Light Damage in the Developing Retina of the Albino Rat: An Electroretinographic Study

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The albino rat retina is severely damaged by exposure to bright light. The degree of damage depends upon the intensity of the exposure and its duration. In the present study, electroretinographic (ERG) responses were measured in rats exposed at different ages during the period of retinal development to 24 hr of bright light and then transferred to darkness for about 2 months. The ERG data indicated that if the bright-light exposure was done prior to 20 days of age, the retina was resistant to the light damage, and the dark-adapted ERG responses measured later were normal. In rats older than 20 days, light damage increased with age until, in rats exposed to light at age 30 days, the ERG was unrecordable. Further experiments showed that light exposure did damage the 15-day-old rat retina; however, complete recovery was attained within 15 days postexposure. Invest Ophthalmol Vis Sci 27:164-167, 1986

The deleterious effects of light exposure on the retina of the albino rat have been extensively studied ever since they were first reported.1,2 Two different types of damage have been identified.3,4 Type I damage is caused by a relatively brief (up to 48-hr) exposure to bright light and is characterized by loss of pigment epithelium and photoreceptors.3,4,5 Anatomical studies have shown that the severity of type I retinal damage depends on the age at which the rats are exposed to the bright light. Until 3–4 wk of age, the albino rat retina is unaffected at the light-microscopic level. Thereafter, in rats up to 24 wk of age, retinal damage becomes progressively more severe as the rats age.6 Electron-microscopic techniques support the conclusion of reduced susceptibility to light of the developing retina.6 A 3-day exposure to bright light (500 foot-candles) of 2-wk-old rats causes damage to the photoreceptors, mainly to the outer segments, followed by limited recovery. Similar exposure of 3-wk-old rats causes severe degeneration in whole photoreceptor cells, with no subsequent recovery of outer-segment material.6

Type II light damage is produced by a long-term exposure to low-intensity illumination.3,4,7 This type of damage is characterized by diffuse loss of photoreceptors and preservation of the pigment epithelial cells.3,4,7 Hormonal studies of type II light damage suggest that sexual maturation increases the susceptibility of the rat retina to long-term dim illumination.8

In this study, rats were exposed for 24 hr to bright light at different postnatal ages. ERG responses measured at 2 months of age indicated minimal retinal damage in rats that were younger than 20 days at time of exposure. Thereafter, the severity of the damage increased as the rat's age at time of exposure increased until, in rats subjected to the light exposure at age 30 days, the ERG responses were unrecordable, even with the brightest test flash. ERG data obtained at short time intervals after termination of the light exposure of 15-day-old rats indicated that the light damaged the retina, but complete recovery of retinal function was attained within 15 days postexposure.

Materials and Methods

Animals

Albino rats were raised in constant darkness from birth. At a certain postnatal day, the offspring were separated from the mother and transferred to a ventilated light-exposure chamber. During the light exposure, the rats were housed in clear plastic cages, each containing one or two rats, placed underneath a fluorescent-light source. All experimental rats were subjected to the same light exposure of 24-hr duration and 260-foot-candle intensity. Following the light-exposure...
period, the rats were returned to their mother and kept in complete darkness until electroretinographic (ERG) measurements. Normal ERG data were measured in 10 rats kept in complete darkness from birth.

**Electroretinogram**

The dark-adapted rat was anesthetized by intraperitoneal injection of sodium pentobarbital (40 mg/kg body weight). The eyes were fully dilated by cyclopentolate hydrochloride (0.5%). The ERG was recorded differentially with a cotton-wick electrode filled with saline solution leading to a chlorided silver wire placed on the cornea. The reference and ground electrodes were attached to the ears.

Light stimulation consisted of an electronic camera flash attenuated by calibrated "neutral" density filters (Schott). The test flash formed a uniform image 3 cm in diameter, covering a section of a ping-pong ball placed on the rat's eye. All preparations were done under dim red-light illumination while keeping the animal's body temperature constant with a heating pad.

All of the procedures used in this study conform to the ARVO Resolution on the Use of Animals in Research.

