ADRENOCORTICAL FUNCTION IN RATS ADAPTED TO THE EXPERIMENTAL ENVIRONMENT DURING PASSIVE AVOIDANCE LEARNING

N. A. Kulikova and V. G. Kassil'

UDC 612.833.81-08:612.453

KEY WORDS: corticosterone; adaptation; passive avoidance learning; memory.

There is little information on the state of adrenocortical function during defensive conditioning, and what there is has been obtained entirely by the use of a model of active avoidance of a nociceptive stimulus [5, 7, 13].

In the investigation described below the concentration of 11-hydroxycorticosteroids (11-HCS) was studied in the blood plasma of rats during formation and extinction of a conditioned passive avoidance reflex (CPAR). Since factors such as handling the animals and putting them in a new environment have an activating effect on the adrenal cortex [4, 5, 8-12], the rats were preadapted to the experimental conditions.

EXPERIMENTAL METHOD

Experiments were carried out on 166 male Wistar rats aged 2 months from the nursery of the Pavlov Institute. CPAR was formed by the method of Bureš and Burešova [6] in the following modification. As a first step, daily for 9 days the rate were placed for 3 min in a large, lit chamber from which they went of their own accord through a circular hole into a small dark experimental chamber (inborn preference for dark and small places). On the 8th day after the rat had stayed 3 min in the small chamber the opening was closed and 20 volleys of ac pulses (50 Hz, 1 mA, duration of volley 0.5 sec) were applied in the course of 1 min to the wire mesh floor. CPAR was considered to have developed if during the subsequent experiments the rat, placed in the large chamber, either did not go into the small chamber, or stayed there not longer than 15 sec [2]. The presence of CPAR was tested 1 and 5 days after electrodermal stimulation (EDS). Adapted rats not subjected to EDS were used as controls. In the group of unadapted animals the experiment with EDS was not preceded by preliminary adaptation of the rats to the experimental conditions. The 11-HCS concentration was determined fluorometrically [1] in blood plasma obtained after decapitation of the rats in the animal house (basal level), 15 min after EDS, and after a stay of 3 min in the small or large (with CPAR preserved) chamber. The experimental results were subjected to statistical analysis by Student's t test.

EXPERIMENTAL RESULTS

As Table 1 shows, in rats placed for the first time in the experimental chamber (unadapted) the 11-HCS concentration was significantly higher than the basal level ($P < 0.01$). This rise was so great that it did not differ significantly from that induced by EDS ($P > 0.1$). The role of novelty of the situation as a powerful stress factor [11, 12] and the appropriateness of the preliminary adaptation of the animals to the experimental conditions which we suggested during CPAR were thus confirmed.

Adaptation of the rats for 7 days was shown not only to lower the basal 11-HCS level ($P < 0.02$), but also to lead to a more marked hormonal response to EDS compared with that to saying in the small chamber ($P < 0.001$). It must be pointed out that 24 h after electrical stimulation the basal 11-HCS level in the adapted animals was raised, but it likewise did not differ from that in the unadapted rats. The relatively unstable effect of adaptation for 7 days will be evident, for only 5 days after its end the basal 11-HCS level and their con-

Laboratory of Ontogeny of Higher Nervous Activity, I. P. Pavlov Institute of Physiology, Academy of Sciences of the USSR, Leningrad. (Presented by Academician of the Academy of Medical Sciences of the USSR N. P. Bekhtereva.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 93, No. 4, pp. 6-7, April, 1982. Original article submitted July 22, 1981.

