Effects of Priming Flash Parameters and Dark Interval on Red-induced Afterpotential in *Balanus* Photoreceptors

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**Abstract**

The sequence (a) priming flash, (b) dark interval, and (c) red light induces a long-lasting afterdepolarization (PDA) in *Balanus* photoreceptors. The inward flow of membrane current associated with the decay of PDA was independent of red test flashes, provided that PDA had plateaued at a particular intensity. The influence of wavelength and duration of the priming flash and their interaction with the dark interval were investigated. Increasing the duration of the priming flash produced a systematic increase in PDA duration. The dark interval plays a crucial role in PDA induction. The priming flash duration and the dark interval were reciprocally related, i.e., short flashes followed by long dark intervals induced as much PDA as long priming flashes followed by short dark intervals. The action spectrum for the priming flash was found to correspond to that of the primary photopigment (VP537).

**Introduction**

With a suitable experimental regimen consisting of a priming flash, dark interval, and intense red light, a long-lasting receptor afterpotential can be initiated in *Balanus* photoreceptor (Hochstein et al., 1973). A similar phenomenon has been demonstrated to occur in a variety of invertebrate photoreceptors (barnacle: Hochstein et al., 1973; Brown and Cornwall, 1975a; *Limulus*: Nolte and Brown, 1972; Minke et al., 1973; flies: Muijser et al., 1975; Hamdorf and Razmjoo, 1977; Tsukahara et al., 1977; scallop: Cornwall and Gorman, 1979). This afterpotential was first referred to as the prolonged depolarizing afterpotential (PDA) by Hochstein et al. (1973). The ionic mechanism of PDA in *Balanus* has been shown to be the same as that for the receptor potential (Brown and Cornwall, 1975a), which makes the phenomenon interesting because the long time course of PDA permits a variety of experimental manipulations that might help elucidate the visual transduction mechanism.

Different mechanistic models of PDA have appeared. Some investigators proposed that excitatory/inhibitory events occur that are linked to the transition of the photopigment states (Hochstein et al., 1973). Other investigators...
have proposed that a single excitatory substance linked to the primary visual pigment can account for the phenomena observed (Brown et al., 1974; Hamdorf and Razmjoo, 1979). Two important concepts have evolved from the first model. First, the observation that the color of the priming flash cannot be orange or red suggested that the action spectrum of the priming flash should be blue-shifted from the action spectrum of the receptor potential. Second, short-term inhibition (short compared with the duration of PDA) was proposed to occur when the cell is illuminated with light in the blue-green region of the spectrum, because longer dark intervals produced more PDA. This was thought to be associated with a shift of the metarhodopsin, M_{495}, to the primary rhodopsin form R_{532} (Minke and Kirschfeld, 1978). Thus, in the process of pre-adapting or priming the cell to elicit PDA, M_{495} light should prove most effective. Evidence was also presented to indicate that this same pigment transition is associated with an inhibitory process that terminates PDA. The present investigation was addressed primarily to the question of the dependence of PDA induction on the wavelength of the priming flash and the time interval between the priming flash and inducing flash. Contrary to expectation, it was found that 537 nm was the most effective priming wavelength and that the duration of PDA was dependent on dark intervals up to 30 min.

MATERIALS AND METHODS

Barnacles (Balanus eburneus) were obtained from Marine Biological Laboratories, Woods Hole, MA, and stored at least 48 h in a seawater tank before they were used. Selection of an animal for recording purposes was based on a rapid cirra withdrawal reflex.

The procedure for recording from barnacle photoreceptors has been described previously (Brown et al., 1970). Briefly, the procedure involves removal of the lateral ocellus from the barnacle and dissection of the pigment epithelium and the tapetum to expose the three photoreceptor cells. The preparation was placed corneal-side down on a quartz-bottomed chamber (thickness = 0.5 mm) and suffused with normal barnacle saline. One of the photoreceptors was impaled under visual control with a 3 M KCl-filled micropipette (resistance 6–10 MΩ in 3 M KCl). The membrane potential was recorded differentially between this electrode and a similar extracellular electrode and displayed on an oscilloscope and chart recorder. Cells that were voltage-clamped were impaled with a second KCl electrode led off to a suitable feedback circuit (Brown and Cornwall, 1975b). The steady state resting potential of the photoreceptors was usually about −50 mV after 1 h of dark adaptation.

The normal barnacle saline (NBS) contained: 462 mM NaCl, 8 mM KCl, 12 mM MgCl₂, and 20 mM CaCl₂, buffered to pH 7.65 with 10 mM of Tris(hydroxymethyl)aminomethane. The bath temperature was maintained at 18°C with a Peltier cell.

