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SIGNAL TRANSMISSION FROM RODS TO GANGLION CELLS IN RAT RETINA AFTER BLEACHING A PORTION OF THE RECEPTIVE FIELD

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SUMMARY

1. Recordings from single axons of retinal ganglion cells in the rat's optic tract in response to small flashing test lights were used to follow the course of dark adaptation after exposing half of the receptive field to a bleaching light.

2. The recovery of log sensitivity followed an exponential time course in the exposed and unexposed half-fields. The curves had different time constants, with the exposed side taking longer to recover.

3. The time constants of recovery were increasing functions of exposure, but the rate of increase was different in the exposed and the unexposed half-fields. Direct exposure increased the time constant at a greater rate than did indirect exposure.

4. Comparison of the time constants of recovery in the exposed half-fields with those for pigment regeneration suggests that sensitivity recovers with the time course of rhodopsin regeneration.

5. Increment thresholds were determined using steady backgrounds which illuminated half of the receptive field. A greater threshold elevation was produced in the directly illuminated half-field compared with the half-field illuminated only by scattered light. Comparisons of the threshold-raising capacity of direct and indirect illumination were used to establish an 'upper bound' on the magnitude of light scatter. The time courses of the recovery of sensitivity after two different bleaches were compared. First, thresholds were measured in the unexposed half-field after a half-field bleach. Secondly the recovery of sensitivity after direct bleaching-exposure to the predetermined scatter 'upper bound' was measured. Recovery was more rapid in the latter case than the former, thus indicating that adaptation spreads laterally *via* some process other than light scattering.

INTRODUCTION

Dark adaptation, the slow recovery of sensitivity following exposure to bright light, is common to both human and animal vision. It has long been known that it is the retina which is desensitized for a considerable period of time. For example, the responses of retinal ganglion cells of rats and cats show a profound and long-lasting reduction in visual sensitivity following exposure to light which bleaches measurable

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amounts of visual pigment (Granit, 1941; Barlow, Fitzhugh & Kuffler, 1957). The exact nature of the effects that lead to this persistent loss of responsivity is not yet known, but recovery often parallels the time course of pigment regeneration (Dowling, 1960: Rushton, 1961). This in itself might tend to implicate the photoreceptor outer segments as the site of visual adaptation. Several attempts have been made to test whether the loss in sensitivity is restricted to the exposed photoreceptors (Rushton & Westheimer, 1962; Andrews & Butcher, 1971; Barlow & Andrews, 1973: Bonds, 1974; Cicerone & Green, 1980*b*). While there is far from complete accord on the outcome of these experiments, there is considerable agreement on one point: the desensitizing effects due to bleaching a particular region of the retina are not restricted to the region directly illuminated. This suggests, but does not prove, that adaptation is not entirely a local event. The difficulty is that it is virtually impossible to confine light to a restricted region of retina. The remote effects might be entirely due to stray light.

In our previous experiments we determined whether bleaching a small area of the receptive field centre of a rat retinal ganglion cell reduced sensitivity globally, throughout the receptive field, or only locally, near the bleached photoreceptors (Cicerone & Green, 1980*b*). We found that the desensitization measured at positions near the bleach was of greater magnitude and longer duration than that at far positions in the receptive field. The portion of the receptive field not directly illuminated, however, did suffer a sensitivity loss which persisted for many minutes.

The experiments reported here were designed to answer the question of whether this occurs trivially because of stray light or because of neural interactions which can extend beyond the areas directly bleached. We present two lines of evidence in favour of neural mechanisms. One of these takes advantage of the empirical observation that the direct and remote effects of bleaching are graded differently with different intensities of exposure. If sensitivity were controlled entirely by the amount of visual pigment bleached locally then a light which produces a given amount of bleaching in the rods will have the same effect on the resulting dark-adaptation curve whether the bleach results from direct illumination or from stray light. Moreover, if light scatter is the only reason for lateral spread of the effects of bleaching, then increasing the bleach luminance by some factor should increase the scattered light by the same factor. If two dark-adaptation curves were matched after a weaker bleach they should still coincide after a stronger bleach. They do not.

