Effect of Neurotransmitters on Movement of Screening Pigment in Insect Superposition Eyes

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Z. Naturforsch. **44c**, 992–998 (1989); received August 17, 1989

Compound Eye, Pigment Migration, Adrenaline, Octopamine, Noradrenaline

The light-induced expansion of the screening pigment in the compound eyes of the sphingid moth *Deilephila* and the owlfly *Ascalaphus* is counteracted by local application of noradrenaline or octopamine. At a critical concentration, similar for both drugs, the expanding effect caused by a light stimulus is completely neutralized, and at higher drug concentrations light stimulation induces a contraction of the screening pigment. The contracting effect of noradrenaline and octopamine is counteracted by adrenaline. None of the other putative neuroactive substances tested (acetylcholine, GABA, histamine, melatonin, serotonin and taurine) has a comparable effect on light-induced pigment movement. It is hypothesized that pigment migration is controlled by a system of antagonistically acting catecholamines, similar to the noradrenaline/adrenaline system present in vertebrates.

Introduction

The light flux reaching the photoreceptors in the superposition eye of many insects, is regulated by movements of screening pigment granules [1–5]. The largest movements occur within specialized screening pigment cells. In the dark-adapted eye the pigment is contracted peripherally between the crystalline cones, and light from a point source entering many facets is focused on the rhabdomes of an individual ommatidium. Light not absorbed by the visual pigment is reflected by the tracheoles surrounding the rhabdome, and can be observed as an "eye glow" or "pupil" [6]. If the eye is exposed to adapting lights the pigment granules disperse, whereby light passing obliquely in the eye is increasingly absorbed. Less light reaches the rhabdomeres [1], and simultaneously the eye glow gradually disappears. Changes in glow, *i.e.* in the intensity of the reflected light, thus can be used as an index of pigment migration.

The spectral sensitivity of the pigment migration in the moth *Deilephila* [7, 8], but not in the related moth *Manduca* [9], is markedly different from that of the photoreceptor layer. Furthermore, in *Deilephila* pigment movement can be elicited in preparations consisting of only the screening pigment cells, dioptric structures and small, inactive visual cell rests [7, 8]. It is therefore possible that in *Deilephila* a photopigment triggering pigment expansion is located within cells distal to the visual cells, *e.g.* within the screening pigment cells or the cone cells. By varying the angle of incidence of light stimulating the compound eye of the sphingid moth *Theretra,* Land [10] confirmed the existence of a mechanism located near the cornea that triggers pigment migration. Nilsson [5] using a similar optical technique concluded that in some moth species there is a general retinal control of pigment migration as well as a local, distal control. Pigment movements may thus be regulated by at least two morphologically separated cell types.

If pigment expansion is not exclusively triggered by a photopigment located within the screening pigment cells themselves, signal transduction from the other cell types controlling pigment migration can be accomplished either by electric coupling between triggering cell and pigment cell, or by modulation of the extracellular composition of divalent cations, or by the release of neurohumoral substance. The observation that in *Deilephila* excitation of the green sensitive visual cells has little influence on pigment expansion [7, 8] makes it unlikely that in this species pigment expansion is controlled by modulation of the extracellular ion composition. Electric coupling is also unlikely, since pigment movement starts at least 10 sec after the completion of a flash-induced photo-

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Verlag der Zeitschrift für Naturforschung, D-7400 Tübingen
0341–0382/89/1100–0992 $ 01.30/0

receptor response [8]. A neurohumoral control seems possible, since the movement of the screening pigment in response to light stimuli can be modulated by locally applied catecholamines [11].

The present study was made firstly, to quantitatively test the influence of some neurotransmitters and neurohumoral substances on screening pigment movement, and secondly, to obtain an indication whether an effect of such substances on pigment movement is a general phenomenon in superposition insect eyes.

Materials and Methods

The experimental technique has been described in detail [8] and is summarized here. The measurements were made on the compound eye of the moth *Deilephila elpenor* (Lepidoptera: Sphingidae) or the dorsal compound eye in the owlfly *Ascalaphus macaronius* (Neuroptera: Ascalaphidae). The effect on the pigment migration of the following substances (up to 10 mM solutions) was tested: acetylcholine, gamma-amino-butyric acid (GABA), histamine, melatonin, serotonin and taurine.

