Action spectrum for photophobia

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Thresholds for photophobia (light-induced discomfort) were determined at wavelengths from 440 to 640 nm for three subjects. Photophobia was assessed by means of electromyography, which was used to measure subjects’ level of squinting. After correction for absorption by macular pigment and the ocular media, subjects’ functions displayed a trend of increasing sensitivity with decreasing wavelength. We propose that the corrected function is indicative of increased sensitivity to potential retinal damage by short-wavelength light. It is therefore suggested that photophobia serves a function of biological protection. Results also suggest that photophobia is significantly mitigated by macular pigment in the short wavelengths. © 2003 Optical Society of America

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1. INTRODUCTION

Photophobia results from exposure to light that is of sufficient intensity to produce discomfort. Typical behavioral responses to this discomfort include squinting, wincing, directing the eyes down toward the ground (in the case of intense sunlight), or simply looking away from an intense light stimulus. The perceived level of discomfort is typically commensurate with the level of aversive response. In the clinical literature, photophobia has been defined as resulting in the induction or the exacerbation of pain upon exposure to light. Little is known about the neurophysiological processes that mediate photophobia. Because pain-signaling fibers of the trigeminal nerve terminate in the dilator and constrictor muscles of the iris, it has been suggested that the vasodilation accompanying the trigemino-pupillary reflex is the sensitizing factor that makes the pupillary light reaction painful. Indeed, it has been demonstrated that an intact trigeminal nerve is necessary to experience photophobia. Iris constriction, as might be expected, has been shown to increase with increasing illumination; at the point when the light intensity becomes truly discomforting, however, the iris appears to constrict and dilate irregularly every few seconds. This phenomenon is known as hippus and is thought to occur because of simultaneous, antagonistic activation of sympathetic and parasympathetic pupillary responses. More recently, it has been shown that hippus is not consistently associated with subjective reports of visual discomfort. King suggested that the rapidity of iris constriction, and intense stretching under lighting conditions that produce maximal constriction, appear to be closely related to visual discomfort. Unfortunately, these factors have proved difficult to measure and thus cannot be reliably used as objective measures of visual discomfort.

In the clinical literature, photophobia is recognized as a symptom of various pathological conditions such as iritis, achromatopsia, conjunctivitis, trigeminal neuralgia, tumors compressing the anterior visual pathways, and, most prominently, migraine headache. Photophobia is also commonly noted as a symptom in albinism and has also been found to coincide episodically with panic disorder and depression. In the migraine headache literature, it has been suggested that photophobia experienced by migraineurs between attacks may be due to abnormalities in spatial and temporal visual processing. Given that there are considerable backprojections from the striate cortex to the lateral geniculate nucleus, it has been suggested that the apparent subcortical processing abnormalities (i.e., spatial and temporal) in migraineurs produce a geniculostriate feedback loop, which results in cortical hypersensitivity and thus abnormal sensitivity to light.

A more thoroughly studied phenomenon related to photophobia is discomfort glare. Discomfort glare is generally defined as a subjective impression of discomfort upon exposure to light. Researchers studying discomfort glare have adopted subjective rating scales, termed visual analog scales. The most popular rating scale used is the de Boer visual analog scale, with subjective ratings ranging from “just noticeable” to “unbearable.” There have been a few studies (e.g., Refs. 19 and 20), however, reporting the use of electromyography as an objective measure of discomfort. Investigators from these studies measured the muscular activity associated with squinting and blinking around the ocular orbit upon exposure to a glare stimulus and found good agreement with subjective ratings of discomfort. General findings from discomfort glare studies (see Ref. 21) include the following: (a) Increases in luminance give rise to increases in visual discomfort; (b) increases in pretest adaptation luminance result in a decrease in visual discomfort; (c) increases in size of the test stimulus, while maintaining a specified luminance level, result in increased visual discomfort; (d) visual discomfort is greater for stimuli viewed centrally rather than peripherally; and (e) discomfort thresholds measured psychophysically are especially variable among subjects.

Whereas discomfort glare is certainly related to photophobia, it is difficult to make a direct comparison between the two. In discomfort glare studies, subjects are rarely,
if ever, exposed to lights that are subjectively judged as "intolerable." By contrast, photophobia involves an acute intolerance to light, typically marked by some sort of behavioral aversion (e.g., squinting or closing the eyes).