0007-4888/82/9304-0385$07.50

TABLE 1. Plasma 11-HCS Concentration (in μg %) in Rats at Various Times after Adaptation to Experimental Situation and CPAR (M ± m)

Experimental conditions	Unadapted animals	Adapted (for 7 days) animals				
		after period of adaptation		after EDS		
					5 days	
		1 day	5 days	1 day	24 h after absence of CPAR	24 h after presence of CPAR
Control (basal level)	25,0±3,05 (8)	15,5±1,63 (8)	22,9±2,34 (7)	26,9±2,03 (17)	26,4±6,59 (5)	28,4±6,67 (5)
After staying 3 min in experimental chamber:						
small	44,3±4,55 (8)	24,7±2,27 (7)	37,0±5,44 (6)	44,9±2,90* (20)	37,5±3,44* (13)	31,1±5,53* (8)
large (CPAR present)	—	—	—	33,8±3,23 (21)	—	36,8±2,54 (20)
15 min after EDS	55,2±4,66 (5)	41,9±2,64 (8)	—	—	—	—

Legend. *) Absence of CPAR; numbers of animals shown in parentheses.

centration in the blood plasma after the rats had stayed in the small chamber were comparable with the characteristic values for unadapted animals.

A period of adaptation thus evidently affects not only the 11-HCS level, but also CPAR and its retention. In the absence of adaptation, 24 h after a single application of EDS 80-90% of the rats exhibited CPAR, which lasted for 3 weeks [3]. In our own experiments, the presence of CPAR was observed 24 h after EDS in only 55% of rats, whereas 5 days after EDS it was present in 71% of animals exhibiting PAS on the first day.

Very significant differences in the 11-HCS concentration were found 24 h after EDS in rats exhibiting and not exhibiting CPAR. In animals with CPAR (i.e., not visiting the small chamber) the 11-HCS level did not differ from the basal level ($P > 0.05$) and, a matter to which special attention must be paid, it was considerably lower than in rats with absence of CPAR ($P = 0.001$). Differences in the corticosteroid concentration between the two groups of animals disappeared 5 days after EDS.

In our opinion the absence of elevation of the 11-HCS level in the plasma of rats with CPAR as early as 24 h after EDS is due to the absence of a negative emotional reaction to the experimental situation. The marked increase in the corticosteroid concentration in animals not exhibiting the reflex, i.e., animals which visited the small chamber, can be explained by fear evoked by the memory of nociceptive stimulation received in that chamber. If CPAR is used as a model of long-term memory, both the behavioral and emotional responses, which are not always parallel in their manifestation, must evidently be taken into account.

LITERATURE CITED

1. Yu. A. Pankov and I. Ya. Usvatova, Publications on New Apparatus and Methods, First Moscow Medical Institute [in Russian], Vol. 3, Moscow (1965), p. 137.
2. V. K. Fedorov, M. KH. Sitdikov, and N. V. Shiryaeva, Zh. Vyssh. Nerv. Deyat., 22, 624 (1972).
3. N. V. Shiryaeva, "Some physiological and biochemical features of passive avoidance learning," Author's Abstract of Candidate's Dissertation, Leningrad (1974).
4. R. Ader and S. B. Friedman, Neuroendocrinology, 3, 378 (1968).
5. J. R. Basset, K. D. Cairncross, and M. G. Kind, Physiol. Behav., 10, 901 (1973).
6. J. Bureš and O. Burešova, J. Comp. Physiol. Psychol., 56, 268 (1963).
7. K. D. Cairncross, A. Wren, C. Forster, et al., Pharm. Biochem. Behav., 10, 355 (1979).
8. J. Dunn, L. Scheving, and P. Milliet, Am. J. Physiol., 223, 402 (1972).
9. J. W. Hennessy, R. Levin, and S. Levine, J. Comp. Physiol. Psychol., 91, 770 (1977).
10. Z. Malatova, S. I. Ahler, and M. Praslička, Biologia (Prague), 33, 195 (1978).
11. H. P. Pfister, Physiol. Behav., 23, 649 (1979).
12. H. P. Pfister and M. G. King, Physiol. Behav., 17, 43 (1976).
13. I. Pollard, J. R. Basset, and K. D. Cairncross, Aust. J. Biol. Sci., 32, 237 (1979).