The photoreceptor was stimulated with rectangular light pulses, 2.5, 5, 10, 15, and 20 s in duration, delivered from an electronically shuttered 150-W tungsten-halogen lamp. Two calibrated fiber optic bundles, 3 mm in diameter, were used (a) to transmit light to within 0.5 mm of the preparation, and (b) to transmit light to a calibrated radiometer. The output of the radiometer was amplified and monitored on an oscilloscope and chart recorder. Interference filters (half-intensity bandwidth 40 nm) and neutral density filters (Balzers, Hudson, NH) were used to vary the wavelength
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and intensity of the light source. The unattenuated intensity of 453 and 650 nm light incident on the preparation was $3.4 \times 10^{14}$ and $1.3 \times 10^{16}$ photons cm$^{-2}$ s$^{-1}$, respectively. Reproducibility of the amplitude and duration of PDA required carefully controlled priming flash (PF), dark interval (DI), and test flash (TF). In the experiments described here, the following procedure was adopted: (a) dark adaptation of the photoreceptor for 1 h after impalement; (b) adaptation of the photoreceptor to a bright red flash (602 nm); (c) dark adaptation of the photoreceptor for 3 min; (d) test stimuli to assay remaining PDA; and (d) presentation of the priming flash. A dark adaptation period of 3 min was always allowed after termination of PDA in each experimental run. This procedure ensured very good repeatability, i.e., the same control PDA amplitude and duration could be obtained over 10 h, provided there was no deterioration of the preparation.

PDA could be elicited in 90% of the cells studied. The photoreceptor cells with no PDA usually had a small resting membrane potential (-10 to -20 mV) immediately after impalement, which slowly improved with time. These cells were also less sensitive to light and the receptor potential produced with 520-625-nm wavelengths showed a slow rising transient phase that did not overshoot zero. PDA could not be elicited in these cells even when using a combination of maximal energy flashes $10^{15}$-$10^{16}$ photons cm$^{-2}$ s$^{-1}$ with long intervals in darkness (DI = 30 min-1 h).

Lantz et al. (1977) have reported variable results for PDA under experimental conditions similar to the present study. We did not experience this degree of variability; however, the phenomenon can be very unpredictable based on the steeply rising function of PDA duration vs. dark interval between priming flash and PDA induction. They reported that PDA decay follows a single time course, which is supported by the present results from voltage-clamped cells.

RESULTS

PDA was monitored by applying red test flashes during PDA (Brown et al., 1974). Stimulation of the photoreceptor with red test flashes after PDA is fully developed does not affect the time course of PDA, i.e., the initial few flashes are the only important ones for the process. This has also been demonstrated for fly photoreceptors by Hamdorf and Razmjoo (1977). Fig. 1 compares the decay of PDA in darkness (A) and during a series of 602-nm flashes at 5-s intervals (B). In both cases, the first red flash produced maximum PDA; in B, the test flashes were presented during decay of PDA but the intensity was reduced to reduce the afterhyperpolarization that follows the receptor potential (Koike et al., 1971). The priming flash and dark interval were the same in both cases. The endpoint of PDA was relatively easy to determine with the red test flashes because as PDA decays, the amplitude of the receptor potential increases, reaching a stable value when the membrane potential repolarizes to the resting level (arrow).

Fig. 1C shows the time course of PDA decay with (▲) and without (Ο) the red test flashes. Data were plotted from Figs. 1A and B. The value for the time constant in this cell was 2 min; the mean time constant from seven different cells under the same conditions was $2.2 \pm 0.2$ min. The amplitude for PDA varied between 25 and 30 mV in these cells.

Membrane Current Associated with PDA

When the photoreceptor was voltage-clamped to the resting membrane potential and stimulated with red light, there was a flow of inward current across
the membrane during PDA (Fig. 2B). The membrane potential changes in the same cell under the same conditions are shown in Fig. 2A. The time course of the membrane current flow in a voltage-clamped cell was studied in more detail by inducing PDA with sequences of 2–10 flashes. At the end of each series, the red light was interrupted to allow PDA to decay (not illustrated). As shown in Fig. 2C, peak PDA current was saturated after eight flashes and any number of flashes greater than this produced the same amplitude and time course (12 s). The same decay slope was obtained with as few as four flashes, but two or three flashes resulted in PDA that decayed with a time constant of ~5 s.

These results are in agreement with a previous report showing that the decay of PDA is exponential (Lantz et al., 1977). However, we found that the time constant of PDA was similar for several different preparations (P < 0.01), provided that the same illumination parameters were used. Previous experiments have shown that PDA decays with a slow time course that is independent of additional red light, provided that PDA is near saturation at that particular intensity (Hochstein et al., 1973; Laiwand et al., 1979).

