Effect of Preparations from Potentiated Ethanol on the Content of Biogenic Monoamines and Metabolism of Ethanol in Tissues of Rats during Alcoholization

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The effect of homeopathically potentiated ethanol (C30 and C200) on ethanol metabolism was studied in alcoholized rats. We measured ethanol concentration in the blood, alcohol dehydrogenase activity in the liver, and contents of biogenic monoamines in the hypothalamus, septum, and whole blood. Potentiated preparations of ethanol were efficient after long-term treatment and delayed ethanol elimination from the blood. Preparation C200 increased alcohol dehydrogenase activity. Potentiated preparations of ethanol (particularly C200) produced a positive effect on catecholaminergic and serotoninergic systems of the brain, i.e. they enhanced protective and adaptive reactions.

Key Words: *ethanol; ultralow doses; alcoholization; biogenic monoamines*

Here we studied the effect of homeopathically potentiated ethanol in dilutions of C30 and C200 (PE-30 and PE-200, respectively) on ethanol metabolism and changes in the content of biogenic monoamines in brain tissues and blood in alcoholized rats.

MATERIALS AND METHODS

Acute and chronic experiments were performed on 50 male rats. The rats received 5% ethanol in a dose of 1.5-2.0 g/kg perorally and were decapitated after 60 min. We measured ethanol concentration in the blood [1] and alcohol dehydrogenase (ADH) activity in the liver [2]. The contents of biogenic monoamines (dopamine, norepinephrine, epinephrine, and serotonin) in the hypothalamus, septum, and whole blood were estimated spectrofluorometrically [3].

The results were analyzed by Student's *t* test.

RESULTS

One hour after ethanol intake blood concentration of ethanol in rats was 8.46±0.95 mmol/liter, while ADH activity in the liver slightly decreased (Table 1). Single administration of test preparations in combination with alcohol did not change blood ethanol concentration. It should be emphasized that liver ADH activity in rats receiving PE-200 far surpassed ADH activity in alcoholized animals and practically did not differ from the control (intact rats).

Long-term administration of ethanol (14 days) was followed by a significant increase in its blood concentration (to 340%) and less pronounced by significant increase in liver ADH activity (to 157%). Administration of test preparation in combination with ethanol had no effect on ADH activity. However, blood ethanol concentration and ADH activity in rats receiving PE-200 were higher than in other animals.

In rats receiving ethanol for 4 months its blood concentration after 24-h withdrawal was low (2.17± 0.24 mmol/liter). Administration of test preparations in combination with ethanol for 6 days increased blood ethanol concentration to 240-270% (compared to alcoholized animals). ADH activity significantly increased in rats receiving PE-200.

Thus, potentiated preparations of ethanol were efficient after long-term treatment and delayed ethanol elimination from the blood. PE-200 also increased ADH activity in the liver.

Single ethanol administration changed the content of biogenic monoamines in rat hypothalamus and septum, the structures most rapidly reacting to exogenous alcohol. Single administration of ethanol decreased the contents of dopamine and epinephrine (Table 2). Administration of test preparation in combination with ethanol did not modulate changes in epinephrine con-

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TABLE 1. Effects of PE-30 and PE-200 on Ethanol Concentration in the Blood and ADH Activity in Liver in Rats (*M*±*m*, *n*=10)

Series		Ethanol concentration, mmol/liter	ADH activity, nmol/mg protein/min
Intact rats		0	5.00±0.41
Single administration of ethanol	control	8.46±0.95	3.85±0.19
	+PE-30	6.29±1.08	4.05±0.17
	+PE-200	6.08±1.15	4.65±0.28^{+}
Chronic administration of ethanol (14 days)	control	28.86±3.47	7.85±0.57*
	+PE-30	23.44±1.52	6.62±0.37*
	+PE-200	37.54±2.39^{+o}	8.40±0.60^{*+}
Chronic administration of ethanol (4 months)	control	2.17±0.24	6.97±0.68*
	+PE-30	5.68±0.93^{+}	7.05±0.78*
	+PE-200	5.21±0.82^{+}	9.75±0.80^{*+o}

Note. Here and in Tables 2 and 3: $p<0.05$: *compared to intact rats; $^{+}$compared to the control; ^{o}compared to PE-30.

tent, but prevented the decrease in dopamine concentration and markedly increased the content of norepinephrine (to 161-172%). It should be emphasized that PE-200 produced most pronounced effect and increased serotonin concentration to 132%.

Chronic alcohol intake (14 days) decreased the content of all biogenic monoamines in rat hypothalamus and septum. PE-30 potentiated this effect of ethanol. PE-200 did not decrease catecholamine concentration (compared to alcoholized animals) and increased serotonin content to the level observed in intact rats.

The peripheral effects of 14-day alcohol intake differed from its central action. Ethanol increased blood catecholamine concentration. The increase in epinephrine concentration was most pronounced (to 273%). These shifts attested to activation of the sympathoadrenal system (Table 3). Blood serotonin level remained unchanged. Combination administration of the test preparations and ethanol prevented the increase in blood catecholamine content in rats. PE-200 was more effective in this respect.

Our results indicate that potentiated preparations of ethanol were efficient after long-term treatment.

TABLE 2. Effects of PE-30 and PE-200 on the Content of Biogenic Monoamines in the Hypothalamus and Septum in Rat Brain (nmol/g, *M*±*m*, *n*=10)

Series		Dopamine	Norepinephrine	Epinephrine	Serotonin
Intact rats		7.18±0.72	3.37±0.21	1.31±0.13	9.30±0.41
Single administration of ethanol					
	control	4.31±0.72*	3.13±0.50	0.38±0.07*	8.51±0.18
	+PE-30	6.20±0.93	5.44±0.93^{*+}	0.49±0.11*	11.17±1.46
	+PE-200	7.05±0.40	5.79±0.81^{*+}	0.55±0.06*	12.30±0.52^{*+}
Chronic administration of ethanol (14 days)	control	3.85±0.56*	1.89±0.33	0.71±0.09*	6.97±0.51*
	+PE-30	3.00±0.65*	0.83±0.12^{*+}	0.33±0.07^{*+}	4.03±0.31^{*+}
	+PE-200	4.57±0.64	1.60±0.29^{*o}	0.71±0.11^{*o}	9.53±0.78^{+o}

TABLE 3. Effects of PE-30 and PE-200 on the Content of Biogenic Monoamines in the Whole Blood from Rats after Chronic Administration of Ethanol (nmol/liter, *M*±*m*, *n*=10)

Series		Dopamine	Norepinephrine	Epinephrine	Serotonin
Intact rats		53.5±4.1	45.7±4.1	32.7±3.8	1067±27
Ethanol	control	84.7±13.3*	75.2±14.2*	89.4±12.7*	856±103
	+PE-30	53.0±6.7^{+}	30.3±6.6^{+}	26.1±6.5^{+}	967±114
	+PE-200	69.7±12.9	38.4±7.3^{+}	28.9±5.3^{+}	1281±221

Both preparations produced similar changes in test parameters. They prevented activation of the sympathoadrenal system produced by ethanol and delayed ethanol elimination from the blood. Higher efficiency of PE-200 manifested in parallel increase in liver ADH activity. It should be emphasized that the effects of these preparations on the central neurotransmitter systems increased with dilution (potentiation). PE-200 produced the positive effect not only on the catecholaminergic, but also on the serotoninergic system in the brain. These data suggest that potentiated preparation of ethanol enhance protective and adaptive reactions.

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