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CHOLINERGIC AND MONOAMINERGIC MECHANISMS ASSOCIATED WITH CONTROL OF BIOLUMINESCENCE IN THE CTENOPHORE *MNEMIOPSIS LEIDYI*

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SUMMARY

1. The effects of cholinergic and monoaminergic drugs and blocking agents on luminescence responses of the comb-jelly *Mnemiopsis leidyi* were investigated, using isolated strips of meridional cells.

2. Catecholamines elicited dose-dependent flash activity and adrenalin was the most patent. The adrenalin response was abolished by propranolol ($0{\cdot}1$ mmol l^{-1}), but not phentolamine. Reserpine ($0{\cdot}1$ mmol l^{-1}) suppressed the flash response to electrical stimulation without affecting the adrenalin response.

3. Acetylcholine (ACh) elicited flash activity which was propagated along the meridional canals. Eserine ($0{\cdot}01$ mmol l^{-1}) potentiated the flash response to either ACh or electrical stimulation.

4. Tubocurarine reduced or abolished responses to either ACh or electrical stimulation. Atropine elicited intense flash activity and potentiated the response to electrical stimulation, but failed to block the ACh response.

5. Prolonged exposure of meridional canals to serotonin (5-HT) depressed or abolished flash responses to ACh, adrenalin and electrical stimulation.

6. The ACh flash response was abolished by propranolol but the response to adrenalin was not altered by tubocurarine. It is concluded that nicotinic cholinergic and beta-adrenergic mechanisms are interrelated and indirectly involved in excitation of luminescence in *Mnemiopsis*.

INTRODUCTION

Luminescence in the comb-jelly *Mnemiopsis* originates in the endodermal meridional canals underlying the eight ciliary comb plate rows (Moore, 1924; Harvey, 1952; Chang, 1954; Freeman & Reynolds, 1973). The photocytes are localized on the testis side of the gastrodermal wall in this hermaphroditic species, and are distributed throughout the entire length of the canals except for interruptions where the comb plates are inserted (Freeman & Reynolds, 1973). This constitutes the cellular basis of the luminescence flashes elicited by electrical or mechanical stimulation, a response

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which is propagated along the canals and was postulated to involve a nerve net (Moore, 1924; Harvey, 1952; Chang, 1954).

More recent investigations by Labas (1977*a*,*b*) provided evidence that bioluminescence, muscular and ciliary (locomotory) activities are coordinated in the ctenophore *Bolinopsis*. Electrical stimulation elicited luminescence flashes and contractile events while simultaneously causing ciliary arrest (Labas, 1977*a*). All three responses are depressed by increasing Mg^{2+} and decreasing Ca^{2+} in the medium (Labas, 1977*b*), thus supporting the involvement of a neural substrate for coordination. Neuronal elements fulfilling the morphological criteria of organized nerve nets have been described in the ectoderm and mesogloea of several ctenophore species (Hernandez-Nicaise, 1973*a*,*b*).

Pharmacological investigations using putative neurotransmitters may provide additional evidence that luminescence in ctenophores is a neurally controlled event. This approach may also provide insight into the poorly understood neurotransmitter mechanisms of primitive metazoans such as the coelenterates (see review by Martin & Spencer, 1983). An earlier attempt using this approach has led to the demonstration of monoaminergic transmission mechanisms in the anthozoan *Renilla* (Anctil, Boulay & LaRivière, 1982). In ctenophores, Chase (1941) briefly mentioned that luminescence in *Mnemiopsis* was sensitized by eserine and acetylcholine. Similarly, an electrophysiologically identified conduction system in *Pleurobrachia*, associated with an increase in ciliary beat frequency, was sensitized by eserine and blocked by curare (Satterlie, 1978). The present pharmacological study was undertaken in an attempt to elucidate the neurochemical control mechanisms subserving luminescence in *Mnemiopsis*. Potent and selective effects of cholinergic and monoaminergic drugs on luminescence are reported in this species.

MATERIALS AND METHODS

Specimens of *Mnemiopsis leidyi*, measuring 4·5–8·0 cm in length (long axis), were collected in Woods Hole, Massachusetts and maintained for several days in the laboratory in circulating sea water. All experiments were carried out at room temperature (20–23 °C).

Segments of comb plate rows, including 4–8 comb plates with underlying meridional canals, were excised under a dissecting microscope. To examine the effects of drugs on luminescence, excised segments were placed in filtered sea water in assay vials of 0·5 ml capacity to fit the light-tight chamber of an Aminco Chem-Glow photometer. At least 5 min was allowed to elapse for the preparation to dark adapt (see Anctil & Shimomura, 1984) before onset of experiments, as *Mnemiopsis* luminescence is readily photo-inhibited (Harvey, 1952; Ward & Seliger, 1976; Anctil & Shimomura, 1984).

Each drug was dissolved in filtered sea water as a stock solution and diluted to a concentration such that its injection into the assay vial, whose sea water volume was known, produced the final concentration desired. The luminescence output of the photometer was recorded on a Pharmacia chart recorder and its stability checked with a solution containing a [^{14}C]beta source (activity: 1 μCi). The beta source was sealed inside a vial identical to those used for assays. Sea water alone was also injected into the

assay vial prior to the drug in each experiment to rule out effects unrelated to drug activity. The amount of sea water injected was then removed from the vial before drug delivery.

