THE EFFECTS OF OMEGA-3 FATTY ACIDS ON ANTIOXIDANT ENZYME ACTIVITIES AND NITRIC OXIDE LEVELS IN THE CEREBRAL CORTEX OF RATS TREATED ETHANOL

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The toxic effect of ethanol on the cerebral cortex and protective effects of omega-3 fatty acids against this neurotoxicity were investigated. Twenty eight male Wistar-albino rats were divided into 4 groups. Rats of the ethanol and ethanol withdrawal groups were treated with ethanol (6 g/kg/day) for 15 days. Animals of the ethanol+omega-3 group received omega-3 fatty acids (400 mg/kg daily) and ethanol. In rats of the ethanol group SOD activity was lower than in animals of the control group. In rats treated with omega-3 fatty acids along with ethanol, SOD activity increased. GSH-Px activity and MDA levels in animals of all groups were similar. In ethanol treated rats NO levels significantly decreased as compared to the animals of the control group (6.45±0.24 nmol/g vs 11.05±0.53 nmol/g, $p<0.001$). In rats receiving ethanol+omega-3, there was a significant increase in the NO level as compared to animals of the ethanol group (13.12±0.37 nmol/g vs 6.45±0.24 nmol/g, $p<0.001$). Thus, ethanol administration leads to oxidative damage and a decrease in the NO level. Omega-3 fatty acids have a protective role against ethanol induced oxidative damage and normalize the NO level.

Key words: superoxide dismutase; alcohol withdrawal; brain

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INTRODUCTION

Ethanol is the primary active component of alcoholic beverages widely consumed in many countries. Studies performed on humans consuming alcohol and on experimental animals convincingly indicate that alcohol consumption causes direct or indirect damage to nearly all organ systems of the body [1]. One of the systems that is highly affected by harmful effects of alcohol is the central nervous system. Results of experimental and clinical studies indicate that acute or chronic ethanol consumption is related to several neurological problems [2]. Reactive oxygen species (ROS) formed in all tissues exposed to ethanol, promote development of oxidative stress largely responsible for the harmful effects of ethanol in the brain [3]. The development of oxidative stress is often accompanied by accumulation of nitric oxide (NO) molecules, which are converted to peroxy-nitrite (NOO^-) radicals deteriorating manifestations of oxidative stress [4].

Omega-3 fatty acids, especially docosahexaenoic acid (DHA), are an important component of neuronal cell membranes in the brain. The structure and constituents of the cell membranes are essential for carrying out its normal functions [5]. DHA accounts for 15–20% of the lipid content in the cerebral cortex. DHA also plays an extremely important role in the development of nervous system. By preserving the axonal structure, it contributes to proper conduction of electrical impulses through the nerve cells [6]. There is ample evidence that omega-3 fatty acids reduce oxidative stress and inflammation in the brain and other body tissues [7]. It has been proven in numerous studies in humans and animals that omega-3 fatty acids supplementation have a neuroprotective effect in neurodegenerative diseases. However, there is no pharmacological preparation of omega-3 fatty acids approved by pharmaceutical authorities for the treatment of any neurological disorder [8].

In this study, the protective effects of ω-3 fatty acids against ethanol-induced cerebral cortex damage were evaluated by analyzing superoxide dismutase (SOD), glutathione peroxidase (GSH-Px) enzymes activities, malondialdehyde (MDA) and NO levels.

MATERIALS AND METHODS

Twenty-eight Wistar-albino male rats, 250-300 g, were used in experiments. All experiments were conducted in the Mustafa Kemal University Animal Laboratory. Animals were kept in a room maintained at an ambient temperature and humidity (25±5°C, 55±5%) under a day/night regime (day 7:00–19:00 and night 19:00–7:00) and allowed a commercial standard rat diet and water *ad libitum*.

