J. Exp. Biol. (1974), **60**, 795–805
With 6 text-figures
Printed in Great Britain

NEURONAL MODULATION OF PHOTORECEPTOR ACTIVITY IN THE BEE MOTH *GALLERIA MELLONELLA*

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(*Received* 19 *October* 1973)

INTRODUCTION

Recent studies on light-adaptation indicate that major changes in receptor sensitivity occur at the receptor-cell level. However, the basic mechanisms underlying these changes in sensitivity have not been elucidated. Most of the photoreceptor cells which show decreases in sensitivity following exposure to light have been in vertebrate preparations (Dowling, 1960; Rushton, 1965; Naka & Rushton, 1968; Werblin, 1971; Dowling & Ripps, 1972; Sillman, Owen & Fernandez, 1973).

Adaptation, however, is quite complex and probably involves a number of different events. Apparent changes in sensitivity could also be due to movement of screening pigments or integration activities in higher-order neurones. Studies which involve adaptation in lepidopteran visual systems have focused on the role that screening pigments play during changes in sensitivity (Bernhard & Ottoson, 1960*a*, *b*, 1964; Bernhard, Höglund & Ottoson, 1963; Höglund, 1963, 1966). In a number of other visual systems, changes in sensitivity following exposure to light appear to involve inhibitory feedback mechanisms. Naka & Kishida (1966) recorded decreased responsiveness of single cells in honeybee drone eye and suggested inhibitory feedback mechanisms within the membrane to explain these reductions in sensitivity. More recently, Duncan & Croghan (1973) have put forward a model which could explain these aspects of adaptation in terms of changes in membrane time constants. Although changes in membrane characteristics could be directly evoked by activity in the photoreceptor cell itself, it is also possible that synaptic interactions could play a part in the process. Synaptic feedback systems on to the receptor cells in the dragonfly ocellus have been described (Dowling & Chappell, 1972). The role this feedback might play in adaptation, however, does not appear to have been considered. Other visual synaptic systems are well known; lateral inhibition has been described in vertebrate eyes (Werblin & Dowling, 1969; Baylor, Fuortes & O'Bryan, 1971), *Limulus* (Hartline & Ratliff, 1956) and in insect eyes (Zettler & Jährvilehto, 1972). A self-inhibitory mechanism has also been described in *Limulus* eccentric cell (Dodge, Shapley & Knight, 1970; Purple & Dodge, 1966).

During the course of previous investigations on the photic response in the bee moth, *Galleria mellonella* (Koopowitz, Stone & Martinez, 1973; Stone & Koopowitz, 1974), we began to suspect that certain changes in sensitivity might be independent of

screening pigment action. In the present paper we report on a fast light-adaptation mechanism which appears to involve inhibitory synapses on the receptor cells in the lepidoteran *Galleria mellonella*.

METHOD

Recording and stimulating methods have been described previously (Koopowitz *et al.* 1973). Adult *Galleria* moths were waxed to a metal plate. The recording electrode was inserted through the cornea into the retina and the indifferent electrode into the abdomen; animals were then allowed to dark-adapt for 1½ h. Stimuli were 10 μsec flashes of white light of intensity 0·501 mJ/cm² at the surface of the eye. Flashes were delivered by a Grass PS 2 physiological photostimulator which was in turn programmed with a Grass S 48 square-wave stimulator.

Stimuli were delivered in pairs and the interstimulus interval (ISI) ranged between 0·2 sec and 10 sec. Although the stimulus intensity was moderately strong, the preparation generally recovered to its dark-adapted value within 20 sec. The time between pairs of flashes was 3·0 min. ISIs to be tested were chosen at random. Recording utilized d.c. amplification. The sweep of the CRO beam was triggered simultaneously with the onset of the light flash and the response was then measured from the trace displayed on a storage CRO. In all of these experiments the recording electrode was positioned on the retinal side of the basement membrane of the eye.

A number of drugs were used. These were made up in insect Ringer (Pringle, 1939) and applied through a hole in the exoskeleton of the animal's head. The sheath surrounding the brain and optic ganglion was torn open to facilitate access of the fluid. Where treatment consisted of Ringer solutions containing drugs, or where the ionic concentrations were changed, the treatment was preceded by application of insect Ringer acting as the control for that particular experiment. When we changed ion concentrations we were careful to add ions which were made up as solutions isotonic to the normal Ringer solution.

