Absolute Thresholds in Human Infants Exposed to Continuous Illumination

Russell D. Hamer,* Velma Dobson,* and Melanie J. Mayer†

Continuous illumination at low-to-moderate photopic levels can cause damage to the visual system in nonhuman species. Therefore, the authors sought to determine whether behaviorally measurable visual deficits occurred in young human infants who had been exposed to long-term, continuous illumination in a neonatal intensive care unit (NICU). Using the forced-choice, preferential-looking technique, the authors measured absolute thresholds for detection of a 502 nm stimulus in nine preterm infants who had been exposed to 13 to 46 days of continuous room illumination. Nine infants born at term, who had experienced ≤5 days of continuous illumination, served as controls. The thresholds for the light-exposed and control infants did not differ. In addition, the light-exposed infants did not differ from control infants in their performance on a rapid acuity screening under photopic conditions. Thus, the present data provide no evidence of functional damage to either rod or cone vision in infants who had been exposed to continuous illumination in an NICU. Some limitations to the generality of these conclusions are discussed. Invest Ophthalmol Vis Sci 25:381-388, 1984

Infants born prior to term often require the sustained medical intervention provided by neonatal intensive care units (NICUs). Infants in the NICU experience an environment quite different from that of the typical newborn who goes home 3 days after birth. One difference that may influence visual development is that, because lights in the NICU remain on at all times to allow the medical staff to monitor infants' color and other vital signs, the infants are exposed to continuous illumination from birth until they are discharged from the NICU.

In the past 15 years, a large body of research has provided overwhelming evidence that continuous light, even at intensities far below those necessary to cause thermal damage to the retina, can cause significant damage to the visual system in several nonhuman species, including primates. This damage has been documented anatomically, physiologically, and psychophysically (see Lanum for a review).

Figure 1 shows some examples of light levels and exposure durations that have been found to cause damage in animals (downward-pointing arrows), compared with representative light levels measured in a local NICU (upward-pointing arrows) and the range of light levels reported for phototherapy units used to treat hyperbilirubinemia in infants (thickened portion of axis). The adult albino rat appears to be the most sensitive to damage, probably because of its lack of pigmentation. Young newborn albino rats seem to be somewhat less susceptible to light-damage due, in part, to the recovery mechanisms that appear to be stronger in immature rats than in adult rats. However, even the young albino rat will sustain irreversible damage to its visual system, if exposed to moderate levels of illumination for a long period of time.

Studies from adult and infant primates are presumably most relevant to humans. Unfortunately, none of these have looked for evidence of damage at low illuminance levels and long exposure durations comparable to those found in an NICU. The lowest illuminance level and longest exposure duration tested on adult primates was 550 ft-c of cool fluorescent light applied for 12 hr. Messner, Maisels, and Leure-DuPree (1978) have performed the only study, to date, that has investigated infant primates' suscepti...
ILLUMINATION LEVELS CAUSING DAMAGE IN ANIMALS

**ADULT ALBINO RAT** (12 DAYS)  
Rapp & Williams (1980)

**ADULT ALBINO RAT** (4 DAYS)  
O'Steen & Anderson (1971)

**INFANT MONKEY** (12 HOURS)  
Messner et al. (1978)

**ADULT MONKEY** (12 HOURS)  
Sykes et al. (1981)

Fig. 1. Representative illumination levels that have been found to cause damage in animals (downward-pointing arrows), along with three representative levels measured in the NICU (upward-pointing arrows). The horizontal bars associated with each upward-pointing arrow represent the range of levels measured under each condition. The axis is in ft-c and is spaced logarithmically. The thickened portion of the axis represents the illumination level of typical phototherapy units. For each of the animal studies, the shortest duration of exposure found to cause damage at each of the indicated illumination levels is given in parentheses. Damage reported in each case was: loss of photoreceptor cells in the form of a criterion of 35% reduction of outer nuclear layer (ONL) thickness (Rapp and Williams); outer segment (OS) destruction, ONL damage and reduced amplitude of evoked potentials recorded in the optic tract, lateral geniculate nucleus and visual cortex (O'Steen and Anderson); damage to rod and cone inner segments and nuclei (Messner et al.); and damage to cone OSs (Sykes et al.).

ability to light-damage, exposing neonatal macaques to 400 ft-c of cool fluorescent light for 12 hr to 7 days. Their results showed damage to the pigment epithelium and the outer segments and cell nuclei in both rods and cones after only 12 to 24 hr of exposure. These authors claim that at these short exposure durations, damage was more evident in rods than in cones. However, after 7 days of continuous illumination, advanced degeneration of both rod and cone outer segments and nuclei was noted. However, lower light levels and longer exposure durations were not explored, and postexposure recovery period was not varied.

