359.73 | 346.93 |
| Mean Peak Contrast Sensitivity at 15 Hz | 159.91 | 221.87 | 178.29 |
| SEM | 6.39 | 37.91 | 12.87 |
| Median | 152.76 | 229.51 | 191.71 |
| Mean Peak Contrast Sensitivity at 24 Hz | 58.37 | 65.99 | 72.10 |
| SEM | 4.16 | 12.67 | 13.10 |
| Median | 54.02 | 68.02 | 60.71 |

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Table B5. Spatio-temporal Contrast Sensitivity: Ratios of Sensitivities at 0.6 cpd to Maximum Contrast for each Temporal Frequency

| Group | Normal | Acquired | Genetic |
|---|---|---|---|
| Mean ratio of low-to-peak sensitivity at 1 Hz | 0.36 | 0.30 | 0.24 |
| SEM | 0.03 | 0.04 | 0.02 |
| Median | 0.40 | 0.33 | 0.26 |
| Mean ratio of low-to-peak sensitivity at 4 Hz | 0.05 | 0.08 | 0.12 |
| SEM | 0.01 | 0.01 | 0.00 |
| Median | 0.05 | 0.08 | 0.11 |
| Mean ratio of low-to-peak sensitivity at 8 Hz | 0.67 | 0.62 | 0.72 |
| SEM | 0.04 | 0.03 | 0.08 |
| Median | 0.67 | 0.61 | 0.73 |
| Mean ratio of low-to-peak sensitivity at 15 Hz | 0.75 | 0.53 | 0.76 |
| SEM | 0.03 | 0.08 | 0.08 |
| Median | 0.73 | 0.54 | 0.79 |
| Mean ratio of low-to-peak sensitivity at 24 Hz | 0.79 | 0.75 | 0.70 |
| SEM | 0.03 | 0.06 | 0.06 |
| Median | 0.81 | 0.74 | 0.66 |

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Table B6. Spatio-temporal Contrast Sensitivity: Spatial Frequencies at Maximum Contrast for each Temporal Frequency

| Group | Normal | Acquired | Genetic |
|---|---|---|---|
| Mean Spatial Frequency of Peak Contrast Sensitivity at 1 Hz | 3.48 | 4.09 | 4.38 |
| SEM | 0.35 | 0.40 | 0.38 |
| Median | 3.23 | 3.73 | 4.98 |
| Mean Spatial Frequency of Peak Contrast Sensitivity at 4 Hz | 1.93 | 1.71 | 3.13 |
| SEM | 0.18 | 0.25 | 0.51 |
| Median | 1.57 | 2.09 | 2.79 |
| Mean Spatial Frequency of Peak Contrast Sensitivity at 8 Hz | 1.69 | 1.50 | 1.47 |
| SEM | 0.21 | 0.16 | 0.30 |
| Median | 1.57 | 1.36 | 1.57 |
| Mean Spatial Frequency of Peak Contrast Sensitivity at 15 Hz | 1.43 | 2.09 | 1.05 |
| SEM | 0.11 | 0.25 | 0.12 |
| Median | 1.36 | 2.09 | 1.17 |
| Mean Spatial Frequency of Peak Contrast Sensitivity at 24 Hz | 1.62 | 1.47 | 2.24 |
| SEM | 0.24 | 0.21 | 0.54 |
| Median | 1.02 | 1.57 | 1.81 |

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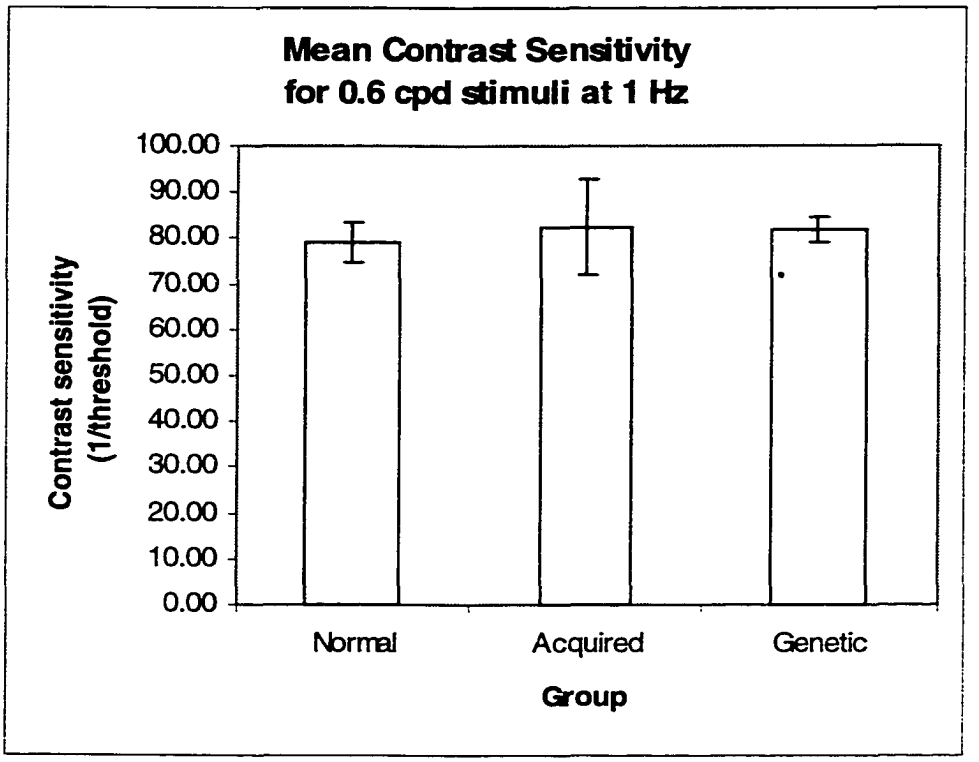


Figure B53. Mean contrast sensitivity for a 0.6 cpd sinusoidal grating modulated at a frequency of 1 Hz for each group. Error bars equal +/- 1 SEM.

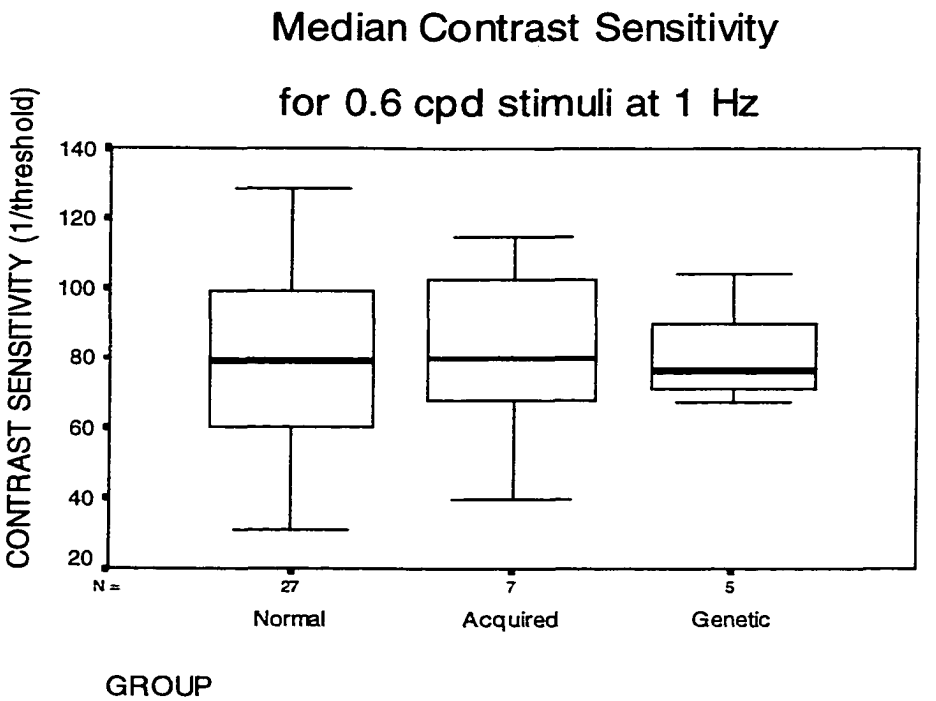


Figure B54. Box-and-whisker plot of contrast sensitivity for a 0.6 cpd sinusoidal grating modulated at a frequency of 1 Hz for each group. As in Figure B1.

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normals. This trend was maintained at the level of group medians (see Table B4 and Figures B55 and B56). Individual genetic group members were quite consistent with one another regarding their maximum sensitivity (less one observer), and the overlap in sensitivities with the other groups' was limited to the top of the normal and acquired's range. The generated mean low-to-peak ratios at this frequency was highest for the color-normal group and lowest for the genetic color-deficient group. Nearly equal differences can be seen among the groups' medians (see Table B5 and Figures B57 and B58). It should be noted, however, that the interobserver variability within the normal group was extensive, the range of sensitivities encompassing that of both the acquired and genetic groups'. The spatial frequency at which the aforementioned maximum sensitivity occurred was, on average, highest for the genetic color-deficients and lowest for the color-normals. These observed differences remained when comparing group medians (see Table B6 and Figures B59 and B60). Variability was, again, large among the normal observers' sensitivities, and all groups' ranges overlapped with one another. One-way ANOVAs, run separately for each variable, failed to be significant. Following a ranking of the group data, a Kruskal-Wallis test was applied. For a significance level of $\alpha = 0.5$, the resulting values of $H$ were also not found to be statistically significant for any of the measures, although maximum contrast sensitivity approached significance ($p = 0.071$).

At a temporal frequency of 4 Hz, the acquired color-deficient group showed a superior mean contrast sensitivity at 0.6 cpd compared to that of the genetic color-deficient and color-normal groups (who were found to be quite similar). This trend was maintained among group medians, although the normals now eclipsed the genetics with slightly higher sensitivities (see Table B3 and Figures B61 and B62). The acquired group

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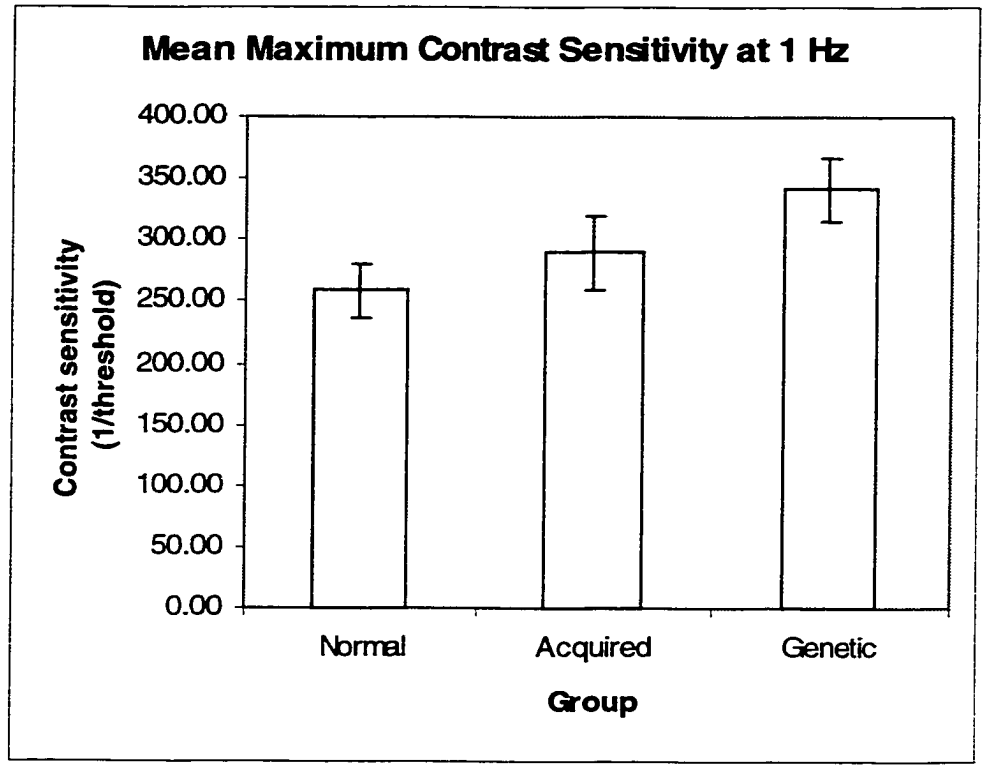


Figure B55. Mean maximum contrast sensitivity for a sinusoidal grating modulated at a frequency of 1 Hz for each group. Error bars equal +/- 1 SEM.

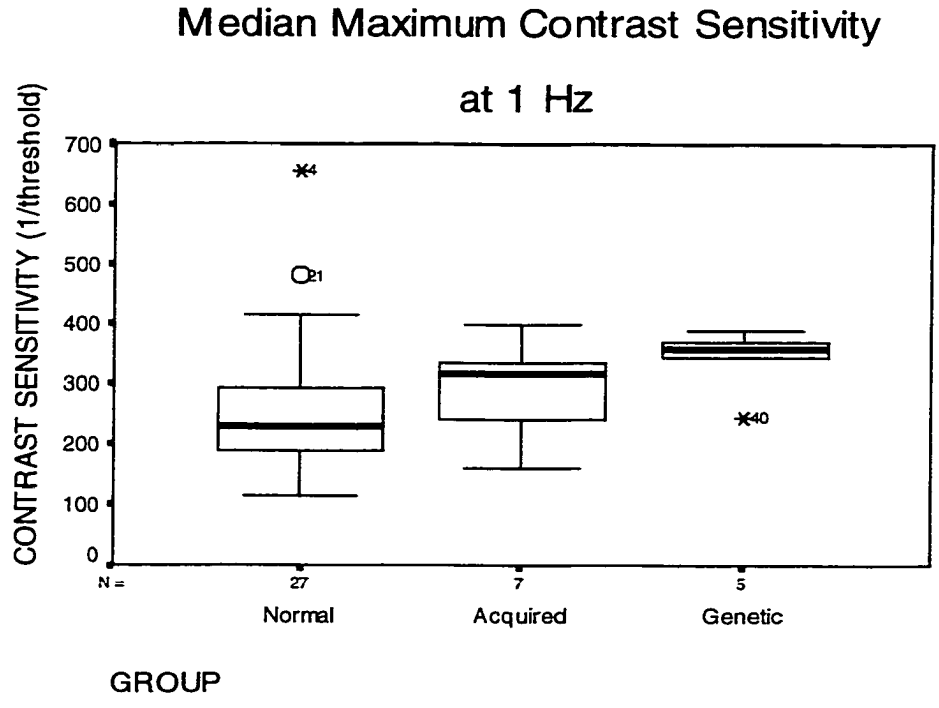


Figure B56. Box-and-whisker plot of maximum contrast sensitivity for a sinusoidal grating modulated at a frequency of 1 Hz for each group. As in Figure B1.

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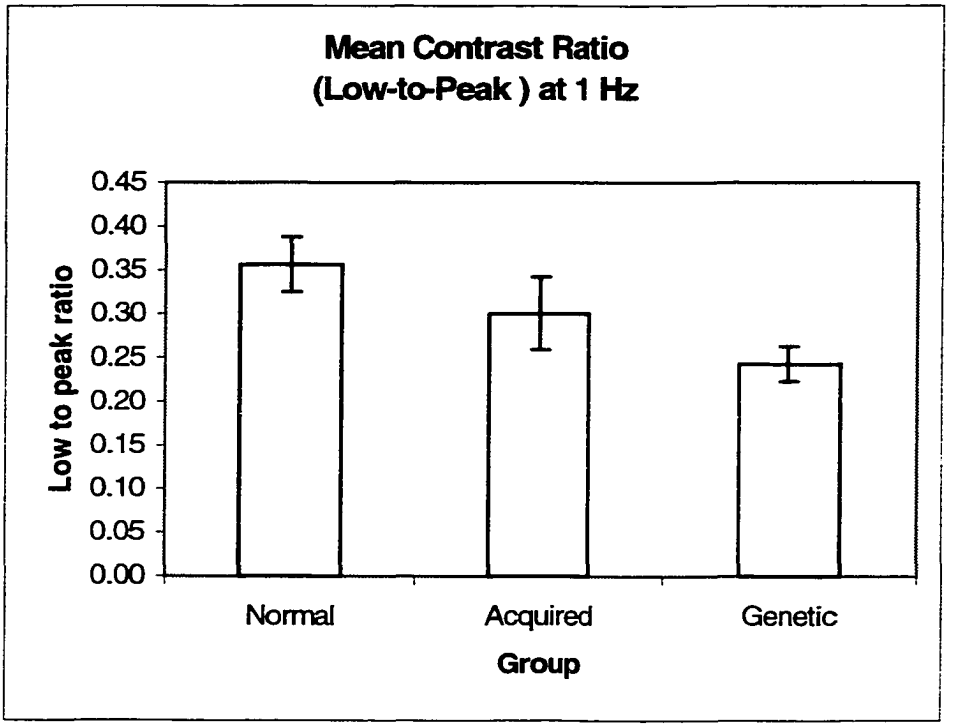


Figure B57. Mean low-to-peak contrast sensitivity ratio (sensitivity at 0.6 cpd/maximum sensitivity) at 1 Hz for each group. Error bars equal +/- 1 SEM.

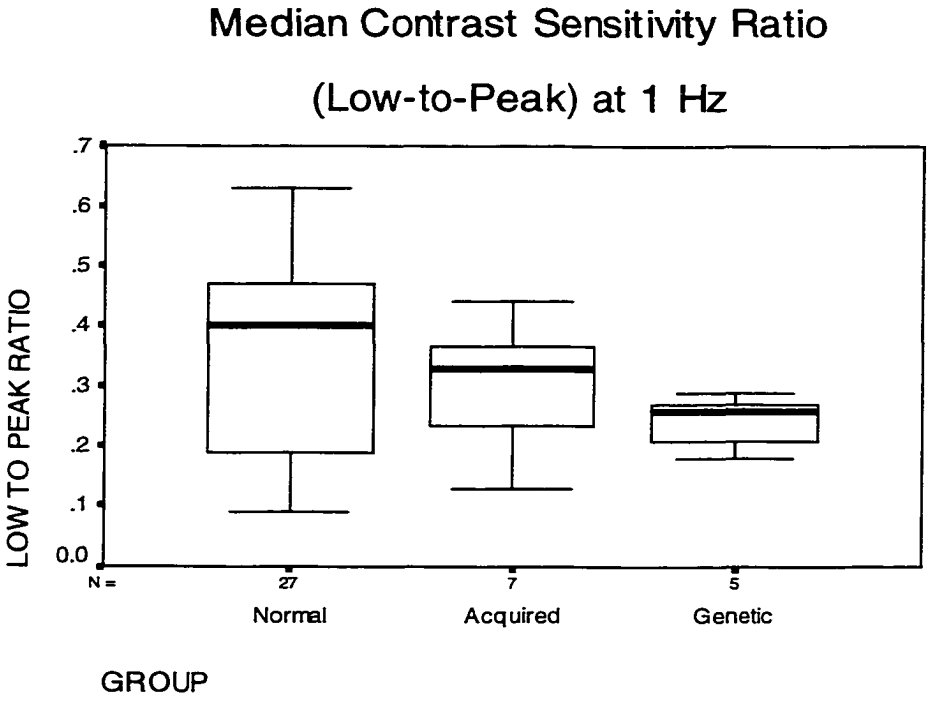


Figure B58. Box-and-whisker plot of contrast sensitivity ratio (sensitivity at 0.6 cpd/maximum sensitivity) at 1 Hz for each group. As in Figure B1.

