Ageing effects on colour vision ~Changed and unchanged perceptions~

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ABSTRACT

Physiological age-related changes in the human visual system have been reported such as an increase in the density of the lens, a reduction of pupil size, a reduction of photon absorption efficiency of cones and losses of retinal ganglion cells. Although the retinal stimulus and neural signals are strongly affected by such age-related changes, colour appearance is stable throughout the life-span because of parallel losses of cone sensitivities and other compensation mechanisms related to colour constancy and long-term chromatic adaptation. There is, however, a loss of sensitivity in colour discrimination. The results of experimental studies by the author and colleagues indicate that this loss in discrimination is not only caused by the reduction of retinal illuminance, but also by a decrease in the signal-to-noise (S/N) ratio in neural pathways processing signals from S-cones and antagonistic signals from L- and M-cones. This change can be considered as a trade-off between maintaining a constant signal levels from the cones across the life-span and decreasing the signal-to-noise ratio in the human visual system. Additionally, the analysis of our experimental results indicate no correlation between age-related increment of lens density and the quantity of cone signal reduction.

1. INTRODUCTION

The primary cause of age-related changes of visual performance are physiological. Therefore, I briefly describe physiological changes and their influence.

The first question of this paper is whether or not senescent physiological changes cause changes in colour appearance. I summarize studies of colour appearance and describe possible mechanisms to maintain the stability of colour appearance under such age-related changes. With respect to colour discrimination, it is well known that there are age-related losses in the ability to discriminate colours in many ordinary situations. Thus, the next question is whether or not all age-related loss of colour discrimination is caused only by the reduction of retinal illuminance or whether there are also losses resulting from receptoral and postreceptoral changes. We found that if stimulus intensity is relatively high, the change in colour discrimination is little, but the influence of signal reduction becomes critical around thresholds. It is concluded that although colour appearance changes little with age, the ability to discriminate colour is reduced significantly with age under certain conditions.

The last question considered in this paper considers the correlation between lens density and quantity of signals from cones that were measured psychophysically with some assumptions. There were two possible hypotheses that could lead to the existence of a correlation between them. A positive correlation could be expected because ultraviolet light exposure may accelerate aging of both lens and cones simultaneously. On the other hand, a negative correlation also could be expected because the increment of lens density may reduce the additional damage to cones because the absorption of short wavelength light and ultraviolet light would be increased. The analysis, however, indicated no correlation between them.

2. INFLUENCE OF AGING FROM A PHYSIOLOGICAL POINT OF VIEW

2.1 PRE-RETINAL AGE-RELATED CHANGES AND THEIR INFLUENCE.

About preretinal changes, the ocular media density increases with age, mainly caused by absorption changes in a crystalline lens. The reduction of pupil size with age, sometimes called "senile miosis," also decreases the retinal illuminance of stimuli. Spectral radiance of stimulus changes so that light absorption in short wavelength region increases as the lens density increases.

These changes cause not only a reduction in retinal illuminance, but also changes the spectral radiance of stimuli on the retina.

Although all senescent changes cannot be explained by the density increment of the ocular media density and pupil miosis, when accuracy of data and quantity of dispersion between observers are considered, most of results of visual performance change with aging can be explained by the amount of light change on the retina caused by these two factors in experiments with natural conditions of viewing.

For example, experimental findings such as sensitivity decrement of scotopic and photopic spectral luminous efficiency functions specified at the cornea for short wavelength stimulus can be explained well. Furthermore, age-related changes of colour-matching function and colour discrimination are also influenced by the increment of absorption at the ocular media. About colour discrimination, the score of Farnsworth-Munsell 100-hue tests decreased with aging.[1] The loss of colour discrimination was similar to colour vision deficiency associated with S-cone loss (tritanopia). This type of the decline in discrimination was also observed when stimuli were viewed by young observers through a short wavelength absorption filter.[1]

In the results[2,3] that precisely measured spectral luminous efficiency function by Heterochromatic Flicker Photometry (HFP) method using observers over a wide age, approximately the same function shape was obtained in the long wavelength region, but sensitivity fell with age at short wavelengths. This sensitivity decline is almost consistent with a change of ocular media density by aging. But because a difference of absolute sensitivity between observers cannot be probed by the HFP method, only relative sensitivity between different wavelengths could be measured. To the extent that cone sensitivity itself deteriorates, this result indirectly indicates that sensitivities of L-cone and M-cone sensitivities decrease at the same rate.[4]

