The effect of anti-SC-serum on liposomes containing membranes of lymphocytes with the SC-antigen could not be explained by an antigen-antibody reaction on the surface of the liposomes, for anti-Thyl-serum did not induce such an effect for liposomes containing thymocyte membranes carrying the Thyl-antigen (Table 2), although ELISA confirmed the presence of this antigen on the liposome membrane (Table 1).

Thus membranes of SC-positive lymphocyte can effect *c*AMP transport through the artificial liposomal membrane. They may also possess a similar function when in the composition of cells. Since we know that *c*AMP is a regulator of differentiation of SC-cells [3], it can be tentatively suggested that exogenous *c*AMP can exert a marked influence on the activity of these cells, which exhibit the properties of cells regulating proliferation in various experimental systems. It can also be postulated that the SC-antigen found on lymphocyte precursor cells may participate directly in *c*AMP transport through the cell membrane of these cells.

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Na,K-ATP-ASE ACTIVITY OF INDIVIDUAL STRUCTURAL COMPONENTS OF THE GUINEA PIG VISUAL SYSTEM DURING HYPOXIA

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The study of the ATPase systems in various organs and organelles during hypoxia has attracted great attention [2, 4, 7-9, 12]. The increased interest in this problem in recent years is connected with the fact that hypoxia leads to ATP deficiency and to disturbance of ionic gradients, which occupy a special place in pathogenetic injury to biological membranes [5]. Nevertheless, there are no data as yet on changes in ATPase activity in individual structural components of the visual system during hypoxia. These data are essential for our understanding of the physicochemical mechanism of cell damage in the visual system during exposure to hypoxia, for depression of the components of the electroretinogram (ERG) and of the visual cortical evoked potential in various types of hypoxia has been established [3, 10, 13, 14].

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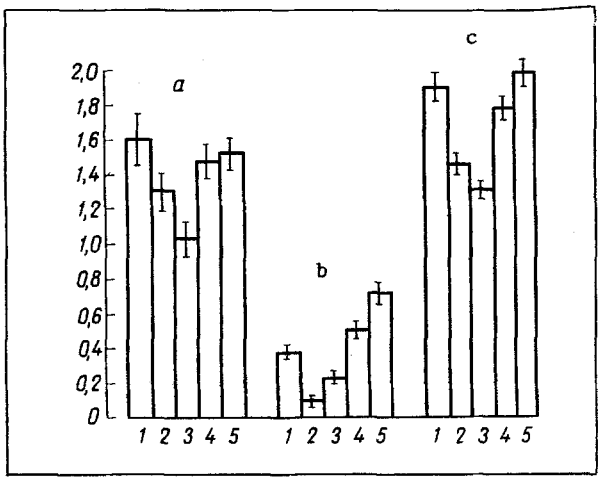

Fig. 1. Changes in Na,K-ATPase activity in individual structural components of guinea pig visual system during hypoxia: a) retina, b) PE, c) VC. 1) Control, 2) hypoxia, 3) hypoxia + reoxygenation, 4) hypoxia + vitamin E, 5) hypoxia + reoxygenation + vitamin E. Ordinate, specific Na,K-ATPase activity (in μmoles P_i/mg protein/h).

Considering that the system of active Na^+ transport plays a role in the formation of the receptor potential of the retina [15], it can be postulated that a disturbance of the work of the Na,K-pump is one cause of depression of electrical responses during hypoxia. Accordingly, the aim of the present investigation was to study activity of Na,K- and Mg-ATPases in the retina, the pigmented epithelium (PE), and the visual cortex (VC) in acute hypoxia.

EXPERIMENTAL METHOD

Experiments were carried out on 62 guinea pigs. Acute hypoxia was produced by gradual replacement of the air in an airtight chamber with a capacity of 0.12 m^3 by nitrogen in the course of 14-16 min. Reoxygenation took place by blowing oxygen into the chamber under low pressure from a cylinder in the course of 15 min. Vitamin E was injected intramuscularly in a dose of 300 mg/kg 16 h before exposure to hypoxia. Immediately after hypoxia and hypoxia with reoxygenation the animals were decapitated and the retina, PE, and VC were removed. Activity of Na,K- and Mg-ATPases was determined as in [6]. Total protein was determined by the method in [11].