Our second experiment, modelled after an earlier one of Rushton & Gubisch (1966) and Alpern, Rushton & Torii (1969), uses the idea that if adaptation is a local event it should not matter whether a steady light falling on the region tested is due to direct illumination or light scatter, as a given amount of light should have the same effect in raising threshold. Using field adaptation with a half-field we established an 'upper bound' on the magnitude of light scatter and then applied this result to the bleaching experiments. Recovery after direct exposure to this 'upper bound' was compared with recovery in the 'unbleached' half-field. Sensitivity was restored after direct exposure considerably more rapidly than after remote exposure. Thus both experiments show that light scatter cannot be the only factor in the spread of bleaching adaptation.

METHODS

Apparatus

The methods are as described before (Cicerone & Green, 1980*a*, *b*) and are summarized here.

The adapting source was a Kodak Carousel, model 800, with a 500 W lamp back-projected onto the tangent screen. Light from this source was modified by neutral density filters and was varied in its spatial extent by stops that provided full-field, half-field or small-spot illumination of receptive fields. The test stimuli were two 1° circular fields, one derived from a 150 W xenon arc lamp and the other from a 150 W tungsten lamp.

Calibrations

The luminance of the white bleaching light was measured with an SEI photometer that had been calibrated against a standard lamp (Macbeth illuminometer). A luminance measurement was made for each bleaching light as it was presented during the course of an experiment. Whenever possible, multiple measurements were made and averaged.

The following procedure was used to estimate the quantal absorptions and the amount of pigment bleached. The retinal illumination in terms of scotopic trolands was calculated from the photometric measurements by taking into account the area of the pupil (19 mm^2 with the fully dilated 5 mm diameter natural pupil) and the photopic to scotopic conversion for our 3000 °K tungsten source (1·5). In man, one scotopic troland of 500 nm retinal illumination produces 5×10^6 quanta/mm^2 sec incident on the retina. Retinal illumination is inversely proportional to the square of the posterior nodal distance. The posterior nodal distance of the rat eye is 2·97 mm (Block, 1969) as compared with 16·7 mm in man (Le Grand, 1957). The ratio of the squares of the nodal distances was applied to obtain $1{\cdot}6 \times 10^8$ quanta/mm^2 sec incident on the rat retina due to each scotopic troland. Using the figures of 4×10^5 rods/mm^2 for the rat and 25 % of the incident quanta absorbed (Cone, 1963), gives 100 quanta *absorbed* by a rod for each scotopic troland of retinal illumination. The fraction of pigment bleached in a rod was calculated using $1-\exp(-It/N)$, where I is the quantal absorption per second, t is the bleach duration in seconds and N ($= 3{\cdot}2 \times 10^7$) is the number of rhodopsin molecules (Cone, 1963). For example, the strongest bleaching stimulus used in the experiment shown in Fig. 2 produced a photopic luminance of 54 cd/m^2 on the tangent screen. For a tungsten source and the 5 mm diameter pupil, this stimulus would produce 3·2 log scotopic trolands of retinal illumination and each rod would absorb 5·2 log quanta/sec. Using $t = 60$ sec the fraction of pigment bleached is calculated directly as 0·26. The above is equivalent to taking Q_e, the bleaching energy that leaves 1/e of the dark-adapted rhodopsin unbleached, to be 15·70 log *incident* quanta/cm^2. This agrees well with Perlman's (1978) empirically measured value of $15{\cdot}9 \pm 0{\cdot}4$ for the normal rat.

Procedure

A ganglion cell's receptive field profile was determined by placing a small (1°) spot of light in various locations and measuring the light necessary to evoke a response of 6 spikes/sec above base line firing rate (Cicerone & Green, 1980*a*). This same criterion was used to track the recovery of sensitivity during dark adaptation. Before measuring dark adaptation, test lights were positioned so as to fall upon two equally sensitive positions to the right and left of receptive field centre. In each dark-adaptation run, following 60 sec exposure of a half-field, two curves were obtained by measuring thresholds alternately in each half-field.

Curve fitting

Simple exponential curves were routinely fitted to all dark-adaptation curves in such a way as to satisfy a least squares criterion. This was done by using a fast, efficient computer search routine (Chandler, 1965). Data points at times less than 1 min were not used. The fits were constrained to return to within 0·1 log units of the absolute threshold determined before the dark adaptation run.

