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Fundamentals of colour awareness: a literature review

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Abstract

A description of some of the basic or fundamental aspects of the colour sensory mechanism will be provided here, based on modern ideas and literature, with reference specifically to the likely origins and evolution of colour vision. The molecular basis for colour awareness and the human colour pathway will also be considered in some detail. This paper intends to provide the theoretical and philosophical basis for further papers that will introduce a modern and original computer-based method for more comprehensive colour vision assessment. This new approach, to be fully described in later manuscripts, may contribute towards improvements in understanding and knowledge of human colour perception and its measurement, still perhaps a relatively under-explored or neglected field of study within optometry and ophthalmology.

Keywords: Colour perception, colour vision, mechanisms of colour vision, colour deficiency, colour vision tests.

Introduction

Together with apes and baboons, humans are Old World primates that are all essentially cone trichromats.That is, these primates all have three types of retinal cones that respond selectively to short (S), medium or middle (M), and long (L) wavelengths of visible light (roughly ranging from 400 to 700 nm). The spectral sensitivities of such primates tend to overlap significantly with peaks at about 440, 530 and 560 nm for the S, M and L cones respectively.[1] Other non-primate mammals may be dichromats with only S and L cones[1], only M and L cones or even monochromats with only L cones (for instance, bushbabies or marine mammals). Certain other species may have more than three cones, for example, the Australian lungfish with four cone types or visual pigments and thereby tetrachromatism.[1, 2] Some invertebrates such as mantis shrimps apparently have sixteen types of photoreceptor cells, of which twelve are involved with their colour sense[1]; thus these creatures have a much more highly developed colour awareness than for humans, for instance. The primate colour mechanism evolved many millions of years ago (approximately 40 MYA); some believe mainly as an adaptive mechanism in response to apparently colourful and edible plants and fruits but recently studies suggest that colour vision may have been present for a long period prior to the evolutionary appearance of plants and flowers (some estimates of the possible time interval involved are 400 to 700 MYA in the earliest vertebrates).[2] It is generally accepted that primates developed trichromacy from their dichromat ancestors.[2] Naturally, colour has also been considered as important in mate selection, and in hunting and for the avoidance of predators. Little is known of the evolution of the colour sense prior to 700 MYA, a time of the divergence of vertebrates and invertebrates, because of scarcity of suitable specimens or fossils for

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examination.[2] Electron microscopy of the retinas of, for instance, southern hemisphere lampreys, named *Geotria australis* (eel-like, jawless creatures believed to have their ancestors amongst the first vertebrates dating back to beyond 540 MYA) supports the idea that the colour sensory mechanism had an early origin in vertebrate evolution, but the specialization of rods and cones is less certain in vertebrate evolution.[2] For example, *G. australis* has different identified photoreceptors types, at least five, possibly providing scotopic and photopic sensitivity (methods such as electroretinography have been used with lampreys to establish the spectral sensitivities of the different cellular types).[2] But, in some species such as hagfish, there remains some controversy regarding the presence of two independent and morphologically different vision sensory systems specialized for scotopic and photopic sensitivity (the so-called *duplicity theory of vision*). In some primitive species morphological distinctions between cell types may not be clear enough to easily distinguish between rods and cones.[2] (Hagfish, like the lampreys, are believed to be sole survivors of very early jawless creatures (living about 540 MYA) and present day hagfish have only very poorly developed eyes, with relatively undifferentiated retinas and often aphakia.) Complicating the issue is that some rodents, birds and fish have receptors that extend their spectral sensitivity into regions outside the visible spectrum, for example, the ultraviolet region.[2] Besides morphological examination of the shapes and differences of receptor types such as rods and cones, molecular investigation of photoreceptor visual pigments provides valuable information or knowledge of the possible evolution of the colour sense. Vertebrate visual pigment consists of a protein (opsin) and a chromophore from Vitamin A (occurring in two forms, A_1 or retinal in mammals, and A_1 and A_2 in fish and reptiles).[2] The photoreceptor rhodopsin (*Rh1*) is located in rods. The photopigments are situated within the discs of the outer segments of the relevant photoreceptors and differences in photoreceptor spectral sensitivity relate to the presence of different chromophores and biologic, genetic regulation of opsin. Microspectrophotometry is used to study the spectral characteristics of photoreceptor types and for the southern hemisphere lamprey spectral sensitivity curves and peak absorbances for three of the five photoreceptor types have been measured by Collins, Hart, Shand and Potter[3]. Such investigations[2, 3] suggest in vertebrates that true rod photoreceptors, and thus sensitivity to low luminance levels, might even be a more modern evolutionary development (namely in jawed vertebrates such as fish, mammals, birds and reptiles) than cone photoreceptors and sensitivity to colour and higher levels of ambient luminance. The very early ancestral, jawless, vertebrates are mostly believed to have lived in shallow water environments where a diverse and wide light spectrum and complex environment encouraged evolutionary development of different visual pigments. Early land mammals developed visual systems that responded to dim light and modern nocturnal rodents still have retinas which mostly contain rods with far fewer cones. (Cones are often mostly concentrated in an area such as the area centralis, as for cats and dogs or sometimes a horizontal visual streak such as in rabbits, squirrels and turtles[4].) Today there is also evidence that different opsin genes can be expressed in the same photoreceptor rather than only a single opsin.[2] Physiologic expression of the opsin gene thus may be more dynamic or plastic than previously believed and different hormonal or environmental cues may modify receptor response. There thus remains the need for a great deal of further investigation to develop and improve our understanding of the evolution and photodynamics of retinal photoreceptors and the higher pathways of the colour sensory mechanism.

