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IONIC EFFECTS ON THE MEMBRANE POTENTIAL OF HYPERPOLARIZING PHOTORECEPTORS IN SCALLOP RETINA

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SUMMARY

1. The effects of different external ionic conditions and of metabolic inhibitors on the membrane potential of hyperpolarizing photoreceptors in the retina of the scallop *Pecten irradians* were examined in the presence and absence of light.

2. Changes in extracellular K^+ have a greater effect on membrane potential in the light than in darkness. The receptor potential is increased in amplitude when $[K]_o$ is reduced and decreased when $[K]_o$ is elevated. It is hyperpolarizing when $[K]_o$ is less than the estimated value for $[K]_i$ and depolarizing when this condition is reversed.

3. The complete replacement of $[Na]_o$ causes a significant hyperpolarization of membrane potential in darkness, whereas it has a much smaller hyperpolarizing effect on the peak of the receptor potential.

4. The ratio of Na^+ to K^+ permeabilities (P_{Na}/P_K) decreases during bright illumination. Our results suggest that P_K is seven times that for P_{Na} in the dark but is 57 times greater than P_{Na} in light.

5. The metabolic inhibitors DNP and NaCN cause membrane potential in the dark to hyperpolarize. This hyperpolarization is associated with a decrease in the P_{Na}/P_K ratio similar to that found during illumination.

6. High $[Ca^+]_o$ also causes membrane potential in the dark to hyperpolarize. This hyperpolarization is associated with an increase in membrane conductance.

7. The results indicate that the hyperpolarizing receptor potential of the distal photoreceptor is produced by a light-evoked increase in K^+ permeability.

INTRODUCTION

Some forty years ago, Hartline (1938) showed that the distal cells in the scallop retina are primary photoreceptor cells which are inhibited by light and excited by its removal. Since that time other examples, of what has come to be called the 'off receptor', have been reported (Kennedy, 1960; Barber & Land, 1967; Land, 1968; Mpitsos, 1973; Wiederhold, MacNichol & Bell, 1973). We showed (McReynolds & Gorman, 1970*a*) that the response of the distal cell to light was a membrane hyperpolarization and that darkness depolarized these cells. Similar responses are found in photoreceptors in the vertebrate retina (Bortoff, 1964; Tomita, 1965; Werblin & Dowling, 1969), but unlike the vertebrate photoreceptor where the hyperpolarizing

receptor potential is produced by a decrease in membrane conductance, the response of the distal photoreceptor is associated with a conductance increase (McReynolds & Gorman, 1970*b*). The purpose of the present paper is to provide evidence that the hyperpolarizing response of the distal cell to light is due primarily to an increase in membrane K^+ permeability. Preliminary evidence for this conclusion has been reported previously (McReynolds & Gorman, 1974; Gorman & McReynolds, 1974).

METHODS

The anatomy and physiology of the distal photoreceptors of the scallop *Pecten irradians* have been described previously (McReynolds & Gorman, 1970*a*, *b*). Individual eyes were removed from the mantle and, after the cornea and lens were dissected away to expose the retina, the eye was held firmly by suction in a channel of a special chamber which has been described elsewhere (Gorman & Marmor, 1970). Artificial seawater (ASW) flowed continuously through the channel. Changes in the perfusing solution were made by manually turning a valve located upstream from the retina, with about 15 sec of dead time between switching the valve and the arrival of a new solution at the eye. Any resulting changes in membrane potential began at that time and were complete within the succeeding 30 sec.

Recordings were made using a capacity compensated electrometer with intracellular micro-electrodes filled with 3 M-KCl (50–100 MΩ resistance). All recordings were referenced to an Ag–AgCl electrode connected through an agar bridge to the flowing ASW in the chamber downstream from the preparations. Changing the perfusing solution with various test solutions caused little (<2 mV) or no change in the junction potential between the micro-electrodes and the reference electrode. Signals were simultaneously displayed on an oscilloscope screen for photography and on a rectilinear pen recorder. An active bridge circuit was used to measure changes in membrane resistance under various conditions. To facilitate penetration of photo-receptor cells a mixture of 1 % pronase and 0·5 % collagenase was applied to the retina for 1–2 min before the experiment.

