The Dual-Channel System For Human Color Vision - A Theoretical Approach

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1. Introduction

The human eye is the finest camera invented and has been whetted relentlessly by mother nature over millions of years of evolution. It captures natural themes with such a high degree of fidelity and allows us to enjoy the world in color. Over the years, great effort has been devoted to understand the cellular basis of vision, especially the retina of the eye. For the simplicity of description for this paper, the structure of the retina is roughly divided into three layers (Figure 1). The inner layer, facing the pupil of eye, consists of ganglion cells, whose axons form optical nerves that carry highly processed visual information to the central brain. The dendrites of ganglion cells receive signal inputs from the middle layer consisting of bipolar cells, which receive visual information from the outer photoreceptor layer. It's the photoreceptor layer that generates the raw electric potentials upon exposure to light.

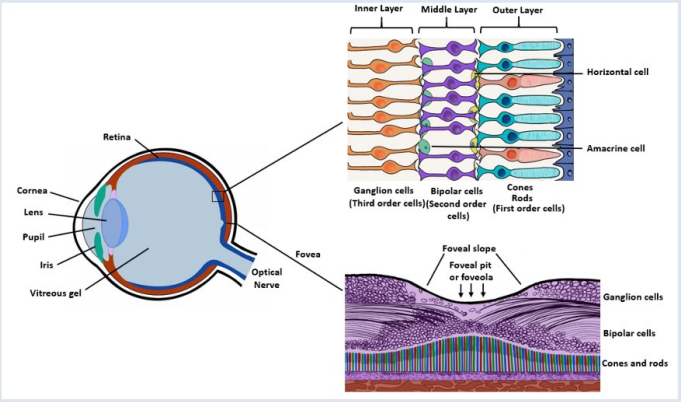

Figure 1. Schematic diagrams of the eye, three-layer architecture of the retina and fovea.

The tissue space between the photoreceptor layer and bipolar cell layer is called the outer plexiform layer(OPL), and the tissue space between the middle and inner layers where bipolar cell axons and ganglion cell dendritic terminals meet is called the inner plexiform layer(IPL). IPL is divided into two functionally discrete sub-laminae. The sub-lamina a is located at the middle layer side, and the sub-lamina b located at the inner layer side. The axon terminals of a given bipolar cell type and the dendritic terminals of a given ganglion cell type usually end either in sub-lamina a or sub-lamina b (mono-stratified), although some cell types span their axon or dendritic tree into both sub-lamina a and sub-lamina b (bistratified). Such a meaningful division of tissue space is of great biological significance.

The fovea is a small depression of about 1.5 mm in diameter in the central retina and is a functionally and morphologically specialized structure in primates only. In humans, the flat center of the fovea, called foveola or foveal pit, sees around the central 1.5 degrees of the visual field, which is the ultimate

view with the highest acuity that allows us to read and discriminate very tiny differences. Vision from the foveal area will be the focus of this paper.

Two types of photoreceptor cells are found in the photoreceptor layer, cone and rod. Their light reactivity comes from the light absorbing chromophore, 11-cis retinal-opsin complex. When 11-cis retinal couples to different types of opsin molecules, it absorbs light in different wavelength ranges. All of primate color vision comes down to three different types of cones, each of which contains a unique type of opsin. S (short) cone is responsible for light in the short wavelength range, M (medium) cone for light in the medium wavelength range, and L (long) cone for light in the long wavelength range. The opsin found in rod cell is called rhodopsin. Because there is only one form of rhodopsin for all rod cells, the rod cell is not color-capable. Figure 2 shows the normalized absorption spectral curves for the three cone types plus the rod cells. There are considerable overlaps in the wavelength ranges in which the three cone types react to generate the initial visual signals.

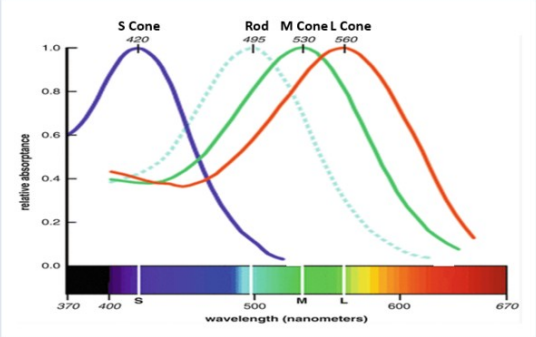

Figure 2. The normalized absorption spectra of the three cones plus rod.

The principle of univariance states that a single cone type can be excited by light of different combinations of wavelength and intensity. By increasing the intensity, a weakly absorbed wavelength can evoke a cone response that will be indistinguishable from the response evoked with strongly absorbed wavelength at a lower intensity. Therefore, a single cone type is not sufficient to differentiate between a response change in wavelength and a response change in intensity. The visual information must be extracted by comparing the responses across different types of cones.

In this paper, by taking a theoretical approach, I will present mechanisms to explain how the principle of univariance is implemented in primate retina. Hopefully it will allow us to think of vision from an alternative angle with the goal to gain a better understanding of vision and facilitate the interpretation of research data.

2. Wavelength and Intensity Are the Dual Properties of Light That Determine Color

In photochemistry, the chance that a photoreaction will occur depends on the wavelength of the light that the reactant is sensitive to, and the degree of the photoreaction triggered by that wavelength is determined by the intensity of that light. Therefore, the wavelength and intensity are the dual properties of light that determine if a photoreaction will occur and occur in what degree, which, in color vision, determine the color of light. Figure 3 shows that the color green changes its tone as the light intensity decreases, while the green wavelength is unchanged.

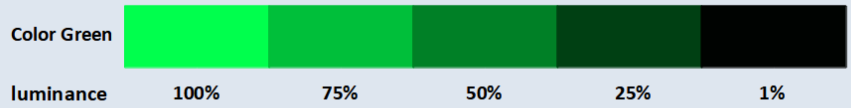

Figure 3. The rectangles represent the tones that change from light green to dark green as their luminance decreases from 100% to 1% while green wavelength is unchanged.

Vision is essentially a photoreaction followed by the phototransduction pathway to convert light into an electric potential $\mathcal{V}$ across the membrane of a cone, referred to as hyperpolarization, which is the very initial vision signals. In the dark, a photoreceptor is partially depolarized to release neurotransmitter glutamate to impact the downstream neurons. By contrast, a hyperpolarized photoreceptor reduces its glutamate release. This seemingly simple and trivial consequence of phototransduction encapsulates all the information that allows us to see in color.

The electric potential $\mathcal{V}$ is the only product that a cone produces in response to light exposure and its size is determined by the absorption spectral curves of the cone type as well as the number of photons that trigger the photoreactions. Although red light is poorly absorbed by all cone types, it is as brilliant as colors green and yellow to our eye. The retina must not only capture the light in the form of $\mathcal{V}$s, but also possess mechanisms to reduce the $\mathcal{V}$s into signals that truly reflect the wavelength and intensity of the incoming light in an unbiased way. How does the retina find out cones of different types and compare their responses to extract visual information? How does the retina distinguish if the $\mathcal{V}$ is due to the impact of green or red or light of other wavelengths? How does the retina represent all wavelengths equally across the visible spectrum despite great spectral variation? Any vision theory should provide at least some reasonable answers to these very fundamental questions.

