Failure of Vitamin E to Protect the Retina against Damage Resulting from Bright Cyclic Light Exposure

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Cumulative light-mediated damage to the retina over a long time period may be involved in the development of age-related retinopathies. Light is thought to produce retinal damage by initiating antioxidative reactions among the molecular components of the retina. Experiments were therefore conducted (1) to confirm that long-term differences in cyclic light intensity affect the rate of age-related photoreceptor cell loss from the retina; and (2) to determine whether the antioxidant, vitamin E, is an effective inhibitor of damage to the retina by bright cyclic light. Albino rats were fed a basal diet either supplemented with or deficient in vitamin E. Each dietary group was divided into two light-treatment groups which were exposed to 12 hr cyclic light of either 15 lux or 750 lux. After 10 and 17 weeks of treatment, retinal photoreceptor cell densities were determined for animals in each group. Vitamin E deficiency resulted in moderate decreases in photoreceptor cell densities in the dim-light groups after both 10 and 17 weeks. Rats exposed to the bright-light condition suffered a pronounced loss of photoreceptor cells by 10 weeks, and an even greater cell loss by 17 weeks. Vitamin E deficiency did not enhance the effect of bright cyclic light in reducing photoreceptor cell densities. Thus, it appears unlikely that retinal damage by cyclic light occurs via an antioxidative mechanism.


The development of age-related retinal pathologies has been proposed to result at least in part from the cumulative effects of damage to the retina by visible radiation.1,2 A variety of evidence has been cited to support this hypothesis. For example, it has been reported that light iris pigmentation is associated with a greater risk for developing senile macular degeneration.3,4 In addition, wearing corrective lenses that reduce transmission of short-wavelength light to the retina beginning at an early age appears to lower the probability that an individual will suffer from age-related retinal degeneration.5 On the other hand, apha-kia, which results in increased transmission of blue and ultraviolet light to the retina, appears to potentiate the development of macular abnormalities.6 Visible light is thought to produce retinal damage by initiating antioxidative reactions that are deleterious to molecular and cellular components of the retina, particularly the photoreceptor cells.7-10 The susceptibility of the retina to antioxidative damage has been demonstrated clearly in numerous studies in which the detrimental effects of impaired antioxidant protection on retinal structure and function have been elucidated.11-14 Whether light exposure of the retina is involved in the effects of antioxidant deficiency has not been previously determined. If light damages the retina by promoting or initiating antioxidative reactions, impaired antioxidant protection of the retina would be expected to increase the susceptibility of the retina to light damage. To test this prediction, the effect of vitamin E deficiency on the susceptibility of the retina to damage from bright cyclic light was studied.

Materials and Methods

Male Fisher 344 rats were purchased at 21 days of age from Harlan Sprague-Dawley, Inc. (Indianapolis, IN). At the supplier, the animals were born and housed under 12 hr:12 hr cyclic illumination from cool-white fluorescent tubes. Illumination at the cage levels was between 1 and 5 lux. Upon arrival, the animals were randomly divided into four treatment groups designated +E/B, +E/D, -E/B, and -E/D. All four groups of rats were housed in the same room under 12 hr:12 hr cyclic illumination from Sylvania F40/CW fluorescent tubes (Danvers, MA). Light levels were determined with a Lutron model LX-101 light meter (Markson Science, Phoenix, AZ) with the measuring probe placed face-up on the bottoms of the animal cages. During the light phase of the light-
ing cycle, average illuminance measured at the cage bottoms was 15 lux for the +E/D and -E/D groups and 750 lux for the +E/B and -E/B groups. Differences in light intensity were achieved by placing the cages in different locations relative to the light sources. Total darkness was maintained during each 12 hr dark cycle. The two -E groups were fed a vitamin E-deficient basal diet that was described previously. The rats in the +E groups were fed the same basal diet supplemented with 250 mg of dl-α-tocopheryl acetate per kilogram diet.