Concentration-response relationships were examined by adding the lowest test concentration of the drug first, and testing successively higher concentrations in a cumulative fashion. Putative blocking agents were added to the preparations at least 5 min prior to testing the drug under study. The amplitude of individual flashes was measured in arbitrary units by reference to the stable light source. Since the responses appeared as flashes of relatively fixed duration (463 ± 14 ms, $N = 10$), the amplitudes of consecutive flashes were integrated by summation over a fixed time scale for quantitative assessments of drug effects. This time scale usually comprised the first 5 min of flash activity of a drug-induced response (Fig. 1).

To examine the effects of drugs on the luminescence response to electrical stimulation, segments of comb plate rows were placed in a Lucite chamber and immobilized over a Sylgard-coated bottom with two platinum electrodes. The latter were connected to a stimulator usually delivering stimuli of suprathreshold intensity: 12-V pulses of 20 ms duration at 20 Hz for 400 ms at a time. A gravity-fed perfusion system irrigated the chamber with sea water alone and also delivered dissolved drugs. The flow rate was adjusted to prevent displacement of the fragile comb plates. Luminescence was measured using a fibre light guide (which viewed the entire preparation) connected to an EMI 9781B photomultiplier tube supplied with 700 V by a high voltage power source. The anodal signal of the photomultiplier was displayed on a Pharmacia chart recorder. Quantification of drug effects for comparative purposes was achieved by integration of the surface circumscribed by the chart pen deflection and the baseline.

Drugs used were the following from Sigma: acetylcholine HCl, L-adrenalin bitartrate, gamma-aminobutyric acid (GABA), atropine sulphate, dopamine HCl, eserine sulphate, D-glutamic acid, glycine, 5-hydroxytryptamine creatine sulphate (serotonin, 5-HT), L-noradrenalin HCl, DL-octopamine HCl, DL-propranolol, reserpine, and *d*-tubocurarine. Nicotine was obtained from Matheson Co., and phentolamine from Ciba-Geigy. All drugs were prepared in filtered, artificial sea water from freshly made stocks of 1–10 mmol l^{-1}. The less water-soluble drugs were dissolved first in dimethyl sulphoxide (DMSO) and diluted to the desired concentrations with sea water. DMSO (1% or less in sea water) had no noticeable effect on the luminescence responses of *Mnemiopsis*.

RESULTS

Luminescence activity

The segments of meridional canals used in the following account displayed considerable variability in their ability to luminesce spontaneously. The preparations used for electrical stimulation showed little or no spontaneous activity. While some of the preparations placed in the photometer vials were completely inactive, most displayed flashes of varying amplitude and frequency. The spontaneous activity of comb plates unrestrained in the assay vials may be due to mechanical disturbances in

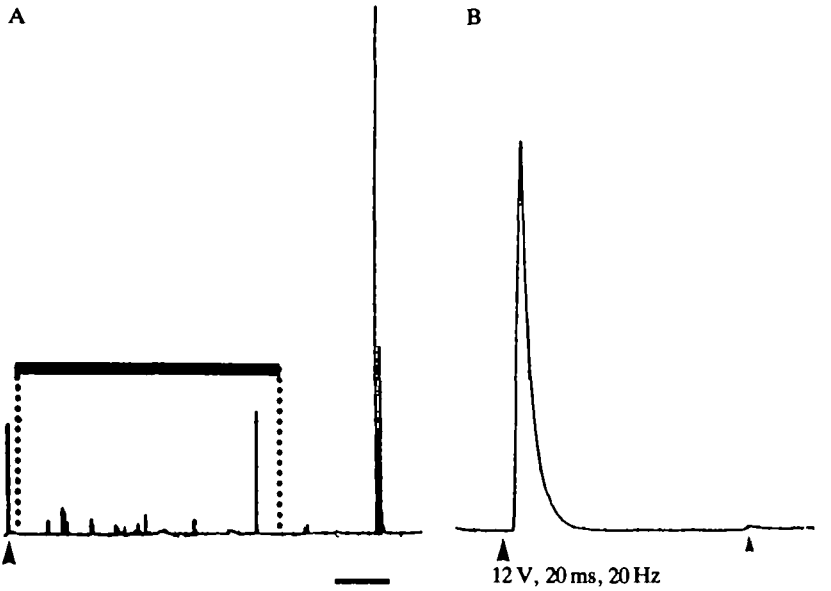

Fig. 1. Recordings of luminescence of pieces of *Mnemiopsis* comb plate rows following mechanical (A) and electrical (B) stimulation. Time of stimulation is marked by large arrowheads. Duration of electrical stimulation was 400 ms. Photomultiplier gain differed in these two recordings. Note that injection of sea water in (A) immediately produced a strong flash and that the ensuing flash activity is atypically high, including the very large flash on the right. The responses in Table 1 and Figs 2 and 5 were integrated by summation of the individual flash amplitudes over the time span shown as a horizontal bar in (A). A small spontaneous flash is indicated by small arrowhead in (B). Time scale: 60 s (A) and 2 s (B).

these preparations, or to the higher sensitivity of the photometer system. These activities were monitored for several minutes prior to drug addition and subtracted from the activities measured after drug addition.

Spontaneous and mechanically induced (by injection of sea water) luminescence appeared as flashes (Fig. 1A) lasting between 400 and 500 ms at 20–23 °C. Threshold electrical stimulation (10 V, 20 ms, 5 Hz, 100 ms pulse trains) of segments of comb plate rows induced similar flashes, while stronger, suprathreshold stimuli, such as those used in drug experiments, usually produced compound flashes of longer duration (1–2 s, Fig. 1B). The properties of flashes induced by electrical stimulation have been described in detail by Chang (1954). These responses were eliminated by replacing sea water with equal volumes of sea water and $MgCl_2$ (0·36 mol l^{-1}). KCl isotonic with sea water elicited a long-lasting glow, and superimposed flashes.