White light from a tungsten quartz-iodide lamp was passed through an electrically operated shutter and a field aperture which was focused to an evenly illuminated spot that covered the entire retina (approximately 750 μm in diameter). The retina was illuminated with flashes of light of constant intensity, 100 msec in duration presented at 10 sec intervals. A photocell monitored the light stimulus at a point between the shutter and the retina. The irradiance in the range 400–700 nm was approximately 10^{-4} W/cm^2 which was sufficient to produce just saturating receptor potential responses in the distal photoreceptors (see McReynolds & Gorman, 1970*a*).

The composition of the normal ASW solution (m-mole/l.) was Na^+, 425; K^+, 9; Ca^{2+}, 9; Mg^{2+}, 48; Cl^-, 496; SO_4^-, 26; HCO_3^-,2. Solutions of different K^+ concentration were made by substituting K^+ for Na^+ on a one-to-one basis. Sodium-free ASW was made by replacing Na^+ with either tris (hydroxy-methyl) aminomethane (Tris), choline or Li^+. For solutions containing Tris as a Na^+ substitute, the total Cl^- concentration was reduced by 21 %, but since removal of external Cl^- has no effect on the distal cell receptors (McReynolds & Gorman, 1974) this is not an important change. Replacement of choline or Li^+ for Na^+ was done on an equimolar basis. In high Ca^{2+} solutions Ca^{2+} was exchanged for Mg^{2+} on an equimolar basis. For these solutions 10 mM-Tris was used as a buffer. All solutions were kept at the same temperature (22–23 °C). The normal ASW solution given above has an osmolarity of 904×10^{-3} osmoles and a pH of 7·8. The pH of various test ASW solutions ranged from 7·7 to 7·85 and the osmolarity from 880 to 1000×10^{-3} osmoles. No correction has been made for these differences which are relatively minor. The metabolic inhibitors 2–4 dinitrophenol (DNP) and NaCN were added directly to ASW solutions and the pH of these solutions adjusted to 7·75.

RESULTS

The membrane response to changes in $[K]_o$

A change in the extracellular potassium concentration $[K]_o$ affects both membrane potential in the dark and at the peak of the receptor potential in response to brief flashes of light, quickly and reversibly (Fig. 1). Although the effect of a change in $[K^+]_o$ on membrane potential in the dark and on the response to illumination are qualitatively the same, there are differences. Fig. 1 shows that an increase in $[K]_o$ to approximately three times normal had a more pronounced effect on the receptor potential than on membrane potential in the dark. This difference is apparent over a wide range of concentrations and is illustrated in Fig. 2 where absolute membrane potential values under the two conditions are plotted as a function of the logarithm of $[K]_o$. The receptor potential became more negative when $[K]_o$ was reduced, and more positive with increases in $[K]_o$, whereas membrane potential in the absence of light was only slightly affected by a reduction in $[K]_o$ or by small increases in $[K]_o$. Membrane potential in both light and dark were significantly depolarized at higher values of $[K]_o$.

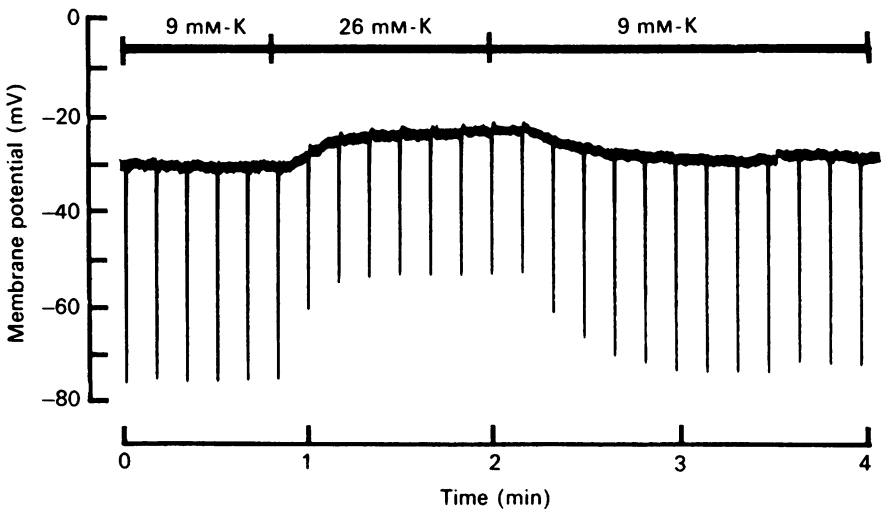

Fig. 1. The effect on membrane potential in darkness and light of changing $[K]_o$ from normal to about 3 × normal. Continuous line is membrane potential in darkness, downward deflexions are hyperpolarizing responses to 100 msec flashes of light.