Since the illumination levels in the NICU fall within the range found to cause damage in animals, it would seem prudent to examine visual function in human infants who have been exposed to continuous illumination in the NICU. One previous study used electroretinogram (ERG) amplitudes and ERG measurement of the rate of dark adaptation to examine rod function, and visual acuity tests in order to examine cone function in 4-year-old children who, as infants, had been exposed to 6 days of continuous illumination in a premature nursery. No evidence of rod or cone damage was found in these children.

The primary purpose of the present study was to examine rod function in infants exposed to continuous illumination. To assess rod function, we compared the absolute thresholds of a group of light-exposed infants born prior to term with those of a control group of infants born at term, who were exposed to much shorter durations of continuous illumination. We also made a rough check of each infants' cone function by means of a rapid acuity screening procedure. The present study differed from the previous study in three important ways. First, subjects were exposed to longer durations of continuous illumination (13 to 46 days vs 6 days in the previous study). Second, subjects were tested at 3 weeks postterm age (vs 4 years). Finally, a behavioral rather than an electrophysiologic technique was used to measure visual function.

### Materials and Methods

#### Procedure

The procedure used was virtually identical to the modification of Teller's forced-choice, preferential-looking (FPL) technique used by Powers et al. to measure scotopic spectral sensitivity in 1- and 3-month-old infants. A dark-adapted infant is held in front of a large translucent screen upon which a stimulus is projected from the rear. On each trial, the stimulus is presented either to the left or to the right of the center of the infant's field of view by an adult experimenter. Side of presentation (left or right) and intensity of the stimulus are designated by a predetermined, pseudo-random sequence. On each trial an adult observer, who is uninformed as to the position and intensity of the stimulus, views the infant's face via an infrared-
sensitive television monitor system and judges the position of the stimulus by watching the infant’s eye and head movements. The observer receives feedback after each trial.

In the present study, each infant was tested with four or five stimulus intensities. The intensities were separated by about 0.5 log unit (lu), spanning a 1.5 to 2.0 lu range.

Each experimental session began with a minimum of 12 min of dark adaptation, during which time the only illumination in the room was a 60 watt tungsten source covered by a red filter (−0.7 log cd/m²). After the dark adaptation period was completed, the red light was turned off and testing proceeded in total darkness. Breaks were taken whenever necessary to accommodate the infant’s need for food, comfort, etc, during which time the red light was turned on. Testing continued in this fashion until either 1 hr had elapsed or the infant was no longer in a testable state (too sleepy or too fussy). The stimulus sequence was continued in the next session (usually the following weekday); this process continued until a minimum of 20 trials at each of the test intensities were completed.

Apparatus, Stimulus and Calibrations

The apparatus has been described previously. The stimulus consisted of 502 nm light produced by passing light from a 45 W tungsten-halogen source through an interference filter (half-bandwidth = 10 nm). Stimulus intensity was varied using a combination of Oriel and Wratten #96 neutral density filters. An opaque stop placed in the collimated portion of the projection beam produced 5 large vertical stripes (three light and two dark) in the circular stimulus as it was projected onto the screen. The entire stimulus, when viewed from the plane of the subject’s eyes, subtended 13.5° of visual angle, with each stripe subtending 2.7°. The center of the stimulus was positioned 26° to the left or right of the center of the screen.

The actual attenuation (for the 502 nm narrow-band light) of all the neutral density filters used in this study was measured using a calibrated silicon photodiode (United Detector Technology, PIN 10 DPF).