Although the photons are no longer relevant after photoreaction, the rate of the phototransduction pathway should depend only on the number of photons that trigger the photoreactions, which determines the level of cone output – a reduction in glutamate release. If the efficiency of phototransduction pathway fluctuated, the sizes of electric potentials $\mathcal{V}$s would fluctuate as well, making it impossible to interpret the end results in a meaningful way. Borrowed from mathematics, the consistent efficiency of phototransduction pathway is expressed as a coefficient $\boldsymbol{c}$.

The rate $\mathbf{r}$ at which the photons are absorbed by 11-cis retinal-opsin complex is determined by the wavelength of the photons. The $\mathbf{r}$ will be smaller if the photon carries a wavelength farther away from the absorption peak, but it will be a constant for a particular wavelength in a particular cone type. The electric potential $\mathcal{V}$ or hyperpolarization could be approximately described as the result of the rate $\mathbf{r}$ times $\mathbf{I}$, the number of photons or the intensity of light in a unit of time, times $\boldsymbol{c}$, the coefficient of phototransduction pathway shown below:

$$\mathcal{V} = \mathbf{r}\boldsymbol{c}\mathbf{I}$$

Since $\mathbf{r}$ and $\boldsymbol{c}$ are constants, they are combined into another constant $\mathbf{R}$ to have the following equation:

$$\mathcal{V} = \mathfrak{R}I \qquad (1)$$

Wavelength appears in the constant $\mathfrak{R}$ only implicitly, making it an intrinsic property of light. In contrast, intensity determines the value $\mathcal{V}$ of a given wavelength in a positive fashion by directly being part of the calculation. Above equation shows that the same level of hyperpolarization can be achieved with different combinations of wavelength and intensity. A single cone type is unable to differentiate a change due to wavelength or due to intensity.

A pair of M-cone and L-cone are named lambda pair when they are exposed to the same light, and the electric potentials $\mathcal{V}$s produced by the lambda pair are called lambda $\mathcal{V}$s. By applying Equation (1) to the lambda pair, we have the following equation pair:

$$\mathcal{V}_M = \mathfrak{R}_M I$$
$$\mathcal{V}_L = \mathfrak{R}_L I$$

By taking the division of two equations, we have the equation below:

$$\frac{\mathcal{V}_M}{\mathcal{V}_L} = \frac{\mathfrak{R}_M I}{\mathfrak{R}_L I} = \frac{\mathfrak{R}_M}{\mathfrak{R}_L}$$

The ratio of the lambda $\mathcal{V}$s is equal to the ratio of $\mathfrak{R}_M$ to $\mathfrak{R}_L$, the result of which is a constant. Therefore, the ratio of the lambda $\mathcal{V}$s is a constant as well. Since $\mathcal{V}$s are the end products of phototransduction and the very initial signals of color vision, the constant nature of the ratio suggests that it's the neural form of wavelength that the visual system generates to represent the physical wavelength of the incoming light and that the central brain uses to perceive colors. It is denoted $\boldsymbol{\lambda}$**.** We have a definition of $\boldsymbol{\lambda}$:

$$\lambda = \frac{\mathfrak{R}_1}{\mathfrak{R}_2} = \frac{\mathcal{V}_1}{\mathcal{V}_2} \qquad (2)$$

Subscripts 1 and 2 in Equation (2) mean M-cone or L cone in a lambda pair. The constant $\boldsymbol{\lambda}$ is independent of light intensity, allowing the visual system to achieve the color constancy, the ability to discriminate objects that differ in the spectral characteristics of surface reflectance. Figure 4 shows two highly hypothesized response lines, blue for M-cone and orange for L-cone. The yellow line that represented the ratio of L-cone to M-cone responses was flat and constant when both cone responses were linear. It displayed a unique intensity-independent nature like physical wavelength that is independent of the intensity of the natural light.

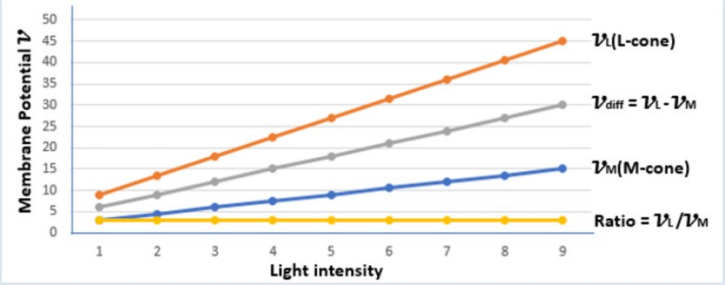

Figure 4. Hypothesized light responses by an M-cone and an L-cone in terms of electric voltage or membrane potential $\mathcal{V}$. $\mathcal{V}_{Diff}$ (gray) and Ratio (yellow) are derived from the original data lines.

If the retina keeps the order of division consistent when deriving the **λ**, for example, ν_M over ν_L, the **λ** values would represent almost all wavelengths that the M-cone and L-cone respond to. If $\nu_M > \nu_L$, all **λ**s are for blue side wavelengths; if $\nu_L > \nu_M$, all **λ**s are for red side wavelengths. They are separated by the **λ** derived from the condition $\nu_L = \nu_M$.

Based on equation (1), the intensity **I** could be calculated only if ν and **R** were known. However, **R** is conceptual and doesn't really mean anything to the retina. It isn't possible for the retina to accurately determine the intensity from the measurable end products νs. Nevertheless, the intensity of light, like wavelength, must be converted into a neural form that the visual system generates to represent the physical intensity of the incoming light and that the central brain uses to perceive colors. The neural form of intensity is denoted **i**. Neural **λ** and neural **i** are the dual signals for color vision, corresponding to the dual properties of light – wavelength and intensity.

How does the retina extract intensity information from the end products νs? The neural **λ** is an absolute value derived from the lambda νs, but the neural **i** could be an approximation only. If we take the differences ν_{diff} of the lambda νs, and plot them against the increasing amount of intensity, we got the gray line shown in Figure 4. It's clear that the ν_{diff} of a lambda pair is linear as well against the increasing amount of intensity. By applying Equation (1) to M-cone and L-cone, we have the following equation after doing the ν differences and rearrangement:

$$I = \frac{\nu_1 - \nu_2}{R_1 - R_2} = \frac{\nu_1}{\Delta R} - \frac{\nu_2}{\Delta R}$$

Although ΔR is unknown to the retina, it is constant for light of a fixed wavelength. Appearance of ΔR implies that both ν_1 and ν_2 can't be used directly out of cones for neural **i**, but must undergo a certain change to take ΔR into consideration. ΔR is called lambda effect. The lambda effect is purely due to spectral variations attributed to wavelengths. If the retina resolved the lambda effect on a ν, it gets a new ν denoted ν_λ:

$$\nu_\lambda = \frac{\nu}{\Delta R} \qquad (3)$$

We use $\nu_{\lambda 1}$ and $\nu_{\lambda 2}$ to express the lambda νs after resolving the lambda effect ΔR. By ignoring the lambda effect, the ν_{diff} of a lambda pair is at least a reasonably estimated neural **i**, as it removed the large absorption variations and made neural **i** more closely reflect the true intensity of the incoming light across the entire visible spectrum. Although the significance of lambda effect can't be stated from Equation (3), with lambda $\nu_{\lambda s}$, neural **i** could be calculated to the highest accuracy shown in Equation (4):

$$i = \nu_{\lambda 1} - \nu_{\lambda 2} \qquad (4)$$

The Equations (3) and (4) indicate that the retina must do extra work to harness transmembrane potentials νs before it could use them for neural **i**. In contrast, generation of **λ** is a straight forward operation using νs directly from cones.

