The Spectral Sensitivity of Black and White

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White light consists of all wavelengths of visible light, however, our perception of white has a spectral tuning defined by the responses of the univariant cone photoreceptors. The neurons mediating white and how they combine the three cone types remains controversial. Here, we investigate the neural substrates for black and white through their contribution to the variation in perceived saturation with wavelength. From classic saturation experiments, we identify experimental conditions where black-white can be isolated from hue. This enables directly comparing the spectral sensitivity of black-white to the spectral signatures of retinal ganglion cells. Paradoxically, we find cone-opponent neurons can account for black-white sensations.

1. INTRODUCTION

Our experience of color can be defined by three parameters: hue, brightness and saturation. Some of the most fundamental questions in color vision research concern how these features relate to the physical attributes of incoming light and how they are represented by neurons within the visual system. Of these three parameters, saturation has been the most difficult to link to an underlying neural substrate.

Saturation is the perceived distance of a color from white, gray or black. For example, a monochromatic blue light appears very saturated, producing little or no white sensation. Between unique blue and equal energy white, the perceived color becomes increasingly desaturated. When matched for brightness, colored lights have two components: hue and white, with the relative strengths of these two components defining perceived saturation [1, 2].

The relative amounts of hue and white perceived in a color varies reliably as a function of wavelength. Trichromats universally agree that of the four fundamental hues (red, green, blue and yellow), yellow is the most desaturated (i.e. contains the most white). Meanwhile blue is the most saturated. Red is perceived as slightly less saturated than blue while green is perceived slightly more saturated than yellow.

This phenomenon is quantified in classic saturation experiments measured by scaling or threshold detection (**Figure 1A, 1B, 1D**). The results are interpreted as reflecting the relative activity of the neural substrates for hue and white as a function of wavelength. However, the different experimental paradigms for measuring saturation produce widely different results (**Figure 1A, 1B, 1D**) [3–6], indicating that they may be measuring different aspects of saturation. Moreover, though undoubtedly related to saturation, the results of some saturation experiments do not perfectly match our perceptual experience of saturation [3]. Here we revisit the underlying assumptions made by past models and we argue that the saturation experiment in **Figure 1A** reflects a measure of the spectral sensitivity of the neurons mediating black and white sensations.

Our analysis begins by accounting for the activities of hue and black-white underlying the threshold change in saturation. While the "first step from white" values plotted in **Figure 1A** do reflect a threshold change in saturation, because the participant adjusted the intensity of a spectral light until a just noticeable difference in hue occurred (see **Methods**), the neural substrates for hue are being held at a constant value. Thus, we argue that because the neural substrates for the hue component are held constant, *the saturation discrimination function reveals the spectral sensitivity of black and white sensations*.

This interpretation reveals an opportunity. The neural substrates for both hue and black-white remain highly controversial [7, 8] and psychophysical experiments measuring saturation, hue or black-white are often limited by the need to make some assumption regarding the neural substrates for one or more of these percepts. By identifying a situation where the neural substrates for hue are held constant and their effects can be discounted, the neural substrates mediating black-white can be studied in isolation, without assuming any specific color vision theory.

2. METHODS

A. Saturation experiments

Here we focus on a classic experimental paradigm for quantifying the wavelength dependence on saturation, measured as the "first step from white"[6] or the "chromatic threshold with respect to white"[3]. A white light is presented to the entire field

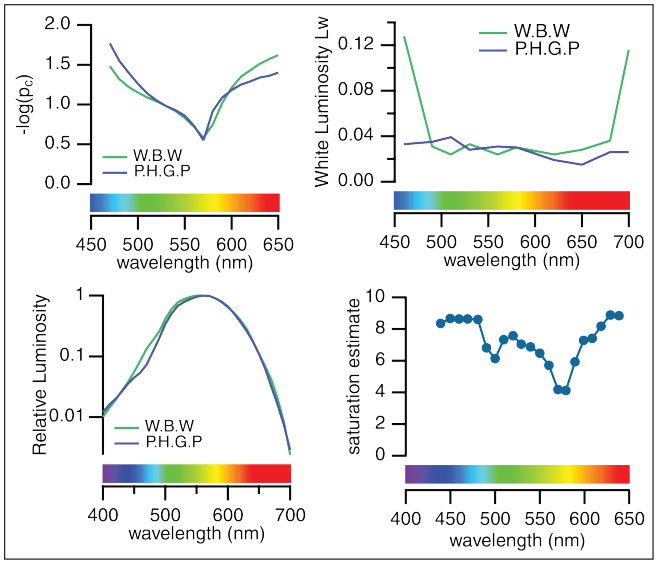

Fig. 1. Saturation and luminance measurements for two observers. **(A)** Saturation discrimination curves from Wright & Pitt (1937) [9]. The y axis is the negative log of the chromatic purity (p_c) of the minimum amount of a spectral light that is distinguishable from white (Eq. (1)). **(B)** First step from the spectrum from Wright & Pitt (1935) [10]. **(C)** Spectral luminosity functions from the same two observers, obtained from Wright & Pitt (1935) [10]. **(D)** Saturation scaling experiment from Jacobs (1967) [5].

while a monochromatic test light is added to just the circle in the center. The strength of the test light is increased until the hue is just noticeably different from the white light. In other words, until a just noticeable difference in saturation occurs. Importantly, the two lights are also matched for brightness. Then the least perceptible colorimetric purity (p_c) can be obtained by:

$$p_c = \frac{L_\lambda}{L_w + L_\lambda} \tag{1}$$

where L_λ is the intensity of the spectral light and L_w is the intensity of the white light. This metric allows expressing saturation independent of both the intensity of the white light and the wavelength of the test light. By convention, the results are plotted as the inverse log, $-log(p_c)$, so the y-axis reads out as a measure of saturation as a function of wavelength. In other words, **Figure 1A** can be read as saying the participant had to increase the intensity of the yellow 570 nm light far more than the intensity of the blue 470 nm light to produce a discriminable change in saturation. While the experiment measures a detection threshold and requires the neural substrates for hue to exceed some critical value, that critical value depends on the strength of the white percept. Thus, the paradigm has been called a saturation discrimination experiment and the results are called the saturation discrimination function.