Variation of illumination in shallow water, forests and other environments producing shadows and patches of differing luminance probably constituted a very important factor in influencing the evolutionary development of the colour sense.[2] Colour naturally is used for the identification, discrimination and differentiation of objects including fruits, leaves, *et cetera*. But not only can colour be used in this manner but there is also the need to extract information about coloured objects under differing illumination conditions, that is, humans and other creatures typically need to recognize or identify coloured objects under different luminance situations (where colours may

appear to have changed relating to variation in luminance). Thus there is the need to perceive specific colours or objects as being the same or unchanged irrespective of the variance in ambient luminance. So, for example, humans can identify a red apple as having the same or constant colour under varying luminance conditions and this phenomenon is known as colour constancy. This phenomenon means that the complicated process of object recognition not only requires chromatic information but also issues of surface reflectance and variation in object and background illuminance must be understood. Possibly the presence of trichromacy, rather than dichromacy, contributes to this process and may explain to some extent the evolutionary advantages of this more complicated photoreceptor mechanism which more reliably allows proper identification of objects under varying luminance and viewing conditions.[1] Even under low luminance conditions, trichromatism may also be more beneficial than dichromatism.[2]

The general neural pathway for the human colour sense

The Retina

Understanding retinal function is a very complicated process even when one partially ignores the rest of the visual pathway. The retina is only about 0.5 mm thick[4] with the retinal pigment epithelium (RPE) providing molecules of retinal or vitamin A to the photoreceptor opsin proteins. The melanin granules of the RPE also absorb stray photons of light and protect the photoreceptors from excessive exposure to light. Very simplistically, the primate retina can be thought of as essentially consisting of three cellular or nuclear layers (that is, the photoreceptors, bipolar and ganglion cells) separated by two synaptic layers (namely the outer and inner plexiform layers) forming links between the cellular layers. Thus the anatomical retinal region containing synapses between the photoreceptors, and bipolar and horizontal cells is known as the outer plexiform layer (OPL) whereas the synaptic region joining bipolar and amacrine cells with ganglion cells is the inner plexiform layer (IPL). Plexiform, that is, resembling or forming a plexus or interlacing network of nerves in this instance. Then, of course, there is also the RPE, outer and inner limiting membranes (the latter forming part of the vitreoretinal interface) and the nerve fibre layer making up a total of ten layers. Within the different layers the primate retinal structure is very interesting and complicated with, depending upon species concerned, up to 20 different types of ganglion cells, 11 types of bipolar cells, 30 types of amacrine cells and four types of bipolar cells.[4] Different neurotransmitters have been identified and glutamate for example is important in transmission of signals from photoreceptors to bipolar to ganglion cells.[4] Other cells, such as amacrine and horizontal cells, use excitory and inhibitory substances including amino acids, peptides, catecholamines and also nitric oxide.[4] Humans and other Old World primates are believed to have a similar colour sense[5] and so a general colour model can be developed where, for the healthy vision system, trichromatism is the rule (that is, red-, green- and blue-sensitive cones are involved in primate colour awareness). But most mammals only have two cone types, usually green- and blue-sensitive cones and their retinas contain predominantly rod photoreceptors. (As mentioned earlier, some non-primate retinas may have as many as five different cone types[4].)