Adult *Deilephilae* were used 3 to 5 days after emergence. The animals were kept in darkness for at least 12 h before the experiment. Most experiments were made on intact animals secured to a block of Styropor. A maximal glow confirmed that the screening pigment was contracted [1]. A contact gel (Siemens; normally used for electrocardiography) was applied to the cornea. In most experiments a drug had been added to the gel. In some experiments 2 drugs had been added. Due to the contact gel, ions and the drugs could diffuse through the cornea. Light reflected by the tracheoles was recorded using an equipment based on a Leitz Ortoplan fluorescence microscope. Pigment movement was induced by a monochromatic blue stimulus (λ_{max} = 430 nm; xenon light source XBO 1600 W; monochromator Bauer BM, bandwidth 2 nm; intensity = 3.4×10^{15} photons $\times$ cm$^{-2} \times$ s^{-1}) usually causing a reduction in reflected light flux to about 0.5. The change in eye glow (number of photons $\times$ s^{-1}) reflected from the tracheoles through 30 to 40 corneal facets was recorded microphotometrically during at least 8 min by projecting onto the eye a linearly polarized red light ($\lambda > 630$ nm; edge filter Schott RG 630, 3 mm; tungsten lamp, 6 V, 5 A) itself causing no reduction in eye glow. The light reflected by the tracheoles passed through a second polarization filter with its polarization plane at right angles to the first. The highly polarized light reflected by the cornea was thereby eliminated, and only the unpolarized light reflected by the tracheoles was measured.

The *Ascalaphi* were caught wild as adults and used within 3 weeks after capture. The animals were kept in darkness for at least 12 h before the experiment. Drugs were added to the corneal gel. Measurements on intact eyes were made as on *Deilephila* with the exception that pigment movement was induced either by an ultra-violet stimulus (λ_{max} = 370 nm; half bandwidth about 80 nm; mercury lamp source HBO 200 W; filter Schott UG 11, intensity = 2.4×10^{16} photons $\times$ cm$^{-2} \times$ s^{-1}) or a blue stimulus (λ_{max} = 445 nm; half bandwidth about 50 nm; mercury lamp source HBO 200 W; filters Schott BG 12, GG 435 and KG 1; intensity = 3×10^{16} photons $\times$ cm$^{-2} \times$ s^{-1}).

Most of the experiments on *Ascalaphus* were made on eye preparations, similar to those earlier used on *Deilephila* [8], consisting of screening pigment cells, dioptric cells and small visual cell rests only. The preparation was made under dim red light by a tangential section cut through the cornea of a dark-adapted eye distally to the rhabdomes. It was transferred onto an agar gel (thickness 2 mm) containing KCl 200 mM, and glucose 50 mM, and kept in a glass chamber with a cover glass. Drugs were added to the agar gel. Pigment movement was induced either by the ultra-violet or by the blue stimulus also used on intact *Ascalaphus* eyes. The change in the transmission of red light ($\lambda > 630$ nm; edge filter Schott RG 630, 3 mm; tungsten lamp, 6 V, 5 A) through the preparation was recorded using the same microscopic equipment as that used for reflectometry. The preparation was inspected before the recordings (using dim red light) and after the recordings (using bright white light) to confirm that no tracheoles were present. (The tracheoles surround the rhabdomes. Absence of tracheoles thus can be used as a criterion that at least the major part of the rhabdomes is absent.)

Results

Measurements on intact eyes in Deilephila

Noradrenaline. The amplitude of screening pigment movement (expressed as change in reflection) after a constant light stimulus varied with the con-

