Possibilities of Regulating Disorders in Functional Activity of the Retina with Antioxidants under Conditions of High-Intensity Light Exposure

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We studied the effects of natural (α-tocopherol and SOD) and synthetic (potassium phenosane and hydroxypyridine) antioxidants on the recovery of disordered parameters of the electroretinogram under conditions of high-intensity light exposure. Potassium phenosane and tocopherol acetate more effectively normalized parameters of electroretinogram in monotherapy. The use of a complex of antioxidants completely prevented the disorders in the electroretinogram parameters in retinopathy after exposure to intense light.

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

The destructive effect of high-intensity light on the retina has been noted not once [3,4,6]. It was found that accumulation of LPO products is an important factor in the development of functional retinal disorders during high-intensity light exposure [2]. Administration of antioxidant to a certain measure restored functional activity of the retina in various retinal pathologies. Suppression of LPO intensification by antioxidants under conditions of exposure to high-intensity light presumably creates conditions for prevention of functional disorders and for restoration of electrical activity of the retina [2,6].

We studied the effects of various antioxidants on the restoration of disordered electroretinogram (ERG) parameters in rabbits with diabetic retinopathy (DR) under conditions of exposure to high-intensity light.

MATERIALS AND METHODS

The effects of natural and synthetic antioxidants on the formation of ERG parameters under conditions of high-intensity light exposure were studied on 36 rabbits. Biopotentials of the eye were recorded through silver chloride electrodes fixed in the lens and connected to the amplifier assembled on a KP544UD integral microcircuit and recorded by an S1-69 oscillographer.

The following antioxidants were used: α-tocopherol (α-TP), potassium phenosane (PP), taurine, SOD, and hydroxypyridine (HP-6). Experimental animals were divided into 8 groups. Group 1 (controls) were intact rabbits daily (10 days) exposed to an 80-min illumination of 45×10^3 lx intensity. In group 2 animals, DR was induced, after which they were exposed to 45×10^3 lx light, similarly as in group 1. In groups 3-8, the rabbits were subjected to the same procedures as in group 2 and were treated with α-TP, taurine, SOD, HP-6, or antioxidant complex, respectively.

Diabetic retinopathy was reproduced by inducing experimental diabetes as described previously [1].

Antioxidants were injected every other day for 1 month: α-TP in a dose of 20 mg/kg, PP, HP-6, and taurine in a dose of 4 mg intramuscularly, and SOD in a dose of 15 McCord units parabulbarly. The data were statistically processed using Mann–Whitney's *U* test.

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TABLE 1. Effects of Antioxidants on ERG Values in Rabbits with Retinopathy under Conditions of High-Intensity Light Exposure

ERG waves, μV		Days of light exposure			Days after light exposure			
		1	5	10	15	20	30	40
Intact rabbits (*n*=3)	a-wave	17.1±1.4	3.0±0.3	0	0	0	8.9±0.7	21.0±1.2
	b-wave	205.2±8.3	90.5±3.5	46.2±3.0	0	0	39.2±3.6	121.2±7.8
Rabbits with DR (*n*=3)	a-wave	13.1±1.1*	20	0	0	0	3.1±0.2**	9.1±1.2**
	b-wave	43.3±1.7**	0	0	0	0	10.2±0.6**	22.2±1.3**
α-TP (*n*=5)	a-wave	16.9±1.8^{+}	0	0	0	0	6.8±0.3^{++}	17.1±1.2^{++}
	b-wave	78.9±7.1^{++}	10.8±1.2^{++}	0	0	0	28.8±2.5^{++}	58.7±6.7^{++}
PP (*n*=5)	a-wave	18.4±2.1	2.1±0.2^{+}	0	0	0	13.8±1.2^{++}	21.6±1.9^{++}
	b-wave	85.9±5.9^{++}	19.8±3^{++}	0	0	0	41.1±4.5^{++}	74.8±5.5^{++}
HP-6 (*n*=5)	a-wave	15.2±1^{++}	0	0	0	0	5.7±0.5	14.2±1.2^{++}
	b-wave	58.7±5.1^{++}	8.9±0.6^{++}	0	0	0	30.0±3.4^{++}	48.7±4.5^{++}
SOD (*n*=5)	a-wave	14.4±0.2^{++}	0	0	0	0	4.1±0.2	12.0±1.3
	b-wave	51.6±4.8	7.1±0.2	0	0	0	25.1±1.4^{++}	38.7±3.5^{++}
Antioxidant complex (*n*=5)	a-wave	22.2±1.5^{+}	6.0±0.3^{++}	0	0	6.4±1.0^{+}	15.6±1.6^{++}	30.6±2.3^{++}
	b-wave	115.6±7.7^{++}	40.2±2	10.1±2.0^{+}	0	26.6±2.6^{++}	63.2±5.0^{++}	124.2±9.2^{++}

Note. **p*<0.05, ***p*<0.01 compared to intact rabbits; $^{+}$*p*<0.05, $^{++}$*p*<0.01 compared to rabbits with DR (data for taurine are not presented).

RESULTS

Exposure of intact animals to bright light caused drastic disorders in the formation of ERG a- and b-waves. The a-wave disappeared completely after 10 days of the experiment and reappeared on day 30 after the exposure was over. Under these conditions the b-wave with amplitude of 205.2±8.3 μV after intense illumination started reducing after 15 days of exposure and disappeared, gradually normalizing on day 40 of experiment. In contrast to intact animals, the a- and b-waves of ERG were not detected in animals with DR as early as on day 5 of the experiment and their amplitudes did not reappear at the end of the experiment (Table 1). These results indicate that injection of antioxidants during exposure to bright light prevented to some measure suppression of ERG amplitudes in rabbits with DR. In rabbits with DR treated with α-TP, the a-wave was completely suppressed not after 5, but only after 15 days of the experiment and was partially restored at the end of the experiment, while b-wave did not reach its initial value by the end of the experiment (Table 1). Treatment with PP under conditions of exposure to bright light led to rapid recovery of ERG a- and b-waves in rabbits with DR, surpassing the initial levels: at the end of experiment (40 days) a-waves reached 21.6±1.9 μV, b-waves 74.8±5.5 μV.

HP-6 prevented reduction of ERG a- and b-waves and on day 40 restored their amplitudes to 14.2±1.2 and 48.7±4.5 μV, respectively. The efficiency of SOD was inferior to all the studied antioxidants by the time and amplitudes of ERG waves (Table 1).

The complex of natural (α-TP and SOD) and synthetic (PP and HP-6) antioxidants most effectively restored ERG parameters after exposure to bright light. The amplitudes of a- and b-waves at the end of experiment were restored sooner and more markedly than after antioxidant monotherapies (Table 1). Treatment with the antioxidant complex in rabbits with DR exposed to bright light led to an increase of a-wave amplitude to 30.6±2.3 μV and of b-wave to 124.2±9.2 μV, which corresponded to the ERG values in intact animals.

Hence, the results indicate that antioxidant treatment in DR promoted normalization of electrical activity of the retina disordered after exposure to high-intensity light. The most effective of the antioxidants in monotherapy were PP and α-TP. Treatment with the antioxidant complex almost completely prevented the disorders in ERG in rabbits with DR after intense light exposure.

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