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CORRELATION BETWEEN ACTIVITY OF ANTIOXIDANT SYSTEMS AND ENDOGENOUS LIPID PEROXIDATION IN THE LEFT AND RIGHT VENTRICULAR MYOCARDIUM

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It has been conclusively proved in recent years that activation of lipid peroxidation (LPO) caused by insufficiency of the antioxidant regulatory systems or excessiveness of the systems generating active forms of oxygen and lipid peroxides, plays an important role in injury to the heart muscle during stress [3] and infarction [4]. The harmful action of LPO in the muscle cell is based on inhibition of the work of the ion pumps responsible for creating and maintaining transmembrane gradients of Na^+ and Ca^{++}, and, consequently, for regulating muscle contraction [1]. This suggests that, other conditions being the same, those parts of the heart which contract more rapidly and strongly may also be characterized by greater power of their antioxidant systems, the more efficient utilization of oxygen in oxidative phosphorylation and, as a result, comparatively low intensity of release of active oxygen and LPO products. The left ventricular myocardium is known to contract faster and more strongly than the right ventricular myocardium [5].

In the investigation described below a comparative study was made of activity of antioxidant enzyme systems and the concentration of LPO products in the myocardium of the left and right ventricles in rats.

EXPERIMENTAL METHOD

Male Wistar rats weighing 180-200 g were used. Lipids were isolated by Folch's method [10]. Accumulation of lipid hydroperoxides (primary LPO products) was assessed by absorption of a solution of the lipids in a methanol: hexane (5:1 v/v) mixture at 232 nm [7], using a Perkin-Elmer (USA) spectrophotometer. The level of LPO end products (Schiff bases) was determined from the intensity of fluorescence of solutions of the lipids in chloroform at 440 nm and excitation at 360 nm [8] on the MPF-1 spectrofluorometer (Hitachi, Japan).

Superoxide dismutase (SOD) activity was estimated by the method in [11] and glutathione peroxidase (GP) activity by Emerson's method in the modification of Lankin et al. [2]. Catalase activity was determined by Luck's method [2].

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TABLE 1. Activity of Enzymes of Antioxidant System and Content of LPO Products in Myocardium of Left and Right Ventricles of Rat Heart (M ± m)

Parameter tested	Left ventricle (n = 18)	Right ventricle (n = 18)
Activity of:		
SOD, relative units	301±15	212±23*
GPI, nmoles NADH/mg protein/min	293±14	216±18**
GPII, nmoles NADH/mg protein/min	213±17	156±9*
catalase, nmoles H_2O_2/mg protein/min	306±10	211±8**
Level of:		
Schiff's bases, relative units	1±0,15	1,64±0,12**
Diene conjugates, relative units	1,05±0,09	1,56±0,15*

Legend. n) Number of animals. * $P < 0.01$, ** $P < 0.001$ compared with left ventricle.

EXPERIMENTAL RESULTS

The intensity of endogenous LPO in the left ventricular myocardium of the rats was significantly lower than in the right ventricular myocardium (Table 1). The concentrations of primary (lipid hydroperoxides) and secondary (Schiff's bases) LPO products in the right ventricular myocardium, for instance, were about 50% higher than in the left ventricular myocardium. A study of the antioxidant systems showed that SOD, GPI, GPII, and catalase activity were all significantly higher in the left ventricular myocardium: SOD by 42%, GPI by 36%, GPII by 37%, catalase by 45%.

It was suggested previously that the higher degree of inotropism of the muscle tissue of the left ventricle corresponds to a faster rate of metabolism [5]. The facts discovered in this investigation show that the intensity of endogenous LPO in the cardiomyocytes correlates negatively with the level of their functional activity.

Two mechanisms of restriction of LPO in the cardiomyocytes of the left ventricle are possible in principle: 1) reduced activity of endogenous generators of active forms of oxygen and of lipid peroxides; 2) increased activity of enzymic and nonenzymic systems responsible for maintaining a stationary concentration of active forms of oxygen and lipid peroxides.

The abundant data in the literature [5, 6, 9] show beyond all doubt that the first of these causes does not apply in the cardiomyocytes of the left ventricle. In fact, the left ventricular myocardium is characterized by a higher level of utilization of molecular oxygen and a higher concentration of electron carriers, capable of reducing it by a single electron, i.e., generating active forms of oxygen, which are LPO initiators.

It can thus be concluded that the lower steady-state level of endogenous LPO in cardiomyocytes of the left ventricle than of the right is maintained on account of increased activity of antioxidant systems and that it plays a role in ensuring the more efficient contractile function of this part of the heart.

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