For the experiments reported here we have mainly concerned ourselves with the tonic negative transient. The origin of this component is the least controversial and most workers consider it to represent the photoreceptor potential. We measured the amplitude of this potential from the resting level of the eye in the dark to the peak of the slow negative-going wave. As stimuli were generally presented as pairs of flashes we have tended to graph the results as the ratio of the amplitude of the second response (R_2) over the amplitude of the first (R_1). Where the value of R_2/R_1 is 1·0 there is no change in responsiveness. The smaller the ratio, the greater the depression of the second response. All experiments have been repeated five or six times, and unless otherwise stated the results obtained were similar.

RESULTS

Effects of ISI on Response Amplitude

The typical *Galleria* visual response is triphasic (Koopowitz *et al.* 1973) with a positive-going component as well as phasic and tonic negative transients.

The results displayed in Fig. 1 show the relative changes in amplitude to the second flash with varying ISI (interstimulus interval). With increasing ISI's up to 1·0 sec there is a progressive decrease in amplitude of the second response for both negative transients (Fig. 1 B, C). In these preparations recovery was rapid and complete within

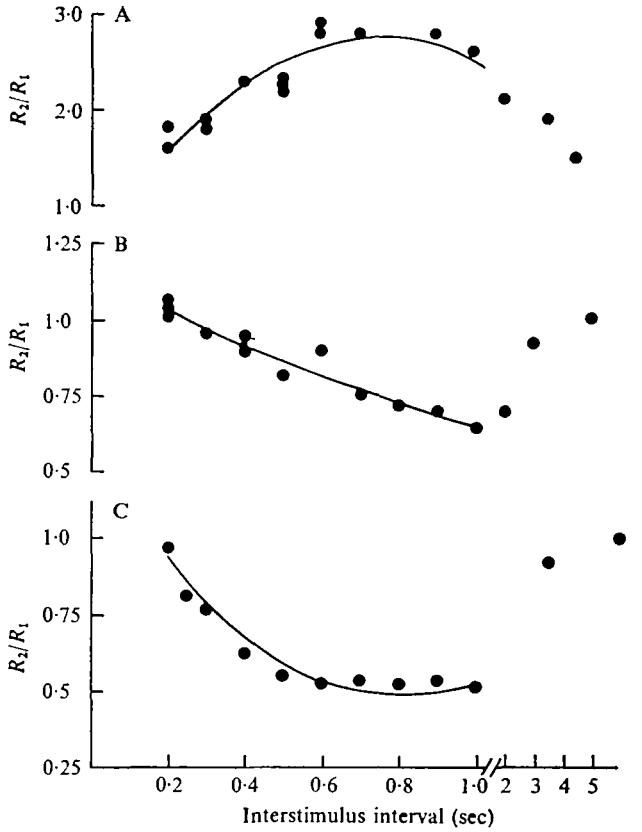

Fig. 1. Effects of light-adaptation on the components of the visual response following paired stimuli given at various intervals. (A) The positive component. (B) The phasic negative transient. (C) The tonic negative transient. In this and all the following figures the ordinates are the amplitudes of the test response divided by the amplitude of the dark-adapted response. Three minutes were allowed between each pair of stimuli. The abscissae are the interstimulus intervals in seconds. Note that the abscissae have two scales.

6·0 sec. The amount of stimulus depression seen tends to vary from preparation to preparation; typical values range from 25 to 50%. The fact that there is a gradual build-up of the effect indicates that the decreased responsiveness is not due to bleaching or lack of photopigment. For the same reasons depression of the second response cannot be due to refractoriness within the photoreceptor membrane.

The positive-going potential (Fig. 1 A) shows facilitation rather than depression. However, as this potential is usually superimposed on the phasic negative transient (Koopowitz *et al.* 1973) the facilitation may be more apparent than real; i.e. reductions in amplitude of the phasic negative component could unmask the positive-going potential during the test flash.

Effect of CO_2 on adaptation

It is possible to partially, or completely lift the depression by a variety of treatments. CO_2 is thought to block ganglionic components of the visual response, and we have found (Fig. 2) that in CO_2-treated preparations the depression is significantly lifted.