Effects of cholinergic drugs and blocking agents

Intact specimens of *Mnemiopsis* responded to acetylcholine (ACh, 10 mmol l^{-1}) with conducted displays of luminescence. Specimens were placed in a seawater dish such that the site of ACh injection was above seawater level. In three preparations that were unresponsive to injection of 0·1 ml of sea water just underneath the epidermis over the meridional canal, injection of an identical volume of 10 mmol l^{-1} ACh in the same location of the air-exposed area elicited, within 5 s, 2–4 luminescence flashes that were propagated along the comb plate row from the site of injection on that row. In

one specimen, flashes propagated also to an adjacent row, but in no case did luminescence spread to more, or all 8, rows of the animal.

Isolated segments of comb plate rows were responsive to ACh and prior addition of 10 μmol l^{-1} eserine sensitized the luminescence response to ACh (Table 1). The response typically included multiple flashes of varying amplitude and irregular occurrence (Fig. 3A). The flashes were usually initiated within 1 min of drug addition, and activity was maintained for up to 15–20 min before washing. The threshold for ACh responses was 0·1 μmol l^{-1} and the integrated luminescence response increased with ACh concentrations as a sigmoid curve (Fig. 2). However, the responses to ACh concentrations between 10 μmol l^{-1} and 10 mmol l^{-1} were highly variable, and the concentration of maximal response varied from one preparation to another.

Nicotine induced similar flashes in segments of comb plate rows. In comb plate segments exposed consecutively to both drugs, the integrated response to 1 μmol l^{-1} nicotine was ten times greater (9·4 ± 1·9, $N = 5$) than that to 1 μmol l^{-1} ACh. The response to ACh was considerably and irreversibly reduced by tubocurarine (Fig. 3A) but not atropine (Table 1). On the contrary, 0·1 and 1 mmol l^{-1} atropine induced multiple flashes delivered at high rates (Fig. 3B). Atropine at concentrations of 10 μmol l^{-1} or less was ineffective in eliciting luminescence. The integrated atropine response was approximately equivalent to that of the ACh response at equimolar concentrations (1 mmol l^{-1}).

The luminescence response to electrical stimulation was augmented significantly in the presence of 10 and 100 μmol l^{-1} eserine (Table 1). Tubocurarine (0·1 mmol l^{-1}) caused a sharp reduction of the response 15 min after its addition to the bath (Table 1), but only after potentiating the response in the form of multiple, small flashes (Fig. 4A). Tubocurarine (1 mmol l^{-1}) abolished irreversibly the response to electrical stimulation, but was ineffective at 10 μmol l^{-1}. Atropine (10 μmol l^{-1}) caused a four-fold increase in the amount of light produced by electrical stimulation (Fig. 4B; Table 1).

Table 1. *Effects of drugs on luminescence responses to electrical stimulation, acetylcholine and adrenalin*

Drugs	Concentration (μmol l^{-1})	Stimuli: Electrical stimulation	Stimuli: Acetylcholine (1 μmol l^{-1})	Stimuli: Adrenalin (1 μmol l^{-1})	Luminescence induction
Eserine	10	177·8 ± 27·2 (5)	193·6 ± 18·6 (4)	NA	−
Tubocurarine	100	8·24 ± 1·43 (4)	10·90 ± 1·35 (3)	95·21 ± 7·82 (3)	−
Atropine	10	422·1 ± 89·7 (3)	107·3 ± 11·1 (4)	NA	+ (0·1, 1 mmol l^{-1})
Reserpine	100	253·6 ± 46·2 (3)*	NA	93·47 ± 8·84 (3)	−
		11·23 ± 2·17 (3)**	NA	NA	
Propranolol	100	5·65 ± 1·79 (5)	0 (3)	0 (5)	+ (0·5, 1 mmol l^{-1})
Phentolamine	100	88·28 ± 9·17 (4)	NA	104·5 ± 7·9 (5)	−
Serotonin (5–HT)	1000	0 (3)	0 (4)	0 (3)	+ (1 mmol l^{-1})

Data expressed as percentage of control responses: $\bar{x}$ ± S.E.M. (number of experiments, 15 min after exposure to drug except for reserpine where preparations were tested 5 (*) and 30 min (**) after addition of drug.

NA, test not attempted. Presence (+) or absence (−) of luminescence inducing ability is indicated, as well as concentrations for responsive preparations.