Experimental Protocols

The rats were divided randomly into four groups consisting of seven rats each. Animals of the control group received intra-gastric saline as a vehicle. Ethanol treated rats received a daily total dose of 6.0 g/kg of 30% ethanol solution (twice a day) via an oesophageal probe for 15 days. Rats of the ethanol+ω-3 group received ω-3 fatty acids along with ethanol (6.0 g/kg) on a daily basis. Omega-3 fatty acid (Marincap capsule®, "Koçak Pharmaceuticals", Turkey) was administered through gavage pathway, at a dose of 400 mg/kg per day [9]. The fatty acid composition of the Marincap capsule is Salmon fish oil concentrate and included eicosapentaenoic acid (EPA; 18%), DHA (12%). Ethanol withdrawal rats, received a daily dose of 6.0 g/kg of 30% ethanol solution (twice a day) via an oesophageal probe for 15 days. At the end of the 15-day experimental period, 28 h after the last administration of ethanol in the ethanol withdrawal group, 4 h after the last administration of ethanol in the ethanol and other groups, all rats were sacrificed by decapitation and cerebral cortex samples were removed for biochemical analysis.

Biochemical Analysis

For biochemical analyses, tissue samples were extracted quickly, washed with cold (4°C) 0.15 M KCl solution and dried with a paper towel. The dried tissue samples were homogenized in an ice container containing 0.15 M KCl solution, by a homogenizer (Ultra Turrax Type T25-B, "IKA Labortechnic", Germany) at 16000 rpm for 3 min. The homogenates were centrifuged at 5000 *g* for 1 h (4°C). Resultant supernatants were aliquoted frozen and stored at -40°C (within 1 week) for analysis of superoxide dismutase (SOD), glutathione peroxidase (GSH-Px) activities. The levels of malondialdehyde (MDA) and NO were analyzed spectrophotometrically (UV-1800, "Shimadzu Corp.", Japan) in the homogenates.

SOD activity was determined by the method modified by Sun et al. [10]. This method is based on reduction of nitroblue tetrazolium (NBT) by the xanthine-xanthine-oxidase system, generating superoxide radicals. In our study, SOD activity was expressed as unit/mg (U/mg) of tissue protein. Determination of glutathione peroxidase activity was based on the method proposed by Paglia and Valentine [11]. In presence of hydrogen peroxide GSH-Px catalyzes oxidation of reduced glutathione (GSH) to its oxidized form (GSSG). The GSSG obtained by means of GSH-Px in the presence of hydrogen peroxide, may be further reduced to GSH in the NADPH-dependent reaction catalyzed by glutathione reductase. GSH-Px activity was measured as the change in absorbance during oxidation of NADPH to $NADP^+$ measured at 340 nm, calculated and expressed as unit/gram (U/g) tissue protein.

Lipid peroxidation was determined by the method of Esterbauer and Cheeseman [12]. The MDA reacts with thiobarbituric acid (TBA) forming a pink chromogen during incubation 90–95°C. After 15-min incubation and cooling the absorbance of samples was measured spectrophotometrically at 532 nm. The results were expressed as nmol/g tissue protein. NO and nitrate converted to nitrite by the Griess reaction, were measured by means of a spectrophotometric method; the results were expressed in nmol/g [13]. Protein measurements were performed in tissue homogenate according to the method of Lowry et al. [14].

Statistical Analysis

The statistical analyses of the results were performed using the statistical program "SPSS 18 for Windows". The distribution of results was evaluated by the non-parametric one-sample Kolmogorov-Smirnov Test. Since results in the groups did not show a normal distribution, the Kruskal-Wallis non-parametric test was used for the comparison of the values. For the comparisons between groups, the Mann-Whitney U test was applied. Differences were considered as statistically significant at $p<0.05$. The data obtained were expressed as arithmetic means ± the standard error of the mean (SEM).

RESULTS

In rats of the ethanol group SOD activity was lower than in animals of the control group (Table 1). In rats treated with ω-3 fatty acids along with ethanol SOD activity significantly increased as compared to SOD activity in rats of the ethanol group. GSH-Px activity and MDA levels in all groups were similar according to the statistics (Table 1).

