

Original Research Article

Protective influence of ginger on hematological parameters and antioxidant system in the blood of rats subjected to withdrawal from long term ethanol consumption

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Abstract

The effects of chronic ethanol exposure and acute ethanol withdrawal on the blood antioxidant defense system, lipid peroxide concentration and hematological parameters, as well as the possible protective role of ginger extract administration were studied. Male wistar albino rats (3 months old) were treated with 20% ethanol (2g/kg b.w. o.p.) for 42 days; the withdrawal group is also treated the same way but stopped from ethanol administration for 72 hrs after the last dose; parallel groups are maintained that received ginger extract (200mg/kg b.w., o.p.) for both the cases mentioned above. The hematological parameters were assessed: red blood cell counts, hematocrit value and hemoglobin concentration were significantly decreased in the blood of EtOH - treated rats, whereas mean corpuscular volume (MCV) significantly increased in withdrawal rats. We also observed increased activity of antioxidant defense enzymes: copper zinc containing superoxide dismutase, catalase, glutathione peroxidase, glutathione reductase and glutathione-S-transferase as well as concentrations of non-enzymatic components of antioxidant defense system: reduced glutathione, vitamin C and vitamin E in rats pre- treated with ginger extract. It is concluded that ginger exhibited a protective role against the pro-oxidant effects of ethanol and ethanol withdrawal on the hematological values as well as on enzymatic and non-enzymatic components of blood antioxidant defense system.

Keywords: Blood, Enzymatic and Non Enzymatic Antioxidants, Ethanol, Ethanol Withdrawal, Ginger

1. Introduction

Free radicals are constantly produced as a consequence of the endogenous reactions. High level of exogenous or endogenous free radicals could lead to destruction of major components of cellular structure, including nucleic acids, proteins, amino acids, lipids, and carbohydrates and would affect various cell functions such as membrane function, metabolism and even gene expression, and they can cause a number of other pathological conditions (Young and Woodside, 2001). As such, they are an important antioxidant defense in nearly all cells exposed to oxygen. Toxic effects of ethanol on animals and humans have been well documented and confirmed by biochemical researches and by morphological and clinical examinations (Dobrzynska et al., 2005; Gabbianelli et al., 2007; Zhao et al., 2001). It was shown that ethanol metabolism is accompanied by free radicals formation and by changes in antioxidant status, which leads to oxidative stress that is very dangerous to erythrocytes (Zima et al., 2001). Erythrocytes are particularly susceptible to oxidative stress as a result of the high concentration of oxygen and haemoglobin. The latter is a potentially powerful promoter of oxidative processes (Cimen, 2008).

Under physiological conditions, erythrocytes and other cells can generate a continuous flux of superoxide radicals (O₂^{•-}) and H₂O₂ at a low level (Pryor et al., 2006). The reactions of these particles with haemoglobin destabilise the haeme and globin structure and release free iron ions that play a significant role in the generation of reactive oxygen species. Both haeme compounds and free iron ions can be active catalysts of lipid peroxidation processes. These products induce site-specific oxidative changes in the erythrocyte membrane's phospholipid bilayer organisation (Dobrzynska et al., 2005). Reduced levels of vitamins in general and Vitamin E in particular (Dupont et al., 2000) have been found in serum of alcoholics. This may reduce antioxidant capacity and promote generation of free radicals and lipid peroxides resulting in tissue damage and disease. Thus the role of nutrition in favor of an anti oxidant defense mechanism cannot be ruled out.

The red cell has been a central focus of this research, because it is thought to undergo a high endogenous rate of H₂O₂ production from hemoglobin autoxidation, which can be markedly increased in cells with unstable hemoglobins. In addition, the red cell is exposed to H₂O₂, superoxide and NO in under certain conditions like exposure to pro oxidants like ethanol etc (Johnson et al., 2000). Earlier studies have demonstrated a close association between oxidative damage and alcohol abuse.

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It has been suggested that free radical intermediates produced during ethanol metabolism might be responsible for causing oxidative damage (Clot et al., 1994). Alcohol withdrawal itself is known to induce oxidative stress in the early recovery phase after alcohol abstinence (Marotta et al., 1997). Hematological assays may provide an index of the physiological status of the organism. Leucocyte count, erythrocyte count, hematocrit and hemoglobin are particularly recommended as tests that could be performed on a routine basis to monitor the health of the patient. Hematological indices including Hb and HCT are influenced by various factors. The studies show that pulsed electric field, magnetic field exposure (Amara et al., 2006), plant extracts (Obaji et al., 2009; Devaki, et al., 2012), drugs, chemicals (Starek-Świechowicz et al., 2012; Komarek, 1984), and opioids (Llorente-García et al., 2009) can influence hematological parameters including Hb and HCT.