The model focuses on the "first step from white paradigm", but also references two alternative experimental paradigms. The "first step from the spectrum" experiments in **Figure 1B** measure the minimal amount of white that must be added to a spectral light to produce a just-noticeable decrease in saturation [4, 6]. The saturation scaling paradigm in **Figure 1C** asks participants to rate the saturation of equiluminant spectral lights on a fixed scale [5, 11, 12]

B. Model

The design of classic saturation discrimination experiments did not require any assumptions about the underlying neural substrates. To determine whether midget ganglion cells can account for black-white sensations, we created a model achromatic channel that represents black and white sensations as the combined activity of OFF and ON cone-opponent midget ganglion cells, respectively. The activity of each spectral subtype of midget ganglion cell as a function of wavelength (λ) was calculated as a linear sum of the center and surround spectral sensitivities [13].

$$L_{on}(\lambda) = \alpha * L(\lambda) - (1-\alpha) * M(\lambda) \tag{2}$$

$$M_{on}(\lambda) = \beta * M(\lambda) - (1-\beta) * L(\lambda) \tag{3}$$

$$M_{off}(\lambda) = (1-\gamma) * L(\lambda) - \gamma * M(\lambda) \tag{4}$$

$$L_{off}(\lambda) = (1-\delta) * M(\lambda) - \delta * L(\lambda) \tag{5}$$

$$S_{off}(\lambda) = (1-\epsilon) * (L(\lambda) + M(\lambda)) - \epsilon * S(\lambda) \tag{6}$$

where L, M and S represent the L-, M- and S-cone spectral sensitivities, respectively, corrected for optical density and macular pigment. The Neitz photopigment template was used, assuming peak sensitivities of 559, 530 and 419 nm and optical densities of 0.3, 0.3 and 0.35 for the L-, M- and S-cones, respectively [13]. The center-surround balance is represented by α, β, γ, δ and ϵ for M-ON, L-ON, M-OFF, L-OFF and S-OFF midget ganglion cells, respectively. While these equations appear to maximize spectral opponency, the presence of, for example, L-cones in the surround of the L-OFF midget ganglion cell is accounted for by decreasing the α parameter.

Perceived saturation, Y, as a function of wavelength, λ, was then calculated as the OFF midget ganglion cell activity subtracted from the ON midget ganglion cell activity:

$$Y(\lambda) = \theta * (\rho * f(L_{on} + M_{on}) - \phi * f(L_{off} + M_{off}) \tag{7}$$

where ρ, ϕ and ω represent the gains of the L/M-ON, L/M-OFF and S-OFF midget ganglion cells, respectively. Separate gains for the S-OFF midget ganglion cell and L/M-OFF midget ganglion cells were used to account for both the lower density of S-cones and the possibility for their signals to be amplified downstream [14, 15]. To simplify fitting, the L/M-ON gain, β, was set to 1. Each ganglion cell's response was half-wave rectified, represented by $f()$. The resulting curve was scaled by θ and translated along the y-axis by y_0 to align with the minima of the saturation discrimination data.

A second model based only on L/M-cone midget ganglion cells omitted the S-OFF midget ganglion cell terms.

$$Y(\lambda) = \theta * (\rho * f(L_{on} + M_{on}) - \phi * f(L_{off} + M_{off}) - \omega * f(S_{off})) \tag{8}$$

The models were fit to published saturation discrimination curve data listed in tables [1, 3, 9, 16] in MATLAB (Mathworks) using the `lsqcurvefit` function. The gain of the ON channel was fixed to an arbitrary value of 1 while the OFF channel gain(s), σ and ω, were allowed to vary. The models with and without the S-OFF midget ganglion cell had eight and six free parameters, respectively. The model code will be made publicly available upon publication at https://github.com/neitzlab/SaturationModels.

C. Serial Electron Microscopy

The preparation, processing and annotation of the serial electron microscopy (EM) volume are described here briefly and in

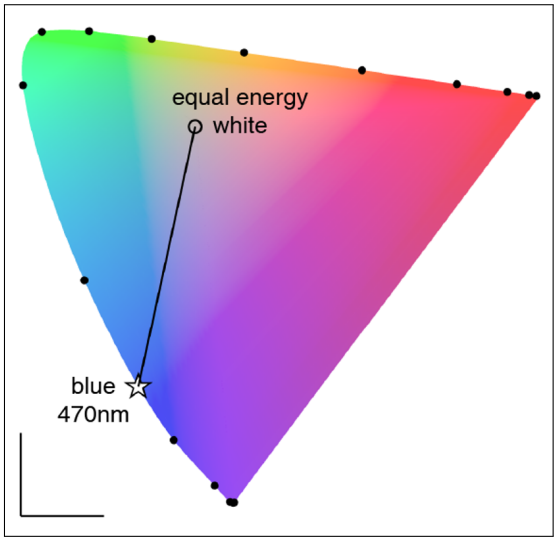

Fig. 2. Percepts of white vary in an equiluminant plane. The chromaticity coordinates for a blue 470 nm light and an equal energy white light, plotted on a 1976 CIE diagram. If the neural substrates mediating black-white percepts had spectral sensitivities matching $V(\lambda)$, all points in the equiluminant CIE diagram would appear equally white.