Table 1. Activities of SOD and GSH-Px and the MDA level in the cerebral cortex of rat of the studied group

Group	SOD (U/mg protein)	GSH-Px (U/g protein)	MDA (nmol/g protein)
Control	0.135±0.013	1.359±0.706	19.174±3.865
Ethanol	0.114±0.049[a]	1.001±0.506	20.718±0.986
Ethanol+ω-3	0.170±0.017[ab]	1.534±1.512	18.879±6.492
Ethanol withdrawal	0.133±0.052	0.951±0.157	16.335±3.244

Each group contained 7 rats. a – $p<0.05$ as compared to the control group, b – $p<0.01$ as compared to the ethanol group.

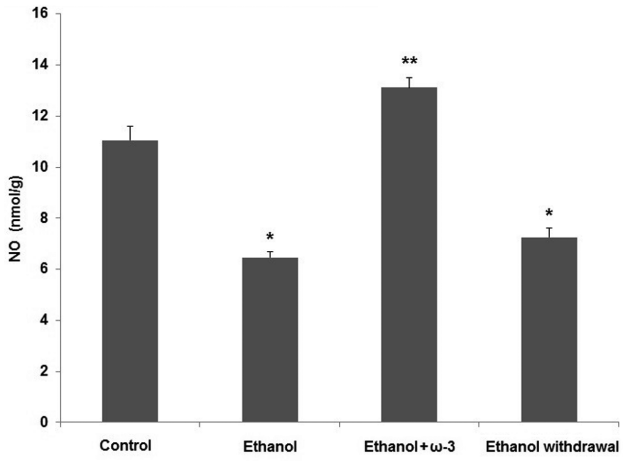

Figure 1. The cerebral cortex NO levels in rats of control, ethanol, ethanol+ω-3, and ethanol withdrawal groups. * – $p<0.001$ versus the control group. ** – $p<0.001$ versus the ethanol group.

In rats treated with ethanol the NO level was significantly lower than in animals of the control group (Fig. 1). In the rats treated with ω-3 fatty acids along with ethanol the significantly increased and even slightly increased the NO level in rats of the control group.

DISCUSSION

Free radicals may be generated in the body under physiological conditions or as a result of a pathological processes. However, there is a balance between free radicals produced and activity of the antioxidant defense system [3]. In the case, when the balance is lost in favor of free radicals, oxidative stress develops. Living organisms protect themselves against oxidative damage by means of the enzymatic and non-enzymatic antioxidant defense systems. Catalase (CAT), SOD, and GSH-Px are among the most effective enzymatic antioxidant defense system [7]. Macit et al. found a significant decrease in the activity of SOD and GSH-PX in the hippocampal tissue of rats exposed to chronic ethanol treatment [15]. In this study we found a statistically significant decrease of the SOD activity in the brain cortical tissues of rats exposed to ethanol, while a decrease in GSH-Px did not reach the level of statistical significance. Certain evidence exists that the changes in antioxidant enzyme activities may depend on duration, dose, and route of ethanol administration and tissue responsiveness [16]. MDA generated as an end product of lipid peroxidation, is one of the widely used parameter indicating oxidative damage [17]. MDA levels were significantly higher in many studies of ethanol-induced oxidative stress. Researchers reported an increase in MDA levels in the hippocampus of rats exposed to chronic alcohol and this rise was found to be statistically significant [15]. Lipid peroxidation products were reported to increase as a result of ethanol treatment in a few studies on different rat species [18, 19]. In this study, MDA levels were not changed in rats of the ethanol group. Jurczuk et al. showed that the MDA level increased in the liver but not in the kidney of animals treated with ethanol for 12 weeks (5 g/kg/day) [20].