Over the last six or so decades studies of the electrophysiology of the primate retina have shown that when photoreceptors are stimulated by light a slow cell membrane hyperpolarization (rather than a depolarization) or increase in transmembrane potential occurs, and these so called *S potentials* represent electrical changes involved with neural transmission of information from photoreceptors to bipolar and horizontal cells.[4] If the stimulating light is removed depolarization occurs. That is, rods and cones release neurotransmitters in the dark (cell membranes are depolarized and sodium ions can flow easily across the cellular membranes of photoreceptors) and continue to do so until light is shone onto them (causing cellular ion channels to close and membrane hyperpolarization with no neurotransmitter release).[4] The process thus is opposite to what one might expect, namely, that light stimulation would produce an increased release of neurotransmitters. This slow hyperpolarizing response is

found both with rods and cones but, as we know, cones react to bright light and rapid changes in luminance conditions while rods respond to dim light and slow changes. Rods and cones are said to have very narrow receptive fields to light falling on them whereas horizontal cells which receive input from multiple cones have larger receptive fields.[4] As for the photoreceptors, some bipolar cells react to rapid variation in visual signals while others respond to slow variation in signals allowing for the development of OFF and ON pathways. Very basically, the ON pathway is involved with detection of light against dark while the opposite is true for the OFF path. This contrast of light and dark is very fundamental in our ability to see and, so neural processing in the retina is already able to break up the received image into component parts which can be differentiated, and then eventually re-integrated and understood by the viewer concerned. Horizontal cells add an opponent signal to bipolar cells so that a *centre surround* receptive field is formed for each bipolar cell.[4] Thus the centre of a bipolar cell may respond to either an ON or OFF signal and, similarly, for the cell periphery. Horizontal cells also feed information back to photoreceptors via electrical synapses known today as *hemi gap junctions*[4], thus further increasing the complexity in understanding events or processes occurring in the retina. Much is still not known about retinal function (for example, recently a type of ganglion cell was found which appears to function without input from rods or cones, that is, it seems to function as if it is itself a type of photoreceptor[4]). In terms of ganglion cells, amacrine input is believed to improve or sharpen boundaries between receptive field centre and surround.[4] (Amacrine cells can basically be divided according to those using either glycine or gamma-aminobutyric acid (GABA) neurotransmitters. Glycinergic amacrine cells are involved with rods and scotopic vision while GABA amacrine cells are thought to play a role in retinal function under varying light conditions such as encountered at different times during the day or night. But, rod function will not be discussed much further in this paper.) As with bipolar cells, ON-centre ganglion cells respond to light falling in the centre of their receptive fields and they are deactivated to light on the periphery of their receptive fields. Similarly, OFF-centre ganglion cells respond to light in their receptor field periphery and they are deactivated by light in their receptor field centre. So the electrical (and other chemical and physiologic) activity of bipolar or ganglion cells is related to the nature of light distribution across their receptive fields. In understanding the whole process, one must also realize that the cellular nature and their distribution varies across the retina so that in the fovea, for example, a single cone is linked to a so-called midget bipolar and midget ganglion cell in a one-to-one relationship. Thus each red-sensitive foveal cone can convey an ON (light-on-dark) or OFF (dark-on-light) signal to a single ganglion cell and the same applies to green-sensitive cones. Thus the foveal neural signal to the brain contains both wavelength-dependent, or spectral, information and spatial information.[4] Not only that but additionally the process eventually provides for interpretation and understanding of highly detailed images of very fine resolution. Blue-sensitive cones, on the other hand, are believed to be older in evolutionary terms[4] and they link to ganglion cells whose receptive fields react to blue ON and yellow OFF signals.[4]

