Camp. Biochem. PhysioL Vol. 60C, pp. 137-143.
Pergamon Press Ltd 1978. Printed in Great Britain

03064492-78-0701-0137502.00/0

THE STIMULATION OF ISOLATED PHOTOPHORES *(ARGYROPELECUS)* BY EPINEPHRINE AND NOREPINEPHRINE

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(Received 18 *July* 1977)

Abstract-1. Photophores isolated from the ventral region of the bathypelagic fish *Argyropelecus hemigymnus* respond to epinephrine and norepinephrine by a long-lasting luminescence: the maximal light emission occurring at 10^{-6} M for epinephrine and 10' M for norepinephrine, is estimated at 8 x 10" quanta/sec per photophore.

2. The LD_{50} value of the light response calculated from the dose-response curves to both neuromediators. is 2 x 10^{-7} M for epinephrine and 2 x 10^{-6} M for norepinephrine.

3. The light response to epinephrine is depressed by propranol and phentolamine 10' M, and the maximum is shifted to concentration higher than 10^{-6} M. Phentolamine completely blocks the light evoked by norepinephrine at a concentration lower than 10' M and shifts the light maximum to 10^{-3} M.

4. Photophores show a greenish fluorescence when excited with 365 nm u.v. light; the fluorescence intensity, is largely decreased after the light emission.

INTRODUCTION

Harvey (1931) was the first to observe a luminescence of a bathypelagic luminous fish *(Echiostoma ctenobarba)* triggered by an intraperitoneal injection of epinephrine. A similar injection in the deep-sea fish *Linophryne arborifera* failed to stimulate the photophores. After injection of epinephrine in a specimen of *Argyropelecus olfersi,* Bertelson & Grontved (1949) observed a yellowish-green luminescence appearing from the photophores. Anctil (1972) induced a luminescence of the light organs by an injection of epinephrine in 3 specimens of *Maurolicus muelleri* but none in either of 2 specimens of *Benthosema glaciate* and 1 specimen of *Myctophum punctatum.* According to Barnes & Case (1974), an intramuscular injection of norepinephrine in swimming myctophids *Stenobrachius leuropsarus* induces a luminescence from all their photophores; however, these authors made control experiments in which saline injected into the fish induced luminescence as well. On the other hand, photophores isolated from *Argyropelecus hemigymnus* did not luminesce when stimulated with 10^{-3} M epinephrine (Anctil, 1972; Baguet & Marechal, 1976).

We have re-investigated the problem by studying the luminescent response of photophores isolated from *Argyropelecus hemygymnus* and stimulated by epinephrine. We confirmed our previous finding (Baguet & Marechal, 1976) but we found that all the photophores responded to concentrations of epinephrine or norepinephrine as low as 10^{-}' M: the organs are thus inhibited by high concentration of epinephrine (10^{-3} M). Dose-response curves have been established for the 2 neurotransmitters: photophores are 200 times more sensitive to epinephrine than to norepinephrine; their responses are inhibited by propranolol (a fi-adrenergic inhibitor) and by phentolamine (an a-adrenergic inhibitor). These results suggest strongly that epinephrine is the normal neurotransmitter of the photophores of *Argyropelecus hemigymnus.*

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METHODS

1. Collection of fish

In the strait of Messina, during the maximum tide, the deep-water currents violently deflected upwards by the barrier shallow, drag to the surface living luminescent fish among which *Argyropelecus hemigymnus* is the most numerous (Baguet & Marechal, 1975); when the south wind (Sirocco) blows strongly, the fish are brought to the Sicilian coast and stranded on a shore line extending from Ganzirri to Torre di Faro (Genovese *et al.,* 1971). Most specimens of *Argyropelecus* were collected on the shore soon after their being stranded but some of them were captured in a handnet from the eddies areas of the strait produced by the deep-water currents. All specimens showed 3 characteristics which disappear rapidly some hours after death: (i) the head and the trunk have play of light on the scales; (ii) the muscles of the caudal region are soft and translucent; (iii) the iridophores located above the caudal photophores shine a brilliant golden colour.

