Ultraviolet-Induced Sensitivity to Visible Light in Ultraviolet Receptors of *Limulus*

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ABSTRACT In the UV-sensitive photoreceptors of the median ocellus (*UV* cells), prolonged depolarizing afterpotentials are seen following a bright UV stimulus. These afterpotentials are abolished by long-wavelength light. During a bright UV stimulus, long-wavelength light elicits a sustained negative-going response. These responses to long-wavelength light are called repolarizing responses. The spectral sensitivity curve for the repolarizing responses peaks at 480 nm; it is the only spectral sensitivity curve for a median ocellus electrical response known to peak at 480 nm. The reversal potentials of the repolarizing response and the depolarizing receptor potential are the same, and change in the same way when the external sodium ion concentration is reduced. We propose that the generation of repolarizing responses involves a thermally stable intermediate of the UV-sensitive photopigment of *UV* cells.

INTRODUCTION

One class of photoreceptors (*UV* cells) in the median ocellus of *Limulus* is sensitive to ultraviolet light (Nolte et al., 1968; Nolte and Brown, 1972). In these cells, an ultraviolet stimulus elicits a change in membrane voltage, making the inside of the cell more positive (i.e. the depolarizing receptor potential). Under certain circumstances, *UV* cells are also sensitive to longer-wavelength light (Nolte et al., 1968). After an intense UV stimulus, the depolarizing receptor potential is followed by a depolarizing afterpotential which may last for many minutes. An intense visible-light stimulus can abolish such an afterpotential. In addition, a cell which is depolarized by a UV stimulus responds to a superimposed visible-light stimulus with a sustained hyperpolarization.

Light-induced hyperpolarizations have been found in photoreceptor cells of some molluscs and chordates (e.g. McReynolds and Gorman, 1970 *a*), as

well as in all those vertebrate photoreceptors from which intracellular records have been obtained (see Tomita, 1970). However, the light-induced hyperpolarization of molluscs and chordates seems to be generated by a conductance increase mechanism (McReynolds and Gorman, 1970 *b*; Toyoda and Shapley, 1967), while that of vertebrates is generated by a conductance decrease mechanism (Tomita, 1970).

The repolarizing response of median ocellus *UV* cells is the only sustained light-induced negative-going response known to occur in an arthropod photoreceptor. We have tried to determine whether a conductance increase mechanism, a conductance decrease mechanism, or perhaps a third type of mechanism is involved in the generation of repolarizing responses.

METHODS

Both the preparation and procedures for stimulating and recording have been described elsewhere (Nolte and Brown, 1969). In all experiments except those on spectral sensitivity (see below), the light source was a 150 w xenon arc lamp (Bausch & Lomb, Inc., Rochester, N. Y.). The UV stimulus was the 360 nm output (10 nm half bandwidth) of a grating monochromator (Bausch & Lomb, Inc.) coupled to the arc lamp. The long-wavelength stimulus was the output of the same arc lamp passed through a Jena GG-14 filter (Fish-Schurmann Corp., New Rochelle, N. Y.). This filter blocks UV light but transmits longer wavelengths; its transmission is down to 10% by 480 nm. We did not measure the absolute intensity of either UV or long-wavelength light impinging on the photoreceptors. However, the unattenuated UV stimulus was 10^6–10^7 times threshold intensity for *UV* cells; the unattenuated long-wavelength stimulus was approximately 10 times more intense than the unattenuated UV stimulus. These lights were not bright enough to elicit photoelectric potentials (PEP's) (Smith and Brown, 1966) in either *UV* cells or *VIS* cells.

To determine the reversal voltage of receptor potentials a single receptor cell was impaled with two microelectrodes. One electrode was used to pass current, the other to monitor membrane voltage.

The normal seawater used in these experiments was "M.B.L. Formula" artificial seawater (Cavanaugh, 1964, p. 55), buffered to pH 7.8 with 15 mM tris(hydroxymethyl)aminomethane (Tris)–HCl. To change the sodium ion concentration of the seawater, sodium chloride was replaced by choline chloride. The seawater bath was changed by a gravity-feed system; overflow was removed by an aspirator. The volume of the recording chamber was 2 ml, and we perfused with 100 ml of new solution when changing the sodium concentration.

We used a median ocellar nerve preparation to determine the spectral sensitivity of the repolarizing response. The nerve was dissected free of surrounding tissue and divided into several small bundles of axons. A single bundle was drawn into a polyethylene suction electrode. Impulse activity from single units (or occasionally two units) was amplified by conventional electronics. The average spike frequency was measured with a digital ratemeter (Baird-Atomic, Inc., Bedford, Mass., Model 425A). Steady UV illumination was provided by a Zeiss 100 w mercury arc illuminator, the output of which was passed through a Jena UG-1 filter (Fish-Schurmann Corp.).