Because photophobia is usually thought of as a symptom of pathology, its assessment in normal subjects has been limited.\(^8,22,23\) As a result, little is known about how basic stimulus variables affect photophobia. For example, it is not known how the wavelength of light affects photophobia thresholds, and that is the purpose of the present study: to determine the action spectrum for photophobia. Discomfort, not photophobia, thresholds to lights of various spectral compositions have been documented in a few studies. Main et al.\(^{24}\) obtained subjective discomfort thresholds in both migraineurs and healthy subjects for three broadband spectral stimuli, which were judged to correspond to "blue," "green," and "red" lights. Their subjects directly viewed light from a slide projector at a distance of 0.6 m. The intensity of the lights was increased by the experimenter until the subject noted that the stimulus was uncomfortable to view. Whether there was an aversion of some sort by the subjects was not stated. The method and the rate of increasing the intensity were not specified by the authors. Additionally, the angular size of the stimulus was not disclosed (although we calculate the angle, assuming a standard projection lens diameter, to be approximately 4.8 deg). Their healthy control subjects were found to have lower discomfort thresholds, measured in photometric units, for the short- and long-wavelength weighted stimuli, relative to the medium-wavelength weighted stimulus. Our effort to transform their photometric data into radiometric data, in order to provide a rough energy-based action spectrum, proved to be heavily dependent on assumptions (e.g., spectral emission of the light source). It is therefore difficult to speculate on actual sensitivity. Moreover, because broadband (not monochromatic) stimuli were used, it was not possible to generate a true action spectrum from these data.

Flannagan et al.\(^{25}\) measured discomfort glare to six 1-deg, monochromatic lights (ranging from 480 to 650 nm), presented 7 deg peripheral to fixation. The researchers used a slide projector and interference filters to present the stimuli. The subjects viewed light from the slide projector directly from a distance of 2.69 m. Subjects were instructed to rate, using a modified de Boer visual analog scale, the various stimuli according to relative subjective discomfort. Like Main et al.,\(^{24}\) they found that relative discomfort (based on photometric measures) was greater in the short- and long-wavelength regions as compared with that in the middle-wavelength region. To examine their results as an action spectrum, we used radiometric data provided by Flannagan et al.\(^{25}\) and converted ratings of "disturbing" into sensitivity measures. The resulting function is negatively sloped, indicating increasing sensitivity with decreasing wavelength.

The aforementioned studies have attempted to characterize the effect of intense lights of various spectral compositions on visual discomfort. Although it appears that the action spectrum for visual discomfort may be different from \(V(\lambda)\) or any other common spectral sensitivity function, the specific characterization of the wavelength dependence of photophobia has yet to be sufficiently determined. An action spectrum for photophobia could reveal the receptor mechanisms involved in mediating photophobia. Furthermore, the shape of the function could yield information about postreceptoral contribution. For the present study, we consider photophobia to be an experience of intolerable light intensity that induces a behavioral aversive response. We hypothesize that the squinting response would provide such a measure of aversion. The squinting response would seem to be an ecologically valid measure of discomfort, as there would be no other explanation for a person to squint in the presence of an intense light other than to alleviate the discomfort associated with that light. From this reasoning, we derived our operational definition of photophobia: Photophobia is experienced at the point when a light becomes sufficiently intense so as to elicit a criterion squinting response. In a manner similar to that used by Berman et al.\(^{19}\) and Murray et al.,\(^{20}\) we used an objective method, electromyography (EMG), to record squinting responses to briefly presented, intense lights.

2. METHODS

A. Subjects

Two of the authors (males, aged 27 and 30 yr) served as subjects. Additional data were obtained from a third subject (female, aged 29 yr), who was naïve to the specific aims of the experiment. All subjects were normal trichromats with no history of visual pathology. To ensure normal trichromacy, subjects' color vision was assessed by using the Ishihara Pseudoisochromatic Test Plates and the Farnsworth Dichotomous Panel D-15 Test. The study was approved by the University of New Hampshire's Institutional Review Board for the Protection of Human Subjects, and all subjects willingly consented to participate in the experiments.

B. Apparatus

A three-channel standard Maxwellian-view system with a 1000-W xenon arc lamp was used. In one channel, two low-level red fixation points, oriented diagonally about the center of the dark-adaptation stimulus, were used as a guide for subjects' fixation. The second channel was used for the presentation of a shuttered low-intensity white light, which served to assess subjects' pretest dark-adaptation level. The third channel provided the test stimulus. Lights in the test stimulus channel were rendered monochromatic by Ditric Optics interference filters (mean half-bandwidth of 8.5 nm, ranging from 7 to 10.5 nm). A neutral-density wedge was used to adjust the intensity of the test stimulus. Photophobia-inducing energy levels were measured after each session with a UDT radiometer (Optometer 61).

