AENSI Journals

Advances in Environmental Biology

ISSN-1995-0756 EISSN-1998-1066

Journal home page: http://www.aensiweb.com/AEB/

Effect of Acute and Chronic Exposure to Ethanol Inembryonic Stage on Homocysteine of New Hatching Chick

[1]Mohabbat Ahmadi and [2]Mahnaz Taherianfard

[1]MS graduated, Department of Physiology- School of Vet Med, Shiraz University- Shiraz, Iran.
[2]Professor, Department of Physiology- School of Vet Med, Shiraz University- Shiraz Iran.

ARTICLE INFO

Article history:
Received 2 April 2014
Received in revised form
13 May 2014
Accepted 28 June 2014
Available online 23 July 2014

Keywords:
Acute ethanol, chronic ethanol, homocysteine, chick.

ABSTRACT

Introduction: Elevated circulating total homocysteine concentrations are associated with a higher prevalence of ischemic heart disease and it seems to be related to ethanol consumption. So, the aim of present study was to investigate the effect of exposure to acute (70%) and chronic (%10) evaporated ethanol in embryonic stage on plasma concentration of homocysteine in new hatching chick. Methods: Forty five fertilized eggs were used. Eggs were divided in 3 groups, 1- Control 2- experimental that acute exposure to ethanol 3- experimental that chronic exposure to ethanol. Plasma concentration ofhomocysteine were measured with the biochemistry (enzymatic)method. Data were analyzed by one-way ANOVA and Duncan as post-hoc. The level of significant was considered $P < 0.05$. Results: Exposure to acute ethanol significantly ($P < 0.05$) decreases plasma concentration of homocysteinein new hatching chick, but exposure to chronicethanol did not any significant effect on plasma concentration of homocysteine in new hatching chick. Conclusion: Present results indicated that exposure to acute ethanol by evaporated in embryonic stage of chicken can change the plasma concentration of homocysteine.

To Cite This Article: Mohabbat Ahmadi and Mahnaz Taherianfard., Effect of Acute and Chronic Exposure to Ethanol Inembryonic Stage on Homocysteine of New Hatching Chick. ***Adv. Environ. Biol., 8(12),*** *239-241, 2014*

INTRODUCTION

Hyperhomocysteinemia is an independent risk factor for cardiovascular disease such as ischemic disease, stroke and myocardial infarction, and arterial and venous thrombotic events [14]. Disturbance of maternal and fetal homocysteine metabolism has been associated with fetal neural tube defects, Homocysteine is an amino acid that is involved in several key metabolic processes, including the methylation and sulphuration pathways [5].

Chronic alcoholism in humans is associated with the development of hyperhomocysteinemia, among the causes of hyperhomocysteinemia is depletion of folate, vitamin B12, or vitamin B6 [15]. Vitamin B12 is an essential cofactor in homocysteinemeta bolisem to methionine and vitamin B6 by cystathionine ϐ synthase converts homocysteine to cystathionine [10]. Alcohol intake disorders absorption of these vitamins and there for decrease of these materials can increases plasma concentration of homocysteine [6].

On the other hand many of the effects of alcohol is through of its product acetaldehyde(9).Acetaldehyde is established from alcohol dehydrogenase in liver [11]. This material causes defection ofmethionine synthase that converts homocysteine to methionine and there for decreases concentration of homocysteine [2]. Alcohol intake produces furtheracetaldehyde by P450-2E1 and increases plasma concentration of homocysteine [12]. According to some researches, there is special relation between plasma homocysteine and rate of alcohol consumption and it's plasma density. so that, it has been reported significant decrease of homocysteine concentration in alcoholic patients after alcohol withdrawal [4,8].

There are many literature data that linking ethanol concentration in acute and chronic form of consumption to an alteration of plasma homocysteine, but a few study were done on the effect of acute and chronic ethanol abused on new borne of alcoholic mothers even in experimental animals. On the other hand it seems that conversion pattern of methionine to homocysteine is similar in mammals and birds [7]. The aim of present study is the effects of exposure to both acute and chronic ethanol on chick plasma concentration of homocysteine in new hatching chick.

