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ROLE OF THE ADRENOCORTICAL REACTION TO PHYSICAL LOAD IN INCREASE IN THE WORKING CAPACITY OF THE BODY

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Translation of "Rol' Reaktsii Kory Nadpochechnikov na Fizicheskuyu Nagrazu v Povyshenii Rabotosposobnosti Organizma" Byulleten' Eksperimental'noy Biologii i Meditsiny, No 7, 1976, pp 774-776
The maximal duration of swimming by rats with a load of 3% of their body weight increased after 5 weeks of training. This time did not increase in animals receiving dexamethasone in the process of training. The blood corticosterone level of these rats with the maximal load increased less than that of animals trained without administration of dexamethasone.
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Moscow BYULL. EKSPERIMENTAL'NOY BIOLOGII I MEDITSINY in Russian No 7, 1976 received by editors 1 Dec 75 pp 774-776

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The maximal duration of swimming by rats with a load of 3% of their body weight increased after 5 weeks of training. This time did not increase in animals receiving dexamethasone in the process of training. The blood corticosterone level of these rats with the maximal load increased less than that of animals trained without administration of dexamethasone. (BYULL. EKSPER. BIOL., 1976, No 7, p 774).

Key words: hypophysial-adrenocortical system; physical load; training.

It is well known that under stress effects on the body the adrenocortical activity increases /16/. This also occurs during considerable physical loads /1, 8-10, 20/ which, if repeated systematically, increase working capacity. In adrenalectomized /12, 13/ or hypophysectomized /14/ animals repetition of physical loads not only fails to increase working capacity, but leads to hypoglycemia and death. Adaptation to loads is also disturbed if atrophy of the adrenal glands develops as a result of prolonged administration of DOCA /15/. According to the data of other authors, adrenalectomized animals retain the ability to adapt themselves to repeated physical loads /7, 11/, but their level of adaptation remains lower than in intact animals /16/. In adrenalectomized rats training does not increase muscular working capacity and does not increase the glycogen content in the muscles /21/.
We investigated the effect of pharmacological blocking of adrenocortical activity during training loads on the development of muscular working capacity.

Experimental method. The experiments were conducted on male rats of the Wistar line swimming at a water temperature of 32° for 5 days a week during 5 weeks. During the first week the duration of swimming was 30 minutes a day. Every successive week the duration of swimming was increased by 5 minutes a day. The rats were trained either without additional effects, or against the background of intramuscular dexamethasone injections (125 µg each) given on the first and second or fourth and fifth days of each week 2 hours before the beginning of swimming. In the dose used dexamethasone effectively blocks the adrenocortical reaction to stress /4/. The blocking effect is manifested after 1½ hours and lasts more than 4 to 8 hours /18/. The maximal working capacity was determined from the time of swimming by the animals in water at a temperature of 33-34° with a load of 3% of their body weight until complete immersion for a period of more than 10 seconds. This test was carried out 2 days after the last training. Immediately after the test the animals were decapitated. The corticosterone level in the blood plasma and adrenal gland homogenate was determined fluorometrically /39/. Thin-layer chromatography was used to purify methyl chloride extracts of plasma.

Experimental results and discussion. Prolonged training significantly increased the maximal working capacity of the rats. However, administration of dexamethasone in the process of training prevented an increase in the working capacity of the animals (table 1).

Table 1. Maximal Duration of Swimming by Rats in Water at a Temperature of 33-34° With a Load of 3% of Body Weight (Min)

<table>
<thead>
<tr>
<th>Animals</th>
<th>Number</th>
<th>Weight (in g)</th>
<th>Duration of Swimming (in min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Untrained</td>
<td>8</td>
<td>270±9</td>
<td>271±5</td>
</tr>
<tr>
<td>Trained:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>without block</td>
<td>5</td>
<td>259±12</td>
<td>320±23</td>
</tr>
<tr>
<td>block on the first and second days of each week</td>
<td>5</td>
<td>249±8</td>
<td>251±23</td>
</tr>
<tr>
<td>block on the fourth and fifth days of each week</td>
<td>5</td>
<td>257±10</td>
<td>230±28</td>
</tr>
</tbody>
</table>

The reason why the positive effect of training was absent against the background of dexamethasone administration could be either a negative effect of excess of the glucocorticoid, or blocking of the adrenocortical reaction to the training load. The absence of differences in the body weight of the trained rats receiving and not receiving dexamethasone apparently indicates that a combination of training loads with administration of dexamethasone did not lead to sharp predominance of catabolic processes.
It was shown previously that the adrenocortical activity increases during the fulfillment of training loads chiefly during the first 3 days of each week /3/. By the fourth or fifth day the body is adapted to the particular load and the need for increased adrenocortical activity disappears. An increase in the load at the beginning of each subsequent week leads to a new intensification of adrenocortical activity on the first days.

Apparently, prevention of this adaptive reaction by dexamethasone disturbs the process of increase in working capacity. At the same time, it is important to take into consideration that after administration of glucocorticoids their increased blood level continues for only about 60 minutes /17, 18/, which rules out the possibility of existence of increased blood dexamethasone concentration during a training load. It is more difficult to explain the effect of dexamethasone in rats which received it at the end of the week, when training loads were no longer accompanied by an increase in adrenocortical activity. At the same time, it has been established that the recovery period after physical loads is also accompanied by long periods of increased adrenocortical activity /2, 8/. Therefore, the effect of dexamethasone in the animals of the last group could be connected with the blocking of these adaptive reactions. However, it is not ruled out that intensified catabolism due to the peripheral action of a temporary excess of dexamethasone could also play a certain role.

The corticosterone level in the blood plasma and adrenal glands (table 2) after control swimming with an additional load increased substantially in all animals and, in particular, in the blood of rats trained without administration of dexamethasone. Evidently, administration of the latter in the process of training restricts the reactivity of the hypophysial-adrenocortical system. The importance of this fact is emphasized by the presence of statistically significant correlation between the maximal duration of swimming and the blood corticosterone level after such a load (r=0.638; P<0.05).

Table 2. Corticosterone Level in Blood Plasma and Adrenals After Swimming for Maximal Duration With Load of 3% of Body Weight (M±m)

<table>
<thead>
<tr>
<th></th>
<th>Before Test of Maximal Working Capacity</th>
<th>After Test of Maximal Working Capacity</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Plasma (in µg%)</td>
<td>Adren. glands (in µg/g)</td>
</tr>
<tr>
<td>Without block</td>
<td>17.1±1.1 (5)</td>
<td>10.0±0.9 (5)</td>
</tr>
<tr>
<td>Block:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>on the first and second</td>
<td>20.3±2.4 (5)</td>
<td>9.3±1.4 (5)</td>
</tr>
<tr>
<td>days of each week of</td>
<td></td>
<td></td>
</tr>
<tr>
<td>training</td>
<td></td>
<td></td>
</tr>
<tr>
<td>on the fourth and</td>
<td>19.4±2.2 (5)</td>
<td>7.8±1.6 (5)</td>
</tr>
<tr>
<td>fifth days of each</td>
<td></td>
<td></td>
</tr>
<tr>
<td>week of training</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Remark: Number of animals given in parentheses.
BIBLIOGRAPHY


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