Fish lacking any one of these three characteristics were discarded.

2. Dissection of the photophores

The row of ventral photophores (labeled "v" in Fig. I of Baguet & Marechal, 1976) has 12 organs on each side. The 18 posterior photophores were isolated from the trunk (under microscope) with scissors and fine forceps for iridectomy, and they were cut into a posterior half, containing

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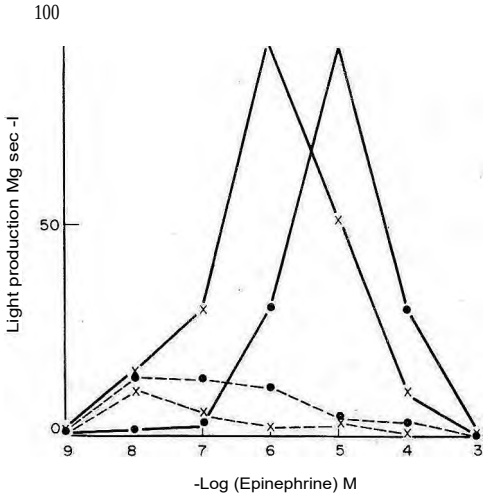


Fig. 1. Dose-response curves of isolated ventral photophores of *Argyropelecus hemigymnus* to epinephrine. The continuous lines correspond to the A-type photophores, the stippled lines correspond to the B-type photophores (x , rear photophores; et, front photophores). Ordinate: light intensity (Mq sec^{-1}). Abscissa: negative log of epinephrine concentration.

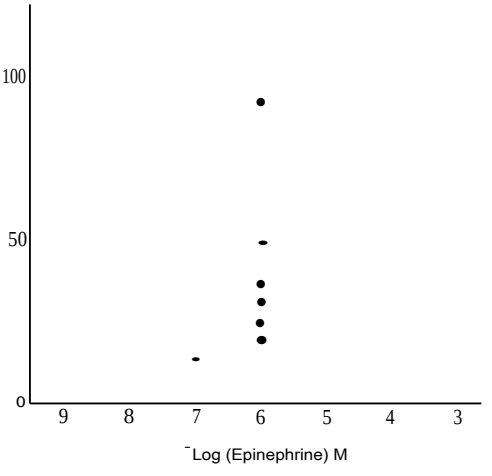


Fig. 2. Distribution of the light maxima of 24 isolated ventral photophores of *Argyropelecus hemigymnus* for different epinephrine concentrations. Ordinate and abscissa as in Fig. 1.

10 organs, and an anterior half containing 8 organs. These 2 preparations are called in this paper the *"rear photophores"* and the *"front photophores"*. The rear photophores are somewhat smaller than the front photophores, so that the total areas of each half are about equal.

The amount of luciferin contained in these preparations was assessed by an indirect method. Baguet & Nicolas (1976) have observed a greenish fluorescence of the photophores excited at 365 nm, which was inversely proportional to the time during which the organ can maintain the light emission; they suggested that this fluorescence arose from the excitation of the luciferin of the photophores. We have also observed a similar fluorescence in the photophores of *Argyropelecus,* using a fluorescence microscope (Galileo); the intensity of the greenish fluorescence, called here "fluorescence index" is estimated semi-quantitatively by visual observation. The organs which did not fluoresce well were discarded; those which fluoresced well were examined again at the end of the experiments.

3. Measurements of the light emission

The preparation is transferred to a small Perspex chamber, and laid perpendicularly to the photomultiplier in order that the emitting area of the photophores faces the photocathode at a distance of 2.0 cm. The luminescent response obtained from the specimen is sensed by a photomultiplier PM 270C (International light) operating with a IL 760 power supply, and it is displayed on a 20 cm strip-chart recorder. Between each measurement, which lasted about 10 sec, the organs were kept in Petri dishes containing saline with the pharmacological agents. The light production was calibrated with a standard light source ("Betalight" source; peak at 470 nm) held at the place of the tissue.