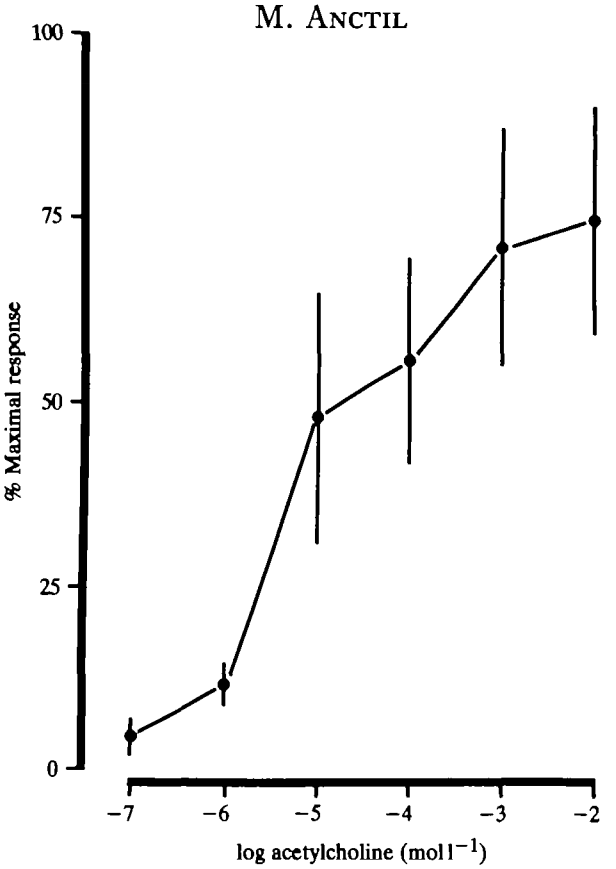

Fig. 2. Concentration-response relationship for ACh-induced flash activity of a segment of comb plate row. Curve represents total light integrated from flash amplitudes cumulated over a fixed time span (see Materials and Methods and Fig. 1) and expressed as the means ± S.E.M. ($N = 4$) of percentage of the maximal luminescence response (= 100 %). The preparations were pretreated with 10 μmol l^{-1} eserine prior to ACh injections.

Effects of adrenergic drugs and blocking agents

Intact specimens of *Mnemiopsis* responded to subepidermal injections of 0·1 ml of noradrenalin (NA, 10 mmol l^{-1}) with local luminescence episodes. In quiescent preparations, local instillation of NA invariably induced a single local flash of low intensity, whereas addition of an identical volume of sea water was ineffective. In hyperactive preparations, where both conducted (3–5) and local (1–2) flashes were elicited within 5–30 s by adding 0·1 ml of sea water, instillation of the same volume of NA (10 mmol l^{-1}) failed to influence the number of conducted responses elicited, but increased the number of local responses to 4 to 6 ($N = 4$) within 1 min of application. Local responses were identified as symmetrical flashes around a 2–3 mm strip of meridional canal where the drug was applied, and lasting approximately 1 s.

Isolated segments of comb plate rows flashed in response to adrenalin at concentrations as low as 1 nmol l^{-1}. The response pattern was similar to that elicited by ACh, involving multiple but separate flashes of short duration and variable amplitude, appearing after a delay of 10–60 s. The adrenalin-induced luminescence disappeared after washing the preparation in fresh sea water. A cumulative concentration-response curve is shown in Fig. 5. The response was maximal at 1 μmol l^{-1} and decreased at