Longer-wavelength test stimuli were provided by a 75 w xenon arc lamp and a series of interference filters. The intensity of both beams could be varied independently with neutral density filters. The two beams were finally combined into a single beam by means of an interference mirror (Spectro-Film, Inc., Winchester, Mass.) which reflected most of the UV light incident at 45° and transmitted most of the long-wavelength light incident at 45°. The combined beam was focused on the ocellus with a quartz lens.

The intensity of the steady UV illumination was kept constant during any particular experiment. This steady UV light remained on throughout the experiment and produced a maintained spike activity in the nerve bundle; a superimposed longer-wavelength stimulus caused a diminution of this maintained spiking rate. We chose the difference between the spiking rates during and after a long-wavelength stimulus as the response parameter. For each interference filter in the series, we found the response magnitude for several stimulus intensities. Using this data, we calculated a spectral sensitivity curve; we have described the procedure for this calculation elsewhere (Nolte and Brown, 1969).

RESULTS

1. *The Repolarizing Response*

Ultraviolet-sensitive photoreceptors ("*UV* cells") in the median ocellus respond to a UV stimulus by generating a depolarizing receptor potential. After the termination of an intense stimulus, the membrane potential returns very slowly to the original resting value. The rate of return depends on both the intensity and the duration of the stimulus. As the duration of a constant-intensity stimulus increases, or the intensity of a fixed duration stimulus is increased, the rate of return decreases (Fig. 1). The minimum light intensity required to produce this effect is approximately 10^5–10^6 times the intensity required to elicit a depolarizing response from a dark-adapted cell. An example of the most extreme form of this slow after-depolarization is shown in Fig. 2 A. In this cell, there was no significant change in membrane potential at the end of the stimulus, and the potential began to return slowly to the resting value at a rate of less than 1 mv/min. The return to resting potential is always monotonic.

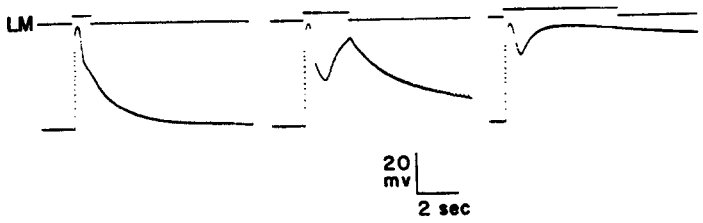

FIGURE 1. The effect of stimulus duration on the depolarizing afterpotential in *UV* cells. All stimuli were of the same intensity, and all responses were from the same cell. The upper traces are the light monitor (*LM*).

The rate of return to resting potential after a UV stimulus can be increased by stimulation with longer-wavelength light. The cell in Fig. 2 was still more than 25 mv depolarized 20 sec after a UV stimulus; presentation of a bright long-wavelength stimulus caused the membrane potential to return to the resting value in several seconds (Fig. 2 B). We call this long-wavelength–induced potential change a "repolarizing response."

Repolarizing responses can also be observed during a prolonged UV stimulus. Responses of this type are shown in Fig. 3. During the steady phase of a UV-elicited depolarizing receptor potential, long-wavelength light elicits a slow repolarizing response which lasts as long as the long-wavelength stimulus. The repolarizing response usually reaches a maximum; then the membrane slowly depolarizes to a new steady voltage (Fig. 3 C). This steady value is al-

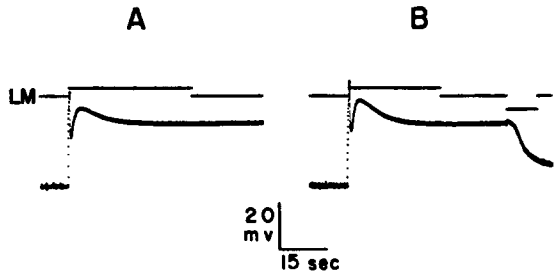

FIGURE 2. Abolition of the depolarizing afterpotential in a *UV* cell by long-wavelength ($\lambda > 480$ nm) light. The upper trace (*LM*) is a light monitor positioned at zero membrane voltage when the light was off. Upward deflections indicate 360 nm light, downward deflections indicate long-wavelength light. The intensity of the 360 nm stimulus is the same in (A) and (B). (A) After a bright UV stimulus, the membrane remains depolarized. (B) Same cell as in (A). The depolarizing afterpotential following a bright UV stimulus is abolished by bright long-wavelength light. There is a brief biphasic receptor potential at the onset of the long-wavelength stimulus.

ways more negative than it would be in the presence of the UV stimulus alone. If the UV stimulus remains on, and the long-wavelength stimulus is turned off, the membrane depolarizes again. Frequently the membrane depolarizes beyond its original steady-phase value before stabilizing at that voltage.