RESULTS

Local adaptation

Previously we have reported that when a sub-area of a ganglion cell's receptive field is bleached the desensitizing effects of exposure are not confined to the exposed area (Cicerone & Green, 1980*b*). This finding is illustrated in Fig. 1, which shows results from a single ganglion cell axon after half of its receptive field centre was bleached. Two equally sensitive locations 1·7° on either side of the receptive field

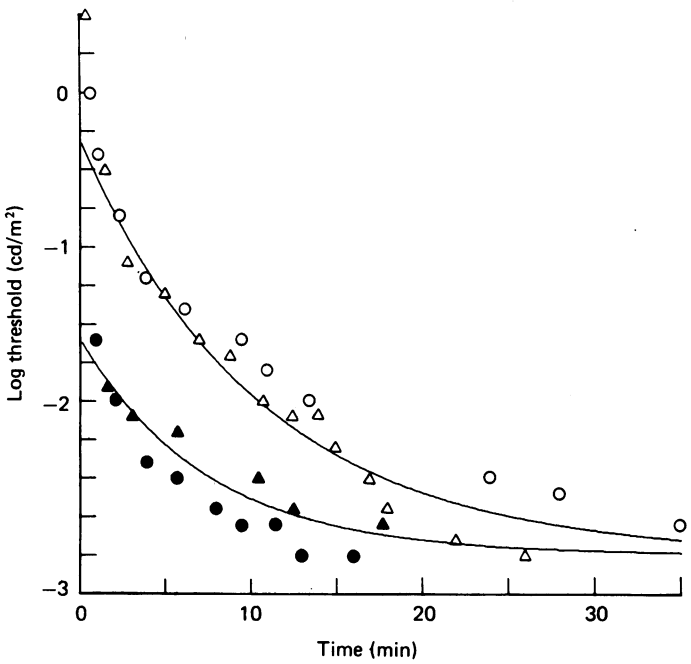

Fig. 1. Dark adaptation of a single retinal ganglion cell after exposing half of the receptive field to a bleaching stimulus. The recovery of sensitivity on the bleached side of the field (open symbols) proceeds with a different time course to that of the unbleached side (filled symbols), independently of whether one side (circles) or the other (triangles) is exposed. The smooth curves are simple exponential decays with time constants of 11·5 min (bleached) and 6·1 min (unbleached).

centre were chosen. At these locations the fully dark-adapted sensitivity determined with the 1° test lights was a factor of 2 (0·3 log units) below the maximum determined at the centre. The retina was then exposed for 60 sec to a semicircular bleaching stimulus approximately 12° in the radius which bisected the receptive field. After the bleach, dark-adaptation curves were obtained for the test lights placed in the bleached and in the unbleached half-fields. Following complete recovery, the conjugate half-field was bleached and a second pair of dark-adaptation curves was measured. The two curves measured in the bleached half-fields (open symbols) were similar and showed a slower time course of recovery than the thresholds measured in the unbleached half-field (filled symbols). Exponential functions with time constants of 11·5 and 6·1min adequately fit the data obtained in the bleached and

unbleached half-fields respectively, independently of whether the left or the right half was bleached.

Strength of the bleach

Fig. 2 shows our measurements of dark adaptation following graded amounts of bleach. Three pairs of dark-adaptation curves are illustrated, which were measured on a single unit by determining thresholds in bleached and unbleached half-fields after exposure to three different bleach luminances, estimated to bleach 3·0, 9·0 and 26 %

