ROLE OF THE HEXOKINASE REACTION IN THE INFLUENCE OF THE GLUCOCORTICOID HORMONES ON THE RATE OF RIGOR MORTIS OF SKELETAL MUSCLES

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The rate of rigor mortis depends on the intensity of glycolysis, providing for phosphorylation. Glycolytic phosphorylation, in turn, is regulated by the activity of "the least rapid enzyme"—hexokinase, under the influence of which the primary phosphorylation of hexose takes place at the expense of the labile phosphate groups of adenosine triphosphoric acid [2, 4].

Certain hormones exert an influence on the hexokinase activity: hydrocortisone inhibits the hexokinase reaction [6, 7], while insulin removes this inhibition [9]. The time of rigor mortis of the skeletal muscles is sharply lengthened after adrenalectomy, while the administration of cortisone returns it to normal [3]. Evidently, the lengthening of the time of stiffening after adrenalectomy is related to the removal of the inhibiting influence of cortisone on hexokinase.

In this work, the role of muscle hexokinase was studied in relation to the influence of glucocorticoid hormones on the rate of rigor mortis.

PROCEDURE

The experiments were carried out with white rats (males weighing 150-170 g). The adrenal glands were removed 10-15 days before the beginning of the experiments. In the postoperative period the rats were kept on a normal diet. They were given physiological saline to drink.

In each experiment an adrenalectomized and a control rat were killed by decapitation (decapitation was carried out under light ether anesthesia). Directly after decapitation, the first sample (a 1 g muscle sample) was taken from the posterior surface of the thigh muscle of the left leg. At the onset of rigor mortis (the latter was determined according to the method of L. I. Tank [5]) a second muscle sample (1 g) was taken from the hind surface of the right thigh. In the weighed samples the hexokinase activity was determined according to the method of Long [1, 8]. Glucose was determined according to the method of Hagedorn-Jensen and its decline was calculated per mg of protein contained in the extract (the protein was determined by the biuret reaction).

RESULTS OF THE EXPERIMENTS

It was established that in adrenalectomized rats, rigor mortis sets in considerably more slowly than in the control animals (Fig. 1, a, b) which is in agreement with the literature data [3]. It was shown that after decapitation, the hexokinase activity of the muscles in the adrenalectomized rats was higher than in the intact animals (Fig. 2). By the time complete rigor mortis had set in, the hexokinase activity in the adrenalectomized rats had decreased to aproximately the activity determined in the control animals in the first test. In the control animals at the maximum rigor mortis, the hexokinase activity was somewhat increased. These data were statistically reliable.

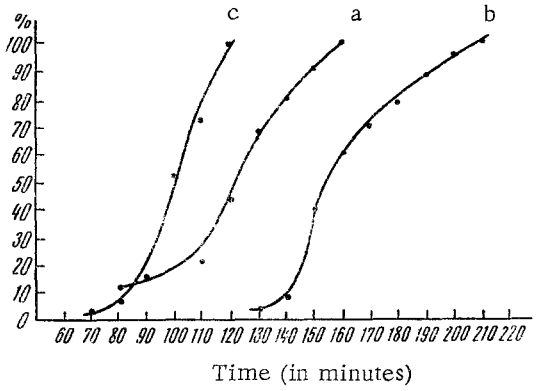

Fig. 1. Rate of onset of rigor mortis in the tails of rats. a) Control animals; b) animals subjected to adrenalectomy 10-15 days before the experiment; c) animals obtaining cortisone acetate at a dose of 4 mg/100 g intraperitoneally for three days. The curves were set up on the basis of the data obtained from 25 animals.

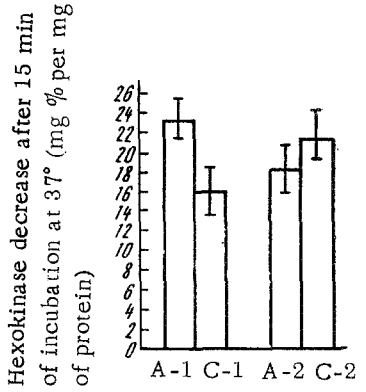

Fig. 2. Hexokinase activity in muscle extracts of adrenalectomized (A) and control (C) rats after decapitation (1) and rigor mortis (2). (There were 26 animals in each group).

Since it follows from the literature data [3] that cortisone speeds up rigor mortis both in adrenalectomized and in healthy animals, the interaction of this phenomena with the influence of cortisone on the activity of hexokinase was investigated. In this series of experiments 4 mg/100 g doses of cortisone acetate were administered intraperitoneally to 25 white rats for three days. The rats were decapitated two and a half hours after the third cortisone acetate injection. A muscle sample for the determination of hexokinase activity was taken from 10 animals immediately after decapitation. The time of stiffening of the skeletal muscles was measured as usual according to stiffening of the tail.

The data obtained in these experiments confirmed the opinion that excess glucocorticoids are capable of speeding up rigor mortis in rats in comparison with control animals, and this difference is statistically reliable (Fig. 1, c). The rate of the hexokinase reaction in a muscle pulp of rats subjected to the action of cortisone acetate was depressed in comparison with the control figures: the average value of 10 determinations for the experimental rats was 13.5 mg %, while for the controls it was 16.05 mg %, but this difference proved statistically unreliable. The causes of this phenomena are not sufficiently clear.

The detection of a higher hexokinase activity in adrenalectomized animals permits one to assume that this increased activity is responsible for the lengthening of postmortem glycolysis, maintaining the level of high-energy compounds in the muscle that is necessary for the prevention of rigor mortis.

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