Equations (2) and (4) have effectively eliminated the impact of the great spectral variations on **λ** and **ί**, and as a result, strongly absorbed light didn't have any advantage over weakly absorbed light after transformed into ultimate visual signals.

By separating visual signals into **λ** and **ί**, the retina could generate and deliver far more detailed visual information to central brain, allowing the central brain to distinguish colors that differ only subtly in wavelength and/or intensity. In this way, the vision system has achieved a combinatorial power to make it possible to perceive millions of colors. **λ** is of chromatic nature, while the intensity of light or **ί** is of achromatic nature. An **ί** could act on any **λ** to weaken or strengthen a color tone specific to that **λ** as shown in Figure 3. If it is right, the chromatic **λ**s allow the visual cortex to render base colors, while achromatic **ί**s allow the visual cortex to modulate the tones of colors set by the **λ**s.

Hyperpolarization of photoreceptors as the result of photon stimulation may be of great biological significance. Although single wavelength light is limited roughly to between 400 nm and 700 nm, light composed of multi-wavelengh is almost unlimited, when combined with the intensity of each wavelength. Action potentials as the result of depolarization are largely responses that are threshold controlled and too coarse for the eye to distinguish a million of colors largely due to tiny differences in wavelength and intensity. Therefore, the cone system requires a mechanism to generate membrane potentials that are granular enough to distinguish one light from another with only small differences. Hyperpolarization has the advantage of being threshold independent, and changes in $\boldsymbol{\nu}$ can be continuous even in the sense of numeric values, faithfully reflecting the wavelength composition and the overall intensity that make up the incoming light.

3. Visual Pathway – a Dual-Channel System for the Dual Properties of Light.

In a logical design of work flow, each step is given a specific task to accomplish. The later step will continue what's left by the earlier steps and will never repeat the tasks completed by the earlier steps unless there is a need to repeat. All living organisms are highly efficient in utilizing the resources available from the environment, and the retina won't waste the resources either. The visual signals are processed in retina in a highly sequential and logical order, hence, later cell types can't be possibly performing the tasks already performed by earlier cell types.

The term "algorithm" is used in computer science to describe an ordered set of operations to transform an input into an output. If an algorithm, for example, quick sort algorithm, applies to a set of data hundreds of time, it always generates the same ordered list of data using the same amount of time. In the biological system, nervous activities seem to be controlled by certain algorithms much like a computer program. Retina of the eye and cochlea of the ear can actually be regarded as biological computers, and they convert physical light or sound into electrical impulses and further transform the initial electrical impulses into final formats that the central brain can use to perceive light or sound. Since the entire process of visual or auditory signal transformation is so reliable, predictable, and precise, it can be achieved only by strictly following an ordered and pre-defined set of operations. We termed such a set of operations neural algorithm. Neural algorithm, just like a computer algorithm, when applied to a specific neural task, it always generates the same result using the same amount of time. Without neural algorithms, we would perceive the same color or same sound differently every

time we see that color or hear that sound. Neural algorithms are important to our nervous system just like computer algorithms are important to computer programs.

The retina consists of three layers of cells. There are also accessory cell types found between these layers. Horizontal cells serve the visual pathway at OPL, and amacrine cells play roles at IPL. All cell types after photoreceptors have complicated morphology, all have many subtypes, and each subtype has its own complexity in morphology and unique cellular localization. They seem entangled to form messy intercellular networks, and can express different neurotransmitter receptors and release different neurotransmitters with opposite effects on the recipient cells. Their true faces and functions are still waiting to be revealed.

3.1 **Bipolar Cells Are the Gates to the λ and ί Channels**

The bipolar cells exist between photoreceptor layer and ganglion cell layer, and make connections to cells in both layers via synapses and gap junctions. They relay visual signals from the photoreceptors to the ganglion cells. In the fovea, one bipolar cell receives input from a single photoreceptor, but not vice versa. One cone cell transmits output to two bipolar cells. These two bipolar cells belong to two types. The first type is called invaginating bipolar cell or IBC because a dendritic terminal of it is sandwiched between two dendritic terminals of horizontal cells in a special structure called an invagination at the bottom of the cone axon terminal (see Figure 5). The second type is called flat bipolar cell or FBC because its dendritic terminals make flat synaptic contacts at area next to the invagination. IBC and FBC respond to light stimulation in completely opposite manners.

There are two main types of glutamate receptors expressed on the dendritic tips of bipolar cells: flat bipolar cells bear ionotropic AMPA/kainate glutamate receptors or iakGluRs, and invaginating bipolar cells bear metabotropic glutamate receptors or mGluRs. The iakGluRs are themselves cation channels opened by glutamate. A decline in glutamate concentration due to reduced release closes iakGluRs, making flat bipolar cells more negative inside to become hyperpolarized. The mGluRs close a cation channel called TRPM1 upon binding of glutamate. Lower concentrations of glutamate open TRPM1, making the invaginating bipolar cells more positive inside to become depolarized. Flat bipolar cells are called negative or N bipolar cells, while invaginating bipolar cells are called positive or P bipolar cells.

The visual signals fork into P and N bipolar cells upon leaving the photoreceptors both in fovea and peripheral. However, there is one difference: each bipolar cell in the peripheral gets its input from multiple cones instead of a single cone in the fovea. The axon terminals of a P bipolar cell end up in sub-lamina b, where they make synapses with the dendrites of ganglion cells that also end up in sub-lamina b. These ganglion cells depolarize upon receiving neural signals from P bipolar cells; hence they are positive or P ganglion cells. Similarly, the axon terminals of an N bipolar cell end up in sub-lamina a, where they synapse with the dendrites of ganglion cells that end up also in sub-lamina a. These ganglion cells hyperpolarize upon receiving neural signals from N bipolar cells; hence they are negative or N ganglion cells. There seem to exist two channels in the visual pathway; the positive or P channel conveys neural messages via action potential, while the negative or N channel conveys neural messages via hyperpolarization. The P channel and N channel make up the dual channels of the visual pathway.

We could say that the dual-channel system is a simple mechanism that the evolution has chosen to separate a complex problem into two parts so that each part can be processed and conducted in its own space without interfering with each other. The visual signals are separated into **λ** signal and **ί** signal to increase the color resolution in the central brain. The dual channels are essentially the **λ** and **ί** channels, and the dendritic terminals of bipolar cells are the entry points where signals flow into these two channels. A dual channel system provides a cellular structural basis to make it possible to keep **λ** signal constant as indicated by Equation (2), while allow **ί** signals to undergo extra processing to resolve the lambda effect as shown by Equations (3) and (4).

3.2 **Current Understanding of Horizontal Cells in the Visual Signal Transformation Pathways**

Horizontal cells are a type of interneurons, and their flat arborized dendritic terminals lie underneath the axon terminals of cones. A horizontal cell makes synaptic contacts with many photoreceptors, and a single cone cell can be contacted by several horizontal cells. Horizontal cells thus form a complex interneuron network with cones. Horizontal cells also form synaptic contacts with P bipolar cells. Horizontal cells express iakGluRs and hence they hyperpolarize upon cone exposure to light.

A cone cell has multiple invaginations at its axon terminals. Each invagination houses two dendritic tips from two horizontal cells with a dendritic tip of P bipolar cell in between. Inside the cone axon terminal is another specialized structure called ribbon. The ribbon synapses can release glutamate into the synapse cleft in a fast and steady fashion. The complex that consists of a ribbon synapse, two horizontal cell dendritic tips and a P bipolar cell dendritic tip is called triad (Figure 5). The triad functions as a micro-environment for the retina to perform important functions during visual signal transformation.