Corresponding Author: Mahnaz Taherianfard, Professor, Department of Physiology- School of Vet Med, Shiraz University- Shiraz Iran.
E-mail: taherian@shirazu.ac.ir

Advances in Environmental Biology, 8(12) July 2014, Pages: 239-241

MATERIAL AND METHODS

Forty five Cubb's fertilized eggs were purchased from Fars Company of chicken were used. Eggs were randomly divided into three groups of 15 each: 1 - Control group: 15 eggs, cotton dipped in clean water and remove surface contaminants, and then incubated in normal conditions of temperature (37.2-37.7 C°) and humidity set (60 % - 70 %) and put the chicks hatched after 21 days, the chicks were sacrificed; plasma was collected from the jugular vein of the neck and frozen the samples. 2- chronic group: 15 eggs, in normal conditions similar to control, but the difference is that humidity produced by ethanol 10% instead of water in control group during 21 days. the chicks were sacrificed, plasma was collected from the jugular vein of the neck and frozen the samples. 3 - Acute group: 15 eggs, in normal conditions similar to control, but the difference is that humidity produced by ethanol 70% on days 6, 13 and 20 of incubation instead of water. The chicks were sacrificed, plasma was collected from the jugular vein of the neck and frozen the samples. In the samples obtained from each group, the level of Homocysteine were measured by biochemistry (enzymatic) method.

SPSS software was used for data analysis. Data were analyzed by one way ANOVA and Tukey as post-hoc test. Significance level ($P < 0.05$) is considered. Data are presented as mean ± standard error in the results section.

Results:

Our data were shown that acute exposure to ethanol in embryonic stages significantly ($P<0.05$) decrease plasma concentration ofhomocysteine in newborn hatching chick; but chronic exposure to ethanol in embryonic stages did not significant effect on plasma concentration ofhomocysteine in newborn hatching chick (figure 1).

Discussion:

In the present study, exposure to acute ethanol in embryonic stages caused a significant decreases plasma concentration of homocysteine in new hatchingchick. Ayaori *et al* reported that After 4 weeks of alcohol withdrawal in healthy men, the levels of serum homocysteine did not change [1]. Stickel *et al* studies indicate that chronic alcohol consumption produces hyperhomocysteinemia in rat by a mechanism that is related to interference with one-carbon metabolism [15].

Walcher *et al* reported that ethanol-induced increased endogenous homocysteine levels in developing chick brain [17]. Van der Gaag *et al* have proved that serum homocysteine increases after moderate consumption of red wine and spirits [16]. Barak *et al* have shown that chronicethanol consumption increases homocysteine accumulation inhepatocytes by impairing in methionine synthetize activity[13]. On the base of some studies, homocysteine has antioxidant role that is resulting from its thiolgroupe [13,14].

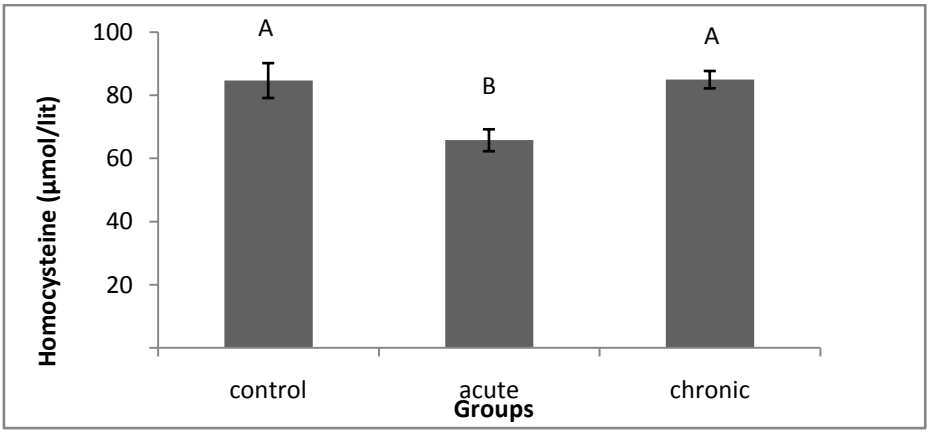

Fig. 1: Effect of acute and chronic ethanol on plasma concentration ofhomocysteine in new hatching chick. Different character represented significant difference at $P<0.05$.

REFERENCES

[1] Ayaori, M., T. Hisada, H. Yoshida, H. Shige, T. Ito, K. Nakajima, K. Higashi, A. Yonemura, T. Ishikawa, F. Ohsuzu, K. Saionji, S. Tamai and H. Nakamura, 2000. Effect of alcohol intake on the levels of plasma homocysteine in healthy males. *J. Nutr. Sci. Vitaminol. (Tokyo)*, 46: 171-174.