Ambient Illumination Measurements

An estimate of the ambient illumination in the neonatal intensive care unit was made with a calibrated Gamma Scientific radiometer (Model #2400) used in its photometric configuration. For each reading, we measured the maximum the light reflected from a standard, diffuse reflecting surface (reflectance = 0.81) by using a small cosine detector with the radiometer. Measurements were made in the daytime, on both sunny and cloudy days, and at night at several representative stations throughout the nursery over the course of 4 months (April through July, 1980). The highest and lowest light levels measured, average daytime and nighttime levels, and an overall (average) ambient illumination estimate are summarized in Table 1. We have made some estimates of the retinal irradiance in a premature infant’s eye during a typical sunny day in the NICU (Table 1). Since these estimates involved many assumptions and calculations, these are presented separately in an Appendix.

Acuity Screening

Upon completion of the scotopic threshold test procedure, each infant’s acuity was screened using the diagnostic stripes procedure developed by Dobson, Teller, Lee, and Wade. In this procedure, the forced-choice, preferential-looking (FPL) technique is used to determine whether or not an infant can detect an acuity grating that is detected easily by most infants of the same postterm age. We used a grating composed of black-and-white stripes 40 min of arc (.75 cy/deg) in width, which previous research has shown can be detected by approximately 95% of both full-term infants and preterm infants tested at 1 month after their due date.

Infants

Nine infants who had stayed in the hospital nursery for 17 to 49 days (mean = 30 days) comprised the experimental (light-exposed) group. Seven of these infants underwent phototherapy for several days (Table

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Table 1. Luminance measurements* in neonatal intensive care unit

<table>
<thead>
<tr>
<th>Sunny day</th>
<th>Direct sunlight entering room</th>
<th>No direct sunlight entering room</th>
<th>Overcast day</th>
<th>Night</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>630.0</td>
<td>91.4</td>
<td>114.6</td>
<td>14.9</td>
</tr>
<tr>
<td>Maximum</td>
<td>3124.0</td>
<td>452.0</td>
<td>274.0</td>
<td>28.1</td>
</tr>
<tr>
<td>Minimum</td>
<td>82.2</td>
<td>10.3</td>
<td>61.0</td>
<td>4.5</td>
</tr>
</tbody>
</table>

* Measurements were made between April and December, 1980. All numbers are in cd/m². The mean of the 3 daytime measurements is 279 cd/m². The grand mean of daytime plus nighttime measurements is 147 cd/m². Much of the animal literature reports illumination levels in ft-c. The number of cd/m² multiplied by 0.36 is approximately equal to the equivalent number of ft-c (our luminance measurements were made using a standard reflector with reflectance = 0.81). All measurements were made outside of the isolettes; therefore, they do not reflect isolette attenuation. Attenuation of a typical isolette under a typical band of fluorescent lights was found to be <0.1 log units. An estimate of the retinal irradiance (for wavelengths between 300 nm and 700 nm) on a sunny day (no direct light), assuming a luminance of 91.4 cd/m², is 0.33 μW/cm² (see Appendix).

† One preterm infant (Casey) was not tested on the acuity screening task.
Table 2. Infants' medical history

<table>
<thead>
<tr>
<th>Infant</th>
<th>Sex</th>
<th>Birth weight (grams)</th>
<th>Estimated gestational age (weeks)</th>
<th>Apgars (1 min, 5 min)</th>
<th>Phototherapy (days)</th>
<th>Duration of stay in hospital (days)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Casey</td>
<td>M</td>
<td>1600 (AGA)*</td>
<td>32.5</td>
<td>8, 9</td>
<td>3</td>
<td>32</td>
</tr>
<tr>
<td>Eric</td>
<td>M</td>
<td>1350 (AGA)</td>
<td>29.5</td>
<td>9, 9</td>
<td>3</td>
<td>49</td>
</tr>
<tr>
<td>Ryan</td>
<td>M</td>
<td>1250 (BSGA)†</td>
<td>31.5</td>
<td>5, 7</td>
<td>4</td>
<td>17</td>
</tr>
<tr>
<td>Pamela</td>
<td>F</td>
<td>1510 (AGA)</td>
<td>31.0</td>
<td></td>
<td>3</td>
<td>49</td>
</tr>
<tr>
<td>Amanda</td>
<td>F</td>
<td>1900 (AGA)</td>
<td>33.5</td>
<td>7, 9</td>
<td>2</td>
<td>24</td>
</tr>
<tr>
<td>Auburn</td>
<td>F</td>
<td>1930 (AGA)</td>
<td>32.0</td>
<td>9, 9</td>
<td>4</td>
<td>24</td>
</tr>
<tr>
<td>Taylor</td>
<td>M</td>
<td>1880 (AGA)</td>
<td>34.0</td>
<td>8, 9</td>
<td>8</td>
<td>22</td>
</tr>
<tr>
<td>Lisa</td>
<td>F</td>
<td>1780 (AGA)</td>
<td>33.0</td>
<td>7, 9</td>
<td>0</td>
<td>22</td>
</tr>
<tr>
<td>Lori</td>
<td>F</td>
<td>1340 (BSGA)</td>
<td>33.0</td>
<td>7, 8</td>
<td>0</td>
<td>27</td>
</tr>
</tbody>
</table>