If the greater dependence of the receptor potential on $[K]_o$ occurs because light increases the K^+ permeability (P_K) of the receptor membrane, driving the membrane potential toward the K^+ equilibrium potential (E_K), then from the Nernst equation for K^+ ions (where $E_K = RT/F \ln [K]_o/[K]_i$), the response to light should be a hyperpolarization as long as the internal K^+ concentration $[K]_i > [K]_o$. Conversely, when $[K]_o > [K]_i$, the receptor potential should reverse polarity. For the cell shown in Fig. 2 the peak of the receptor potential and membrane potential in the dark converged to a value of zero when $[K]_o = [K]_i$, at approximately 350 mM-$[K]_o$. When $[K]_o$ was further increased to 425 mM the membrane potential in the dark was positive rather than negative with respect to an external reference electrode and the photoreceptor response became depolarizing (Fig. 3).

Membrane response to changes in $[Na]_o$

Both choline and Tris are sufficiently large that they are unlikely to penetrate easily through Na^+ channels in the membrane and therefore should be effective replacements for $[Na]_o$. Fig. 4 shows the effect of complete replacement of $[Na]_o$ on membrane potential in the dark and on the peak of the photoreceptor's response to

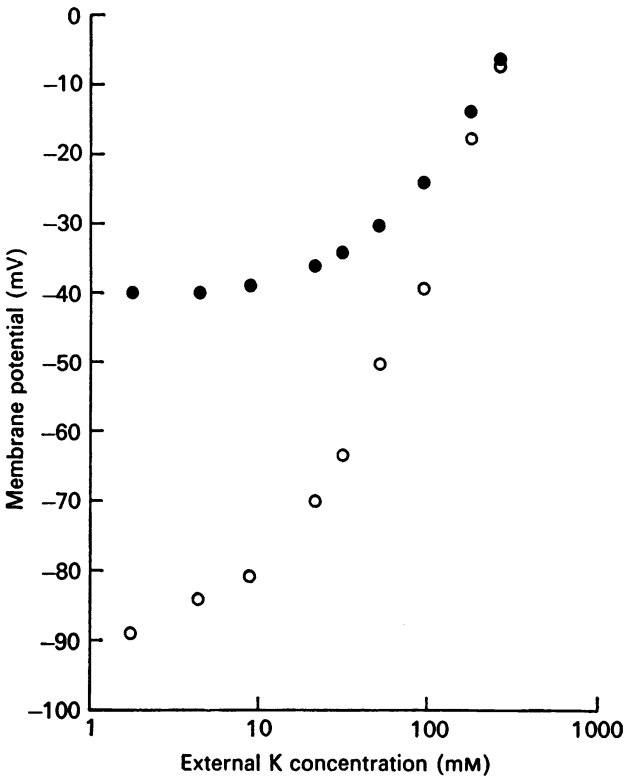

Fig. 2. Plot of membrane potential in the dark (filled circles) and at the peak of the light-induced receptor potential (open circles) *vs.* log $[K]_o$ at 22 °C.

brief flashes of light. Replacement with choline produced an average hyperpolarization of membrane potential in the dark of $-19{\cdot}1 \pm 2{\cdot}4$ mV (S.E. of the mean, nine experiments), and replacement with Tris gave similar results ($-22{\cdot}2 \pm 2{\cdot}2$ mV, S.E. of the mean, ten experiments). Fig. 4. shows that the hyperpolarizing effect of removal of $[Na]_o$ on the receptor potential was much smaller than its effect on membrane potential in the dark. Choline produced an increase in the hyperpolarization of the peak photoreceptor response of $-4{\cdot}5 \pm 1{\cdot}2$ mV (S.E. of the mean) and Tris a hyperpolarization increase of $-2{\cdot}0 \pm 0{\cdot}8$ mV (S.E. of the mean). In three experiments the effect of replacing $[Na]_o$ with Li^+ was compared to the effects produced by choline or Tris. In one experiment Li^+ had no effect, but in the other two instances its effect was equal to that for choline or Tris.