**Effect of Dark Interval on PDA Development**

Figs. 3 and 4 show the effect of varying the dark interval between a priming flash and red inducing flashes on the time course of PDA. Fig. 3 is from two different cells that show the characteristic types of PDA observed. In the first cell, A–G, the dark interval was systematically varied from 1 to 64 min. The parameters of the priming flash that preceded the dark interval and the red inducing flashes were the same for each trace. At dark intervals >4 min, PDA was essentially at its maximum amplitude after the first red inducing flash.
Nevertheless, the time for PDA to decay to the baseline increased systematically as the dark interval was increased. This occurred even though some decrease in PDA amplitude occurred after the longest dark intervals (traces F and G). There is another interesting feature in this record. PDA and the
receptor potential are not additive, i.e., the receptor potential appears to recover before PDA decays, although the recovery phase appears to take longer, the longer the dark interval. The insert at the right shows PDA in a different cell at a dark interval >1 min. At 10 and 15 s after the priming flash there was little evidence of PDA. In C', which shows a 30-s dark interval, there was some PDA that lasted for several minutes. In this cell, the development of PDA took many more flashes than it did in the cell at the left, as is evident in the tracings in D' taken at longer dark intervals. This phenomenon has been interpreted as a strong inhibitory effect produced by the priming flash (Hochstein et al., 1973). In this case, PDA induction was comparatively slow except after a dark interval of 64 min. Nevertheless, PDA amplitude

![Diagram](image-url)
increased systematically with dark interval, and the maximum amplitude occurred more rapidly at longer dark intervals. As in the first cell, the decay time was dependent on the time allowed between the priming and inducing flashes. It is evident that the time course of PDA development can be different in different cells but the decay time is always proportional to dark interval.

The relation between PDA duration and dark interval between priming and inducing flashes is shown in Fig. 4. Different symbols represent different cells. It is clear from these relations that for a given cell, PDA duration is a steeply rising function of the dark interval for dark intervals <20 min. For dark intervals between 20 and 30 min, the relation is less acute, and after ~30 min there is no further effect of an increasing dark interval. This relation appeared to hold for weak as well as strong priming flash intensities (from $1 \times 10^{14}$ to $2 \times 10^{15}$ photons cm$^{-2}$ s$^{-1}$).

![Graph showing PDA duration as a function of dark interval.](image)
Reciprocity was observed between the priming flash duration or intensity and the dark interval after the priming flash. That is, a weak priming flash can produce the same amount of PDA as a strong priming flash if the dark interval is sufficiently long. This is shown in Fig. 5A. In this experiment, the priming flash duration was reduced from A through D. The dark interval was increased to obtain a PDA with the same duration. It was found that there was a systematic relation between the priming flash duration and dark interval necessary to obtain a certain duration of PDA. For example, in the traces shown, the priming flash duration was reduced by one-half for each successive record and it was found that doubling the dark interval produced the same duration of PDA. This held for dark intervals from ~32 min down to ~1 min. The relation between priming flash duration and dark interval is shown in Fig. 5B. The priming flash duration is plotted on the y ordinate and the reciprocal of the dark interval, min⁻¹, is plotted on the x ordinate. The plot yields a linear relation over a large span of dark intervals and the same relation was found for 15 different cells. Any combination of priming flash duration and dark interval that falls on the straight line produced the same amount of PDA; these combinations showed little variation throughout the entire experiment, provided that there was no deterioration of the cell.

**Wavelength of Priming Flash**

Another parameter of the priming flash that has an effect on PDA duration is the wavelength of the priming flash. A series of records is shown in Fig. 6 in which the priming flash wavelength was varied between 453 and 572 nm, but the quantum content of each of the flashes was adjusted so that they were equal. The dark interval was kept the same in each case (10 min). Using the criterion of the time required for PDA to decay to the control level (arrows), it is clear from the records shown that 537 nm was the most effective wavelength for the maximum duration of PDA. This is the same peak as the action spectrum for the receptor potential (Shaw, 1972), for the light-induced current (Brown and Cornwall, 1975b), and for the primary photopigment (Minke and Kirschfeld, 1978). It should be noted that the amplitude of PDA after the first flash was greater for priming flashes at 520 and 499 nm than it was at 537 nm. However, such a criterion does not prove to be systematic because the amplitude of PDA after the first flash is about the same at 537, 474, or 572 nm.