Light-exposed infants

<table>
<thead>
<tr>
<th>Infant</th>
<th>Birth weight (grams)</th>
<th>Estimated gestational age (weeks)</th>
<th>Apgars (1 min, 5 min)</th>
<th>Phototherapy (days)</th>
<th>Duration of stay in hospital (days)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rebecca</td>
<td>F</td>
<td>4536</td>
<td>40.0</td>
<td>8, 9</td>
<td>0</td>
</tr>
<tr>
<td>Laura</td>
<td>F</td>
<td>3203</td>
<td>40.0</td>
<td>9, 10</td>
<td>0</td>
</tr>
<tr>
<td>Tami</td>
<td>F</td>
<td>3685</td>
<td>40.0</td>
<td>9, 9</td>
<td>0</td>
</tr>
<tr>
<td>Tristan</td>
<td>M</td>
<td>3133</td>
<td>40.0</td>
<td>7, 9</td>
<td>0</td>
</tr>
<tr>
<td>Christopher</td>
<td>M</td>
<td>3615</td>
<td>40.0</td>
<td>9, 8</td>
<td>0</td>
</tr>
<tr>
<td>Abraham</td>
<td>M</td>
<td>3643</td>
<td>40.0</td>
<td>8, 9</td>
<td>0</td>
</tr>
<tr>
<td>Beth Ann</td>
<td>F</td>
<td>3657</td>
<td>40.0</td>
<td>9, 9</td>
<td>0</td>
</tr>
<tr>
<td>Lilli</td>
<td>F</td>
<td>3487</td>
<td>40.0</td>
<td>9, 9</td>
<td>0</td>
</tr>
<tr>
<td>J.H.</td>
<td>M</td>
<td>3799</td>
<td>40.0</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Control infants

* AGA = appropriate for gestational age.
† BSGA = borderline small for gestational age.

2). As a direct result of animal studies, infants' eyes now are patched routinely during phototherapy treatment. Therefore, a conservative estimate of the actual duration of exposure to continuous illumination for each infant was obtained by subtracting the number of days of phototherapy from the number of days in the hospital. The resulting exposure durations ranged from 13 to 46 days (mean = 27 days).

The light-exposed infants were all born at least 6 weeks prior to term, as assessed by Dubowitz exam. Nine infants who stayed in the hospital 5 days or less following delivery served as controls. These infants were all born at term (within ±10 days from due date, by parents' report).

In order to minimize the likelihood that the vision of the light-exposed infants would differ from that of the control infants for reasons other than the suspected variable (continuous illumination), the light-exposed infants had to meet the following two criteria:

1. They had received little (<6 hr) or no supplemental oxygen during the perinatal period. This minimized the risk of testing infants with retinopathy of prematurity (retrolental fibroplasia, RLF), and indeed, ophthalmologic examination when the infants were discharged from the hospital showed no evidence of RLF in any of the infants. In addition, the exam revealed no gross changes in the posterior pole.

2. They had no history of visual or other neurologic dysfunction.

All control infants had been born without medical complications. Details of each infant's medical history are shown in Table 2.

Light-exposed infants who met the criteria for the study were recruited by direct contact with the parents. Control infants were recruited from birth announcements in a local newspaper.

