Regulation of Lipid Peroxidation in the Retina under the Effect of Bright Light

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Changes in LPO intensity under the effect of exposure to bright light and the possibility of their correction with antioxidants were studied on rabbits with diabetic retinopathy. It was found that enhanced LPO caused by exposure to bright light in rabbits with diabetic retinopathy can be corrected with antioxidants. Phenosan potassium salt, α-tocopherol, and oxypyridine were more effective than SOD and taurine in preventing MDA accumulation. A complex of natural and synthetic antioxidants was most efficient in correcting LPO under conditions of exposure to bright light.

Key Words: *retina; LPO; antioxidants; retinopathy*

Exposure to bright light enhances free-radical lipid oxidation in photoreceptor membranes of the retina [4,5]. Previous studies showed that accumulation of LPO products in the retina is associated with its dysfunction manifesting in a decrease of electroretinogram amplitudes [4]. Since patients with eye disease are often exposed to bright light during diagnostics and treatment, these problems deserve special attention. However, the efficiency of various natural and synthetic antioxidants in correcting LPO caused by exposure to bright light in the retina of patients with retinopathy remains poorly studied. At the same time, the results of these studies can help to develop a new scheme of treatment preventing light-induced damage and to promote introduction of antioxidants into ophthalmological practice for correction of aftereffects of cataract surgery in patients with diabetic retinopathy (DR) and in patients with macular edema and intravitreal hemorrhage.

Here we studied peculiarities of correction of LPO intensity in the retina of rabbits with DR exposed to bright light.

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MATERIALS AND METHODS

The effects of natural and synthetic antioxidants on LPO intensity in the retina were studied on 62 rabbits. The animals were divided into 8 groups.

Group 1 rabbits (controls) were daily exposed to bright light (45×10^3 lux, 80-min exposure) for 10 days; in group 2 rabbits, DR was modeled and then exposure to bright light was performed as in group 1; rabbits with DR of groups 3-8 received α-tocopherol (TP), phenosan potassium salt (PP), taurine, SOD, oxypyridine (OP-6), or a complex of antioxidants, respectively. The intensity of LPO was evaluated by MDA content measured as described elsewhere [3]. Antioxidants were administered in the following doses: PP, taurine, and OP-6 in a dose of 4.0 mg dissolved in 0.3 ml physiological saline (parabulbar injection), SOD 15 Mac cord units (corresponds to 4 mg enzyme protein; parabulbar injection), and α-TP intramuscularly in a dose of 20 mg/kg body weight. DR was modeled by injection of dithizone as described previously. The data were processed statistically using Student *t* test [1].

RESULTS

In rabbits with DR, exposure to bright light (45×10^3 lux) induces considerable accumulation of MDA com-

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TABLE 1. Effect of Antioxidants on MDA Content (nmol/mg protein) in the Retina of Rabbits with DR under Conditions of Exposure to Bright Light (*M*±*m*)

Antioxidant treatment	Day of experiment							
	exposure to bright light			after exposure to bright light				
	3	5	10	20	30	40	50
Intact animals (*n*=11)	0.142±0.013	0.14±0.01	0.161±0.014	0.221±0.011	0.256±0.016	0.146±0.012	0.141±0.013
DR (*n*=9)	0.211±0.019*	0.422±0.033**	0.714±0.056**	0.511±0.012**	0.463±0.031*	0.412±0.011*	0.389±0.02*
DR+α-TP (*n*=6)	0.14±0.09^{+}	0.310±0.011^{++}	0.44±0.10^{+}	0.36±0.06^{+}	0.36±0.01^{+}	0.28±0.03^{+}	0.25±0.03^{+}
DR+PP (*n*=6)	0.12±0.01^{++}	0.26±0.02^{++}	0.40±0.04^{++}	0.32±0.02^{+}	0.31±0.01^{++}	0.20±0.05^{++}	0.20±0.04^{++}
DR+taurine (*n*=7)	0.21±0.01^{+}	0.40±0.05^{++}	0.61±0.03^{+}	0.48±0.10^{+}	0.41±0.03^{+}	0.36±0.01^{+}	0.320±0.016^{+}
DR+SOD (*n*=8)	0.19±0.02^{+}	0.36±0.10^{+}	0.56±0.02^{++}	0.43±0.02^{++}	0.36±0.02^{++}	0.31±0.01^{+}	0.28±0.01^{++}
DR+OP-6 (*n*=8)	0.17±0.01^{+}	0.33±0.04^{+}	0.50±0.02^{++}	0.38±0.03^{++}	0.30±0.04	0.28±0.01	0.275±0.020^{+}
DR+antioxidant complex (*n*=7)	0.12±0.02^{++}	0.32±0.04^{++}	0.40±0.01^{++}	0.31±0.02^{++}	0.24±0.01^{++}	0.16±0.02^{+}	0.16±0.03^{++}

Note. $^{*}p<0.05$, $^{**}p<0.01$ compared to intact animals; $^{+}p<0.05$, $^{++}p<0.01$ compared to rabbits with DR.

pared to intact rabbits. On day 12 of the experiment, MDA content in intact rabbits and animals with DR increased to 0.161±0.014 and 0.714±0.050 nmol/mg protein, respectively.

Isolated administration of antioxidant considerably suppressed MDA accumulation under conditions of light exposure. By the end of the experiment, MDA content in the retina of rabbits with DR receiving α-TP and PP was 0.25±0.03 and 0.20±0.07 nmol/mg protein, respectively. Taurine and SOD less effectively suppressed LPO in the retina compared to other antioxidants used in the experiment.

The inhibiting effect of OP-6 was comparable to that of PP: it reduced MDA content by the end of the experiment to 0.275±0.020 nmol/mg protein (Table 1). After administration of taurine and SOD, MDA content by the end of the experiment was 0.320±0.016 and 0.280±0.010 nmol/mg protein, respectively (Table 1); the efficiency of OP-6 in inhibiting LPO in the retina of rabbits with DR during light expose was also noted.

Complex administration of antioxidants produced more pronounced inhibitory effect on MDA accumulation in the retina of rabbits with DR after exposure to bright light.

By the end of the experiment, MDA content in rabbits receiving a complex of natural and synthetic antioxidants approached the level observed in intact animals (Table 1).

Our findings drove us to the following conclusions: exposure to bright light considerably increases the intensity of LPO in the retina of rabbits with DR; PP, α-TP, and OP-6 more markedly suppressed MDA accumulation in the retina than natural antioxidants SOD and taurine; the maximum suppression of MDA was observed after administration of a complex of natural and synthetic antioxidants.

These data substantiate the need of using antioxidant complex in the treatment of patients with DR.

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