After 17 weeks of treatment, five to seven animals in each group were anesthetized with diethyl ether, and 1 to 1.5 ml blood samples were obtained from the tail veins. Blood coagulation was prevented by collecting the samples into tubes each containing approximately 1 mg of disodium ethylenediaminetetraacetic acid. The plasma fraction was separated from each blood sample and used for vitamin E determinations. Plasma vitamin E concentrations were determined with high performance liquid chromatography, as described previously, except that detection was achieved with a Hewlett Packard model 1046A fluorescence detector (Palo Alto, CA). The limit of detection for α-tocopherol with this technique was 8.5 pmol per analysis. The largest volume of plasma analyzed for an individual animal was 200 μl. Thus, as little as 43 pmol of α-tocopherol per milliliter plasma could be measured with the analytical procedure employed.

The α-tocopherol contents of the neural retinas were also determined in five rats from each of the treatment groups at 17 weeks. Animals were euthanized with CO₂ and their eyes were enucleated immediately. The neural retina was dissected from one eye of each animal and retinal α-tocopherol contents were measured essentially as described by Katz and colleagues, except that detection was performed with a Hewlett Packard model 1046A fluorescence detector. A maximum of 85% of each sample was extracted for α-tocopherol determinations. Thus, the limit of detection for α-tocopherol was 10 pmol per retina.

After both 10 and 17 weeks of treatment, animals from each experimental group were euthanized with CO₂, and their eyes were immediately enucleated. One eye from each animal was placed in a cacodylate-buffered mixed aldehyde fixative at room temperature and the corneas, irides, and lenses were removed. Fixation was continued for 1 hr at room temperature with constant gentle mixing, and the tissues were then stored in fixative at 4°C for a minimum of 24 hr. Following fixation, the eyes were bisected in the superior-inferior direction about 1 mm temporal to the optic nerve head. The nasal portion of each eye was embedded in glycol methacrylate. Sections along the superior-inferior meridian passing through the optic nerve were cut at a thickness of 2 μm with a Sorvall JB-4 microtome (Bio-Rad, Richmond, CA) and stained with Toluidine Blue.

Using a Zeiss Photomicroscope I equipped with a calibrated ocular micrometer, the cross-sectional lengths of retinal sections from each eye were measured from the edges of the optic nerve head to the superior and inferior ora serrata. Six regions of each retinal section were photographed with Kodak KM 135 slide film at a magnification of ×67. The regions photographed were centered on points one-quarter, one-half and three-quarters of the distances from the superior and inferior ora serrata to the edges of the optic nerve heads. The micrographs were projected onto a white surface, and the numbers of photoreceptor nuclei in 300 μm lengths of each retinal region were determined. Photoreceptor cell densities for each animal were estimated from the average of nuclei counts on two sections from the same eye. Eyes from five to seven rats per treatment group were used for photoreceptor density determinations.

The experiments described above conform to the ARVO Resolution on the Use of Animals in Research.

Assessments of the significance of treatment effects on the various measured parameters were performed using analysis of variance for an experiment with a 2 × 2 × 2 factorial design. Comparisons between individual treatment groups were performed using analysis of variance as described by Winer.

**Results**

After 17 weeks of treatment, neither the -E/D nor the -E/B animals had detectable levels of vitamin E in either plasma or retina (Table 1). The rats fed the diet supplemented with vitamin E had mean plasma α-tocopherol levels of approximately 33 nmol per ml. The light environment had no influence on plasma vitamin E concentrations (Table 1). Among animals fed the vitamin E-supplemented diet, those exposed to the bright light condition had a mean retinal α-tocopherol content that was 26% lower than that of rats maintained under dim cyclic light (Table 1). This difference in retinal vitamin E content between the +E/D and +E/B groups was statistically significant (P = 0.003).

Vitamin E deficiency resulted in moderate decreases in photoreceptor cell densities in some regions of the retinas from rats in the dim-light groups after 10 weeks of treatment (Figs. 1, 2, Tables 2, 3). At this time point, the photoreceptor cell density in the equatorial region of the superior retina was an average of
14% lower in the -E/D group than in the +E/D animals (P < 0.01) (Table 2). Photoreceptor cell densities in the other examined regions of the superior retina of the dim-light group were not affected by vitamin E deficiency after 10 weeks (Table 2). In the equatorial region of the inferior retina, the mean photoreceptor cell density was 6.5% lower in the -E/D group than in the +E/D rats after 10 weeks (P < 0.05) (Table 3). Vitamin E deficiency resulted in a smaller loss of photoreceptor cells from the posterior retina of the dim-light reared rats after 10 weeks, while no vitamin E-related cell loss was observed in the peripheral retinas of the -E/D rats (Table 3). The vitamin E deficiency-related decreases in cell densities in the dim-light group were not the result of differences in the rates of eye growth, and thus in the degree of retinal stretching, between the treatment groups. The cross-sectional lengths of the retinas were the same for all treatment groups at 10 weeks. The mean length of the superior retina along the measured meridian was 4870 ± 28 μm at 10 weeks, and the mean retinal length from the optic nerve head to the inferior ora serrata was 4873 ± 26 μm.