Modern developments in technology have allowed for a much improved understanding of the anatomy and physiology of the colour sense and, for instance, it has been possible to selectively stain the population of blue-sensitive or S cones,[5] which constitute about 5-10% of the retinal cones, being rare or absent at the fovea. Otherwise, the S, M and L cones appear to be relatively randomly arranged.[5] Retinal horizontal cells (named type H1), in the outer plexiform layer link L and M cones but not S cones while the other horizontal (type H2) cells mostly link S cones with more limited contact with L and M cones. Exactly how these horizontal cells contribute to chromatic processing is unclear but they are considered to be important due to their connectional specificity and they are believed to contribute to outer retinal spatial processing or with gain control.[5] In the fovea, the red-green pathway involves L and M cones, midget bipolar cells and thereafter midget ganglion cells that

link with the parvocellular (PC) layers of the lateral geniculate nuclei (LGN). Single M and L cones link with single midget ganglion cells through the midget bipolar cells and modern thinking is that all the midget ganglion cells are red-green opponent cells.[5] Midget ganglion cells may be red or green ON-centre or OFF-centre. In the retinal centre and periphery the dendritic trees of neighbouring ganglion cells of the midget- PC system do not overlap even though the one-to-one connectivity changes from the fovea to a many-to-one relationship at the retinal periphery.[5] There is a relation between the extent of overlap of retinal dendritic trees and points in the visual space, that is receptive fields and the term coverage factor is used to indicate how many dendritic trees cover specific points in visual space (overlap is probably desirable since input from separate cells can be compared and intensity and location of small stimuli can be determined to a fraction of the dendritic tree size). So the dendritic trees of central midget ganglion cells do not overlap, there is one-to-one connectivity of receptor to ganglion cell and the coverage factor is one or unity. Towards the retinal periphery (about 30^{o} eccentricity) the dendritic trees for neighbouring ganglion cells still do not overlap although the coverage factor now is between 20 and 30 with many bipolar midget cells supplying input to single ganglion cells.

The blue-yellow pathway involves differentiation of S cone signals from summed M and L cone signals. The +S−(L+M) cell is the small bistratified ganglion cell and has a relatively large dendritic tree. The luminance channel relates to parasol (or larger) ganglion cells (receiving input from M and L cones) that send input to the magnocellular (MC) layers of the LGN.[5] They also react to red-green chromatic input and so overlap with the red-green pathway somewhat.[5]

The Lateral Geniculate Nuclei

The primate lateral geniculate nuclei (LGN) generally are regarded as having six primary layers separated by interlaminar or konicellular (KC) regions (*koni*, from a Greek word meaning dust[6]). The parvocellular (PC) cells, in layers 1 to 4, of the LGN respond mainly to red-green opponent stimulation. These cells have small receptive fields responsive to high contrast achromatic stimuli or low contrast chromatic stimuli.[6] Magnocellular (MC) layers (5 and 6 in the LGN) relate to cells with large receptive fields and high contrast sensitivity. Parasol ganglion cells in the retina are the dominant factors for MC input (and thereafter to layers 4A and 4C in primary visual cortex area V1) to the LGN while retinal midget ganglion cells are the comparable factor with the PC layers of the LGN (and thence to layers 4A and 4C also).[6] The KC layers in the LGN consist of interneurons and relay neurons and are believed to play a role in blue-yellow opponent responses (for example, blue-ON responses appear to be primarily segregated to the KC layers).[6, 7] Small bistratified ganglion cells in the retina link to the KC layers in the LGN and thence to layers 2 and 3 of layer IV of primary visual cortex area V1 (see Figure 1). Studies of the LGN with 'single-unit or neuron' fine electrodes, Nissl stain and calcium binding proteins such as calbindin help researchers in their explorations of LGN function, including response to chromatic or achromatic stimuli.[6]