[2] Banerjee, R.V. and R.G. Matthews, 1990. Cobalamin-dependent methionine synthase. *FASEB J.*, 4: 1450-1459.

[3] Barak, A.J., H.C. Beckenhauer, K.K. Kharbanda and D.J. Tuma, 2001. Chronic ethanol consumption increases homocysteine accumulation in hepatocytes. *Alcohol.*, 25: 77-81.
[4] Bleich, S., K. Spilker, C. Kurth, D. Degner, M. Quintela-Schneider, K. Javaheripour, E. Ruther, J. Kornhuber and J. Wiltfang, 2000. Oxidative stress and an altered methionine metabolism in alcoholism. *Neurosci Lett.*, 293: 171-174.
[5] Hague, W.M., 2003. Homocysteine and pregnancy. *Best Pract Res Clin Obstet Gynaecol.*, 17: 459-469.
[6] Halsted, C.H., J.A. Villanueva, A.M. Devlin, O. Niemela, S. Parkkila, T.A. Garrow, L.M. Wallock, M.K. Shigenaga, S. Melnyk and S.J. James, 2002. Folate deficiency disturbs hepatic methionine metabolism and promotes liver injury in the ethanol-fed micropig. *Proc. Natl. Acad. Sci. USA*, 99: 10072-10077.
[7] Hathcock, J.N. and M.L. Scott, 1966. Alterations of methionine to cysteine conversion rates and nutritional muscular dystrophy in chicks. *Proc. Soc. Exp. Biol. Med.*, 121: 908-910.
[8] Hultberg, B., M. Berglund, A. Andersson and A. Frank, 1993. Elevated plasma homocysteine in alcoholics. *Alcohol. Clin. Exp. Res.*, 17: 687-689.
[9] Israel, Y., H. Orrego and F.J. Carmichael, 1994. Acetate-mediated effects of ethanol. *Alcohol. Clin. Exp. Res.*, 18: 144-148.
[10] Kang, S.S., P.W. Wong, H.G. Bock, A. Horwitz and A. Grix, 1991. Intermediate hyperhomocysteinemia resulting from compound heterozygosity of methylenetetrahydrofolate reductase mutations. *Am. J. Hum. Genet.*, 48: 546-551.
[11] Klyosov, A.A., L.G. Rashkovetsky, M.K. Tahir and W.M. Keung, 1996. Possible role of liver cytosolic and mitochondrial aldehyde dehydrogenases in acetaldehyde metabolism. *Biochemistry*, 35: 4445-4456.
[12] Lieber, C.S., 1990. Interaction of alcohol with other drugs and nutrients. Implication for the therapy of alcoholic liver disease. *Drugs*, 40(3): 23-44.
[13] Matte, C., F.M. Stefanello, V. Mackedanz, C.D. Pederzolli, M.L. Lamers, C.S. Dutra-Filho, M.F. Dos Santos and A.T. Wyse, 2009. Homocysteine induces oxidative stress, inflammatory infiltration, fibrosis and reduces glycogen/glycoprotein content in liver of rats. *Int. J. Dev. Neurosci.*, 27: 337-344.
[14] Perna, A.F., D. Ingrosso and N.G. De Santo, 2003. Homocysteine and oxidative stress. *Amino Acids*, 25: 409-417.
[15] Stickel, F., S.W. Choi, Y.I. Kim, P.J. Bagley, H.K. Seitz, R.M. Russell, J. Selhub and J.B. Mason, 2000. Effect of chronic alcohol consumption on total plasma homocysteine level in rats. *Alcohol. Clin. Exp. Res.*, 24: 259-264.
[16] van der Gaag, M.S., J.B. Ubbink, P. Sillanaukee, S. Nikkari and H.F. Hendriks, 2000. Effect of consumption of red wine, spirits, and beer on serum homocysteine. *Lancet*, 355: 1522.
[17] Walcher, B.N. and R.R., Jr. Miller, 2008. Ethanol-induced increased endogenous homocysteine levels and decreased ratios of SAM/SAH are only partially attenuated by exogenous glycine in developing chick brains. *Comp. Biochem. Physiol. C Toxicol. Pharmacol.*, 147: 11-16.