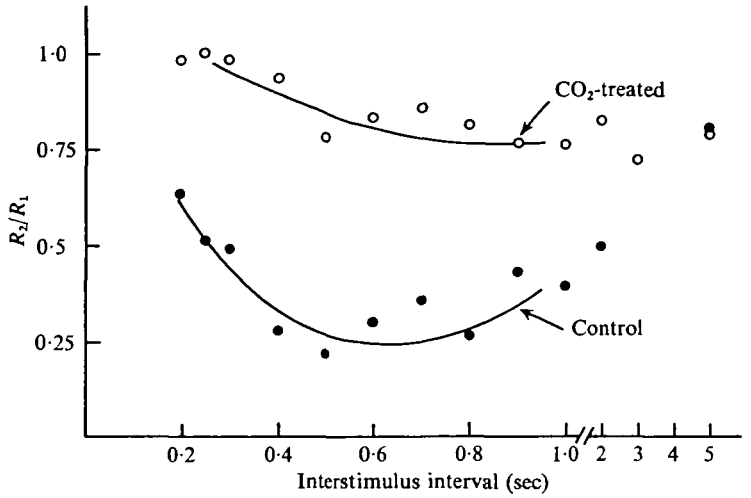

Fig. 2. Changes in amplitude of the tonic negative transient under CO_2 anaesthesia. ●, Results from the untreated preparation. O, Results following the application of CO_2 for 5 min.

In Fig. 2 reduction seen in the second flash was 75 % of its original amplitude at 0·5 sec. This is more than is usually found. With application of CO_2 the maximum depression recorded was only 25 %. In this preparation the absolute magnitude of the response to the initial flash was reduced, e.g the control value was −6·0 mV for the initial flash (R_1) and −2·6 mV for the test flash (R_2) at an ISI of 0·9 sec. After application of CO_2 the amplitude of R_1 was −5·0 mV and R_2 was −3·8 mV for the same ISI. Under the influence of CO_2 the test stimulus response (R_2) was larger even though the amplitude of the initial response was reduced. CO_2 did not always decrease the amplitude of R_1; in fact, in four of seven cases it was either unchanged or increased.

Effects of K^+ on Adaptation

On the assumption that the receptor axons running into the ganglion might have K^+-dependent resting potentials, we attempted to block ganglionic events by depolarizing the axons and ganglion with KCl. This was done by adding KCl, isotonic to Ringer solution, to the ganglion. KCl causes a decrease and eventual loss of the phasic negative component of the visual response. The remaining tonic negative transient also tends to be reduced in amplitude. As can be seen in Fig. 3, the remaining negative transient does not decrease in amplitude in response to the second flash. Trains of up to 15 stimuli with ISI of 0·8 sec did not produce reductions in amplitude of the test flashes.

De-ganglionation and adaptation

An obvious experiment to test the idea of a ganglionic feedback mechanism would be to sever the connectives between the retina and the ganglion. Although this is a fairly easy operation to perform, the axons of the retinula cells themselves are severed, which means that the photoreceptors are injured. Consequently, the majority of preparations become rather 'spiky' and it is difficult to measure response amplitudes very accurately. In three preparations, however, large responses could still be measured from the de-ganglionated eye. In two of these preparations the size of the initial response was

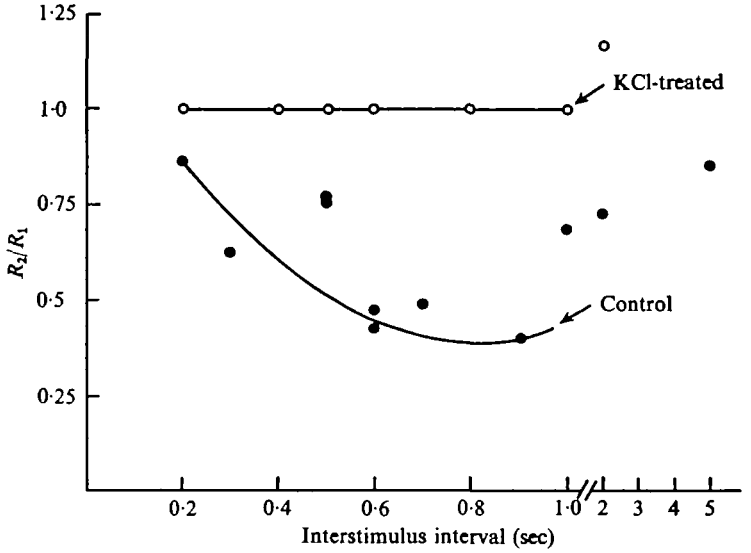

Fig. 3. Effects of KCl on light-adaptation of the tonic negative transient. ●, Results from the preparation with Ringer applied to the ganglion. ○, Results following addition of KCl isotonic to Ringer.

