MONOAMINERGIC AMYGDALAR MECHANISMS OF CONDITIONED REFLEXES OF VARIED BIOLOGICAL MODALITY

I. V. Komissarov and A. N. Talalaenko

UDC 612.825.1:612.822.2

Microinjection of dopamine and serotonin into the dorsomedial zones of the rat amygdala led to an increase in the latent period of conditioned defensive and motor–food reflexes, spontaneous motor activity, and the number of pushing movements and the magnitude of the food-grasping reflex. Noradrenalin had no effect on the parameters of the conditioned food reflex but facilitated the conditioned avoidance reflex, reducing its latent period considerably. The results are evidence of the specificity of the neurochemical mechanism responsible for reflexes of varied biological modality at the amygdalar level in rats.

KEY WORDS: amygdala; conditioned reflexes of varied biological modality; monoaminergic mechanisms.

There are no experimental data on the neurochemical mechanisms of activity of the amygdala as a structure responsible for the conditioned food and defensive behavior of animals [4, 8, 12] although the presence of adrenergic, dopaminergic, and serotoninergic axons has been demonstrated histochemically in it [13] and neurons sensitive to catecholamines and serotonin have been discovered [2, 11]. However, some similarity can be assumed between the neurochemical mechanisms of positive and negative conditioned reflexes in the amygdala, as is shown by the convergence of excitation of different biological modalities on the same amygdalar neurons [2] and disturbance of the conditioned-reflex interchange of conditioned reflexes of different types in rats after destruction of the amygdala [7].

The object of this investigation was to study the functional role of noradrenalin (NA), dopamine (DA), and serotonin (5-HT), when injected into the dorsomedial zones of the rat amygdala, in conditioned-reflex responses of varied biological modality.

EXPERIMENTAL METHOD

Experiments were carried out on two groups of sexually mature male rats. Motor–food conditioned reflexes were investigated [5] in the animals (13) of group 1. The conditioned stimulus (a 1000 Hz buzzer), 10 sec in duration (acting alone for 5 sec), was applied 5 times daily at intervals of 60 sec. The unconditioned reinforcement consisted of pellets of powdered meat and bone with the addition of bread, which the animals obtained by opening a door. The motor–food conditioned reflexes (FCR) were quantified by an apparatus which transmitted the opening and shutting of the door to the axis of a potentiometer [6]. Three indices of the FCR were recorded: latent period (the time from sounding the buzzer to a push on the door), magnitude (the total angle of deviation of the door in response to the conditioned stimulus), and the number of pushing movements (the number of movements made by the animal in an attempt to open the door). In the animals (22) of group 2 a conditioned avoidance reflex (CAR) was produced by the method described previously [10]. After stabilization of the FCR and CAR, the animals were anesthetized with ether and a guiding cannula was inserted into the dorsomedial region of the right amygdala, using an atlas of the rat brain [3], by means of which solutions of the test substances could be injected in a volume of 2-5 μl from

---

Department of Pharmacology, Donetsk Medical Institute. (Presented by Academician of the Academy of Medical Sciences of the USSR V. V. Zakusov.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 80, No. 11, pp. 3-5, November, 1975. Original article submitted March 14, 1975.

TABLE 1. Effect of Monoamines, Injected into the Rat Amygdala, on CAR, Muscle Tone, Motor Activity, and FCR (M ± m)

Preparation	Dose (in μg base)	Latent period of CAR after 30 min (in sec)	Motor activity	Muscle-relaxing action (% of animals slipping off shaft)	Change in indices of FCR† after 30 min		
					latent period of reflex (in sec)	magnitude of reflex	number of pushes
Control	—	0,94 ± 0,05	51 ± 3,16	0	—(0,09±0,011)	+(12,9±2,8)	—(0,3±0,195)
DA	2	1,21 ± 0,08	64 ± 7,95	0	—(0,09±0,018)	+(21,1±3,0)	+(0,3±0,073)*
	5	2,51 ± 0,16*	75 ± 4,19*	0	+(0,24±0,031)*	+(42,6±6,59)*	+(1,5±0,24)*
NA	2	0,69 ± 0,03*	53 ± 3,16	0	—(0,1±0,025)	+(14,7±3,47)	—(0,3±0,07)
	5	2,24 ± 0,13*	33 ± 2,49*	0	+(0,28±0,043)*	—(20,4±3,198)*	—(1,5±0,288)*
5-HT	2	1,2 ± 0,06	63,2 ± 5,54	0	—(0,09±0,017)	+(21,7±4,67)	+(0,6±0,157)*
	5	2,33 ± 0,17*	73,4 ± 4,2*	0	+(0,22±0,032)*	+(46,8±6,57)*	+(1,4±0,27)*

*Differences from control statistically significant ($P < 0.05$).

