

NONSPECIFIC ANTIHYALURONIDASE IN THE BLOOD SERUM OF RATS WITH EXPERIMENTAL HYPER- AND HYPOTHYROSIS

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Ya. I. Dumanskii and N. B. Lutsyuk

Department of Children's Diseases (Head—Docent D. I. Ogorodnik) and Department of Hygiene (Head—Professor O. V. Perov), Ternopol' Medical Institute

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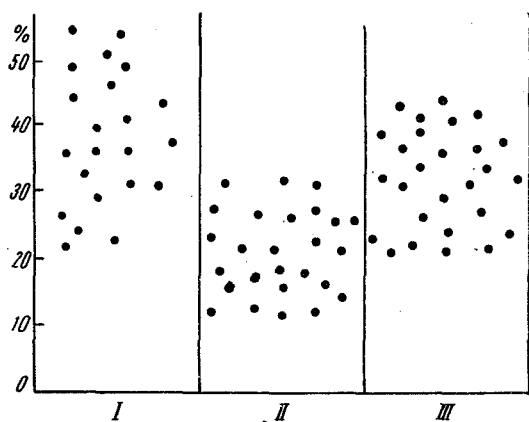
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In one of our publications [1], we studied the influence of vitamin A deficiency and A hypervitaminosis on the activity of nonspecific antihyaluronidase of rat blood serum. It was found that both excess and insufficient uptake of vitamin A by the animal organism leads to a distinct, statistically reliable increase in the nonspecific antihyaluronidase activity. The mechanism of this effect remained obscure. Noteworthy is the fact that large loads of the vitamin and vitamin A deficiency cause a distinct weakening of the function of the thyroid gland [2, 3]. The possibility remains that the action of vitamin A described on the nonspecific antihyaluronidase is connected with its influence on the thyroid gland.

There are few examples demonstrating the influence of hormones on the tissues through the corresponding enzyme systems. Of definite interest is the dependence of the hyaluronidase enzyme activity on the hormonal influences of the thyroid gland. In this work we studied the activity of nonspecific antihyaluronidase of the blood serum of white rats during artificial hypo- and hyperthyrosis.

EXPERIMENTAL

The experiments were conducted on young male white rats. All the experimental animals were kept on the mixed laboratory food. We used 75 animals, comprising three experimental groups. The animals of the first group



Indices of activity (in %) of blood serum antihyaluronidase of rats with experimental hypo- and hyperthyrosis.

I) First group ($\mu 35.7\%$, $\sigma \pm 11.4\%$, $m \pm 3.42\%$); II) second group ($\mu 21.6\%$, $\sigma \pm 6.93\%$, $m \pm 2\%$, $t 3.58$) III) third group ($\mu 32.9\%$, $\sigma \pm 8.28\%$, $m \pm 2.39\%$, $t 0.7$).

(22 rats) were controls. Experimental hyperthyrosis was induced in the animals of the second group (26 rats) by daily oral administration of 0.01 g of thyroindin per 100 g of body weight for a period of ten days. The animals of the third group (27 rats) received oral administrations of 6-methylthiouracil in doses of 10 mg per 100 g of body weight daily during a period of 10 days. Thus, the animals of the third group were in a state of artificial hypothyrosis by the end of the experiment (listlessness, lag in growth). At the end of the experiment, which lasted 10 days, the rats were bled from the large veins of the neck. The nonspecific antihyaluronidase activity was determined in the serum obtained by a viscosimetric method [4], based on the ability of blood serum antihyaluronidase to weaken the effect of hyaluronidase on hyaluronic acid in the test tube. The antihyaluronidase activity was expressed in percent (calculated according to the formula of Glick and Gollan).

RESULTS

In the animals of the control group, the indices of the antihyaluronidase activity fluctuated within rather

substantial limits (22.1-55.1%), with an average value of 35.7% (see figure). The indices of the antihyaluronidase activity in animals with artificial hyperthyrosis lay within the range 11.5-33% (average value 21.6%). The difference in the antihyaluronidase activities of the animals of the first and second groups is statistically reliable. Thus, when the amount of circulating thyroid hormone in the animal organism is increased, the activity of the nonspecific hyaluronidase inhibitor decreases. In the animals of the third group, the values of the activity of the enzyme antihyaluronidase fluctuated from 21.9 to 44.6% (average value 32.9%). The statistical treatment of the experimental results indicates the absence of any reliable differences between the indices of the enzyme activity in the animals of the first and third groups.

The data obtained on the decrease in the activity of rat blood serum nonspecific antihyaluronidase under conditions of thyroid hyperthyrosis indicate the need for considering the role of the thyroid hormones in the explanation of metabolic processes that occur in the organism, in particular, in the connective tissue. The results of the work do not confirm the hypothesis that the stimulating effect of vitamin A on antihyaluronidase is related to its inhibition of the thyroid function.

LITERATURE CITED

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