Between 10 and 17 weeks, photoreceptor cell density in the +E/D group decreased between 2 and 11% in the superior retina, and between 10 and 22% in the inferior retina, depending on the region examined (Tables 2, 3). In the -E/D group, photoreceptor density decreased between 8 and 19% in the superior retina and between 16 and 21% in the inferior retina from 10 to 17 weeks. All of these age-related decreases in photoreceptor density were statistically significant at a confidence level of greater than 0.95, except that in the superior peripheral retinas of the +E/D rats. The decreases in cell density that occurred between 10 and 17 weeks could be attributed partially to retinal stretching resulting from eye growth between these two time points. At 17 weeks, the length of the superior retina along the superior-inferior meridian had increased to an average of 4975 ± 23 μm for all treatment groups, and the length of the inferior retina had increased to a mean of 5085 ± 29 μm. As at 10 weeks, there were no significant differences in retinal cross-sectional length among the treatment groups at 17 weeks. The age-related decrease in photoreceptor cell density between 10 and 17 weeks was greater in the -E/D than in the +E/D animals for most regions of the retina. Thus, after 17 weeks of treatment, vitamin E deficiency had resulted in significant reductions in photoreceptor cell densities in all regions of the retinas of the dim-light groups that were examined, with the exception of the inferior-equatorial region (Tables 2, 3).

An increase in cyclic light intensity from 15 to 750 lux resulted in substantial decreases in photoreceptor cell densities in animals fed both the vitamin E-supplemented and -deficient diets (Figs. 1, 2, Tables 2, 3). When the bright-light groups were compared to the +E/D animals, vitamin E status was found to have no significant influence on the degree of light-induced photoreceptor cell loss; there were no significant differences in photoreceptor cell densities between the +E/B and -E/B groups at either 10 or 17 weeks (Tables 2, 3).

The inferior retina was more severely affected by bright cyclic light than was the superior retina. After 10 weeks of treatment, the photoreceptor cell densities in the inferior retinas of the +E/B group were reduced by 69 to 79% relative to the photoreceptor cell densities in the corresponding retinal regions of the +E/D rats (P < 0.005 for all regions). Likewise, photoreceptor cell densities in the inferior retinas of the -E/B group were reduced by 61 to 75% relative to those in the corresponding regions of the +E/D rats after 10 weeks (P < 0.005 for all regions). In the superior retinas, on the other hand, light-related decreases in photoreceptor density after 10 weeks were between 40 and 67% when the +E/B and +E/D groups were compared, and between 38 and 54% when the -E/B and +E/D groups were compared (P < 0.005 for all regions).

The light effect on the retina was more pronounced after 17 weeks than after 10 weeks of treatment. After 17 weeks, superior retinal photoreceptor cell densities were 62 to 82% lower in the +E/B group than in the +E/D rats (P < 0.005 for all regions). In the -E/B rats, mean photoreceptor cell densities in the superior retina were reduced by 56 to 86% relative to those in corresponding regions of the retinas of the +E/D rats (P < 0.005 for all regions). As at 10 weeks, light-related photoreceptor cell loss was greater in the inferior than in the superior retinas after 17 weeks. The light-related reduction in mean photoreceptor cell density in the inferior retinas of the +E/B rats was between 87 and 95% relative to the cell densities in the retinas of the +E/D rats (P < 0.005 for all re-
Fig. 1. Light micrographs illustrating the effects of light and diet on photoreceptor cell densities in the equatorial region of the superior retina. After 17 weeks of treatment, vitamin E deficiency alone resulted in a small decrease in photoreceptor cell density in rats maintained under dim cyclic light (–E/D). Photoreceptor loss was much more severe in animals maintained under bright light, regardless of diet.

regions). The –E/B animals had photoreceptor densities that were 92 to 96% lower than those in corresponding regions of the inferior retinas of the +E/D animals after 17 weeks (P < 0.005 for all regions).