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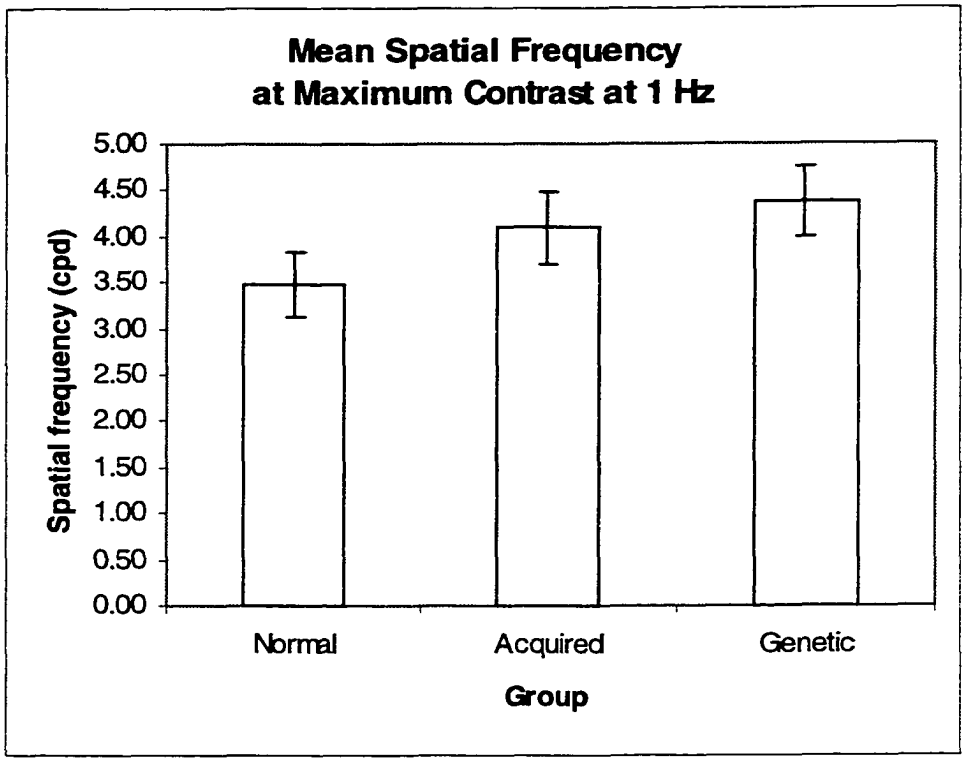


Figure B59. Mean spatial frequency at which maximum contrast sensitivity occurred at 1 Hz for each group. Error bars equal +/- 1 SEM.

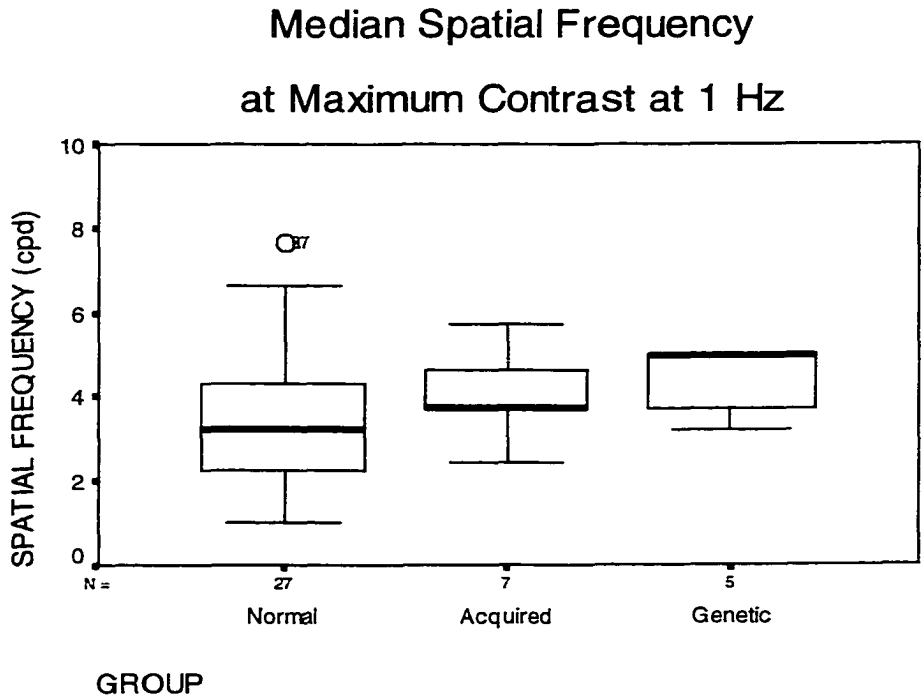


Figure B60. Box-and-whisker plot of spatial frequency at which maximum contrast sensitivity occurred at 1 Hz for each group. As in Figure B1.

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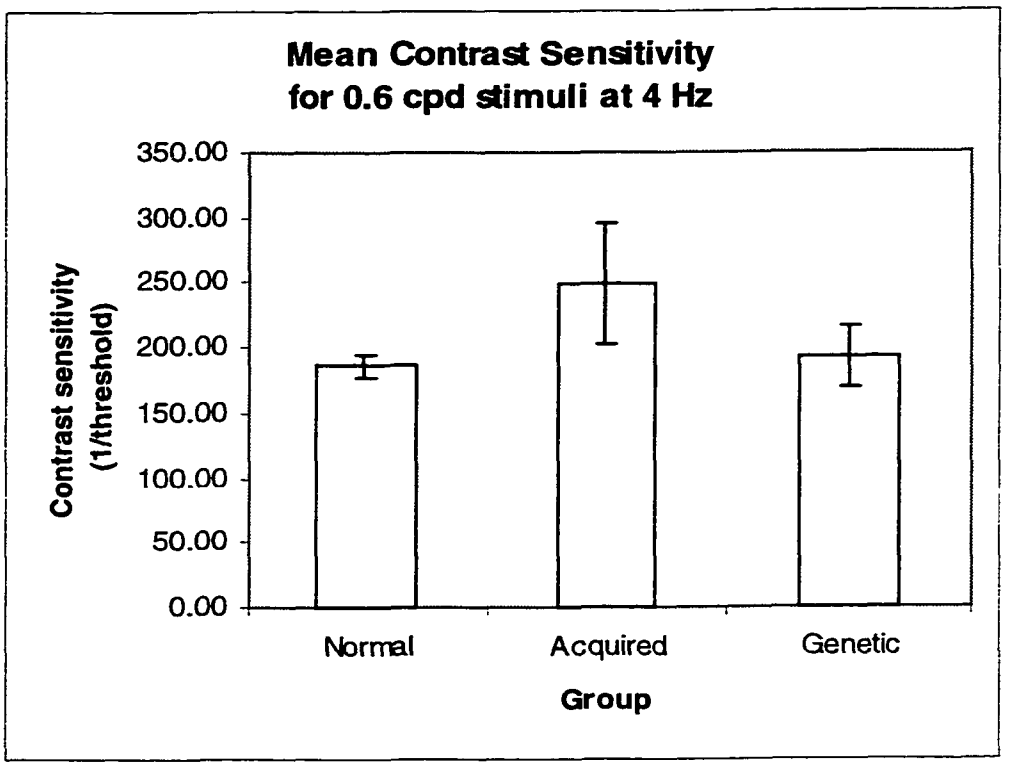


Figure B61. Mean contrast sensitivity for a 0.6 cpd sinusoidal grating modulated at a frequency of 4 Hz for each group. Error bars equal +/- 1 SEM.

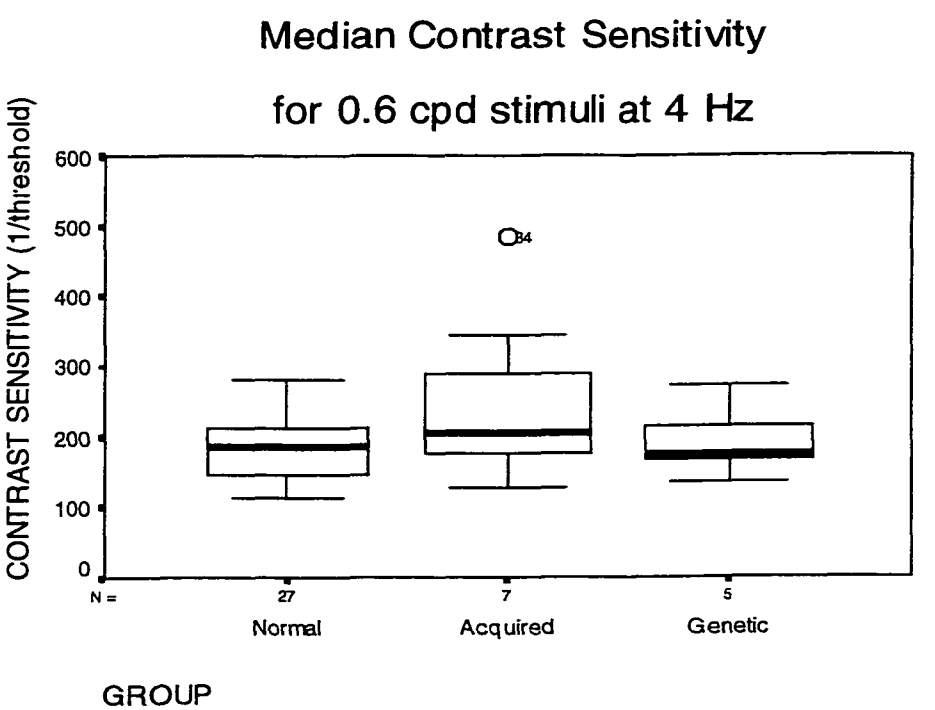


Figure B62. Box-and-whisker plot of contrast sensitivity for a 0.6 cpd sinusoidal grating modulated at a frequency of 4 Hz for each group. As in Figure B1.

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showed the most interobserver variability, and the ranges of all three groups were found to overlap. Maximally at 4 Hz, the acquired group's mean sensitivity was also found to be well above that of either the normal's or genetic's (which were, again, relatively comparable). Group differences decreased in extent when medians were considered, the genetic group also now showing higher sensitivities than the normals (see Table B4 and Figures B63 and B64). There was a marked degree of variability among acquired group members, although the highest and lowest recorded sensitivities in this group were still above those of the other groups'. Overlap among sensitivities across groups was moderate. The resulting mean sensitivity ratios were highest for the genetics and lowest for the normals (by more than a factor of 2), as were the medians (see Table B5 and Figures B65 and B66). The range of sensitivities was much larger for the normal and acquired groups, with relatively little overlap with that of the genetics. The peak sensitivity (see Table B6 and Figures B67 and B68) coincided with a higher mean spatial frequency for genetic color-deficients, compared to color-normals and acquired color-deficients. The median spatial frequency of the acquired group surpassed that of the normal group. The degree of variability of sensitivities among group members was large for genetics and normals. While the range of sensitivities for each group did overlap with one another, the upper 75% of genetic observers' sensitivities coincided only with the top 25% of the normals' (and none of the acquireds'). A one-way ANOVA revealed significant differences between groups on the measures of low-to-peak sensitivity ratios, $F(2, 36) = 11.18$, $p < 0.01$, and spatial frequency at maximum contrast sensitivity, $F(2, 36) = 4.12$, $p < 0.05$. Both sensitivity at the low spatial frequency and maximum contrast sensitivity approached significance ($p = 0.097$ and $p = 0.096$, respectively).

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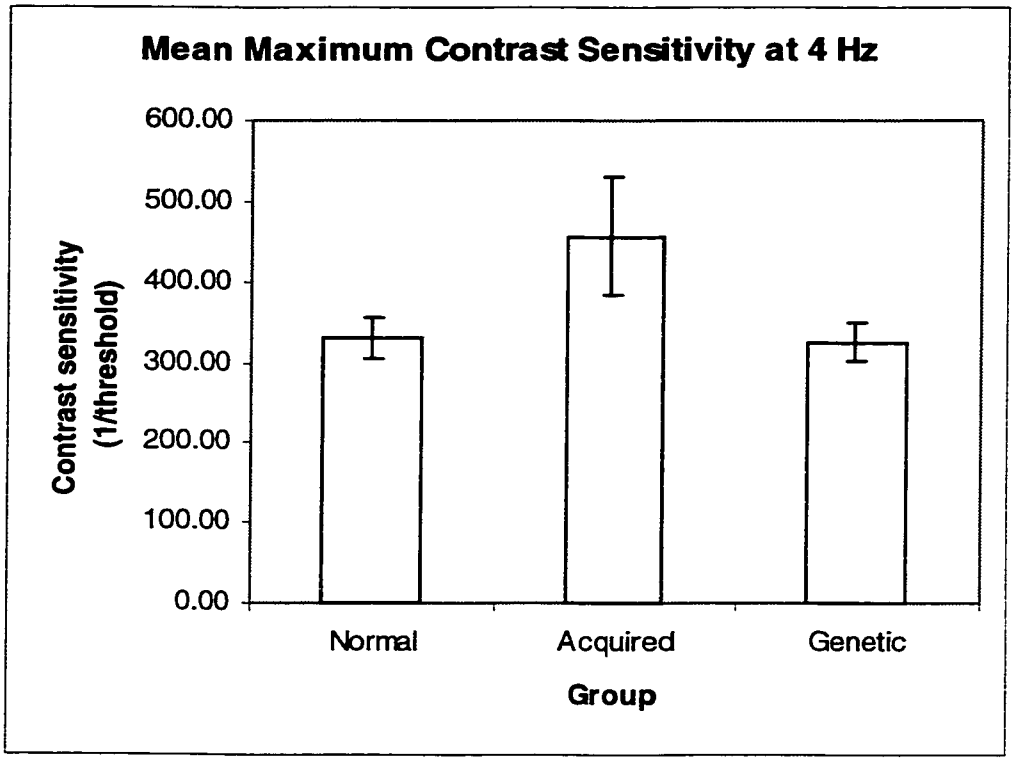


Figure B63. Mean maximum contrast sensitivity for a sinusoidal grating modulated at a frequency of 4 Hz for each group. Error bars equal +/- 1 SEM.

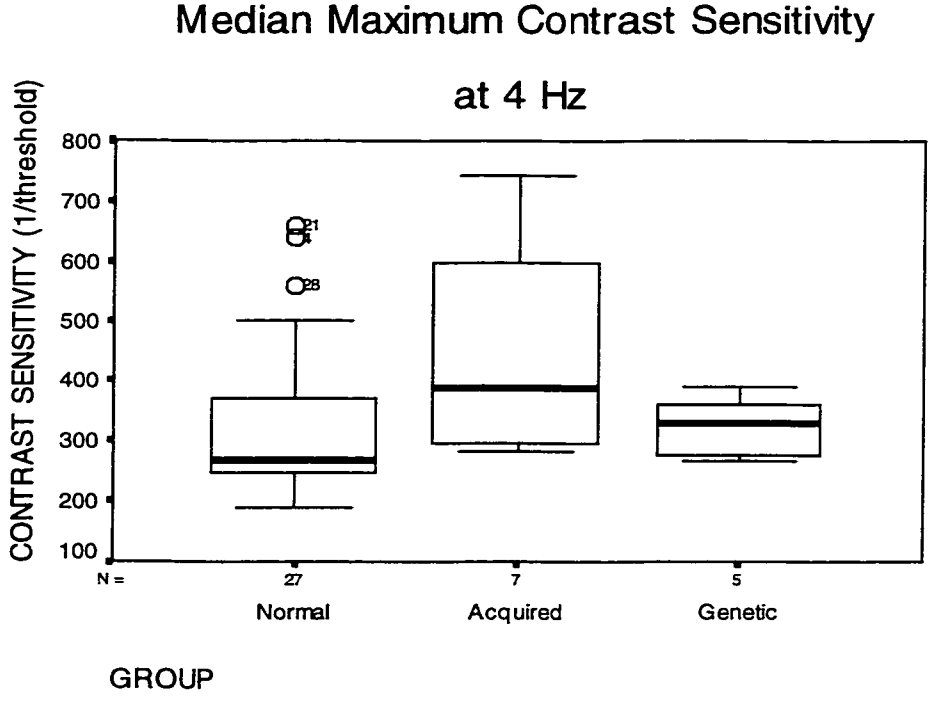


Figure B64. Box-and-whisker plot of maximum contrast sensitivity for a sinusoidal grating modulated at a frequency of 4 Hz for each group. As in Figure B1.

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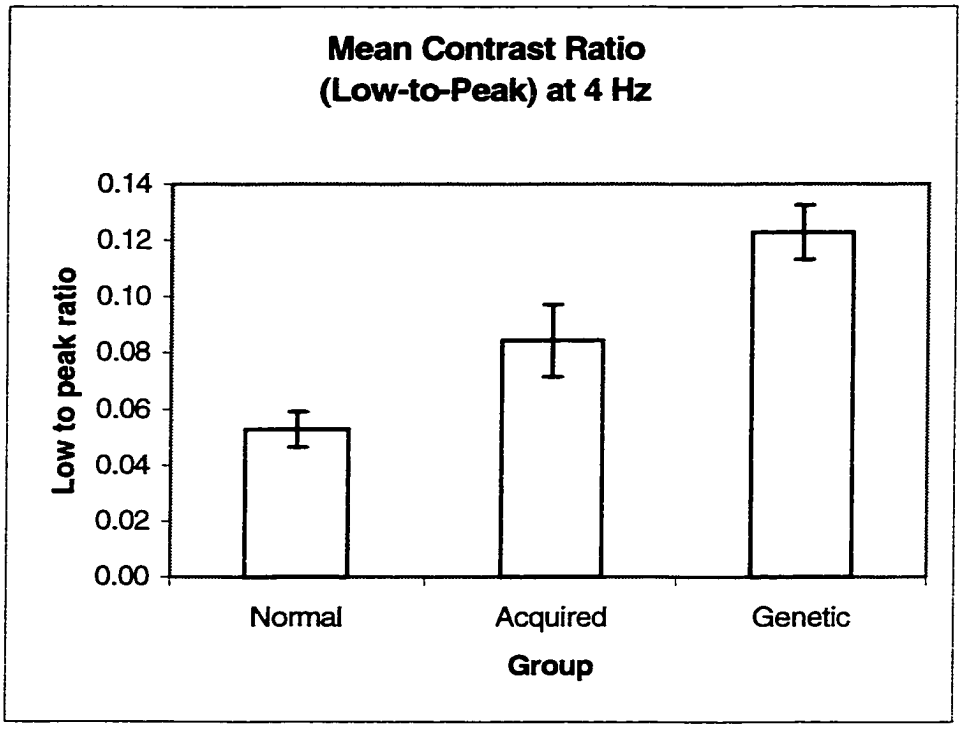


Figure B65. Mean low-to-peak contrast sensitivity ratio (sensitivity at 0.6 cpd/maximum sensitivity) at 4 Hz for each group. Error bars equal +/- 1 SEM.