The increase in the permeability ratio P_{Na}/P_K during light

The shape of the two curves in Fig. 2 suggests that they could be fitted with a constant field type equation (Goldman, 1943; Hodgkin & Katz, 1949). Since neither membrane potential in the dark nor at the peak of the photoreceptor response to light is affected by changes in $[Cl]_o$ (McReynolds & Gorman, 1974) and if we assume that $P_{Na}[Na]_i \ll P_K[K]_i$, then membrane potential (V) can be expressed as

$$V = \frac{RT}{F} \ln \frac{[K]_o + (P_{Na}/P_K)[Na]_o}{[K]_i} \quad (1)$$

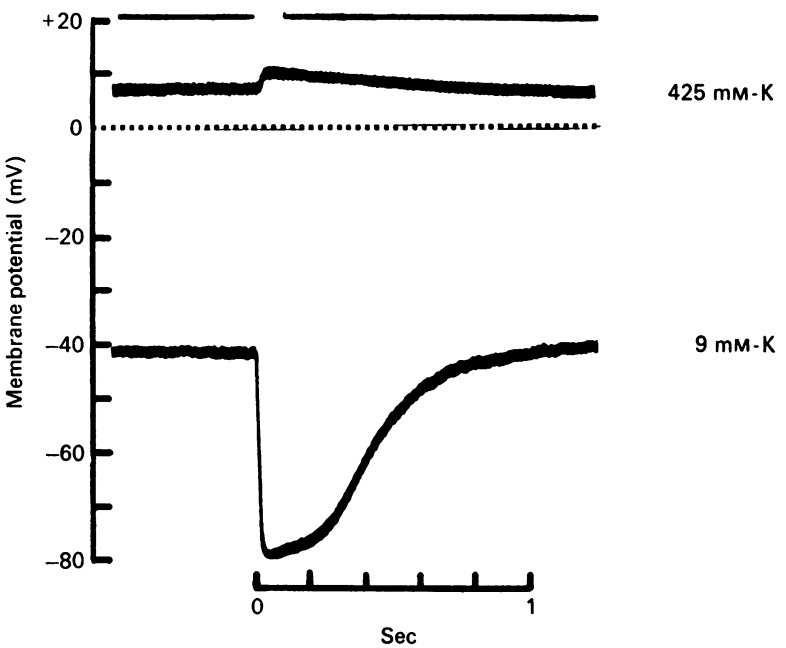

Fig. 3. Effect of high $[K]_o$ or membrane potential. The two traces show the response of membrane potential to a brief flash of light at the indicated external K^+ concentration.

where P_{Na}/P_K represents the membrane permeability ratio for Na^+ and K^+ ions, the subscripts denote the internal (i) and external (o) concentrations of these ions and R, T and F have their usual meaning.

Agreement or disagreement with eqn. (1) can be seen more clearly if eqn. (1) is rewritten in exponential form (Moreton, 1968; Gorman & Marmor, 1970) so that $e^{VF/RT}$ is a linear function of $[K]_o$

$$e^{VF/RT} = \frac{[K]_o + (P_{Na}/P_K)[Na]_o}{[K]_i}. \quad (2)$$

If data are plotted in this manner, points satisfying eqn. (2) will fall along a straight line whose slope allows an estimate of $[K]_i$ and whose y intercept allows an estimate of P_{Na}/P_K.

Fig. 5 shows part of the data given in Fig. 2 replotted as $e^{VF/RT}$ *vs.* $[K]_o$. In agree-

ment with eqn. (2) the experimental points representing membrane potential under conditions of light and darkness fall on two parallel straight lines but with different intercepts (also see Fig. 6). Measurements were made from plots of $e^{VF/KT}$ *vs.* $[K^+]_o$ between the ranges of 0 to 100 mM in ten different cells, using a minimum of four values of $[K]_o$ for each cell. For these ten cells (at normal $[K]_o$) the average value of membrane potential was $-34{\cdot}2 \pm 2{\cdot}46$ mV (S.E. of the mean) in darkness and $-77{\cdot}9 \pm 1{\cdot}98$ mV