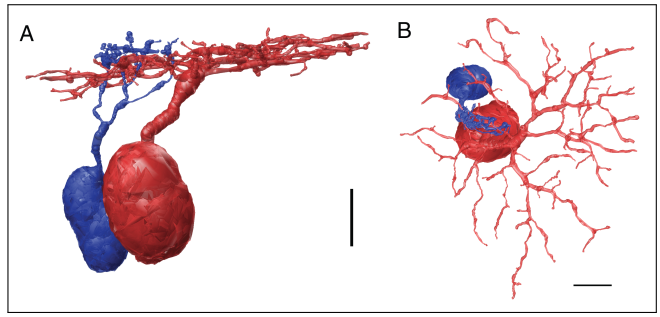

Fig. 3. Two views of the dendritic fields of neighboring OFF midget (blue) and parasol (red) ganglion cells in the macaque central retina. Note the edge of the volume runs along the left side of **B**, so the parasol reconstruction is not complete. Scale bars are 10 microns.

depth in our previous work [17]. An eye was obtained from a terminally anesthetized adult male macaque (*Macaca nemestrina*) through the Tissue Distribution Program at the Washington National Primate Research Center. All procedures were approved by the Institutional Animal Care and Use Committee at the University of Washington. A block of inferior parafoveal retinal tissue at ∼1 mm eccentricity from the foveal center was imaged using a Zeiss Sigma VP field emission scanning electron microscope equipped with a 3View system and sectioned in the horizontal plane. In each 90 nm section, an area approximately 200 microns on a side was imaged as a 5 X 5 montage at a resolution of 7.5 nm/pixel. At ∼1 mm eccentricity, the displacement of ganglion cells from cone pedicles was minimized while still remaining in a region where most midget ganglion cells received direct input from a single cone.

The volume contained 1893 horizontal sections spanning from the ganglion cell layer through the cone pedicles. Image registration was performed using Nornir (https://nornir.github.io). Annotations were performed using Viking, a web-based, multiuser software developed in the Marc and Jones labs (https://connectomes.utah.edu)[18]. Processes were annotated by placing a circular disc spanning the entire process at the center of mass and linking it to annotations on neighboring sections. Figures were created using SBFSEM-tools, an open source 3D visualization toolbox for connectomics data developed in the Neitz lab (https://www.github.com/neitzlab/sbfsem-tools)[17]. The 3D reconstructions will be made publicly available upon publication at https://www.github.com/neitzlab/SaturationModels.

3. RESULTS

A. Defining the "achromatic" channel

Hurvich & Jameson were the first to model saturation as an interaction between chromatic and achromatic channels defined by specific transformations of the cone photoreceptors. Their theory was based on the assumption that saturation represents the ratio of activity in the independent chromatic and achromatic channels, or hue and black-white [19, 20]. In their opponent process theory, Hurvich & Jameson's achromatic channel refers to black, white and gray, or the absence of a hue sensations. However, for vision scientists, "chromatic" neurons are spectrally-opponent and compare the activities of different cone types. "Achromatic" neurons are assumed to be spectrally non-opponent, summing the responses of different cone types, and cannot distinguish changes in wavelength from changes in intensity.

Accordingly, Hurvich & Jameson (1955) assumed the mechanisms mediating black-white percepts (their "achromatic channel") matched $V(\lambda)$, the spectral luminosity function. Thus, "achromatic" has often been equated with luminance, $V(\lambda)$, L+M-cone activity, and later, the activity of non-spectrally-opponent neurons, particularly the parasol ganglion cells ([21] but see [22]). Interestingly, there are other $L+M$ ganglion cells in the primate retina with similar response properties to parasol ganglion cells [23] and the responses of $L-M$ midget ganglion cells could also explain $V(\lambda)$ [24].

Saturation has proved to be difficult to account for by models assuming black and white percepts are mediated by neurons with spectral sensitivities matching $V(\lambda)$. **Figure 1A** and **Figure 1C** shows the saturation discrimination and luminosity functions for the same two observers [9, 10]. The saturation discrimination function reliably diverges from $V(\lambda)$ in two key features. The minima in saturation occurs in a sharp peak at about 570nm while $V(\lambda)$ has a broad peak around 555 nm. Second, $V(\lambda)$ predicts long-wavelength reds will be the most saturated, while the saturation discrimination function shows it is indeed short-wavelength blues that are the most saturated.

Figure 2 shows a simple demonstration of the independence of luminance and black-white percepts. A CIE diagram is an equiluminant plane, meaning that all points in the plane should elicit equal activity in parasol ganglion cells and other neurons with spectral sensitivities matching $V(\lambda)$. Thus, if $V(\lambda)$ were responsible for white percepts, all points in this plane should appear equally white. Yet between equal energy white and unique blue, there is a change in percept of white. In other words, a change in white without a change in luminance, or the activities of neurons with spectral sensitivities matching $V(\lambda)$.

This distinction is well summarized by a review on luminance by Lennie and colleagues (1993) : "V(λ) is a useful and well-established standard for photometry, but beyond that its significance for visual function is unclear" [24]. Indeed, the

standard methods for measuring $V(\lambda)$, heterochromatic flicker and the minimum border technique, do not involve judgements about the perceived whiteness or blackness of the stimuli. Moreover, experiments measuring perceived whiteness and blackness match the saturation discrimination function in **Figure 1A**, not $V(\lambda)$ [25]. Taken together, it is clear that the spectral tuning and underlying neural substrates for black-white are distinct from those typically associated with luminance and $V(\lambda)$.

