LIPID PEROXIDATION IN THE COURSE OF EXPERIMENTAL TOXICO-ADRENAL ENCEPHALOPATHY

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Activation of lipid peroxidation (LPO), a universal mechanism of damage to biological membranes and of metabolic disturbances [3, 8, 10], occupies an evident place in the pathogenesis of progressive degenerative diseases of the brain [1, 7]. These observations suggested that the neurohistological features of cell membrane destruction and metabolic disturbances in the brain, discovered in experimental toxico-adrenal encephalopathy (ETAE) [4] are connected with intensification of LPO.

On the basis of this hypothesis it was decided to investigate levels of thiobarbituric acid-active products (TBAP) in the blood plasma and of unsaturated fatty acids (UFA) in erythrocyte membranes, and also the peroxide resistance of erythrocytes (PRE) in dogs at different stages during the course of the ETAE.

EXPERIMENTAL METHODS

Experiments were carried out on seven mature mongrel dogs weighing 8-15 kg. ETAE was induced by the method in [4]. Blood was taken from a subcutaneous limb vein during the first half of the day before feeding, before the onset (background) and again 1, 7, 15, 30, 45, and 60 days after the beginning of ETAE. TBAP in the blood plasma were determined by the method in [9], UFA in the erythrocyte membranes by the method in [2], and PRE by the method in [6]. The results were subjected to statistical analysis.

TABLE 1. TBAP Level in Blood Plasma (in nmoles/ml), UFA Level in Erythrocyte Membranes (in nmoles/ml of suspension) and PRE (in % of hemolysis) during the Course of ETAE (M ± m, n = 7)

Experimental conditions	TBAP	UFA	PRE
Control	2,2±0,09	23,3±0,24	7,2±0,04
Duration of ETAE, days			
1	6,0±0,40*	25,1±0,33*	10,0±0,35*
7	6,4±0,51*	28,6±0,40*	12,0±0,46*
15	8,9±0,14*	29,3±0,24*	15,7±0,24*
30	8,9±0,10*	30,8±0,62*	31,9±0,44*
45	3,5±0,30*	28,7±0,14*	28,7±0,26*
60	2,8±0,13*	28,9±0,24*	12,3±0,28*

Legend. *$p < 0.05$ compared with control.

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EXPERIMENTAL RESULTS

At all times in the course of ETAE the plasma TBAP level and the UFA level in erythrocyte membranes were raised, whereas PRE was lowered (Table 1). These data, as well as data in the literature showing that PRE is directly dependent on the antioxidant supply of the membranes [6], suggest that activation of LPO is connected with inhibition of the antioxidant system. Considering that intensification of LPO in the brain is reflected in an increased lipoperoxide concentration in the peripheral blood [5], lysis of neurons observed in ETAE can be explained by activation of LPO. The absence of any direct dependence of the time course of the structural changes in the brain on values of the parameters of LPO intensity investigated at different stages of development of ETAE will be noted. Intensification of the neurohistological features of aggravation of the degenerative changes can be observed throughout a 60-day period of investigation [4]. The increase in the intensity of LPO takes place only during the first 30 days, and in the later stages a tendency is found toward normalization of the plasma TBAP, erythrocyte membrane UFA, and PRE levels. This divergence of the parameters of severity of the pathological process can probably be attributed to the inequality of the relative contribution of LPO activation to the pathochemistry of ETAE during the different periods of its course. In the early stages it probably occupies a determinant position, but later a more important role passes to other types of disturbances of brain metabolism.

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ROLE OF THE MICROSOMAL ETHANOL-OXIDIZING SYSTEM IN REGULATION OF LINOLEYL-CoA-DESATURASE ACTIVITY AFTER LONG-TERM ALCOHOLIZATION

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Long-term exposure to ethanol is characterized by various disturbances of lipid metabolism, one of which is lowering of desaturase activity [2, 10]. The fatty acid desaturase is the terminal electron acceptor in the NADH-cytochrome b_5-dependent microsomal oxidation system, and for that reason the level of activity of this enzyme is closely linked with function of this electron transfer chain. Activity of the latter is linked, in turn, with the NADPH-cytochrome P-450-dependent microsomal oxidation system which, in the opinion of some workers [10], can be attributed to the absence of high specificity of these systems for oxidation

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