BIOPHYSICS AND BIOCHEMISTRY

Effects of Low-Energy Infra-Red Laser Radiation on Intact Rats

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The effects of low-energy infra-red laser radiation of varied power on hormonal and hematological status are examined in rats. It is shown that laser radiation impairs protein metabolism, the degree of impairment being dependent on the power of radiation. The use of laser radiation in nontherapeutic doses leads to hypoxia and inadequate functional activity of the insular apparatus.

Key Words: *low-energy laser radiation; hormonal metabolic status; hematological status*

Low-energy laser radiation (LELR), which exerts a variety of effects on biological structures, has been extensively used in medicine during the last decade. Infra-red radiation it preferable because of its deeper penetration into tissues.

An important feature of laser radiation is that the photoactivating effect from a small area spreads to remote areas and internal organs [6].

Although the effects of lasers on humans and animals have been extensively studied, the mechanisms of their action remain unclear. Along with various beneficial changes [1,7] LELR produces adverse effects on living systems. These effects may be caused by inadequate doses and individual differences in the susceptibility to LELR.

Our objective was to find out how LELR of varied power acts on intact rats. Hormonal status, morphofunctional characteristics of blood formed elements, acid-base status, and changes in blood electrolytes were studied in rats exposed to various regimes of LELR.

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MATERIALS AND METHODS

Experiments were carried out on 200 intact random-bred male rats weighing 200 g. Their anterior abdominal wall was irradiated once for 1 min using a Skalyar semiconductor infra-red laser in a constant mode. The radiation power (dose) was 5, 20, or 40 mW.

Blood for examination was sampled from the portal vein of Nembutal-premedicated rats at 1, 3, 5, and 7 days postirradiation.

Blood levels of insulin and cortisol were determined by radioimmunoassay. Serum levels of glucose, triglycerides, cholesterol, albumin, lactate dehydrogenase, and creatine phosphokinase were determined using a biochemical analyzer and Corning kits. Lipid peroxidation (LPO) products were quantitated by the luminescence method, and the total antioxidant activity of blood serum was evaluated by its ability to inactivate the Fe^{2+}-initiated LPO of egg yolk phospholipids. The ceruloplasmin content was measured by the method of Revin (1982) with modifications. The parameters of acid-base equilibrium and the levels of blood gases were measured in an AVL-995 gas analyzer. The contents of K, Na, and Ca were measured in an AVL-9845

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ion analyzer. Hematological parameters: leukocyte, platelet, and erythrocyte counts, hemoglobin concentration, hematocrit, and mean erythrocyte volume were determined in a cell analyzer.

RESULTS

There was no linear relationship between changes in insulin and cortisol levels after irradiation with different doses of LELR (Tables 1-3). An increase in the cortisol concentration one day after irradiation at 5 or 40 mW paralleled an increase in the triglyceride content, while the albumin and cholesterol concentrations remained unchanged. These findings suggest that cortisol produces no metabolic effect manifesting itself as an increase in the cholesterol and phospholipid contents and a decrease in the triglyceride content [8].

A tendency toward a rise of glucose and a fall of insulin levels became significant 5 days after irradiation at 5 mW and 1 day after irradiation at 20 mW. Presumably, an increase in the glucose content is associated with inadequate activity of the pancreatic insular apparatus.

There is evidence that LELR induces conformational changes in the plasma membrane proteins and, consequently, in the membrane potential and membrane sensitivity to biologically active substances [3]. Since insulin receptors are present on the plasma membrane [9], it is possible that in our experiments the functional activity of insular apparatus was impaired as a result of conformational changes in membranes.

An increase in the lactate dehydrogenase activity observed on day 3 after irradiation at 5 and 20 mW and on day 1 after irradiation at 40 mW reflects activation of anaerobic glycolysis, the rate of which had increased to compensate for inadequate catabolism. However, the functioning of the anaerobic pathway of glucose oxidation was limited due to insulin deficiency (both relative and absolute). Energy deficiency is probably compensated by intensified utilization of the intracellular substrate for the synthesis of ATP [2]. This was reflected by increased activity of creatine phosphokinase.

A slight increase in blood pH unassociated with blood gases observed after irradiation at 5 mW and 20 mW may reflect an inadequate function of the common catabolic pathway. This pathway serves mainly to supply hydrogen of organic substances to the respiratory chain. Changes in the parameters of acid-base status on day 5, the development of pronounced subcompensated acidosis in rats irradiated at 5 mW or 20 mW, and the absence of blood alkalinization in rats irradiated at 40 mW, may be due to increased contents of lactate and its metabolites (variations of these contents over time reflect the activity of lactate dehydrogenase).

All regimes of laser irradiation intensified oxygen metabolism, as evidenced by increased pO_2, percent of hemoglobin saturation with oxygen, and oxygen content in venous blood. Increased oxygenation of venous blood may be caused by reduced utilization of oxygen for oxidative phosphorylation in the mitochondria. Presumably, a decrease in the erythrocyte count and hematocrit recorded on day 3 after irradiation at 5, 20, and 40 mW and the tendency toward a decrease in hemoglobin after exposure to 5 mW, which became significant after exposure to 20 mW, reflect a compensatory response aimed at preventing excessive oxygenation.