4. Pharmacological agents

Stock solutions of epinephrine hydrochloride (10^{-3} M) and norepinephrine hydrochloride (10^{-3} M) (Fluka) dissolved in saline (NaCl, 13.5 g; KCl, 0.6 g; $CaC1_2$, 0.25 g; $MgC1_2$, 0.35 g per 1. solution adjusted to pH 7.3 with Tris buffer) containing a reductor (Na_2SO_3 1%) were stored at 5°C and diluted as required before use. In some experiments, phentolamine and propranolol (a and fi-adrenergic inhibitors) were added to the solutions at a final concentration of 10^{-5} M.

5. Design of the experiments

The preparation was immersed after dissection into 1 ml of saline at 14°C for 30 min. It was transferred every 30 min into 1 ml saline with epinephrine at concentrations that increased from 10^{-9} to 10^{-3} by steps of 10^{-1}. The measurements of the light production were performed at each transfer. At the end of the experiments, the fluorescence index was estimated.

The experiments with norepinephrine were performed in exactly the same way, except that norepinephrine was substituted for epinephrine.

When inhibitors (propranolol or phentolamine) were used, the preparations were first immersed for 30 min in saline containing the inhibitor (10^{-5} M), and then they were stimulated by epinephrine or norepinephrine in the presence of 10^{-5} M inhibitor.

RESULTS

1. The light response to epinephrine

Thirty minutes after the dissection, most of the organs, which had been kept in saline, produced a small light emission of 0.8 Maquanta/sec, or about 0.1 Maquanta/sec per photophore. This is about 300 times less than the light production during a flash evoked by an electrical stimulation (cf. Fig. 1 of Baguet & Marechal, 1976); this very low light production explains why Baguet & Marechal (1976) state that no excised photophores luminesce spontaneously.

The light production increases when the organs are stimulated with epinephrine, even if its concentration is as low as 10^{-9} M. Figure 1 shows the dose-response relationship obtained from the photophores of 2 fish. The continuous lines (example A) are the dose-response curves for front (circles) and rear (crosses) photophores (of the same fish). The 2 curves are simi-

Table 1. Light production (Mq sec^{-1}; S.E.M. = standard error of mean)

Epinephrine	Log (M) bath	-9	-8	-7	-6	-5	-4	-3	*N*	S.E.M.
Type B	0.7	5.6	14.7	11.4	7.7	3.6	1.8	1.5	9	0.9
Type A	0.9	6.4	16.1	20.3	45.8	63.7	23.0	2.9	14	3.5
All data	0.8	6.1	15.5	16.8	30.8	40.2	14.7	2.3	23	2.2

lar: the light production increases with the epinephrine concentration, up to 10^{-6}-10' M; and decreases for still higher concentrations of epinephrine. Extinction is complete at 10^{-3} M. The stippled lines (example B) represent the dose-response curves of front and rear photophores of another fish: the light production is maximal at a much lower epinephrine concentration (10^{-8} M); there is no difference between the front and the rear photophores. However, the intensity of the light is similar to that produced by the organs of example A stimulated by the same dose of epinephrine, and it is much lower than the maximal light intensity obtained in type A for 10' M epinephrine.

Such dose-response curves have been obtained on 24 samples: 12 front photophores, and 12 rear photophores, dissected from 12 fish. The intensity of the light was maximum for various epinephrine concentrations (Fig. 2). Fourteen samples produced more than 30 Mq/sec for epinephrine concentrations of 10^{-5} or 10^{-6} M, and 9 of them produced a maximal light emission which was smaller than 30 Mq/sec, for epinephrine concentrations of 10^{-8}-10^{-7}. One organ had a feeble light maximum for 10^{-3} M epinephrine and this result has been discarded in the following calculations. On the basis of these results, we divided the dose-response curves into 2 groups, called respectively A and B. The mean light production for each epinephrine concentration is shown in Table 1 for the 2 classes, together with their standard errors.

