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Effect of Acute and Chronic Exposure to Ethanol in Embryonic Stage on Lipid Profile of Chick After Hatching

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ABSTRACT

Introduction: The lipid profiles are well-established indicator for the risk of cardiovascular 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 triglyceride, total cholesterol, HDL- cholesterol, LDL- cholesterol and VLDL-cholesterol immediately after hatch of 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 of triglyceride, total cholesterol, HDL- cholesterol, LDL-cholesterol and VLDL-cholesterol were measured with the radioimmunoassay kit. 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 ($P < 0.05$) increases plasma HDL-cholesterol, LDL- cholesterol and total cholesterol concentration immediately after hatch of chick. Exposure to chronic ethanol significantly ($P < 0.05$) increases plasma LDL- cholesterol, VLDL-cholesterol, triglyceride and total cholesterol concentration immediately after hatch of chick. **Conclusion:** Present results indicated that exposure to acute and chronic ethanol by evaporated in embryonic stage of chicken can change the plasma concentration of lipid profile.

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INTRODUCTION

Disorders related to alcoholism is the main causes of the loss of health. According to reports, this material decreases the age of concumers at least 10 years. Based on the experience of life insurance companies, it have proven that lifetime in alcohol addicts person is about 25 % -30 % lower than the others. According to a study from the United States in 1963, about 100000 deaths were associated with alcohol consumption [1].

Many of the chronic effects of alcohol is through it's metabolism by alcohol dehydrogenase and microsomal pathways of ethanol oxidizing system. Alcohol dehydrogenase pathway, which metabolizes alcohol to Acetaldehyde is then converted to acetate. In both of these reactions , NAD are reduced to NADH. Excessive NADH leads to metabolic disorders such as increased in uric acid, hypoglycemia, hyperlipidemia and Hypoproteinemia [2]. Alcohol in Krebs cycle is increased the reaction of oxaloacetate to maleate so it increase the maleate. As a result , the Krebs cycle activity decreases and inhibits fatty acid oxidation. On the other hand, acetate that produced from ethanol oxidation convert into acetyl- coenzyme A, the precursor for the synthesis of fatty acids, so the lipid production is increased [3].

There are many litrature data that linking ethanol concentration in acute and chronic form of consumption to an alteration of plasma lipids, but a few study were done on the effect of acute and chronic ethanol abused on newborne of ahcholic mothers even in experimental animals (*1). On the other hand it seems that chick is better than rat for this study; because the main location of lipid metabolism in chick and man is liver, but in rat is fatty tissue [4].

The aim of present study is the effects of exposure to both acute and chronic ethanol on chick plasma lipid levels, including the level of HDL - cholesterol, LDL - cholesterol, VLDL cholesterol, total cholesterol and triglycerides in new haching chick.

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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 icubation 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 total cholesterol, triglycerides, and total amount of HDL - cholesterol, LDL - cholesterol and VLDL - cholesterol were measuread by an enzymatic calorimetric method using biochemistry kits.

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 and chronic exposure to ethanol in embryonic stages significantly ($P<0.05$) increses plasma concentration of total cholesterol in newborn hatching chick; also plasma concentration of total cholesterol was significantly higher in acute than chronic groups (figure 1). Chronic exposure to ethanol in embryonic stages significantly ($P<0.05$) increses plasma concentration of triglyceride in newborn hatching chick; but acute exposure to ethanol in embryonic stages had no effect on plasma concentration of triglyceride (figure 2). Acute exposure to ethanol in embryonic stages significantly ($P<0.05$) increses plasma concentration of HDL cholesterol in newborn hatching chick; but chronic exposure to ethanol in embryonic stages had no effect on plasma concentration of HDL cholesterol (figure 3). Acute and chronic exposure to ethanol in embryonic stages significantly ($P<0.05$) increses plasma concentration of LDL cholesterol in newborn hatching chick; also plasma concentration of LDL cholesterol was significantly higher in acute than chronic groups (figure 4). Acute and chronic exposure to ethanol in embryonic stages significantly ($P<0.05$) increses plasma concentration of VLDL cholesterol in newborn hatching chick (figure 5).