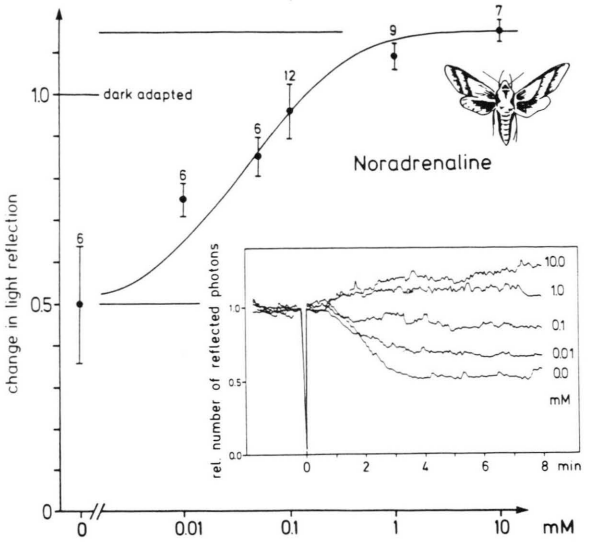

Fig. 1. Effect of noradrenaline concentration on movement of screening pigment in intact compound eye of moth *Deilephila*. *Graph:* Change in intensity of light reflected from tracheoles with concentration of noradrenaline (0.01, 0.1, 1.0, 10.0 mM) in gel applied on cornea. Pigment movement, causing change in reflection, elicited by stimulation of dark adapted eye by blue light ($\lambda_{max} = 430$ nm) of constant intensity (3.4×10^{15} photons $\times$ cm$^{-2} \times$ s^{-1}) and duration (4 s) and measured by $\lambda > 630$ nm. Stimulation in absence of noradrenaline (0.0 mM) reduced reflection to about 0.5. Vertical bars are SD, and adjacent figures show number of experiments. *Inset:* Time course of change in intensity of reflected light (arbitrary units) after same light exposure as in graph. Stimulation indicated by artifact at time 0 min. Concentration of noradrenaline (0.0 to 10.0 mM) indicated at right of traces. Note in graph that empirical data (dots) could be approximated by first order function (continuous line), and that concentration of noradrenaline just canceling pigment movement in response to light stimulus was about 0.2 mM, and in inset that both pigment expansion and contraction started after latency of about 20 to 30 s.

centration of noradrenaline in the contact gel applied on the cornea (Fig. 1, inset). In the absence of noradrenaline (0.0 mM), the 4 s light stimulus within 3–4 min caused a decrease in reflection to about 0.5, indicating a specific, moderate expansion of the screening pigment layer. The reflection then gradually increased to reach the original dark adapted value within 20–30 min (not shown in Fig. 1). The expansion was reduced when noradrenaline (0.01 mM or 0.1 mM) had been added to the gel. A comparison of the traces shows that a critical concentration between 0.1 and 1.0 mM should have completely prevented a light induced movement of the pigment granules. At higher concentrations the light stimulus caused a reversed granule movement (*i.e.* an additional contraction of the pigment layer, recorded as an increase in reflection). Fig. 1 also shows that pigment expansion as well as contraction started after a latency of about 20 to 30 s. The variation with drug concentration in direction and amplitude of the pigment movement recorded 3–4 min after the light stimulus (Fig. 1, graph) could be approximated by a first order function. The light induced pigment expansion decreased with increase in noradrenaline concentration up to about 0.2 mM. Above this critical concentration the light stimulus caused a contraction of the pigment layer.

Octopamine. Observations analogous to those made after application of noradrenaline were made when octopamine had been added to the corneal gel (Fig. 2). Similarly to noradrenaline, raising the con-

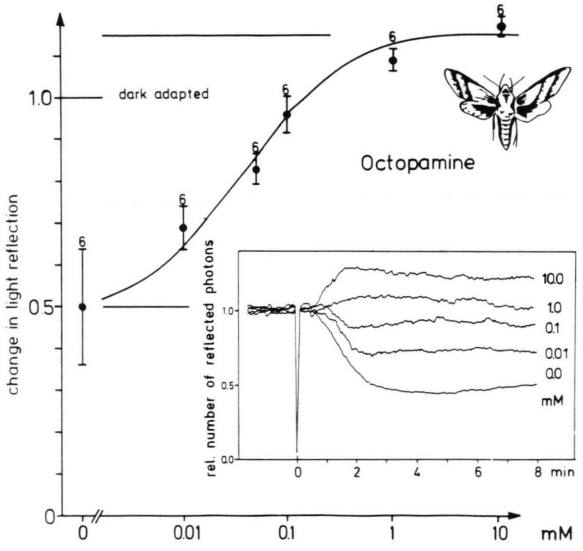

Fig. 2. Effect of octopamine concentration on movement of screening pigment in intact compound eye of moth *Deilephila*. Graph shows change in intensity of light reflected from tracheoles with concentration of octopamine (0.01, 0.1, 1.0, 10.0 mM) in gel applied on cornea. Light stimulus and drug concentrations same as in Fig. 1. Stimulation in absence of octopamine (0.0 mM) reduced reflection to about 0.5. Vertical bars are SD, and adjacent figures show number of experiments. *Inset:* Time course of change in intensity of reflected light (arbitrary units) after same light exposure as in graph. Stimulation indicated by artifact at time 0 min. Concentration of octopamine (0.0 to 10.0 mM) indicated at right of traces. Note in graph that empirical data (dots) could be approximated by first order function (continuous line), and that concentration of octopamine just canceling pigment movement in response to light stimulus was about 0.2 mM, and in inset that both pigment expansion and contraction started after latency of about 20 to 30 s.

centration of octopamine to 0.1 mM caused a reduction in the amplitude of the light induced pigment expansion, and a further rise in octopamine concentration induced a considerable additional pigment contraction. The relation between drug concentration and response amplitude using octopamine (Fig. 2) was almost identical to that using noradrenaline (Fig. 1). A critical concentration of both drugs about 0.2 mM completely prevented pigment movement.