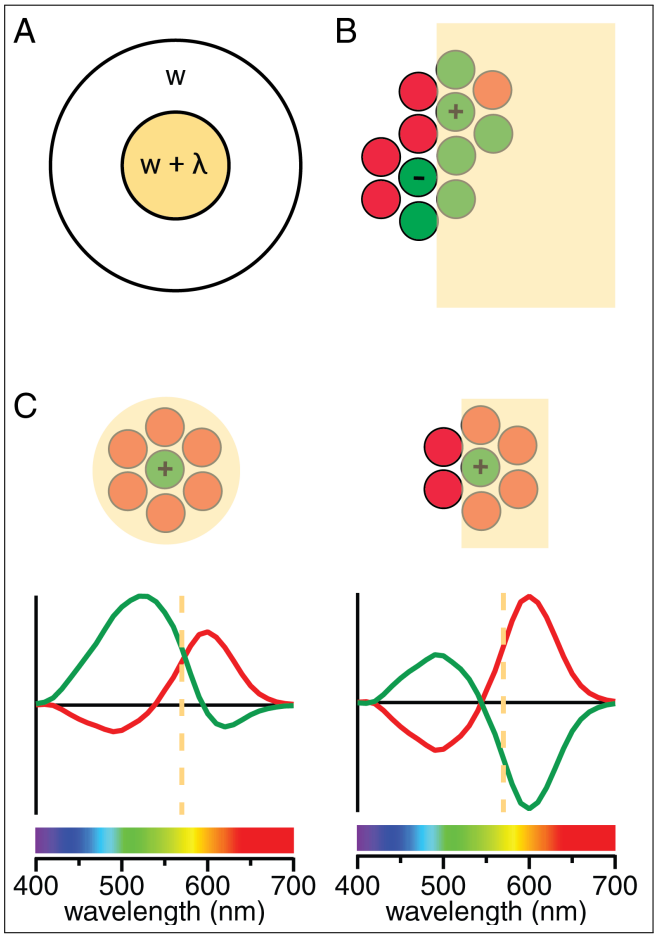

Fig. 4. (A) Example saturation discrimination experiment in which a monochromatic spectral light (λ) is added to a full-field white background (w) until just noticeably different from white (i.e. a threshold change in saturation or hue). **(B)** L- and M-cone inputs to midget ganglion cells responsible for detecting the edge of the test light. An ON-center cell responds best at the inner edge of the test light while an OFF-center cell responds best outside the test stimulus. Note that the model also includes a midget ganglion cell of the opposite polarity at each center cone. **(C)** Top: Illustration of full-field and edge stimuli superimposed on the receptive field of a sample M-ON midget ganglion cell. Bottom: L-ON and M-ON midget ganglion cell activity for two types of stimuli. Yellow dashed line at 570 nm.

B. Can midget ganglion cells account for black-white sensations?

Increasing evidence supports the idea that midget ganglion cells mediate spatial vision (reviewed by Lennie & Movshon (2005) [22]). We next tested whether black and white sensations can be explained by spectrally opponent midget ganglion cells. Each L- and M-cone photoreceptor in the central retina is represented twice in the retinal output, by both an ON and OFF midget ganglion cell [26]. The single cone makes up the midget ganglion cell's center receptive field, while horizontal cell feedback from neighboring cones forms the surround. This center-surround receptive field structure is key to the midget ganglion cell's role in spatial vision and is ideal for edge detection computations [8, 27]. This is important because black and white sensations are defined by the changes in intensity that occur at edges.

The size of a neuron's receptive field defines their spatial resolution. During the course of a larger series of serial electron microscopy experiments in the macaque central retina, we reconstructed a neighboring OFF parasol and OFF midget ganglion cell (**Figure 3**). Although their dendritic field sizes and density are well-established [28, 29], previous methods have not independently labeled neighboring cells for direct comparison, as shown in **Figure 3**. We include these data to further emphasize the disparity in their dendritic field sizes, which serves as a proxy for the center receptive field size. The OFF midget ganglion cell received input from a single cone via an OFF midget bipolar cell. Midget ganglion cells are the only neurons with the necessary density to mediate our high resolution, black-white vision [30–32].

However, given their classification as "chromatic" neurons, midget ganglion cells have long seemed an unlikely substrate for black and white sensations. In trichromats, the difference in spectral tuning of the center and surround receptive fields, such as those in **Figure 4B**, results in spectral opponency [33]. The midget ganglion cell's spectral tuning is often thought of as an detriment to edge detection because it enables midget ganglion cells to detect not only edges defined by intensity, but also by changes in spectral contrast (reviewed by Patterson, Neitz, Neitz (2019)[8]). From a single midget ganglion cell's binary spike output, it is impossible for a downstream neuron to distinguish whether the cell responded to a black-white edge or some chromatic-white edge. Thus, spectral sensitivity of midget ganglion cells defines the range of chromatic edges that a spectrally opponent neuron will confuse with black or white. This should be apparent as "distortions" in our sensations of white [25], relative to our intuition that white light is equal across the visual spectrum. It follows that *if spectrally opponent midget ganglion cells are indeed responsible for black-white percepts, the unintended consequences of their spectral tuning should be apparent in our perception of color saturation.* In other words, the sensitivity of midget ganglion cells to chromatic edges may be the neural mechanism responsible for the striking variations in white and saturation with wavelength.

C. Accounting for the spatial aspects of the stimulus and the midget ganglion cell's receptive field

Models of midget ganglion cell activity that include both their spectral and spatial opponency are rare. In particular, the effects of spatial opponency on midget ganglion cell spectral tuning is often overlooked. If only spectral opponency is considered, white sensations mediated by L-ON and M-ON midget ganglion cells do not seem to account for the saturation discrimination curve (**Figure 4A**). If 570 nm lights appear the most desaturated, then the neural substrates for white should be responding maximally at this wavelength. However, the approach in **Figure 4A** considers only the spectral tuning of cone inputs to midget ganglion cells.

To be successful, the model must also account for the spatial arrangement of those cone inputs, as well as the spatial aspects

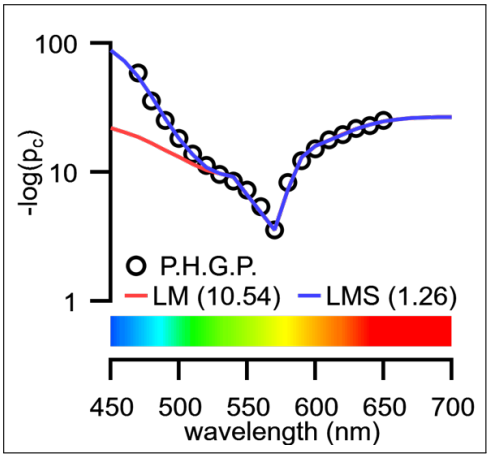

Fig. 5. Midget ganglion cell models with (blue curve) and without (red curve) the S OFF midget ganglion cell contribution fit to the saturation discrimination data for one observer, P.H.G.P., from Wright & Pitt (1937) [9], with the root mean square error shown in parentheses.

of the stimulus. Black and white sensations are defined by the changes in intensity that occur at edges. Similarly, hue sensations are defined by the changes in spectral contrast at object boundaries. Thus, the saturation of the test light is defined by the receptive fields of neurons located along an object's boundaries and the rest is handled by "filling in" at the level of the cortex [34]. If the receptive field is located at an edge and only some of the surround is stimulated, the contributions of both types now could sum to signal white at 570 nm (**Figure 4B**).