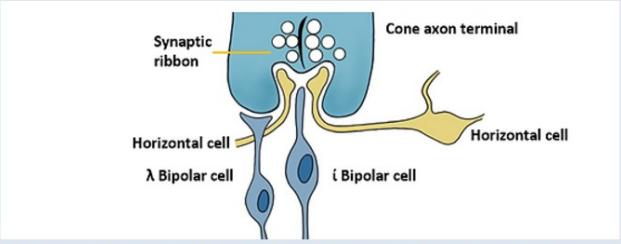

Figure 5. schematic organization of the cone synapse with bipolar cells and horizontal cells. Two dendritic terminals from two horizontal cells, one dendritic terminal of an **ί** or P bipolar cell and an invagination in the cone axon terminal form a specialized structure called triad. The **λ** or N bipolar cell also shown next to the triad.

Horizontal cells have been believed to play a number of roles in visual signal processing: contributing to contrast enhancement and color opponency, generating center-surround receptive fields, and providing lateral inhibition, feedback and feed-forward interactions to photoreceptors and bipolar cells. This list includes almost every major purported visual processing feature that contributes to color vision. Is it possible for a single cell type to play roles in all of them? It has been believed for over 30 years, and it continues to be believed so today. Nevertheless, cells would not assume roles imposed or given by people who are interested in them.

There is a fundamental difference between neural research and biological research in general. In general biological research, results obtained from individual cells or individual parts of cells are largely valid when putting back into the system. For example, results from in vitro transcriptional regulation study is relevant to the in vivo transcriptional regulation in question. However, results from individual neurons

or a group of neurons aren't necessarily sufficient to gain a general picture of how these neurons work and function in vivo as a whole. In neural research, the sum is greater than, even much greater than, the parts. When we try to figure out the roles horizontal cells play in visual signal transformation, we must realize that there is no single cell type that can be a master of all, contributing to every aspect of the system. More specialized a cell becomes, more limited functionality the cell has. Highly specialized horizontal cells can't be exception. Otherwise it will lead research to nowhere. Over-interpretation of research results obtained from limited conditions in a way detached from the big picture will also obscure our understanding of the true nature of these cells.

As a result, despite decades of research, the horizontal cells' roles in visual signal transformation are still vague and elusive. By paying attentions to their major neural characteristics while overlooking tidbit details, this paper attempts to investigate the roles horizontal cells play in visual signal transformation, especially the roles of triad structure in the process with a goal to paint a better picture of how horizontal cells contribute to color vision in fovea.

3.3 **Horizontal Cells Control the Gate to the ί Channel**

The visual information flows from the cones to bipolar cells to ganglion cells. There are accessory cells to modulate the process every time the information changes hands. The direct contacts between horizontal cells and photoreceptors pose time and space constraints on the roles horizontal cells can play, which means that it's unlike that horizontal cells could play many roles in the vision process as they have been believed to play. Furthermore, visual signals that horizontal cells receive from the cones are at the very early stage in the visual pathway, and it seems impossible for the horizontal cells to figure out what role a signal from a particular cone will be in the whole visual field and take action to make necessary changes for the sake of final images, for example, contrast enhancement. Nevertheless, the fact that a horizontal cell contacts multiple cones and a cone is contacted by multiple horizontal cells reveals an important function – possible signal comparisons over the area covered by horizontal cells, which obviously requires the input of information from multiple cone cells. The co-localization of cone axon terminals and horizontal cell dendrites suggests that the signals that have just reached the cone axon terminals must be processed before they enter the bipolar cells.

Equations (3) and (4) imply that generation of $\boldsymbol{\acute{\imath}}$ signal is at least a two-step task and the first step will be divided into two sub-tasks. The first sub-task would be to modulate the membrane potential $\boldsymbol{\nu}$ so that the lambda effect $\Delta\mathbf{R}$ would be factored into the final $\boldsymbol{\nu}_{\boldsymbol{\lambda}}$. $\Delta\mathbf{R}$ is a vague concept solely due to Equation (3). It's at retina's discretion how to resolve the lambda effect. The second sub-task would be to group cones into the lambda pairs as required for deriving the $\boldsymbol{\lambda}$ and $\boldsymbol{\acute{\imath}}$. When we think of the functions of horizontal cells, we must think of how retina fulfills this two sub-tasks required by the Equations (3) and (4).

Let's first look at the signal passage from cones to N bipolar cells. Expression of iakGluRs on N bipolar cells means that they hyperpolarize like a cone upon receiving signals from it. As a result, N bipolar cells receive a copy of the original signal from the cone axon terminal. This sign and quantity conserving property of the N bipolar cells fulfills the requirement of Equation (2) for calculating $\boldsymbol{\lambda}$. The use of unaltered lambda $\boldsymbol{\nu}$s for $\boldsymbol{\lambda}$ indicates that there is no need for any mechanism to modulate this process. Indeed, there is no modulation mechanism present in this signal transfer process.

If the cone signals were passed to P bipolar cells like the cone signals were passed to N bipolar cells, P bipolar cells would receive a copy of cone signal except the signal sign was converted to positive. The P bipolar cells could still use raw-like signals to generate **ί**, but it's only an approximation of the intensity due to the lambda effect, which wouldn't be good enough for the highest color resolution. A complex end can't be achieved with simple means.

The triad structure implies that an end result must be achieved through such a complex structure. The key questions are why the retina needs such a structure at the cone-P bipolar junction and why the triad contains two dendritic tips from two different horizontal cells and a single dendritic tip from a P bipolar cell arranged in such a way under the ribbon? The answers seem to be that all it is to fulfill the two sub-tasks of the Equations (3) and (4): resolving lambda effect and matching lambda pair.

It appears that the triad structure evolved to resolve the lambda effect. A horizontal cell's dendritic tips express iakGluRs, which also consume glutamate released from the ribbon synapse. A direct consequence of this is that glutamate pool in the synapse cleft available to the P bipolar cells are effectively reduced by a certain amount, which in turn amplifies the positive signals on the P bipolar cells by a corresponding amount. Having horizontal cell's dendritic tips in the triad seems to be a venue to modulate $\boldsymbol{\nu}$s by controlling the amount of glutamate available to the P bipolar cells. The end result is turning lambda $\boldsymbol{\nu}$s into lambda $\boldsymbol{\nu}_{\lambda}$s.

There were more factors in the triad micro-environment to change the concentration of glutamate in the synapse cleft, further changing the depolarization of the P bipolar cells. How exactly these factors influence each other seemed to be instructed by a special neural algorithm – resolve algorithm – encoded in the triad for the resolution of lambda effect. At the end of the resolve algorithm, any given input lambda $\boldsymbol{\nu}$s were transformed predictably and consistently into the corresponding output lambda $\boldsymbol{\nu}_{\lambda}$s, and the output lambda $\boldsymbol{\nu}_{\lambda}$s were solely determined by the input lambda $\boldsymbol{\nu}$s. The likely outcome of the resolve algorithm is that the lambda $\boldsymbol{\nu}$s were modulated to broaden the range of **ί**s, enabling our eyes to distinguish color tones due to subtle differences in intensity. Even if resolution of the lambda effect brought big changes to $\boldsymbol{\nu}$s, it had no impact on **λ.** That's clearly the reason why evolution separates visual signals into two channels.