Fig. 3. Effect of mixture of octopamine and adrenaline on movement of screening pigment in intact compound eye of moth *Deilephila*. Traces show time course of pigment movement, recorded as change in relative intensity of light reflected from tracheoles, after constant light stimulus (same as in Fig. 1). Trace 1: octopamine (1.0 mM). Trace 2: mixture of octopamine (1.0 mM) and adrenaline (1.0 mM). Trace 3: mixture replaced by octopamine (1.0 mM) alone. Note that addition of adrenaline to octopamine (trace 2) led to expansion of pigment layer, and that this reaction was canceled by octopamine alone (trace 3).

Adrenaline. An antagonistic action of adrenaline and octopamine is indicated by the recordings illustrated in Fig. 3. As in the experiment seen in Fig. 2, after addition of 1.0 mM octopamine the light stimulus caused the pigment layer to contract slightly. When 1.0 mM adrenaline together with 1.0 mM octopamine had been applied, the stimulus caused a moderate pigment expansion, similar to that seen when no drug had been added. Again applying 1.0 mM octopamine only, caused the pigment granules to remain almost immobile after the light stimulus. A similar result was obtained using a mixture of adrenaline and noradrenaline.

Acetylcholine, GABA, histamine, melatonin, serotonin and taurine. No effect of these substances on the light induced pigment movement was detected. Addition of the substance up to 10.0 mM to the corneal gel did not, or only very slightly, affect the time course of the movement. The contracting effect of octopamine on light induced pigment movement (Fig. 2) was also not clearly altered when this substance was mixed with acetylcholine, melatonin or serotonin.

Measurements on intact eyes in Ascalaphus

To obtain an indication whether an influence of catecholamines on granule movement in screening pigment cells is a general phenomenon in insect superposition eyes, the time course of screening pigment movement, and the effect of noradrenaline and octopamine, were tested on an ancient insect species, the neuropter *Ascalaphus macaronius*. This species has a superposition compound eye which is divided into two parts. The photoreceptors in the dorsal eye are selectively sensitive to ultra-violet light [12]. Fig. 4 shows that in *Ascalaphus*, as in *Deilephila* [8], the reflectance of the retina in the intact eye temporarily decreased after exposure to an ultra-violet light stimulus, indicating an expansion of the screening pigment. As in *Deilephila*, also blue stimuli elicited a decrease in reflectance, in spite of the visual cells in the dorsal *Ascalaphus* eye responding to ultra-violet light only. The amplitude of the reflectance change increased with the duration of the light stimulus (Fig. 4). The time course of the reflectance

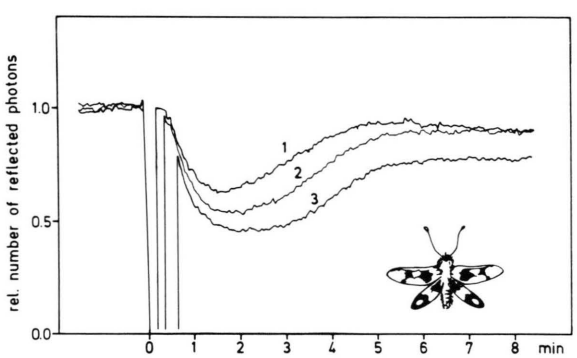

Fig. 4. Time course of pigment movement in intact dorsal superposition eye of neuropter *Ascalaphus*. Traces show change in intensity (arbitrary units) of light reflected from tracheoles. Pigment movement, causing change in reflection, elicited by stimulation of dark adapted eye by ultra-violet light ($\lambda_{max} = 370$ nm, filter Schott UG 11) of constant intensity (2.4×10^{16} photons $\times$ cm$^{-2} \times$ s^{-1}) and measured by $\lambda > 630$ nm. Light stimulation indicated by artifact. Stimulus duration 10 s (trace 1), 20 s (trace 2) or 40 s (trace 3). Note that decrease in reflection, indicating expansion of screening pigment, increased with stimulus duration, and that minimal reflection was reached about 2 min after onset of all stimuli.