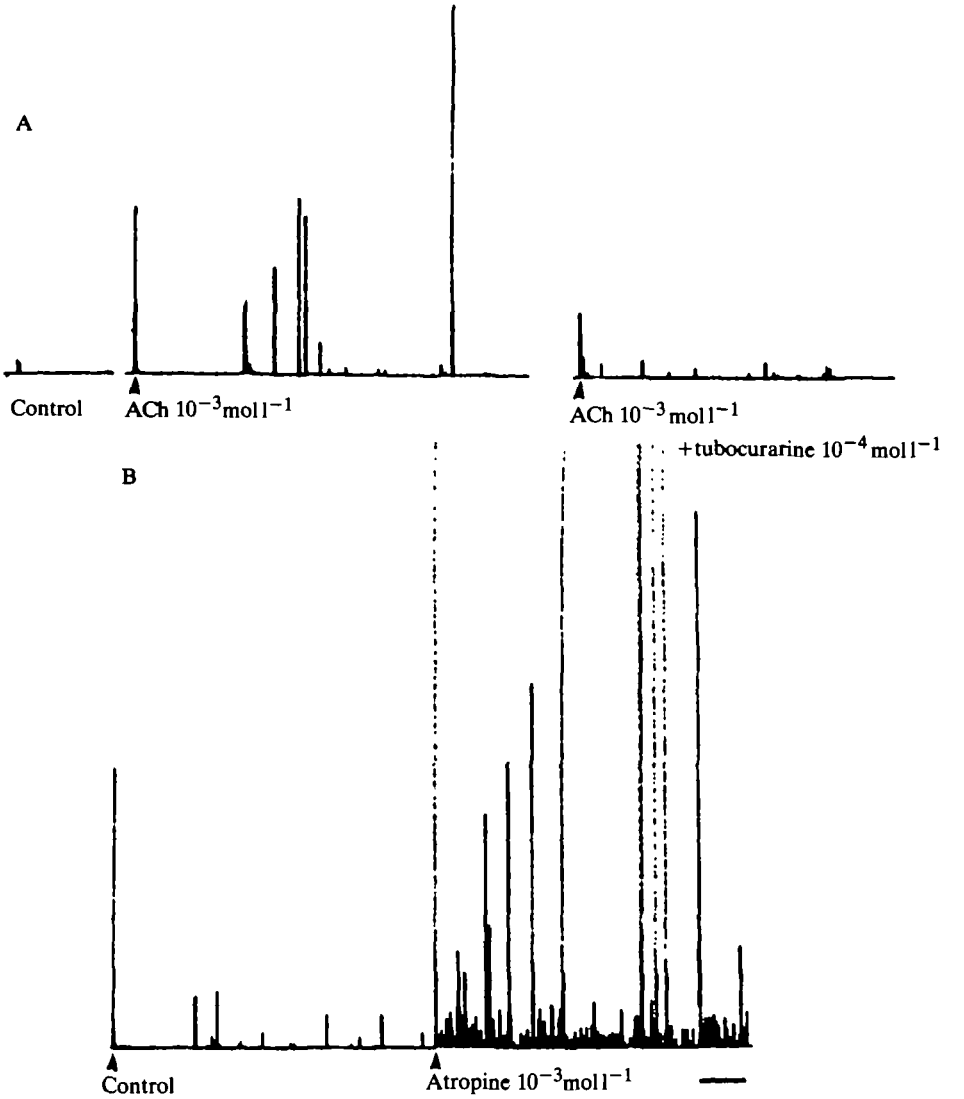

Fig. 3. (A) Effect of tubocurarine on ACh-induced luminescence in an isolated segment of comb plate row. Flash activity elicited by ACh injection (arrowheads) is shown before (middle trace) and 15 min after treatment with tubocurarine (right trace). The sea water control displayed little activity. (B) Luminescence-inducing activity of atropine in another preparation. Note flash activity following injection of sea water (Control) and high rate of small flash activity after atropine. Scale: 60 s.

higher concentrations. It is not clear whether desensitization and/or other processes may account for the loss of potency at concentrations above 1 μmol l^{-1} but application of 1 mmol l^{-1} adrenalin to freshly prepared strips of comb plate rows elicited flash responses whose integrated amplitude was similar to that measured after 1 μmol l^{-1} adrenalin. Noradrenalin and dopamine (0·1–1 mmol l^{-1}) also induced flash activity, but were ineffective at concentrations lower than 0·01 mmol l^{-1}.

The flash response to adrenalin (0·1 and 1·0 mmol l^{-1}) was abolished by pretreating isolated preparations with propranolol (0·1–0·5 mmol l^{-1}) (Fig. 6; Table 1).

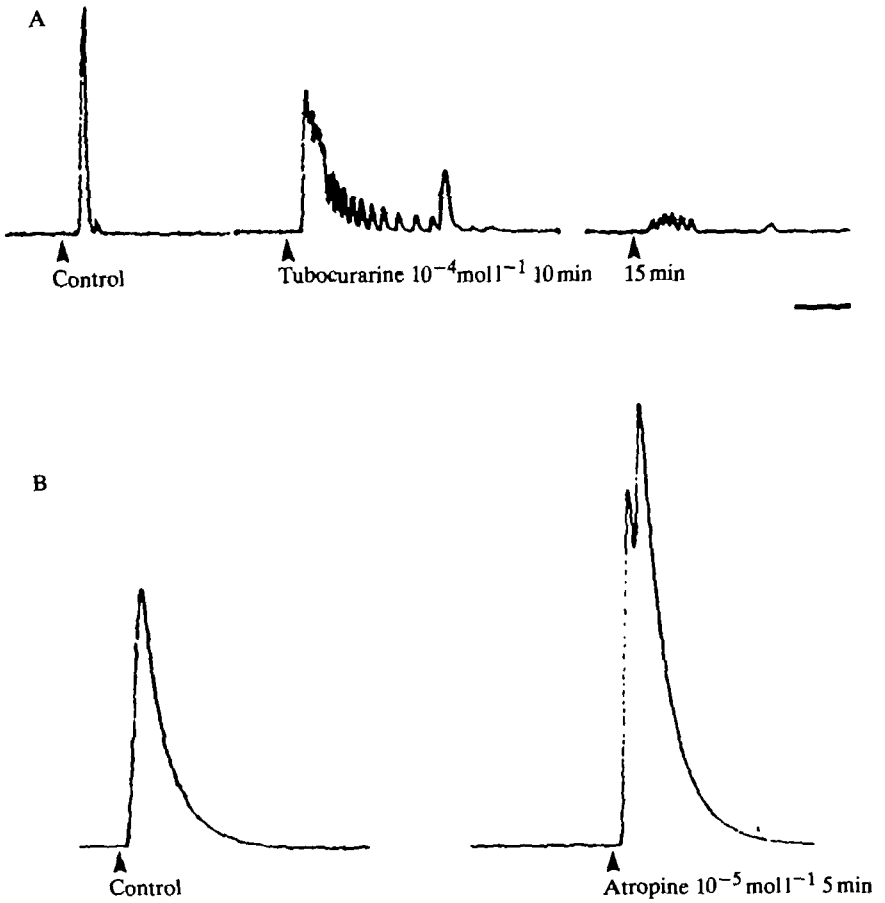

Fig. 4. Effects of tubocurarine (A) and atropine (B) on luminescence induced by electrical stimulation (arrowheads: 12 V, 20 ms, 20 Hz, 400 ms pulse train) in isolated segments of comb plate row. In (A), responses before (left), 10 min (middle) and 15 min after exposure to tubocurarine are shown. In (B), responses before (Control) and 5 min after addition of atropine (right) are shown. Scale: 2 s.

Propranolol, by itself, induced multiple flashes only at 0·5–1·0 mmol l^{-1} (Fig. 6B), and flashes were accompanied by a long-lasting glow at 1 mmol l^{-1}. Phentolamine (10 and 100 μmol l^{-1}) failed to influence the adrenalin response (Table 1).

Adrenalin (10 μmol l^{-1}) considerably enhanced the luminescence response of isolated preparations to electrical stimulation. Reserpine (0·1 mmol l^{-1}) similarly potentiated this response to 200–300 % of control values within 2–5 min after addition to the bath (Table 1), but prolonged (30 min) exposure to reserpine led to a sharp reduction of electrically induced luminescence (Table 1). In contrast, reserpine failed to affect the adrenalin response (Table 1). Propranolol (0·1 mmol l^{-1}), but not phentolamine, considerably reduced or sometimes abolished irreversibly the response to electrical stimulation (Fig. 7A; Table 1).