A Grass Instruments model 7P3B EMG preamplifier/amplifier was coupled to one channel of a Gould Brush 220 chart recorder for the analysis of squint muscle potentials. For precise determination of the portion of the chart recording that corresponded to stimulus presentation, a photocell was positioned orthogonally to a beam splitter through which light from the test channel passed.
Output from the photocell provided input for the second channel of the chart recorder.

C. Procedure
Subjects maintained a stable alignment by biting down on a premade dental impression and leaning their head against a forehead rest assembly. A pupillary alignment procedure was performed to ensure that the light from the optical system was in focus in the plane of the subject’s pupil and passing through the center of the subject’s pupil. A constant pretest dark-adaptation level was ensured by subjects’ detection of a 5.0-log cd/m², 1-s flash of a white-light stimulus, presented every 6 s to the central 5.75 deg of the retinas of their right eyes. Subjects were instructed to fixate the center of an imaginary line drawn between the two low-level, diagonally oriented, red fixation points (separated by 7 deg) and attempt to detect the low-level, white-light stimulus upon shuttered presentation. Upon detection of three consecutive flashes, the subject signaled the experimenter. The scotopic-level white light was then presented continuously for 1 min, and the subject was instructed to maintain fixation. This was done to ensure equal adaptation of the retinal area to be stimulated by the test field. After the 1-min adaptation period, the subject was presented with the test stimulus, a light of predetermined wavelength and intensity, subtending 5.75 deg of visual angle, for 5 s.

EMG was used to record muscle potentials associated with subjects’ squinting responses. Surface electrodes were attached to the right temple (reference), to the upper cheek, below the right eye’s lateral canthus (test), and on the back of the neck (ground). EMG was used solely for the determination of a threshold photophobia response; the criterion threshold photophobia response was based on the amplitude and the duration of squinting. A continuous squinting response that lasted at least half the duration of the test stimulus presentation and that reached, at any point during the continuous squinting, a 4:1 signal-to-noise ratio (squinting activity/baseline) was considered a threshold photophobia response. Occasionally, a subject blinked or exhibited a “startle squint” upon presentation of the test stimulus, followed by normal fixation or some squinting (depending on the discomfort caused by the test stimulus) for the remainder of the stimulus presentation. We attributed the startle reaction to the photic startle response. On traces where the photic startle response was evident, we did not factor the startle amplitude or duration into the photophobia threshold criteria. Figure 1 shows an example of a typical criterion photophobia response.

The method of ascending limits was employed. A relatively low-intensity test stimulus was initially presented, and, in steps separated by dark adaptation to a constant level, the intensity of the stimulus was increased in order to reach the photophobia threshold.

The intervening dark-adaptation trials extended the duration of the experimental session considerably. As a way of expediting each photophobia threshold measurement, the following procedure was employed. During the experiment, after each trial, subjects were instructed to rate the discomfort level of the test stimulus on a scale from 1 to 10, 10 being their subjective experience of photophobia. The rating was then inserted into a simple equation,

\[
I = (10-R) \times 0.05, \tag{1}
\]

where \(I\) is neutral density to be removed from the test channel and \(R\) is subjective rating. For example, if the subjective rating of the test stimulus were 7 on a given trial then, for the subsequent trial, 0.15 log unit of neutral density would be subtracted from the test beam by means of the neutral-density wedge. This procedure was repeated until the subject reached the criterion photophobia response, as described above. A rating of 10 was not considered a measure of threshold photophobia—the EMG criteria described above were exclusively used to determine photophobia thresholds. In our many trials, however, we had only one coincidence of a 10 rating and nonphotophobia EMG criteria. In that case, we subtracted 0.10 log unit of neutral density from the test beam; the ensuing trial produced a threshold photophobia response, as determined by EMG.

Wavelengths from 440 to 640 nm in steps of 20 nm were assessed. Because of the extreme length of each session (typically 2–3-h duration), three sessions were needed to complete an action spectrum; curves from each session were least-squares fitted on the basis of photophobia thresholds at three normalizing wavelengths, obtained during each session: 520, 600, and 640 nm. In this way, any response variability arising from differences in elec-
trode placement, subjects' day-to-day absolute sensitivity, or output of the optical system could be offset by a scalar shift along the axis of ordinates. Two action spectra were completed for each subject.