change (decrease as well as ensuing increase) in *Ascalaphus*, which is active in daylight, was somewhat faster than in *Deilephila*, which is active in dim light, and the amplitude of the reflectance change was somewhat smaller than in *Deilephila* [8]. Minimal reflectance was reached 1.5 to 2 min after onset of the light stimulus. Application of noradrenaline or octopamine to the intact eye affected the light induced reflectance change similarly to *Deilephila*. The effect of the drugs on intact eyes was however, more difficult to determine quantitatively in *Ascalaphus* than in *Deilephila*. It was more easily recorded on eye preparations containing distal cells only.

Measurements on distal cells in Ascalaphus

In *Ascalaphus*, as in *Deilephila* [7, 8], ultra-violet and blue light stimuli elicited a transient decrease in transmission through preparations consisting of screening pigment cells, dioptric cells and small inactive visual cell rests only, indicating that the light stimulus caused a transient expansion of the screening pigment (Fig. 5, trace 1). As in the intact eye, minimal transmission was reached about 2 min after the onset of the light stimulus. In the presence of adrenaline the same light stimulation led to a much larger decrease in transmission. After application of noradrenaline (10.0 mM), the light stimulus triggered an increase in transmission that disappeared within a few min (Fig. 5, trace 2), indicating a transient contraction of the screening pigment. The time course of this reversed light reaction was faster in *Ascalaphus* than in the corresponding preparation made from *Deilephila* eyes. A similar reaction of higher amplitude was seen after application of octopamine (Fig. 5, trace 3).

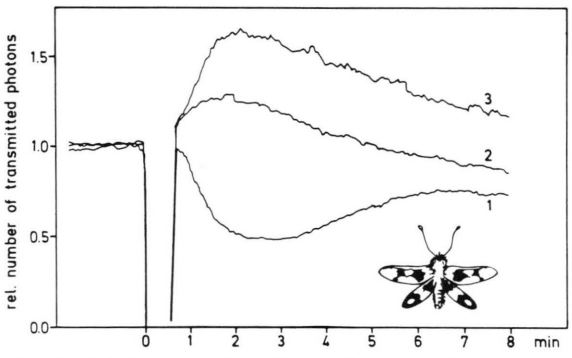

Fig. 5. Effect of noradrenaline and octopamine on movement of screening pigment in preparation containing only distal cells from dorsal compound eye of neuropter *Ascalaphus*. Traces show change in light transmission (arbitrary units) through preparation. Change in transmission, indicating pigment movement, elicited by blue light ($\lambda_{max} = 445$ nm; filters Schott BG 12, GG 435 und KG 1) of constant intensity (3×10^{16} photons $\times$ cm$^{-2} \times$ s^{-1}) during 30 s. Light stimulation indicated by artifact. Drug (10.0 mM) applied to agar gel on proximal side of preparation. Trace 1: untreated preparation. Trace 2: preparation treated by noradrenaline. Trace 3: preparation treated by octopamine. Note that in untreated preparation light stimulus caused decrease in transmission, indicating expansion of screening pigment, while in drug treated preparation transmission increased, indicating contraction of pigment, and that octopamine was slightly more effective than noradrenaline.

Discussion

In some moth species, pigment expansion is triggered by a photopigment located distal to the visual cells [5, 7, 8, 10]. Two experimental results of the present study demonstrate the presence of a distally located trigger also in the dorsal superposition eye of the neuropter *Ascalaphus*. Firstly, the visual cells in this eye are sensitive to ultra-violet light only [12], while pigment expansion can be elicited by blue light (Fig. 5) as well as by ultra-violet light (Fig. 4). In *Ascalaphus*, as in *Deilephila* [7, 8], the spectral sensitivity of pigment expansion thus markedly differs from that of the rhabdomes, and in both species expansion is elicited by ultra-violet and by blue light. Secondly, in both species pigment expansion is elicited in preparations consisting of screening pigment cells and dioptric structures but no active visual cells (Fig. 5). It thus seems that pigment expansion in *Ascalaphus*, similarly to *Deilephila*, is triggered by an ultra-violet-blue sensitive photopigment that is located distally to the photoreceptors near the crystalline cones. (This conclusion obviously does not exclude the existence of another trigger located more proximally.) The pigment movement in *Ascalaphus*, which is active in bright sunshine, is somewhat faster, and less extended, than in *Deilephila*, which is active in dim ambient light. The pigment movements in *Ascalaphus* probably permit the eye to adapt to rapid changes in ambient bright light, *e.g.* due to clouds. Not only the spectral sensitivity of the pigment expansion, and the location of the mechanism triggering pigment expansion, but also the effect of some neuroactive substances is similar in *Ascalaphus* and *Deilephila*.