D. Model

To systematically investigate the effect is **Figure 4A**, we created a model "achromatic channel" that represents the spectral sensitivity of black-white sensations as the combined activity of ON and OFF midget ganglion cells (see **Methods**). Briefly, the spectral tuning of each midget ganglion cell subtype was modeled as a weighted linear sum of the center and surround spectral sensitivities. For example, in Eq. (2), the weight parameter α represents the balance between L-cone in the center and M-cones in the surround. This parameter can be varied to account for the edge condition (**Figure 4C**).

While this equation appears to maximize spectral opponency, the presence of M-cones in the surround receptive field can be accounted for by decreasing alpha, which decreases the strength of the center M-cone. This term also reduces the number of assumptions we need to make about crucial unknown variables, such as the relative strength of the center and surround receptive fields and the participant's L:M-cone ratio. Similar equations are used for the L-ON, L-OFF and M-OFF midget ganglion cells and their outputs were rectified to simulate the nonlinearity imposed by the spike threshold of a ganglion cell. By combining the equations for each spectral subtype of midget ganglion cell, we calculated saturation as a function of wavelength by subtracting the activity of the OFF cells mediating black from the ON cells mediating percepts of white.

The results align with the demonstration in **Figure 4**. L vs. M cone-opponency provides a clear explanation for the sharp minimum at 570nm. However, the model fails to account for the slope of increasing saturation for short-wavelength lights. This model would predict blue and red would be equally saturated. To account for the short-wavelength component of the saturation discrimination curve requires contribution from S-cones.

E. S-OFF midget ganglion cells

There have been reports of a rare S-cone OFF midget ganglion cell subtype that could bridge this gap. However, there has been considerable controversy regarding the existence of S-OFF midget ganglion cells and these neurons have not been incorporated into models of visual perception. However, there has also been considerable variability in the methods, species and eccentricities involved. We recently provided both anatomical and physiological confirmation of the existence of S-OFF midget ganglion cells in the primate central retina [17]. Briefly, we confirmed a single OFF midget bipolar cell contacted each S-cone, which in turn, contacted a single OFF midget ganglion cell. We recorded from S-OFF midget ganglion cells in the central retina and confirmed that, like the more common L vs. M-cone midget ganglion cells, S-OFF midgets have a center-surround receptive field consistent with a role in spatial vision.

We added an S-OFF midget ganglion cell term to the saturation model (Eq. (6), Eq. (8)). The S-OFF term was given a separate gain control from the L-OFF and M-OFF to account for the sparsity of S-cones and the potential for their signals to be amplified downstream [14, 15]. Including the S-OFF midget ganglion cell allowed our model to fit the short-wavelength region of the saturation discrimination curve. In fact, our model provided excellent fits to many saturation discrimination datasets (**Figure 6**) [1, 3, 9, 35]. This result is consistent with black and white sensations mediated by midget ganglion cells, and surprisingly, a potential role for S-OFF midget ganglion cells in signaling percepts of blackness.

4. DISCUSSION

Here we have argued that the saturation discrimination functoin reflects conditions where sensations of black-white can be isolated from hue and from controversial assumptions on the neural substrates of hue. Second, we incorporate the spatial aspects of the stimulus and midget ganglion cell receptive fields into our spectral model. This allowed us to compare the spectral tuning of black-white to the spectral sensitivities of parasol and midget ganglion cells. While our results are not causal evidence for midget ganglion cells mediating black-white sensations, we establish L vs. M and S - LM cone-opponent neurons provide the most accurate account of the neural substrates for black-white percepts and their contributions to saturation.

A. Interpreting the saturation discrimination experiment

The logic used to interpret the results of saturation experiments is as follows. The saturation of colored light depends not only on the strength of the hue response, but also the strength of the white response. If saturation is the relative portions of hue and white in a monochromatic light, then saturation experiments should measure how the relative amounts of hue and white vary as a function of wavelength. These variations as a function of wavelength reflect the spectral sensitivities of the neurons mediating hue and black-white sensations. It follows that saturation discrimination experiments can be interpreted as *a comparison between the spectral sensitivities of the neural substrates mediating our percepts of hue and white*.

This interpretation leads to the idea proposed by Hurvich & Jameson's model: saturation can be represented by the ratio of activity in the neural substrates for hue and black-white [20].

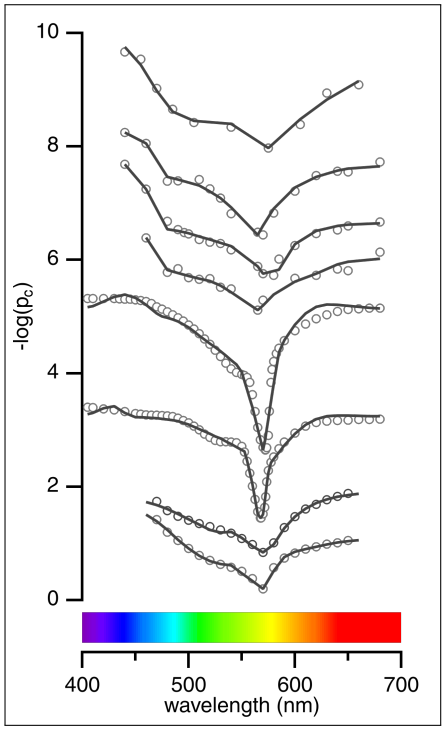

Fig. 6. Model fits (green) to classic saturation discrimination datasets. From top to bottom: D.P. from Purdy (1931) [3], W.J.M., L.C.M. and F.L.W., from Martin et al (1933) [1], I.G.P. and F.G.B. from Priest & Brickwedde (1938) [35], W.D.W. and P.H.G.P. from Wright & Pitt (1937) [9].