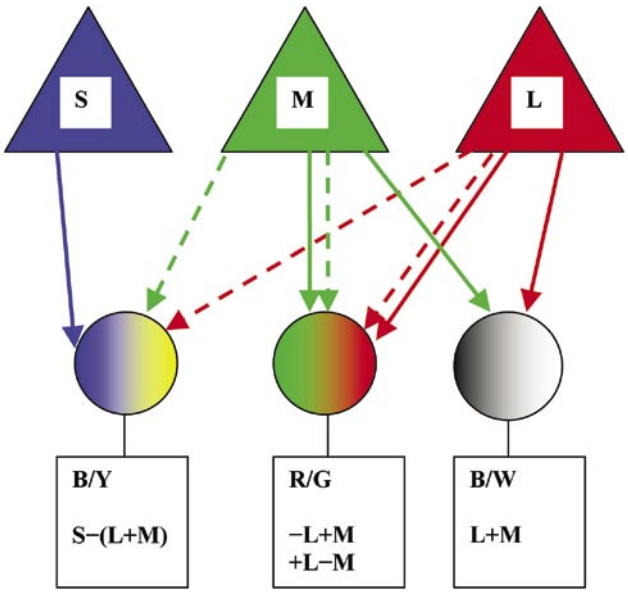

Figure 1: An extremely simplified model of the early stages of the human colour sense is indicated. Summation and subtractive characteristics of the retinal cones (triangles in the figure) and their input to the retinal ganglion cells (indicated with circles) and the lateral geniculate nuclei (indicated with squares) are suggested. Light stimulates the retinal photoreceptors and signals from the short (S), middle (M) and long (L) wavelength sensitive cones (indicated with triangles

and corresponding colours for peak cone sensitivities) are added (solid lines) and subtracted (dashed lines) to produce three opponent signals namely red-green, blue-yellow and black-white or dark-light (the achromatic or luminance signal). Readers should remember that although the different types of cones have a maximal sensitivity to a specific wavelength, that their spectral sensitivity curves overlap one another and so the response of each type of cone is not simply only, to say, red for the L cones (as suggested in the simplified figure above). The L cones respond to other wavelengths (in man, roughly between about 475 to 675 nm) but have a maximal sensitivity (λmax) to wavelengths at about 560-570 nm. Similarly, the M cones respond to wavelengths roughly between about 425 to 625 nm with λmax = 530-550 nm while the S cones respond to wavelengths roughly between about 400 to 525 nm with λmax = 440-450 nm.[1, 13] Other species naturally have their own spectral sensitivity characteristics. In the LGN one group of neurons (the R/G colour opponent cells) is excited by red light and inhibited by green light.[8] These cells react to signals from L and M cones in opposite polarities (+L–M or –L+M). Other neurons (B/Y colour opponent cells) react to blue and yellow light.[8] Signals from the LGN input into the primary visual cortex, namely area V1 (Brodman's area 17 or the striate cortex) in the occipital lobe.This figure was adapted from Boynton GM. Colour vision: How the cortex represent color. *Curr Biol* **12** R838 - 840.

Midget and parasol cells are believed to constitute about 90% of the retino-geniculate pathway in primates and thus are responsible for much of the input to the LGN.[6] It is the midget-parvocellular pathway that is apparently responsible for high-acuity spatial signals that are so important in vision function in primates.[6]

The Visual Cortex

Functional magnetic resonance imaging (fMRI) can measure blood-oxygen-level-dependendent (BOLD) variation in cerebral vasculature.[8] The variation relates to underlying neuronal events or responses, for instance, to presentation of chromatic stimuli. The BOLD reaction is considered to be an averaged response of small regions (essentially a few millimetres) of cortical tissue over short intervals of time measured in seconds. Together with other methods, fMRI allows researchers to study many phenomena such as reaction and adaptation to chromatic light (that is, changes in cone sensitivity or gain allows for light adaptation[8]). Thus by manipulating background or foreground conditions fMRI measurements of area V1 can assist in understanding neuronal activity and the colour sense.