3. RESULTS

The two photophobia action spectra \([P(\lambda)]\) for observers JS and AW are presented in Fig. 2. Peak sensitivities (on an energy basis) are normalized to zero. The similarity of the curves within and between subjects suggests that the methods used facilitated reliable measures. Both subjects’ data exhibit a large notch, with a minimum sensitivity point corresponding to 460 nm. Both subjects’ curves are relatively broad when compared with either \(V(\lambda)\) or \(V'(\lambda)\). In fact, an envelope of the combination of \(V(\lambda)\) and \(V'(\lambda)\) makes for an approximate fit to our observer’s \(P(\lambda)\) functions when either \(V(\lambda)\) or \(V'(\lambda)\) is allowed to shift along the Y axis for a least-squares fit (Fig. 3). The prominent notch, however, remains as a major discrepancy between \(P(\lambda)\) and the combined \(V(\lambda)\) and \(V'(\lambda)\) functions. The primary difference between the two subjects’ curves is the wavelength of peak sensitivity. JS’s function peaks at 500 nm, whereas AW’s peaks at 520 nm.

Of particular interest to us was the large notch centered at 460 nm. Common spectral sensitivity functions such as \(V(\lambda)\) and \(V'(\lambda)\) do not show this pronounced notch. To explore further the nature of the notch and to examine more closely the possible trend of increasing photophobia sensitivity with decreasing wavelength below 460 nm, we had both observers participate in a session in which photophobia thresholds were assessed at wavelengths from 440 to 500 nm in steps of 10 nm. Additionally, a third observer was enlisted to confirm the overall shape of \(P(\lambda)\), including three wavelengths within the region of the notch. The results of these supplemental sessions are presented in the top panel of Fig. 4, where it can be seen that 460 nm maintained its position as the minimum-sensitivity point relative to the other short wavelengths tested. The curve of the third observer, NK, closely approximated that of the other subjects, including the appearance of the pronounced notch at 460 nm.

From the results presented in the top panel of Fig. 4, it was apparent that, at wavelengths below 460 nm, our subjects’ sensitivity increased with decreases of wavelength. In an attempt to determine if subjects’ sensitivity would continue to increase at wavelengths shorter than 440 nm, we tested at 420 nm. We did not have
enough energy at 420 nm to elicit a criterion squinting response in either subject, but both subjects viewed this wavelength at maximum transmittance and each rated the experience a 9. Because we had obtained scaling data during the experiment, we were then able to take relative energy measurements for all wavelengths at their 9 rating. For the few wavelengths that were never rated a 9 but were rated 8 and 10, a linear interpolation was used to determine our criterion response of 9. Although this procedure is admittedly crude, the resulting action spectra bear similarity to those derived by EMG, as shown in the bottom panel of Fig. 4. In fact, a strong significant correlation was found between subjects’ ratings of threshold photophobia and EMG-derived thresholds (JS: $r = 0.976, p < 0.001$; AW: $r = 0.982, p < 0.001$). More importantly, the results suggest that sensitivity to lights below 440 nm increases.

### 4. DISCUSSION

A possible explanation for the pronounced notch found in our subjects’ $P(\lambda)$ functions could be the absorption of light by macular pigment (MP). The wavelength of peak absorption for MP is 460 nm, which is precisely where the point of minimum sensitivity is found in our observers’ $P(\lambda)$ functions. MP is symmetrically distributed about the fovea; the optical density of MP decreases exponentially from its peak in the fovea outward to an asymptote at approximately 5.5 deg of angular subtense. Although the exact function of MP is not certain, a protective role against damaging short-wavelength light has been hypothesized.

MP optical density (MPOD) is conventionally measured psychophysically by the method of heterochromatic flicker photometry (HFP). HFP thresholds are largely determined by receptors stimulated by the edges of the test fields used. Thus the derived optical density values are for MP at the retinal loci corresponding to where the edges of the field are. During the course of our study, we found preliminary evidence for spatial summation of the photophobia response over relatively large field sizes (from 1 to 6 deg). Preliminary data collected for an upcoming study also indicate that the photophobia response exhibits robust spatial summation throughout a range of stimulus sizes (from 5 to 30 deg). We therefore hypothesize that photophobia thresholds are determined by retinal mechanisms summed over the total area stimulated by the test field, not just those located at the edges of the field. Thus the attenuation factor by MP would be considerably greater than simply the HFP-derived MPOD value, which reflects optical density at only the edges of the test field.

To correct our observers’ $P(\lambda)$ functions for the attenuation of MP, we mathematically integrated MPOD over the area stimulated by the test field. This was done by using spatial profiles of MPOD (previously obtained for the temporal retina), measured at 460 nm, for subjects JS and AW. The integration was then simply doubled to account for the absorption of light by MP over the nasal half of the retina stimulated by the test stimulus. In obtaining the total absorption for MP in this way, one assumes that MP is distributed symmetrically about the foveola.

This has generally been found to be the case. A further assumption is that the putative spatial summation effect for photophobia is linear. Our preliminary data regarding spatial summation of the photophobia response suggest an approximately linear relationship between stimulus energy and stimulus area for stimuli subtending from 5 to 30 deg.