A recent systematic review and meta-analysis showed that omega-3 supplementation had an important role in enhancing the antioxidant defense system in various tissues. Therefore, omega-3 supplementation can have very positive effects in improving many pathological conditions, especially those caused by oxidative stress [21]. Good evidence exists that omega-3 supplementation ameliorated the ethanol-induced damage of different organs [7]. In our study treatment with ω-3 fatty acids normalized SOD activity in animals of the ethanol+ω-3 group. Since GSH-Px activity and MDA remained unchanged in rats of the ethanol group, omega-3 supplementation influenced only the parameter (SOD) changed by the ethanol treatment.

The original finding of this study consists in elucidation of the effects of ethanol and omega-3 fatty acid administration on the cerebral cortex NO level. Data in the literature indicate that ethanol plays an important role in regulating NO at various levels. For example, blood NO levels increased after acute or chronic ethanol consumption in animals or humans [22]. On the contrary, very a few studies demonstrated the ethanol-induced decrease of NO. For example, chronic alcohol consumption may lead to the decrease in the brain NO levels [23]. In a recent study, nitrite levels were significantly lower in the hippocampus and prefrontal cortex of animals treated with ethanol [24]. Similarly, in this study the cerebral cortex NO level significantly decreased in rats of the ethanol group. The lack of an increase in the cerebral cortex NO may require looking at the harmful effects of ethanol from another perspective. It is well known that NO is a very important neurotransmitter and neuromodulator in the brain, playing a critical role in maintaining normal brain functions. NO is also known as a survival-promoting molecule that can protect various cell types, especially neurons, against different toxic conditions and insults [25]. Some authors suggest that chronic ethanol consumption may decrease NO production via a reduction of nNOS expression in cerebral cortex or other tissues [26]. Treatment with ω-3 fatty acid significantly recovered NO levels in cerebral cortex of rats treated with ethanol. The effects of ω-3 on NO levels are contradictory. Some authors reported that ω-3 supplementation reduced NO production by inducible NO synthese [27]. Other researchers found an increase in the NO levels in brain tissues

in both juvenile and adult ω-3 deficient rats [28]. On the other hand, ω-3 fatty acid supplementation increased NO bioavailability in patients with cardiovascular problems, but not in healthy subjects [29]. In another study, omega-3 supplementation for 3 weeks resulted in increased basal serum NO as compared with pretreatment levels in athletes [30]. Omega-3 supplementation for 3 months also increased plasma NO levels in both sedentary and athletes in university students [31]. According to these data, ω-3 fatty acids can correct neuropathological damage in the brain by normalizing the NO level.

Many studies do not distinguish between ethanol exposure and ethanol withdrawal and thus it is not clear whether the reported injuries/mechanisms are associated with ethanol toxicity, ethanol withdrawal or both [32]. In this context researchers clearly stated, we have added an ethanol withdrawal group to the experimental protocol. Indeed, oxidative stress parameters did not change in animals of the alcohol withdrawal group, but the NO level decreased. The latter is consistent with a previous report that ethanol withdrawal induced oxidative stress and reduced NO levels in vasculature [33]. Since SOD activity decreased moderately in this study it rapidly recovered to the initial (control) level in rats of the ethanol withdrawal group (Table 1). In this study, the abstinence period was 28 h. This suggests that in cases where severe oxidative stress does not develop, as in this study, SOD enzyme activity can recover much faster than other parameters [34].

CONCLUSIONS

In conclusion, the present study showed that ethanol administration was accompanied by a decrease in the cerebral cortex SOD activity and the NO level. Omega-3 fatty acids supplementation normalized ethanol-induced changes in the SOD activity and the NO level.

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COMPLIANCE WITH ETHICAL STANDARDS

The experiments were performed in accordance with the Guide for the Care and Use of Laboratory Animals [35]. This research is approved by a Local Ethics Committee.

CONFLICT OF INTEREST

The authors declare no conflicts of interest.