2.2 AGE-RELATED RECEPTORAL AND POST-RECEPTORAL CHANGES AND THEIR INFLUENCE

Both reduction of cone density on the retina and sensitivity loss of each cone type can be considered as factors contributing to the age-related loss of cone sensitivity for the light that reaches to the retina. About the cone density, the foveal cone density does not change by aging significantly.[5] However, a loss in cone density is reported in peripheral region.[5] In each cone, anatomical shape changes occur in visual cell outer-segment. Losses in photon absorption with age may be due to decrements of cone outer-segment length, change of alignment of disk-like membranes, disorder of direction of outer segments and degeneration phenomenon of follicle.[6] Because they produce decrements in the quantity of absorption for photons passing cones, cone sensitivity decreases with aging. S-, M- and L-cones sensitivities at the cornea were measured psychophysically for observers from 10 years old to 80 years old using Stiles' two-colour threshold method and a monotonic sensitivity decline of about 0.13 log unit per decade was observed without depending on a type of cone.[4] Regarding post-receptoral changes, age-related loss of ganglion cells occurs in the range of 11 degrees in central retina, and it is reported that the decrement of it was about 15-25% between about 30 and 70 years .[7]

3. SENESCENT CHANGES IN COLOUR APPEARANCE

3.1. COLOUR APPEARANCE IS RELATIVELY STABLE ACROSS THE LIFE SPAN

Age-related changes in colour appearance have been measured with several different methods and in different regions of colour space. The wavelengths of spectral unique hues were measured for 50 observers ranging from 13 to 74 years of age in natural (Newtonian) view.[8] Results show that there is no senescent change in the wavelengths of unique blue and unique yellow. On the contrary, the wavelength of unique green shifted slightly toward shorter wavelengths with increasing age. This result can be explained by a nonlinearity in signal processing by the yellow-blue chromatic opponent channel. Overall, unique hues of monochromatic lights are relatively stable throughout the life span.

Neutral points were measured in terms of additive mixtures of two wavelengths corresponding to unique blue and unique yellow, and verified with mixtures of a long wavelength monochromatic light and the complementary wavelength.[9] There is no change with age in the chromaticity coordinates of these neutral points. Thus, the white point is not affected by the age-related changes of spectral transmittance of the lens.

Hue and saturation of OSA colour chips were measured by a colour-naming method with natural viewing conditions.[10] This experiment investigated the change of colour appearance of broadband surfaces. The results showed that the red-green and yellow-blue proportions do not change with age. This implies that the appearance of (object) colour is stable with age. However, the proportion of the chromatic component relative to the achromatic component was slightly reduced with age on the colour chips of lower values. These results mean that there is no hue shift with age, but saturation is reduced if it is measured with some colour chips in natural view.

For further investigation, the saturation function was precisely measured with monochromatic lights in Maxwellian view with equal (corneal) illuminance and with equal retinal illuminance.[11] The result indicated that there is no statistically-significant difference in the saturation function between young and old age groups. Thus, it is expected that small age-related reductions of the chromatic component in the colour-naming method are caused by a change in the spectral distribution of colour chips on the retina or possible influences of stray light that increases with age. This kind of reduction of the chromatic component was also observed in recent study.[12]

3.2. WHY COLOR APPEARANCE IS SO STABLE ACROSS THE LIFE-SPAN?

As indicated by the experimental data described above, even though the physical change in stimulation on the retina is relatively large with increasing age, there is little age-related change in colour appearance. There are three possible hypotheses that may explain this stability of colour appearance.

(1) Parallel loss of cone sensitivities

There are significant losses of cone sensitivities with age but the decline of sensitivities in each cone type is nearly the same.[4] This parallel loss of cone sensitivities helps to balance relative responses of cones as long as responses of chromatic-opponent channels are linearly related to the signals from cones, because colour appearance is basically determined by the relative intensity of signals from the three types of cone.

(2) Colour constancy type of compensation

Age-related changes in the spectral radiance on the retina can be compensated theoretically by colour constancy mechanisms, such as a von Kries type of adaptation. In the von Kries type of adaptation, responses of cones to the stimulus are affected by adaptation to background illumination. Under this model, the appearance of Munsell colour chips plotted in the cone response coordinates can be almost the same for different age groups.[13] Also, the appearance test on a D65 white stimulus was performed on young observers by changing the chromatic coordinates and luminance on a CRT to simulate age-related increases in light absorption by the lens from 50 to 80 year-old people.[13] The result showed that at least for the white stimulus, the short-term colour constancy mechanism can explain the stability of colour appearance for old observers under 66 years old.