Because the relative absorption of MP as a function of wavelength has been well documented, we were able to correct for MP attenuation at the other wavelengths used in the study, relative to our 460-nm correction value. We used the MP absorption template of Bone et al. to accomplish this. Ocular media absorption was corrected for by using the values recommended by Wyszecki and Stiles. The MP- and ocular-media-corrected $P(\lambda)$ functions are presented in Fig. 5. With the correction for MP attenuation and ocular media absorption, the shape of the $P(\lambda)$ functions is essentially monotonic—a general trend of increasing sensitivity with decreasing wavelength is apparent. As a general test of our correction, we determined thresholds for photophobia to 460- and 540-nm lights at a retinal eccentricity outside that which contains MP. To elicit photophobia, we increased the stimulus to 22 deg. The subject was instructed to fixate a small point of light located 31 deg temporal to the center of the test stimulus. This stimulus configuration ensured that the entire test stimulus was placed beyond the spatial extent of MP. In support of our correction, the relationship between 460 and 540 nm was found to be reversed with respect to whether viewing was foveal or peripheral (Fig. 6). Owing to the large difference in stimulus size and potential differential wavelength effects of spatial summation, however, direct comparisons between central and peripheral viewing conditions regarding MP should be made tentatively.

The negative slope of the corrected functions is similar to the discomfort sensitivity data from Flannagan et al. plotted for comparison in Fig. 5. It is important to note that, in their study, the glare source was presented 7 deg in the periphery (a retinal region that contains no MP). For this reason, we can make a direct comparison be-
between our corrected functions (which reflect zero absorption by MP) and their function. We plotted data for only the three wavelengths (out of six) from the study of Flannagan et al. for which radiometric data were provided that corresponded to ratings of “disturbing” (i.e., equivalent to reaching photophobia threshold). Because Flannagan et al. presented their stimuli in free view, pupillary modulation of light entering the eyes may be the source of the difference in slope between the two.

Our corrected functions are fitted to a first approximation by the rhesus macaque threshold-energy retinal damage function reported by Ham et al. (Fig. 5). They used laser illumination of various wavelengths at a parafoveal location to produce a threshold change in fundus appearance, which came about primarily as a result of retinal pigment epithelium damage. Ham et al. note that their action spectrum approximates the absorption spectrum of melanin; melanosomes in the retinal pigment epithelium appeared to be involved in the lesions. Their testing at a parafoveal region of the retina effectively precluded the absorptive effects of MP. Although all four of the exposure-time-based sensitivity functions of Ham et al. fit our data well, their 1000-s data were used for comparison, as those light levels were the closest in intensity to the light levels used in our study, albeit substantially more intense. In particular, the general similarity between the functions of increased sensitivity with decreasing wavelength is striking. A similar relationship has been established by Gorgels and van Norren (Fig. 5). They suggested that melanin absorption spectra of these pigments can be compared with absorption spectra of retinal melanin, melanopsin, and metarhodopsin II. Pigments have been adjusted on the Y axis to allow for a composite best fit to $P(\lambda)$.

Corrected $P(\lambda)$. We suspect, however, that this very approximate fit is the result of curve fitting and not the revelation of a physiological basis of photophobia. Given our results, one could hypothesize that the function of photophobia is biological protection. An aversive response (squinting) that is biased in sensitivity toward potentially damaging short-wavelength light is suggestive of this. Moreover, the close approximation of our subjects’ corrected $P(\lambda)$ functions to the threshold-energy retinal damage function in rhesus monkeys found by Ham et al. further supports this conclusion.

Our data suggest that MP plays a major role in the attenuation of photophobia, substantially greater than what would be expected from spatial averaging of MPOD. For photophobia, it appears that MP acts as a spatially integrated filter. For this reason, even a small amount of spatially integrated MP could prove to significantly reduce photophobia mediated by central viewing.

With respect to MP’s role in attenuating photophobia, if we assume the $P(\lambda)$ notch to be primarily the result of MP, then, hypothetically, a subject with very little or no MP might produce a $P(\lambda)$ function that indeed decreases as a function of wavelength in a fashion similar to that exhibited by our corrected $P(\lambda)$ functions. The authors intend to pursue this point in a future study.

We maintain that the squinting response is a valid measure of photophobia. However, the high correlation between subjects’ ratings of threshold photophobia and objectively determined thresholds for photophobia suggests that, in future studies, photophobia thresholds may confidently be determined psychophysically by using carefully controlled experimental procedures and scaling methods such as those described in the present study.

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REFERENCES


