Effect of Low-Intensity Laser Radiation on Working Capacity of Animals with Modified Endocrine Status

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Translated from *Byulleten' Eksperimental'noi Biologii i Meditsiny*, Vol. 124, No. 8, pp. 165-167, August, 1997
Original article submitted May 29, 1997

The endocrine factor plays an important role in the mechanism underlying the increase in working capacity of organism. Other factors also contribute to this increase.

Key Words: *hypothyroidism; hyperthyroidism; adrenalectomy; hydrocortisone; dexasone*

Although the effects of low-intensity laser radiation (LILR) on the organism have been extensively investigated, much remains unclear. There is evidence that these effects are mediated by the endocrine system, primarily, via hypothalamic—hypophyseal—thyroid and hypothalamic—hypophyseal-adrenal axises [1,2].

Previously, we showed that LILR increases working capacity of animals more than 2-fold with concomitant activation of thyroid gland and adrenals. Since thyroid and glucocorticoid hormones are involved in the maintenance and preservation of stability of organism exposed to various pathogenic factors, we decided to evaluate their role in the mechanism responsible for the LILR-induced increase in the working capacity of rats, using the models of hypo- and hyperthyroidism, adrenalectomy, and treatment with dexasone and hydrocortisone.

MATERIALS AND METHODS

Experiments were performed on 169 male outbred albino rats (initial body weight 160-180 g).

Physical load was modeled with using of an 8-track treadmill with automatically regulated track speed, which allowed us to record the time during which the animals could run and the covered "distance." In this study track speed was 20 m/min.

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An Uzor semiconductor low-intensity laser was used as a source of radiation (wavelength 0.89 μ, output capacity 4 W, pulse frequency 1500 Hz, irradiation time 8 min). The lumbosacral region was epilated and exposed to LILR.

Thyrotoxicosis was modeled by intraperitoneal injection of triiodothyronine (55 μg/100 g) for 9 days. Hyperthyroidism was identified by aggressiveness, body weight loss (20-30 g), and increased rectal temperature (1-2°C).

Control rats were injected with normal saline.

Hypothyroidism was attained by administration of laser-irradiated normal saline for 7 days and by thyroidectomy under ether anesthesia. The animals were taken in the experiment 7 days after surgery.

Hyperfunction of adrenal cortex was modeled by a single injection of hydrocortisone in a dose of 5 mg/200 g body weight 2-3 h before experiment.

Adrenal hypofunction was modeled by two methods:

— bilateral adrenalectomy under ether anesthesia 7 days before experiment;
— administration of dexasone (1 mg/100 g), which blocks the production of endogenous glucocorticoids, 2-3 h before experiment.

RESULTS

Working capacity (time of running and covered distance) decreased to a greater extent (1.5-fold, $p<$

0007-4888/97/0008-0768$18.00

0.05) after adrenalectomy than after thyroidectomy (Table 1). It should be noted that LILR increased working capacity of rats with experimental hypothyroidism to the normal level. In adrenalectomized rats LILR also increased it (2-fold, $p<0.05$) but not to the control level.

From this finding it can be concluded that the absence of the contribution of adrenal hormones to the effect of LILR on working capacity is more significant than that of thyroid hormones.

In order to gain more insight in the influences of glucocorticoid hormones it was interesting to compare the effects of LILR in adrenalectomized and dexasone-treated rats. Labor capacity of dexasone-treated was 2-fold higher than that of adrenalectomized rats and 2-fold lower that of control rats ($p<0.05$).

Low-intensity laser radiation prolonged the period of running (up to 32 min), which was 30% longer than that in the control group ($p<0.05$).

These findings suggest that not only glucocorticoids but other adrenal hormones, primarily epinephrine, are involved in the realization of the LILR effect. This suggestion requires more experimental support.

Both hyperthyroidism and administration of hydrocortisone markedly (1.5- to 1.7-fold, $p<0.05$) decreased working capacity in comparison with the control.

It should be noted that hyperthyroidism and hydrocortisone produced similar effects, judging from the time of running and covered distance. Laser radiation had no effect on working capacity of thyroidectomized rats. In hydrocortisone-treated rats, LILR prolonged the period of running 3-fold, which was 2-fold greater than that in the control group ($p<0.05$).

Consequently, changes in the organism caused by high doses of thyroid hormones block the effect of LILR on working capacity.

Since uncoupling of oxidation and phosphorylation are a manifestation of hyperthyroidism, it can be suggested that the effect of LILR on working capacity is realized if the optimal level of energy metabolism is preserved.

Presumably, this effect of thyroid hormones restricts the effect of LILR. This hypothesis is supported by the finding that LILR increases working capacity of hydrocortisone-treated rats. In fact, both thyroid hormones and high doses of hydrocortisone produce a catabolic effect, but, in contrast to thyroid hormones, hydrocortisone has no direct effect on energy metabolism.

TABLE 1. Effect of LILR on Working Capacity in Various Models ($M\pm m$)

Experimental conditions	Time of running, min	Covered distance, m
Control (n=28)	20.4±0.5	403±0.09
Laser (n=16)	56.0±0.9	1120±0.9
Thyroid gland		
Thyroidectomy (n=11)	9.5±0.9	190±1.0
Thyroidectomy+laser (n=13)	20±0.4	400±0.3
Hypothyroidism (administration of laser-irradiated normal saline, n=9)	4.9±0.7	98±0.03
Thyrotoxicosis (n=19)	14.4±1.8	288±1.9
Thyrotoxicosis+laser (n=13)	14.9±0.7	298±0.8
Adrenal gland		
Adrenalectomy (n=10)	6.2±0.7	124±0.6
Adrenalectomy+laser (n=18)	10.2±0.3	204±0.3
Dexasone (n=10)	12±1.3	240±1.2
Dexasone+laser (n=10)	31.9±2.8	638±1.7
Hydrocortisone (n=6)	12.6±1.3	252±1.3
Hydrocortisone+laser (n=6)	40±2.1	800±1.9

Thus, LILR has no effect on working capacity of rats with hyperthyroidism, normalizes it in rats with hypothyroidism and increases it in adrenalectomized and dexasone- (2-fold) or hydrocortisone-treated (3.2-fold) rats.

Although endocrine component plays a substantial role in the mechanisms underlying the LILR-induced increase in working capacity, it is not the sole factor determining this effect.

REFERENCES

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