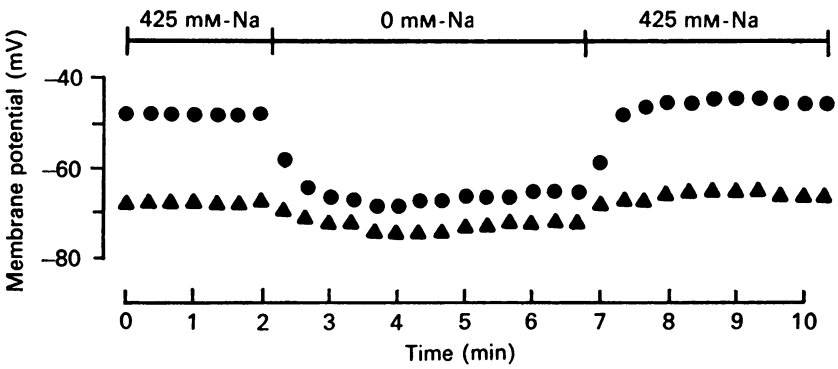

Fig. 4. The effect of $[Na]_o$ removal on membrane potential. The circles represent membrane potential in the dark. The triangles represent the peak of the photoreceptor response to brief (100 msec) flashes of light. $[Na]_o$ was replaced with choline at the times indicated above the plot.

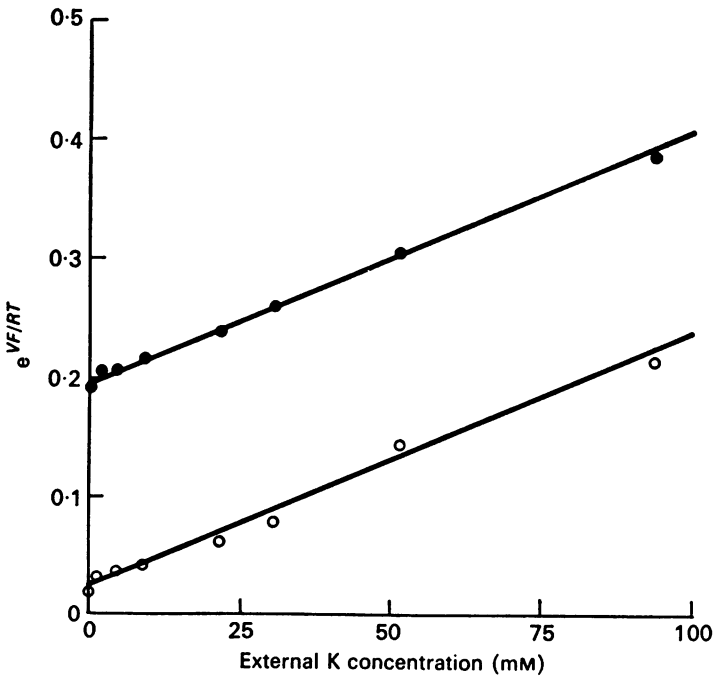

Fig. 5. The relationship between $e^{VF/RT}$ and $[K]_o$ in the dark (filled circles) and at the peak of the photoreceptor response to light (open circles). Data from Fig. 2 replotted as $e^{VF/RT}$. The parallel straight lines are drawn through the experimental points, showing the change in intercept produced by light (see text for further discussion).

(S.E. of the mean) in light (measured at the peak of the receptor potential response). From the plots, the values of P_{Na}/P_K were $0{\cdot}140 \pm 0{\cdot}022$ (S.E. of the mean) in darkness and $0{\cdot}017 \pm 0{\cdot}001$ (S.E. of the mean) at the peak of the receptor potential. $[K]_i$ was $289{\cdot}6 \pm 30{\cdot}5$ mM (S.E. of the mean), and at normal $[K]_o$ the calculated average value