Their model was designed to find the chromatic and achromatic channel responses necessary to give a just-noticeable change in saturation (set as a constant saturation ratio of 0.1, relative to 0 for a completely desaturated white light) [36]. Like our model, they assumed that a threshold change in saturation is correlated with a constant visual effect. However, our model decomposed the threshold change in saturation into its two components, hue and white. This led to the insight that in **Figure 1A**, Thus, **Figure 1A** can be interpeted as a situation where hue is held constant and only black-white sensations vary.

B. Different saturation experiment paradigms measure distinct aspects of saturation.

The idea that the different saturation experiments in **Figure 1A, 1B, 1D** might be measuring different aspects of saturation is relatively unexplored. In an early study, Purdy (1931) concluded the "first step from the spectrum" results are closely related to saturation, but do not completely match our perceptual experience of saturation [3]. Here, we reason they match measurements of black-white instead [25].

The most accurate experiment for measuring our perceptual experience of saturation may be the results of scaling experiments like **Figure 1D** because these tasks involve true judgements of perceived saturation. Most group the results of the "first step from white" and scaling experiments together as "generally uniform" [19]. However, there are clear, reliable differences. The scaling experiments capture both the desaturation of yellows (570-580 nm) and cyans (490-500nm), whereas **Figure 1A** emphasizes only the desaturation around 570nm. We propose that, unlike the "first step from white" experiments, scaling experiments engage both the neural substrates for hue and black-white simultaneously. Thus, a minima in saturation can occur because the white response is relatively strong, or because the hue response is relatively weak. Comparison with opponent response functions [20] indicate the latter is the case for the 490-500 nm region. Hurvich and Jameson's model fits scaling data best because their model of the threshold change in saturation involves the activities of both hue and black-white.

Our approach may also be able to explain the striking differences in saturation measured as the first step from spectrum (**Figure 1B**) [3, 4, 6] as a situation where black-white sensations are held constant and only the neural substrates of hue are predicted to vary. Thus, this experimental paradigm measures a distinct process (the neural substrates of hue) and would not be expected to match the results in **Figure 1A** and **1D**. Although future experiments sampling more wavelengths are necessary, this idea could explain changes in the results of the "first step from the spectrum" experiment reported by Yeh and colleagues (1993) at very low light levels [6].

Finally, saturation is not only the perceived distance from white, but also black and grey. Saturation experiments measuring perceived distance from white were the focus of this work because they are the most straightforward to perform and interpret. As detailed above, our sensations of black and white are defined by the changes in intensity that occur at edges. Because white percepts are relative, rather than absolute, the neural substrates for black are engaged even in the "first step from white" experiments. That being said, certain measurements of induced blackness and grayness diverge from $V(\lambda)$ [37] and resemble the saturation discrimination function [38, 39], suggesting the analysis presented here could be applicable.

C. Black-white sensations can be accounted for by spectrally-opponent midget ganglion cells

Our model demonstrated that L vs. M cone opponency can explain the sharp minima in saturation around 570 nm. Additional support for the idea that cone-opponent neurons mediate black-white can be seen in the saturation discrimination functions of dichromats [16, 40–42]. The effect of a missing L-, M- or S-cone on the spectral luminosity function, $V(\lambda)$ is predictable - the peak shifts towards the remaining long-wavelength cone type (**Figure 7B**). However, the effect of dichromacy is more complex for saturation discrimination functions. As shown in **Figure 7A**, the sharp decrease in saturation around 570 nm is absent for protanopes and deuteranopes, but remains desaturated for tritanopes (**Figure 7D**) [41]. Interestingly, for deuteranopes and protanopes, the saturation discrimination functions are closer to matching the physical nature of white (i.e. equal sensitivity at all wavelengths) than the spectral luminosity functions.

Surprisingly, S-OFF midget ganglion cell contribution was essential to fully explain the saturation discrimination function. The idea that S-OFF midget ganglion cells contribute to sensations of blackness is surprising, but consistent with the receptive field properties of S-OFF midget ganglion cells in the central retina [17]. Evidence for blue cone contribution to edge and form vision [43]. Though S-OFF midget ganglion cells are far rarer than L/M-OFF midget ganglion cells, their contribution may be stronger because there is no opposing response from S-ON midget ganglion cells. An interesting avenue for future research will be to explore whether the S-OFF midget ganglion cells can explain any of the well-documented asymmetries in the S-cone ON and OFF pathways.

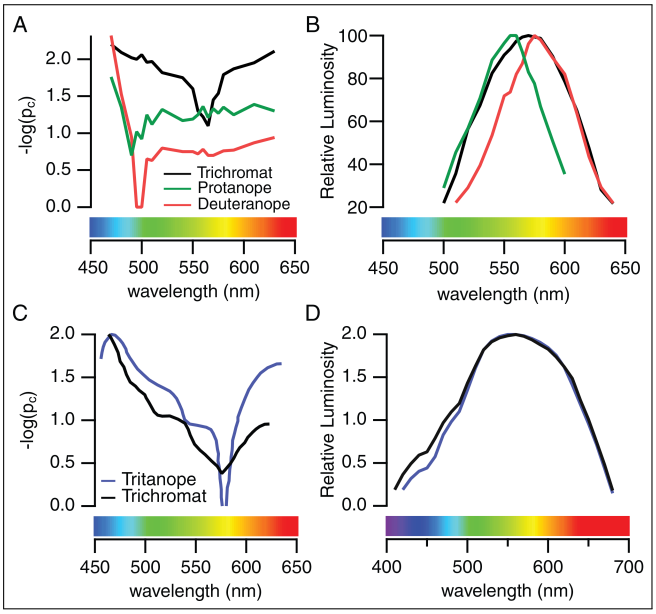

Fig. 7. (A) Saturation discrimination and **(B)** spectral luminosity functions for trichromatic (black), deuteranopic (red) and protanopic (green) observers, from Chapanis (1944) [16]. **(C)** Saturation discrimination function for a tritanope, from Cole (1965)[41]. **(D)** Average spectral luminosity functions for seven tritanopes compared to a trichromatic observer obtained under the same conditions, from Wright (1952) [42].