In an era of increasing tendency towards the use of traditional medicine against synthetic drugs, a variety of medicinal herbs are known to stimulate phagocyte cells including ginger, garlic, curcumin and turmeric (*Curcuma longa*) etc. (Dugenci et al., 2003; Nya and Austin, 2009; Nya and Austin, 2011; Behera et al., 2011; Alambra et al., 2012). The rhizome of ginger (*Zingiber officinale*) has been reported to possess a broad-spectrum of prophylactic and therapeutic activities (Ernst and Pittler, 2000). Ginger is found effective in the control of a range of bacterial, viral, fungal and parasitic diseases (Agrawal et al., 2001; Martins et al., 2001; Endo et al., 1990). In addition, ginger is effective as an immunomodulatory agent in animals and fish and helps to reduce the losses caused by diseases in aquaculture (Nya and Austin, 2009; Ali et al., 2008; Zhou et al., 2006; Tan and Vanitha, 2004).

The aim of our study was to investigate a possible protective influence of ginger extract pretreatment in conditions of chronic ethanol exposure and ethanol withdrawal on hematological parameters and antioxidant defense system (AOS) in the blood of rats subjected to acute ethanol withdrawal. The following parameters were determined: red blood cells count (RBCs), hematocrit value (Ht), hemoglobin (Hb), RBCs copper zinc containing superoxide dismutase (CuZn SOD, EC 1.15.1.1), catalase (CAT, EC 1.11.1.6), glutathione peroxidase (GSH-Px, EC 1.11.1.9), glutathione reductase (GR, EC 1.6.4.2) and plasma glutathione-S-transferase (GST, EC 2.5.1.18) activities, as well as blood GSH and plasma Vit C and Vit E concentrations.

2. Material and methods

2.1 Extract preparation

Ginger extract was prepared from locally purchased ginger rhizome using double distilled water by Soxhlet extraction method.

2.2. Animals and experimental design

In our experiments, 3-month-old male Wistar albino rats weighing 220 ± 30 g were used. The animals were kept at

28 ± 5 °C and exposed to a 12h light - 12h dark cycle. All rats were housed in individual cages and given a standard diet and water ad libitum. The Ethic Committee respecting the Control and Supervision of experiments on Animals (CPCSEA), Ministry of Social Justice and empowerment, Government of India, approved all the experiments. The animals were divided into the following groups:

- control group (n=6) was treated intragastrically with 2ml of physiological saline every day for six weeks;

- AGE group(n=6) treated with ginger extract 200 mg/kg bw orogastrically for six weeks;

- alcohol group (n=6) was treated intragastrically with 2 ml of 20% ethanol every day for six weeks.

- alcohol+extract group(n=6) was treated intragastrically with 2 ml of 20% ethanol every day for six weeks and was simultaneously treated with ginger extract 200 mg/kg bw orogastrically.

- withdrawal group (n=6) was treated intragastrically with 2 ml of 20% ethanol every day for six weeks and then stopped ethanol administration and kept under observation for the next 72hrs.

- extract and withdrawal group (n=6) was treated intragastrically with 2 ml of 20% ethanol every day along with ginger extract administration 200 mg/kg bw orogastrically for six weeks and then stopped ethanol administration and kept under observation for the next 72hrs .

After six weeks of the experiment, animals were sacrificed by decapitation and fresh blood was immediately collected into heparinized test tubes. Haematological parameters such as RBC, haemoglobin (HGB), haematocrit (HCT), RBCs, WBCs, Hbs and platelet count, mean corpuscular volume (MCV), mean corpuscular haemoglobin (MCH), and mean corpuscular haemoglobin concentration (MCHC) were determined by using haematological analyser ADVIA 2120 (Siemens).

Blood for the determination of antioxidant status was centrifuged to separate plasma and RBCs. Plasma specimens were used for determination of Vit C by the method of Day et al. (1979), while Vit E was determined by the method suggested by Desai (1984). The MDA content was assayed as thiobarbituric acid reactive substances (TBARS) in the blood according to Ohkawa et al. (1979). Concentration of GSH in whole blood was measured by a standard method according to Beutler (1975).