Of the photoreceptor cells remaining in the superior retinas of the bright-light groups after 17 weeks of treatment, few had normal-appearing inner or outer segments (Fig. 1). The distance between the outer limiting membrane and the RPE was greatly reduced in both groups maintained under the higher light intensity, and the cell components remaining in this region often appeared greatly swollen and lacked the substructure indicative of the presence of discs in the outer segments. Vitamin E did not appear to be effective in preventing these light-induced morphological changes in the inner and outer segments. In the inferior regions of the retinas from the animals maintained under the bright light condition, the few remaining photoreceptor nuclei had no evidence of inner or outer segment remnants.

Because vitamin E deficiency led to decreases in photoreceptor cell densities in the dim-light groups, the magnitudes of the light-induced photoreceptor cell losses in some retinal regions were significantly lower in the –E animals than in the rats fed the +E diet. For example, after 10 weeks, the light-related loss of photoreceptors in the equatorial region of the superior retina was about 28% when the –E/D and –E/B groups were compared (P < 0.01). The difference in photoreceptor cell density between the +E/D and +E/B groups, however, was about 40% in the same retinal region (P < 0.01). After 17 weeks, the magnitude of the light-induced photoreceptor cell loss was also greater in the vitamin E-supplemented than in the vitamin E-deficient group. For example, the +E/B rats had an average of 62% fewer photore-
Fig. 2. Effects of light and diet on photoreceptor cell densities in the equatorial region of the inferior retina after 17 weeks of treatment. Vitamin E deficiency alone had no effect on photoreceptor cell densities in animals maintained under dim cyclic light (−E/D). Light-related photoreceptor cell loss in the +E/B and −E/B groups was more severe in the inferior than in the superior retina.

cceptor nuclei in the superior equatorial retina than did the +E/D rats. Among the animals fed the vitamin E-deficient diet, on the other hand, the mean light-induced photoreceptor cell loss from the superior equatorial retina amounted to only about 51%.

Table 2. Influence of diet and light on photoreceptor cell density in the superior retina

<table>
<thead>
<tr>
<th>Retinal location</th>
<th>Weeks of treatment</th>
<th>Number of photoreceptor cell nuclei per 300 μm</th>
<th>+E/D</th>
<th>+E/B</th>
<th>−E/D</th>
<th>−E/B</th>
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<tr>
<td>Posterior*</td>
<td>10</td>
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<td>496 ± 12§</td>
<td>163 ± 50</td>
<td>494 ± 9</td>
<td>230 ± 62</td>
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<tr>
<td>Equatorial†</td>
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<td>537 ± 8</td>
<td>324 ± 15</td>
<td>460 ± 8</td>
<td>331 ± 17</td>
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<tr>
<td>Peripheral‡</td>
<td>10</td>
<td></td>
<td>441 ± 16</td>
<td>234 ± 22</td>
<td>451 ± 15</td>
<td>273 ± 35</td>
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<tr>
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<td>459 ± 9</td>
<td>81 ± 28</td>
<td>409 ± 8</td>
<td>63 ± 22</td>
</tr>
<tr>
<td>Equatorial</td>
<td>17</td>
<td></td>
<td>476 ± 9</td>
<td>180 ± 30</td>
<td>423 ± 8</td>
<td>208 ± 36</td>
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<tr>
<td>Peripheral</td>
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<td></td>
<td>431 ± 18</td>
<td>127 ± 40</td>
<td>364 ± 9</td>
<td>104 ± 21</td>
</tr>
</tbody>
</table>

* Centered 75% of distance from superior ora serrata to optic nerve head.
† Centered 50% of distance from superior ora serrata to optic nerve head.
‡ Centered 25% of distance from superior ora serrata to optic nerve head.
§ Mean ± SE (n = 5–7).