D. Parasol ganglion cells are not necessary to explain black-white percepts

We find black-white percepts can be explained entirely by midget ganglion cells. While we cannot rule out minimal parasol ganglion cell contribution at some level, the responses of $L + M$ neurons are ill-suited for mediating the elementary sensations of black-white. As demonstrated in **Figure 1C** and **2**, the correlation between parasol ganglion cell responses and $V(\lambda)$ is a separate issue distinct from sensations of black and white [21].

There is increasing evidence for and acceptance of the idea that midget ganglion cells mediate spatial vision, the high-resolution edge detection that defines our percepts of black and white [31, 32, 44–46]. An open question is what the function of parasol ganglion cells could be, if not spatial vision. One possibility is that parasol ganglion cells may be better suited for motion than high resolution spatial vision. Lesions of the magnocellular pathway do not alter spatial vision but do impair motion detection [47, 48]. The speed and trajectory of a moving object can be decoded from the responses of a population of parasol ganglion cells [49] and we recently demonstrated motion sensitivity in individual parasol ganglion cells [50].

5. CONCLUSIONS

In a classic study on saturation discrimination, Purdy (1931) remarked, "No theory of color vision, however, can be regarded as adequate if it does not account for the laws of saturation as well as those of hue."[3]. We demonstrate that an black and white percepts mediated by spectrally opponent midget ganglion cells provides the most accurate account to date of the physiological mechanisms underlying saturation.

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8. DISCLOSURES

The authors declare no conflicts of interest.

REFERENCES

1. L. C. Martin, F. L. Warburton, and W. J. Morgan, "Determination of the sensitiveness of the eye to differences in the saturation of colours," Gt. Britian Med. Res. Counc. Special Rep. Ser. **188**, 1–41 (1933).
2. S. Hecht and S. Shlaer, "The color vision of dichromats II. Saturation as the basis for wavelength discrimination and color mixture," J. Gen. Physiol. **20**, 83–93 (1935).
3. D. Purdy, "On the saturations and chromatic thresholds of the spectral colours," Br. J. Psychol. **21**, 283–313 (1931).
4. P. K. Kaiser, J. P. Comerford, and D. M. Bodinger, "Saturation of spectral lights," J. Opt. Soc. Am. **66**, 818–826 (1976).
5. G. H. Jacobs, "Saturation estimates and chromatic adaptation," Percept. & Psychophys. **2**, 271–274 (1967).
6. T. Yeh, J. Pokorny, and V. C. Smith, "Chromatic discrimination with variation in chromaticity and luminance: Data and theory," Vis. Res. **33**, 1835–1845 (1993).
7. J. Neitz and M. Neitz, "Evolution of the circuitry for conscious color vision in primates," Eye. **31**, 286–300 (2016).
8. S. S. Patterson, M. Neitz, and J. Neitz, "Reconciling Color Vision Models With Midget Ganglion Cell Receptive Fields," Front. Neurosci. **13**, 865 (2019).
9. W. D. Wright and F. H. G. Pitt, "The saturation-discrimination of two trichromats," The Proc. Phys. Soc. **49**, 22 (1937).
10. W. D. Wright and F. H. G. Pitt, "The colour-vision characteristics of two trichromats," Proc. Phys. Soc. **47**, 205–217 (1935).
11. K. Fuld, "The contribution of chromatic and achromatic valence to spectral saturation," Vis. Res. **31**, 237–246 (1991).
12. J. Gordon, I. Abramov, and H. Chan, "Describing color appearance: Hue and saturation scaling," Percept. & Psychophys. **56**, 27–41 (1994).
13. B. P. Schmidt, M. Neitz, and J. Neitz, "Neurobiological hypothesis of color appearance and hue perception." J. Opt. Soc. Am. A, Opt. image science, vision **31**, 195–207 (2014).
14. A. Roorda and D. R. Williams, "The arrangement of the three cone classes in the living human eye," Nature **397**, 520 – 522 (1999).
15. R. L. De Valois, N. P. Cottaris, S. D. Elfar, L. E. Mahon, and J. A. Wilson, "Some transformations of color information from lateral geniculate nucleus to striate cortex." Proc. Natl. Acad. Sci. United States Am. **97**, 4997–5002 (2000).
16. A. Chapanis, "Spectral saturation and its relation to color-vision defects," J. Exp. Psychol. **34**, 24–44 (1944).
17. S. S. Patterson, J. A. Kuchenbecker, J. R. Anderson, A. S. Bordt, D. W. Marshak, M. Neitz, and J. Neitz, "An S-cone circuit for edge detection in the primate retina," Sci. Reports **9**, 11913 (2019).
18. J. R. Anderson, S. Mohammed, B. Grimm, B. W. Jones, P. Koshevoy, T. Tasdizen, R. Whitaker, and R. E. Marc, "The Viking viewer for connectomics: Scalable multi-user annotation and summarization of large volume data sets," J. Microsc. **241**, 13–28 (2011).