Isolated RBCs were washed three times with 3 volumes of ice-cold 155 mmol/l NaCl and hemolysates containing about 50 g Hb/l (prepared according to McCord and Fridovich 1969) were used for the determination of SOD, CAT, GPx and GR activities. For the determination of SOD activity by the epinephrine method of Misra and Fridovich (1972), lysates were diluted with distilled water (1:7 v/v) and treated by chloroform-ethanol (0.6:1 v/v) in order to remove hemoglobin (Tsuchihashi, 1923).

CAT activity was determined according to Beutler (1982), while the activity of GPx was assayed by the subsequent oxidation of NADPH at 340 nm with t-butyl-hydroperoxide as a substrate (Maral et al. 1977). The activity of GR was determined by measuring NADPH oxidation at 340 nm in the presence of oxidized glutathione (Glatzle et al. 1974). GST activity towards 1-chloro-2,4-dinitrobenzene (CDNB) as a substrate was measured according to Habig et al. (1974).

2.3 Statistical analysis

The data obtained were expressed as mean \pm SD. The data were analysed by use of standard statistical analyses, two way ANOVA test for multiple comparisons to determine significance between different groups. The level of significance was set at $P < 0.05$.

3. Results

Table 1 clearly show that ethanol withdrawal resulted in significant decreases of RBCs count, Ht value and Hb concentration ($p < 0.05$) when compared to control animals as well as chronic ethanol ingested group. Pretreatment with ginger diminished the negative effects of ethanol indicating that ginger prevents anemia caused by chronic ethanol consumption. Lipid peroxide concentration was significantly increased in the blood of rats after acute withdrawal from ethanol ($p < 0.05$), while ginger pretreatment reversed this change to control values.

The data presented in Table 2 shows significant changes in the activities of AOS enzymes during the treatment of rats with ethanol and ethanol withdrawal under the influence of ginger extract. In animals exposed to ethanol withdrawal the activities of SOD and CAT ($p < 0.05$)

(Table 2), as well as the activities of GPx, and GST ($p < 0.05$) (Table 2) were significantly decreased in comparison to controls. The pretreatment with ginger prior to ethanol intoxication as well as withdrawal from chronic ethanol consumption partially reversed these changes. The activities of SOD, CAT and GR ($p < 0.05$) were significantly decreased as compared with animals given ethanol alone and also in withdrawal animals without ginger treatment.

The concentration of serum malondialdehyde was increased during the whole examined period after chronic intoxication of ethanol and after 72 hours of withdrawal from ethanol MDA concentration in the serum is found to be further elevated but in groups that were treated with ginger extract a significant decreased MDA levels are recorded (Table 2). Pretreatment with ginger extract in ethanol withdrawal group reversed the concentrations of GSH, Vit E and Vit C to the control levels.

Ethanol metabolism is accompanied by the generation of free radicals and acetaldehyde. This aldehyde can readily react with amino and sulphhydryl groups of protein molecules (Braun et al., 1995) and disturb their structure. It leads to a statistically significant decrease in

the activity of all examined antioxidant enzymes. It is especially evident by in vitro experiments, when the enzyme activities were strongly decreased after acetaldehyde treatment. The decrease in the activity of Cu, Zn-SOD selectively metabolises superoxide anion and enhances this radical level. The superoxide anion is the primary free radical species involved in various tissue injuries. It undergoes spontaneous enzymatic dismutation to hydrogen peroxide. ROS react mainly with sulphhydryl and aromatic groups of proteins (Dean, 2008) which lead to changes in the structure and functions of protein molecules, including enzymes. In the presence of iron ions hydroxyl radicals are generated from superoxide anions and hydrogen peroxide, which results in enhanced lipid peroxidation that is evaluated by measurement of MDA level. The decrease in CAT activity should not disturb redox balance because hydrogen peroxide is removed from the erythrocytes mainly by the GSH-Px system (Wakimoto et al., 1998). However, the level of GSH-Px co-substrate-reduced glutathione is significantly decreased after acetaldehyde as well as ethanol reaction.

