Besides inhibition of GK activity, activity of GPD-1 and GPD-2 in the brain tissue was inhibited by 52 and 14% respectively. Depression of GK and GPD-2 activity in the brain was accompanied by a marked fall in the brain GP reserves. Under these circumstances the decrease in GPD-1 activity was assessed as compensatory preservation of the reserves of GP – the original compound in reactions of phosphatide synthesis.

An increase in GPD-1 activity (by 40%) in the liver tissue in all probability was dependent on intensification of the processes of gluconeogenesis in that organ in diabetes, whereas activation of GPD-2 was evidently directed toward replenishing the GP reserves in response to the inhibited state of GK.

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HYDROPEROXIDES OF FATTY ACIDS, FLUORESCENT PRODUCTS, AND TOCOPHEROL CONCENTRATIONS IN TISSUES OF RABBITS AFTER EXPOSURE TO COLD

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KEY WORDS: hydroperoxides of fatty acids; fluorescent products; tocopherol; peroxidation of lipids; liver; lungs; erythrocytes.

The study of the molecular mechanisms of the harmful action of cold on biological tissues is an essential condition for the understanding of the etiology and pathogenesis of diseases arising during or made worse by the action of cold.

Being an essential component of practically every form of stress, including cold stress, hydroperoxides of fatty acids formed during reactions of free-radical oxidation (FRO) of lipids, may act as agents disturbing the molecular organization of cell membranes [9] and may contribute to the onset of pathological changes. An indicator of such disturbances is the appearance in the tissues of fluorescent compounds, polymerization products of a protein–lipid complex [2]. The appearance of fluorescent compounds reflects profound structural and functional disturbances of cell membranes and of oxidative processes in the cells and tissues as a whole.

The object of this investigation was to study the possible role of FRO reactions of lipids in the development of destructive processes in the lungs of rabbits exposed to low temperatures.

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TABLE 1. Concentration of Hydroperoxides, Fluorescent Products, and Tocopherol in Lung and Liver Tissue Homogenates from Animals Exposed to Cold (M ± m)

Group of animals	Number of experiments	Tissue homogenate of lungs			Tissue homogenate of liver		
		E_{233}	fluorescent products, relative units	tocopherol, mg/100 g	E_{233}	fluorescent products, relative units	tocopherol, mg/100 g
1) intact	21	0,025±0,001	15,3±1,0	1,63±0,15	0,079±0,003	13,8±1,0	3,07±0,13
2) 15-th day of experiment	11	0,052±0,002*	18,00±0,08*	2,36±0,20	0,140±0,008*	22,0±2,7*	3,74±0,30
3) 27-th day of experiment	15	0,065±0,002*	20,70±0,17*	4,12±0,40	0,29±0,01*	16,3±0,8	1,94±0,15*

*P < 0.05 compared with control.

Legend. Here and in Table 2: E_{233} stands for optical density at 233 nm.

TABLE 2. Peroxidation of Lipids (POL) and Tocopherol Concentration in Lipids of Rabbit Erythrocytes (M ± m)

Group of animals	Number of experiments	POL of erythrocytes, E_{233}					Content of tocopherol, mg%
		min/M	I	5	10	15	
1) intact	35	0,060±0,003	0,086±0,003	0,098±0,004	0,117±0,007	0,126±0,005	0,75±0,06
2) 15th day of experiment	11	0,120±0,004*	0,180±0,006*	0,222±0,005	0,250±0,007*	0,258±0,007*	1,16±0,07*
3) 27th day of experiment	15	0,57±0,05*	0,60±0,02*	0,81±0,08	1,06±0,08*	1,51±0,09*	1,1±0,1*

*P < 0.05 compared with control.

Legend. I) Initial state of addition of ascorbate. [As in Russian original – Ed.]

EXPERIMENTAL METHOD

Male rabbits weighing 2-2.5 kg were used. Daily for 27 days the animals were kept in a climatic chamber for 3 h at −25°C. Before the beginning of the experiment and on the 15th and 27th days some animals were killed by air embolism. The lungs and liver were removed and perfused with cold physiological saline, after which the following parameters were recorded: hydroperoxides in tissue homogenates [6, 8], fluorescent products [7], and the tocopherol concentration [10]. Ascorbate-dependent oxidation was initiated in erythrocytes [1].