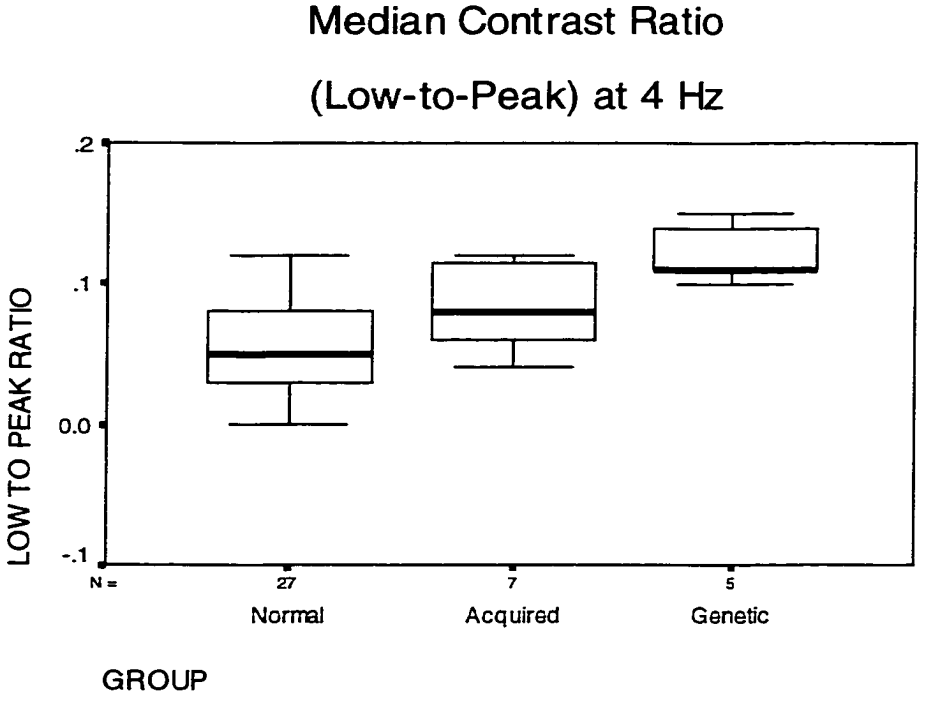


Figure B66. Box-and-whisker plot of low-to-peak contrast sensitivity ratio (sensitivity at 0.6 cpd/maximum sensitivity) at 4 Hz for each group. As in Figure B1.

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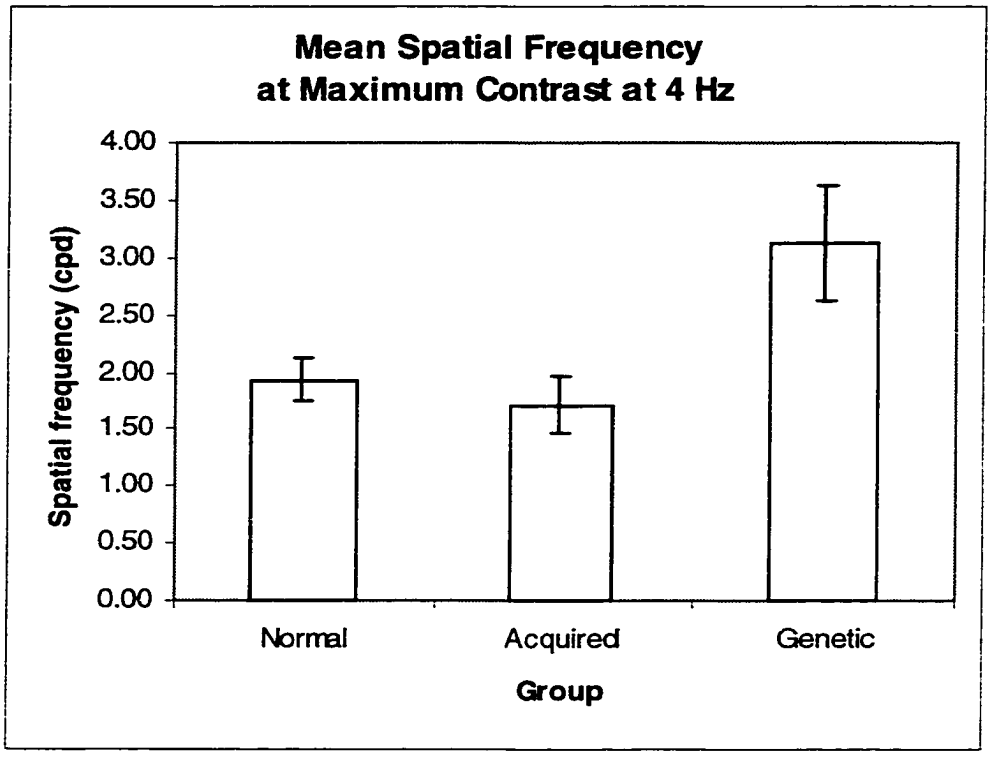


Figure B67. Mean spatial frequency at which maximum contrast sensitivity occurred at 4 Hz for each group. Error bars equal +/- 1 SEM.

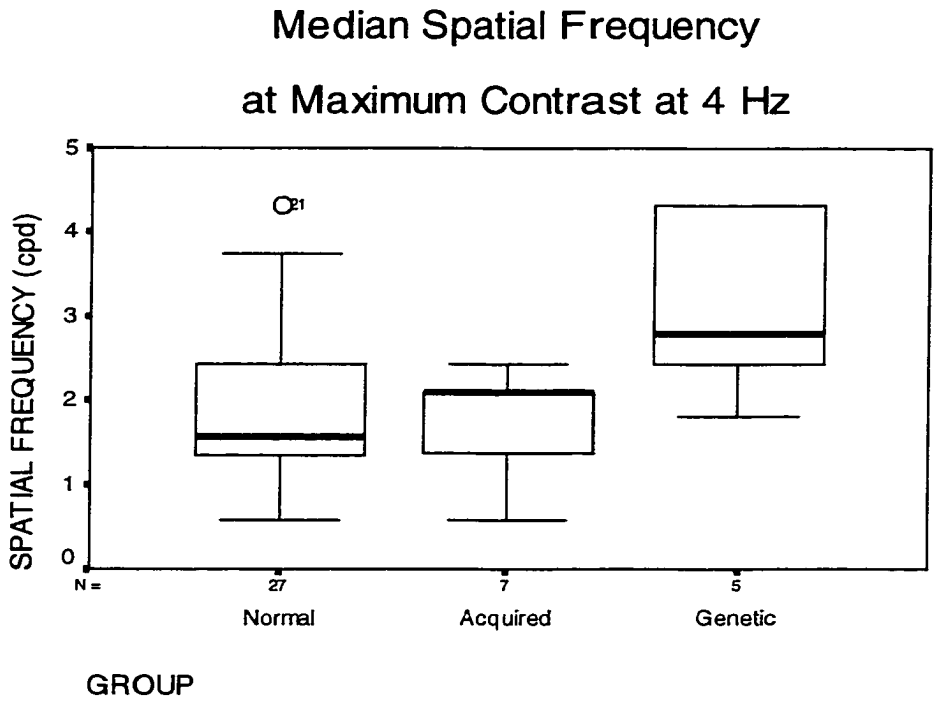


Figure B68. Box-and-whisker plot of spatial frequency at which maximum contrast sensitivity occurred at 4 Hz for each group. As in Figure B1.

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When a Kruskal-Wallis test was applied to the ranked data, maximum contrast sensitivity approached significance ($p = 0.067$), and spatial frequency at maximum contrast fell just shy of significance, $H(2, N = 39) = 5.911$, $p = 0.052$. The differences in sensitivity ratio, however, was significant, $H(2, N = 39) = 13.312$, $p \leq 0.001$.

A 0.6 cpd stimulus at 8 Hz yielded comparable group mean contrast sensitivities for the normal and acquired groups, and a higher sensitivity for the genetic group. This distinction was maintained at the level of group medians, with acquireds more sensitive than normals (see Table B3 and Figures B69 and B70). There was less variability than was seen in previous measures, although the majority of sensitivities of individual color-normal and acquired color-deficient observers greatly overlapped. The mean maximum sensitivity at this temporal frequency was also highest for the genetic group, followed by the normals and acquireds. The group differences, however, were smaller. The median maximum contrast sensitivity of the acquired group slightly exceeded that of the genetic group, and was somewhat distinct from the normal group (see Table B4 and Figures B71 and B72). Interobserver variability was greatest among the color-normals. The ranges of each of the groups overlapped extensively with one another. The low-to-peak sensitivity ratios produced were found to average in a consistent manner from a high of 0.72 for the genetic group to a low of 0.62 for the acquired group, the normal group equidistant between them. Group medians paralleled in this way, as well (see Table B5 and Figures B73 and B74). Variability was largest for the color-normals and genetic color-deficients, with ranges coinciding across all groups. The peak sensitivity was located, on average, at a spatial frequency greatest for normals and least for genetics. These two groups, however, showed equivalent medians (the difference in means attributable to

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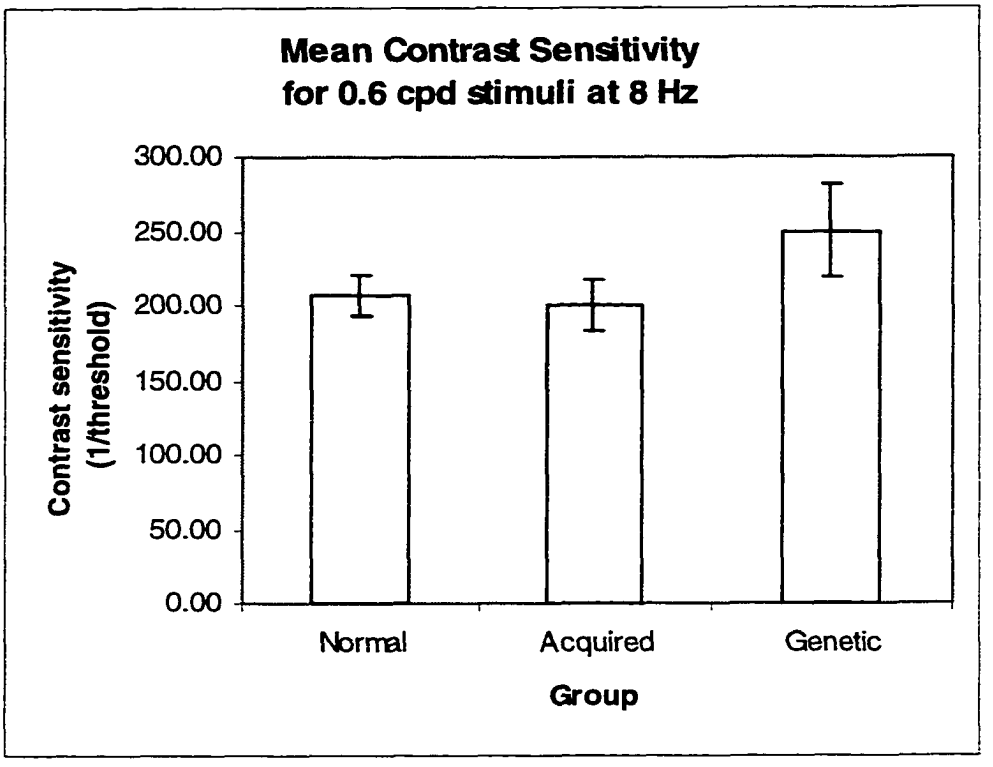


Figure B69. Mean contrast sensitivity for a 0.6 cpd sinusoidal grating modulated at a frequency of 8 Hz for each group. Error bars equal +/- 1 SEM.

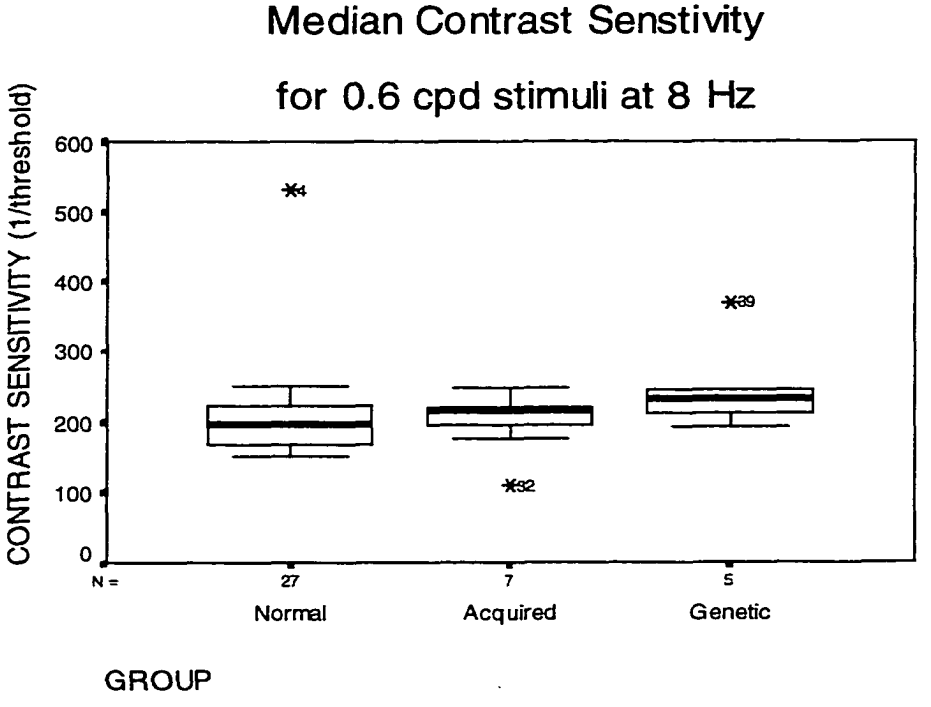


Figure B70. Box-and-whisker plot of contrast sensitivity for a 0.6 cpd sinusoidal grating modulated at a frequency of 8 Hz for each group. As in Figure B1.

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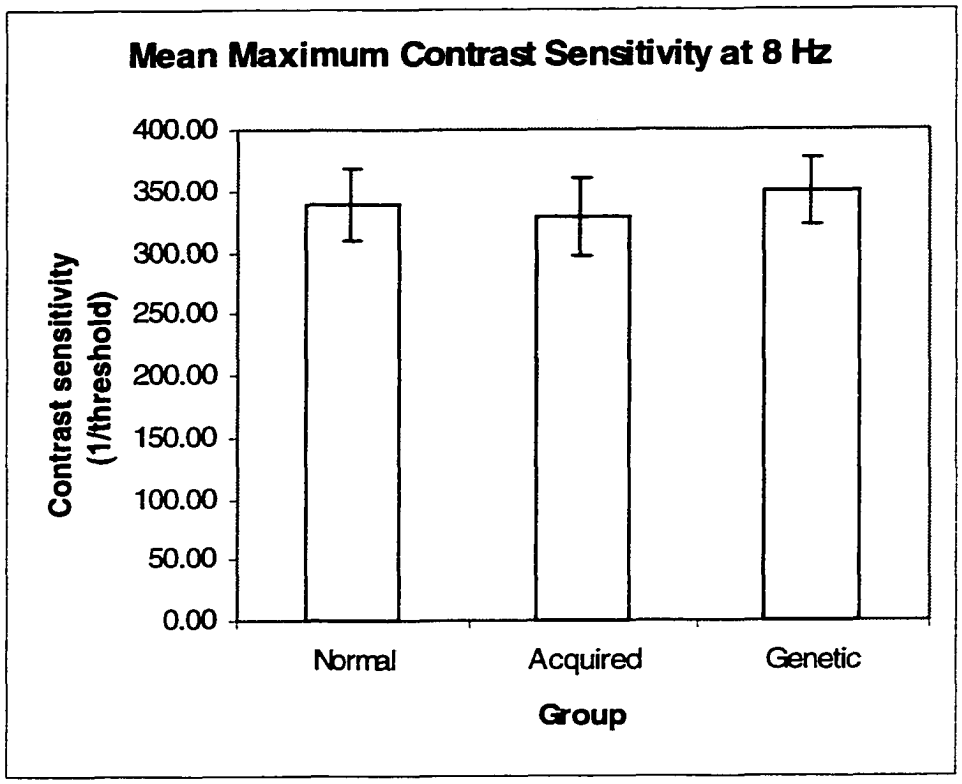


Figure B71. Mean maximum contrast sensitivity for a sinusoidal grating modulated at a frequency of 8 Hz for each group. Error bars equal +/- 1 SEM.

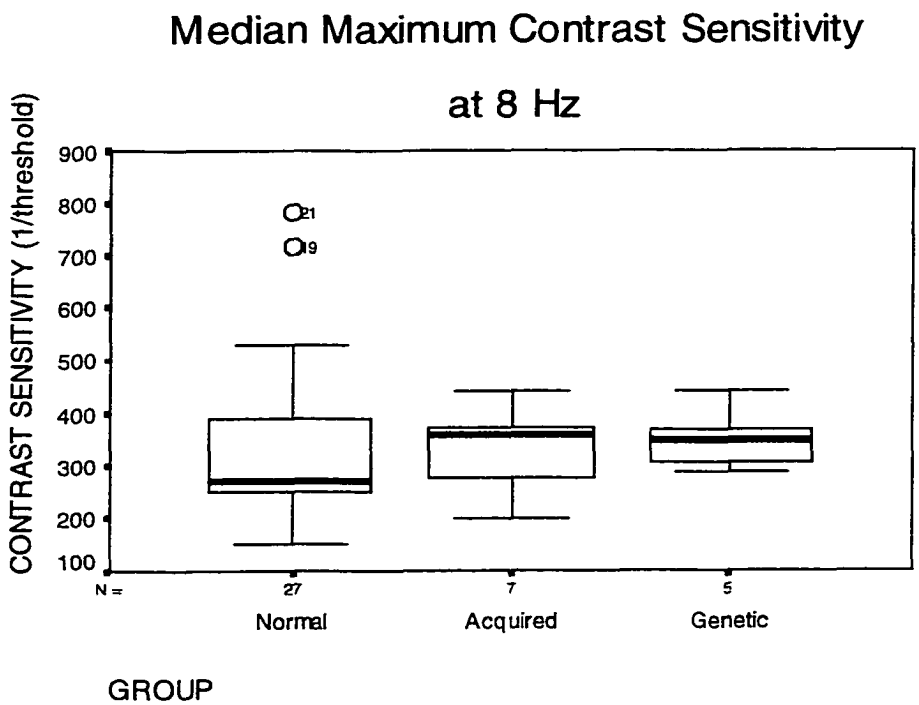


Figure B72. Box-and-whisker plot of maximum contrast sensitivity for a sinusoidal grating modulated at a frequency of 8 Hz for each group. As in Figure B1.