The light production is the same in type A and type B for low epinephrine concentrations (10^{-9} and 10^{-8}), being 6-20 times above that observed in the absence of epinephrine stimulation. For higher concentrations of epinephrine the response of type A photophores increases, while that of type B photophores decreases; this suggests that type B photophores are unable to maintain a light emission lasting longer than 1 hr. The light production of type A photophoresis maximal for 10^{-5} M epinephrine, and it is equal to 63.7 Mq/sec; this is about 8 Mq/sec per photophore, a value which is not much smaller than that observed during an electrical stimulation, (25-50 Mq/sec according to Baguet & Marechal 1976). From the results shown in Table 1, the ED" is evaluated as 4 x 10^{-7} M. For concentrations higher than 10' M, the light production is inhibited: that produced by 10^{-3} M epinephrine is only half that obtained with 10^{-9} M epinephrine.

Table 2 shows the dose-responses for front and rear photophores which are similar. The light maxima are respectively 45.9 and 42.3 Mq/sec; the rear photophores are perhaps somewhat more sensitive to epinephrine than the front ones, as their light maximum occurs for 10^{-6} M epinephrine, and their ED_{50}' at 2 x 10^{-7}, while the front ones have theirs respectively at 10' M and 2 x 10^{-6}. However, the light production of the rear photophores is not statistically different from that of the front ones, except at 2 epinephrine concentrations (10^{-6} M and 10^{-5} M).

2. *The effects of inhibitors on the light response to epinephrine*

(a) *Propranolol.* Twenty-two dose-response curves have been obtained on organs dissected from 11 fish and stimulated by epinephrine in the presence of 10^{-5} M propranolol. Figure 3 shows the maximal light responses, plotted as a function of the epinephrine concentration that evoked them; they range from 10^{-9} M to 10^{-4} M; the scatter is wider than that observed when epinephrine acts alone (cf. Fig. 2) and it is questionable whether the subdivision into types A and B which was used to analyse those results can also be used to analyse the results obtained with propranolol. However, the comparison of Fig. 3 with Fig. 2 suggests that propranolol inhibits the A-type photophores, by decreasing their light maximum, and by shifting it to *higher* concentrations of epinephrine; however the drug increases somewhat the light maximum of the B-type photophores, and shifts it to *lower* concentrations of epinephrine. The dose-response curves have been grouped according to the criterion of the localization of their light maximum, into type A (light maxima for epinephrine concentrations higher than 10^{-8} M, *N* = 17) and type B (maxima for epinephrine concentrations lower than 10^{-8} M, *N = 5).* Table 3 shows their mean responses: propranolol depresses by 59.5% the light maximum of the A-type photophores, and shifts their ED" to 4 x 10^{-6} M (i.e. 10 times); it increases 2.7 times the light maximum of the B-type and shifts it by an order of magnitude to *lower* epinephrine concentrations.

Table 2. Light production (Mq sec^{-} S.E.M. = standard error of mean)

	Epinephrine Log (M) -9	-8	-7	-6	-5	-4	-3	S.E.M.
4 front photophores	4.8	14.2	14.3	21.5	45.9	15.6	2.6	3.1
5 rear photophores	6.9	15.8	17.9	42.3	31.1	12.6	2.7	3.2
All data	6	15.0	16.3	33.0	37.4	13.9	2.6	3.2

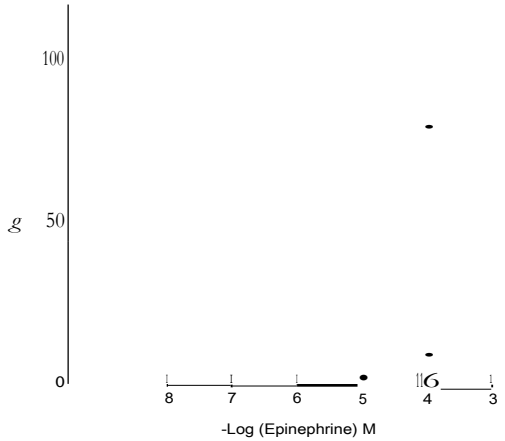


Fig. 3. Effects of propranolol 10' M on the distribution of the light maxima of isolated ventral photophores as a function of the epinephrine concentration. Ordinate and abscissa as in Fig. 1.