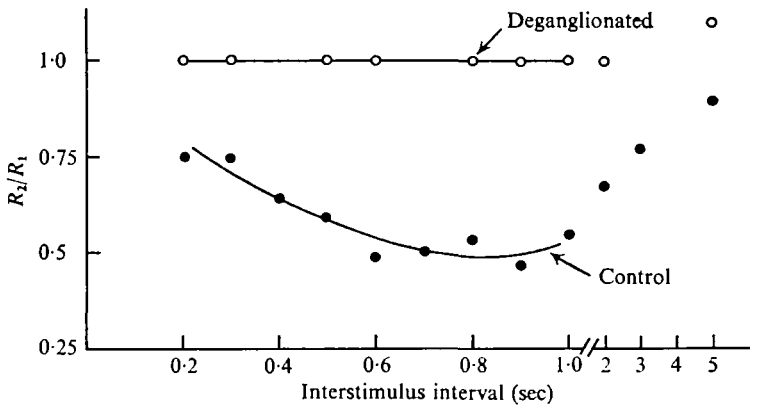

Fig. 4. Effects of de-ganglionation on light-adaptation of the tonic negative potential. ●, Results obtained from the untreated preparation. ○, Results after severing neuronal connexions between the retina and ganglion.

actually greater following the operation. In one preparation the initial response (R_1) increased in amplitude from $-0{\cdot}7$ to $-2{\cdot}7$ mV, while in the second preparation a much smaller increase from $-0{\cdot}73$ to $-1{\cdot}0$ mV was measured. In the third preparation there was a drastic decrease in amplitude from $-1{\cdot}3$ to $-0{\cdot}3$ mV. In all three cases, however, there was no depression of the response to the test flash ($R_2/R_1 = 1{\cdot}0$). Fig. 4 is a representative plot of the data.

Effects of Ca^{2+} and Mg^{2+} on adaptation

Among its multitude of effects Ca^{2+} is thought to promote transmitter release at synapses and neuromuscular junctions (Katz, 1970). Ca^{2+}, isotonic with Ringer, solution applied to the ganglion abolishes the negative-going transients of the visual