19. D. Jameson and L. M. Hurvich, “Some Quantitative Aspects of an Opponent-Colors Theory. I. Chromatic Responses and Spectral Saturation,” J. Opt. Soc. Am. **45**, 546–552 (1955).
20. L. M. Hurvich and D. Jameson, “An opponent-process theory of color vision,” Psychol. Rev. **64**, 384–404 (1957).
21. B. Lee, P. Martin, and A. Valberg, “The physiological basis of heterochromatic flicker photometry,” J. Physiol. pp. 323–347 (1988).
22. P. Lennie and J. A. Movshon, “Coding of color and form in the geniculostriate visual pathway (invited review),” J. Opt. Soc. Am. A **22**, 2013–2033 (2005).
23. J. D. Crook, B. B. Peterson, O. S. Packer, F. R. Robinson, P. D. Gamlin, J. B. Troy, and D. M. Dacey, “The smooth monostratified ganglion cell: evidence for spatial diversity in the Y-cell pathway to the lateral geniculate nucleus and superior colliculus in the macaque monkey.” The J. Neurosci. **28**, 12654–12671 (2008).
24. P. Lennie, J. Pokorny, and V. C. Smith, “Luminance,” J. Opt. Soc. Am. A **10**, 1283–1293 (1993).
25. K. Fuld, T. A. Otto, and C. W. Slade, “Spectral responsivity of the white–black channel,” J. Opt. Soc. Am. A **3**, 1182 (1986).
26. H. Kolb and D. Marshak, “The midget pathways of the primate retina,” Documenta Ophthalmol. **106**, 67–81 (2003).
27. D. Marr and E. Hildreth, “Theory of edge detection,” Proc. Royal Soc. Lond. Ser. B **207**, 187–217 (1980).
28. V. H. Perry, R. Ohler, and A. Cowey, “Retinal ganglion cells that project to the dorsal lateral geniculate nucleus in the macaque monkey,” Neuroscience **12**, 1101–1123 (1984).
29. D. M. Dacey and M. R. Petersen, “Dendritic field size and morphology of midget and parasol ganglion cells of the human retina.” Proc. Natl. Acad. Sci. United States Am. **89**, 9666–9670 (1992).
30. D. Dacey, “The mosaic of midget ganglion cells in the human retina,” J. Neurosci. **13**, 5334–5355 (1993).
31. E. A. Rossi and A. Roorda, “The relationship between visual resolution and cone spacing in the human fovea,” Nat. Neurosci. **13**, 156–157 (2010).
32. R. Sabesan, B. P. Schmidt, W. S. Tuten, and A. Roorda, “The elementary representation of spatial and color vision in the human retina,” Sci. Adv. **2**, e1600797 (2016).
33. J. Verweij, E. P. Hornstein, and J. L. Schnapf, “Surround antagonism in macaque cone photoreceptors,” J. Neurosci. **23**, 10249–10257 (2003).
34. S. Grossberg and E. Mingolla, “Neural dynamics of form perception: Boundary completion, illusory figures, and neon color spreading,” Psychol. Rev. **92**, 173–211 (1985).
35. I. G. Priest and F. G. Brickwedde, “The minimum perceptible colorimetric purity as a function of dominant wavelength,” J. Opt. Soc. Am. **28**, 133–139 (1938).
36. D. Jameson and L. M. Hurvich, “Some Quantitative Aspects of an Opponent-Colors Theory. II. Brightness, Saturation and Hue in Normal and Dichromatic Vision,” J. Opt. Soc. Am. **45**, 602–616 (1956).
37. J. S. Werner, C. M. Cicerone, R. Kliegl, and D. DellaRosa, “Spectral efficiency of blackness induction,” J. Opt. Soc. Am. A **1**, 981 (1984).
38. R. M. Evans and B. K. Swenholt, “Chromatic Strength of Colors: Dominant Wavelength and Purity,” J. Opt. Soc. Am. **57**, 1319 (1967).
39. D. L. Bimler, G. V. Paramei, and C. A. Izmailov, “A whiter shade of pale, a blacker shade of dark: Parameters of spatially induced blackness,” Vis. Neurosci. **23**, 579–582 (2006).
40. J. H. Nelson, “Anomalous trichromatism and its relation to normal trichromatism,” Proc. Phys. Soc. **50**, 661–702 (1938).
41. B. Cole, G. Henry, and J. Nathan, “Phenotypical variations of tritanopia,” Vis. Res. **6**, 301–313 (1966).
42. W. D. Wright, “The characteristics of tritanopia,” J. Opt. Soc. Am. **42**, 509–521 (1952).
43. R. M. Boynton, R. T. Eskew, and C. X. Olson, “Blue cones contribute to border distinctness,” Vis. Res. **25**, 1349–1352 (1985).
44. R. L. De Valois and K. K. De Valois, “A multi-stage color model,” Vis. Res. **33**, 1053–1065 (1993).
45. J. D. Crook, M. B. Manookin, O. S. Packer, and D. M. Dacey, “Horizontal Cell Feedback without Cone Type-Selective Inhibition Mediates "Red-Green" Color Opponency in Midget Ganglion Cells of the Primate Retina,” J. Neurosci. **31**, 1762–1772 (2011).
46. P. R. Martin, E. M. Blessing, P. Buzás, B. A. Szmajda, and J. D. Forte, “Transmission of colour and acuity signals by parvocellular cells in marmoset monkeys,” The J. Physiol. **589**, 2795–2812 (2011).
47. W. H. Merigan and J. H. R. Maunsell, “Macaque vision after magnocellular lateral geniculate lesions,” Vis. Neurosci. **5**, 347–352 (1990).
48. W. H. Merigan, C. E. Byrne, and J. H. R. Maunsell, “Does primate motion depend on the magnocellular pathway?” J. Neurosci. **11**, 3422–3429 (1991).
49. E. J. Chichilnisky and R. S. Kalmar, “Temporal resolution of ensemble visual motion signals in primate retina,” J. Neurosci. **23**, 6681–6689 (2003).
50. M. B. Manookin, S. S. Patterson, and C. M. Linehan, “Neural Mechanisms Mediating Motion Sensitivity in Parasol Ganglion Cells of the Primate Retina,” Neuron. **97**, 1327–1340 (2018).