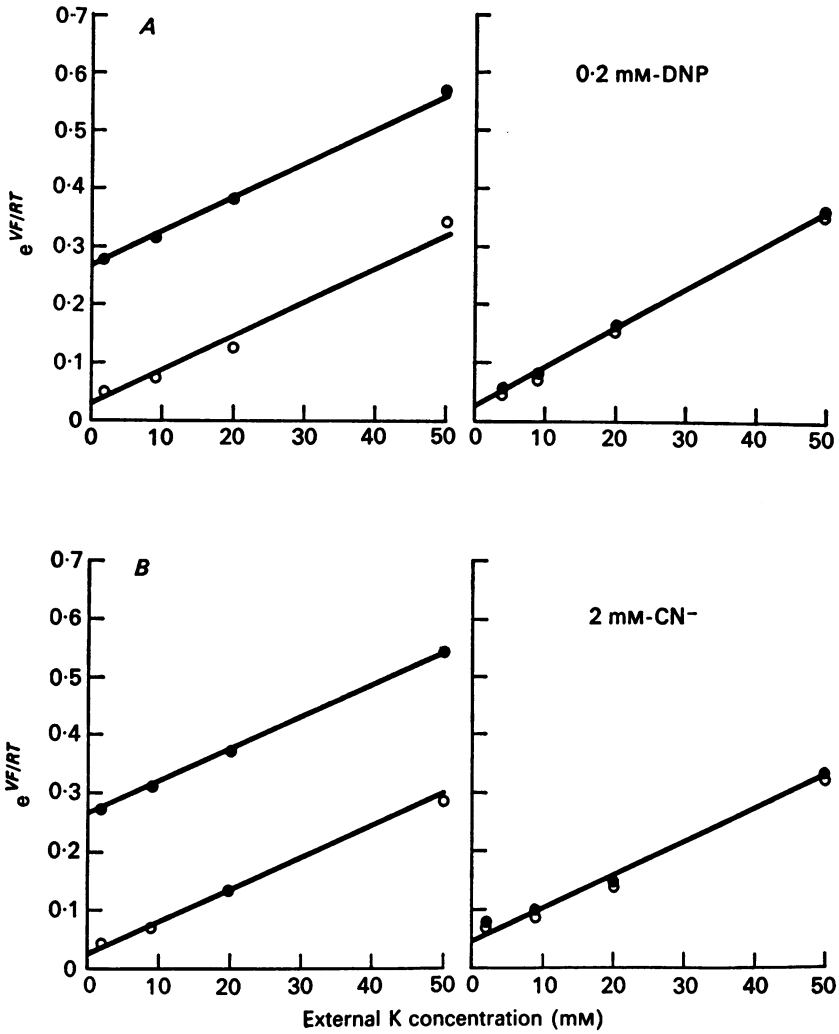

Fig. 6. The relationship between $e^{VF/RT}$ *vs.* $[K]_o$ in the dark (filled circles) and at the peak of the photoreceptor response to light (open circles) before and during the presence of metabolic inhibitors. In *A*, average data from six cells before (left side)and during (right side) 0·2 mM-2·4 dinitrophenol (DNP). In *B*, average data from four cells before and during 2·0 mM-NaCN. The straight lines are drawn to fit the experimental points.

for E_K was $-87{\cdot}5 \pm 2{\cdot}4$ mV (S.E. of the mean). These results suggest that $[K]_i$ remains reasonably constant and that light exerts its main effect on the P_{Na}/P_K ratio which is decreased by a factor of eight in light. The results also show that even in saturating light intensities, the peak of the cell's response to light approaches, but never equals, the estimated value for E_K.

The membrane response to metabolic inhibitors

On a number of occasions cells with normal resting potentials in the dark and sizeable responses to light were observed to spontaneously hyperpolarize to approximately -60 to -80 mV with a decrease in total cell resistance and a concomitant loss of light response. Often this occurred after prolonged recording periods. This effect, although unpredictable, was not due to a general deterioration of the eye because other cells in the same retina continued to show normal membrane potential and response properties. Since K^+ ions carry much of the current across the photoreceptor membrane in the dark, the spontaneous hyperpolarization and decrease in cell resistance suggests that there may be a mechanism within the cell or cell membrane which normally keeps P_K at a low value in the dark.

