DISTURBANCES IN THE LIPID PEROXIDATION SYSTEM OF BLOOD NEUTROPHILS IN PATIENTS WITH TURNER'S SYNDROME

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A characteristic feature of Turner's syndrome (TS) is premature aging, accompanied by certain biochemical disturbances and, in particular, changes affecting lipid metabolism and an increase in the frequency of hyperlipidemia [2]. Disturbances also are observed in metabolism of lipid peroxides (LP). The blood neutrophils in this disease have a high LP level and reduced activity of one enzyme of the antioxidant system, namely myoloperoxidase [1].

The aim of this investigation was to study the functional state of the lipid peroxidation (LPO) system of neutrophilic leukocytes.

EXPERIMENTAL METHOD

Venous blood with heparin was used. Neutrophils were obtained by the method described previously [1], suspended in 1 ml of 0.025 M Tris-HCl with 175 mM KCl, pH 7.4, and disintegrated in a glass homogenizer. The state of LPO was studied during oxidation of the cell homogenate by blowing air through it [3] and on the addition of adrenalin as antioxidant [4] in a concentration of $5 \cdot 10^{-4}$ M. The difference in oxidation was recorded as malonic dialdehyde (MDA) formation in the reaction with 2-thiobarbituric acid [5]. The content of total lipids was determined by a modified method in [6].

EXPERIMENTAL RESULTS

Altogether 15 patients with TS were studied, 11 of them with the 45, X karyotype, and four with chromosomal mosaicism or structural changes in the X chromosome. The patients' age varied from 17 to 40 years. Eighteen clinically healthy individuals constituting the control group were comparable with the patients in age.

The investigation showed (Table 1) that the difference in the fall in the level of LPO products on addition of adrenalin *in vitro* to homogenates of neutrophils was considerably more marked in patients with TS than in the control. The relative decrease in the MDA con-

TABLE 1. Changes in LPO System of Blood Neutrophils of Patients with TS Observed on the Addition of Adrenalin (M ± m)

Group of subjects tested	Number investigated	MDA*, μmoles/mg lipids	Relative decrease in MDA content, %
Healthy (control)	18	0,23±0,02	24,4±2,7
Patients with TS	15	2,11±0,5	36,8±3,9
P		<0,001	<0,05

Legend. Asterisk indicates difference in MDA level after oxidation of neutrophil homogenates for 1 h without and with adrenalin.

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tent (in percent) in the cells of these patients under the influence of exogenous antioxidants also was greater than in normal subjects.

Disturbances found in TS are evidence of a shift of equilibrium in the LPO system of the neutrophils, which is probably a sign of their functional insufficiency. The test with loading of the neutrophils with adrenalin revealed that activation of LPO takes place in TS, and the functional activity of endogenous antioxidants is probably depressed. This fact is in agreement with previous observations showing a decrease in the activity and thermostability of myeloperoxidase, one component of the neutrophil antioxidant system [1]. It can also be postulated that the sensitivity of the neutrophils to the exogenous antioxidant adrenalin is undisturbed in TS.

It follows from these results that the MDA level in blood neutrophils, in the test system used, was much higher in patients with TS than normally. The altered response of the cells to addition of the antioxidant in patients with TS confirms the writers' hypothesis of a functional defect of the neutrophils. The hormonal imbalance characteristic of TS, and due to an anomaly of the sex chromosomes, may perhaps have an aggravating action on this function of the blood neutrophils.

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EFFECT OF TEMPERATURE *IN VIVO* AND *IN VITRO* ON OXIDATION AND PHOSPHORYLATION IN ALBINO RAT MYOCARDIAL MITOCHONDRIA

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It is now known that cold has a significant effect on the energy metabolism of the myocardium [1, 2]. It has been suggested that this effect is based on changes in functional activity of the mitochondria as the main energy-producing structures. For instance, it has been found in experiments on mitochondria isolated from the liver of albino rats exposed to acute chilling that the rate of respiration in them in all metabolic states and the rate of phosphorylation throughout the period of cooling were raised by 24-50% during oxidation of succinate and certain other substrates [5]. Meanwhile direct cooling of heart muscle, its homogenates, or isolated mitochondria was accompanied by a considerable decrease in the rate of oxidation substrates of the Krebs cycle in all metabolic states and by inhibition of phosphorylation [8]. Dependence of respiration and phosphorylation on temperature has been studied in greater detail in liver mitochondria, and changes in that dependence have been found in muscle mitochondria during adaptation to cold [6, 7, 9-11]. As regards cardiac mitochondria, no such information is available.

The aim of this investigation was to measure oxidation and phosphorylation in mitochondria of the myocardium both when subjected to the direct action of temperature on them and during acute and chronic exposure of the whole animal to cold.

EXPERIMENTAL METHOD

Experiments were carried out on mature noninbred male albino rats weighing 250-270 g. The animals were adapted to cold at 3-4°C for 4-5 weeks. Control and cold-adapted animals

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