HORMONAL INFLUENCES ON RETINAL PHOTODAMAGE

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Photic stimuli influence the function and behavior of animals in a number of ways. As analyzed by Wurtman (1), photic information serves at least three functions in mammals: 1) it acts as a stimulus for the complex phenomenon of vision, 2) it regulates optic autonomic reflexes, and 3) it controls certain "neurovegetative functions", such as gonadal maturation and some endocrine rhythms. Environmental photoperiodicity participates in the regulation of sexual development of the rat (2). Diurnal and seasonal photic changes can synchronize, or even generate, rhythms in endocrine function, such as reproductive hormone cycles. The origin and continuation of the rhythms are dependent in many instances on feedback mechanisms involving gonadal hormones and hypothalamic and pituitary vascular connections. Whether pituitary glands and gonadal hormones have feedback functions directly with the photoreceptor organ, the eye, is unknown at this time.

While studying the effects of continuous photoperiod and intraocular serotonin metabolism on reproductive cyclicity in the laboratory rat, a severe and statistically significant reduction in the number of retinal photoreceptors was observed in the eyes of animals exposed to low-intensity visible illumination (3). Animals reared in and exposed to cyclic photoperiods of the same intensity did not have this retinal defect. A search of the literature related to this study indicated that Noell et al. (4) previously had described this phenomenon after exposing several kinds of animals to more intense visible light, but its association with continuous exposure to a relatively low intensity level of illumination had not been observed previously. Several questions arose as the result of these reports and were related to the influence of photically-damaged retinal receptor cells on...
visual activity, photically regulated endocrine responses, visually and optically mediated and guided behavioral responses, and behavior of the animals, in general. Other considerations have been concerned with the causative mechanism of receptor damage and subsequent degeneration and whether the damage could be experimentally reversed or prevented by manipulating pharmacologically the light-exposed animals.

LOW-INTENSITY VISIBLE LIGHT AND RETINAL DAMAGE

Noell et al. (4) reported a functional deficit in the visual system of rats examined one week after exposure to high levels of illuminance (800 Ft-C) for 24 hours. Examination of the retina of these animals revealed a destruction of portions of the photoreceptor and pigment epithelial layers. The damaging effect of light exposure was shown to be related to the body temperature of the rats during the exposure period, the length of the exposure time and the intensity of the fluorescent light source. Similar changes were reported by Gorn and Kuwabara (5) in rats exposed to high levels of white (2000 Ft-C) and colored (200 Ft-C) light for periods ranging from two hours to more than seven days. A later electron microscopic study (6) of retinas from similarly treated rats showed that some of the earliest changes caused by intense light exposure include a separation and vacuolation of the lamellar structure of the outer segments of the photoreceptors. The damaged outer segments increased in diameter two-fold, and they were irregularly distorted in shape. When an extremely bright light was applied, or the environmental temperature was elevated, the destruction of the receptor cell body preceded the damage to the outer segment, an observation made earlier by Noell et al. (4) with the light microscope. After five days of exposure, pigment epithelial cells showed a marked increase in height and a lengthening of their microvilli. Kuwabara and Gorn (6) found that the retinal tissue showed a great capacity for structural and functional recovery after five days of exposure, if the animals were placed in dim light. These investigators did not describe the events related to the regenerative process.

Albino rats kept in constant illumination at an intensity comparable to that found in many animals rooms, that is, 18 Ft-C as reflected from the cage floor or 75 Ft-C direct lighting, develop a retinal degeneration, which in its early stages apparently is limited to the photoreceptor cells (3). Although receptor damage can be detected with the light microscope during the first few days of exposure, rapid destruction of these cells occurs during the first few weeks; after 30 days of exposure to this low-intensity illuminance, no intact photoreceptors were observed in the retina. Remnants of photoreceptors, particularly cone nuclei, have been described in the retinas of rats exposed for long periods of time (7). Contrary to the effects of high levels of illuminance and