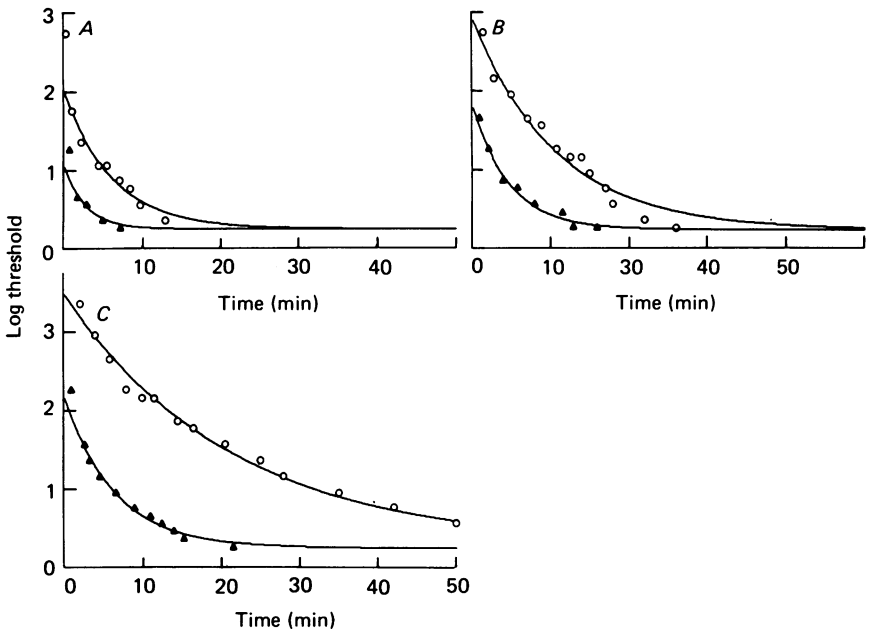

Fig. 2. Dark adaptations following various bleaching exposures. Same unit as in Fig. 1. Exposure was increased in 0·5 log unit steps. The highest exposure (*C*) was estimated as 6·9 log quanta absorbed per rod and was calculated to bleach 26 % of the rhodopsin. The smooth curves are exponentials fitted using a least squares criterion.

of the pigment respectively. Exponential decays describe the time course of recovery reasonably well except for times less than 1 min. The time constant of the best-fitting exponential increased with bleach strength. In the exposed half-field the time constants obtained after the three bleaches were 6·1, 10·5 and 21·0 min respectively. The recovery curves in the unbleached half of the field had time constants of 2·8, 4·6 and 6·4 min at the three corresponding bleach levels. In comparing the 9 to the 26 % bleach it can be seen that the time constant doubled in the bleached half-field and increased by a factor of 1·4 in the unbleached half-field. As bleach strength was increased, a more pronounced slowing of dark adaptation in the directly exposed half-field compared with the spared half-field was a property of all the cells we have tested in this way. Fig. 3 summarizes all of our information on this point. The time constants of recovery are plotted from data measured on single units in bleached and unbleached locations after exposing half-fields to bleaches of varying effectiveness.

The time constants were all obtained by fitting a single exponential function to each set of dark-adaptation measurements.

The results in Fig. 4 provide further evidence on the direct and indirect effects of graded bleaching. All threshold measurements shown were made on the same unit at the same test location. Half of the receptive field, opposite the half where the test stimulus was placed, was exposed to a light which bleached 9 % of the visual pigment.

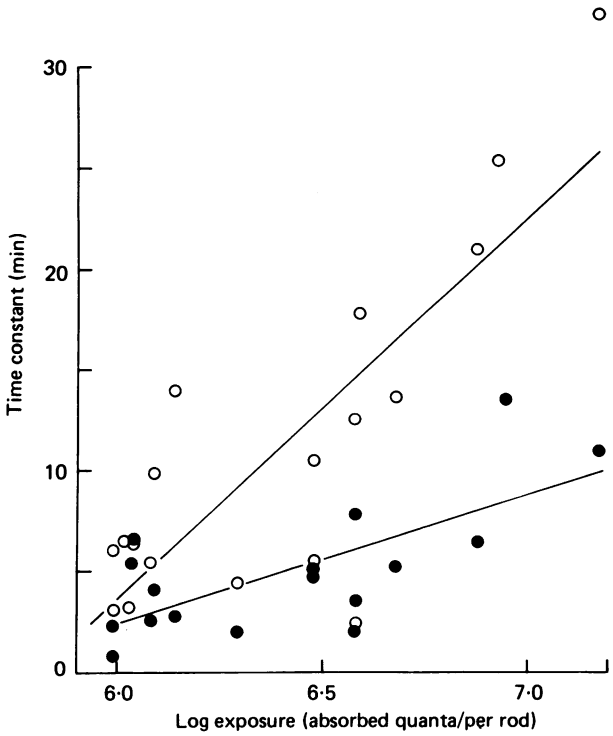

Fig. 3. Rate of recovery of sensitivity in the bleached (○) and unbleached (●) half-fields after exposure to various bleaching intensities. The points are the time constants of the exponential fit to the measurements of the recovery of sensitivity. Linear regression lines are drawn through the two sets of data.