Discussion

Age-related retinopathies, particularly those with macular involvement, are among the major causes of visual impairment in developed countries. Normal
aging is accompanied by a loss of retinal neurons, including photoreceptor cells, from the retinas of both humans18 and rodents.16,20,21 In most cases, this cell loss is probably partially responsible for a modest loss of acuity,22,23 and perhaps contributes somewhat to the age-related decrease in threshold light sensitivity.24 Severe vision loss, associated with age-related macular degeneration, probably represents an end-stage of this normal aging process. This interpretation is supported by the fact that aging is the predominant risk factor for developing degenerative macular disease.18,25 Thus, it is probable that an understanding of the etiology of age-related macular degeneration can be achieved by characterizing the factors that regulate normal age-related changes in the retina.

Because factors that increase the lifetime exposure of the retina to light appear to increase the risk for developing age-related retinopathies, it is likely that light plays a role in the etiology of these diseases. This possibility is supported by the current finding that photoreceptor cell loss is greatly accelerated by increasing the cyclic light intensity to which animals are exposed for a long period of time. Acceleration of the age-related loss of retinal photoreceptor cells by cyclic light has also been reported by other investigators.20,21,26

In the present study, the animals were transferred from a cyclic light environment of 1-5 lux to brighter light environments at 21 days of age. This sudden increase in environmental light intensity could have had an acute effect that contributed somewhat to the observed loss of photoreceptor cells in the bright light-reared rats, at least at the 10-week time point. The progression of photoreceptor cell loss between 10 and 17 weeks indicates, however, that chronic exposure to bright cyclic light played the major role in the observed cell losses. Additional experiments will be required to determine whether an acute effect contributed to the observed loss of photoreceptor cells in the animals exposed to bright cyclic light.

The mechanism by which light induces photoreceptor cell death is currently unknown. It has been proposed that the retina may be damaged by photosensitized autoxidation of retinal components.7-10,27,28 If this hypothesis is correct, one would predict that impairment of the antioxidant protection of the retina would increase the susceptibility of this tissue to the damaging effects of light. Previous experiments have produced results inconsistent with this prediction.29-31 For example, Stone and colleagues29 reported that dietary vitamin E and selenium deficiencies failed to enhance the susceptibility of the retina to damage from acute light stress. Both vitamin E and selenium function to protect the retina from autoxidative damage,4,6 and retinal levels of both compounds are lowered by reducing their levels in the diet.15,29,32 Thus, it appears unlikely that acute damage to the retina by high intensity light occurs primarily via photosensitized autoxidation of retinal components.

It is possible, however, that the mechanisms involved in acute retinal light damage differ from the mechanisms by which cyclic light of moderate intensity promotes age-related photoreceptor cell loss. Indeed, acute retinal damage from constant light exposure is characterized by a more pronounced loss of photoreceptors from the superior retina than that which occurs inferior to the optic nerve head.33 The photoreceptor cell loss resulting from bright cyclic light exposure, on the other hand, is most severe in the inferior retina (Tables 2, 3). This topographical difference in cell loss suggests that the acute and chronic damaging effects of light may occur via different mechanisms. Therefore, experiments were conducted to evaluate the possibility that photosensitized autoxidation is involved in the loss of photoreceptor cells resulting from chronic exposure to bright cyclic light. Photoreceptor cell densities after long-term exposure to cyclic light of 750 lux intensity were found to be similar regardless of whether animals

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<td>495 ± 23</td>
</tr>
<tr>
<td>Posterior</td>
<td>17</td>
<td>480 ± 10</td>
</tr>
<tr>
<td>Equatorial</td>
<td>17</td>
<td>423 ± 9</td>
</tr>
<tr>
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‡ Centered 25% of distance from inferior ora serrata to optic nerve head.
§ Mean ± SE (n = 5-7).
were fed vitamin E-supplemented or -deficient diets. Because vitamin E deficiency resulted in a loss of photoreceptor cells in the animals maintained under dim cyclic light, the light-related differences in photoreceptor cell densities were actually somewhat less among the vitamin E-deficient rats than in the animals fed the vitamin E-supplemented diet. For example, after 10 weeks of treatment, the photoreceptor cell density in the superior-equatorial retina was 40% lower in the +E/B group than in the +E/D animals; however, the photoreceptor cell density in the same retinal region of the —E/B group was only 28% lower than in the —E/D rats. Since the effects of light exposure and vitamin E deficiency are less than additive, it appears that either bright light exposure inhibits the effects of vitamin E depletion, or that vitamin E deficiency may actually decrease the susceptibility of the retina to the damaging effects of bright cyclic light. The latter phenomenon might occur as a result of the fact that vitamin E deficiency lowers neural retina vitamin A concentrations; vitamin A deficiency has been shown to protect against acute light damage to the retina. In addition, the apparent reduction in sensitivity to the effect of bright cyclic light that occurs as a result of vitamin E deficiency may be partially due to the existence of a subpopulation of photoreceptor cells that are particularly sensitive to destruction as a result of either vitamin E deficiency alone or of the damaging effects of light alone. Elimination of this subpopulation of sensitive cells as a result of vitamin E deficiency would leave a retina with a higher than normal proportion of cells that are resistant to the damaging effects of light.