It is noteworthy that 0·1 mmol l^{-1} propranolol abolished the luminescence response to 1 mmol l^{-1} ACh as well as the response to adrenalin (Table 1). In contrast, 0·1 mmol l^{-1} tubocurarine failed to block the response to 1 mmol l^{-1} adrenalin (Table 1) while reducing the response to ACh.

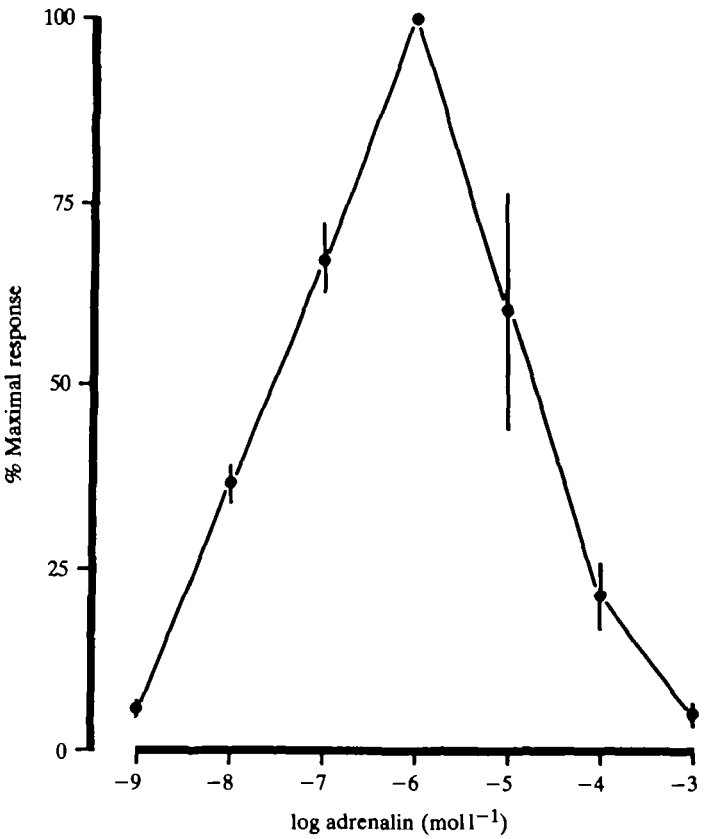

Fig. 5. Concentration-response relationship for adrenalin-induced flash activity of isolated segments of comb plate rows. Curve was plotted as in Fig. 2. In all four preparations, maximal integrated response was obtained at 1 μmol l^{-1}.

Effects of serotonin (5-HT) and other drugs

5-HT (0·1 and 1·0 mmol l^{-1}) elicited a small luminescence response within 10–15 s after addition of the drug to the bath. The response resembled that elicited by ACh and adrenalin, i.e. a burst of flashes of variable amplitude (Fig. 8B). This response lasted 3 min and was followed by a period of inhibition during which the luminescence responses of the isolated preparations to mechanical, electrical (Fig. 7B), adrenalin (Fig. 8) and ACh stimulations were greatly reduced or abolished by 1 mmol l^{-1} 5-HT (Table 1), but not 0·1 mmol l^{-1} 5-HT. The responses to electrical stimulation, 1 mmol l^{-1} ACh and 1 mmol l^{-1} adrenalin were restored after washing the preparations in fresh sea water.

Glutamate, glycine, GABA and octopamine failed to elicit luminescence or to influence electrically stimulated luminescence of isolated preparations at concentrations up to 1 mmol l^{-1}.

DISCUSSION

The results reported above provide evidence that cholinergic and monoaminergic mechanisms are associated with transmission of luminescence excitation in *Mnemiopsis*.