It would be interesting to see if different individuals resolve lambda effect equally well. If not, then a better ability to resolve the lambda effect could confer individuals to enjoy more vivid colors. The good thing is that if individuals never sees vivid colors due to their not-so-good ability to resolve the lambda effect, they wouldn't even know that colors could be viewed more vividly. Therefore, the red in one's eye may not be exactly identical to the red in other's eye. As long as colors remain the same to an individual, it's alright for the colors to be different to different people.

The horizontal cells are also responsible for the second sub-task, matching lambda pairs. There must be a match algorithm to instruct the matching process. The match algorithm works by gathering $\boldsymbol{\nu}$s from cones covered by horizontal cells and then comparing $\boldsymbol{\nu}$s to determine which pairs meet the matching requirements.

The sandwich algorithm is an easy-to-conceive match algorithm for matching the lambda pair. Lambda pairing requires minimum of three cones in straight line with the middle cone being a different type (Figure 6A). In human, cones in the foveal center are arranged in a hexagonal configuration to pack a maximum number of cones in a given area. The hexagonal configuration seems to make the sandwich algorithm more likely to work (Figure 6B). A primate photoreceptor can have many ribbon structures, and a single photoreceptor cell can be covered by multiple horizontal cells, making it possible for the sandwich algorithm to identify all lambda pairs in the fovea. The match algorithm used νs from cones, but match result was to have the P bipolar cells grouped into lambda pairs.

It was assumed that the retina could divide a visual scene into clusters in the cone mosaic, and cones in each cluster were exposed to light of the same dual properties. Such a cluster would be heterogeneous in cone types, and would have no fixed shape and size, as it reflected a homogeneous part in the visual scene at a given moment. This kind of cone clusters is named H clusters (Horizontal cells, Heterogeneous in cone types, and Homogeneous in light exposure). After cones were matched into lambda pairs, adjacent pairs could fuse into a H cluster if they shared the same νs. This process could take place in the entire fovea to delineate the cone mosaic into H clusters. Any cone of one type in a H cluster could pair with any cone of different type in the same H cluster as lambda pairs. When νs entered P bipolar cells, cone H clusters become P bipolar cell H clusters. By forming a sensor network to execute the sandwich algorithm, which essentially was a voltage mediated cell sorting process, horizontal cells have every cone and bipolar cell mapped into a particular H cluster.

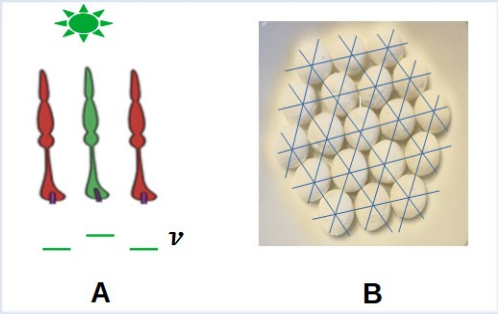

Figure 6. A). Illustration of the sandwich algorithm for matching delta pairs. If the middle M-cone is flanked by two L-cones with the identical potential νs, then M-cone must be exposed to the same light source as the two flanking L-cones. **B).** Each rice ball represents one cone, and 19 rice balls are arranged in a hexagonal configuration. In such an arrangement, each rice ball can be assessed by sandwich algorithm from all six angles.

To this point, the retina has well prepared bipolar cells for subsequent signal transformation. This preparation must be completed at the time the signals diverge into λ and ι channels for economical and space reasons. Horizontal cells control the gate to ι channel to ensure that the initial visual signals have lambda effect resolved, that each cone and P bipolar cell is mapped into their own H clusters, and that P bipolar cells are ready for the second step to calculate ι values. Figure 7 summarizes how the retina converts νs from a lambda pair into two separate, independent parameters λ and ι, and conveys them in their own channel to central brain where they are merged and translated into color.

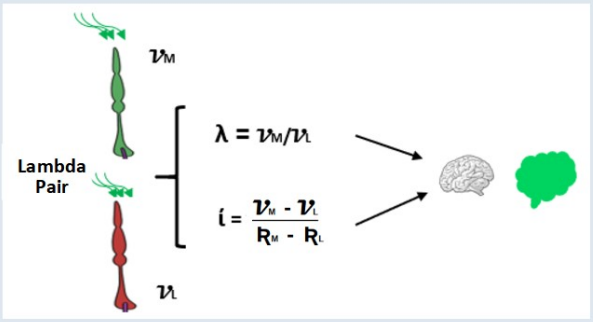

Figure 7. Summary of conversion of cross membrane electric potential νs from the two cone types (lambda pair) into neural wavelength λ and neural intensity $\acute{\imath}$, which are the ultimate neural signals transmitted to the brain where they are decoded into color that we perceive.

Vision acuity varies greatly among individuals. There seem a few major factors that could affect the vision acuity. Smaller diameters of cone cells would allow more cone cells to be packed into the hexagonal configuration in the fovea, enhancing space acuity. A balanced ratio and distribution of M-cones and L-cones could increase color acuity by increasing the efficiency and accuracy of the execution of match algorithm. Lastly, a better ability to resolve lambda effect could also enhance color acuity.

3.4 Amacrine Cells Do the Rest of Work

Amacrine cells, like horizontal cells, are a type of interneurons in the retina. They exist in many subtypes with various shapes and sizes, and are arborized to cover small to large visual fields depending on their tissue locations. Amacrine cells are strictly either mono-stratified or bi-stratified in the IPL region, where the axon terminals of bipolar cells and the dendrites of ganglion cells meet. They make extensive and intricate connectivity to other cells in their reaches. Amacrine cells perform important functions both in local and global scopes.

After visual signals enter bipolar cells, N bipolar cells carry hyperpolarization signals, ready for action to generate neural wavelength λ. P bipolar cells carry depolarization signals, ready for action to produce neural intensity $\acute{\imath}$. Both actions will take place independently in their own channel and sub-lamina space with the help of amacrine cells sitting in between.

Neural λ is produced by dividing lambda νs (Equation (2)), and neural $\acute{\imath}$ is produced by subtracting lambda νs (Equation (4)). Here division and subtraction are operations borrowed from mathematics to make a point that νs of a lambda pair will be combined in certain ways to generate new membrane potentials in the ganglion cell. Productions of λ and $\acute{\imath}$ must be predictable and precise in output and time for every λ and $\acute{\imath}$. This two processes, like matching lambda pairs and resolving lambda effect, would be instructed by their own algorithms – λ algorithm for λ generation, and $\acute{\imath}$ algorithm for $\acute{\imath}$ generation.

In fovea execution of λ algorithm occurs in sub-lamina a, where the axons of N bipolar cells contact dendrites of N ganglion cells. Execution of $\acute{\imath}$ algorithm occurs in sub-lamina b, where the axons of P bipolar cells contact dendrites of P ganglion cells. λ algorithm is independent of $\acute{\imath}$ algorithm, but $\acute{\imath}$ algorithm might require input of signals produced by the λ algorithm. Since hyperpolarization signals reach the sub-lamina a earlier, the execution of λ algorithm could occur ahead of $\acute{\imath}$ algorithm.