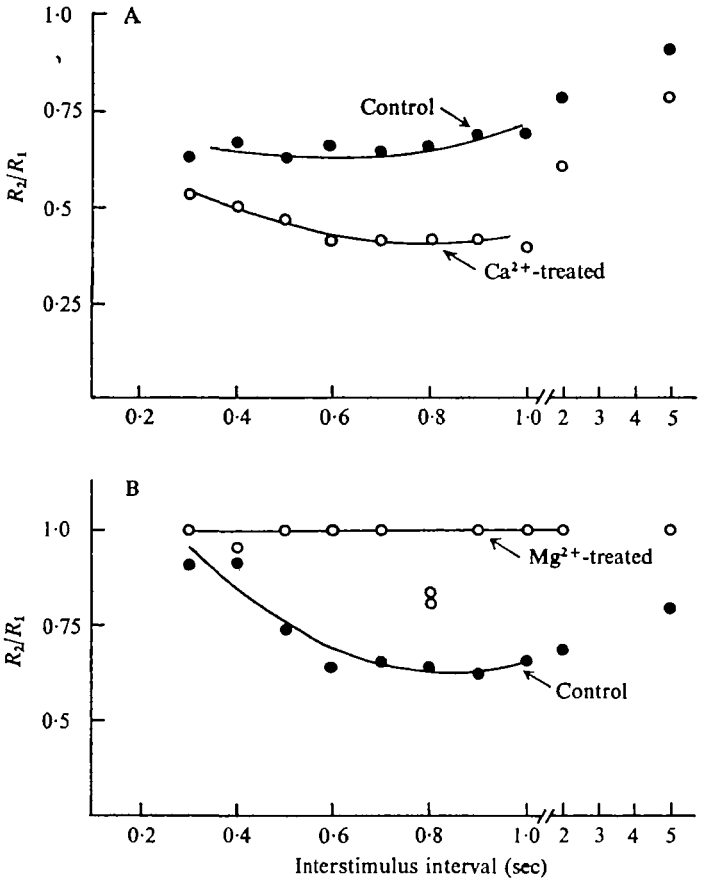

Fig. 5. (A) Increases in light-adaptation of the tonic negative component with increased Ca^{2+} concentrations. ●, Results following application of Ringer to the ganglion. ○, Results after addition of 1 part isotonic $CaCl_2$ to 8 parts Ringer. (B) Effects of Mg^{2+} on light-adaptation of the tonic negative transient. ●, Results from the untreated preparation. ○, Results following application of $MgCl_2$, isotonic to Ringer, to the ganglion.

response. Addition of small quantities of isotonic $CaCl_2$ to Ca^{2+}-free Ringer solution (1 part $CaCl_2$ to 8 parts Ringer) tended to increase the depression. In Fig. 5A the amplitude of the initial response (R_1) was reduced by the Ca^{2+} treatment from about $-3{\cdot}0$ to $-2{\cdot}3$ mV. Although we measured consistent increases in depression, the amplitude of the initial response (R_1) following Ca^{2+} treatments were unpredictable. In some cases there were increases in amplitude while in other cases there were decreases in amplitude of the dark-adapted (R_1) value. Mg^{2+} acts as an antagonist to Ca^{2+} and is often used to block synapses. When Mg^{2+}, isotonic to Ringer solution, was added to the ganglion both negative components were rapidly abolished leaving only the positive-going component. If isotonic MgCl was diluted with Ringer (1 part $MgCl_2$ to 8 parts Ringer) the slow negative component was reduced in amplitude but not abolished. Depression was lifted (Fig. 5B). In this experiment with an ISI of 0·5 sec the initial response of the control (R_1) was $-1{\cdot}35$ mV and the response to the test flash (R_2) was $-1{\cdot}0$ mV. Following treatment with Mg^{2+} the response to both flashes (R_1 and R_2) was $-0{\cdot}5$ mV. In the experiment, displayed in Fig. 5B, there was some

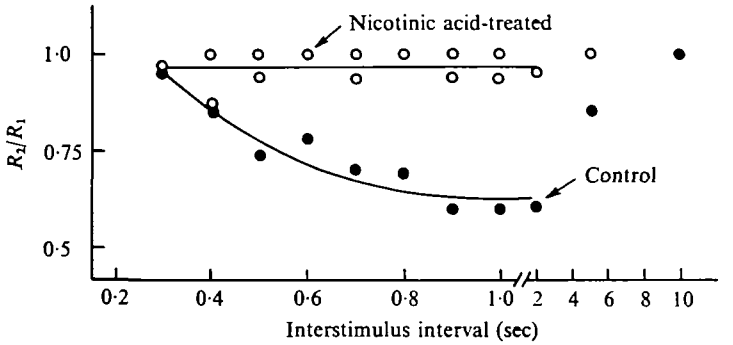

Fig. 6. Effects of nicotinic acid on the amplitude of the tonic negative transient. ●, Results with addition of Ringer to the ganglion. ○, Results following application of 1×10^{-5} M nicotinic acid.

depression at an ISI of 0·8 sec. In the four other preparations depression was only partially lifted for all ISI values, e.g. with an ISI of 0·8 sec, R_2/R_1 ratios for controls: experimentals were 0·6:0·75, 0·525:0·725, 0·44:0·6, 0·4:0·53.

Nicotinic acid and adaptation

When nicotinic acid is applied to the ganglion in concentrations of 1×10^{-5} M in Ringer solution we find that not only is adaptation abolished but the dark-adapted response increases markedly in amplitude. In one experiment the average value for R_1 before drug treatment was $-0{\cdot}86$ mV. After addition of nicotinic acid it had increased to a mean value of $-3{\cdot}87$ mV. At higher concentrations of nicotinic acid these increases in amplitude are not always seen or they may be rather transitory in nature. In Fig. 6, when readings were taken $1\frac{1}{2}$ h after application of the drug, adaptation was not completely abolished. Twenty-five minutes after the initial readings were taken, adaptation no longer occurred. R_2/R_1 values less than 1·0 were obtained prior to this time. Although concentrations of up to 1×10^{-2} M nicotinic acid have been used and these abolish adaptation, the entire visual response is usually lost after about 90 min.

