

EFFECT OF BIOTIN ON TISSUE RESPIRATION OF CERTAIN ORGANS OF ALBINO RATS WITH EXPERIMENTAL HYPERCHOLESTEREMIA

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The effect of biotin on the tissue respiration of the organs of normal animals, and more especially in pathological conditions, has been inadequately studied. For this reason experiments were carried out to study the tissue respiration of certain organs of albino rats under the influence of biotin in normal and pathological conditions. The first experiments showed that biotin stimulates the tissue respiration of all investigated organs [1]. The pathological condition chosen was experimental hypercholesteremia, in which the intensity of the tissue respiration is depressed [2].

EXPERIMENTAL METHOD

Altogether 40 experiments were carried out on 40 albino rats weighing 162-208 g. The tissue respiration was measured in the following organs: the heart, brain, liver, kidneys, spleen, and muscle tissue. The rats were decapitated, and the tissue to be investigated was quickly extracted and miced in the cold. A sample of tissue weighing 100 mg was placed in a Warburg's flask and 2 ml of Ringer's solution (pH 7.35) were added. The tissue respiration was determined by the usual manometric method in an atmosphere of oxygen at 37.5°. The index of intensity of the tissue respiration was the volume of oxygen in cubic millimeters absorbed per hour per milligram of tissue dried to constant weight.

In the experiments of series I the effect of biotin was studied on the tissue respiration of the organs in normal conditions. The biotin solution was injected subcutaneously in a dose of 0.3 μ g/100 g body weight/day for 20 days. The controls were rats kept in the same conditions but not receiving biotin. In series II experimental hypercholesteremia was caused in the albino rats by feeding them on cholesterol in a dose of 0.5 g/kg body weight with meat and fat for 120 days. Under these conditions the total cholesterol concentration in the blood serum rose on the average to 123 mg %. After the 120th day the rats began to receive subcutaneous injections of biotin in a dose of 0.3 μ g/100 g body weight once daily for 20 days. The controls were rats with experimental hypercholesteremia not receiving biotin.

EXPERIMENTAL RESULTS

Initially the tissue respiration of the organs of normal rats was studied after subcutaneous administration of biotin for 20 days. Analysis of the results obtained showed that in these conditions the absorption of oxygen by the heart in the first hour of observation increased to 18.5 ± 0.9 (compared with the normal 6.7 ± 0.46), or by 276%, absorption by the brain—to 11.6 ± 0.8 (normal 4.8 ± 0.42), or by 241%, by the liver—to 28.7 ± 1.7 (normal 5.3 ± 0.42), or by 541% by the kidneys—to 13.2 ± 1.1 (normal 8.4 ± 0.5), or by 157%, and by the spleen—to 14.2 ± 0.7 (normal 6.7 ± 0.4), or by 211% of the normal value. Similar results were obtained during the study of the intensity of the tissue respiration at the second hour of observation. The absorption of oxygen by the heart increased to 13.4 ± 0.9 (in the control 3.1 ± 0.3), or by 432%, by the brain—to 8.6 ± 0.5 (control 2.8 ± 0.1), or by 307%, by the liver—to 24.8 ± 1.5 (control 3.2 ± 0.3), or by 775%, by the kidneys—to 11.0 ± 0.8 (control 6.3 ± 0.4), or by 174%, and by the spleen—to 11.1 ± 0.7 (control 4.1 ± 0.4), or by 264% (of the initial level).

In chronic experimental conditions biotin thus stimulated the tissue respiration of all the investigated organs. The increase in the absorption of oxygen by all the organs was statistically significant.

In the next series of experiments the effect of biotin was studied on the tissue respiration of the organs of rats with experimental hypercholesteremia. In these conditions biotin stimulated the absorption of oxygen by the heart to 15.8 ± 0.4 (in the control 3.6 ± 0.08), or by 338%, by the brain—to 7.1 ± 0.4 (control 2.5 ± 0.1), or by 184%, by the liver—to 19.0 ± 0.6 (control 6.8 ± 0.1), or by 179%, by the kidneys—to 8.9 ± 0.6 (control 5.0 ± 0.4), or by 78%, by the

spleen—to 10.2 ± 0.8 (control 2.6 ± 0.1), or by 292%, and in muscle tissue—to 3.6 ± 0.32 (control 1.4 ± 0.4), or by 157%. All the differences are statistically significant.

Hence, in the conditions of experimental hypercholesteremia, biotin also stimulates the processes of biological oxidation.

LITERATURE CITED

1. V. N. Ivanov, In the book: Proceedings of the 1st Scientific Conference of Young Graduates of Chita Medical Institute [in Russian], Chita (1963), p. 87.
2. L. A. Malyshev, Vopr. med. Khimii, No. 2, 142 (1963).