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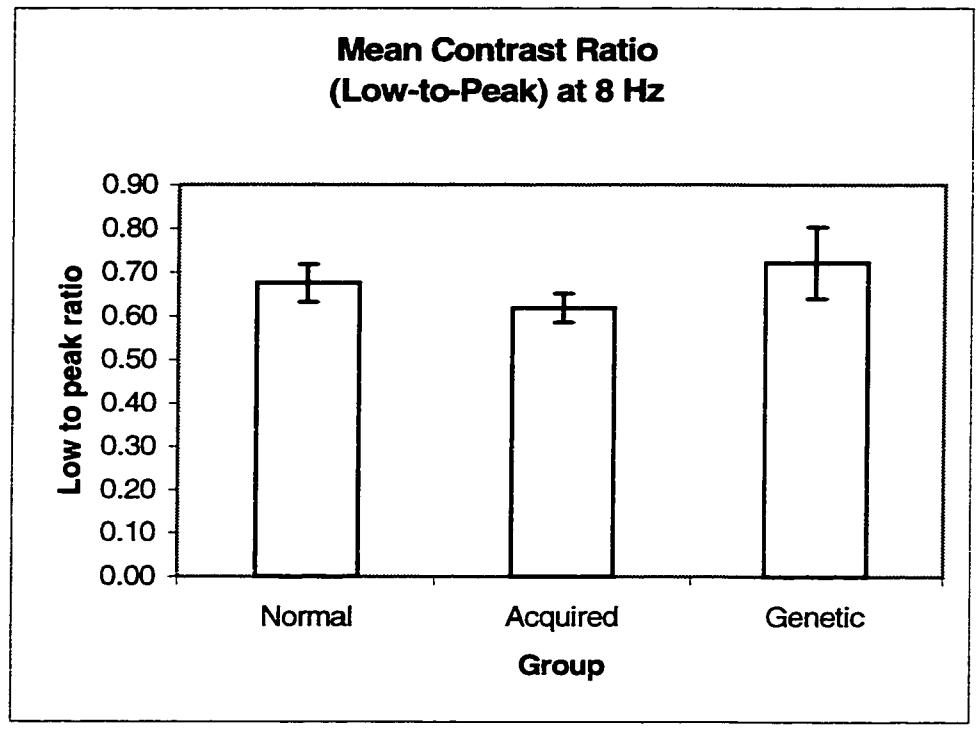


Figure B73. Mean low-to-peak contrast sensitivity ratio (sensitivity at 0.6 cpd/maximum sensitivity) at 8 Hz for each group. Error bars equal +/- 1 SEM.

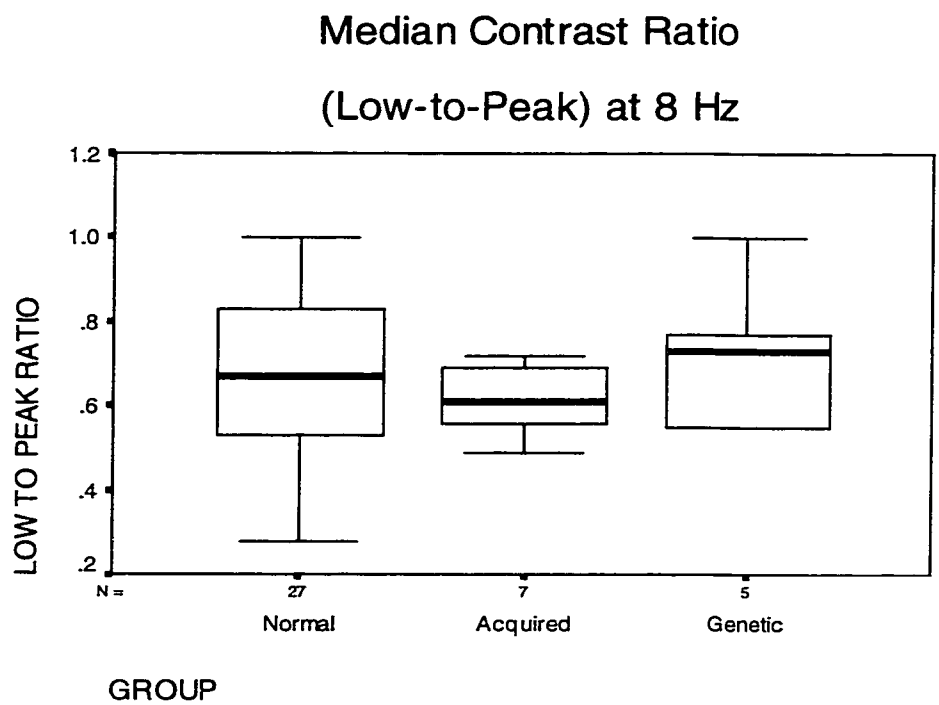


Figure B74. Box-and-whisker plot of low-to-peak contrast sensitivity ratio (sensitivity at 0.6 cpd/maximum sensitivity) at 8 Hz for each group. As in Figure B1.

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three extreme values), slightly above that of acquireds (see Table B6 and Figures B75 and B76). Variability in sensitivities among observers in each group was moderate, with considerable overlap in range. No measure was found to be significant according to either a one-way ANOVA or to the Kruskal-Wallis test ($p > 0.05$).

A 15 Hz modulated grating produced a mean contrast sensitivity that was highest among genetic color-deficient observers and lowest among acquired color-deficient observers, with color-normals occupying an intermediate value. This was retained at the level of group medians given the proximity of the means and medians (see Table B3 and Figures B77 and B78). The degree of interobserver variability within each group was somewhat modest, although ranges did overlap. The peak contrast sensitivity at this temporal frequency averaged out to be greatest for the acquired group, distancing itself from the genetic and normal groups, which placed second and third. There was less of a disparity between the acquired and genetic groups' medians, but more of one between the genetic's and the normal's (see Table B4 and Figures B79 and B80). Variability of sensitivities among acquired color-deficient group members was quite large, the range completely enveloping the other groups'; however, this group's median value was well above the highest recorded threshold in the genetic group, and was just shy of the normal's upper limit. Group mean sensitivity ratios were nearly equivalent for the color-normals and genetic color-deficients, with acquired color-deficients' well below. This continued among group medians, except for genetics pulling slightly ahead (see Table B5 and Figures B81 and B82). The amount of intragroup variability was nearly equal. The central 50% of acquired observer's sensitivities, however, matched only the bottom 25% of normals' and genetics'. Maximum sensitivities were found to occur at a substantially

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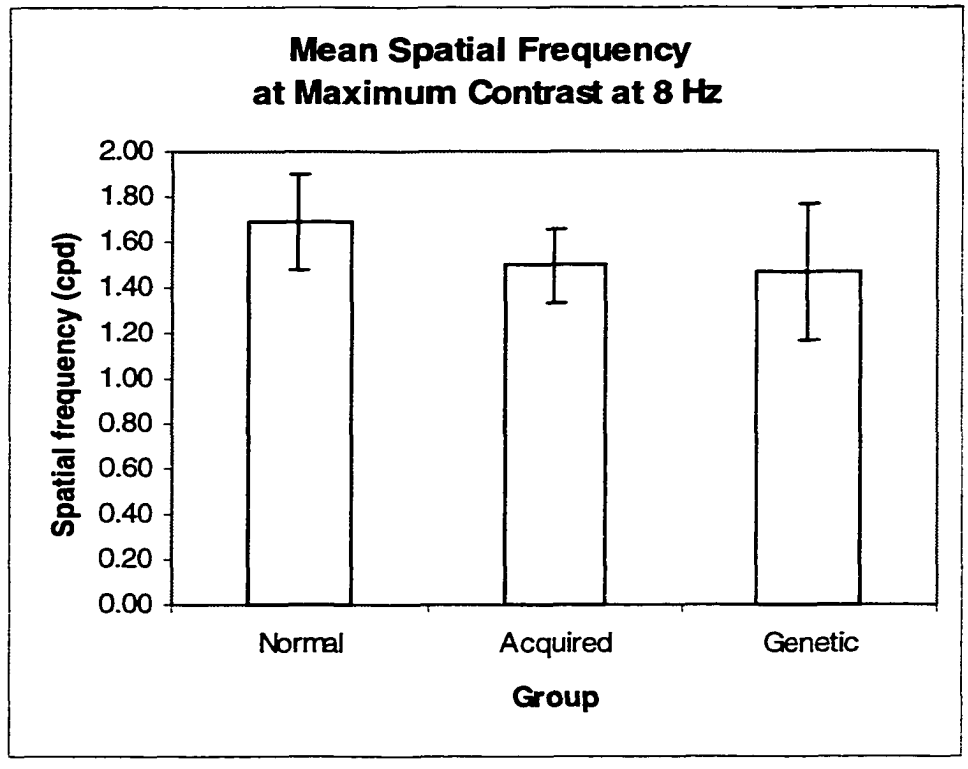


Figure B75. Mean spatial frequency at which maximum contrast sensitivity occurred at 8 Hz for each group. Error bars equal +/- 1 SEM.

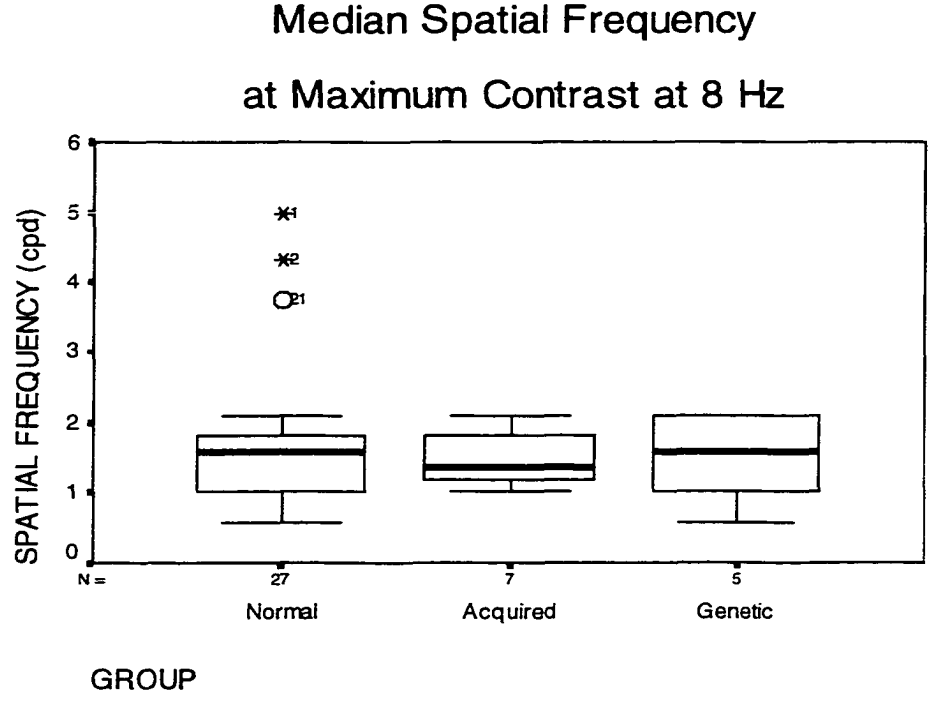


Figure B76. Box-and-whisker plot of spatial frequency at which maximum contrast sensitivity occurred at 8 Hz for each group. As in Figure B1.

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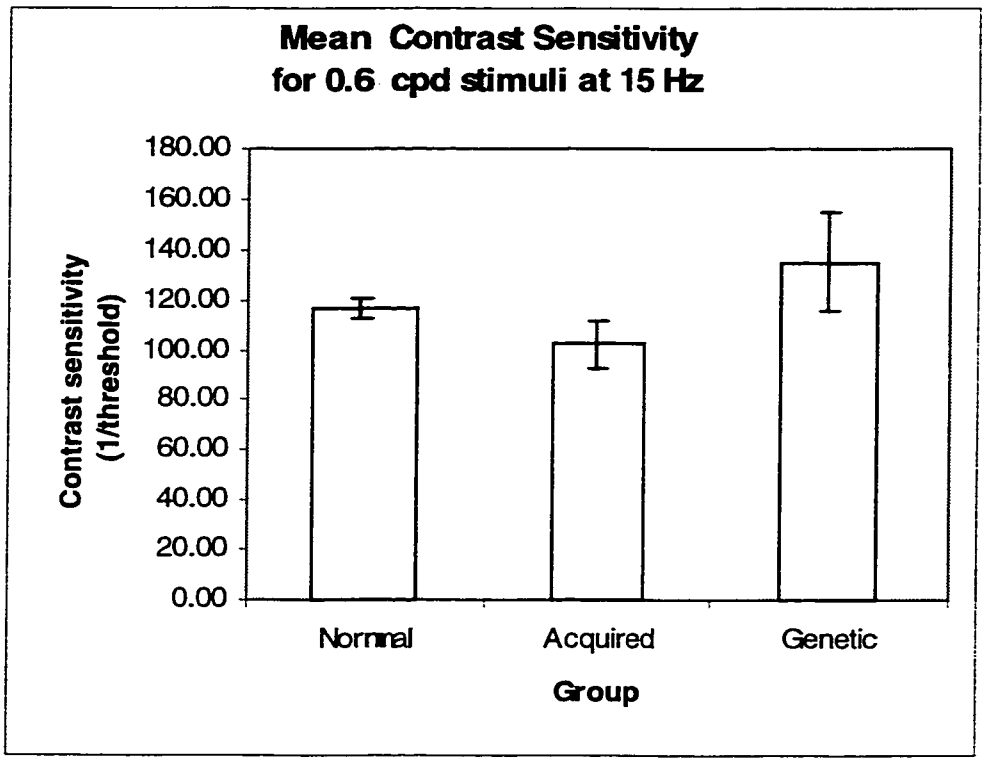


Figure B77. Mean contrast sensitivity for a 0.6 cpd sinusoidal grating modulated at a frequency of 15 Hz for each group. Error bars equal +/- 1 SEM.

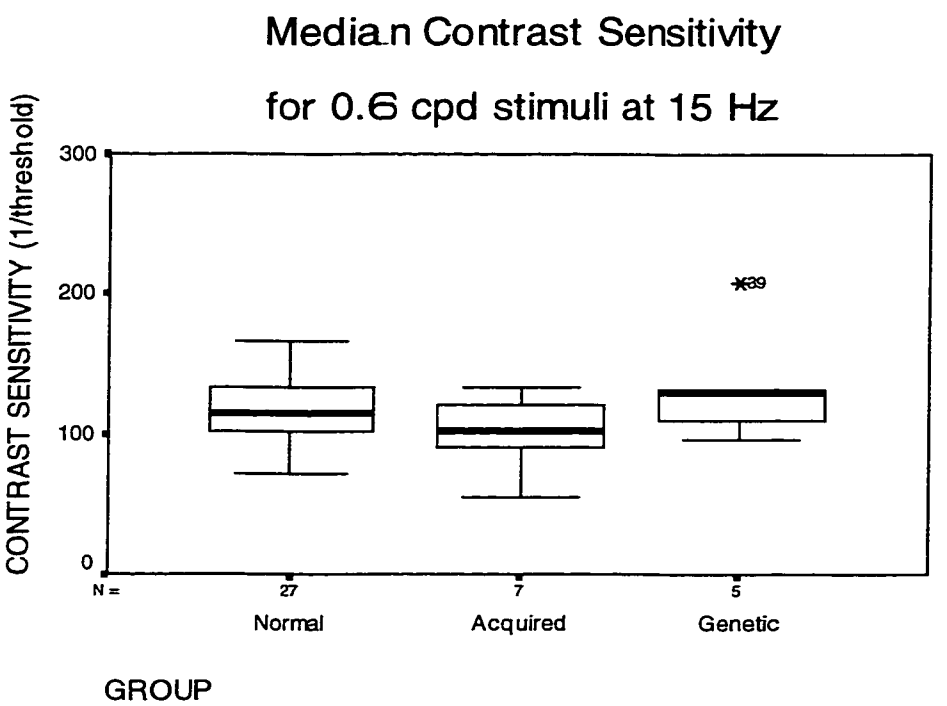


Figure B78. Box-and-whisker plot of contrast sensitivity for a 0.6 cpd sinusoidal grating modulated at a frequency of 15 Hz for each group. As in Figure B1.

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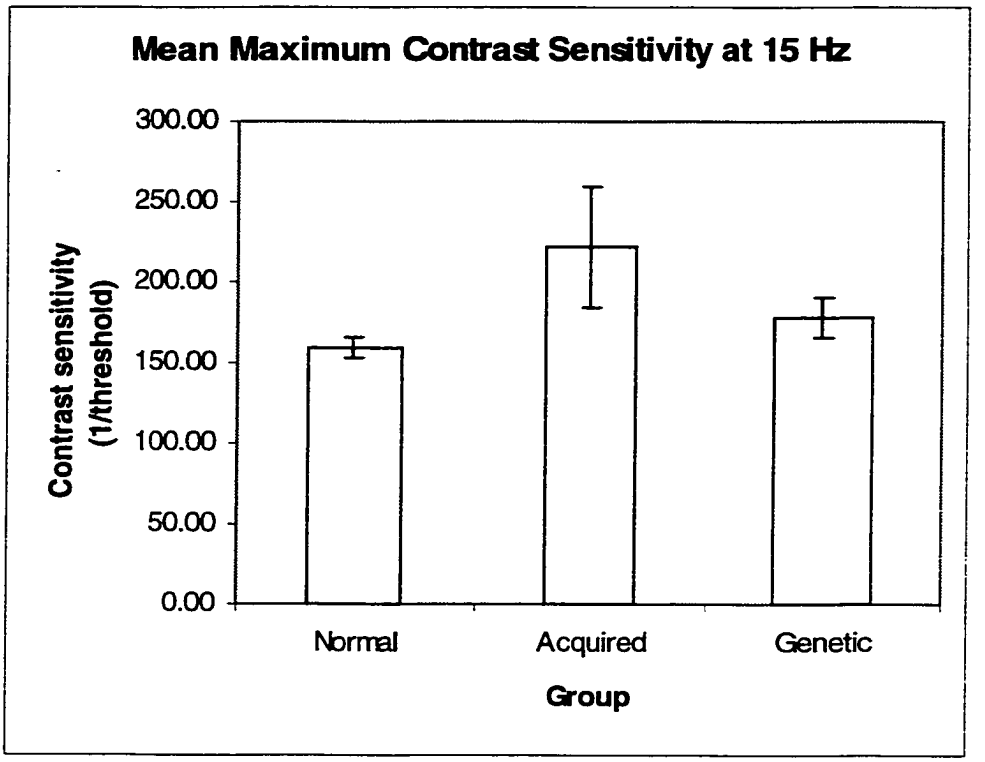


Figure B79. Mean maximum contrast sensitivity for a sinusoidal grating modulated at a frequency of 15 Hz for each group. Error bars equal +/- 1 SEM.

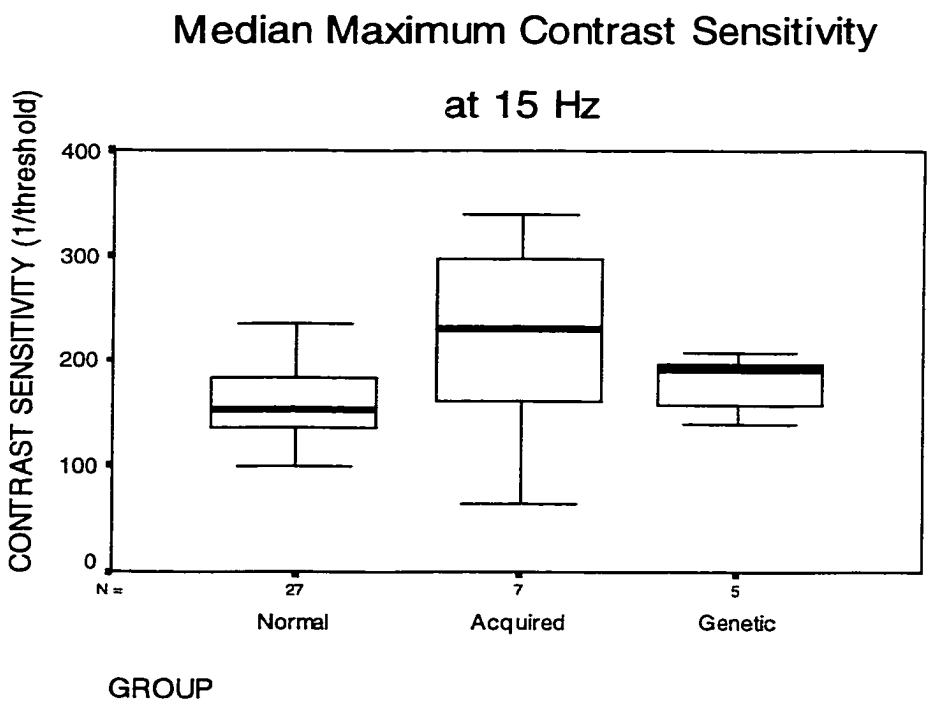


Figure B80. Box-and-whisker plot of maximum contrast sensitivity for a sinusoidal grating modulated at a frequency of 15 Hz for each group. As in Figure B1.