†+and − represent increase and decrease respectively in indices of FCR recorded.

a microinjector. The effect of the monoamines was estimated 30 min after their injection. Bidistilled water (5 μl) was injected into the amygdala of the control rats. At the end of the experiments the brain was removed and examined histologically.

EXPERIMENTAL RESULTS AND DISCUSSION

Microinjection of 2 μg NA into the amygdala had different effects on the positive and negative conditioned reflexes in the rats. In this dose, NA did not alter the muscle tone or the spontaneous motor activity (SMA) of the experimental animals, but facilitated CAR, shortening the latent period of the reflex. Meanwhile, NA had no significant effect on the latent period, number of pushes, and magnitude of the FCR (Table 1). In a dose of 5 μg, NA increased the latent period of CAR and inhibited FCR, as shown by a marked decrease in the number of pushes and in the magnitude of the food-getting reflex. The depriming effects of NA on conditioned reflexes of varied biological modality are not specific but are evidently brought about through the inhibition of the motor responses of the experimental animals. In a dose of 5 μg, NA in fact inhibited the SMA of the rats.

The results indicate differences in the neurochemical mechanism of the dorsomedial amgydala responsible for conditioned reflexes of different types. The participation of NA is evidently not important in the mechanism of reflexes to biologically positive stimuli. At the same time, this amine facilitates excitation of a biologically negative modality, producing a CAR at the level of the dorsomedial zones of the amygdala. This conclusion is supported by data showing that adrenergic systems of the amygdala participate in the activation of the trigger mechanism of aggressive behavior [1] and the modulating effect of NA on the transmission of excitation in the rat amygdala from a system of "negative" reinforcement [2].

Microinjection of DA and 5-HT (2 μg) into the amygdala did not alter SMA of the rats, muscle tone, or the latent period of CAR. In this dose, these amines likewise did not affect the magnitude or latent period of the FCR, but they increased the number of pushes. As a result of injection of 5 μg DA and 5-HT the latent period of CAR was lengthened appreciably and the latent period of FCR was increased. Unlike NA, the depriming effect of these amines on conditioned reflexes of different types in rats is specific, for SMA of the animals increased clearly after microinjection of 5 μg DA and 5-HT (Table 1). It might be supposed that the increase in the latent period of FCR produced by DA and 5-HT was connected with inhibition of food motivation. However, this is contradicted by the fact that microinjection of the amines into the amygdala produced a significant increase in the number of pushes and in the magnitude of the conditioned food-getting reflex. Both parameters reflect an overall increase in the strength and number of the animal's movements to the conditioned stimulus and, consequently, they can be regarded as indices of motivated excitation of the animal [9]. The results described suggest that afferent excitation, reflecting information of different biological importance, is transmitted through dopaminergic and serotoninergic neuronal systems of the amygdala, which participate in the modulating effect of the bioamines on conditioned defensive and food-getting reflexes in rats. The ability of 5-HT, when injected into the amygdala, to inhibit the performance of conditioned-reflex responses [12], supports this conclusion.

LITERATURE CITED

1. L. Kh. Allikmets, in: Proceedings of Symposia of the 24th All-Union Conference on Problems in Higher Nervous Activity, Celebrating the 125th Anniversary of I. P. Pavlov's Birth [in Russian], Moscow (1974), p. 39.

2. V. P. Belyi, "Neurochemical analysis of responses of amygdalar neurons to excitation of brain structures of "positive" and "negative" reinforcement," Author's Abstract of Candidate's Dissertation, Moscow (1973).
3. J. Bureš, M. Petran, and I. Zachar, Electrophysiological Methods of Investigation [in Russian], Moscow (1962).
4. L. K. Danilova, Zh. Vyssh. Nerv. Deyat., No. 2, 253 (1972).
5. L. I. Kotlyarevskii, Zh. Vyssh. Nerv. Deyat., No. 5, 753 (1951).
6. N. V. Makarenko and V. A. Troshikhin, Fiziol. Zh. (Ukr.), No. 6, 832 (1965).
7. N. F. Suvorov, Zh. Vyssh. Nerv. Deyat., No. 3, 497 (1974).
8. K. V. Sudakov, Biological Motivations [in Russian], Moscow (1971).
9. A. N. Talalaenko, Byull. Éksp. Biol. Med., No. 4, 3 (1973).
10. S. A. Chepurnov and V. P. Belyi, Neirofiziologiya, No. 4, 407 (1974).
11. V. G. Shalyapina and G. Telegdi, Zh. Vyssh. Nerv. Deyat., No. 1, 104 (1972).
12. K. Fuxe, T. Hökfelt, and U. Ungerstedt, Internat. Rev. Neurobiol., 13, 93 (1970).
13. D. W. Straughan and K. F. Legge, J. Pharm. Pharmacol., 17, 675 (1965).