A dark-adaptation curve was determined in the unbleached half-field. After complete recovery the bleaching stimulus was placed in the same half-field as the test stimulus and attenuated so that it bleached 3·3 % of the pigment, a level which we had estimated, based on experiments like those shown in Fig. 2, would yield a dark-adaptation curve similar to that measured after a 9 % bleach in the opposite half-field. The dark-adaptation curve obtained from this 3·3 % bleach is illustrated in Fig. 4. It closely matched that obtained when the stronger 9 % bleach was confined to the conjugate half-field. Thus, we succeeded in matching the recovery from an indirect exposure to the recovery from a direct but weaker bleach. The time constant of the best-fitting exponential was 3·7 min.

The two bleaches were then increased in luminance by 0·5 log units and the experiments repeated. This increased bleaching to 10 and 26 % in the exposed

half-fields. The two relevant curves, one determined after indirect exposure to the 26 % bleach and the other after direct exposure (10 % bleach), do not match. The time constant of recovery after the direct bleach increased considerably more (to 12·6 min) than that after indirect exposure (to 6·4 min). This result is clearly contrary to what would be expected had scattered light been the sole cause of the remote bleaching effects.

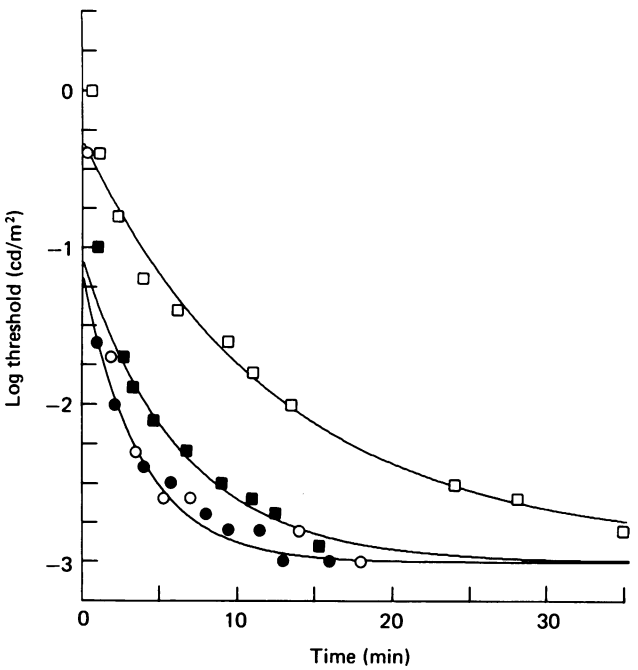

Fig. 4. Recovery of dark adaptation following direct and indirect exposure. Lower curve shows similar recovery of a single retinal ganglion cell after direct exposure to a 3·3 % bleach (○) and an indirect exposure to a 9 % bleach (●). Smooth curve is best-fitting exponential (time constant = 3·7 min). Exposure to three times more light produced unequal dark-adaptation curves. Recovery after indirect exposure (■) has a time constant of 6·4 min and after direct exposure (□) a time constant of 12·6 min.

Perlman (1978) has used retinal densitometry to measure pigment regeneration in the normal albino rat. Following low to moderate partial bleaches rhodopsin regeneration in the dark followed a single-exponential time course. The time constant of regeneration increased with bleaching strength. The filled triangles in Fig. 5 are Perlman's (1978) determinations of the time constants of rhodopsin regeneration after various bleaches. The open circles plot the time constants from our own experiments for the recovery of ganglion cell sensitivity as a function of the estimated fraction of pigment initially bleached. Our estimates of the flux on the retina could easily be systematically off by ±0·1 log unit. This is not a huge error, but bleaching at these intensities is a linear, not a logarithmic, process and consequently 0·1 log unit corresponds to a 20 % error in estimated bleaching. Perlman's data tend to show faster recovery times than do ours. Nonetheless, given the uncertainties in estimating the

quantity of light falling on the retina, the agreement is not bad. The differences could be just a matter of equating the light levels. Thus, as a first approximation, log sensitivity appears to recover at about the same rate as pigment regeneration.