While there is no evidence for the existence of such a subset of photoreceptor cells, the fact that not all cells die at once in response to either bright cyclic exposure or vitamin E deficiency indicates that there is variability in the susceptibility of the photoreceptors to autoxidative damage and the damaging effects of light. Whether the same cells are particularly sensitive to the effects of light and vitamin E deficiency is currently unknown. Thus it is not possible to state with certainty why the decrease in photoreceptor cell density was more pronounced within the +E group than among the —E animals. However, the results clearly indicate that photoreceptors are not protected by vitamin E from the long-term effects of bright cyclic light.

Among animals fed the vitamin E-deficient diet, vitamin E depletion from the retina appeared to be due primarily to dietary deficiency, although light-induced photoreceptor cell loss may have played a minor role. This conclusion is based on the observation that retinal vitamin E was depleted in rats fed diets lacking this nutrient, even when the animals were maintained under dim light conditions. The dim light-reared rats fed the vitamin E-deficient diet had only moderately reduced photoreceptor cell densities relative to the dim light-reared animals that received vitamin E, and photoreceptor morphology was not appreciably altered in the —E/D rats. Thus, the depletion of vitamin E from the retina was not dependent on photoreceptor or outer segment loss. Light-induced photoreceptor cell death was, however, accompanied by a significant decrease in retina α-tocopherol content in the +E animals (Table 1). This is not surprising in view of the fact that photoreceptor cells have a high vitamin E content. Because a high proportion of total retinal α-tocopherol is localized in the rod outer segments, it appears that the light-induced decrease in retinal α-tocopherol content is less than would be expected from the observed loss of photoreceptor cells and outer segment layer destruction in the +E animals maintained under the cyclic bright light condition. Thus, exposure to light that caused severe retinal damage did not result in a specific reduction in retinal α-tocopherol content, as would be expected if damage by cyclic light involved extensive autoxidation. In fact, bright cyclic light exposure actually resulted in an increase in retinal α-tocopherol content relative to photoreceptor cell numbers in the +E rats. This observation is consistent with the finding of Penn and colleagues that the ratio of α-tocopherol to total lipid phosphorous in the retina increased as a result of retinal damage by cyclic light.

Dietary ascorbate supplementation has been shown to be effective in inhibiting damage to the retina from acute light exposures and it has been suggested that this protective effect of vitamin C is due to its antioxidant activity. This hypothesis regarding the mechanism of ascorbate protection remains to be tested definitively. Ascorbate is known to affect many aspects of cellular metabolism, and it could be through one of these other roles that vitamin C protects against acute light damage. While ascorbate may inhibit free radical-mediated autoxidation under certain conditions, it is also capable of promoting autoxidative reactions in vivo. In contrast, the only clearly demonstrated biological function of vitamin E is protection against autoxidative damage, and vitamin E does not promote autoxidation in vivo under any conditions that have been examined. Thus, manipulation of dietary vitamin E intake provides a better model for evaluating the role of photosensitized autoxidation in retinal damage mediated by light than do experiments involving alterations in retinal ascorbate concentrations. The failure of vita-
min E to protect the retina against either acute light damage or the damaging effects of bright clyclic light, and the failure of either type of light effect to produce a specific loss of retinal vitamin E indicate that photosensitized autodisruption is unlikely to play a significant role in light-mediated damage to the retina.

Key words: retina, light damage, vitamin E, aging, cell loss

References