Contacts between bipolar cell axons and ganglion cell dendrites form a large number of synapses, as both axons and dendrites are quite arborized. When the neural signals reached the branching points on bipolar cell axons, they could be partitioned into each axon terminal. Each axon terminal could carry its own threshold to control if the signal it received sufficed to release neurotransmitters into the postsynaptic cells. Amacrine cells, part of the algorithms, would influence greatly the outcome of the algorithms. The central role of amacrine cells could be to either strengthen or weaken the signal that flows from bipolar cells to ganglion cells. The nature of the outcome could be further influenced by information amacrine cells received from other amacrine cells and/or bipolar cells or ganglion cells. Only survived signals could release neurotransmitters to act on postsynaptic dendrites of the ganglion cells. Existing in a large variety of subtypes, visual field sizes, and localization, amacrine cells could exert broader influences in the visual pathway beyond **λ** algorithm and **ί** algorithm, for example, coordinating, integrating, and synchronizing visual signals from large visual areas to enhance the quality of color vision. The ganglion cells finally summate the signals from its dendritic tree into one final bit of information to be transmitted to the central brain.

No matter how complicated the neural division and neural subtraction could be, both would follow the steps set by **λ** algorithm and **ί** algorithm respectively. As a result, the end products are predictable from time to time, and the time consumed to execute the algorithms are constant from time to time. Only through the operations of high precision nature, different visual processings can be synchronized to the highest degree possible across the entire retina to make the entire visual field as a uniform whole.

To this end, the visual signals that originate from a lambda pair of cones in the fovea finally enter the four ganglion cell axons, two carrying the identical **ί** and two carrying the identical **λ**. There should be only one **ί** and one **λ** for the entire H cluster. They are the ultimate signals of vision for the visual cortex that allow us to perceive the world in full color. Should the visual signals **ί** and **λ** carry the same sign as all positive or negative, they could confuse the visual cortex to mistake **ί** for **λ** or vice versa when they are combined and translated into colors.

4. Discussion

Retina is a fascinating masterpiece created by mother nature. The simple, raw signals that start from light exposure in the photoreceptors undergo a long and baffling journey to finally end up in the central brain. In this journey the retina does all the nuts-and-bolts kind of signal processing jobs with only one goal: to reduce the primitive visual signals – electric membrane potential $\mathcal{V}$s – into wavelength and intensity needed for color vision. Let's look at the Equations (1) to (4) again in one place:

$$\mathcal{V} = \mathrm{R}\mathrm{I} \qquad (1)$$

$$\lambda = \frac{\mathrm{R}_1}{\mathrm{R}_2} = \frac{\mathcal{V}_1}{\mathcal{V}_2} \qquad (2)$$

$$\mathcal{V}_\lambda = \frac{\mathcal{V}}{\Delta \mathrm{R}} \qquad (3)$$

$$\text{ί} = \mathcal{V}_{\lambda_1} - \mathcal{V}_{\lambda_2} \qquad (4)$$

Equations (1) to (4) lay the architectural blueprint of the cellular structures for color vision, and the primate retina is the biological implementation of Equations (1) to (4). Equation (1) requires two cell types with different sensitivities to the same wavelength - lambda pair - to produce a pair of electric membrane potentials - lambda $\boldsymbol{\nu}$s. In primates, M-cone/L-cone pair and S-cone/M-cone fulfill Equation (1). Equations (2) and (3)/(4) require separate spaces to process $\boldsymbol{\lambda}$ and $\boldsymbol{\imath}$ as different operations are needed to generate $\boldsymbol{\lambda}$ and $\boldsymbol{\imath}$ respectively. In primates there exist two channels, N channel for processing and conducting $\boldsymbol{\lambda}$ signals, and P channel for processing and conducting $\boldsymbol{\imath}$ signals. Equation (3) requires that the initial electric membrane potentials $\boldsymbol{\nu}$s be modified in certain way for Equation (4). Equations (3) and (4) require separate spaces to produce $\boldsymbol{\nu}_\lambda$ and $\boldsymbol{\imath}$ respectively. In primates, $\boldsymbol{\nu}$s are modulated in the structure triads into $\boldsymbol{\nu}_\lambda$s before engaged in $\boldsymbol{\imath}$ production in sub-lamina b. Through implementation of Equations (1) to (4), the primate retina is fully capable of converting light into simple signals – electric membrane potentials, then reducing them back into the neural forms of wavelength and intensity that represent the incoming light either as hyperpolarization or depolarization.

This paper is intended to explain how the color vision works with an emphasis on the central vision. Wavelength and intensity are the dual properties of visible light. While the composition of wavelengths is intrinsic to light emitted from a given source, the intensity can vary as the source becomes weaker or stronger. When light enters the eye, it strikes individual cones to produce the initial visual signals $\boldsymbol{\nu}$s – the electric membrane potentials. $\boldsymbol{\nu}$s are the sole raw signals left by light stimulation.

Production of $\boldsymbol{\nu}$s in cones is not sufficient for color vision as the sizes of $\boldsymbol{\nu}$s are dependent on the wavelength in such a way that $\boldsymbol{\nu}$s aren't the truthful reflection of the light that entered the eye. The retina must de-correlate $\boldsymbol{\nu}$s with wavelength by transforming $\boldsymbol{\nu}$s to signals that faithfully represent the incoming light free of bias due to cone spectral absorption variations. In addition to wavelength, the intensity of light also greatly changes the $\boldsymbol{\nu}$ values although its impact is quite uniform across the entire visible spectrum.

A single cone type can be excited by light of different combinations of wavelength and intensity, and is not sufficient to differentiate between a response change in wavelength and a response change in intensity. The visual information must be extracted by comparing the responses across different types of cones, which is the principle of univariance. The complexity of the visual pathway is evolved to work around the principle of univariance, and any theories in color vision field should at least be able to give plausible accounts to how different types of cones cooperate to achieve spectral decorrelation.

Equations (1) to (4) not only lay the cellular organizational blueprint for color vision, they also establish how wavelength and intensity of the incoming light should be represented in neural forms free of bias.

Assuming that the production of electric potential $\boldsymbol{\nu}$s by photoreceptors through phototransduction is linear and constant to the given incoming light, the ratio of the lambda $\boldsymbol{\nu}$s would be a constant for a given wavelength, independent of the intensity (Equation 2). This ratio is the neural wavelength or $\boldsymbol{\lambda}$, representing the wavelength of the incoming light. Although the difference of lambda $\boldsymbol{\nu}$s is less sensitive to cone's spectral absorption variations, and is a better, but not the best choice to represent the intensity of incoming light, since the effects of the same wavelength on $\boldsymbol{\nu}$s are not the same to the two cone types. The effect of wavelength on lambda $\boldsymbol{\nu}$s is called lambda effect. The retina needs to resolve lambda effect on each $\boldsymbol{\nu}$ of the lambda pair before taking their difference. This difference is the neural

intensity or **ί**, representing the intensity of the incoming light (Equation (4)). Resolution of lambda effect is the extra step the retina must take to produce **ί**.

A single cone passes its signal to two bipolar cells. One bipolar cell, N bipolar cell, hyperpolarizes on receiving the signal from the cone. This N bipolar cell passes its signal to a N ganglion cell in the same fashion. A N bipolar cell and a N ganglion cell make up the N channel. The N channel is specific to **λ** signals. Another bipolar cell, P bipolar cell, depolarizes on receiving the signal from the cone. This P bipolar cell passes its signal to a P ganglion cell in the same fashion. A P bipolar cell and a P ganglion cell make up the P channel. The P channel is specific to **ί** signals. The presence of two channels, as required by Equations (2) and (4), guarantee that processings of **λ** and **ί** don't interfere with each other. The cellular structure of the retina also strongly suggests that visual signals are partitioned into two channels, N channel and P channel, although there is no clue which is for which.