REFERENCES

1. *Wu D., Zhai Q., Shi X.* (2006) Alcohol-induced oxidative stress and cell responses. J. Gastroenterol. Hepatol., **21**(3), S26-S29. DOI: 10.1111/j.1440-1746.2006.04589.x
2. *Hillbom M., Pieninkeroinen I., Leone M.* (2003) Seizures in alcohol-dependent patients: Epidemiology, pathophysiology and management. CNS Drugs, **17**, 1013-1030. DOI: 10.2165/00023210-200317140-00002
3. *Hernández J.A., López-Sánchez R.C., Rendón-Ramírez A.* (2016) Lipids and oxidative stress associated with ethanol-induced neurological damage. Oxid. Med. Cell. Longev., **2016**, 1543809. DOI: 10.1155/2016/1543809
4. *Shan L., Wang B., Gao G., Cao W., Zhang Y.* (2013) L-Arginine supplementation improves antioxidant defenses through L-arginine/nitric oxide pathways in exercised rats. J. Appl. Physiol., **115**(8), 1146-1155. DOI: 10.1152/japplphysiol.00225.2013
5. S*wanson D., Block R., Mousa S.A.* (2012) Omega-3 fatty acids EPA and DHA: health benefits throughout life. Adv. Nutr., **3**(1), 1-7. DOI: 10.3945/an.111.000893
6. *Michael-Titus A.T., Priestle J.V.* (2014) Omega-3 fatty acids and traumatic neurological injury: From neuroprotection to neuroplasticity? Trends Neurosci., **37**(1), 30-38. DOI: 10.1016/j.tins.2013.10.005
7. *Serrano M., Rico-Barrio I., Grandes P.* (2023) The effect of omega-3 fatty acids on alcohol-induced damage. Front. Nutr., **10**, 544. DOI: 10.3389/fnut.2023.1068343
8. *Chitre N.M., Moniri N.H., Murnane K.S.* (2019) Omega-3 fatty acids as druggable therapeutics for neurodegenerative disorders. CNS Neurol. Disord. Drug Targets, **18**(10), 735-749. DOI: 10.2174/1871527318666191114093749
9. *Zararsiz I., Kus I., Akpolat N., Songur A., Ogeturk M., Sarsilmaz M.* (2006) Protective effects of ω-3 essential fatty acids against formaldehyde-induced neuronal damage in prefrontal cortex of rats. Cell Biochem. Funct., **24**(3), 237-244. DOI: 10.1002/cbf.1204
10. *Sun Y.I., Oberley L.W., Li Y.* (1988) A simple method for clinical assay of superoxide dismutase. Clin. Chem., **34**(3), 497-500. DOI: 10.1093/clinchem/34.3.497
11. *Paglia D.E., Valentine W.N.* (1967) Studies on the quantitative and qualitative characterization of erythrocyte glutathione peroxidase. J. Lab. Clin. Med., **70**(1), 158-169. PMID: 6066618
12. *Esterbauer H., Cheeseman K.H.* (1990) Determination of aldehydic lipid peroxidation products: malonaldehyde and 4-hydroxynonenal. Methods Enzymol., **186**, 407-421. DOI: 10.1016/0076-6879(90)86134-H
13. *Cortas N.K., Wakid N.W.* (1990) Determination of inorganic nitrate in serum and urine by a kinetic cadmium-reduction method. Clin. Chem., **36**(8), 1440-1443. DOI: 10.1093/clinchem/36.8.1440
14. *Lowry O.H., Rosebrough N.J., Farr A.L., Randall R.J.* (1951) Protein measurement with the Folin phenol reagent. J. Biol. Chem., **193**, 265-275. PMID: 14907713
15. *Macit E., Ulusoy G., Celik T., Kayir H., Uzbay T.* (2012) Comparative effects of antioxidants on chronic ethanol-induced oxidative stress in rat hippocampus. J. Neurol. Sci., **29**(2), 329-339.
16. *Ozel Turkcu U., Bilgihan A., Biberoglu G, Mertoglu Caglar O.* (2010) Carnosine supplementation protects rat brain tissue against ethanol-induced oxidative stress. Mol. Cell Biochem., **339**, 55-61. DOI: 10.1007/s11010-009-0369-x
17. *Pompella A.* (1997) Biochemistry and histochemistry of oxidant stress and lipid peroxidation. Int. J. Vitam. Nutr. Res., **67**(5), 289-297.