The magnitude of the repolarizing response elicited by long-wavelength light in the presence of a steady UV light is graded with the intensity of the long-wavelength stimulus (Fig. 4). The response may be as large as 30–40 mv, measured from the UV-elicited steady-phase voltage. However, we have never observed a repolarizing response, in the presence of a steady UV stimulus, which reached the original dark resting potential.

Repolarizing responses, elicited either during or after a UV stimulus, are frequently preceded by a brief biphasic receptor potential (Nolte and Brown, 1972), as can be seen in Figs. 2 B and 3 A. The latency of these repolarizing responses is typically 200–500 msec (Fig. 5), but sometimes is longer than 1 sec.

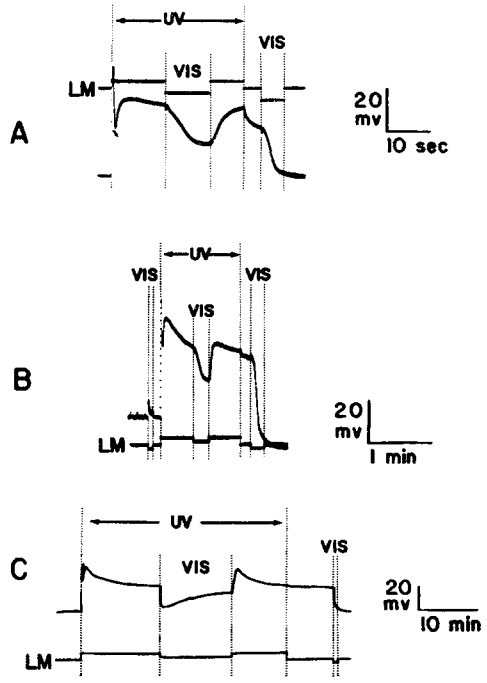

FIGURE 3. Repolarizing responses recorded in three *UV* cells during and after bright UV stimuli. The traces labeled *LM* are the light monitor; upward deflections indicate UV light, downward deflections indicate long-wavelength light. (A) The hyperpolarizing phase of a biphasic receptor potential can be seen at the onset of long-wavelength stimuli. (B) A bright long-wavelength stimulus delivered to a dark-adapted *UV* cell elicits only a small receptor potential. The same stimulus delivered during a bright UV stimulus elicits a repolarizing response. (C) A repolarizing response evoked during a UV stimulus has an initial, maximum hyperpolarization after which it slowly approaches a steady value. After the long-wavelength stimulus is turned off, the cell depolarizes past its original steady-phase voltage, then slowly approaches it.

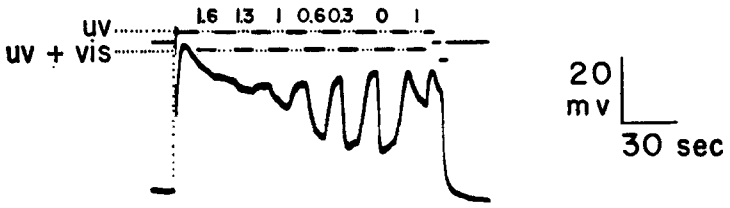

FIGURE 4. Repolarizing responses evoked by long-wavelength ($\lambda > 480$ nm) stimuli of different intensities, during steady UV light. The upper trace is a light monitor; upward deflections indicate UV light, downward deflections indicate long-wavelength light. All long-wavelength stimuli lasted 10 sec; the intensity of each stimulus is indicated above the light monitor trace as the logarithm of the attenuation of the long-wavelength source.

We have never observed any phenomenon analogous to repolarization in the receptor type which generates a depolarizing receptor potential when stimulated by long-wavelength light ("*VIS* cells"). At the end of the most intense stimuli we can deliver, the membrane potential of a *VIS* cell always returns quickly to its original resting value in the dark (Fig. 6). If a bright UV stimulus is delivered to a *VIS* cell during the steady phase of its response to a

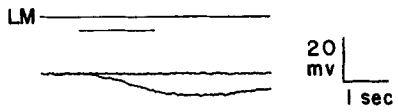

FIGURE 5. Latency of the repolarizing response. A bright UV light was on throughout this experiment. Two sweeps were superimposed; in the second sweep, a long-wavelength stimulus was delivered, evoking a repolarizing response. The upper trace (*LM*) is a light monitor; the downward deflection indicates the presentation of long-wavelength light.