Erythrocytes are particularly susceptible to oxidative damage as a result of high polyunsaturated fatty acid content in their membranes and high concentration of oxygen and haemoglobin, the latter being a potentially powerful promoter of oxidative processes (Cimen, 2008). ROS reactions with haemoglobin destabilise the haeme and globin structure and release free iron ions that play significant role in the generation of ROS. Destructive action of ROS and ethanol metabolites depends on the efficiency of antioxidant system, whose components also undergo ROS reaction. The deleterious consequences of the above mentioned actions have stimulated studies on the mechanisms of action of biologically relevant natural antioxidants such as polyphenolic compounds.

Alcohol has a variety of pathologic effects on hematopoiesis. It directly damages erythroid precursors, thereby contributing to macrocytosis and the anemic state of chronic alcoholics. Ethanol induces sideroblastic anemia, perhaps by direct interference with heme synthesis. Further, chronic ingestion of alcohol can lead to various types of hemolytic anemia caused by alterations in the erythrocyte membrane lipids that occur in association with alcoholic liver disease (Nordmann and Rouach, 1996). It has been reported that ethanol causes hematological disturbances in various clinical and experimental studies (Kanbak et al., 2007; Padmini and Sundari, 2008). The present study demonstrates that chronic ethanol administration induced a decrease in the levels of rats Hb, Ht, MCH, MCHC and RBC and Plt numbers. The reduction in RBCs, Hb and Ht might be due to an inhibition of erythropoiesis and hemoglobin synthesis and to an increase in the rate of erythrocytes destruction (Maruyama et al., 2001). The observed decrease of RBCs, Hb, Ht values and the increase of MCV value in rats exposed to ethanol could be the manifestation of swollen erythrocytes and macrocytic anemia (Harold and Ballard, 1997). In agreement with the above observations a previous study (Alimi et al., 2012a) showed that chronic ethanol ingestion leads to a marked anemia in

Table 1 Comparative hematological values (means \pm S.E.M) during chronic ethanol ingestion and ethanol withdrawal under the influence of ginger extract

Parameter	Controls	AGE group	Ethanol group	Ethanol + AGE group	Ethanol withdrawal group	Ethanol withdrawal + AGE group
RBC ($10^6/\mu\text{l}$)	7.46 \pm 0.32	7.28 \pm 0.42	6.73 \pm 0.25*#	7.16 \pm 0.36	5.97 \pm 0.76*#	7.28 \pm 0.42
Hb (g/dl)	14.38 \pm 0.51	14.25 \pm 0.38	10.31 \pm 0.54*#	13.83 \pm 0.49	9.10 \pm 0.54*#	14.25 \pm 0.38
Ht (%)	43.92 \pm 1.21	44.81 \pm 1.23	37.65 \pm 1.41**	42.37 \pm 1.32	34.99 \pm 1.41***#	44.81 \pm 1.23
MCV (mm^3/RBC)	56.83 \pm 2.89	57.43 \pm 2.34	65.72 \pm 3.41*#	54.68 \pm 2.21	69.31 \pm 3.41*#	57.43 \pm 2.34
MCH (pg/RBC)	20.34 \pm 0.58	20.16 \pm 0.67	17.83 \pm 0.38*#	19.81 \pm 0.49	17.83 \pm 0.38*#	20.16 \pm 0.67
MCHC (g/dl)	33.84 \pm 1.61	33.73 \pm 1.53	28.26 \pm 1.28**	32.65 \pm 1.42	28.26 \pm 1.28*#	33.73 \pm 1.53
Plt ($10^3/\mu\text{l}$)	38.21 \pm 49.3	734.34 \pm 41.1	283.91 \pm 25.7*#	723.49 \pm 36.47	283.91 \pm 25.7*#	734.34 \pm 41.1
WBC ($10^3/\mu\text{l}$)	10.53 \pm 0.16	10.67 \pm 0.23	15.43 \pm 0.91*#	11.23 \pm 0.43	18.38 \pm 0.91*#	10.67 \pm 0.23

Values are expressed as means \pm SD, for eight rats in each group. Significant differences were calculated at * p < 0.05, ** p < 0.01 vs, control group; and AGE <0.05, ## p <0.01 vs, ethanol and withdrawal group. RBC: Red blood cells, Hb: Hemoglobin, Ht: Haematocrit, MCV: Mean corpuscular volume, MCH: Mean corpuscular hemoglobin, MCHC: Mean corpuscular hemoglobin concentration, Plt: Platelet and WBC: white blood cells.