In Fig. 7, PDA duration is shown as a function of the intensity of priming light at seven different wavelengths. The logarithm of the priming flash intensity is shown on the x ordinate. There is a linear relation between the logarithm of the intensity of priming light and PDA duration for a given wavelength of light. Each of the relations is parallel to the others except those obtained at the blue wavelengths, i.e., 474 and 453 nm. From such relations, an action spectrum for a given cell was obtained by taking the reciprocal number of photons necessary to induce a given duration of PDA. This is shown for seven different cells in Fig. 8. The data have been normalized to the peak of the action spectrum (537 nm) and the points represent the mean reciprocal photons necessary to obtain a given amount of PDA. Bars represent
Figure 5. A. Reciprocity between dark interval and duration of the priming flash. PDA was induced by a series of 602-nm wavelength flashes. The duration of the priming flash from top to bottom was 15, 10, 5, and 2.5 s and the duration of the dark interval was adjusted to produce the same decay time for PDA. B. The relation between reciprocal priming flash duration and dark interval. Any combination of priming flash duration and dark interval that falls on the line produces the same duration of PDA.
Figure 6. Effect of priming flash wavelength on PDA duration. Each priming flash was adjusted to $1 \times 10^6$ photons cm$^{-2}$s$^{-1}$. The wavelength of the priming flash varied from A through G. The dark interval after the inducing flash remained constant. The endpoint of PDA is indicated by the arrows.
standard deviations of the measurements. The solid curve was obtained from a Dartnall nomogram at 540 nm.

**Wavelength of PDA Termination**

The priming flash action spectrum was compared with the action spectrum to terminate PDA using a comparable experimental protocol. Fig. 9 shows the envelope of PDA decay produced by light flashes of equal quanta at several different wavelengths. The first flash produced an initial change in the membrane potential that varied depending on the wavelength of light. The membrane potential change was less at 453 and 548 nm than it was for the other wavelengths shown. The greatest initial membrane potential change was produced by a series of 520- or 499-nm flashes. However, after the initial change, the membrane depolarized slightly before the smooth decay phase. These data are shown plotted on semilogarithmic coordinates in Fig. 9B. Different wavelengths are represented by different symbols as shown in the inset. After the first two flashes, the time course of decay was very similar for
each wavelength. The greatest reduction in PDA amplitude was produced by 520- and 499-nm flashes. Although the difference in amplitude is small between 499 and 520 nm, the time required for the membrane to repolarize ($\Delta t$) was consistently greater at 520 ($138.6 \pm 4.2$ s; SD) than at 499 ($118.2 \pm 2.86$) or 474 nm ($101.2 \pm 3.9$). A $t$ test of the $\Delta t$ among these wavelengths for five cells yielded $520 > 499$ or 474 nm ($P < 0.01$) and $499 > 474$ nm ($P < 0.01$).

**DISCUSSION**

The major observations from this investigation are (a) the duration of PDA is strongly governed by time in the dark after a priming flash; (b) the PDA...
Figure 9. A. Effect of a series of flashes at different wavelengths (indicated beside each trace) on the termination of PDA. Each flash contained $1.0 \times 10^{16}$ photons cm$^{-2}$ s$^{-1}$. Priming flash (PF), dark interval (DI), and test flash (TF) were the same for each experimental run and were $2 \times 10^{15}$ photons cm$^{-2}$ s$^{-1}$, 15 min, and $2 \times 10^{16}$ photons cm$^{-2}$ s$^{-1}$, respectively. The final value of the membrane potential 2.5 min after the records shown is represented by the dashed line. B. Semilogarithmic plot of data. Inset shows symbols corresponding to the wavelength of light used to terminate PDA.
duration-log priming flash intensity relation is linear for less than saturating intensity; and (c) the action spectrum for the priming flash corresponds closely to the action spectrum of the late receptor potential. The dark interval between priming and inducing flash is as great a determinant of PDA duration as priming flash duration and/or intensity. The dark interval and quantum content of the priming flash are reciprocally related, i.e., reducing the quanta of priming flash by half and doubling the dark interval will result in equal durations of PDA. Thus, it appears that PDA is not only caused by the number of pigment transitions that occur during the priming flash but is also dependent on dark interval (Hochstein et al., 1973). The events during the dark interval can be conceptualized in at least two ways: (a) the dark interval represents time to allow accumulation of a substance that is triggered by red light for initiation of PDA and (b) the dark interval represents a period during which an "inhibitor" decays that is capable of annihilating the "excitors" responsible for PDA (Hochstein et al., 1973; Minke et al., 1973). Previous studies (Hochstein, 1973; Minke et al., 1973) have suggested that a presumptive inhibitor decays in <5 min. However, PDA amplitude was used in those studies, whereas PDA duration was the focus in the present investigation and suggests that the presumptive inhibitor may last five times as long. It was pointed out in Figs. 3 and 5 that the receptor potential is not fully developed during PDA and this has been viewed as an inhibitory effect of the priming flash. But as Figs. 3 and 5 also show, the longer the dark interval, the longer the receptor potential is attenuated. This observation does not appear to fit the notion of an "inhibition" that decays in darkness. It is also unlikely that it is some form of light adaptation for the reason that the effects of light adaptation should attenuate with time. Rather, the system acts as though the transducer of PDA is used at the expense of transducing the receptor potential. Even this is oversimplified because the membrane potential of the peak and steady phase of the receptor potential recovers more rapidly than PDA decays. This is especially evident for short dark intervals (e.g., 1 min, Fig. 3).