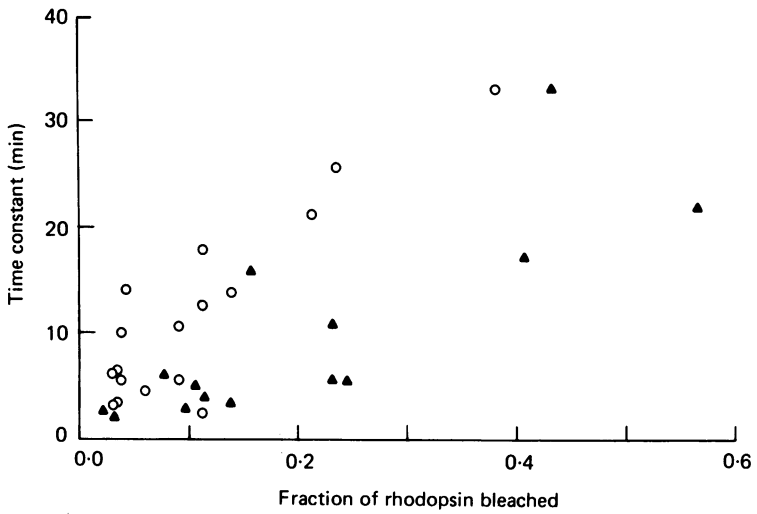

Fig. 5. Comparison of rate of recovery of sensitivity with the rate of rhodopsin regeneration. The open circles are the time constants of recovery in the bleached half-field (replotted from Fig. 3). The triangles are determinations of the time constant of pigment regeneration in normal albino rats from Perlman (1978).

Estimating light scatter

The adaptation we find outside the nominal bounds of our background light could be caused by light scatter and local receptor adaptation, by neural signals which spread laterally and desensitize adjacent photoreceptors, or by changes in the gain of an element in a shared pathway, for example a bipolar cell carrying signals from these photoreceptors to the ganglion cells. Let us assume that adaptation is entirely local and that all of the indirect effect is due to light scatter. Under this assumption we can contrast the direct and indirect effects of an adapting light and, in so doing, obtain an estimate of the upper bound to scattered light. Increment thresholds were determined at two equally sensitive position on either side of the receptive field centre for the unit whose results are show in Fig. 6. A steady, semicircular adapting field was positioned to bisect the receptive field into illuminated and unilluminated halves. Increment thresholds were obtained with a flashing 1° test light in the illuminated half-field and also in the unilluminated half-field. The lines drawn through the two sets of data have unit slope, indicating that under both conditions of this experiment Weber's law is obeyed. The lateral shift along the abscissa (representing background luminance) which is required to superimpose the two data sets reflects the fact that the half-field background less effectively desensitizes receptive field areas outside its boundary. The background is not without effect on the unilluminated half-field, for when the test light is spatially separated from the background a desensitization of up to 2 log units is revealed for this range of background luminances. The lateral

shift of the curves required to bring them to superposition then provides us with an estimate of the amount of light which could be scattered into the unilluminated half-field. For this unit 15 times more light is required to elevate the threshold indirectly than directly. A portion of the indirectly produced change in sensitivity is probably of neural origin (Green, Tong & Cicerone, 1977; Cicerone & Green, 1980*a*), so this estimate provides an upper bound on scattered light.