EXPERIMENTAL RESULTS

The experiments showed that specific Na,K-ATPase activity was highest in VC, namely 1.94 ± 0.11 μmole P_i/mg protein/h, rather lower in the retina (1.6 ± 0.18 μmole/mg/h), and much lower in PE (0.38 ± 0.061 μmole/mg) (Fig. 1). The same rule also was observed with Mg-ATPase. The highest specific activity was found in VC, namely 3.92 μmoles/mg/h, the retina had lower activity (2.75 ± 0.24 μmole/mg/h), and the lowest activity of all was found in PE (2.28 ± 0.21 μmole/mg/h. The ratio between activities of Mg- and Na,K-ATPases was highest in PE (4 times), almost equal in VC (twice) and the retina (1.8 times).

The action of acute hypoxia in all the structures studied led to depression of Na,K-ATPase activity. However, the sensitivity of the enzyme to hypoxia differed in different tissues (Fig. 1): the greatest degree of inhibition of enzyme activity was found in PE (more than threefold, $p < 0.001$) (in individual animals complete suppression of activity of the enzyme was observed), a weaker degree of inhibition was observed in VC (26%, $p < 0.05$), and the weakest degree of all in the retina (18%, $p > 0.05$).

The effect of hypoxia on Mg-ATPase was heterogeneous in character: it either inhibited or activated the enzyme in all three structures.

Much evidence has now been obtained for potentiation of the damaging action of hypoxia after reoxygenation, evidently due to the more significant intensification of lipid peroxidation (LPO) [5]. With this in mind, we studied changes in Na,K-

ATPase activity in individual tissues of the visual system after reoxygenation of guinea pigs for 15 min after a previous exposure to acute hypoxia.

As the results show, reoxygenation after acute hypoxia led to greater depression of Na,K-ATPase activity in the retina and VC. By contrast with those structures, activity of the enzyme in PE was partially restored during reoxygenation, evidently due to the structural and functional characteristics of this tissue.

Preliminary administration of vitamin E to the animals almost completely prevented the depression of Na,K-ATPase activity in the retina and VC, whereas in PE activity of the enzyme exceeded the control level. Incidentally, administration of vitamin E to the control animals caused no significant changes in ATPase activity.

Though much evidence has accumulated in the literature for depression of ATPase activity in various organs and organelles during hypoxia and ischemia, the physicochemical mechanism of this process is still unknown. It is considered most frequently that intensification of LPO in the membrane [4], a universal damaging factor in hypoxia and ischemia, is the cause of depression of activity of these enzymes during exposure to hypoxia. Some workers, however, have observed the absence of correlation between changes in ATPase activity and accumulation of LPO products during hypoxia [7]. Analysis of existing data suggests that contradictions in results relating to this problem are evidently connected with differences in the objects and experimental conditions used, whereas correlation between the intensification of LPO and depression of ATPase activity is undisputed.

Comparative analysis of the results showing changes in Na,K-ATPase activity and the electrophysiological data on depression of the amplitude of individual components of ERG and the evoked potential of VC in hypoxia [3, 10, 13, 14] demonstrates clear correlation between these processes and improves our understanding of the mechanism of hypoxic damage. In particular, there is evidence of high sensitivity of the cells of PE to hypoxia, expressed as significant depression of the amplitude of the *c*-wave of the ERG [10, 14]. We also found that hypoxia induces a more significant degree of depression of Na,K-ATPase activity in PE than in the other components of the visual system (Fig. 1). Similar correlation was observed in VC and the retina between the decrease in Na,K-ATPase activity and depression of components of the ERG and the evoked potential of VC [3]. Taking these facts into consideration it can be postulated that depression of the work of the Na,K-pump is one cause of depression of the electrical responses in individual structural components of the visual system in hypoxia.

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