**Results**

Figure 1 shows representative dark-adapted ERG responses obtained from a control rat and from rats exposed at different ages to 24 hr of bright (260-foot-candle) illumination. The ERG responses were recorded when the rats reached the age of about 2 months. It can be clearly seen in Figure 1 that the ERG responses obtained from rats subjected to the standard light exposure at ages 5, 10, and 15 days were similar to the control ones, while rats exposed to light at 20 and 25 days of age showed subnormal responses. The b- and a-wave amplitudes were averaged for each experimental group and plotted as a function of the test flash intensity, as shown in Figures 2A and 2B, respectively. The two continuous lines describe the normal range (mean ± standard deviation) of a- and b-waves measured in 10 rats raised in darkness from birth. The data points represent mean values obtained from rats subjected to 24-hr light exposure at age 5 days (solid diamonds, n = 8), 10 days (solid triangles, n = 5), 15 days (open squares, n = 8), 20 days (open triangles, n = 6), and 25 days (solid squares, n = 4).

In order to quantify the degree of retinal damage, a- and b-wave ratios were defined. For each test-flash intensity, the mean wave amplitude of the experimental group was divided by the corresponding value of the control group. These ratios, calculated for all test-flash intensities, were averaged for each experimental group to give a single number which described the effect of the light exposure on the ERG waves. In Figure 3, the a-wave (open circles) and b-wave (solid circles) ratios are plotted as a function of the age at which the rats were exposed to the 24 hr of bright light. The a- and b-waves were similarly affected by the light exposure. In rats exposed before reaching the age of 20 days, both ERG waves were normal. Light exposure at older ages reduced both a- and b-waves; the older the rats at time of light exposure, the more severe was the ERG deficit.

The results presented indicate that the developing rat retina is protected from the damaging effects of
Exposure, the b-wave threshold was elevated by as much as 4 log units above the normal control, while the b-wave ratio was reduced by about 90%. This functional deficit cannot be explained by incomplete rhodopsin regeneration. Rhodopsin regeneration after a bleaching exposure in adult and young albino rats is complete within 3 hr, and ERG threshold at that time is significantly elevated above the dark-adapted level.11,12 It is concluded, therefore, that the light exposure produced functional damage to the young retina. As the rats stayed for longer periods in the dark, both the b-wave threshold and the b-wave ratio gradually improved, until at 15 days post-light exposure normal levels were reached for these parameters.

Discussion

The data presented in this report show that the effects of short-term bright-light exposure on the functional integrity of the albino rat retina strongly depend on
the age of the animal at the time of light exposure. No functional damage could be measured in adult rats that were subjected to the light exposure before reaching the age of 20 days. However, if light exposure was done after age 20 days, the adult rat showed an ERG deficit that became more severe as the age of exposure was raised. In rats exposed to light at age 30 days or later, the ERG was unrecordable, indicating severe retinal damage. These electrophysiological data support previous anatomical and ERG findings that point to the resistance of the developing rat retina to light damage.

The outer segments of the rod photoreceptors in the rat retina are continuously renewed. In the normal adult retina, the rod outer segments are maintained at a constant length because the rates of disc synthesis and disc removal are equal. In the developing rat retina, the outer segments are continuously elongated, indicating a greater rate of disc synthesis than of disposal. The data presented here show that the dependence of the degree of light damage on the age at light exposure parallels the time course of outer-segment elongation. It is suggested, therefore, that the light exposure utilized in this study affected primarily the outer segments. In young rats, when the process of outer-segment growth is most prominent, recovery from this light exposure may be complete. However, in mature animals the ability of the outer segments to recover is lost, and therefore light exposure results in irreversible cell death. This ability of the developing rat retina to recover from light exposure through the process of outer-segment renewal is supported by the data shown in Figure 4. In rats exposed to light at age 15 days, severe functional retinal damage was demonstrated after the exposure. However, retinal function recovered completely within 15 days post-light exposure, a time period which parallels the rate of outer-segment renewal. Anatomical measurements show that, following bright-light exposure, the rate of elongation of outer segments in the developing rat retina is increased compared to the normal level and that normal outer-segment length is attained within 12 days.

It is concluded, therefore, that in the developing rat retina, the process of retinal maturation provides a means by which the net addition of outer-segment material can be substantially increased following insults such as intense light exposure, thus allowing recovery from the damage. The question remaining is why in the adult rat retina such recovery is not observed, even though the outer-segment-renewal process is speculated to provide the means for recovery.

Key words: retina, electroretinogram, light damage, development, a-wave, b-wave

References