All infants began testing at 43 weeks gestational age (±7 days). For the light-exposed infants, the average delay between discharge from the hospital and the first day of testing was 47 days (range = 37 to 61 days). On average, control infants began testing on their 18th postnatal day (range = 17 to 20 days). Testing took 1 to 7 days, 1 hr per day. Acuity screening required one additional test session at approximately 44 weeks gestational age. Parents were paid $5 per session, plus parking or bus fare. All parents were informed of the purposes of the study, as well as the methods used and any potential risks and/or benefits that might be derived by participating in the study. Voluntary, informed, written consent for their child to participate in the study was obtained from each parent before any testing began.

Results

Absolute Thresholds

Figure 2 shows sample psychometric functions from four infants in the present study. Figure 2A shows data
from two light-exposed infants, and Figure 2B shows data from two control infants. For both groups, data displayed are those from the most sensitive (hexagons) and least sensitive (triangles) infants. The smooth curves represent a best-fitting cumulative normal curve, estimated by probit analysis. 25 We defined threshold for each data set as the intensity that corresponded to the 75% correct point on the best-fitting cumulative normal curve.

It is apparent that the two distributions of thresholds for detection of a large 502 nm stimulus overlap substantially. This result is depicted more completely in Figure 3. The thresholds for all the light-exposed (E, circles) and control (C, triangles) infants are plotted on the left portion of the figure. The distributions overlap almost entirely except for one outlying point. A one-sided t-test verified that these distributions were statistically indistinguishable (t = 0.722, P > 0.2).

In addition, we have plotted the instantaneous slope of the probit curve at 75% correct for each infant.‡ Since this slope is related inversely to the standard deviation of the best-fitting cumulative normal distribution, we wanted to see if the two groups of infants differed on this dimension. They did not (two-sided t = 1.724, P > 0.10).

Acuity Screening

Of the eight light-exposed infants tested, seven passed the acuity screening when tested at 44 weeks gestational age. One infant (Lori) failed the acuity screening at 44 weeks. She failed again at 48 weeks when tested with a grating appropriate for that age (27 min of arc/stripe, 1.07 cy/deg), but was able to detect the gratings used to screen 44 week old infants (0.75 cy/deg). At 52 weeks, Lori passed the screening when tested with the grating appropriate for her age (20 min/stripe, 1.5 cy/deg).

Seven of nine control infants passed the acuity screening at 44 weeks (4 weeks postnatal). The two who failed (Laura and Lilli) passed when they were retested at 48 weeks with the appropriate grating for that age.

Discussion

In the present study, a behavioral estimate of absolute threshold in very young infants (43 weeks gestational age) revealed no differences between light-exposed, preterm infants and control, full-term infants. This suggests that exposure to continuous illumination caused no extensive deficit in rod function. In addition, the results of the acuity screening suggest that the photopic system of the preterm infants was functioning within normal limits, although more extensive testing would be required to evaluate this finding fully.

Our finding no measurable damage in light-exposed

† A slope is not reported for one infant (Lisa) since the probit analysis program did not converge on a best-fitting cumulative normal curve for this data set. Threshold for this data set was defined as the intensity corresponding to the 75% point of a weighted least-squares fit to the data.

‡ Threshold is defined as the intensity (in log units) corresponding to the median of the normal curve (75% correct), and is indicated in each case by the dashed line and vertical arrow.
dition, the exposure durations were much longer than those tested on 4-year-old children who had been exposed to light to reveal certain kinds of damage. For example, neither of the blue cones, nor highly localized damage would be revealed by either the scotopic thresholds or the acuity screening.

In view of the overwhelming evidence that continuous illumination can damage the visual system in many organisms, including neonatal primates, and that even intermittent light at levels below the threshold for thermal damage can cause damage in primates, it seems unlikely that humans would be uniquely immune to such damage. Therefore, we should be cautious in generalizing from these results. The need for such caution is emphasized by three general considerations.

First, the present technique would not be expected to reveal certain kinds of damage. For example, neither damage to the short wavelength-sensitive mechanism (the blue cones), nor highly localized damage would be revealed by either the scotopic thresholds or the acuity screening.