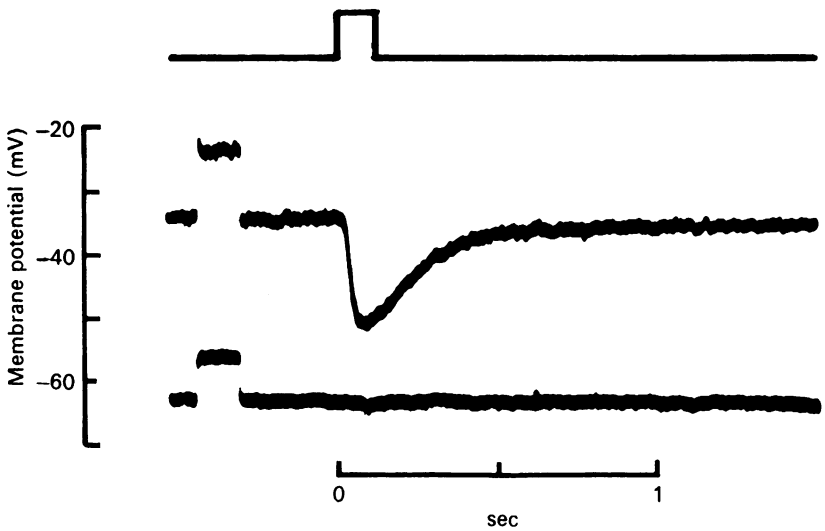

Fig. 7. Effect of increasing $[Ca]_o$ from normal (9 mM) to about $3 \times$ normal (26 mM) on membrane potential. The top trace shows the photoreceptor response to a brief flash of light in 9 mM-$[Ca]_o$ and the bottom trace the response after 26 mM-$[Ca]_o$. The upward deflexion of membrane potential at the beginning of each trace is the membrane response to a current pulse of $1{\cdot}0 \times 10^{-10}$ A. A reduction in response amplitude indicates an increase in membrane conductance.

Both DNP and CN produced a similar hyperpolarization (Gorman & McReynolds, 1974). The effect was rapid and, unlike the spontaneous hyperpolarization, reversible. In the presence of 0·2 mM-DNP or 2 mM-CN both membrane potential in the dark and the peak of the receptor potential showed an initial, transient depolarization. Following this (within 45–60 sec) membrane potential in the dark was hyperpolarized by up to -50 mV to a new maintained value, with a concomitant reduction of receptor potential. In all cases membrane conductance in the dark was increased in the presence of DNP or CN.

Fig. 6 shows the results of changing $[K]_o$ on membrane potential and the receptor potential in the presence of DNP (Fig. 6*A*) and CN (Fig. 6*B*). Membrane potential is plotted as $e^{VF/RT}$ *vs.* $[K]_o$. Both DNP and CN decrease P_{Na}/P_K. For comparison, the results of changing $[K]_o$ on membrane potential in the same group of cells before

treatment with DNP or CN are shown. The plots show the strong similarities between the effects of light and metabolic inhibitors on membrane potential. In Fig. 6*A*, the P_{Na}/P_K ratio decreased from 0·114 to 0·013 in light and to 0·008 in the presence of 0·2 mM-DNP. In Fig. 6*B*, the P_{Na}/P_K ratio decreased from 0·116 to 0·011 in light and to 0·021 in the presence of 2 mM-CN.

Membrane response to changes in $[Ca]_o$

Both DNP and CN block the mitochondrial uptake of Ca^{2+} (Lehninger, Carafoli & Rossi, 1967). An increase in $[Ca]_i$ could increase P_K (Meech, 1974), thereby hyperpolarizing membrane potential in the dark. If the membrane is permeable to Ca^{2+}, increasing $[Ca]_o$ should also lead to an increase in $[Ca]_i$. Fig. 7 shows the effect of increasing $[Ca]_o$ to three times its normal value. The effects are similar to those observed in the presence of metabolic inhibitors. Both membrane potential in the dark and the peak of the receptor response were initially depolarized. Within 30–60 sec this effect was followed by a membrane hyperpolarization and loss of light responsiveness. Neither the loss of the light response or the maintained hyperpolarization were reversible when cells were left in high Ca^{2+} for more than 1 min. Partial recovery occurred when shorter durations were used. Membrane conductance increased during the maintained hyperpolarization and usually persisted when $[Ca]_o$ was returned to normal values. Part of the difficulty in using high $[Ca]_o$ is that most cells were lost when the extracellular Ca concentration was elevated. None of the cells survived treatment with 55 mM-$[Ca]_o$, and of those which survived 27 mM-$[Ca]_o$ treatment only a small fraction showed full recovery.

DISCUSSION

The results presented in this paper, when taken together with those given previously (McReynolds & Gorman, 1970*b*; McReynolds & Gorman, 1974), indicate that the hyperpolarizing receptor potential of *Pecten* distal photoreceptors is due primarily to an increase in K permeability. The hyperpolarization is associated with an increase in membrane conductance and has a reversal potential which is more negative than membrane potential in darkness. Changes in extracellular K^+ have a greater effect on the receptor potential than on membrane potential in the absence of light. The receptor potential is hyperpolarizing when extracellular K^+ is less than the estimated value for intracellular K^+ and depolarizing when this condition is reversed. Moreover, the peak of the response is unaffected by complete removal of Cl^- ions and only minimally affected by removal of Na^+ ions.