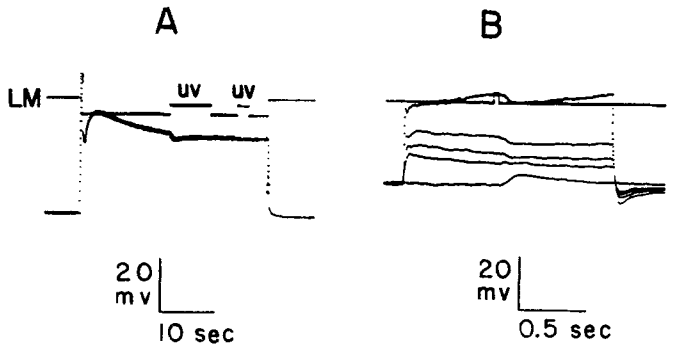

FIGURE 6. UV-evoked hyperpolarizations in a *VIS* cell. The upper trace (*LM*) is positioned at zero membrane voltage when the light is off; upward deflections indicate UV light, downward deflections indicate long-wavelength light. The intensity of all UV stimuli is the same. (A) During the response to a bright, long-wavelength ($\lambda > 480$ nm) light, a UV stimulus evokes a transient hyperpolarization. This response quickly adapts; a second UV stimulus evokes no response. (B) Same cell as in (A). In the presence of a dim long-wavelength light, brief UV flashes were delivered every 30 sec. Membrane potential was displaced by depolarizing current pulses injected through a second electrode. The response to a UV flash is a transient depolarization, which reverses near −20 mv.

bright long-wavelength stimulus, we sometimes see a transient hyperpolarization (Fig. 6). However, we think that this transient hyperpolarization is a different type of response, for the following reasons. It is never maintained for more than a few seconds, whereas repolarizing responses are maintained for the duration of the stimulus (compare Figs. 3 and 6). Also this negative-going response in *VIS* cells is easily light-adapted; a second UV flash delivered soon after the first often elicits no response (Fig. 6). Finally, we find that a similar

UV-elicited, slow negative-going response can be seen in the presence of a dim long-wavelength light, if the cell is depolarized beyond −25 mv with extrinsic current (Fig. 6 B). In this latter case, the response reflects the slow, normally depolarizing, phase of the biphasic receptor potential; since the cell has been depolarized beyond the reversal potential for this phase the polarity of the response is inverted (Nolte and Brown, 1972). The dim long-wavelength background light is necessary to adapt the depolarizing receptor potential of *VIS* cells. Otherwise, the UV stimuli might have elicited a depolarizing receptor potential, although the sensitivity of the cell is much lower to UV than to longer wavelengths (Nolte and Brown, 1969). We have also found that UV-elicited hyperpolarizations in the presence of a bright long-wavelength light (Fig. 6 A) reverse their polarity between −20 and −30 mv (Nolte and Brown, unpublished results), whereas the reversal potential of the repolarizing responses recorded in *UV* cells is at some positive voltage (see below).

Since these slow hyperpolarizations which we sometimes see in depolarized *VIS* cells have the same adaptational properties and reversal potential as the depolarizing phase of the biphasic receptor potential, it seems likely that they are generated by the same mechanisms.

2. *Spectral Sensitivity of the Repolarizing Response*

The monochromatic stimuli available in our system were not sufficiently intense for us to measure reliably the spectral sensitivity of the repolarizing response. Waterman (1953) has shown that action potentials can be recorded from the median ocellar nerve. Virtually all the cells in the median ocellus which generate large action potentials in response to light are UV sensitive, and repolarizing responses can be recorded from them (Nolte and Brown, 1972). All the units in the median ocellar nerves from which we recorded were UV sensitive, and repolarizing responses could be generated in them. Therefore we determined the spectral sensitivity curve for the repolarizing response using the median ocellar nerve preparation. When a long-wavelength stimulus is superimposed on a UV stimulus, the change in spike rate is a linear function of the logarithm of the long-wavelength intensity (Fig. 7 A). Using this response parameter, we determined the spectral sensitivity of the repolarizing response in six preparations. The average curve obtained is shown in Fig. 7 B. We were unable to investigate wavelengths shorter than 425 nm, since they evoked increased spike rates. However, it is clear that the curve peaks near 480 nm.

In one case, we recorded intracellularly from a *UV* cell which was stable and sensitive enough to allow us to determine the spectral sensitivity of the repolarizing response directly. The curve obtained for this cell also peaked near 480 nm.