Visual signals from the cone cells are modulated at a special structure at the bottom of a cone axon terminal when passing to P bipolar cells. This structure, called triad, is formed from a cone axon terminal, a dendritic tip from P bipolar cell and two dendritic tips from two horizontal cells, and is where the lambda effect is resolved and lambda **𝜈**s are refined into lambda **𝜈**λs. In addition, the triad structure is also the place where cones across the fovea are matched into lambda pairs. Triad structures, as required by Equations (3), make sure that the **ί** values are produced to the highest possible accuracy for the best color acuity. Are the presence of triad structure and resolution of lambda effect a pure coincident? No, it isn't. If it is, what's the roles of triads in visual signal processing?

An H cluster is a group of cone cells, heterogeneous in cone types, but homogeneous in light exposed. A visual scene consists of a number of H clusters, dependent on its complexity. Sandwich algorithm is one hypothesized working model to identify lambda pairs. Lambda pairs that share the same set of **𝜈**s fuse into an H cluster, delineating a visual scene into H clusters.

Further processings of lambda **𝜈**s and lambda **𝜈**λs in their own channel generate **λ** and **ί** in their final formats for the central brain. To this end, all ganglion cells descending from the cone cells in an H cluster carry the same **λ** in the **λ** channel and the same **ί** in the **ί** channel. Bipolar cells and ganglion cells seemed to provide a signal passage that allowed horizontal cells and amacrine cells to do their work on signals as signals are traveling down through it.

You can be sick or healthy, happy or sad or mad, young or old, living in cold or hot, you see the same natural scene a million of times without differences as long as the natural scene is not changed physically. The retina is truly a biological computer with extraordinary precision, stability, repeatability, and efficiency. Like a modern computer, the biological computer must rely on some neural algorithms to guarantee that numerous nervous signals are processed, coordinated, integrated, synchronized, and transmitted precisely, repeatably, and efficiently in output, space, and time. At least four neural algorithms – resolve algorithm, match algorithm, **λ** algorithm, and **ί** algorithm – are working to guarantee the transformation of visual signals. With the aid of computer simulation technology, it would be possible to simulate some of these neural algorithms to reveal the true nature of color vision.

A neural algorithm must be supported by special cellular structures, and a special cellular structure may be evolved to support a particular neural algorithm. A special cellular structure could reveal a hint of how the neural algorithm would work. There seems to exist structure-function correlations between the four neural algorithms and their underlying cellular structure. The resolve algorithm is intended to resolve the lambda effect on early visual signal to make it more refined for the **ί** value. It brings small and local changes to the signal and may require information input from nearby cones. As a result, the underlying triad structure provides a micro-environment, in which a set of neural factors influence each other to change the amount of glutamate released into synaptic cleft, thus changing the $\boldsymbol{\nu}$s for the P bipolar cells. The triad structure also has the means to exchange information with nearby cones when needed to support the algorithm. Match algorithm is a sorting algorithm acting on multiple cells to group them into lambda pairs and relies on the triad as its underlying cellular structure. Both **λ** and **ί** algorithms are to merge lambda $\boldsymbol{\nu}$s and $\boldsymbol{\nu}_{\lambda}$s to generate new $\boldsymbol{\nu}$s - **λ** and **ί**. The underlying cellular structures are dense axon-dendrite connections with accessory cells in between to support communication among lambda pair, partition the signals into smaller portions for micromanagement, input information from or output information to other cells for coordination, synchronization, integration, and so on.

Separation of visual signals into **λ** and **ί** have great advantages in signal processing. One question to be asked is that if a single channel, for example, the widely accepted ON channel (see below), could have the capacity to process, carry, and convey visual signals that allow us to distinguish a million of colors, majority of which differ only extremely subtly? With the separation of the signals into **λ** and **ί**, the visual system gains a combinatorial power that would at least double the capacity to process, carry, and convey a much larger amount of visual signals that truly allows us to distinguish a million of colors. In addition, separating **ί** from **λ** gives the visual pathway space to greatly broaden the **ί** range to enhance visual sensitivity to tiny changes in intensity. Lastly separation of visual signals into chromatic **λ** and achromatic **ί** allows the central brain to perceive exact colors much like adjusting chromatic paint to desired color tone by adding or removing achromatic solvent. It also makes color comparison easier and more precise by comparing **λ** with **λ**, and **ί** with **ί**.

There is another possible reason why the visual signals diverge into negative **λ** and positive **ί** channels. If we saw a red rose, we could project the rose in our mind after a few days. However, the projected rose is largely black and white, or colorless. Actually this is true to anything we see in our life. This means that our brain only retains or stores or memorizes the physical contour of an object, not the colors of the object. This black and white physical contour of the rose is very likely the imperfect copy of **ί** signals that represented the rose that entered our eye in the form of light.

Cones respond to exposure to visible light by evoking the phototransduction cascade to generate $\boldsymbol{\nu}$s with their light sensitive chromophores. Nevertheless, not every photon counts. More distant the photon wavelength is to the chromophore absorption peak, smaller the chances the photons have to activate the chromophores, generating smaller generic $\boldsymbol{\nu}$s as a consequence. However, cones are color blind and do not have the capability to distinguish if the $\boldsymbol{\nu}$s are generated with, for example, strongly absorbed green light, or weakly absorbed red light, or any light in between. Should cones have this capability, the retina would have far less complex cellular structures, and the visual pathway would be much simpler.

Evolution of the retina has created different cone types and unique cellular structures to support the visual pathway, through which a logic neural workflow could be executed to transform the generic $\boldsymbol{\nu}$s into meaningful neural signals that incarnate the physical properties of light that determine color. It would be ignorant to think that generic $\boldsymbol{\nu}$s could be modified in the early processing stage before relevant information had been extracted, as this could bear serious consequences of totally distorting the color. In case of contrast enhancement, enhancing color contrast wasn't as easy as saying ABC that the contrast could be enhanced simply by adding something to this $\boldsymbol{\nu}$ and subtracting something from that $\boldsymbol{\nu}$. This sort of thinking is too naive and wishful. To enhance contrast in a visual field, the retina must know what visual signals are involved in contrasting and when these signals are available for contrast enhancement. The contrast enhancement occurs unlikely at the cone-horizontal junction simply because of neighboring $\boldsymbol{\nu}$s being different or of some other reasons. Only after individual pieces of signals related to contrasting had been extracted from the generic $\boldsymbol{\nu}$s, contrast enhancement could take place on these signals if contrast enhancement was part of the visual pathway.

There are a few theories and hypotheses in the field of color vision. However, they can be hardly used to explain even the very basic principle of univariance, not to envision a picture of how our eyes see colors. Color opponency and center surround theories are the most prominent among them, and have gained a wide acceptance. These two theories were supported by experimental observations that were obtained using unique lab technique under a particular set of conditions and then generalized to a great extent. Both theories failed to explain how the generic signals $\boldsymbol{\nu}$s are transformed along the visual pathway to their final forms that the central brain could consume to perceive color. Both theories seem not relevant at all to the principle of univariance which is the foundation of color vision in all organism. These theories, especially the center surround theory, also impose quite rigid cellular organizational structures on retina, which is at odds with eye's unlimited ability to see anything in the nature. Are these theories at work when the eye is looking at a pure color, like pure red, screen? Have these theories been misguiding and, as a result, hindering vision research?