Table 2 Antioxidant system (means \pm S.E.M) in the blood during chronic ethanol ingestion and ethanol withdrawal under the influence of ginger extract (n=6)

Parameter	Controls	AGE group	Ethanol group	Ethanol+ AGE group	Ethanol withdrawal group	Ethanol withdrawal + AGE group
SOD (U/g Hb)	1,534 \pm 109	1,544.03 \pm 1.76	1398 \pm 102a,b,c	1,502 \pm 115 ^b	1,237 \pm 99 ^a	1,602 \pm 115 ^b
CAT (U/g Hb)	136 \pm 8	151 \pm 1.08	129 \pm 10 ^b	134 \pm 10 ^b	108 \pm 8 ^a	141 \pm 10 ^b
GPx (U/g Hb)	31.5 \pm 2.0	35.03 \pm 1.76	27.6 \pm 2.1a,b,c	27.0 \pm 2.1 ^b	22.8 \pm 1.8 ^a	32.0 \pm 2.1 ^b
GR (U/g Hb)	18.2 \pm 1.2	14.96 \pm 0.58	16.6 \pm 1.3b	17.8 \pm 1.3 ^b	14.9 \pm 1.1 ^a	18.8 \pm 1.3 ^b
GSH ($\mu\text{mol/g Hb}$)	7.52 \pm 0.49	8.99 \pm 3.35	6.92 \pm 0.52b	7.50 \pm 0.40 ^b	6.01 \pm 0.48 ^a	7.50 \pm 0.40 ^b
GST (U/g Hb)	6.20 \pm 0.58	6.43 \pm 0.50	4.68 \pm 0.34 ^{a c}	5.93 \pm 0.50	3.41 \pm 0.26 ^{a,b}	6.13 \pm 0.85
Vitamin (mg/L) E	2.47 \pm 0.17	2.94 \pm 0.62	2.21 \pm 0.19a,b,c	2.55 \pm 0.20 ^b	1.97 \pm 0.15 ^a	2.08 \pm 0.20 ^b
Vitamin (mg/L) C	7.94 \pm 2.01	7.74 \pm 2.34	4.28 \pm 0.29 ^a	5.20 \pm 1.77 ^a	3.84 \pm 0.48 ^a	5.45 \pm 0.62
MDA ($\mu\text{mol/L}$)	1.78 \pm 0.12	1.95 \pm 0.56	1.99 \pm 0.16a,b,c	1.68 \pm 0.11 ^{a,b}	2.63 \pm 0.19 ^a	2.22 \pm 0.11 ^{a,b}

^a P <0.05 in comparison to the control group; ^b P <0.05 in comparison to the withdrawal group; ^c P <0.05 in comparison to the control group

rats, evidenced by a large production of deformed erythrocytes, associated to an increase in erythrocyte hemolytic percentage. This last finding could explain the reduction of Hb, MCH and MCHC values observed in ethanol-fed rats. In this study the ethanol-treated rats also exhibited significantly higher WBC number than the control animals.

Vit E is the primary liposoluble antioxidant, which may have an important role in scavenging free oxygen radicals and in stabilizing the cell membranes, thus maintaining its permeability (Bjørneboe et al. 1990, Navarro et al. 1999). Vitamin E may also affect oxidative changes which occur in other cell organelles (Ibrahim et al. 2000). Moreover, it is known that antioxidants, such as Vit E, coenzyme Q, vitamin C (Vit C), glutathione (GSH) and selenium may act synergically, preventing lipid peroxidation and cell destruction (Beyer 1994, Chen and Tappel 1995, Navarro et al. 1999, Lass and Sohal 2000). Vitamin C is a potent scavenger of free oxygen radicals and it has been shown that marginal Vit C deficiency results in intracellular oxidative damage in the guinea-pig (Hudécová and Ginter 1992, Nagyová et al. 1994, Tatara and Ginter 1994). In comparison to the chronic exposure,

the withdrawal group demonstrated much decreased concentrations of Vit C and Vit E. However ginger extract administration was able to restore the levels of these vitamins in the blood.

The current findings also demonstrated decreased activity of erythrocyte antioxidant enzymes during chronic alcohol intoxication and ethanol withdrawal. A decrease in the activity of erythrocyte glutathione peroxidase was earlier reported in rats chronically intoxicated with ethanol (Akkus et al., 1997; Gabbianelli et al., 2007). On the other hand, a decrease in the activity of superoxide dismutase and catalase from different tissues after ethanol intoxication was also observed (Łuczaj et al., 2004, Ostrowska et al., 2004).