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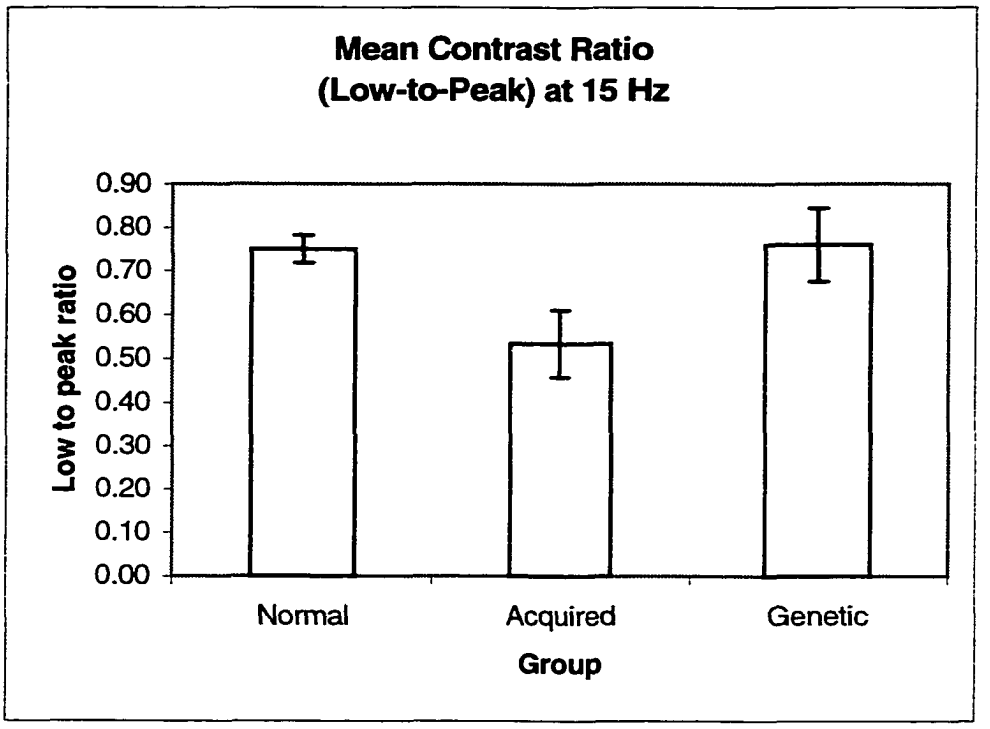


Figure B81. Mean low-to-peak contrast sensitivity ratio (sensitivity at 0.6 cpd/maximum sensitivity) at 15 Hz for each group. Error bars equal +/- 1 SEM.

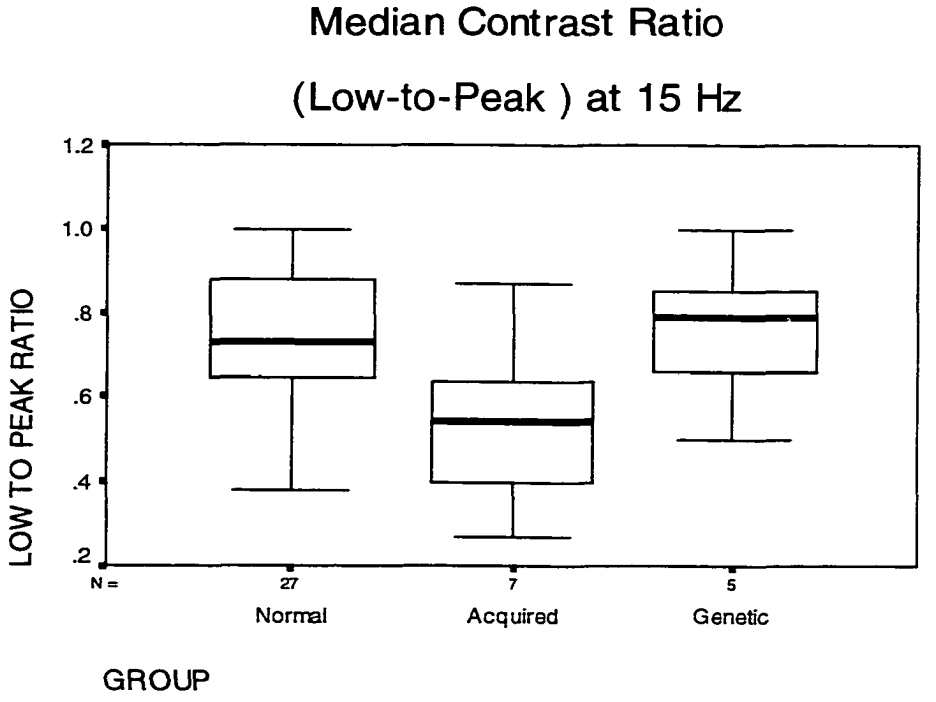


Figure B82. Box-and-whisker plot of low-to-peak contrast sensitivity ratio (sensitivity at 0.6 cpd/maximum sensitivity) at 15 Hz for each group. As in Figure B1.

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higher mean spatial frequency for the acquired group than for either the normal or genetic. This was upheld at the group median level of comparison (see Table B6 and Figures B83 and B84). The color-normal group had the largest amount of interobserver variability, whereas the genetic group essentially had none (4 out of the 5 members had identical data points). The top 75% of the acquired group's sensitivities overlay the upper quartile of the normal group. Using a one-way ANOVA, group differences among the following measures were found to be significant: maximum contrast sensitivity, $F(2, 36) = 4.19$, $p < 0.05$; low-to-peak sensitivity, $F(2, 36) = 4.45$, $p < 0.05$; and spatial frequency at maximum contrast sensitivity, $F(2, 36) = 6.18$, $p < 0.01$. Applying a Kruskal-Wallis test to the ranked data, the difference between groups of the spatial frequency at maximum contrast sensitivity was found to be statistically significant, $H\ (2, N = 39) = 8.667$, $p < 0.05$. The ratio of sensitivities at lowest spatial frequency to maximum contrast was found to approach significance ($p = 0.061$).

For the highest temporal frequency tested (24 Hz), the mean contrast sensitivity at the lowest spatial frequency was greatest for the genetic color-deficient group, decreasing in equal increments down to the color-normal group. The differences between group means, however, were not very large. A similar differential trend was seen among group medians, although the acquired group had a slightly higher sensitivity than genetics (see Table B3 and Figures B85 and B86). The normal group was most variable, and the ranges of sensitivities of each group overlapped considerably. The average maximum sensitivity recorded among the groups also followed the above trend. Here too, the genetic color-deficients had superior sensitivity. This reversed again when examining group medians, with the acquired color-deficient group having higher sensitivities (see Table B4 and

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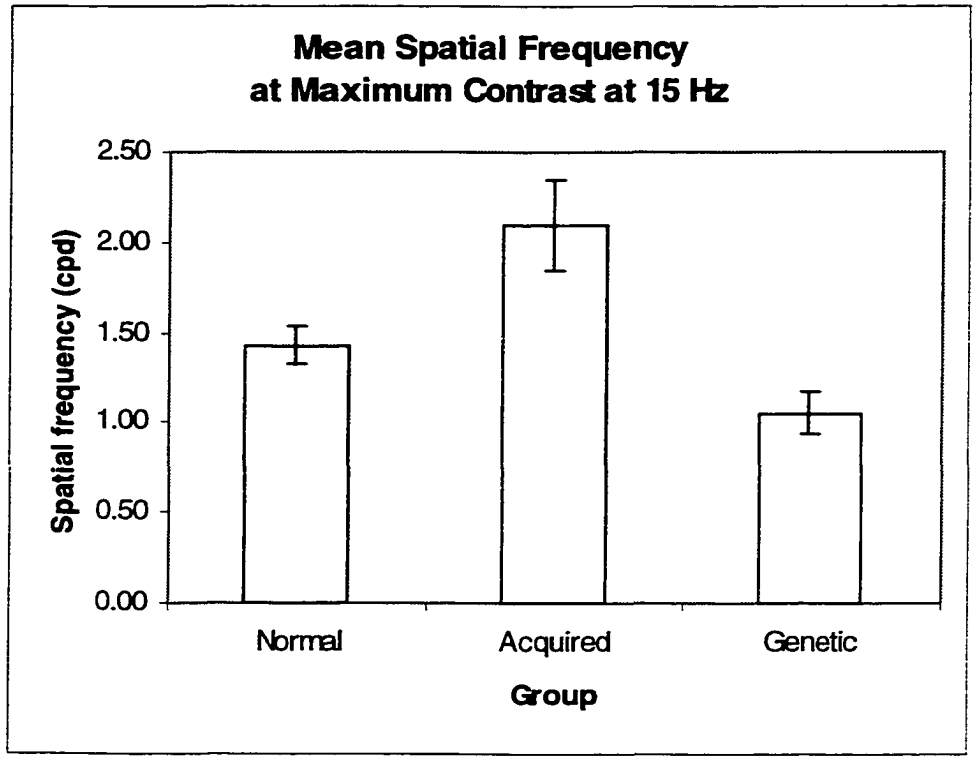


Figure B83. Mean spatial frequency at which maximum contrast sensitivity occurred at 15 Hz for each group. Error bars equal +/- 1 SEM.

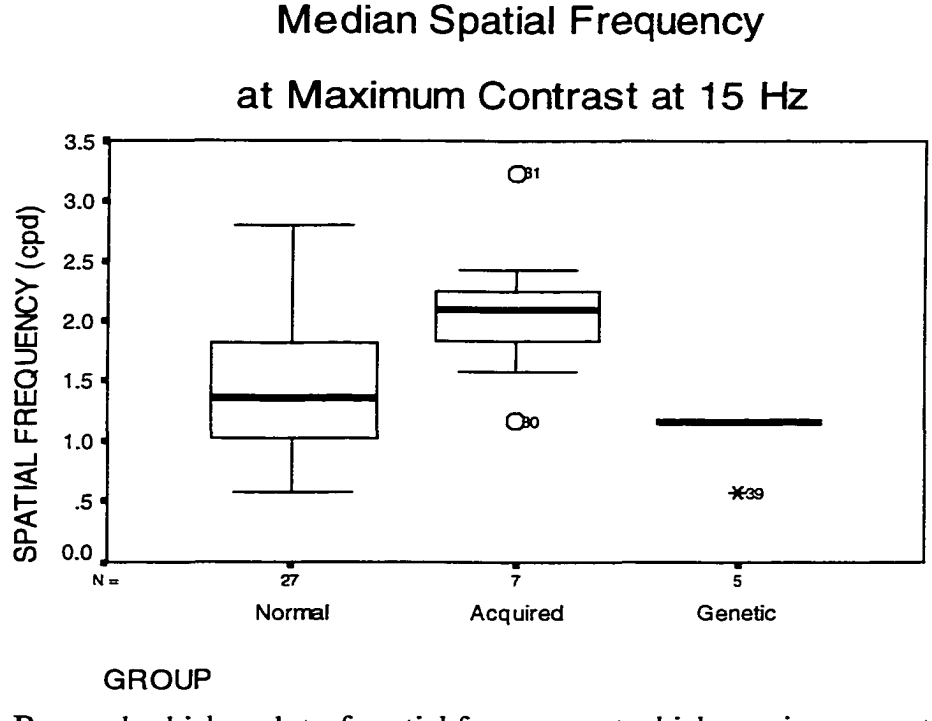


Figure B84. Box-and-whisker plot of spatial frequency at which maximum contrast sensitivity occurred at 15 Hz for each group. As in Figure B1.

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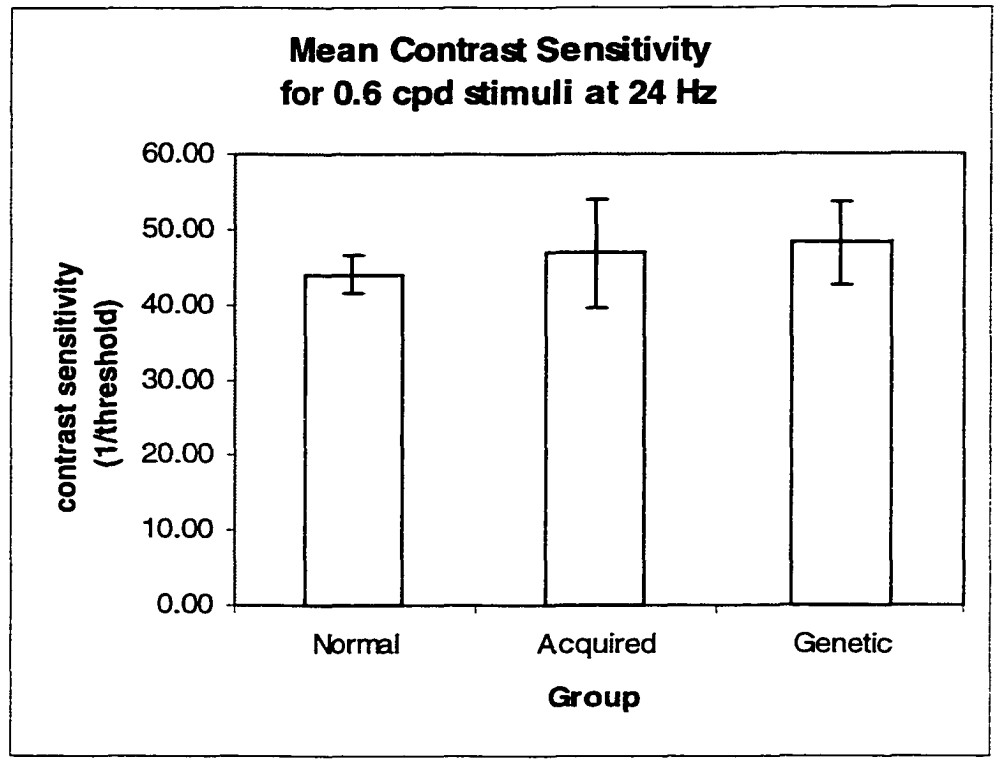


Figure B85. Mean contrast sensitivity for a 0.6 cpd sinusoidal grating modulated at a frequency of 24 Hz for each group. Error bars equal +/- 1 SEM.

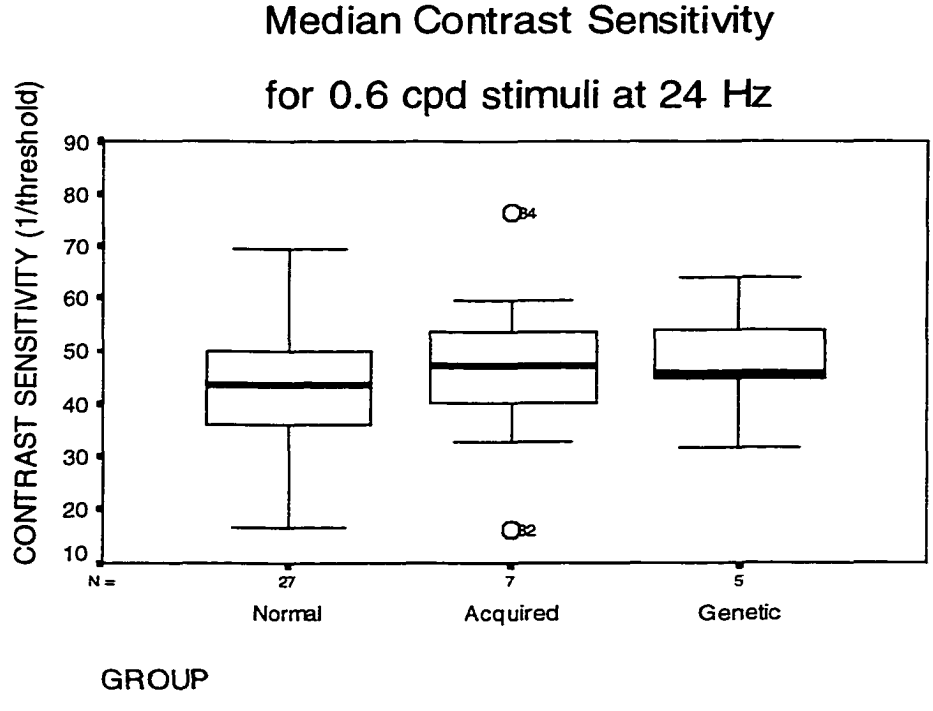


Figure B86. Box-and-whisker plot of contrast sensitivity for a 0.6 cpd sinusoidal grating modulated at a frequency of 24 Hz for each group. As in Figure B1.

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Figures B87 and B88). The acquired group was also most variable between observers, although all groups were found to have overlapping sensitivities. These values yielded mean low-to-peak sensitivity ratios of an opposite trend. Color-normals had the highest ratios, decreasing in almost equal steps down to the lowest ratios in the genetic color-deficients. This was found to be consistent across group medians, as well (see Table B5 and Figures B89 and B90). All three groups had moderate variability, with a large degree of overlap in range. The peak occurred at a mean spatial frequency somewhat higher for the genetic group, compared to the normal and acquired groups, as did the median (see Table B6 and Figures B91 and B92). There was less variability among genetic observers (versus normals and acquireds), but there was definite overlap in the frequencies observed across groups. Group differences among these measures were not found to be significant using either a one-way ANOVA or the Kruskal-Wallis test ($p > 0.05$).

Binocular vision testing. Results of the test for eye alignment (horizontal fusion) are presented in Table B7 and in Figures B93 and B94. The mean number of prism diopters required for horizontal fusion was greatest for acquired color-deficients and least for genetic color-deficients (see Figure B93). Unlike the means, which were somewhat disparate between the color-deficient groups, medians were equivalent between these two groups, and above that needed for color-normals (see Figure B94). According to a one-way ANOVA, as well as to a Kruskal-Wallis test applied to the ranked data, these differences between groups were not significant at $\alpha = 0.05$.