(3) Relatively long-term adaptation

Experimental finding in which colour vision changes by long-term adaptation from 20 to 30 days provides the possibility of plasticity in brain.[14] Recently, this long-term adaptation was more directly measured on patients who had cataract surgery and got intraocular lens (IOL).[15] They measured xy coordinates of achromatic point before surgery and after surgery until one year later. It was found that the achromatic point moves toward a reasonable white and the distance between them were decreased exponentially as a function of logarithm of days after surgery.[15] These results suggest that an adjustment mechanism of nervous system depending on experience is working on. That kind of adjustment mechanism also enables to provide the stability of colour appearance in the life span.

4. SENESCENT CHANGES IN COLOR DISCRIMINATION

My colleagues and I have measured age-related changes in colour discrimination mediated by an S-cone mechanism.[16-18] Stimuli were presented in Maxwellian view and equated in retinal illuminance by individual HFP functions to control for age-related variations in pupil size and to compensate for the change of spectral transmittance of lens between observers, respectively. We also used a temporal two-alternative forced-choice (2AFC) method to measure thresholds in order to avoid possible criterion shifts associated with age.

Figure 1 shows the results plotted in terms of Log ΔS vs. Log S coordinates. The abscissa, Log S, refers to the amount of excitation of S-cones by a standard field, in S-cone trolands. The ordinate, Log ΔS, is the threshold for discriminating between the standard field and a test field, described in terms of the difference in the amount of S-cone excitation. As shown in the figure, the threshold increased with age at low S-cone excitation levels even though relative retinal illuminance was equated for each individual observer. However, there is no difference in threshold between age groups at higher S-cone excitation levels. Because we used constant retinal illuminance for each condition of S-cone excitation level, the age-related change in colour discrimination by S-cones appears only on yellowish colours but not on whitish-blue colours. Equation 2 describes a model under the Weber-Fechner law that we used to analyze the data. In this equation, N denotes the Noise and W denotes the Weber fraction.

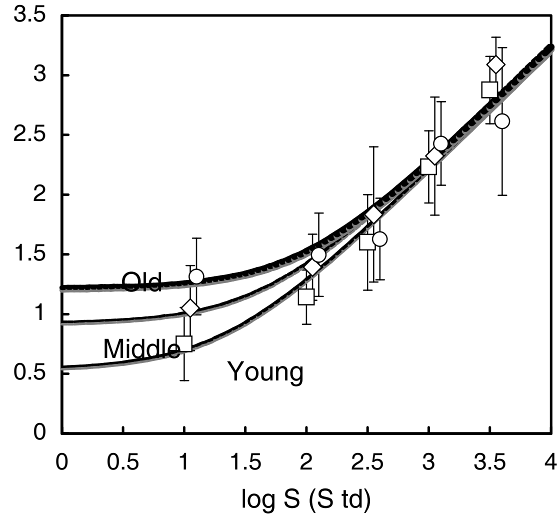

Figure 1: Geometric mean of thresholds in chromatic discrimination mediated by S-cones for young (squares), middle (diamonds) and old (circles) age groups as a function of S-cone stimulation. Error bars represent ±1SD. [Re-plotted from the data in ref.16]

$$\text{S-cone mechanism :}\ \Delta S / (S+N) = W \quad (1)$$

Fits to the data using this model reveal that there is an age-related increase of noise and/or a loss of cone quantal efficiency (in this case, S-cone), but no change in Weber fraction. The sensitivity defined as the Weber fraction is stable with age.

This result means that there are changes of visual discrimination with aging that may be problematic, even if the change of colour appearance is not so large as shown in the previous section. Because cone signal strength decreases, it can not be avoided that signal / noise (S/N) rate turns worse even if it is assumed that there is some compensation.[16,18] Thus, it causes a decrease in the ability of colour discrimination around the level of thresholds. It means that if stimulus intensity is high enough, the change in colour discrimination is little because the Weber-Fechner law is generally maintained. However, the influence of signal reduction becomes critical around thresholds.[16,18,19]

We have also measured age-related changes in wavelength discrimination using a Maxwellian-view optical system, with retinal illuminance equated by individual HFP functions, and use of a spatial 2AFC method.[17,18] The mean difference in threshold at all wavelengths was about 3 nm between groups of young and old observers. The amount of change in thresholds, however, is not systematically related to wavelength. The data were analyzed in terms of a modified version of the Boynton-Kambe colour discrimination equations.[18,20] Analyses of the wavelength discrimination functions using this model indicate that for wavelength discrimination determined by signals from L- and M-cones processed in chromatically-opponent channels, the increase in threshold with age is mainly caused by an increase in the Weber fraction. This means that the sensitivity of the L-M mechanism itself declines with age.