DISCUSSION

Our data can be interpreted to suggest that fast light-adaptation in *Galleria* involves neuronal interactions. This discussion is concerned with the location of the feedback mechanism and its possible mode of action.

Synaptic feedback mechanisms

The visual response as we have recorded it is a mass electrical event recorded extracellularly. Although the recording electrode was placed in the retina, it is probable that ganglionic events are also recorded in the response. We have shown quite clearly that when ganglionic connectives are severed that fast adaptation does not occur. Therefore, these adaptational events are either second-order neurone activities or require first-order axons and perhaps also the integrity of the whole photoreceptor cell. It is possible to rule out a major role played by second-order cells. If adaptation occurred as a decrease in size of the second-order cell response, one would not expect to find increases in absolute amplitude following de-ganglionation or treatment with nicotinic acid.

Many recent studies on adaptation indicate that the first-order photoreceptors themselves are a major site of light-adaptation. Most of these studies have involved a variety of vertebrate photoreceptors (Dowling, 1960; Rushton, 1965; Naka & Rushton, 1968; Werblin, 1971; Dowling & Ripps, 1972; Sillman *et al.* 1973). Intracellular recordings from the photoreceptors of the bee drone (Naka & Kishida, 1966) and of *Aeschna* (Autrum & Kolb, 1972) also demonstrate first-order adaptation. In *Limulus*, Dodge *et al.* (1970) describe self-inhibitory as well as lateral inhibitory events. At the present time it is not known whether the same or similar events underly adaptation in all these cases.

Our data strongly suggests that adaptation involves synaptic events but also occurs in the photoreceptor cells. The evidence that synaptic events are involved is as follows:

(*a*) Mg^{2+} ions are known to block synapses (del Castillo & Engbaek, 1954). When the *Galleria* ganglion was poisoned with Mg^{2+}, adaptation disappeared. (One would expect from our other data that the amplitude of the response with Mg^{2+} poisoning would either remain unchanged or increase in size. The reverse occurred and response was actualiy smaller. We are at a loss to explain this finding.)

(*b*) Ca^{2+} is generally antagonistic to Mg^{2+}, and one might expect Ca^{2+} to depress the response and enhance adaptation, which, in fact, it does. It is also possible, however, that Ca^{2+} plays a direct role in visual adaptation (Lisman & Brown, 1972). Excess Ca^{2+} completely abolishes the negative components, leaving only the positive component. It seems unlikely that this effect could be due to overstimulation of synapses. At lower concentrations the increased depression that we measure could either be synaptic or a direct effect.

(*c*) The best evidence that synaptic mechanisms are involved comes from our experiments with nicotinic acid. Nicotinic acid is known to block synapses (Wiersma & Schalleck, 1948). With its application there is a concomitant increase in response amplitude. The change in amplitude following application of the drug suggests that the normal response may be continuously dampened by some kind of inhibitory event in the normal preparation. Results from the de-ganglionation experiments add weight to this interpretation.

(*d*) CO_2 tends to lift adaptation. We have shown (Stone & Koopowitz, 1974) that in this preparation CO_2 abolishes the phasic negative component leaving the amplitude of the tonic negative component either unchanged or slightly larger. It has been suggested that CO_2 acts as an anaesthetic in insects by blocking synapses (Hoyle, 1960). Abolishing depression with CO_2 could act by interfering with synaptic events, but an interplay between the phasic and tonic negative components of the response could also be involved in the adaptation. Thus, abolishing the phasic potential might also decrease the amount of adaptation. However, this cannot be the sole event during adaptation because adaptation is not abolished completely (Fig. 2) by CO_2.