Given that dioptric power can occur in both the positive and negative directions, the above statistics are not very meaningful for our purposes. Rather, using the criterion

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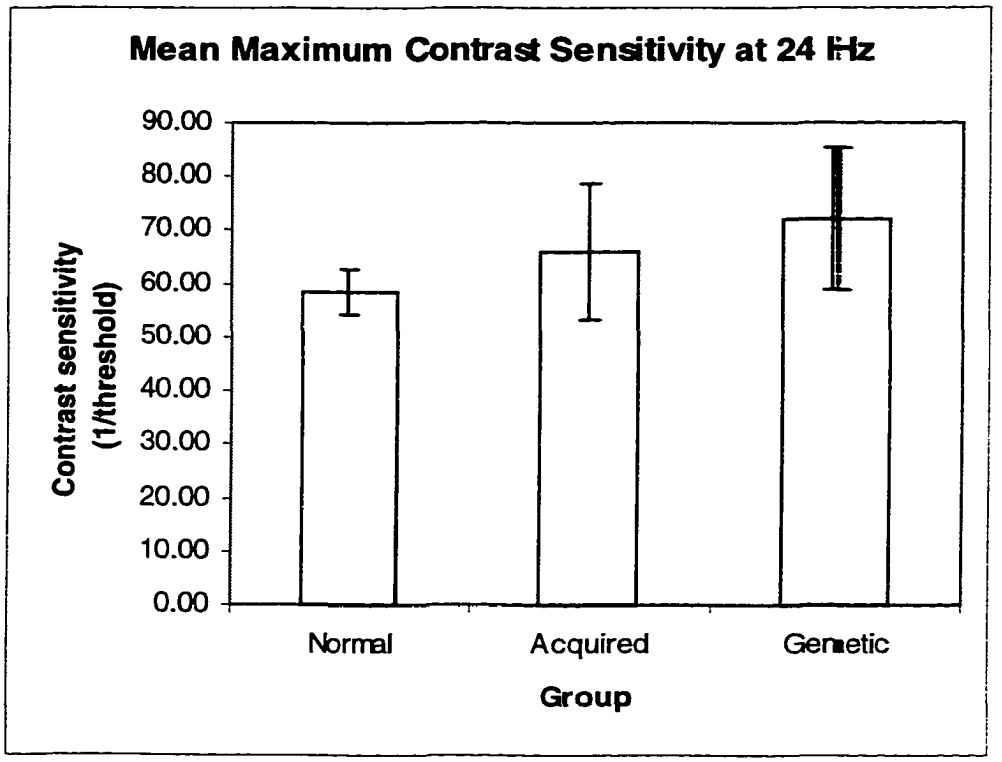


Figure B87. Mean maximum contrast sensitivity for a 0.6 cpd sinusoidal grating modulated at a frequency of 24 Hz for each group. Error bars equal +/- 1 SEM.

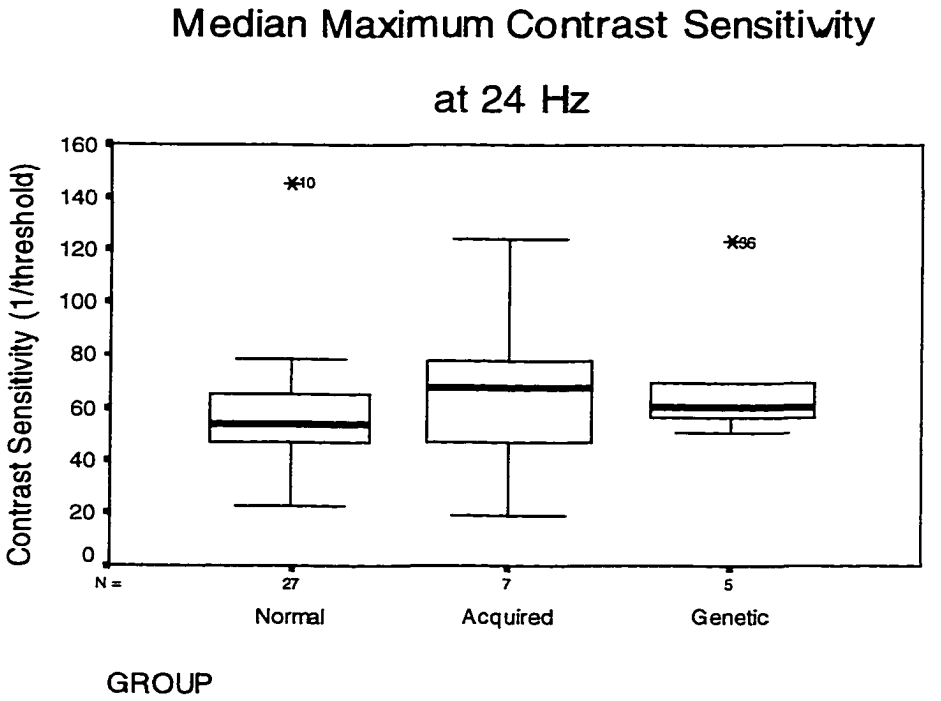


Figure B88. Box-and-whisker plot of maximum contrast sensitivity for a 0.6 cpd sinusoidal grating modulated at a frequency of 24 Hz for each group. As in Figure B1.

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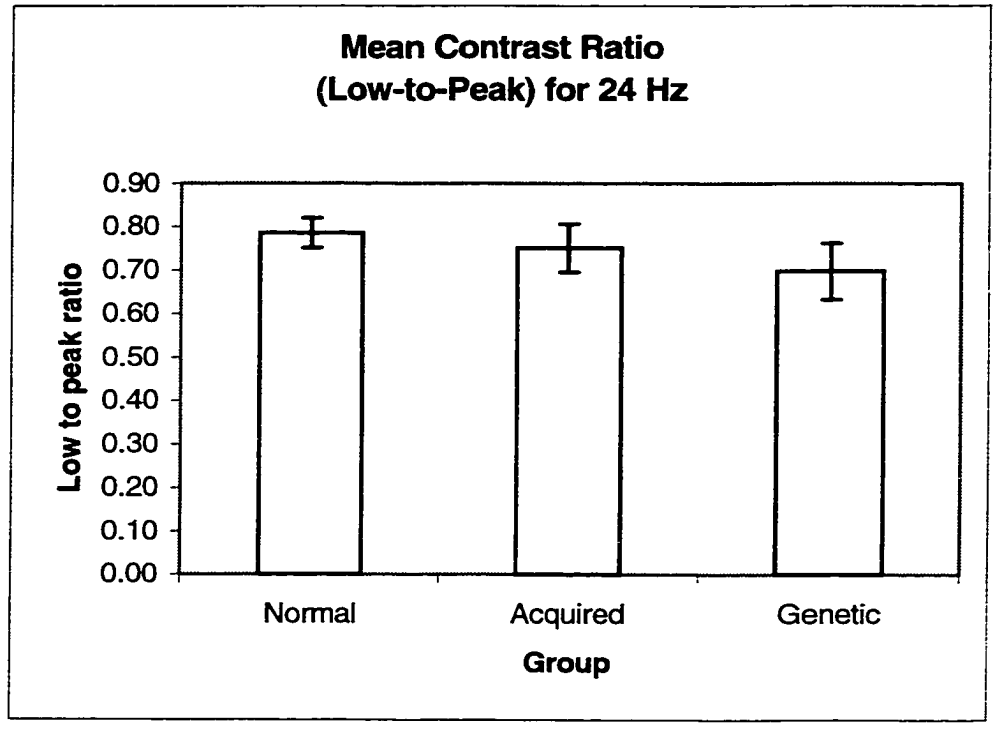


Figure B89. Mean low-to-peak contrast sensitivity ratio (sensitivity at 0.6 cpd/maximum sensitivity) at 24 Hz for each group. Error bars equal +/- 1 SEM.

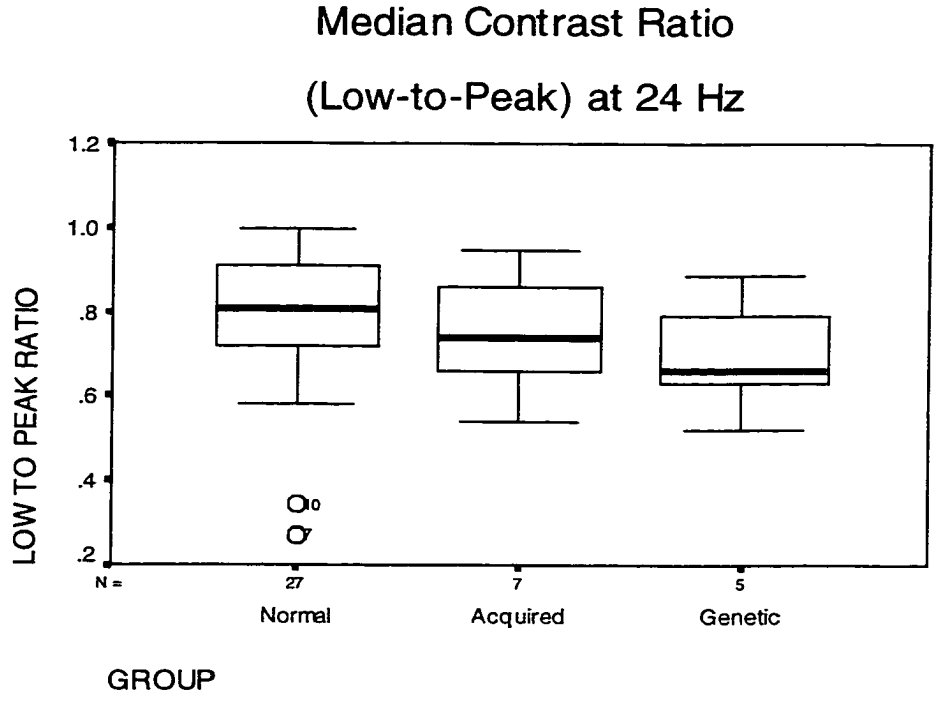


Figure B90. Box-and-whisker plot of low-to-peak contrast sensitivity ratio (sensitivity at 0.6 cpd/maximum sensitivity) at 24 Hz for each group. As in Figure B1.

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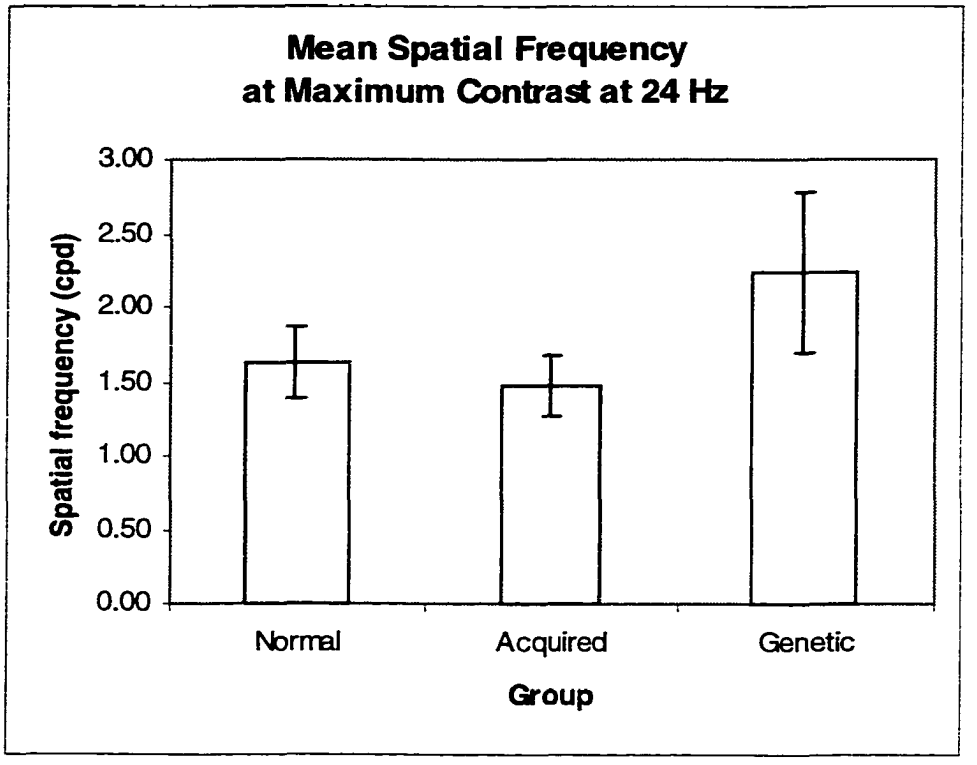


Figure B91. Mean spatial frequency at which maximum contrast sensitivity occurred at 24 Hz for each group. Error bars equal +/- 1 SEM.

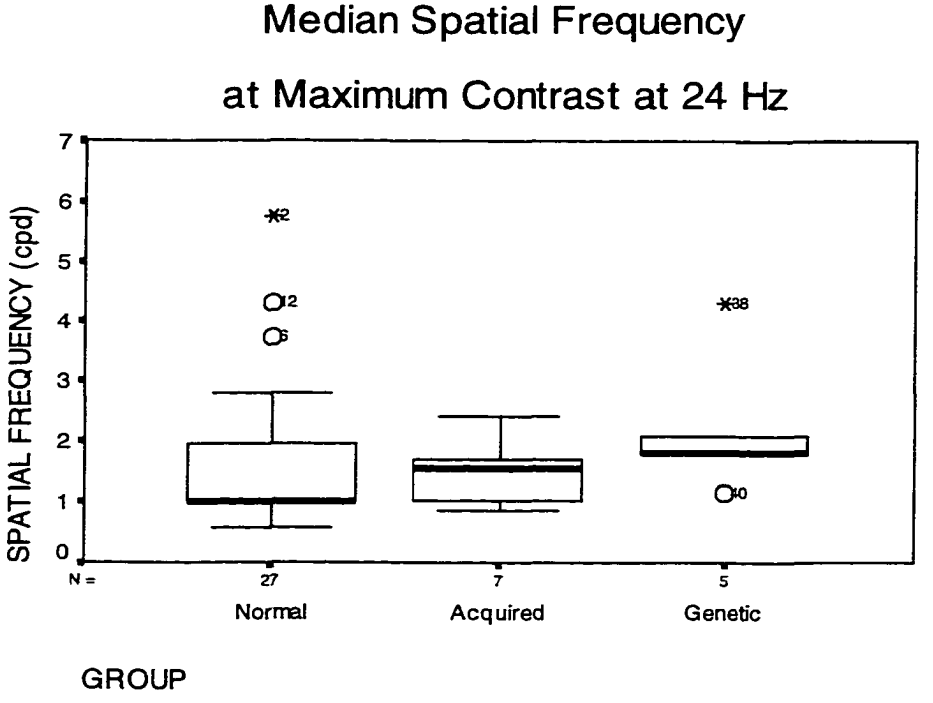


Figure B92. Box-and-whisker plot of spatial frequency at which maximum contrast sensitivity occurred at 24 Hz for each group. As in Figure B1.

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Table B7. Binocular Vision Testing: Eye Alignment (in diopters) and IPD (in mm)

| Group | Normal | Acquired | Genetic |
|---|---|---|---|
| Mean number of prism diopters required for horizontal fusion | 1.04 | 1.21 | 0.90 |
| SEM | 0.80 | 0.96 | 1.81 |
| Median | 1.25 | 2.00 | 2.00 |
| Mean interpupillary distance | 60.68 | 63.79 | 65.60 |
| SEM | 0.62 | 1.66 | 0.71 |
| Median | 61.00 | 66.00 | 64.50 |

whereby a necessity of more than five diopters constituted poor or problematic fusion, a total of six color-normal observers may be described as such.

Fifteen normals were found to be right eye-dominant and 13 were left eye-dominant. All but one observer each were right eye-dominant in the acquired and genetic color-deficient groups.

Mean interpupillary distance (values for which can also be found in Table B7 and in Figures B95 and B96) was found to be largest for the genetic group and smallest for the normal group. A one-way ANOVA found this difference to be statistically significant, $F(2, 37) = 5.96$, $p < 0.01$, as did a Kruskal-Wallis test performed on the ranked data, $H\ (2, N = 40) = 9.604$, $p < 0.01$. Despite the genetic group having the largest mean IPD, it is important to note that the acquired group's median IPD exceeded that of the genetics', and that members of all three groups had overlapping measurements (though variability was low, especially among genetics; see Figure B96).

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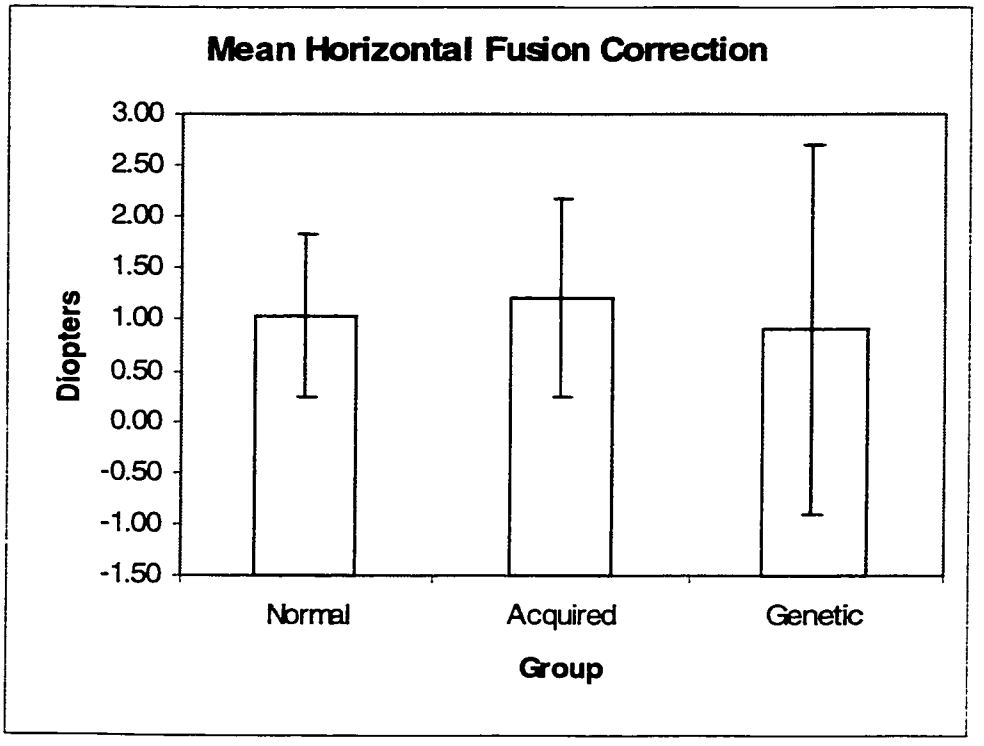


Figure B93. Mean number of prism diopters required for horizontal fusion for each group. Error bars equal +/- 1 SEM.

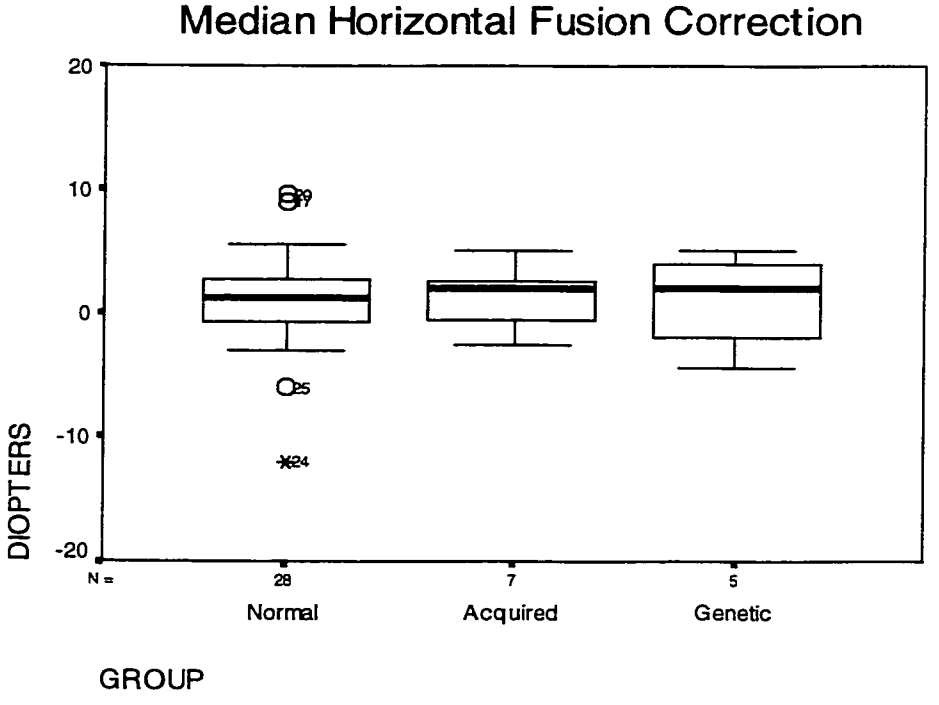


Figure B94. Box-and-whisker plot of number of prism diopters required for horizontal fusion for each group. As in Figure B1.