Discussion:

In the present study, exposure to acute and chronic ethanol in embryonic stages caused a significant increase in plasma total cholesterol chick that this increase was higher in the acute group. Costatin *et al* studies indicate additive effects of acute and chronic ethanol intake on plasma triglyceride levels has been significant increase in the acute group [8] On the other hand, Romeo and colleagues in 2008, based on experiments on some Spanish men and women, reported that moderate alcohol consumption had no effect on plasma total cholesterol [9]. According to the research, the main responsible for the increase in total cholesterol is Acetaldehyde that produced from ethanol oxidation into acetyl-coenzyme A, which is Finally, the precursor for cholesterol synthesis from acetyl coenzyme A [10]. Acute exposure to ethanol had more potent effect on plasma concentration of total cholesterol than chronic exposure to ethanol, it may be because of acute exposure to ethanol in addition to alchol dehydrogenase pathway microsomal pathway also become activated [11].

In the present study, exposure to chronic ethanol in embryonic stages caused a significant increase in plasma total cholesterol chick. Costatin et al studies indicate acute and chronic ethanol intake increased plasma triglyceride levels that was more potent in acute group [8]. In humans, based on experiments indicated that the combination of fat and alcohol has a positive effect on plasma triglyceride concentration, because alcohol can suppress colon cleansing from chylomicrons derivatives [12, 13]. Hojnacki et al, reported that in monkeys high dose of alcohol has no effect on plasma triglyceride levels [14]. This lack of effect has been observed on serum triglycerides in some women and men whose consume medium dose of alcohol [9]. Mennen et al indicated that triglyceride levels (triglycerides by measuring chylomicrons) an increase or no change in the group with moderate consumption of ethanol and unchanged were associated with heavy consumption [15].

In the present study, exposure to acute ethanol in embryonic stages caused a significant increase in plasma total cholesterol chick. Rimm *et al* concluded that alcohol consumption increases the concentration of HDL - cholesterol [16]. Costatin *et al* studies indicate that chronic ethanol Increases the HDL - cholesterol [8]. Research on monkeys has shown that the use of low-dose alcohol does not have effect on HDL – cholesterol, whereas higher doses, increases HDL - cholesterol levels Other study have shown that alcohol consumption increases HDL – cholesterol in two forms, studies on the rate of chronic ethanol Costatin et al showed additive s of, whereas, according to this group, the rate factor in acute and control groups compared to almosthas remained

unchanged in others [15] effects on serum HDL - cholesterol is not, Increased levels of HDL - cholesterol in some women and men who on average consume alcohol sees, have been reported [9], ies in the amount of HDL - cholesterol: 1 - directly increased hepatic production and secretion of apolipoproteins and lipoproteins; 2 – reduction of HDL - cholesterol uptake in circulation [17].

In the present study, exposure to acute and chronic ethanol in embryonic stages caused a significant increase in plasma LDL - cholesterol chick that this increase was higher in the acute group. Hojnacki *et al* also showed that in monkeys high-dose drinking of ethanol was increased LDL – cholesterol whereas lower doses did not have any effect on LDL – cholesterol [14]. Peebles *et al* reported that oral ethanol intake has been associated with decreased in LDL - cholesterol [19]. Chronic ethanol solution in adult rats, lead to severe reduction in LDL – cholesterol; while acute ethanol solution had no effect on LDL – cholesterol [8]. It seems that ethanol lead to inhibition of hepatic lipase, so was increased the LDL – cholesterol [17, 21].

In the present study, exposure to acute and chronic ethanol in embryonic stages caused a significant increase in plasma VLDL - cholesterol chick. Sane *et al* was shown an increase in hepatic VLDL particle production is influenced by acute alcohol [22]. Oral ethanol intake in chickens, is reduced the amount of VLDL - cholesterol [19]. Mennae *et al*, reported that with moderate ethanol consumption the VLDL – cholesterol had no change, but high ethanol consumption is reduced the amount of VLDL – cholesterol [15].

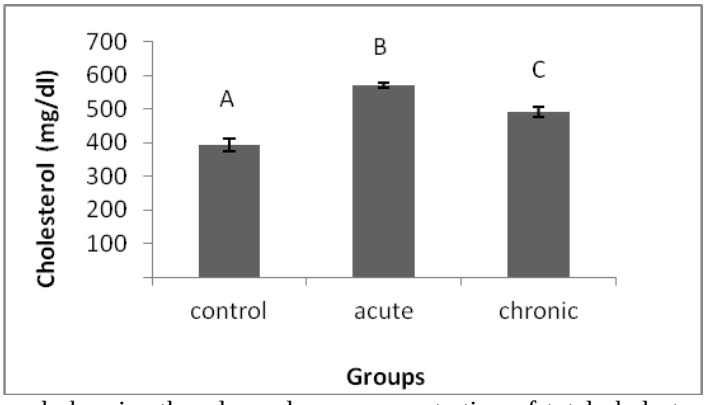

Fig. 1: Effect of acute and chronic ethanol on plasma concentration of total cholesterol in immediately after hatch of chick. Different character represented significant difference at P<0.05.