The presence of synapses on to the retinular cells has not yet been anatomically demonstrated for *Galleria*. Synaptic feedback on to the photoreceptor cells in the dragonfly ocellus has been reported (Dowling & Chappel, 1972). The role that this feedback could play in light-adaptation was not discussed; however, evidence is presented which suggests that the feedback modulates the phasic nature of the visual response in the dragonfly. If feedback synapses exist in *Galleria* one might expect them to be in the lamina ganglionaris. Investigations of the lamina in other lepidoptera have not utilized ultrastructural techniques. Studies with the light microscope (Straus-

feld & Blest, 1970) reveal a number of receptor axon configurations, and it is possible that some of these could involve the kinds of synapses that we hypothesize.

Membrane events during light-adaptation

One of the problems in unravelling adaptation is that there are probably a number of completely isolated events involved in the process of light-adaptation. Bleaching could reduce the quantity of photopigment, there could be migration of screening pigment, neuronal events could occur farther down the system and a variety of membrane events might take place within the receptor cells themselves. We surmise that the adaptation we record is due to a synaptic event. It is difficult to model intracellular events from extracellularly derived data. In our experiments the receptor potential appears to return to the pre-stimulus level while the depression caused by light-adaptation is still present. Until we get into the cells we will not know what the corresponding intracellular changes might be. In some vertebrate photoreceptors one can measure changes in membrane resistance without concomitant changes in membrane potential (Yoshikami & Hagins, 1973). We would like to put forward the following tentative model for fast adaptation in *Galleria*. A light stimulus would act in triggering a synaptic feedback loop on to the receptor axons, probably in the lamina. This would cause a maintained conductance change in the axon's terminal of the photoreceptor cell. If the time and space constants of the axonal membrane are very large and the axoplasmic resistance low, one might expect the synaptic induced currents to be shunted up the axon cylinder into the body of the receptor cell and through the receptor membrane. At present we have absolutely no concrete data on the membrane characteristics of the system. Indeed, even if intracellular measurements could be made it would be difficult to pick out a high-resistance axon from low-resistance membranes at the synapse and cell body.

In second-order cells of the fly *Calliphora*, Zettler & Järvilehto (1971) have found what appears to be non-decremental graded conduction. In other insect species it is apparent that at least the first-order and second-order visual cells also support graded activity. Whether or not these potentials are also transmitted in a non-decremental fashion is unclear. Zettler and Järvilehto have calculated some membrane characteristics for their preparation. We estimate that in *Galleria* the distance from the basement membrane of the retina to the lamina averages 100 μm and the lamina is about 44 μm thick. This does not take into account shrinkage due to fixation, etc. Strausfeld & Blest (1970) found that some lepidopterans' receptor cell axons also travelled to the medulla; this could increase the length of the axons in *Galleria* by a minimum of 77 μm. Zettler & Järvilehto (1971) calculated a space constant for second-order *Calliphora* visual cells of 110 μm. If the same space constants applied to *Galleria* then passive electrotonic antidromic conduction could probably not account for the changes in sensitivity recorded. In their calculations Zettler and Järvilehto considered that their measurements of membrane resistance reflected a uniform resistance throughout the cell and they assumed that there was not a low-resistance membrane at the synapse. In our model we hypothesize the reverse and assume that the axonal membrane resistance would be much higher than at the synapse and cell body.

If our supposition of inhibitory feedback is correct, then the conductance change at the synapse would have to be conducted antidromically from the lamina up to the

receptor cell soma. Intracellular measurements could not distinguish variability in resistance of different parts of a cell unless the parts could somehow be isolated from each other. Until this can be done we will not be able to test our hypothesis.

SUMMARY

1. Pairs of light stimuli given at varying intervals causes changes in fast-adaptation characteristics in the bee moth *Galleria mellonella*. With increasing intervals between the stimuli of up to 1·0 sec there is a decrease in the amplitude of the visual response to the second stimulus.

2. This depression of the response can be affected by a variety of agents which alter synaptic activity. Application of compounds such as Mg^{2+} and nicotinic acid, which block synapses, to the optic ganglion lift depression.

3. K^+ and CO_2 applied to the optic ganglion also abolish adaptation.

4. Severing neuronal connexions between the retina and ganglion also lifts the depression. De-ganglionation and nicotinic acid also result in increases in absolute amplitude of the response.

5. If Ca^{2+} is added to the Ringer bathing the ganglion, adaptation becomes more marked.

6. These results suggest that the decreases in sensitivity following a brief light stimulus are mediated by synaptic feedback onto the receptor cells.

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