We do not exclude the possibility that a light-induced flux of some other ion, or ions, contributes to the receptor potential peak on the basis of the present data. The definitive test would be to examine the reversal potential for the receptor potential at different values of extracellular K^+ to determine if it can be predicted by the Nernst equation for K^+ ions, i.e. does $E_R = E_K$ for all values of $[K^+]_o$. Our data, however, allows us to make some reasonable predictions about the behaviour of the photoreceptor membrane in the light and darkness. For a membrane that is permeable primarily to K^+ and Na^+ ions, membrane potential can be predicted from a simple bi-ionic equation provided that the intracellular and extracellular ion concentrations and the permeability ratio for these ions are known or can be determined. Our results

suggest that in darkness the permeability of the photoreceptor membrane to K^+ is only seven times greater than its permeability to Na^+, whereas at the peak of the receptor potential produced by a just saturating light intensity, the membrane permeability to K^+ is 57 times that for Na^+. At intermediate light intensity the P_K/P_{Na} ratio will fall between these extremes.

If Na^+ were the only ion besides K^+ not in electrochemical equilibrium, and with a permeability comparable to that of K^+, then removal of external Na^+ should cause the potential to follow E_K closely in light and darkness. Experimentally, however, it was found that in the absence of external Na^+ the membrane potential was 10–20 mV positive to the estimated E_K in darkness and 2–6 mV positive to it in light. The simplest explanation is that the membrane has a finite permeability to the Na^+ replacements. This, however, is unlikely to be the full explanation. Substitution of Li^+ for Na^+ had approximately the same hyperpolarizing effect in darkness and light as either tris or choline. The similarity in size of Na^+ and Li^+ when contrasted to their different effects suggests that the membrane is remarkably selective for Na^+ ions. An alternative explanation is that the membrane is also permeable to divalent ions, e.g. Ca^{2+} or Mg^{2+}. Eqn. (1) could, of course, be modified to account for a residual permeability to monovalent Na^+ replacements or a permeability to divalent ions, and thereby provide a better approximation of the membrane behaviour under various conditions, but our present data is insufficient to allow us to make a choice between available explanations.

Metabolic inhibitors and high extracellular Ca^{2+} both apparently produce their effects by increasing K^+ permeability. This action might be explained if we assume the K^+ permeability of the receptor membrane is controlled by the level of intracellular free Ca^{2+} (Gorman & McReynolds, 1974). Both high extracellular Ca^{2+} and the metabolic inhibitors DNP and CN would be likely to increase intracellular Ca^{2+}. High extracellular Ca^{2+} should increase Ca^{2+} influx (Baker, 1972), metabolic inhibitors should increase intracellular Ca^{2+} by reducing or blocking the active uptake of Ca^{2+} by mitochondria (Lehninger *et al.* 1967). If the receptor membrane contains Ca-activated K^+ channels (Meech, 1974) any rise in intracellular Ca^{2+} above a critical level should cause membrane hyperpolarization.

Hyperpolarizing receptor potentials which are caused by an increase in membrane K^+ permeability are not unique to the scallop retina. Similar responses occur in *Paramecium* following mechanical stimulation (Naitoh & Eckert, 1973) and in certain *Aplysia* ganglion cells following stimulation with light (Brown & Brown, 1973). The hyperpolarizing receptor potential of photoreceptors in the eye of the clam *Lima* (Mpitsos, 1973; McReynolds, 1976) is also produced by an increase in K^+ permeability (unpublished results, M. C. Cornwall & A. L. F. Gorman). Other likely candidates are the hyperpolarizing receptor potentials found in the eye of the primitive chordate *Salpa* (McReynolds & Gorman, 1975), in the pineal eye of fish (Tabata, Tamura & Niwa, 1975) and the 'off'-receptors in the eye of the cockle *Cardium* (Barber & Land, 1967) and in the siphon of the clams *Mercenaria* (Wiederhold *et al.* 1973) and *Spisula* (Kennedy, 1960). This list, although far from extensive, suggests that sensory stimuli may cause a selective increase in K^+ permeability in a variety of different sensory receptors throughout the animal kingdom.

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