TABLE 1. Metabolic Effects of Low-Energy Laser Radiation with a Power of 5 mW

Parameter	Intact controls	Observation period, days			
		1st	3rd	5th	7th
Cortisol, mmol/liter	48.4±5.1	90.9±8.5*	57.6±4.2**	27.9±3.1*	35.6±2.9
Insulin, μU/ml	70.8±6.8	54.9±6.3	65.5±5.7	35.2±2.8*	59.5±6.1
Glucose, mg/ml	6.3±0.51	4.8±0.35	7.3±0.93	8.0±0.95	7.7±0.81
Triglycerides, g/liter	88.8±7.8	82.0±7.8	97.5±8.5	122.4±14.7	117.8±12.2
Cholesterol, mg/ml	80.8±7.4	89.3±9.2	77.3±8.4	59.8±7.1	76.5±8.1
Albumin, mg/ml	35.3±2.7	39.6±4.2	40.2±3.1	34.2±2.8	33.3±2.5
Creatine phosphokinase, U/liter	610.6±58.7	644.2±58.3	717.9±69.2	856.3±79.4	996:8±82.4*
Lactate dehydrogenase, U/liter	495.4±43.2	411.1±35.6	1014.2±97.3*	1125.9±92.1*	1526.3±101.4*
Antioxidant activity, %	100	338.7	37.1	257.7	138.9
Ceruloplasmin, %	100	142.1	88.6	171.4	94.1
LPO products, %	100	178.3	60.4	59.4	115.1

Note. Here and in Tables 2 and 3: $p<0.05$: *compared with the control; **compared with preceding measurement.

TABLE 2. Metabolic Effects of Low-Energy Laser Radiation with a Power of 20 mW

Parameter	Intact controls	Observation period, days			
		1st	3rd	5th	7th
Cortisol, mmol/liter	48.4±5.1	44.6±4.7	38.4±4.2	26.5±3.1*	63.1±7.1**
Insulin, μU/ml	70.8±6.8	92.2±8.7	66.3±7.1	42.8±5.3*	55.3±6.2
Glucose, mg/ml	6.3±0.51	6.5±0.58	8.6±0.79	8.4±0.81	7.8±0.83
Triglycerides, g/liter	88.8±7.8	120.3±14.0	115.8±12.7	124.0±11.8	115.8±10.1
Cholesterol, mg/ml	80.8±7.4	74.3±6.8	68.4±5.9	67.3±7.2	88.2±9.1
Albumin, mg/ml	35.3±2.7	38.5±3.4	35.7±2.9	37.5±3.1	34.9±2.8
Creatine phosphokinase, U/liter	610.6±58.7	708.9±69.4	711.5±68.9	978.5±80.1*	1041.8±97.2*
Lactate dehydrogenase, U/liter	495.4±43.2	779.4±81.5	964.0±91.7*	952.7±89.7*	1496.6±107.7*
Antioxidant activity, %	100	257.1	193.1	61.1	96.0
Ceruloplasmin, %	100	132.1	132.7	97.6	74.1
LPO products, %	100	185.8	58.5	81.5	92.4

TABLE 3. Metabolic Effects of Low-Energy Laser Radiation with a Power of 40 mW

Parameter	Intact controls	Observation period, days			
		1st	3rd	5th	7th
Cortisol, mmol/liter	48.4±5.1	271.8±32.7*	44.5±5.2	20.4±3.4*	44.5±5.7
Insulin, μU/ml	70.8±6.8	45.0±3.7*	60.2±4.8	59.1±5.2	43.7±4.8*
Glucose, mg/ml	6.3±0.51	10.8±0.97*	9.4±0.82*	9.4±0.87*	8.8±0.79
Triglycerides, g/liter	88.8±7.8	122.3±10.8	121.8±11.7	143.5±10.9*	103.0±8.9
Cholesterol, mg/ml	80.8±7.4	98.1±8.7	83.2±8.2	57.7±6.2*	101.0±9.3
Albumin, mg/ml	35.3±2.7	35.3±3.1	32.5±2.9	39.9±3.7	35.7±2.8
Creatine phosphokinase, U/liter	610.6±58.7	714.5±65.7	689.2±59.2	912.9±79.8*	1189.1±97.4*
Lactate dehydrogenase, U/liter	495.4±43.2	1201.6±89.6*	714.7±67.5	1363.2±97.8*	1315.1±89.9*
Antioxidant activity, %	100	241.7	141.1	160	97.7
Ceruloplasmin, %	100	137.9	109.0	131.0	65.2
LPO products, %	100	178.3	67.0	81.1	58.4

Insufficiency of common catabolic pool associated with the absence of the metabolic effects of cortisol leads to the activation of anaerobic glycolysis and determines changes in the acid-base status, blood gases, red cell count, and hemoglobin.

Hormonal effects may be abolished as a result of damage to hormone receptors caused by LPO products [4,5]. An increase in the LPO content occurring 1 day after irradiation (irrespective of increased antioxidant activity) is a factor of cell membrane damage. Analysis of our findings revealed a transition to a hypoxic state which may be considered as a factor protecting the body against LPO products and preserving structural integrity of the cells.

This study has shown that LELR impairs metabolic processes in a dose-dependent manner, inducing hypoxia and dysfunction of the insular apparatus when applied in nontherapeutic doses.

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