18. *Almansa I. Barcia J.M., López-Pedrajas R., Muriach M., Miranda M., Romero F.J.* (2013) Naltrexone reverses ethanol-induced rat hippocampal and serum oxidative damage. Oxid. Med. Cell. Longev., **2013**, 296898. DOI: 10.1155/2013/296898
19. *Scolaro B., Delwing-de Lima D., da Cruz J.G.P., Magro D.D.* (2012) Mate tea prevents oxidative stress in the blood and hippocampus of rats with acute or chronic ethanol administration. Oxid. Med. Cell. Longev., **2012**, 314758. DOI: 10.1155/2012/314758
20. *Jurczuk M., Brzóska M.M., Moniuszko-Jakoniuk J., Galażyn-Sidorczuk M., Kulikowska-Karpińska E.* (2004) Antioxidant enzymes activity and lipid peroxidation in liver and kidney of rats exposed to cadmium and ethanol. Food Chem. Toxicol., **42**(3), 429-438. DOI: 10.1016/j.fct.2003.10.005
21. *Heshmati J., Morvaridzadeh M., Maroufizadeh S., Akbari A., Yavari M., Amirinejad A., Maleki-Hajiagha A., Sepidarkish M.* (2019) Omega-3 fatty acids supplementation and oxidative stress parameters: A systematic review and meta-analysis of clinical trials. Pharmacol. Res., **149**, 104462. DOI: 10.1016/j.phrs.2019.104462
22. *Deng X.S., Deitrich R.A.* (2007) Ethanol metabolism and effects: Nitric oxide and its interaction. Curr. Clin. Pharmacol., **2**(2), 145-153. DOI: 10.2174/157488407780598135
23. *Kurban S., Mehmetoğlu İ.* (2008) The effect of alcohol on total antioxidant activity and nitric oxide levels in the sera and brains of rats. Turk. J. Med. Sci., **38**(3), 199-204.
24. *Khan M.I., Nikoui V., Naveed A., Mumtaz F., Zaman H., Haider A., Aman W., Wahab A., Khan S.H., Ullah N., Dehpour A.R.* (2021) Antidepressant-like effect of ethanol in mice forced swimming test is mediated via inhibition of NMDA/nitric oxide/cGMP signaling pathway. Alcohol, **92**, 53-63. DOI: 10.1016/j.alcohol.2021.01.005
25. *Karaçay B., Bonthius D.J.* (2015) The neuronal nitric oxide synthase (nNOS) gene and neuroprotection against alcohol toxicity. Cell. Mol. Neurobiol., **35**, 449-461. DOI: 10.1007/s10571-015-0155-0
26. *Silva S.M., Silva S., Meireles M, Leal S.* (2015) nNOS is involved in cardiac remodeling induced by chronic ethanol consumption. Toxicology, **329**, 98-105. DOI: 10.1016/j.tox.2015.01.009
27. *Mori M.A., Delattre A.M., Carabell B., Pudell C., Bortolanza M., Staziaki P.V., Visentainer J.V., Montanher P.F., del Bel E.A., Ferraz A.C.* (2018) Neuroprotective effect of omega-3 polyunsaturated fatty acids in the 6-OHDA model of Parkinson's disease is mediated by a reduction of inducible nitric oxide synthase. Nutr. Neurosci., **21**(5), 341-351. DOI: 10.1080/1028415X.2017.1290928
28. *Cardoso H.D., dos Santos Junior E.F., de Santana D.F., Gonçalves-Pimentel C., Angelim M.K., Isaac A.R., Lagranha C.J., Guedes R.C., Beltrão E.I., Morya E., Rodrigues M.C., Andrade-da-Costa B.L.* (2014) Omega-3 deficiency and neurodegeneration in the substantia nigra: Involvement of increased nitric oxide production and reduced BDNF expression. Biochim. Biophys.Acta, **1840**, 1902-1912. DOI: 10.1016/j.bbagen.2013.12.023
29. *Balakumar P., Taneja G.* (2012) Fish oil and vascular endothelial protection: Bench to bedside. Free Radical Biol. Med., **53**, 271-279. DOI: 10.1016/j.freeradbiomed.2012.05.005
30. *Żebrowska A., Mizia-Stec K., Mizia M., Gąsior Z., Poprzęcki S.* (2015) Omega-3 fatty acids supplementation improves endothelial function and maximal oxygen uptake in endurance-trained athletes. Eur. J. Sport Sci., **15**(4), 305-314. DOI: 10.1080/17461391.2014.949310
31. *Mostafa Mahmoud A.* (2017) Effect of omega 3 and regular exercise on the muscle performance: Special prevalence of histamine and nitric oxide production. Al-Azhar Med. J., **46**, 739.748.
32. *Jung M.E., Metzger D.B.* (2010) Alcohol withdrawal and brain injuries: Beyond classical mechanisms. Molecules, **15**, 4984-5011. DOI: 10.3390/molecules15074984
33. *Gonzaga N.A., Mecawi A.S., Antunes-Rodrigues J., de Martinis B.S., Padovan C.M., Tirapelli C.R.* (2015) Ethanol withdrawal increases oxidative stress and reduces nitric oxide bioavailability in the vasculature of rats. Alcohol, **49**(1), 47-56. DOI: 10.1016/j.alcohol.2014.12.001
34. *Alexinschi O.-E., Chirita R., Ciobica A., Manuela P., Dobrin R., Prepelita R., Serban I., Chirita V.* (2014) The relevance of oxidative stress status in one week and one month alcohol abstinent patients. J. Med. Biochem., **33**(3), 284-290. DOI: 10.2478/jomb-2014-0008
35. Guide for the Care and Use of Laboratory Animals (1985) National Institutes of Health (NIH).