5. DISCUSSION

5.1. SENESCENT CHANGES IN COLOR APPEARANCE AND DISCRIMINATION

In many cases, colour appearance is stable through the life-span even while there is a loss in the ability to discriminate colour differences. These effects are observed even with compensation for retinal illuminance. Because of senescent anatomical and physiological changes, signals from cones and responses from channels processing antagonistic cone signals have to be amplified more with increasing age to maintain colour appearance. However, this appears to have the consequence of making the signal-to-noise (S/N) ratio worse with age and causes the loss in colour discrimination in certain conditions, when signals are relatively weak, but not for all conditions. This can be considered as a sort of a trade-off between maintaining colour appearance and losing the accuracy of colour discrimination with aging of the human visual system.[21]

5.2. RELATION BETWEEN AGE-RELATED INCREASES IN LENS DENSITY AND REDUCTIONS OF CONE SIGNALS

I examined the relationship between increment of lens density and reduction of signal from cones. I calculated the correlation between the luminance rate between blue and red phosphors and an amplitude of impulse response functions that were measured in our expetiment.[22] At first, I analyzed individual results of the preparation experiment that was performed to correct a luminance change of each phosphor by the lens density change in the experiment.[22] Change of luminance rate between blue and red phosphors is examined when the minimum flicker between each phosphor was measured. I expect that this luminance rate reflects the amount of age-related increment of the lens density.

Secondly, I inspected the result of psychophysical measurement about a decline of signal originated from cones. In our experiment with Maxwellian-view optical system, after the adjustment of luminance, we measured luminous impulse response functions by a double-pulse method.[22] Because signals from cones decrease with aging, threshold value for luminous impulse rose even if spectral correction of stimuli was performed. Shape of individual impulse response functions (IRFs) was derived from threshold changes when an inter-stimulus interval of two pulses was changed. I assume that the positive peak amplitude of IRFs quantitatively reflects the intensity of signals from cones through a luminous pathway.

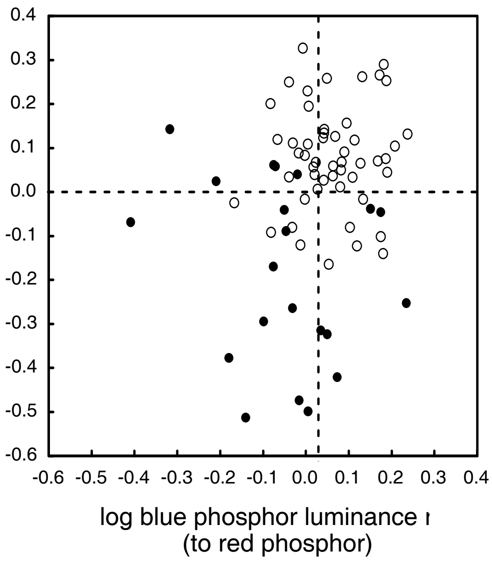

Figure 2: Logarithmic maximum amplitude of individual impulse response function (IRF) nor-malized at the average as a function of logarithmic blue phosphor luminance rate to red phosphor. Dotted lines denote the average of all 72 observers. Filled circles denote the data of 21 observers over 65 years old. [New plot from the data in ref. 22]

Under these assumptions, I plotted the peak amplitude of the IRFs as a function of the logarithm blue phosphor luminance rate. In figure 2, the abscissa refers the logarithmic blue phosphor luminance rate and the ordinate refers to the logarithmic maximum amplitude of individual impulse response functions. The correlation between them is weak as shown in the figure (correlation coefficient =0.19), and a prediction is not possible from the regression line (level of significance 1%). When data points for observers over 65 years old are denoted by filled circles, only a small tendency can be observed in that data points gather in the left bottom. Therefore, the influence of the lens density and the influence of the signal reduction (a cone sensitivity decline) contribute to increase the individual difference separately.

This result is scientifically interesting. Because the increment of absorptions by the lens for short-wavelength light can decrease damage to the retina by ultraviolet light, I expected that it may have a role in preventing an additional decline of the cone signal as rational hypothesis of the lens density increment. If this hypothesis is correct, there should be some correlation between them. Such correlation, however, was not observed. Thus, I cannot say that lens density increases restrain an aging effect of cones and the retina. After all, it is rational to think that damages by ultraviolet light accumulates in both lens and cones, but it affects them independently or not in a simple way.

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