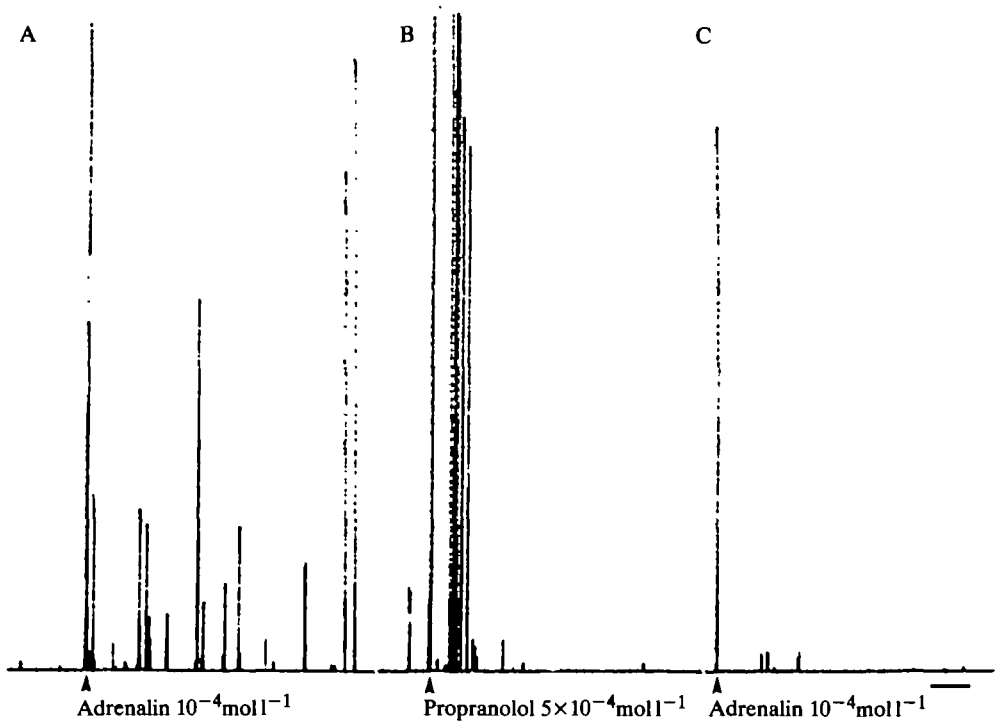

Fig. 6. Effect of propranolol on flash activity induced by adrenalin in an isolated segment of comb plate row. The adrenalin responses before (A) and 15 min after application of propranolol (C) are shown. Note in (B) the intense and transient flash activity shortly after addition of propranolol. Scale: 60 s.

The evidence includes the low concentration threshold of the luminescence responses to ACh and adrenalin, and the selectivity of the effects of agonists and blocking agents on these responses. The neuronal origin of the adrenergic mechanism is suggested by the depression of the luminescence response to electrical stimulation in the presence of reserpine, a neuronal monoamine-depleting drug which failed to affect the response to exogenous adrenalin. The short-term excitatory effect of reserpine may have resulted from the displacement and release of the endogenous catecholaminergic transmitter. Although there is ultrastructural evidence of neurites associated with photocytes of *Mnemiopsis* (Anctil, 1985), there is, as yet, no evidence that these neurites utilize a biogenic amine as neurotransmitter.

Both ACh and adrenalin induced intermittent bursts of flashes but no glow or continuous response in *Mnemiopsis*. This striking feature is puzzling when confronted with the glow and superimposed flash responses induced by KCl in this study. Presumably, KCl depolarized the photocytes directly, hence the glow, but also depolarized neurones whose episodic activity would be transmitted to photocytes as flashes. In the cnidarian *Renilla*, both KCl and adrenalin elicited glows as well as superimposed flashes from the luminescent tissue (Case & Strause, 1978; Anctil *et al.* 1982), and adrenalin induces glow responses in isolated photocytes of *Renilla* (G. Germain & M. Anctil, unpublished). Thus it is likely, but yet to be demonstrated, that neither ACh nor adrenalin act directly on the photocytes of *Mnemiopsis*. However, because only the adrenalin-induced flash responses were found to remain

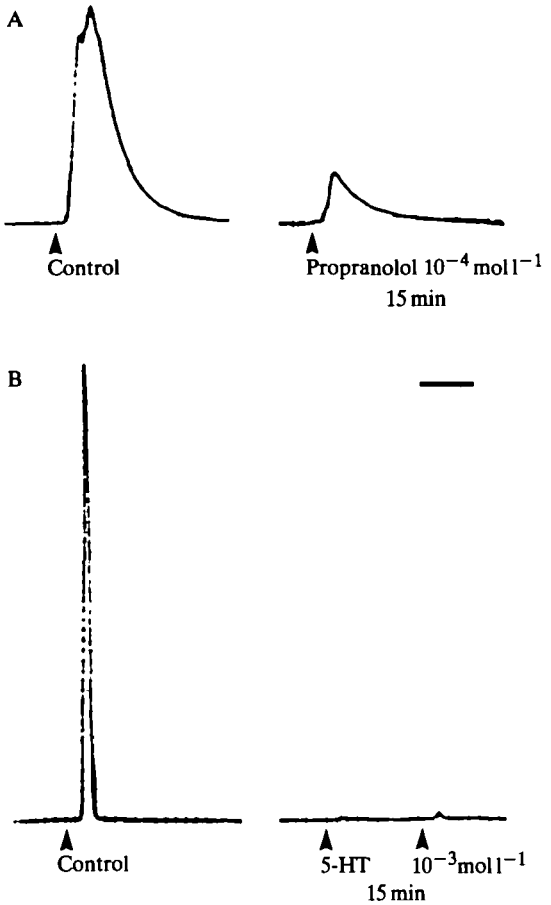

Fig. 7. Effects of propranolol (A) and 5-HT (B) on luminescence induced by electrical stimulation (arrowheads: 12 V, 20 ms, 20 Hz, 400 ms pulse train) in isolated segments of comb plate rows. In (A) and (B) responses are elicited 15 min after adding the drug, and in (B) two stimulation episodes were attempted. Scale: 2 s.

localized to the site of injection, it is presumed that adrenalin acts on a neuronal system or other excitable cells closely associated with the photocytes.

The effects of adrenergic drugs on luminescence activity in *Mnemiopsis* closely resemble those reported in the cnidarian *Renilla* (Anctil *et al.* 1982). As in *Renilla*, luminescence induced either by electrical stimulation or adrenalin is suppressed by propranolol but not phentolamine, thus implying the specific mediation of beta adrenoceptors in this response. Similarly noradrenalin and dopamine are less effective than adrenalin in eliciting the response, and high concentrations of propranolol induced luminescence in both *Renilla* and *Mnemiopsis*. Thus the adrenergic control mechanisms for luminescence are strikingly similar in these two members of primitive phyla. The luminescence-inducing action of high concentrations of propranolol is possibly unspecific as suggested for *Renilla* (Anctil *et al.* 1982), although this issue deserves further investigation. In *Mnemiopsis*, this effect involves both a glow and superimposed flashes as does the effect of KCl. Both agents may induce these responses by affecting ion-dependent membrane properties of the photocytes.