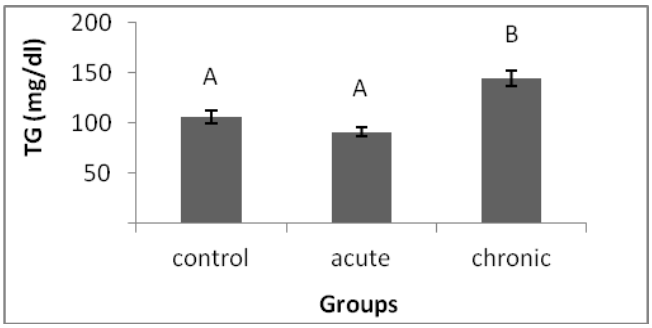

Fig. 2: Effect of acute and chronic ethanol on plasma concentration of troglyceride in immediately after hatch of chick. Different character represented significant difference at P<0.05.

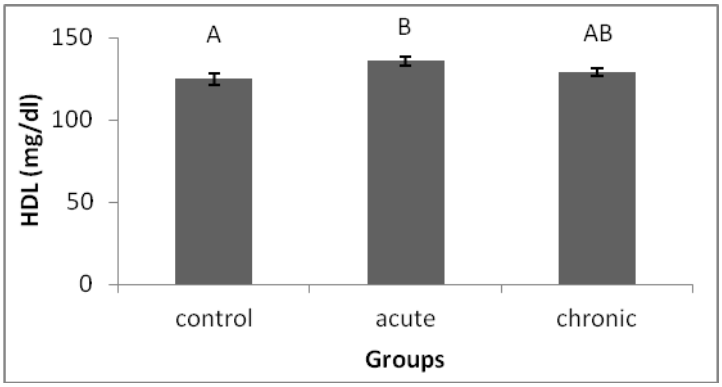

Fig. 3: Effect of acute and chronic ethanol on plasma concentration of HDL cholesterol in immediately after hatch of chick. Different character represented significant difference at P<0.05.

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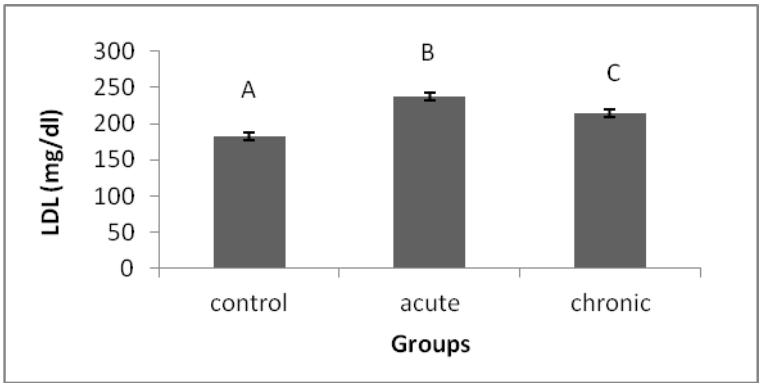

Fig. 4: Effect of acute and chronic ethanol on plasma concentration of LDL cholesterol in immediately after hatch of chick. Different character represented significant difference at P<0.05

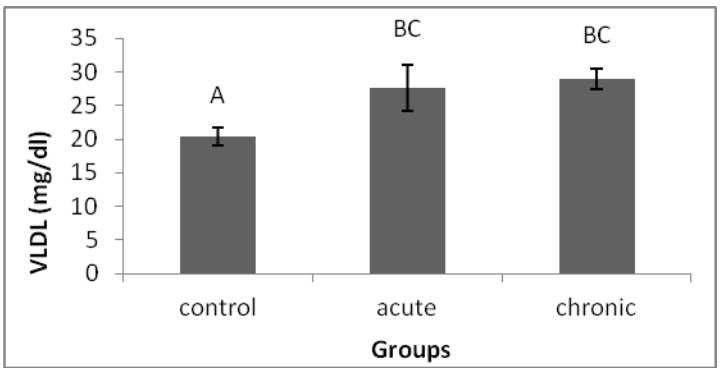

Fig. 5: Effect of acute and chronic ethanol on plasma concentration of VLDL cholesterol in immediately after hatch of chick. Different character represented significant difference at P<0.05

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