In a simplified model neuronal signals pass from retinal S cones to small bistratified retinal ganglion cells, and then to the KC regions of the LGN and to cytochrome oxidase (CO) blobs found in layers 2, 3 and 4A of the primary visual cortex, V1.[7] The midget retinal ganglion cells, on the other hand, relay to the PC layers of the LGN and then to layer 4C$_\beta$ or 4A (of V1). Parasol ganglion cells relay to the MC layers of the LGN and then to layer 4C$_\alpha$ or 4A (of V1).[6] Figure 2 summarises the various ideas above.

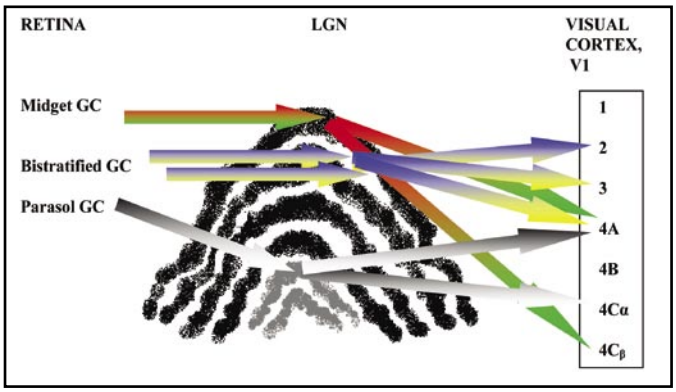

Figure 2: A very simplified representation of retinal ganglion cell (GC) neural input to the lateral geniculate nucleus (LGN) and primary visual cortex, area V1 is indicated. The four parvocellular (PC) layers are indicated in black, the two magnocellular (MC) layers are in grey and the koniocellular (KC) or interlaminar layers are in white. Layer 1 of area V1 is most superficial, that is, nearest the skull or cranium. Coloured arrows are used to roughly indicate the three opponent pathways but for simplicity arrows are only linked to a single PC, MC or KC layer. In reality, all layers would be involved and so, for example, signals from midget ganglion cells would pass to all four PC layers (the black layers in the figure). And, similarly, for the bistratified GC to all KC layers (in white) and for the parasol GC to both MC layers (in grey) for non-chromatic (luminance) signals.

After V1, neural (colour) signals are passed to areas V2, V3 and V4 (the prestriate cortex) and lesions of, for instance, V4 may produce mild colour discriminatory problems but, at the same

time, very poor colour constancy.[7] For such an individual, under varying illuminance conditions a single, constant colour (say a shade of red) would be difficult to recognize as being the same; but separate regions of the same colour (the red shade in our example) when viewed under only a single illuminance level would be easily understood as being the same.

The inferotemporal (IT) cortex also has colour-responsive neurons[7] and lesions here produce severe difficulties with discrimination of most colours or hues but, perhaps somewhat surprisingly, not with discrimination of shades of grey.[7] Colours in humans are generally recognized or described in terms of broad categories and there are said to be about 11 basic colour names commonly used interracially (namely red, pink, orange, yellow, green, blue, purple, white, grey, black and brown).[7] Chimpanzees, apparently recognize similar categories,[9] and suggestions have been made that the IT cortex is involved in a form of categorical discrimination of colour.

Following vascular or other injury to area V4 of the visual cortex (located in the fusiform gyrus) in, for example, cerebral achromatopsia, humans lose their colour sense and see parts or all of their world in dull and "dirty shades of grey".[10] Achromatopsia may be total and bilateral, or may only affect one half of the field of view (producing a strange condition or state known as hemiachromatopsia, in which one half of the world is seen and understood as colourless with the other half normally coloured). In achromatopsia, retinal and even LGN mechanisms for colour awareness are intact. Thus signals are passed properly to the cortex but thereafter the mechanism for understanding and interpreting the world in colour are anomalous. Patients with achromatopsia can read and write, respond to moving objects, have depth perception and discriminate forms but they have a very selective defect to colour. They experience difficulties with recognition of familiar faces (prosopagnosia) and may have speech disorders.[10] Rarely, achromatopsia can be transient with mild vascular insufficiency.[10] Other central anomalies of colour, such as colour anomia (where colours are recognized but cannot be named), can be differentiated from achromatopsia.[10] Studies of individuals with these various anomalies assist in understanding the cerebral contributions to the colour sense.