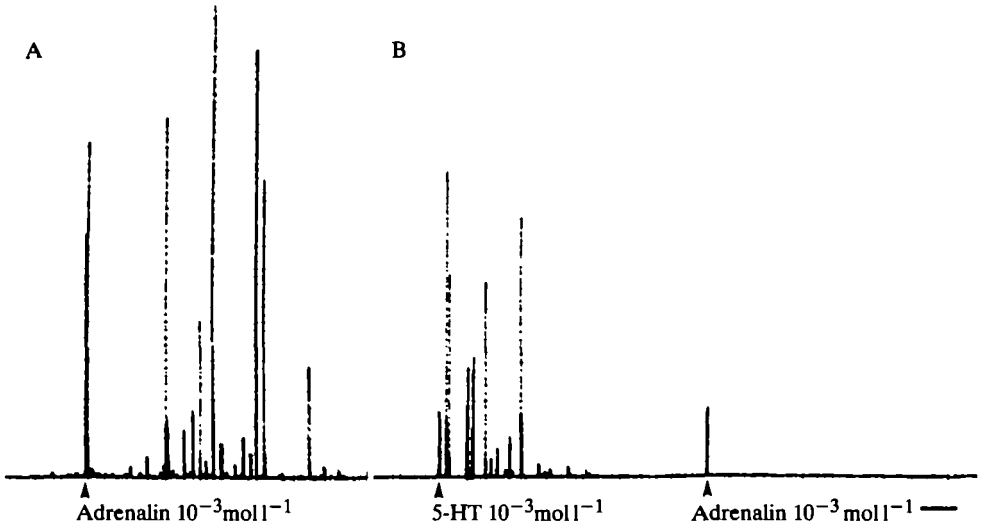

Fig. 8. Effect of 5-HT on flash activity induced by adrenalin in an isolated segment of comb plate row. The adrenalin responses before (A) and 8 min after application of 5-HT (B) are shown. Note in (B) the transient flash activity elicited shortly after adding 5-HT. Scale: 60 s.

ACh and tubocurarine, which were reported to be ineffective on the luminescence system of *Renilla* (Anctil *et al.* 1982), were highly effective on the luminescent tissue of *Mnemiopsis*. ACh appears to excite the system *via* nicotinic receptors since nicotine agonized and tubocurarine antagonized the flash response. The highly excitatory effect of atropine on luminescence may reflect either an unspecific action on photocytes, as postulated for high concentrations of propranolol, or the presence of a muscarinic inhibitory pathway associated with luminescence control. Kass-Simon & Passano (1978) described similar antagonistic effects of tubocurarine and atropine on the frequency of contraction burst pulses in the cnidarian *Hydra*. It is also puzzling that tubocurarine transiently prolonged the electrically stimulated luminescence response of *Mnemiopsis*, and only after 15 min did it cause a marked reduction of the response (Fig. 4A). This unexplained transient effect is either indicative of a relatively poor specificity of tubocurarine in this preparation (although nicotine was effective), or of complex interactions between types of receptors.

The luminescence-inducing activity of ACh and adrenalin is similar except for the higher sensitivity threshold for ACh (100 nmol l^{-1} as opposed to 1 nmol l^{-1} for adrenalin) and the concentration necessary to produce the maximal integrated response. The differences in response sensitivity between the two putative transmitters may reflect differences in photocytic receptor activity, stimulus-response coupling or neural pathways used by ACh and adrenalin. The latter possibility is supported by the ability of propranolol to block the ACh response and the failure of tubocurarine to block the adrenalin response. This observation suggests that ACh may trigger flash responses from photocytes through an intervening adrenergic pathway. That the cholinergic mechanism presumably involved is indirect, possibly through a nerve net, is also supported by evidence that the flash responses to ACh are propagated along the comb plate rows, whereas the adrenergic responses remain local. The postulated presence of multiple synaptic pathways mediating the ACh effect may help explain

the considerable variability of the integrated response amplitudes (Fig. 2). Coupling between photocytes, as suggested by the numerous gap junctions interconnecting them (Anctil, 1985), may also contribute to this variability as well as to luminescence conduction along the meridional canals in *Mnemiopsis*.

The mixed excitatory/inhibitory effects of 5-HT on luminescence raise difficult problems of interpretation. The specificity of these actions remains to be demonstrated since 5-HT-specific antagonists were not available at the time of these experiments. A false transmitter effect of 5-HT could have resulted in displacement of the postulated adrenergic-like transmitter, hence the brief period of flash activity after addition of 5-HT followed by a prolonged period of inexcitability. However, the latter hypothesis is countered by evidence that the flash response to exogenous adrenalin is also blocked in the presence of 5-HT. Clearly, the mechanism of action of 5-HT in *Mnemiopsis* should be investigated further.

In conclusion, cholinergic and adrenergic-like mechanisms seem to be present in *Mnemiopsis* on the basis of pharmacological activities associated with luminescence control. Other activities of ctenophores, ciliary and muscular in nature, also appear to be influenced by these transmitter mechanisms on the basis of presently incomplete pharmacological and histochemical evidence (Hernandez-Nicaise, 1976; Satterlie, 1978). Together with the pharmacological (Anctil *et al.* 1982) and radioautographic evidence (Anctil, Germain & LaRivière, 1984) in the cnidarian *Renilla*, these observations argue for the early emergence of cholinergic and adrenergic neurotransmitter mechanisms in metazoan nervous systems.

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