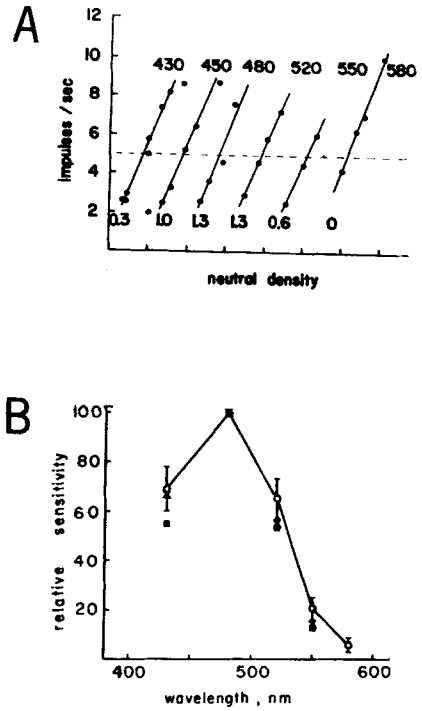

FIGURE 7. Spectral sensitivity of repolarization. (A) Change in impulse frequency elicited by long-wavelength stimuli superimposed on steady UV light. The maintained frequency was about 12 impulses/sec. The frequency during a long-wavelength stimulus is plotted against the stimulus intensity for several different wavelengths. The number at the upper end of each curve is the wavelength. The number at the lower end of each curve gives the logarithm of the stimulus attenuation for the lowest point on that plot. Scale marks on the neutral density axis are 1 log unit apart. (B) Spectral sensitivity of the repolarizing response, determined from data such as that shown in Fig. 7A. Points are the averages of six preparations. Error bars are ±SE of the mean. For comparison, squares are points predicted by Dartnall's nomogram for a visual pigment with λ_{max} at 480 nm. Crosses are values taken from the data of Gogala et al. (1971) and indicate the absorption spectrum of a stable intermediate of the UV-sensitive photopigment of the insect *Ascalaphus macaronius*.

3. *Reversal Potential*

In order to search for a reversal potential for the repolarizing response we impaled single *UV* cells with two microelectrodes; one electrode was used to pass current, the other to monitor membrane voltage.

We found that if the membrane voltage is displaced to relatively large (e.g. 50 mv) inside positive values, the polarities of both the depolarizing receptor potential and the repolarizing response were reversed (Fig. 8 A). That is, if the membrane voltage is initially set at +50 mv, the response to a UV stimulus

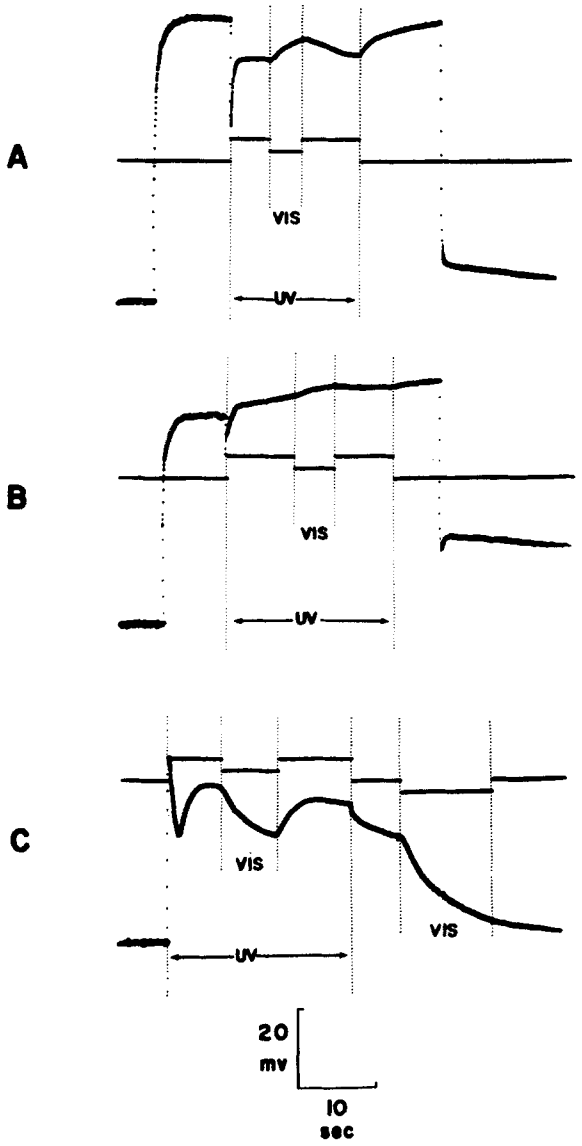

FIGURE 8. Reversal of the repolarizing response. The upper trace is a light monitor positioned at zero membrane voltage when the light was off; upward deflections indicate UV light, downward deflections indicate long-wavelength ($\lambda > 480$ nm) light. (A) When the membrane voltage was displaced to about +40 mv, UV responses and repolarizing responses of reversed polarity were recorded. (B) When the membrane voltage was displaced to about +17 mv, only a very small repolarizing response of reversed polarity was recorded. (C) At resting voltage, normal depolarizing receptor potentials and repolarizing responses were recorded.

makes the membrane voltage less positive; a long-wavelength stimulus presented during this response to UV light causes the membrane voltage to become more positive.