Philosophical Issues Concerning Colour Awareness

In fundamental physics objects are colourless (the philosophical idea or concept referred to as *eliminativism*[11]) but yet the world appears to almost all of us to be filled with colour. The famous painter Paul Cezanne is reported by Merleau-Ponty to have said, that color is the "place where our brain and the universe meet".[12] Many philosophers, including for example, Aristotle, and scientists such as Newton have attempted to reconcile these two apparently contradictory aspects of human experience. The central issue is one of colour realism.[11] That is, the question is one of whether objects indeed have colour or physical properties producing colour (known as *physicalism*), or whether colour is the result of perceptual processes in the observer? Colour realism concerns the issue of whether visual experience represents the world correctly or realistically. An eliminativist would believe that our common experience of the world as being filled with colour is in error or is incorrect; it is largely an illusion. Some eliminativists, or projectivists, believe colour is a projection of our sensory experience while others believe that nothing is coloured, not even sensations.[11] Dispositionalism (from the English philosopher John Locke) suggests that colours are psychological dispositions or tendencies that produce specific visual sensations in particular persons under particular conditions.[11] In *primativism* objects are coloured but the colours are not dispositions or physical properties of the objects.[11] Modern thinking regards the colour sense as the product of colour opponent processes[13-15] and various colour spaces (such as based on the Commission Internationale de l'Eclairage (CIE) chromaticity diagrams[13] of 1931 or later, or that of Krauskopf [14]) have been proposed as theoretical models to assist in more complete understanding of colour perception. Other approaches involving neural network computational models[16] suggest that colour awareness is a self-organizing and plastic structure[16, 17] in which learning, attention and consciousness are important elements.[17] Naisberg proposes a model,

the biophysical vision model[16], that attempts to explain vision and colour in terms of hardware and software elements commonly found in computational systems. Thus concepts familiar to engineers and others such as logic circuits, sensors, gating devices and central processing units (CPU) are increasingly being used to understand human biosensors (rods and cones), conductive neural pathways and the visual cortex.

In Figure 3, a simple colour space, from Krauskopf [14], is represented with three axes. The labels for the colour space axes are different in the work of different authors depending upon their particular interests and emphasis. (Some authors mainly use psychophysical terminology whereas others use electrophysiological terms.) Such color spaces allow for the study of colour detection or discrimination thresholds within or between individuals as well as of various quantities relating to the electrical signals or responses of individuals to chromatic or achromatic stimuli.

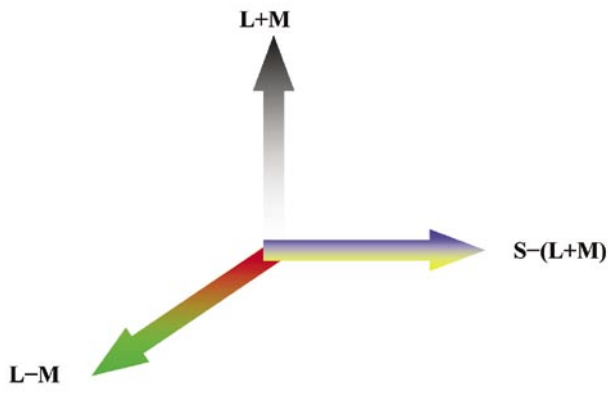

Figure 3: A simple pseudo-3D colour space modified from Figure 16.1 of Krauskopf [14] is indicated with orthogonal axes L−M, L+M and S−(L+M) respectively. The L+M axis is known as the luminance axis while the other axes represent the red-green and blue-yellow chromatic channels or signals.

Conclusion

In future papers this author intends to expand upon the concept of color space, and also to introduce an original approach to the measurement and quantification of the colour sense, and to the understanding of possible deficiencies of colour vision commonly found. This paper serves as background to further understanding the nature of human colour awareness, and its potential relations to the methods to be described later.

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