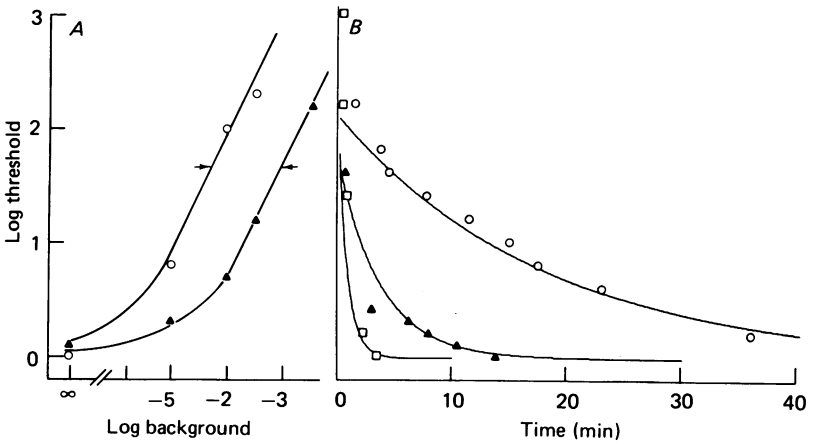

Fig. 6. *A*, increment threshold obtained with half-field illumination of a single retinal ganglion cell. The thresholds in the illuminated and unilluminated halves of the field have the same shape but are shifted relative to each other by 1·2 log units on the log background axis. *B*, the recovery of sensitivity after direct (○) and indirect (▲) exposures to a half-field bleach. The squares show recovery after bleaching with one-tenth of the light. The smooth curves are the best-fitting exponentials.

In Fig. 6*B* are shown two dark-adaptation curves measured following a 60 sec exposure of half of the field to a stimulus estimated to bleach 11·2 % of the pigment. The thresholds were determined in the bleached (○) and unbleached (▲) half-fields after this exposure. The bleaching luminance was reduced by a factor of 10 and a third dark-adaptation curve was determined in the bleached half-field. It is immediately apparent from these data that the recovery following direct bleaching with one-tenth the light is considerably faster (time constant of 0·85 min) than the recovery in the unbleached half-field following the full exposure (time constant of 3·7 min). Clearly, this discrepancy would be greater if one-fifteenth of the light, the estimated upper bound for scattered light, had been used. Thus, the desensitization in the spared half-field is only partially attributable to scattered light.

DISCUSSION

There is general agreement that a bleaching stimulus which falls upon one region of the retina can depress visual sensitivity in another region (Rushton & Westheimer, 1962; Bonds, 1974; Cicerone & Green, 1980*b*). The results we have previously reported are not inconsistent with adaptation confined entirely to localized areas, for the milder effects in remote areas could have been due to light scatter. The experiments we report

here show that this is not so. If adaptation in the unbleached half-field had been due solely to light scatter, then the similarity of the dark adaptation (see Fig. 4) produced first by direct exposure to a bleach and secondly by the indirect effects of a bleaching light three times as bright would imply that the stray light falling on the indirectly illuminated region was one-third of that in the directly illuminated region. When the bleaching light was augmented by 0·5 log unit the corresponding dark-adaptation curves were not the same: the recovery curve for the direct exposure had a much longer time constant than that produced after indirect exposure. This result is incompatible with the hypothesis that the lateral effects of bleaching are due solely to light scatter. The finding that as strength of exposure is increased, local effects grow in magnitude and duration at a faster rate than do the remote effects, seems to be a general property of all the cells we examined. Our data on graded half-field bleaches (Fig. 3) show that the recovery times in the bleached locations are, over a range, described by a linearly increasing function of the logarithm of the number of quanta absorbed from the exposure. The slope of this function is greater than that describing the recovery times in the unbleached half-field.

The measurements in Fig. 6 show that the log increment threshold *versus* log background functions have the same shape, whether or not thresholds are measured in the illuminated or the unilluminated regions. The ratio of direct to indirect illumination required for equal threshold-raising ability, as reflected in the shift required to superimpose the curves, was 1:15. Therefore 7% scatter to the remotely illuminated test region is all that would be required under a scattered-light explanation to account for the results in light adaptation. This is considerably less than the direct exposure of one-third the strength necessary to achieve the same bleaching-recovery curves in the previous experiment. One therefore expects that a one-fifteenth direct exposure will not mimic the lateral effects of bleaching. In Fig. 6 a recovery curve was measured in the spared half-field after an exposure estimated to bleach 11·2% of the pigment in the conjugate half-field. The bleaching luminance was then reduced to one-tenth and applied to the previously spared area. This second dark-adaptation curve, measured after direct exposure to more light than could be scattered by the first bleach, has a significantly shorter time constant than the first curve measured after indirect exposure. A direct bleach reduced by a factor of 15 would undoubtedly recover even more rapidly, so that the recovery of the unbleached half-field after the first exposure is slower than would be predicted if it were solely a consequence of light scatter. The only explanation which seems feasible is that the effects of bleaching can spread neurally, at a site central to photopigment bleaching.