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ВЛИЯНИЕ ω-3 ЖИРНЫХ КИСЛОТ НА АКТИВНОСТЬ АНТИОКСИДАНТНЫХ ФЕРМЕНТОВ И УРОВЕНЬ ОКСИДА АЗОТА В КОРЕ ГОЛОВНОГО МОЗГА КРЫС, ПОДВЕРГНУТЫХ ВОЗДЕЙСТВИЮ ЭТАНОЛОМ

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Исследовали токсическое воздействие этанола на кору головного мозга и защитное действие ω-3 жирных кислот против этой нейротоксичности. Двадцать восемь самцов крыс линии Вистар были разделены на 4 группы. Крысы в группах “этанол” и “отмена этанола” получали этанол (6 г/кг/день) в течение 15 дней. Животные в группе “этанол+ω-3”получали ω-3 жирные кислоты (400 мг/кг в день) и этанол. У крыс в группе “этанол” активность СОД была ниже, чем в контрольной группе. У крыс, получавших ω-3 жирные кислоты вместе с этанолом, активность СОД повысилась. Активность GSH-Px и уровень малонового диальдегида были одинаковыми во всех группах. У крыс, получавших этанол, уровень NO был значительно снижен по сравнению с контрольной группой (6,45±0,24 нмоль/г против 11,05±0,53 нмоль/г, p<0,001). У крыс, получавших этанол+ω-3, отмечено значительное повышение уровня NO по сравнению с животными в группе этанола (13,12±0,37 нмоль/г против 6,45±0,24 нмоль/г, p<0,001). Таким образом, приём этанола приводит к окислительному повреждению и снижению уровня NO. ω-3 жирные кислоты оказывают защитное действие против окислительного повреждения, вызванного этанолом, и нормализуют уровень NO.

Полный текст статьи на русском языке доступен на сайте журнала (http://pbmc.ibmc.msk.ru).

Ключевые слова: супероксиддисмутаза; алкогольная абстиненция; мозг

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