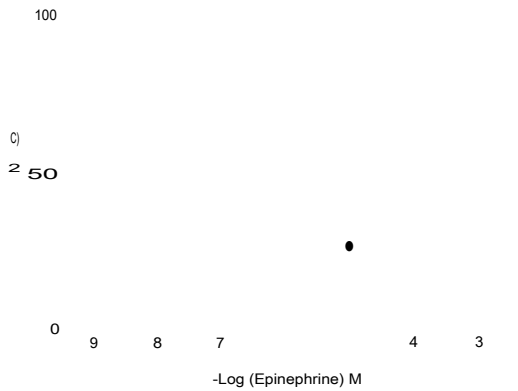


Fig. 4. Effects of phentolamine 10^{-5} M on the distribution of the light maxima of isolated ventral photophores as a function of epinephrine concentration. Ordinate and abscissa as in Fig. 1.

These interesting facts are probably linked to another one: propranolol, without any epinephrine, stimulates strongly the light production which reaches 30.6 Mq/sec ($N = 22$), a value 8 times higher than that observed for organs in saline and higher than the maximum light production with 10^{-5} M epinephrine. With 10^{-5} M propranolol, as a matter of fact, low concentrations of epinephrine (10^{-8} M) *inhibit* the light production triggered by propranolol alone.

No differences were found in the reactions of rear ($N = 11$) or front ($N = 11$) photophores stimulated by epinephrine in the presence of propranolol.

(b) *Phentolamine.* Twenty-two dose-response curves have been obtained on the organs of 11 fish stimulated by epinephrine in the presence of 10' M phentolamine. All the curves were similar, in that the light was maximum for epinephrine concentrations of 10^{-6} M-10^{-4} M (see Fig. 4). Thus no type B photophores were observed. Table 3 shows the mean light production as a function of the epinephrine concentration; it is maximum (23.1 Mq/sec) at 10^{-5} M epinephrine. The inhibitory effect of phentolamine on epinephrine stimulation is thus quite similar to that of propranolol. However phentolamine does not stimulate the photophores in the absence of epinephrine; on the contrary, it inhibits the spontaneous light emission of organs in saline. This effect is the reverse of that evoked by propranolol.

3. The light response to norepinephrine

Figure 5 shows one dose-response curve of a photophore to norepinephrine. Fourteen such experiments have been performed. The distribution of the light maxima is shown in Fig. 6; As is the case for epinephrine stimulation, the experiments were divided into 2 groups: A-type (light maximum for norepinephrine contractions higher than 10^{-6} M; $N = 8$) and B-type (light maximum for epinephrine concentrations lower than 10^{-6} M; $N = 6$). Figure 5 shows that norepinephrine stimulates feebly the organs at all concentrations; it stimulates them strongly in a very narrow range around 10' M; the magnitude of this maximum is not different from those obtained with epinephrine stimulation; the organs' responses at 10' M or at 10^{-3} M do not exceed 5% of the response observed at 10^{-4} M. It may be that this range is still narrower for other samples; this may explain why several of them had low maxima at 10' or 10^{-3} M (cf. Fig. 6). The mean ED_{50} ($N = 8$) for A-type preparations is 2 x 10^{-5} M: this is 200 times higher than that measured for epinephrine. No differences were observed between the responses of the front ($N = 4$) and the rear ($N = 4$) photophores.