There is some membrane voltage at which a long-wavelength stimulus, presented during a UV stimulus, elicits no response. This is the reversal potential for the repolarizing response. We can find two values of membrane voltage, separated by no more than 5–10 mv, at which repolarizing stimuli cause the membrane voltage to move in opposite directions (Fig. 9 A, B). We call the voltage midway between these two bracketing values the reversal potential. This value should be accurate to within ±5 mv. Using this technique we find the reversal potential of the repolarizing response to be +5–15 mv.

The reversal potential of the depolarizing receptor potential was somewhat more difficult to determine, because different methods gave different results. There is no voltage at which a transient phase of the receptor potential is not recorded; as the membrane voltage is displaced to more positive values, the transient phase changes continuously from a positive-going response to a polyphasic response to a negative-going response (Fig. 9 D). We could choose a fixed time after the beginning of the stimulus, and find a membrane voltage for which there was no light-induced voltage change at that time. For example, in Fig. 9 D we chose the time corresponding to the peak of the transient phase of the response for which membrane voltage had not been displaced. We plotted the light-induced change in membrane voltage *versus* membrane voltage before the stimulus was given (Fig. 9 E). We then determined graphically the membrane voltage at which the voltage change at this time was zero. Reversal potential values obtained by this method are usually 0–10 mv, inside positive. However, if the voltage change is measured at earlier times, more positive values of reversal voltage are obtained. Thus, the value of reversal potential is ambiguous, and depends on the time during the response when it is measured.

To avoid this ambiguity, we favor a second method of measuring reversal potential. This is the determination of the voltage at which the fast voltage change seen at the termination of a UV stimulus ("light-off" response) reverses its polarity. By the same sort of bracketing operation used to find the reversal potential of the repolarizing response, we can find the reversal potential of the light-off response (±5 mv). An example is shown in Fig. 9 C. Reversal potential, determined by this method, is usually about 5–15 mv, inside positive. In any given *UV* cell, this reversal potential and that determined for the repolarizing response are the same, to within the uncertainties of our measurements (Fig. 9 A–C).

If 50% of the Na^+ in the bathing sea water is replaced by $choline^+$, then the reversal potential of the depolarizing receptor potential, measured by either of the methods described above, becomes less positive. The change is usually