The evidence from this study indicates that if "inhibition" occurs between a priming flash and PDA in Balanus, it has a much longer decay time than 5 min and occurs by a different mechanism than the "inhibition" that terminates PDA. There were two findings that support this: (a) the longer the dark interval, the longer PDA is; thus "inhibition" appears to have a long time course that is comparable to PDA; and (b) the action spectrum for the priming flash is at ~537 nm rather than at ~495 nm, which corresponds more closely to the action spectrum for "inhibiting" an already present PDA as shown in Fig. 9 (Hochstein et al., 1973; Brown and Cornwall, 1975a; Minke and Kirschfeld, 1978).

As Fig. 6 shows, for moderate intensities of the priming flash, 537 and 548 nm induce PDA for at least 45 s longer than less optimal priming flash wavelengths. At the intensities used in the present study, the action spectrum appears to fit a single pigment nomogram quite closely (Fig. 9). However, previous work using saturating intensities (Hochstein et al., 1973) would indicate a much broader limb at short wavelengths because a 447-nm priming
flash induced more PDA than 584 nm. According to the theory of Hochstein et al. (1973), two opposing effects should be evident with blue-green priming light: (a) an "anti-PDA" that acts to shorten PDA, and (b) an increased rhodopsin concentration that lengthens PDA. Because both effects are maximal at ~495 nm, it is difficult to attribute the present results to either of them. The action spectrum for the priming flash found in this study seems to be closer to the primary photopigment. In another study (Minke et al., 1978) on *B. amphitrite* photoreceptors, the authors conclude that the "saturation spectrum" and "relaxation spectrum" of "preparation for PDA" shows a peak at 510 nm. The present study is not directly comparable with this earlier work because less than saturating priming stimuli were used (equal quanta). Nevertheless, perusal of Fig. 2A (left column) of that paper shows that the amplitude of PDA is greatest after a 543-nm priming flash. In the present study, a linear relation was found between log priming flash intensity and PDA duration. The studies of Minke et al. (1978) and Hochstein et al. (1973) indicate a linear relation between intensity and PDA amplitude. It might be that PDA amplitude and duration are different functions of priming intensity. On the other hand, we find that there is too much scatter in the data of Hochstein et al. (Fig. 4) to justify either relation and the range of intensity of the data of Minke et al. (Fig. 2) is rather narrow (30-fold) compared with the present investigation (2.25 decades).

One criticism of the present experiments is that the action spectrum may have been different had we allowed the dark effect to saturate after each priming flash, i.e., 30 min. This was not possible to evaluate because of the length of the experiments that would have led to deterioration of the preparation. However, experiments involving shorter intervals (5 min) were conducted on five cells and the same action spectrum was found. Thus, there does not appear to be significant interaction between the priming flash wavelength and dark intervals for dark intervals that represent ~50% of the saturated dark interval effect.

The action spectrum for termination of PDA is different than the priming flash. Hochstein et al. (1973) have reported a peak that corresponds to a presumptive metarhodopsin in *B. amphitrite* photoreceptors (490 nm), whereas Brown and Cornwall (1975a) reported a value for the peak of the action spectrum in *B. eburneus* photoreceptors at ~510 nm. The present study shows that the first flash in a series of weak light produces an initial rapid membrane repolarization that is greater in magnitude at 520 nm than it is at shorter (499 nm) or longer (548 nm) wavelengths. Additional flashes of the same intensity do not appear to alter the time course of PDA, i.e., the time course of decay at a variety of different wavelengths was almost the same. Minke and Kirschfeld (1978) have presented evidence that an accessory pigment present in *B. eburneus*, but not in *B. amphitrite*, may explain the experimental differences. The screening pigment has a broad absorbance in the blue region of the spectrum and is present in much greater quantities than the metarhodopsin. Thus, it could shift the apparent action spectrum to the red by an amount predictable from their absorbance measurements.
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