4. The effects of inhibitors on the light response

As all the former experiments showed that there were no differences between rear and front photo-

Table 3. Light production (Mq sec^{-1}; S.E.M. = standard error of mean)

	Before inhibitor	After inhibitor	Epinephrine Log (M) -9	-8	-7	-6	-5	-4	-3	*N*	S.E.M.
Propranolol 10^{-5} M											
Type A	10.2	5.3	39.6	17.2	21.9	14.6	19.2	11.6	1.2	5	0.9
Type B	2.1	38.1	4.4	4.1	3.2	4.1	25.3	25.8	4.2	17	3.5
All data	3.9	30.6	12.4	7.1	7.4	6.5	23.9	22.6	3.5	22	2.2
Phentolamine 10^{-5} M											
Type A	12.3	4.3	1.2	0.7	1.1	7.5	23.1	13.9	0.3	22	2.1

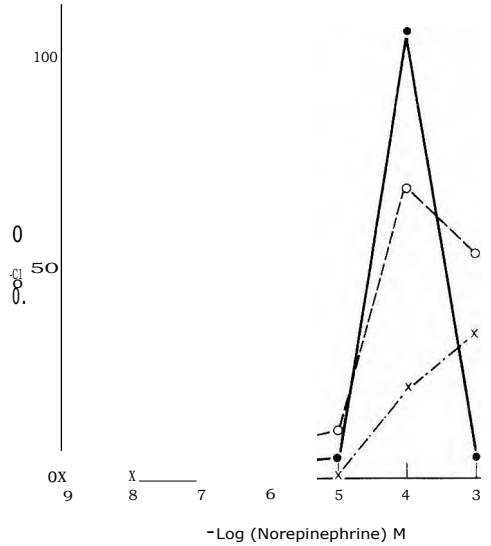


Fig. 5. Typical dose-response curves of A-type photophores to norepinephrine; without inhibitor (0), with propranolol 10^{-5} M (0), with phentolamine 10' M (x). Ordinate: light intensity (Mq sec^{-1}). Abscissa: negative log of norepinephrine concentration.

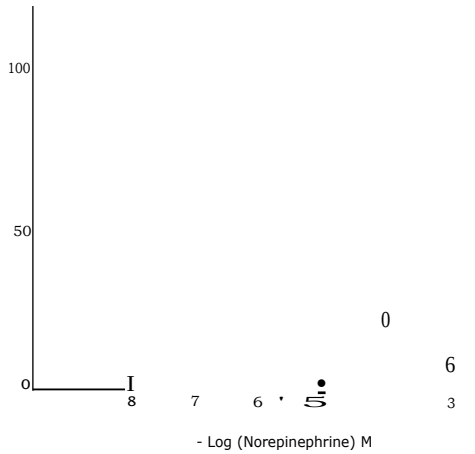


Fig. 6. Distribution of the light maxima of the A-type photophores as a function of norepinephrine concentration. Without inhibitor (0), with propranolol 10^{-5} M (0), with phentolamine 10^{-5} M (x).

phores, the design of the experiments was modified, to test the action of the inhibitors with an increased efficacity. The organs of 12 fish were divided into 12 pairs of rear and front photophores; 1 of 3 treatments was assigned to one member of the pair, in such a way that 8 organs were stimulated by norepinephrine, 8 by norepinephrine with 10' M propranolol and 8 with norepinephrine with 10^{-5} M phentolamine (balanced incomplete block design, Li, 1964). The distribution of the light maxima is shown in Fig. 6 (note that there are 6 supplementary points for norepinephrine: these experiments were performed before those testing the inhibitors). Figure 5 shows 3 dose-response curves for 3 organs; these curves, although experimental ones, are fairly close to the means and illustrate well the conclusions of the statistical analysis performed on the randomized blocks, limited to the cases of the A-type photophores.

Propranolol 10^{-5} M does not shift the light maximum, but depresses it by about 40%; it increases the light production at 10^{-3} M, i.e. it decreases the inhibition of large norepinephrine concentrations.

Phentolamine 10^{-5} M completely blocks the light production evoked by norepinephrine at concentrations lower than 10^{-5} M. The light production is maximum at 10^{-3} M, and equals 35% of that recorded without inhibitor; however this maximum is probably not true, because we have not tested concentrations of norepinephrine higher than 10^{-3} M. Thus phentolamine shifts the light maximum to larger concentrations of norepinephrine by at least one order of magnitude.