N bipolar cells are called OFF bipolar cells and P bipolar cells ON bipolar cells in research literature, and same for ganglion cells. "ON" bipolar cell and "ON" ganglion cells make up the "ON" channel, and "OFF" bipolar cell and "OFF" ganglion cells make up the "OFF" channel. Nevertheless, ON and OFF are not appropriate to describe these two cell types. Hyperpolarized cells should not be considered OFF. We say light stimulated photoreceptors, but light stimulation makes photoreceptors hyperpolarized. Are light stimulated photoreceptors OFF? Hyperpolarization is simply a neural state after a neuron is exposed to certain external factors. As long as an hyperpolarized state could impact downstream neurons, it should not be considered "OFF". Furthermore, an "OFF" neuron could turn the postsynaptic cells "ON" if the cells express receptors that could lead the cells to depolarize upon receiving the pre-synaptic impact. There are no explanations why a cone diverges its signals into "ON" and "OFF" cells, and no clues to the nature of signals that "ON" and "OFF" channels carry respectively. Calling them OFF or ON is misleading, and has been obscuring their true roles in the process of visual signal transformation.

To end this paper, let's use $\boldsymbol{\lambda}$ values to explain how the colors of multi-wavelength light are determined. Naturally light that enters the eye is not of single wavelength, but of mixed wavelengths. The 11-cis retinal-opsin molecule would absorb any photons in its absorption range. Each wavelength in multi-

wavelength light had its own intensity, and stimulated cone cells independent of other wavelengths in the same light. The end result was another $\boldsymbol{v}$, the sum of all voltage changes evoked in the cone by each wavelength. That meant the $\boldsymbol{\lambda}$ would be generated from the lambda $\boldsymbol{v}$s derived from mixed-wavelength: $\boldsymbol{v}_1/\boldsymbol{v}_2$ or $\mathbf{R}_1/\mathbf{R}_2$, where $\boldsymbol{v}_1$ and $\boldsymbol{v}_2$ are composite $\boldsymbol{v}$s, and $\mathbf{R}_1$ and $\mathbf{R}_2$ are composite constants. If this $\boldsymbol{\lambda}$ happened to be equal to the $\boldsymbol{\lambda}$ generated from the lambda $\boldsymbol{v}$s derived from single-wavelength light, the color would be perceived as the single-wavelength color. Because cones discriminate wavelengths only through its retinal-opsin complexes via different absorbance spectra, it would be impossible for the retina to know whether the $\boldsymbol{v}$, so the $\boldsymbol{\lambda}$, was the result of exposure to single wavelength light or multi-wavelength light.

If light contains two wavelengths that are close in the visible spectrum, like half green and half yellow, the total $\boldsymbol{v}_t$ would be the sum of green $\boldsymbol{v}$ and yellow $\boldsymbol{v}$. The size of $\boldsymbol{v}_t$ fell between the two $\boldsymbol{v}$s if the light contains pure green and pure yellow respectively. The $\boldsymbol{\lambda}$ value computed from the lambda $\boldsymbol{v}_t$s fell just within the space close to both green $\boldsymbol{\lambda}$ and yellow $\boldsymbol{\lambda}$, which is an additive color – greenish yellow. On the other hand, if light contains two wavelengths that are not close in the visible spectrum, like half green and half red, the total $\boldsymbol{v}_t$ would still be the sum of green $\boldsymbol{v}$ and red $\boldsymbol{v}$. The size of $\boldsymbol{v}_t$ again fell similarly between the two $\boldsymbol{v}$s if the light contains pure green and pure red respectively. Because the difference between green $\boldsymbol{v}$ and red $\boldsymbol{v}$ was quite large, $\boldsymbol{v}_t$ would be close to neither green $\boldsymbol{v}$ nor red $\boldsymbol{v}$, and the $\boldsymbol{\lambda}$ value computed from the lambda $\boldsymbol{v}_t$s fell within a space between green $\boldsymbol{\lambda}$ and red $\boldsymbol{\lambda}$, but close neither to green $\boldsymbol{\lambda}$ nor red $\boldsymbol{\lambda}$. As a result, mixing green and red wouldn't lead to an additive color – greenish red, but a color between green and red, that was yellow. A similar picture could be drawn for light containing blue and yellow. Yellow and green seem to be two chasms in the visible spectrum, and mixing wavelengths from the two sides of each chasm generates colors that fall within that chasm. This is obvious from the visible spectrum. From color opponency point of view, it seems that wavelengths from the two sides of each chasm opposes each other, but actually, it is simply the fact that mixing two dissimilar things lead to a thing that is dissimilar to both of its parents. There is no such thing as color opponency.

Finally I will attempt to give afterimage an explanation. The central theme of the dual channel system is that the color we perceive through eye is deduced from the sole products of phototransduction – the electric transmembrane potential $\boldsymbol{v}$s – that occurs in M-cones and L-cones. Any visual phenomena must be explained by focusing on what normal visual processes have been disrupted in the retina that have caused the visual phenomena in question. If this is not the case, then the cause could be residing in the visual cortex.

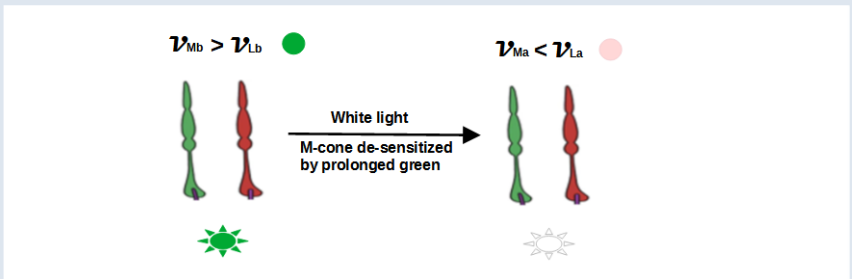

Figure 8. A diagram to illustrate how afterimages can form after looking at the initial image for a prolonged amount of time. An exposure to a particular color for a prolonged time could make the cone cell that absorbs the initial light more strongly ($\boldsymbol{v}_{Mb} > \boldsymbol{v}_{Lb}$) less sensitive to new color exposure, but could make the cone cell that absorbs the initial light less strongly relatively more sensitive to new color exposure ($\boldsymbol{v}_{Ma} < \boldsymbol{v}_{La}$), resulting in a weak reddish afterimage if the initial light is green. Subscripts M: M-Cone, L: L-Cone, b: before, a: after

Figure 8 illustrates how an afterimage could form after a prolonged exposure to green light. When exposing to green light, M-cone, which absorbs green light more strongly than L-cone, produces a membrane potential $\boldsymbol{\nu}_{\mathrm{M}}$, which is greater than the membrane potential $\boldsymbol{\nu}_{\mathrm{L}}$ produced by L-cone. Upon prolonged exposure to green light, M-cone becomes less sensitive to new color exposure, in this case, the white light. This makes L-cone relatively more sensitive to the white light. As a result, the sizes of the membrane potentials are reversed from $\boldsymbol{\nu}_{\mathrm{Mb}} > \boldsymbol{\nu}_{\mathrm{Lb}}$ to $\boldsymbol{\nu}_{\mathrm{Ma}} < \boldsymbol{\nu}_{\mathrm{La}}$, here subscript b means before, and a means after. The direct consequence of this reverse is temporarily shifting $\boldsymbol{\lambda}$ from green zone into the weak reddish zone, which is perceived as a weak reddish afterimage.

Changes in the membrane potential $\boldsymbol{\nu}$s in cone cells could result in the changes in neural $\boldsymbol{\lambda}$, which changes our perception of color, which underpins the notion that the dual channel system of the retina is at work for the color vision.

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