REACTIONS OF LIPID PEROXIDATION IN THE LIVER AND LUNGS OF RATS DURING LONG-TERM ADAPTATION TO COLD

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A condition for adequate response of a living organism to a long-acting unfavorable factor is ability to maintain oxidative processes under steady-state conditions, determined by the power of antioxidant systems [5]. In stress evoked by exposure to low temperatures, just as during the action of other unfavorable factors, reactions of lipid peroxidation (LPO) in the body are activated [2, 3, 7]. During the first day of exposure to cold antioxidant activity (AOA) of the lipids rises, but later this is replaced by a fall and by an increase in the content of LPO products [3, 4]. Next follows an even greater fall in AOA, a stage of exhaustion supervenes [4], and the animals die.

The objective of this investigation was to study a problem that is still largely unexplained, namely how the reactions of LPO and AOA of lipids change when mechanisms of long-term adaptation to the action of moderately low temperatures are formed in animals.

EXPERIMENTAL METHOD

Noninbrad male rats weighing 180-200 g were kept for 49 days at 4-6°C, whereas control animals were kept at 20-22°C on the ordinary animal house diet. Investigations were carried out on the 7th, 30th, and 49th days of exposure to cold. The animals were decapitated. Lipids were extracted with a mixture of chloroform and methanol (3:1) and their AOA was determined by the method in [2]. The content of LPO products in the liver homogenate was established as the concentration of products reacting with 2-thiobarbituric acid (TBA-active products) [10], and the content of fluorescent LPO products in the lungs was determined by the method in [8]. The ability of the liver lipids to take part in the reaction of ascorbate-dependent peroxidation (ADP) was assessed from the accumulation of TBA-active products in 100 mM Tris-HCl buffer, pH 7.4, containing 0.8 mM ascorbate, 12 μM Fe^{++}, and 10% of tissue perfused with 1.15 M KCl, during incubation for 20 min at 37°C. Protein was determined as in [9].

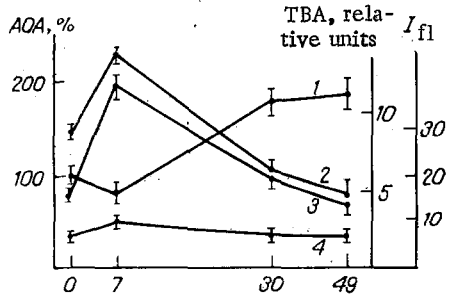

Fig. 1. Changes in intensity of LPO reactions of liver and lungs during long-term adaptation of rats to cold. 1) AOA of liver lipids (in %); 2) increase in concentration of CBA-active products in liver lipids during ADP (in relative units); 3) content of fluorescent LPO products in the lungs (in relative units); 4) content of TBA-active products in the liver (in relative units). Abscissa, time (in days).

KEY WORDS: adaptation; cold; peroxidation of lipids.

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EXPERIMENTAL RESULTS

The results showed that changes in the intensity of LPO reactions in the liver and lungs were phasic in character. As Fig. 1 shows, the intensity of LPO reactions on the 7th day of exposure to cold was increased. The content of TBA-active and fluorescent LPO products increased in the lipids of the liver and lungs. The ability of the liver lipids to take part in the ADP reaction was considerably enhanced. These changes were accompanied by some decrease in AOA of the liver lipids. On the 30th and 49th days of the experiment, i.e., during the period of the steady state of adaptation, the ability of the liver lipids to take part in the ADP reaction was considerably reduced and fell below the control level. AOA of the liver lipids on the 30th and 49th days of exposure to cold was twice as high as on the 7th day of the experiment, and was significantly higher than in the control. The content of fluorescent products in the lungs fell toward the 30th day, but it reached the control level only toward the 4th day. The greater increase in the content of LPO products in the lungs than in the liver is evidence that the respiratory system is included among those systems which are primarily damaged by exposure to cold. Considerable accumulation of LPO products may be a risk factor in the development of pathological processes in the lungs.

The results thus indicate that on the 30th and 49th day of exposure of rats to low temperatures the animal switches to a new steady-state level of oxidative reactions, characterized by high AOA of lipids. The increase in AOA is evidently determined by changes in the composition of the membrane lipids, by the degree of unsaturation of the fatty acids incorporated in them [2]. The increase in AOA leads to a decrease in viscosity of the membrane lipids, which affects the activity of membrane-bound enzymes [1] and the sensitivity of the cells to regulatory influences [6]. Activation of LPO reactions and the decrease in AOA of the lipids taking place in the initial phase of exposure to cold are known to be nonspecific components of any type of stress. They evidently can play an important role in the formation of the specialized response of the organism to a change in the external environmental conditions.

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