In the present experiments the bleaches are relatively slight; they range from 2·9 to 38% in Fig. 5. Our evidence seems to suggest that over this range of exposures the recovery of sensitivity and pigment regeneration follow approximately the same time course, with restoration of sensitivity lagging slightly behind regeneration. The fact that both direct and indirect exposure result in dark-adaptation curves which are exponential, might suggest that sensitivity is regulated in both areas by a common exponentially decaying signal. The difference in time constants requires that the adaptive signal generated in the exposed rods have undergone a power-law transformation with an exponent less than 1 before reaching the site of sensitivity regulation for lateral adaptation. If so, the ratio of the time constant of recovery in

the bleached half-field to that in the unbleached half-field should remain fixed, independent of the bleach. Our data do not provide strong evidence for a fixed ratio of time constants. The ratios for the three pairs of curves in Fig. 2 are 2·2, 2·3 and 3·3 (in parts *A–C*) respectively. The ratios computed from the pairs of measurements in Fig. 4, which come from eleven cells in ten animals, when plotted as a function of log bleaching exposure, are fitted by a linear regression line having a slope of 0·46. This may indicate the existence of another adaptive process which acts locally but not laterally. Such a process is by no means unreasonable. Penn & Hagins (1972) report that the rod photovoltage responses to bright flashes last for long periods of time. As the response to a bright flash declines, responses to weaker test flashes reappear and grow in magnitude, but the large response declines more rapidly than the test responses grow. Thus the amplitude of the rod response remains depressed even after the return to base line of the photovoltage. These observations support the existence of a mechanism regulating sensitivity distal to the plasma membrane, presumably within the outer segment disks themselves, and this could partially explain the difference in the rates of recovery. Moreover if this distal mechanism were paced by rhodopsin regeneration then the close correspondence between the time constants of regeneration and sensitivity-recovery in the bleached areas would also be explained.

After a half-field bleach, the two halves of the field would be expected to dark-adapt at different rates, since the slow recovery of sensitivity on the exposed side would be regulated locally within the photoreceptors and the more rapid recovery on the unexposed side would reflect more global neural events. The rod photovoltage might be the control signal for these lateral adaptive effects. Following weaker exposures two factors would tend to make the differences smaller. On the exposed side pigment regeneration would proceed more rapidly (Perlman, 1978) and the desensitizing effects of bleached pigment, which vary logarithmically, would be less prominent (Dowling, 1960; Rushton, 1961; Green, Dowling, Siegel & Ripps, 1975). Thus, one would expect that recovery on both sides would be neurally controlled to a larger extent.

Recent work on dark adaptation has emphasized the role of photoreceptor mechanisms (Dowling & Ripps, 1971; Penn & Hagins, 1972; Grabowski, Pinto & Pak, 1972; Kleinschmidt & Dowling, 1975; Pepperberg, Brown, Lurie & Dowling, 1978; Pepperberg & Masland, 1978). In this study we investigated the remote actions of bleaching, which we termed *lateral adaptation*. We cannot yet use this term to imply a particular mechanism but only to describe the situation in which bleaching photopigment in one group of receptors affects the sensitivity to stimuli falling on another relatively unexposed region of the ganglion cell receptive field. The mechanism of lateral adaptation might in fact be signals which spread laterally from the exposed receptors, through inter-receptor contacts or horizontal cells, in some way modifying the gain of the receptor-to-bipolar cell synapse. Alternatively, a neurone in the common pathway might pool adaptive signals from a large number of receptors, any one of which is capable of reducing its gain. Still another possibility is that exposure causes the release of a desensitizing chemical agent which, by lateral diffusion, would desensitize areas not directly exposed to light. Whatever the mechanism, there can be no doubt that several processes participate to determine retinal sensitivity during dark adaptation.

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