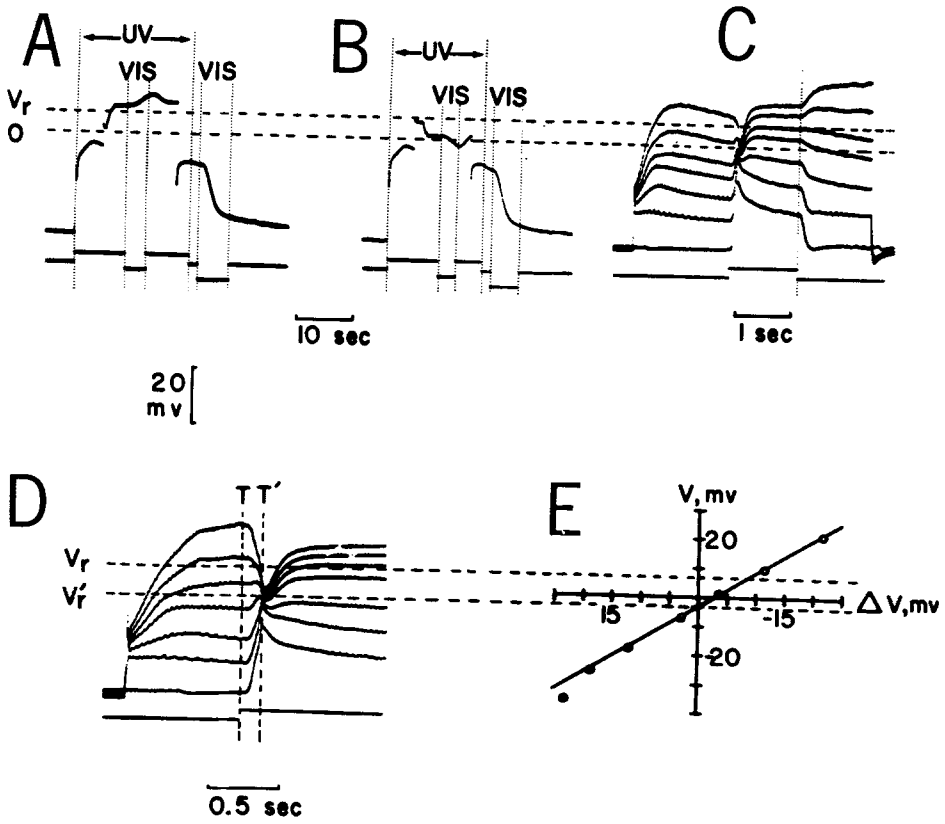

FIGURE 9. Reversal potentials of the depolarizing receptor potential and the repolarizing response. In (A)–(D) the lower trace is a light monitor; upward deflections indicate UV light, downward deflections indicate long-wavelength light. The dashed line marked V_r indicates the estimated reversal potential for the light-off response, determined from (C). The dashed line marked 0 indicates zero membrane voltage. All responses are from the same cell. (A) During the response to a UV stimulus, the membrane voltage was displaced to about +10 mv; a long-wavelength stimulus evoked a response which made the membrane still more positive. (B) During the response to another UV stimulus, the membrane voltage was displaced to about +2 mv; a long-wavelength stimulus evoked a response which made the membrane less positive. (C) UV stimuli were given during depolarizing current pulses. The reversal voltage for the voltage change recorded at the end of the UV stimulus, marked by the vertical dotted line (the light-off response), can be estimated from these records. (D) Same stimulus as in (C). Times T and T' were chosen for a graphical determination of reversal potential, shown in (E). T was the time at the onset of the stimulus; T' was the time at the peak of the receptor potential recorded with no extrinsic current. The dashed line marked V_r' indicates the reversal potential for the transient phase of the depolarizing receptor potential, determined graphically in (E). (E) Graphical determination of the reversal potential of the transient phase of the depolarizing receptor potential. V is the value of membrane voltage at time T. ΔV is the difference between the membrane voltages at times T and T'. The reversal potential determined by this method is about 10 mv negative to that determined in (C).

about 5–6 mv; in any given *UV* cell the reversal potential of the repolarizing response changes by about the same amount as the reversal potential of the depolarizing receptor potential. In most cells, when the Na^+ concentration was reduced by more than 50–60%, the repolarizing response became too small for its reversal potential to be measured accurately.

DISCUSSION

Depolarizing afterpotentials following stimulation with bright light are found in the retinular cells of some insect compound eyes (Hadjilazaro and Baumann, 1968; Naka, 1961) and have also been reported for eccentric cells of the lateral eye of the horseshoe crab *Tachypleus tridentatus* (Tomita et al., 1960). Little is known about the mechanisms underlying any of these afterpotentials. The depolarizing afterpotential which we find in the *UV* cells of *Limulus* median ocelli differs from those found in insect retinular cells, at least in waveform. The *UV* cell afterpotential consists of a slow monotonic return to resting potential after a bright UV stimulus. Insect retinular cell afterpotentials have more complex waveforms, frequently involving an initial partial return to resting potential and a subsequent depolarization (Hadjilazaro and Baumann, 1968; Naka, 1961). Furthermore, the afterpotential is sometimes larger than the steady phase of the receptor potential preceding it (Naka, 1961).

A phenomenon like the repolarizing response which we observe in *UV* cells has not been described for any other type of photoreceptor, to our knowledge.

Any mechanisms proposed for the depolarizing afterpotential and the repolarizing response of *UV* cells must be consistent with the following data: (*a*) only very intense UV lights (at least 10^5 times threshold intensity) are followed by depolarizing afterpotentials; (*b*) repolarizing responses can only be elicited during the response to a very intense UV light (intensity as in [*a*]) or during the subsequent depolarizing afterpotential; long-wavelength stimuli delivered during the response to a relatively dim UV stimulus elicit at most biphasic receptor potentials;[1] (*c*) the spectral sensitivity curve of the repolarizing response peaks near 480 nm and is different from any other spectral sensitivity curve we have measured for electrical responses in receptor cells of the median ocellus (Nolte and Brown, 1969); (*d*) the reversal potential of the repolarizing response is about 5–15 mv, inside positive, which is about the same as the reversal potential of the light-off response of the depolarizing receptor potential. In addition, the reversal potentials of both events change in the same way when the external sodium concentration is reduced.

[1] The most intense long-wavelength stimulus we can deliver is about 5–10 times the intensity of our unattenuated UV stimulus, which in turn is about 10^6–10^7 times the threshold intensity for a completely dark-adapted *UV* cell. It is possible that with brighter long-wavelength stimuli, repolarizing responses could be elicited during the response to relatively dim UV light.