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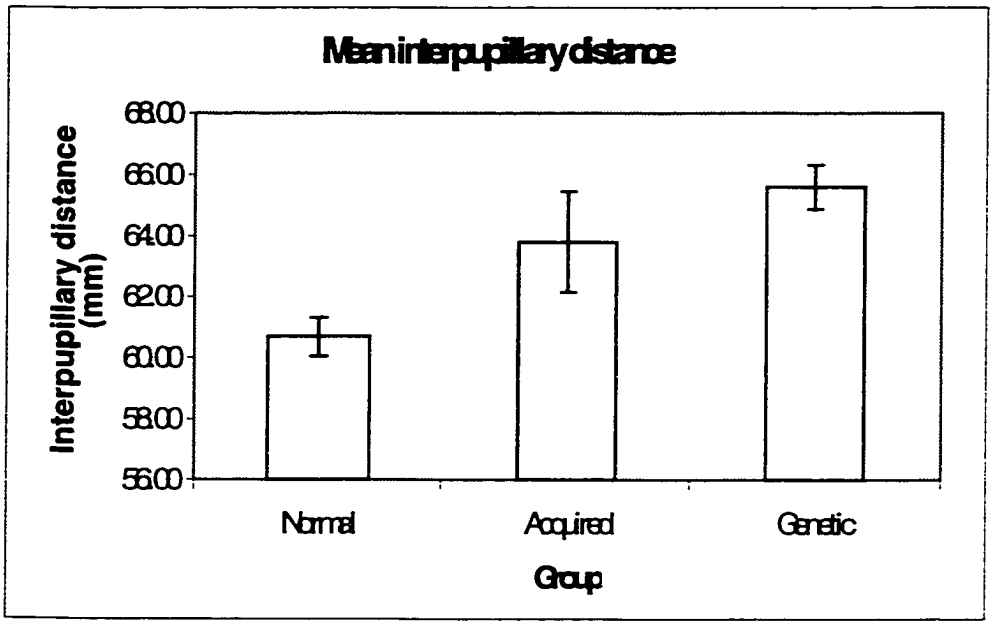


Figure B95. Mean interpupillary distance for each group. Error bars equal +/- 1 SEM.

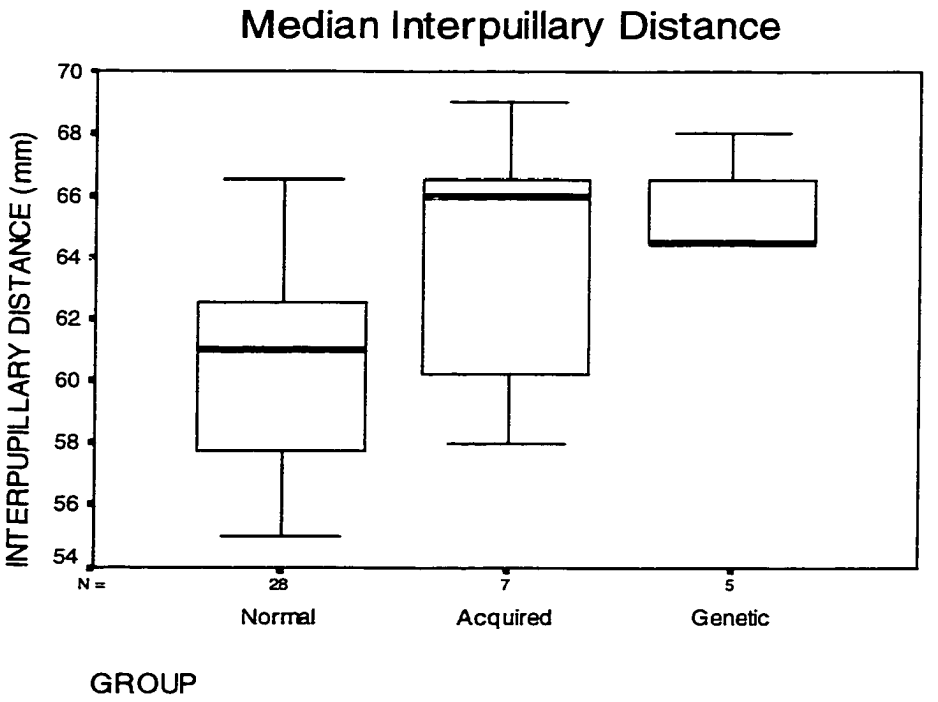


Figure B96. Box-and-whisker plot of interpupillary distance for each group. As in Figure B1.

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The test for ocular rivalry produced 12 right eye-dominant (3 with a strong degree of rivalry, 5 with a weak degree, and 4 unclassified), 14 left eye-dominant (7 rated as strong, 6 rated as weak, and 1 unclassified), and 2 mixed dominance (both strong) color-normals. Nine of these individuals had shown dominance in the opposite eye in the peephole test for dominance. The acquired color-deficient group was comprised of 6 right eye-dominant (3 strongly, 1 weakly, and 2 unclassified) and 1 left eye-dominant (strong) observers, 2 of whom showed opposite results compared to the sighting test. The genetic color-deficient group consisted of one weakly right eye-dominant, three weakly left eye-dominant, and one strongly mixed members. Only two of these observers showed consistent dominance between the two tests. Interestingly, of the five acquired observers maintaining a given dominance across tests, two used their better (possibly non-deficient) eye, while the other three used their poorer eye. Similarly, of the two genetics with identical eye dominance in the two tests, each preferred their "better" eye (though both eyes were quite similar regarding type and degree of severity in each of these observers).

Based on the participant's performance on the stereo-screening tests, all observers were found to have at least some form of stereopsis, except for one individual belonging to the acquired color-deficient group. (This observer attempted the stereopsis experiment, nonetheless; see below and Discussion.) Thus, collectively, binocular categorization was distributed as follows: For the normal group, 6 observers were considered as having poor fusion with stereopsis, and the remaining 22 had good fusion with stereopsis; for the acquired group, the one member described above was classified as having good fusion without stereopsis, with the others all having good fusion with stereopsis; and for the genetic group, all observers were counted as having good fusion with stereopsis.

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Stereopsis. Results from the stereopsis experiment are presented in Table B8. The intergroup differences seen among mean forward stereoacuity was virtually non-existent at the level of group medians, as the medians for all three groups were nearly identical to each other (see Figure B97). Interobserver variability was largely confined to the normals and acquireds, but the range of thresholds overlapped extensively for every group. Similarly, for the backward condition, the median stereoacuity for all three groups was exactly the same (with n = 25, 4, and 5, respectively, for the normal, acquired, and genetic groups; see Figure B98).

Table B8. Stereopsis: Stereoacuity and Fusion Thresholds (in min arc)

| Group | Normal | Acquired | Genetic |
|---|---|---|---|
| Mean Stereo Minimum Front | 0.63 | 0.78 | 0.31 |
| SEM | 0.13 | 0.38 | 0.06 |
| Median | 0.27 | 0.25 | 0.25 |
| Mean Stereo Minimum Back | 0.44 | 0.29 | 0.25 |
| SEM | 0.11 | 0.04 | 0.00 |
| Median | 0.25 | 0.25 | 0.25 |
| Mean Stereo Maximum Front | 44.21 | 49.82 | 49.58 |
| SEM | 2.09 | 4.73 | 3.66 |
| Median | 45.65 | 52.78 | 50.71 |
| Mean Stereo Maximum Back | 30.83 | 31.39 | 39.36 |
| SEM | 2.13 | 7.62 | 5.83 |
| Median | 27.52 | 28.36 | 32.26 |

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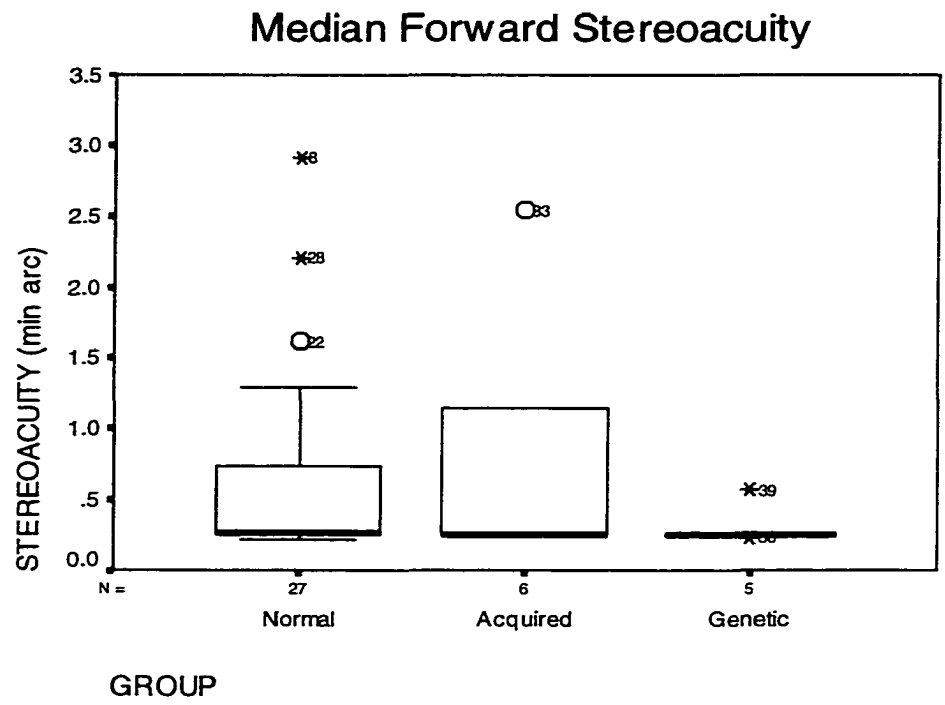


Figure B97. Box-and-whisker plot of forward stereoacuity for each group. As in Figure B1.

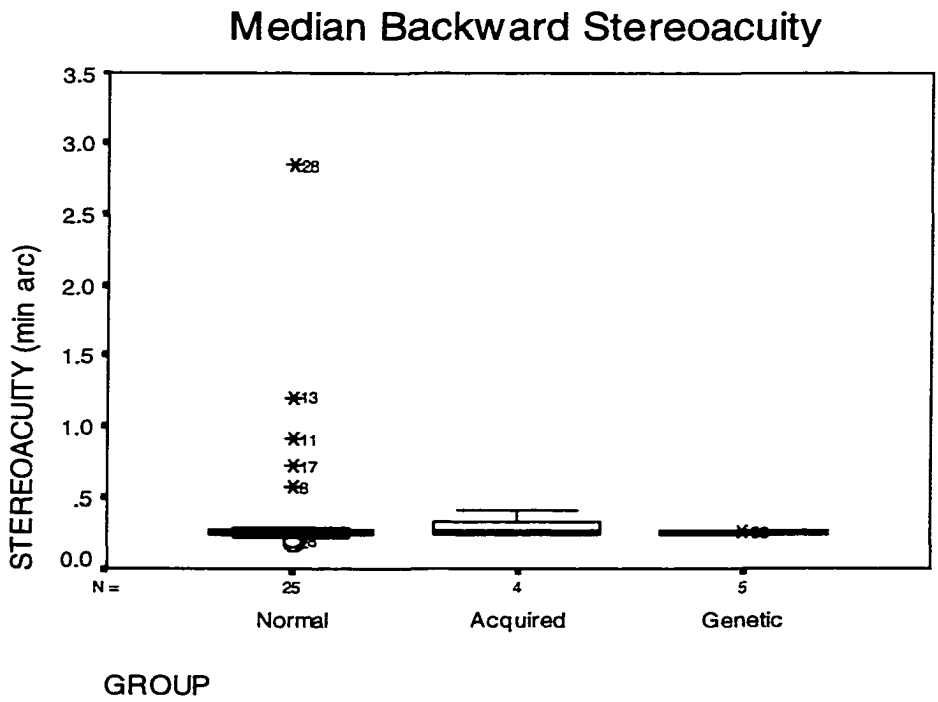


Figure B98. Box-and-whisker plot of backward stereoacuity for each group. As in Figure B1.

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The observed difference between the two color-deficient groups' forward fusion limit was a bit more sizeable when comparing medians than when comparing means (1.07 versus 0.23 min arc, both in favor of the acquired group; see Figure B99). All three groups showed a moderate to large degree of interobserver variability, with the genetic group having both the smallest overall range of thresholds and the lowest maximum threshold (of each group's most sensitive observers) measured across the group ranges. As seen in Figure B100, the genetic group's median backward fusion threshold value was considerably less than their group mean, but the order among the groups' sensitivities was maintained (i.e., with the acquired group still more sensitive than the normals, now by slightly more than 0.8 min arc). Here, too, the groups' ranges of thresholds were considerably large. Yet, the normal and acquired groups' ranges overlapped considerably more so than when the genetic group's range was paired with either of them.

Monocular hyperacuities. Table B9 presents the results for the vernier acuity experiment. Group median vernier acuity, while retaining the rank order, showed a difference of nearly twice the magnitude between normals and acquireds that was found among the means (see Figure B101). Within groups, interobserver variability was large (though less so among genetics), and across groups, ranges overlapped considerably (with normals, for example, presenting both the smallest and second largest of the thresholds recorded).

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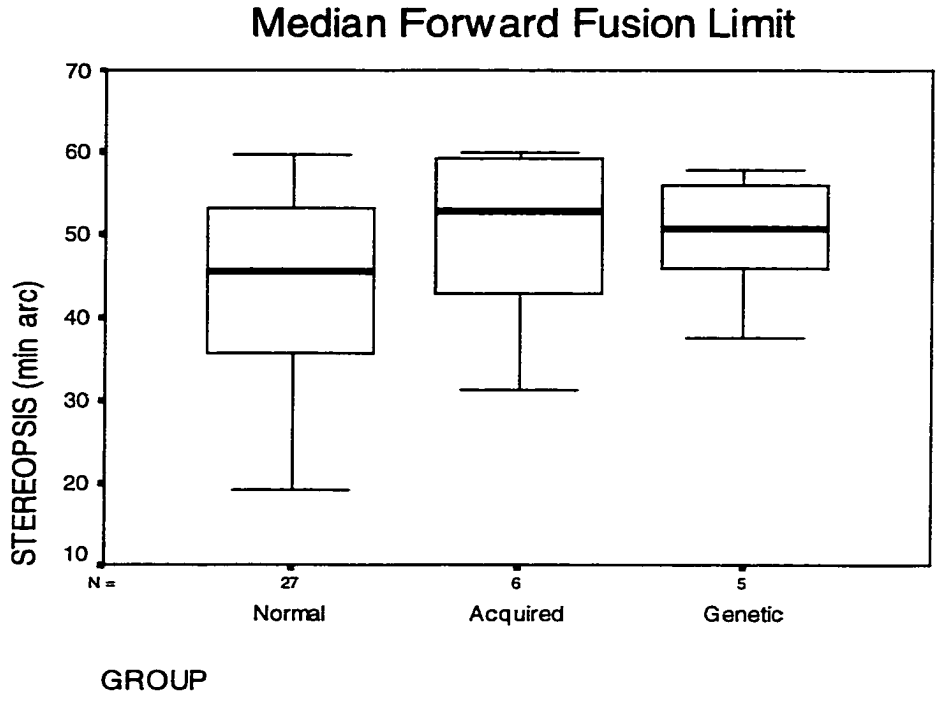


Figure B99. Box-and-whisker plot of forward fusion threshold for each group. As in Figure B1.

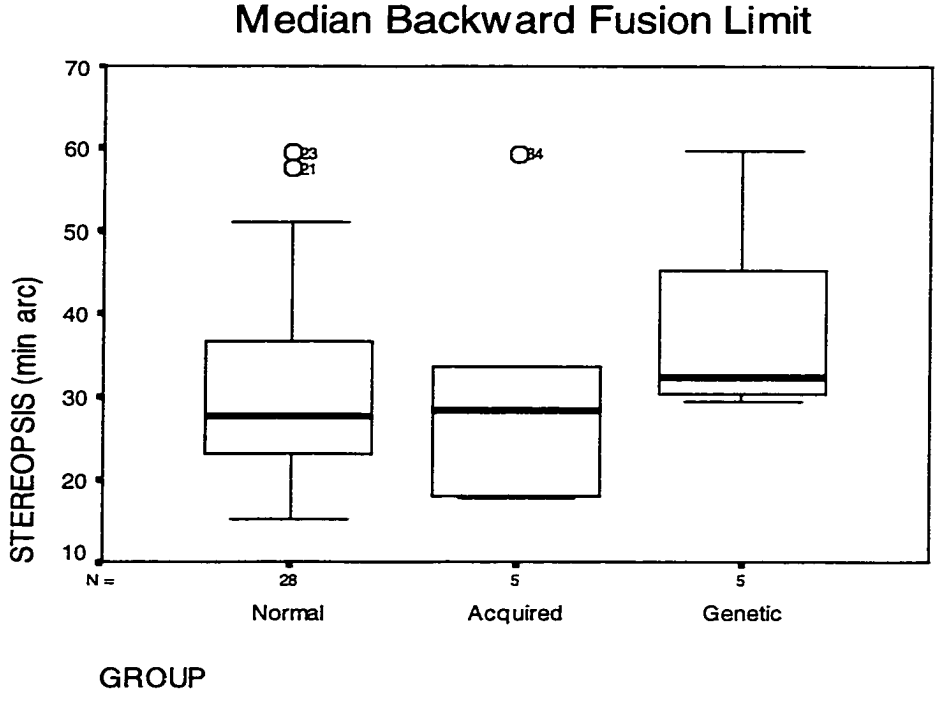


Figure B100. Box-and-whisker plot of backward fusion threshold for each group. As in Figure B1.