Second, it might indeed be the case that the average, relatively healthy premature infant is not at visual risk in this particular nursery setting. Perhaps the average light levels and exposure durations experienced by infants in this nursery are below some critical threshold necessary to cause long-term functional deficits. (The retinal irradiances to which these infants are exposed are probably relatively low, probably \(\leq 0.4 \mu W/cm^2\) averaged over space and time during the middle of a sunny day; see Appendix.) Or perhaps infants’ normal repair mechanisms were able to reverse any damage before we tested them. Even if this were true, however, it would be desirable to have an estimate of the magnitude of the safety margin that we are dealing with. How much higher can the light levels be, and how much longer can the exposure durations be before significant risk is incurred? Unfortunately, as indicated in the introduction, the animal literature does not provide us with enough information to evaluate fully the relative safety or risk of the lighting regimes to which human infants are routinely exposed.

A third, related consideration is that some populations of infants may be more at risk for sustaining light-induced damage than is the typical infant in the nursery. For example, studies of light-exposed rats suggest that infants with hypopigmentation or albinism, with sustained elevation of body temperature, or with larger than normal pupils would be at greater risk. In addition, infants kept in illumination “hot spots” (Table 1) in the nursery or who remained in the hospital for an unusually long period of time may be at greater than average risk.

In summary, we used the forced-choice, preferential- looking technique to assess rod and cone function in nine infants who had been exposed to 13 to 46 days of continuous illumination in an NICU. Rod function was assessed by comparing these infants’ absolute thresholds for 502 nm light with the thresholds of a sample of control infants, born at term, who had been exposed only minimally (\(\leq 5\) days) to similar illumination conditions. A rapid screening of acuity in each infant provided a rough behavioral assessment of cone function. We found no difference between the light- exposed and nonlight-exposed infants on either of these measures. These results are reassuring; for, even if there were some damage in the light-exposed infants’ visual systems, it is not sufficient to disrupt the mechanisms—from photon-absorptions all the way to behavioral output—that are necessary for the light-exposed preterm infants to perform at the same level as the minimally exposed, full-term infants in the FPL task.

However, several considerations warrant caution in generalizing from these results. For example, ambient illumination levels vary somewhat from nursery to nursery (eg, Gottfried et al), and clinically useful estimates of safe combinations of light level and exposure duration are not available yet from the animal literature. Furthermore, even if we had access to such in-
formation and could be reasonably sure that the typical infant in the NICU was not at visual risk, special precautions might still be required for populations of infants who might be expected to be more susceptible than the average infant to light-induced retinal damage.

Appendix

We estimated retinal irradiance in the eye of a typical, healthy 34-week-gestation premature infant during the middle of a sunny day in the NICU (no direct light entering the unit) as follows:

Retinal irradiance \( R_{s1-s2} \) due to an extended stimulus, measured over a wavelength band \( \lambda_1 \) to \( \lambda_2 \), depends on four quantities: \( P \), area of the pupil (cm\(^2\)); \( A_s \), area of the stimulus on the retina (cm\(^2\)); \( T_s \), ocular transmission as a function of wavelength; and \( C \), corneal irradiance (\( \mu \)W/cm\(^2\)) as a function of wavelength. These are related to \( R \) by the expression

\[
R_{s1-s2} = \frac{P}{A_s} \int_{\lambda_1}^{\lambda_2} T_s C d\lambda
\]

Due to a paucity of data on the physiologic optics and retinal anatomy of young infants’ eyes (and especially premature infants’ eyes), \( P \), \( A_s \), and \( T_s \) can only be estimated grossly. In the following discussion we highlight with italics the important assumptions we made to carry out these estimates.

\( C \): Corneal irradiance as a function of \( \lambda \) was measured at the level of a typical premature infant’s eyes in the NICU. Flux measurements were made for 10 nm bands from 300 nm to 700 nm using a small cosine detector pointing at a white wall (distance ~10 feet) in the nursery, and a Gamma Scientific radiometer (Model #DR-1A). The result represents a (presumably) typical admixture of the spectrum of fluorescent lighting with the spectrum of indirect sunlight.

\( P \): Premature infants’ pupils are small. We assumed a pupil diameter of 1.5 mm (Drs. C. Hoyt, and R. Kalina, personal communication).

