ROLE OF THE ADRENOCORTICAL RESPONSE TO PHYSICAL EXERTION IN INCREASED WORKING CAPACITY OF ATHLETES

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The maximal duration of swimming by rats carrying a load of 3% of their body weight was increased after training for 5 weeks. In animals receiving dexamethasone during training, this time was not increased. The blood corticosterone level of these rats carrying the maximal load was increased less than that of animals trained without administration of dexamethasone.

KEY WORDS: pituitary-adrenocortical system; physical exertion; training.

During exposure to stress the adrenocortical activity of the body increases [16]. This occurs also during considerable physical exertions [1, 8-10, 20] which, if repeated systematically, increase working capacity. In adrenalectomized [12, 13] or hypophysectomized [14] animals repetition of physical exertions not only does not increase working capacity, but leads to hypoglycemia and death. Adaptation to exertion is disturbed also if atrophy of the adrenals develops as a result of prolonged administration of DOCA [15]. According to other workers, adrenalectomized animals can still adapt themselves to repeated physical exertion [7, 11], but their level of adaptation is lower than that of intact animals [6]. In adrenalectomized rats training does not increase muscular working capacity and does not increase the glycogen concentration in the muscles [21].

The effect of pharmacological blocking of adrenocortical activity during training exercises on the development of muscular working capacity was investigated.

EXPERIMENTAL METHOD

Male Wistar rats were made to swim for 5 days a week, for 5 weeks in water at a temperature of 32°C. During the first week the duration of swimming was 30 min each day. Every successive week the duration of swimming was increased by 5 min a day. The rats were trained either without additional treatment or with accompanying intramuscular injections of dexamethasone (each of 125µg), given on the first and second or fourth and fifth days of each week 2 h before the beginning of swimming. In the dose used, dexamethasone effectively blocks the adrenocortical response to stress [4]. The blocking effect appears after 1.5 h and lasts more than 4-8 h [18]. The maximal working capacity was determined from the length of time during which the animals could swim in water at a temperature of 33-34°C carrying a load of 3% of their body weight until complete immersion for more than 10 sec. This test was carried out 2 days after the last training. Immediately after the test the animals were decapitated. The corticosterone concentration in the blood plasma and adrenal homogenate was determined fluorometrically [19]. Methyl chloride extracts of plasma were purified by thin-layer chromatography.

EXPERIMENTAL RESULTS AND DISCUSSION

Prolonged training significantly increased the maximal working capacity of the rats. Administration of dexamethasone during training, however, prevented the working capacity of the animals from rising (Table 1).

The reason why the positive effect of training was absent when accompanied by dexamethasone administration could be either a negative effect of excess of the glucocorticoid or blocking of the adrenocortical response

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TABLE 1. Maximal Duration of Swimming by Rats in Water at $33-34^{\circ}$ C Carrying a Load of 3% of Body Weight (M \pm m)

Animals		Weight (in g)	Duration of swim- ming (in min)
Untrained Trained without block block on 1st and 2nd days of each week block on 4th and 5th days of each week	8	270±9	271±5
	5	259±12	320±23
	5	249±8	251±23
	5	257±10	238±28
		1	1

TABLE 2. Corticosterone Concentration in Blood Plasma and Adrenals after Swimming for Maximal Duration with Load of 3% of Body Weight (M \pm m)

Animals	Before test of maximal working capacity		After test of maximal working capacity	
	plasma (in µg %)	adrenals (in µg/g)	plasma (in µg %)	adrenals (in µg/g)
Without block Block: on ist and 2nd days of each week of	17,1±1,1 (5)	10,0±0,9 (5)	42,8±4,6 (5)	23,4±6,5 (5)
training on 4th and 5th days of each week of	$20.3\pm2.4.(5)$	9,3±1,4 (5)	33,0=4,3 (5)	25,2±4,0 (5)
training	19,4±2,2 (5)	7,8±1,6 (5)	31,6±4,6 (5)	25,0=3,7 (5)

Legend. Number of animals given in parentheses.

to the training load. The absence of differences in the body weight of the trained rats receiving and not receiving dexamethasone evidently indicates that a combination of training exercises with administration of dexamethasone did not lead to sharp predominance of catabolism.

It was shown previously that adrenocortical activity rises during training exercises chiefly during the first three days of each week [3]. By the fourth to 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 fresh intensification of adrenocortical activity on the first days.

Prevention of this adaptive response by dexamethasone evidently disturbs the process of increase in working capacity. It is important to bear in mind that after administration of glucocorticoids their increased blood level continues for only about 60 min [17, 18], so that the possibility of continuation of an increased blood dexamethasone concentration during training exertion is ruled out. It is more difficult to explain the effect of dexamethasone in rats which received it at the end of the week, when training exercises were no longer accompanied by an increase in adrenocortical activity. At the same time it has been shown that the recovery period after physical exertion is also accompanied by long periods of increased adrenocortical activity [2,5]. The effect of dexamethasone in the last group of animals could therefore be attributable to the blocking of these adaptive reactions. However, the possibility cannot be ruled out that increased catabolism due to the peripheral action of a temporary excess of dexamethasone could play an important role.

The corticosterone level in the blood plasma and adrenals (Table 2) after a control period of swimming with the additional load increased substantially in all animals and, in particular, in rats trained without administration of dexamethasone. Injection of dexamethasone during training evidently restricts the reactivity of the pituitary-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 concentration after such loading (r = 0.638; P < 0.05).

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AGE DIFFERENCES IN PANCREATIC INSULAR FUNCTION

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Age differences in pancreatic insular function and indices of insulin production during sugar loading tests were studied in experiments on rats. Despite hyperinsulinemia and a higher rate of insulin liberation in old animals the utilization of glucose is worsened, evidence of the development of relative insulin insufficiency. Hyperinsulinemia in old animals is accompanied by a decrease in the total blood insulin activity and an increase in the "coefficient of inactivation" of insulin. The development of relative insulin insufficiency with age may be due to both insular and extrapancreatic factors, leading to a reduction in the biological activity of insulin.

KEY WORDS: insulin; age dynamics; sugar loading.

Homeostasis is maintained during aging by definite changes in the system of neurohumoral regulation [2, 13]. Special attention is attracted to the age evolution of the pancreatic insular apparatus.

Investigations have shown [8, 9, 11] that the endocrine part of the pancreas undergoes definite changes with age; information on age changes in the insulin activity of the blood has also been published [4, 5, 10]. However, data on the age dynamics of the blood insulin level are few in number and contradictory in nature [12, 16, 17].

It was decided to study the response of the insular apparatus to glucose administration and its changes with age.

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