The substances tested in the present study (with the exception of adrenaline and melatonin) are puta-

tive neurotransmitters, neuromodulators or neurohumoral agents in the visual system of insects (reviews in [13] and [14]). The first indication that pigment migration in insects may be controlled by one of these substances was the observation [11] that noradrenaline applied locally in the compound eye of *Deilephila* reverses the reaction of the screening pigment granules to light stimulation. There is good evidence that in other arthropod eyes the related catecholamine octopamine modulates pigment movement. The sensitivity of the ventral and lateral eyes of *Limulus* is probably controlled by efferent octopaminergic neurons [15], and in scorpions the circadian release of octopamine from neurons efferent to the retina causes the screening pigment to move away from the rhabdomeres [16], thereby increasing the sensitivity of the eye during the night.

The present study showed that in *Deilephila* the reaction of the screening pigment to a light stimulus after application of octopamine is very similar to that after application of noradrenaline. None of these drugs affects the pigment position in the dark adapted eye, while both substances counteract light induced pigment expansion. This counteracting effect is strongly dose dependent (Fig. 1 and 2). At a critical concentration, which is about the same for both drugs, the pigment expanding effect of the light stimulus is completely neutralized, and at higher drug concentrations the light stimulus induces a contraction of the screening pigment. – The drug concentrations used in the present study may seem unphysiologically high. The corneal diffusion barrier impedes however, the supply of drug to the effector cell, and due to enzymatic break down the concentration of a physiologically acting substance is continuously reduced.

The similar action of octopamine and noradrenaline in *Deilephila* shows that in this species the OH-group in position 3 on the benzene ring (in noradrenaline) is of little importance for the regulation of pigment position. The present results thus give no indication whether in *Deilephila* octopamine or noradrenaline is more likely to participate in the physiological control of pigment migration. Due to the presence in arthropod eyes of efferent octopaminergic neurons probably affecting pigment position [15, 16], octopamine seems however, to be the more probable candidate for physiological action in *Deilephila*.

The effect of noradrenaline and octopamine on the light induced pigment expansion in *Ascalaphus* (Fig. 4 and 5) is similar to that in *Deilephila*. In *Ascalaphus* the pigment is however, somewhat more sensitive to octopamine than to noradrenaline. This result is an additional indication that octopamine is more likely than noradrenaline to be a physiologically active substance.

None of the other substances tested (acetylcholine, GABA, histamine, melatonin, serotonin and taurine) has an effect similar to that of noradrenaline and octopamine. Adrenaline has the opposite effect. In the presence of this substance the light induced expansion of the screening pigment exceeds that caused by light alone [11]. The synergistic effect between light and adrenaline seems to be specific for this substance, since no other tested substance has a similar effect. Octopamine and noradrenaline counteract the dispersing effect of adrenaline. When equal concentrations of octopamine and adrenaline are applied an almost normal light response is elicited (Fig. 3).

The present observations thus provide further evidence for the hypothesis [11] that light induced pigment movements are modulated by antagonistically acting catecholamines. Adrenaline has not been demonstrated in insect visual systems [13, 14]. In analogy with the noradrenaline/adrenaline system in vertebrates, one can therefore postulate that insects have a system of antagonistically acting catecholamines based on octopamine and, possibly, the methylated form of this substance. If so, octopamine presumably couples to α-receptors on the pigment cells, or triggering cells, whereas the methylated, adrenaline like, substance probably couples to β-receptors. The position of the screening pigment granules in insect eyes would then be influenced by a balance between α- and β-receptor activation. The hypothetical action of catecholamines thus is by α- and β-receptor activation to modulate the light sensitivity of the screening pigment cells, or of the triggering cells, and thereby to modulate the extent of pigment translocation.

Acknowledgements

The research reported in this paper was supported by Deutsche Forschungsgemeinschaft, "Konzell 5", and by Karolinska Institutets Fonder.

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