5. Fluorescence and luminescence

Photophores are fluorescent in green when excited with 365 nm u.v. light. We assigned to each of them, by visual inspection, a number (0-3), proportional to the intensity of the fluorescence. Table 4 reports the mean values of this index, measured *at the end* of the experiments. This index is 2-3 at the *beginning* of the experiments (the organs which did not fluoresce well were discarded). It is thus obvious that in all cases, the fluorescence has decreased quite a lot in the course of the experiments. Type A photophores have higher indexes than type B, in spite of the fact that they have produced a larger amount of light. The organs stimulated by epinephrine have a higher index than those stimulated with norepinephrine: here again it correlates with the total amount of light produced, which is larger if epinephrine is the stimulating agent. The index is also larger for epinephrine and propranolol, than for epinephrine alone.

All these observations are consistent with the idea that the fluorescence at the end of the experiments depends on the magnitude of the light production:

Table 4. Fluorescence index

	Epinephrine			Norepinephrine		
	Free	Propranolol 10^{-5} M	Phentolamine 10^{-5} M	Free	Propranolol 10^{-5} M	Phentolamine 10^{-5} M
Type A	0.30(9)	0.50(5)		0.15(7)	0.11(9)	0(2)
Type B	0.36(14)	0.67(17)	0.14(22)	0.19(9)	0.22(6)	0.28(6)

The number of measurements is shown in parentheses.
N.B.—Before the experiment: 0 = 0.50. After the experiment: 0 = 0.16.

photophores that show a low light production are photophores with a low luciferin content.

DISCUSSION

1. The light response to epinephrine and norepinephrine

Photophores isolated from the ventral region of freshly-captured specimens of *Argyropelecus hemigymnus,* luminesce in the presence of epinephrine 10^{-9}-10^{-5} M; the intensity of the light emission is maximal at 10^{-6} M for posterior photophores and 10^{-5} M for anterior photophores. The comparison of dose-response curves between epinephrine and norepinephrine puts in evidence the high sensitivity of photophores to epinephrine: the ED_{50} of the light response in epinephrine is 200 times lower than in norepinephrine. For concentrations higher than 10^{-5} M (epinephrine) and 10^{-4} M (norepinephrine), both neuromediators inhibit luminescence.

Contrary to phentolamine, propranolol does not influence the luminescence induced by epinephrine or norepinephrine at low concentration; luminescence at high concentration is depressed to the same extent by both inhibitors in epinephrine while in norepinephrine, the inhibiting effect of phentolamine is much more marked than propranolol.

The great sensitivity of isolated photophores to epinephrine suggests that epinephrine is the natural nervous mediator controlling the luminescence of *Argyropelecus hemigymnus.*

2. The nature of the neuromediator

In careful histological examination of well-preserved photophores of *Argyropelecus,* it has been possible to demonstrate that each cephalic photophore is innervated by a small but distinct nerve, this arising from the mandibular, buccal and hyoid branches of the facial nerve, which contains sympathetic efferent fibres (Nicol, 1960). The present pharmacological results support the hypothesis that the ventral serial photophores are innervated by the sympathetic nervous system as well.

Knowledge of the sympathetic origin of photophore innervation is not limited to *Argyropelecus;* the anatomical studies on the nervous system of the epipelagic luminescent fish *Porichthys* (Nicol, 1957) and the recent pharmacological studies on isolated photophores (Baguet, 1975; Anctil & Case, 1976) strongly support the view that *Porichthys* photophores are exclusively innervated by catecholaminergic neurons of sympathetic origin.

However, the characteristics of their light responses differ from those of *Argyropelecus.* in two respects.

(i) The photophores of *Argyropelecus* are able to luminesce continuously for 3 hr in presence of increasing concentrations of epinephrine. Such a long-lasting luminescence is never observed on the isolated photophore of *Porichthys:* the light response to epinephrine, whatever the concentration, lasts 10-20 min, the peak of light occurring 3-5 min after the beginning of stimulation. This light production is followed by a long refractory period; it is absolute for at least 30-40 min after the end of the first response and is relative for a long period afterwards. It cannot be reversed by increasing the epinephrine concentration (Baguet, 1975).