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Table B9. Monocular Hyperacuities: Vernier Acuity Thresholds (in min arc)

| Group | Normal | Acquired | Genetic |
|---|---|---|---|
| Mean Vernier Acuity | 0.38 | 0.47 | 0.30 |
| SEM | 0.03 | 0.09 | 0.05 |
| Median | 0.35 | 0.50 | 0.27 |

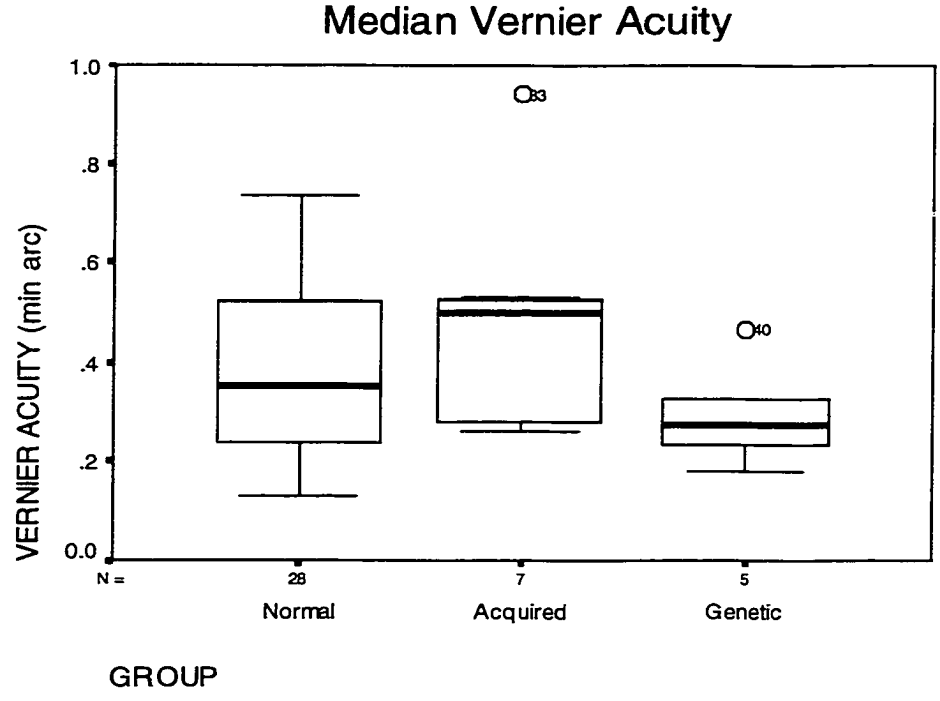


Figure B101. Box-and-whisker plot of vernier acuity for each group. As in Figure B1.

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Motion sensitivity. The results of the motion sensitivity experiment are presented in Table B10. The trend for genetic color-deficients having a superior minimum motion detection threshold compared to color-normals (who, in turn, are superior to acquired color-deficients) was maintained at the level of group medians, with genetics' sensitivities exceeding those of normals' and acquireds' by factors of 2 and 4, respectively (see Figure B102). Variability within groups was generally low, although overlap in range did occur.

Medians for maximum motion detection thresholds were just as discrepant as were the means. However, the genetic and acquired groups now trailed the normals by the same margin of 4 min arc (see Figure B103). Variability was moderate to large for each group, and entire group ranges were found to overlap one another.

Table B10. Motion Sensitivity: Minimum and Maximum Displacement Thresholds (in min arc)

| Group | Normal | Acquired | Genetic |
|---|---|---|---|
| Mean Minimum Motion Sensitivity ($d_{min}$) | 3.49 | 5.31 | 1.04 |
| SEM | 1.12 | 2.14 | 0.26 |
| Median | 1.56 | 3.16 | 0.87 |
| Mean Maximum Motion Sensitivity ($d_{max}$) | 24.65 | 20.74 | 24.18 |
| SEM | 1.16 | 1.82 | 3.82 |
| Median | 25.71 | 21.50 | 21.61 |

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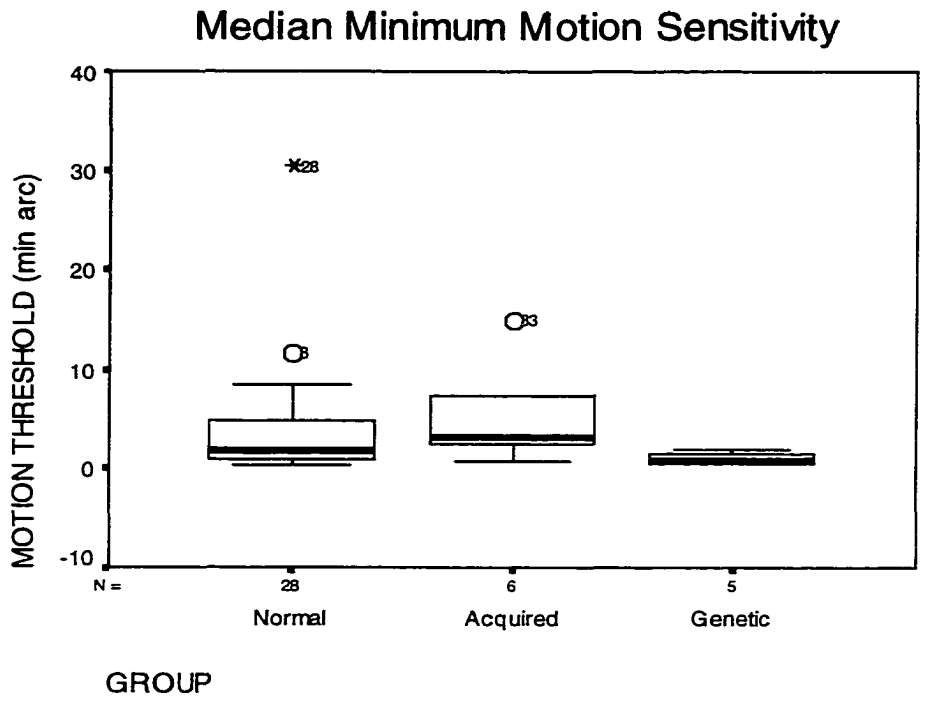


Figure B102. Box-and-whisker plot of minimum motion sensitivity threshold for each group. As in Figure B1.

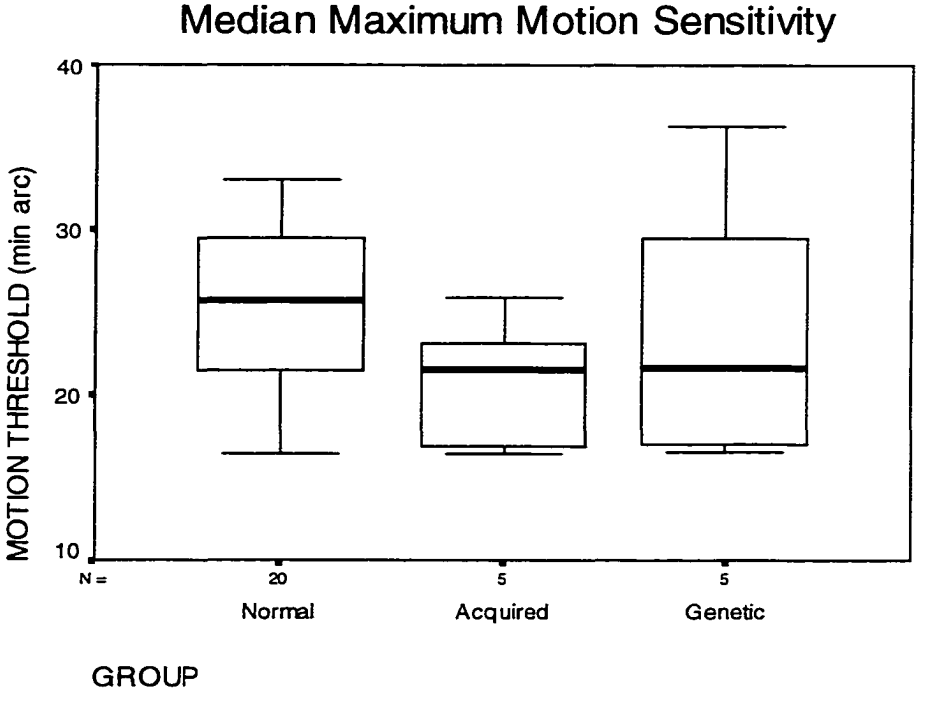


Figure B103. Box-and-whisker plot of maximum motion sensitivity threshold for each group. As in Figure B1.

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Ocular and dermal spectral reflectivity. Group mean (and median) spectral reflectances for iris and skin pigmentation can be found in Table B11 and in Figures B104-B107.

On average, genetic color-deficients' irises gave higher summed reflectances (i.e., tended to be lighter in color) than either color-normals or acquired color-deficients (see Figure B104). This was also found across group medians (see Figure B105). Variability was large within each group, and the ranges of values were found to overlap. Mean dermal summed reflectances were also greatest for the genetic group, while those for the normal and acquired groups were nearly the same (see Figure B106). This trend was also observed when comparing group medians, although the observed differences between groups were less marked compared to the means (see Figure B107). Variability was, again, high for each group, but the overlap in range was limited mostly to the color-normals and acquired color-deficients, as the lower limit of the genetic color-deficient

Table B11. Ocular and Dermal Spectral Reflectivity: Summed Reflectances

| Group | Normal | Acquired | Genetic |
|---|---|---|---|
| Mean reflectivity of iris | 12.23 | 11.40 | 15.94 |
| SEM | 1.25 | 2.06 | 1.82 |
| Median | 10.64 | 11.26 | 16.13 |
| Mean reflectivity of dermis | 76.49 | 76.68 | 92.63 |
| SEM | 3.39 | 6.76 | 5.94 |
| Median | 83.45 | 82.67 | 88.19 |

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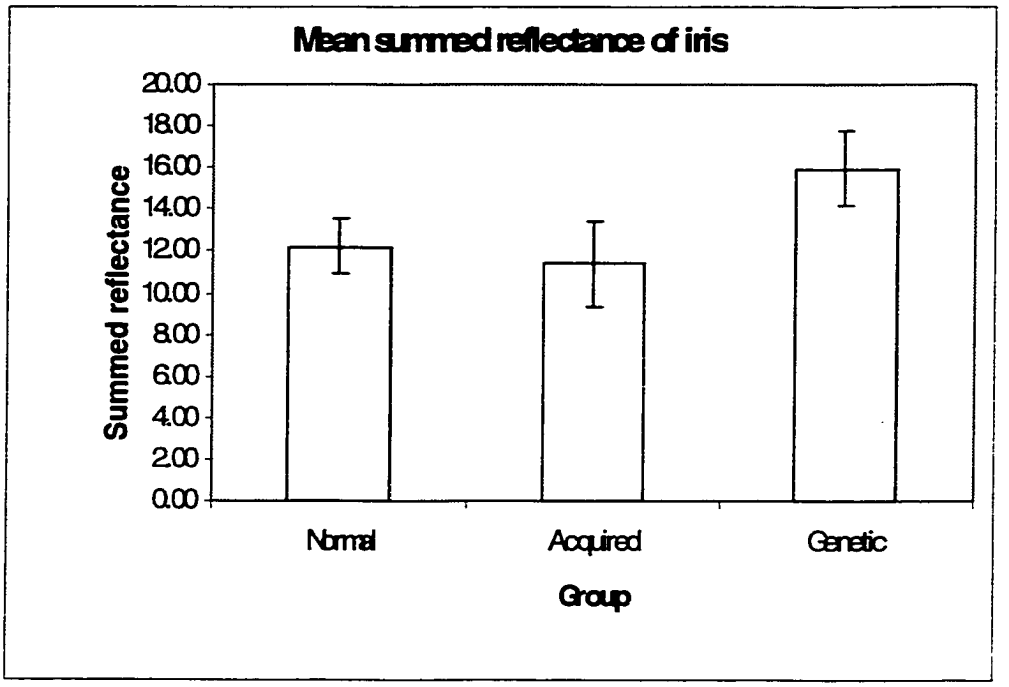


Figure B104. Mean summed reflectance of iris for each group. Error bars equal +/- 1 SEM.

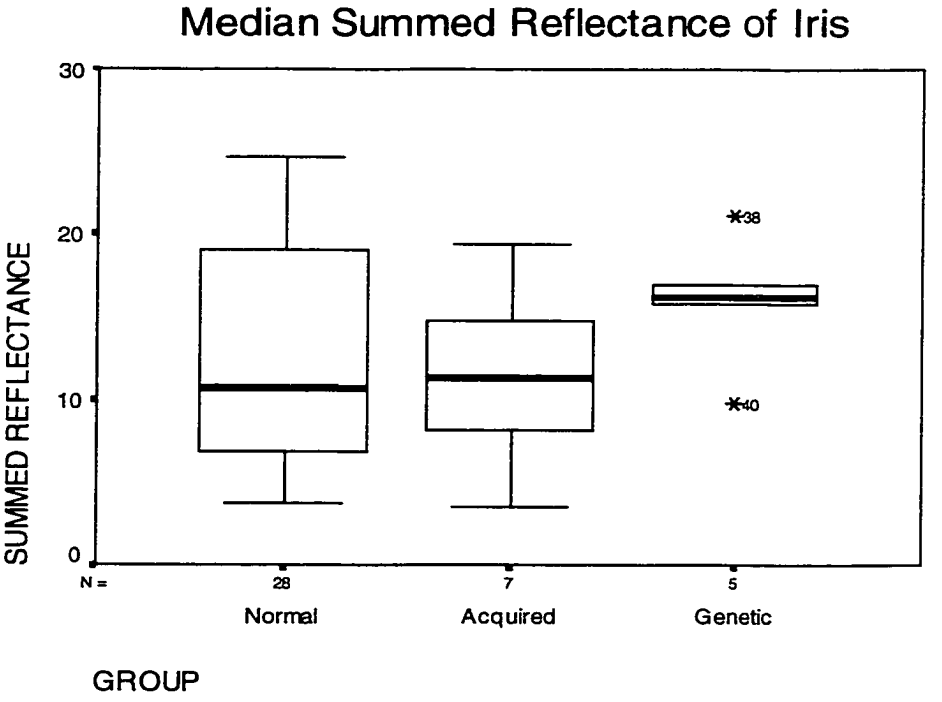


Figure B105. Box-and-whisker plot of summed reflectance of iris for each group. As in Figure B1.

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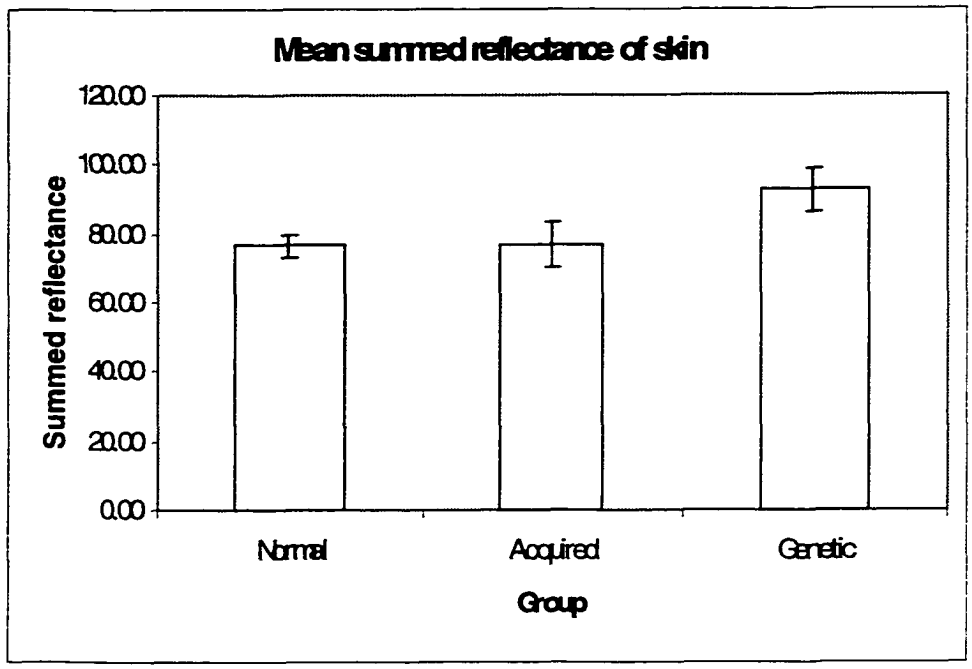


Figure B106. Mean summed reflectance of dermis for each group. Error bars equal +/- 1 SEM.

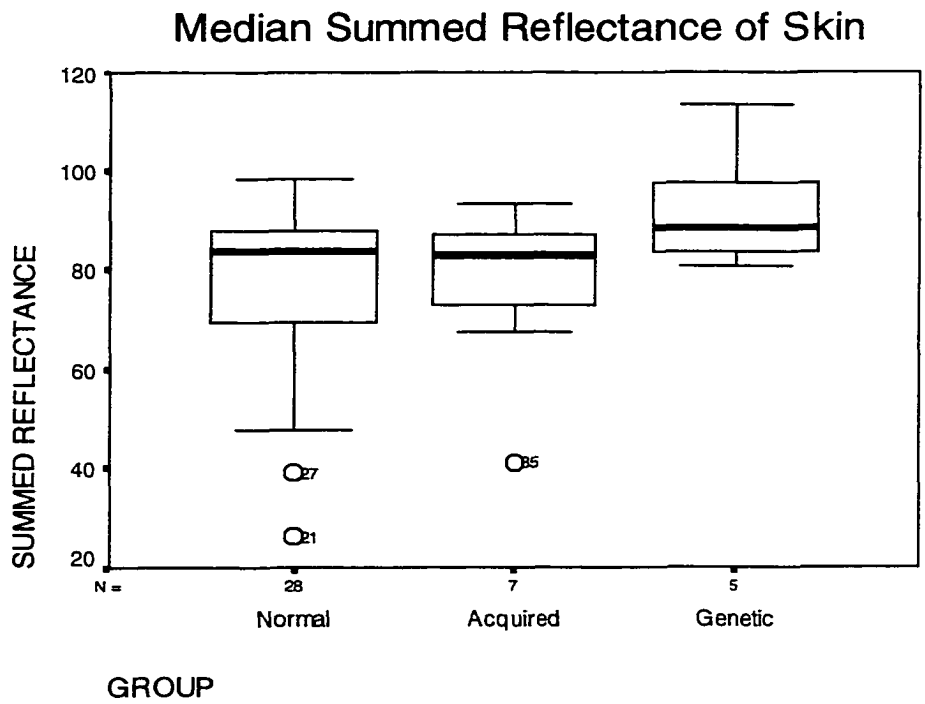


Figure B107. Box-and-whisker plot of summed reflectance of dermis for each group. As in Figure B1.

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group was closer to the normals' and acquireds' upper range limits. Results of one-way ANOVAs and Kruskal-Wallis tests revealed that neither of these variables showed significant group differences ($p > 0.05$).

One of our goals in this research was the use of spectral reflectivity of observers' iris and skin pigmentations as adjuvants to gain a fuller understanding of the underlying genetic transmission and heritability of color vision deficiencies. While there is great potential in determining which visual functions (or deficits in processing of visual information) correlate with varying degrees of pigmentation, we are currently unable to provide any information in addressing this issue. Ocular and dermal pigmentation is largely a function of an individual's racial and ethnic background. While we found there to be no significant differences in spectral reflectivities between our groups of observers, there was a definite overall trend toward lighter irises and skin tones among the genetic color-deficient group members (as seen in Table B11 and Figures B104-B107). (It should be mentioned that there was a significant correlation found between the reflectances of iris and dermis for the full sample, $r(38) = 0.61$, and for the normal group, $r(26) = 0.58$, both at the 0.01 level of significance. These figures still pale in comparison to that of the genetic group, $r(3) = 0.87$, which would certainly reach significance if this trend continues in additional observers.) Yet, to make any conclusions at this point based on the data obtained would be highly subject to the introduction of personal biases. Despite overlap across groups, our genetic color-deficients were found to be quite congruent regarding their genealogies. Each of the group members reported being of first- to third-generation middle- and/or eastern-European descent (including Czechoslovakia, Poland, Romania, Russia, and the Ukraine). With differences in the prevalence of color

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deficiencies according to the population statistics of individual countries and certain world regions known to exist, we need to accumulate data on observers from a more culturally-diverse sample in order to amass any evidence leading to results that are in any way conclusive.

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