The diminution in the antioxidant enzyme activity during chronic ethanol intoxication may be caused by many factors. It may be due to the inhibition of protein molecules biosynthesis, which was earlier observed in ethanol intoxication (Bengtsson et al., 1984). It is known that alcohol decreases the aqueous layer formed on the outer surface of the proteins (Chen et al., 1995), which may lead to the intramolecular interactions of polar groups of protein molecules and may cause conformational changes.

Alterations in the erythrocyte prooxidant and antioxidant levels have also been demonstrated when alcoholic cirrhotic were compared with nonalcoholic cirrhotic patients (Nalini et al., 1999) and with non-cirrhotic controls as well (Hadi et al., 1999). Moreover, significant decrease in the activity of erythrocyte SOD and GPx was observed in alcoholics with no significant change in the activity of these enzymes in plasma (Guemouri et al., 1993).

Treatment with ginger prior to ethanol withdrawal increased erythrocyte SOD, CAT and GR activities indicating that ginger eliminates the toxic effects of ethanol on the activity of these enzymes. At the same time, the activity of plasma GST and blood GSH concentration remain at the level of control values which confirm the protective role of ginger. The erythrocyte GSH-Px activity was markedly increased in ginger extract treated animals. The extract treatment before ethanol withdrawal helped to maintain the erythrocyte GSH content. The role of glutathione peroxidase (GPX) in cellular defense against oxidant attack has been discussed for many years. It is believed that the glutathione peroxidase enzyme, protects the erythrocyte against peroxides that are generated intracellularly or exogenously (Johnson et al., 2000). Glutathione reductase plays an important role in protecting hemoglobin, red cell enzymes, and biological cell membranes against oxidative damage by increasing the level of reduced glutathione (GSSG) in the process of aerobic glycolysis. The enzyme deficiency may result in mild to moderately severe hemolytic anemia upon exposure to certain drugs or chemicals. However, hereditary deficiency of the enzyme is extremely rare (change et al., 1978). Glutathione can be used to "detoxify" reactive oxygen species such as hydrogen peroxide H₂O₂, a process in which glutathione is oxidized to the dimer glutathione disulfide in a reaction catalyzed by glutathione peroxidase. Reduced glutathione in turn is regenerated from glutathione disulfide by glutathione reductase in a reaction requiring NADPH as a cofactor (Hayes and McLellan, 1999).

Earlier reports confirmed the presence of saponin, terpenoid flavonoid, steroid, and cardiac glycosides in ginger (Rozanida et al., 2005; Ghasemzadeh et al., 2010; Rice-Evans et al., 1997; Shukla et al., 2007). These phytochemicals were widely reported as quencher of free radicals in the biological system and in amelioration of various diseases associated with free radicals. Flavonoids exhibits a wide range of biological activities such as antimicrobial, anti-inflammatory, antiangiogenic, analgesic, antiallergic effects, cytostatic, and antioxidant properties (Hodek et al., 2002). Aliyu et al. 2009 reported that phenolic compounds are the major group of compounds that acts as primary antioxidant because it can react with oxygen free radicals such as hydroxyl, superoxide anion radicals and lipid peroxy radicals. A high correlation between antioxidant activity and phenolic compounds is reported (Odabasoglu et al., 2004). Thus the role of phytochemical constituents of ginger in favor of an antioxidant defense mechanism cannot be ruled out. Therefore, it may be inferred that the beneficial effect observed by the administration of *Z. officinale* aqueous

extract in rats can be attributed to these phytochemicals

Conclusion

The study presented here confirms that alcohol dependence leads to oxidative stress in peripheral blood. 72 hrs of alcohol withdrawal and chronic intoxication has induced oxidative damage in erythrocytes that leads to anemia, loss of membrane function by enhancement of LP concentration as well as alteration of the activity of AOS enzymes: SOD, CAT, GSH-Px, GR and GST and concentrations of GSH, Vit C and Vit E. Our results show that ginger extract expressed protective role against prooxidant effect of ethanol withdrawal. Thus the study suggests longer time of supplementation with potential antioxidant is required to restore the prooxidant-antioxidant balance during alcohol rehabilitation.

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Conflict of interest: Nil

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