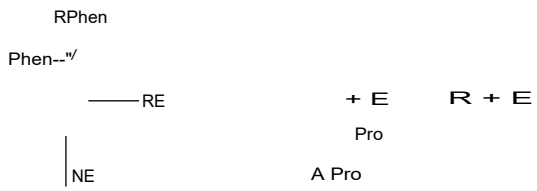
(ii) Epinephrine 10^{-5} M triggers a maximal light emission of 250 Mq/sec per photophore in *Porichthys* (Baguet, 1975) but only of 8 Mq/sec in *Argyropelecus.* The low rate of light production in *Argyropelecus* involves a slow decrease of the substrates for the luminous reaction and consequently, may explain the indefatigability of its light emission, in contrast to that of *Porichthys.*

The meaning of this difference is probably to be found in the following explanation offered for the role of luminescence in fish. According to several authors, the light of the ventrolateral and/or ventral surface of the body, matches the downwelling ambient light in order to obliterate the ventral silhouette of the fish (Fraser, 1962; Clark, 1963; McAllister, 1967; Nicol, 1969). In deep waters, where light intensities are low, the photophores of *Argyropelecus hemigymnus* can match the environmental light by shining at a much lower intensity than the photophores of *Porichthys,* which lives in upper water where the ambient light level is comparatively high. Moreover this difference between epipelagic and bathypelagic fish is emphasized by the number of photophores that are about 200 times as numerous in *Porichthys* as in *Argyropelecus.*

3. The nature of the sympathetic receptor

Sympathetic receptors of mammals are classified in 2 main groups (a and /3), according to their response to epinephrine, norepinephrine and a variety of drugs which stimulate or antagonize these 2 groups. We have attempted to decide whether the sympathetic receptors of the photophores belong to the a- or to the)3-class. It was expected that if they were of a-type they would be more sensitive to norepinephrine than to epinephrine, and that they would be inhibited by phentolamine, and not by propranolol (to a somewhat lesser degree); if they were of the f3-type, they would be more sensitive to epinephrine than to norepinephrine, and they would be inhibited by propranolol, but not by phentolamine. We have obtained mixed results: the photophores are more sensitive to epinephrine than to norepinephrine, and they are inhibited by propranolol (two)3-properties), but they are inhibited by phentolamine (an a-property). These results may be interpreted to mean that both a- and /3-receptors are present on the photophore postsynaptic membranes. However, some observations do not fit easily with this concept: (1) the photophores show a strong pharmacological desensitization, for epinephrine concentrations higher than 10^{-5} M or norepinephrine concentration higher than 10' M; (2) propranolol 10^{-5} M alone triggers the light emission, but phentolamine 10^{-5} M does not; (3) epinephrine and norepinephrine inhibit at low concentrations the propranolol light effect.

We propose the following model:



Adrenergic receptors of photophores are normally in a resting state *R;* they combine with epinephrine, *E* (or with a lower affinity, to norepinephrine, *NE)* and the complex *RE* is activated by a change of the receptor molecule *(A):* the complex *AE* dissociates, triggering the light emission; *A* returns spontaneously to the resting state *R.* Phentolamine inhibits *E* and *NE* by combining with *R;* propranolol can trigger the light emission by combining to *A,* and pulling the reaction *R—i• A* by mass action. As *E* has more affinity to *A* than has propranolol, the inhibiting effect of *E* on propranolol is also explained.

Although this model is very speculative, it can be used to design further experiments to clarify the mechanisms of the nervous control of photophores.

Acknowledgements—This work was made possible by a grant from the "Fonds National de la Recherche Scientifique" of Belgium, and from the "Centre beige d'Oceanographie".

We wish to acknowledge the help of Professor S. Genovese, director of the Istituto di Idrobiologia of the University of Messina (Italy) who provided us with the necessary information and bench space.

We thank Professor Th. Godfraind for his interest and advice during the preparation of the manuscript.

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