EXPERIMENTAL RESULTS

The results are given in Tables 1 and 2. They show that prolonged exposure to low temperatures led to activation of FRO reactions of lipids both in the lung and liver tissues and in the blood. This was reflected as the appearance of both intermediate (hydroperoxides) and end (fluorescent compounds) products of FRO of lipids in the tissues. It is noteworthy that activation of the FRO reactions developed against the background of a rise in the tocopherol concentration, evidence of disparity between the velocity of the reactions generating free radicals and lipid hydroperoxides and the systems of their detoxication, and especially tocopherol. An increase in the tocopherol concentration in the tissues can be regarded as a systemic reaction of the body aimed at maintaining FRO reactions of lipids in the tissues at a steady level. Activation of lipolytic processes, characteristic of stress situations and, in particular, of cold [5] was accompanied not only by an increase in the blood concentration of total lipids and free fatty acids, but also by the liberation of tocopherol into the blood stream. The principal organs concerned with the deposition of tocopherol are the brown adipose tissue and the liver. According to the results, during prolonged exposure to cold the tocopherol concentration gradually increased in the animal's lung tissues and began to fall in the liver. This evidently indicates that during exposure to cold there is a redistribution of the tocopherol concentration in the body. The need for tocopherol increased sharply not only in the body as a whole, but also, in particular, in the lungs. Evidently the important role of the lungs in the regulation of heat production [4] makes them a unique "system-forming factor" in the body during exposure to cold. If hydroperoxides of lipids can participate in the regulation of heat production processes, naturally only up to certain concentrations because of uncoupling of oxidative phosphorylation and respiration [3], excessive "processing" of hydroperoxides may be the cause of development of pathology of the membranes and lung tissue as a whole. The fall in the tocopherol concentration in the liver is evidence, on the one hand, of the ever-increasing demands of the body for tocopherol, and on the other hand, of its ex-

haustion at its depots. The sharp fall in the tocopherol level in the liver against the background of activation of FRO may facilitate the disturbance of liver function, especially of synthetic processes. During exposure to cold, a combination of adaptive reactions thus develops, systemic in character and aimed at maintaining primarily oxidation–reduction reactions in the lungs, while they perform their respiratory and nonrespiratory (temperature-regulating) functions. This evidently is the explanation of the increased vulnerability of the lungs, both in animals and in man, during prolonged exposure to cold.

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DIRECT STIMULATING ACTION OF BLOOD SERUM AND OF VITAMIN D_3 AND ITS HYDROXY-ANALOGS ON CALCIUM TRANSPORT IN THE CHICKEN SMALL INTESTINE in vitro

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Stimulation of absorption of Ca^{++} in the animal intestine is one of the more important functions of vitamin D. In the modern view [1, 8], vitamin D_3 exerts its action through biochemical conversions in accordance with the following scheme: vitamin D_3 → 25-hydroxyvitamin D_3 (25-OHD_3) → 1,25-dihydroxyvitamin D_3 (1,25-$(OH)_2D_3$) → transcription of DNA in cell nuclei of intestinal epithelium – de novo synthesis of calcium-binding protein (CaBP) → absorption of Ca^{++} in the intestine. This mechanisms of the action of the final active form of vitamin D_3, namely 1,25-$(OH)_2D_3$, is similar to the action of steroid hormones which induce in target organs the synthesis of specific proteins which perform a physiological function.

The role of CaBP, synthesized in the intestinal epithelium under the influence of vitamin D, in the process of Ca^{++} absorption has been demonstrated by many experiments [6, 17]. The present writers showed [2] that introduction of exogenous CaBP into the intestine of rachitic chicks restores their disturbed Ca^{++} transport.

However, recent evidence has been obtained [5, 14] that CaBP synthesis does not correlate with the increase in Ca^{++} transport under the influence of 1,25-$(OH)_2D_3$. It has been suggested that this steroid can directly influence the permeability of intestinal epithelial cells for the cation. The question of the action of vitamin D_3 and its analogs on Ca^{++} transport in vitro accordingly arises. One source of active metabolites of vitamin D_3, namely 25-OH_3 and 25-$(OH)_2D_3$, may be the blood serum of animals receiving vitamin D_3 [10].

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