Our data allow us to exclude three possible types of mechanism for the generation of the repolarizing response: (*a*) It cannot be caused by a conductance increase to some ion such as potassium or chloride, like some types of invertebrate IPSP (e.g. Blankenship et al., 1971). A negative-going response generated by a conductance increase mechanism should get larger when the membrane voltage is displaced in the (inside) positive direction, but the repolarizing response gets smaller as membrane voltage is made more positive and finally reverses its polarity at a voltage of 5–15 mv, inside positive. (*b*) It cannot be caused by synapses of *VIS* cells onto *UV* cells, since the spectral sensitivity curve for the depolarizing receptor potential of *VIS* cells peaks at 525 nm (Nolte and Brown, 1969), whereas the spectral sensitivity curve for the repolarizing response peaks near 480 nm. We do not know the spectral sensitivity curve for the depolarizing phase of the biphasic receptor potential of *VIS* cells, but this response adapts quickly and has no maintained component (Fig. 6), whereas the repolarizing response lasts as long as the repolarizing stimulus. (*c*) Finally, it seems very unlikely that it is caused by the action of long-wavelength light on a long-wavelength–sensitive photopigment which is ordinarily present in *UV* cells. The only response to long-wavelength light that can be recorded in a partially dark-adapted *UV* cell is a biphasic receptor potential; neither phase of this receptor potential reverses at 5–15 mv, inside positive (Nolte and Brown, 1972).

The known properties of some inverterbrate photopigments suggest a possible type of mechanism. Many of the arthropod photopigments which have been studied have been found to yield relatively stable intermediates (metarhodopsins) after exposure to light (e.g. Brown and Brown, 1958; Hays and Goldsmith, 1969; Hubbard and St. George, 1958; Wald and Hubbard, 1957). This behavior is in contrast to that of vertebrate visual pigments, which rapidly hydrolyze to opsin and retinal after exposure to light (Hubbard et al., 1965). The acid form of these stable invertebrate metarhodopsins typically has its absorption maximum at 490–500 nm (Brown and Brown, 1958; Hays and Goldsmith, 1969; Hubbard and St. George, 1958; Wald and Hubbard, 1957); rhodopsin can be photoregenerated from these metarhodopsins (Hubbard and St. George, 1958). Gogala et al. (1970) recently extracted a UV-sensitive photopigment from an insect compound eye and found that its properties were similar to those of other invertebrate photopigments: UV irradiation of this pigment resulted in the formation of a stable metarhodopsin (λ_{max} of the acid form = 480 nm) from which the original UV-sensitive pigment could be photoregenerated quantitatively.

We suggest the following model for a possible mechanism for the depolarizing afterpotential and repolarizing response of *UV* cells. We assume that the UV-sensitive pigment (VP360) of *UV* cells yields a thermally stable intermediate (M480) after exposure to light. We assume further that: (*a*) The presence of M480 implies the presence of a "light-activated" patch of membrane. (*b*)

There is a rapid conversion of M480 back to VP360, but the conversion system has a limited capacity. (*c*) VP360 can be photoregenerated from M480.

This model predicts that: (*a*) at low intensities of UV illumination M480 would be converted to VP360 almost as rapidly as it was generated; very little M480 would be present at any time, so no depolarizing afterpotential (and no significant repolarizing responses) would be seen. (*b*) At high intensities of illumination, the capacity of the conversion system would be exceeded and the concentration of M480 would increase. Illumination with 480 nm light during the UV illumination would remove some M480 and hyperpolarize the cell. After the termination of the UV illumination, the cell would remain depolarized until the M480-to-VP360 conversion system could remove all of the M480; the length of time needed for this would depend on the intensity and duration of the UV illumination (see Fig. 1). (*c*) The spectral sensitivity curve of the repolarizing response would peak at 480 nm. (*d*) In terms of its effect on the light-activated membrane, a repolarizing stimulus would be equivalent to termination of a dim UV stimulus. Therefore, the reversal potential of the repolarizing response would be the same as that of the light-off response of the depolarizing receptor potential, and both reversal potentials would change in the same way when the external sodium concentration is altered.

We do not propose this model as unique; there are a number of other satisfactory models and our data do not allow us to distinguish among them. For example, the "light-activated" state of the membrane could correspond to the transition of M480 through the conversion system, rather than to the presence of M480. However, our data do indicate that the depolarizing afterpotential and the repolarizing response are generated in *UV* cells themselves and depend on the production of a stable photoproduct of the UV-sensitive photopigment for their generation.

The authors would like to thank Mr. Paul Brown, and Doctors John Dowling, John Lisman, and Kenneth Muller for their comments and criticism.

This work was supported by National Institutes of Health Grants EY-00151, EY-00312, and EY-00377.

Received for publication 28 June 1971.

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