\( A_s \): Recently, Robb (1982) made postmortem measurements of retinal surface area in infants and children from 26 weeks gestation to 6 years postnatal (Robb made no correction for shrinkage due to fixation). From his Table and Figure 3, it appears that retinal surface area increases linearly with gestational age up through 43 weeks. A linear regression analysis of the first 14 surface area measurements yields the following equation, Surface Area (mm\(^2\)) = 18.45 \times Gestational Age (weeks) − 137.6, correlation coefficient = 0.915. From this we estimate that retinal surface area at 34 weeks gestation (34 weeks is the mean midpoint of the hospital stay) is 4.9 mm\(^2\).

In using this number in equation 1, we have assumed that, averaged over time, the entire retinal area is uniformly irradiated. Of course, the spatial and temporal distribution of flux onto the retina will be quite variable depending on the time of day, on the changes in the distribution of light sources in the visual field, and on head and eye movements. Since it is not possible to specify these spatial and temporal inhomogeneities, we assumed homogeneity over the entire retina. This assumption probably underestimates local (spatial and temporal) fluxes.

\( T_s \): The ocular transmission through premature infants’ ocular media is not known. In addition, the hyaloid vasculature may contribute to absorption in premature eyes. Since this vasculature generally atrophies by 32–34 weeks gestation, we assumed clear media with no absorption by the hyaloid vasculature.

There is evidence that young infants (born at term and tested postnatally) have a much flatter t-function than adults.\(^{26}\) In the extreme, \( T_s = 1.0 \) for all \( \lambda \). To be conservative, we estimated \( R \) for two extremes, one assuming \( T_s = 1.0 \) for all \( \lambda \), and one assuming adult values of \( T_s \) at each value of \( \lambda \). The true \( T_s \)-function is likely to fall somewhere between these two extremes.

For the adult values of \( T_s \) between 380 nm and 700 nm, we used the data of Norren and Vos (1974).\(^{31}\) Table 2. Each density in their Table 2 was corrected to account for small pupils by multiplying by 1.16 (p. 1242, Norren and Vos). Very few data are available on adult ocular transmission at wavelengths less than 380 nm. For wavelengths between 300 nm and 380 nm, our estimate was based on a linear extrapolation of adult lens transmission data (Table 2.7 in Wyszecki and Stiles, 1967).\(^{32}\) These calculated densities were then shifted vertically on the density axis to match Norren and Vos’ data at 380 nm.

Integration of equation 1 between 300 nm and 700 nm (summing over 10 nm increments) yielded the following estimates of \( R \):

1. For \( T_s = 1.0 \):
   \[
   R = 0.33 \mu \text{W/cm}^2;
   \]

2. For \( T_s \): adult transmission:
   \[
   R = 0.27 \mu \text{W/cm}^2;
   \]

Since the retina seems to be most susceptible to damage from middle and short wavelength light (<550 nm),\(^{9,13-14,26,33}\) we have also calculated retinal irradiance for \( X \) as between 300 nm and 550 nm. For conditions 1 and 2 above, respectively, \( R = 0.12 \) and \( 0.08 \mu \text{W/cm}^2 \).

The above are estimates of retinal irradiance homogeneously distributed over the entire retina. Uncontrolled or unknown factors that would attenuate light reaching the infants’ retinas (such as the eyelids, random shading in the nursery, etc) have not been included in the analysis. In addition, we have neglected losses of radiant flux due to reflections at the various refracting surfaces anterior to the retina, as well as sources of radiant flux originating in the external stimulus, but produced by fluorescence of the eye lens and retina.

Although these irradiances are small, relative to those that have been found to cause damage in experimental studies of light-damage (eg, ~200 to 600 \( \mu \text{W/cm}^2 \)), the cumulative exposure durations for the typical premature infant are much longer than those that have been used in the experimental studies. Furthermore, the typical premature infant stays in the hospital longer than any of the premature infants tested in the present study. Finally, note that the peak values of irradiance may exceed these estimates by 1 to 2 orders of magnitude (Table 1).

Key words: continuous illumination, absolute thresholds, human infants, acuity screening, preterm infants

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