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BLOOD CORTICOSTERONE LEVEL IN KRUSHINSKII-MOLODKINA AND WISTAR RATS AFTER SHORT EXPOSURE TO ACOUSTIC STIMULATION

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The effects of acoustic stimulation on rodents have been widely studied by many specialists. The epileptogenic action of sound has received the most attention. Lines of mice and rats particularly sensitive to acoustic stimulation and responding to it by epileptiform fits have been bred [1]. One such line is the Krushinskii-Molodkina (KM) line. Rats of this line respond to short acoustic stimulation by a strong clonicotonic fit. Long exposure to acoustic stimulation leads to severe functional disturbances and often to death of the animal. The high sensitivity of KM rats to sound suggests that this stimulus acts as a stress factor for these animals. Systemic protective-adaptive processes, largely controlled by hormones of the pituitary-adrenal system, are known to be a very important component in the development of stress reactions [7].

In the investigation described below the functional state of the adrenals was investigated in rats exposed to short acoustic stimulation.

EXPERIMENTAL METHOD

Male KM rats, which in 98-99% of cases reacted to acoustic stimulation by clonicotonic fits, and Wistar rats, insensitive to sound, were used in the experiments.

The experimental animals were exposed to the loud ringing of a bell (about 110-115 dB) for 1.5 min. The sensitivity of the rats to sound was assessed on a four-point scale [1].

The state of adrenal function was studied by determining changes in the blood corticosterone level of the rats in response to acoustic stimulation. Blood was taken from the animals by decapitation 5, 15, 30, 60, and 120 min after acoustic stimulation. Intact animals, not exposed to acoustic stimulation, were used to determine the initial blood hormone level.

The corticosterone concentration in the blood plasma was determined by competitive protein-binding analysis using cortisol-1,2-H^3 with rat serum transcortin as binding protein [6]. Different types of stress have been characterized previously on the basis of information on the blood glucocorticoid concentrations of experimental animals: stress associated with immobilization, laparatomy, laser irradiation, and during the formation of the structure of the society [2-4]. This method of assessing the state of function of the pituitary-adrenal system is highly sensitive and it adequately reflects the level of the protective-adaptive reactions in states of stress.

EXPERIMENTAL RESULTS

The plasma corticosterone level of intact Wistar rats did not differ significantly from that of intact KM rats (Table 1). As a result of exposure for 1.5 min to acoustic stimulation 13% of the Wistar rats developed motor excitation and a fit assessed at 2 points on average, actually during exposure to the stimulus. The remaining 87% of Wistar rats were absolutely insensitive to sound (0 point). The KM rats reacted in 99% of cases to the acoustic stimulus with a powerful epileptiform fit assessed at 4 points.

The blood corticosterone concentration of Wistar rats insensitive to sound was increased 30 min after exposure, but 1 h after acoustic stimulation it returned to its initial level (Table 1). The increase in the blood corticosterone concentration in rats insensitive to sound suggests that acoustic stimulation evokes a short-term stressor effect in these animals.

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TABLE 1. Effect of Short Exposure to Acoustic Stimulation on Blood Corticosterone Concentration (in μg%) in Male KM and Wistar Rats (M ± m)

Line of rats	Initial value	Time after acoustic stimulation, min				
		5	15	30	60	120
Wistar: insensitive to sound	8,33±0,67 ($n = 5$)	6,90±0,35 ($n = 7$) $P_1 > 0,05$	7,03±0,40 ($n = 8$) $P_1 > 0,05$	14,87±0,89 ($n = 8$) $P_1 < 0,001$	9,58±0,70 ($n = 6$) $P_1 > 0,05$	5,35±0,71 ($n = 5$) $P_1 < 0,02$
Sensitive to sound		9,43±0,70 ($n = 8$) $P_1 > 0,05$ $P_2 < 0,01$	8,70±0,79 ($n = 6$) $P_1 > 0,05$ $P_2 > 0,05$	14,37±1,29 ($n = 6$) $P_1 < 0,01$ $P_2 > 0,05$	8,30±1,09 ($n = 6$) $P_1 > 0,05$ $P_2 > 0,05$	7,38±1,47 ($n = 5$) $P_1 > 0,05$ $P_2 > 0,05$
KM	6,92±0,65 ($n = 5$) $P_1 > 0,05$	9,46±0,57 ($n = 8$) $P_1 < 0,02$ $P_2 < 0,01$ $P_3 > 0,05$	11,33±0,56 ($n = 6$) $P_1 < 0,001$ $P_2 < 0,001$ $P_3 < 0,05$	23,83±0,50 ($n = 14$) $P_1 < 0,001$ $P_2 < 0,001$ $P_3 < 0,001$	21,73±0,71 ($n = 5$) $P_1 < 0,001$ $P_2 < 0,001$ $P_3 < 0,001$	5,41±1,13 ($n = 6$) $P_1 > 0,05$ $P_2 > 0,05$ $P_3 > 0,05$

Legend. P_1) Compared with data obtained before acoustic stimulation, P_2) compared with data for Wistar rats insensitive to sound, in the same period, P_3) compared with data for Wistar rats sensitive to sound, in the same period; n) number of rats in group.

Possibly not only acoustic stimulation, but also the whole program of manipulations associated with the experiment (shifting the animals from their cages to the chamber, and so on) play a role in this case.

The blood corticosterone concentration of Wistar rats which responded to the bell by a convulsive fit did not differ significantly from the blood level of the hormone in rats insensitive to sound. It will be noted that the blood corticosterone level of the Wistar rats sensitive to sound was significantly higher only compared with animals insensitive to the sound of the bell 5 min after exposure. The absence of any significant difference in the time course of the plasma corticosterone concentration after exposure to the sound of the bell in Wistar rats insensitive to sound and in animals of the same line responding to such stimulation with a convulsive fit shows that the motor excitation in this case evidently did not give rise to substantial hormonal disturbances in the hypothalamo-hypophyseo-adrenal system.

The response of the adrenals of the KM rats to sound differed considerably from that of the Wistar rats. As Table 1 shows, 15 min after acoustic stimulation the blood corticosterone level of the KM rats was higher than in Wistar rats sensitive to sound. The blood corticosterone level of the KM rats was increased 30 min after acoustic stimulation and was much higher than the hormone level at the same time in the Wistar rats. The blood corticosterone level of the KM rats 60 min after acoustic stimulation remained at its previous high level, whereas in Wistar rats it returned to its initial value. It was only 2 h after acoustic stimulation that the blood hormone level in the KM rats did not differ significantly from the corticosterone concentration in the blood of the intact rats. The differences in the time course of the blood corticosterone concentration in the KM and Wistar rats after acoustic stimulation for 1.5 min suggest that in KM rats this type of stimulation evokes a marked stress reaction.

These results are in good agreement with data showing that convulsive fits of audiogenic epilepsy in male rats increase the functional activity of the hypothalamo-hypophyseal system in the manner of a stress reaction [5].

The results suggest that this increased sensitivity of the hypothalamo-hypophyseo-adrenal system of KM rats to acoustic stimulation is a trait that is genetically linked with the high reactivity of the animal to the epileptogenic action of this stimulus. On the other hand the possibility cannot be ruled out that the more marked and persistent stress state in the KM rats than in the Wistar rats is due to the development of a much stronger convulsive fit in the KM rats, to act as an additional stimulus for them.

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