THE EFFECT OF THE LATERAL CIGARETTE SMOKE ON SERUM LIPID PROFILES IN DIABETIC RATS

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ABSTRACT

According to the report of WHO, one person every 8 seconds (annual three million) loses her/his life due to smoking. The lateral smoke of a cigarette is the one that the people around the smoker inhale and it is more toxic than the main smoke of a cigarette. Nonsmokers who inhale cigarette smoke indirectly are called passive smokers. In this study, the effect of passive cigarette smoke inhalation on lipid profiles in diabetic rats was evaluated.

In this intervention study, 24 male Wistar rats weighing 200 to 220 g randomly divided into 4 groups of 6 animals: healthy control, diabetic control, smoker treatment and smoker-diabetic treatment groups. In the diabetic control and treatment groups the diabetes was induced with a single dose (65mg / kg) injection of streptozotocin in the IP form. The blood sugar levels of rats were measured using a glucometer after 24 hours; so, the rats with higher glucose levels than 250 mg / dl were considered as diabetic. The healthy treatment and diabetic treatment groups were exposed to the inhalation of passive smoke of a cigarette burned during 1 to 2 minutes for 30 days routinely. After one month, the rats were decapitated and the blood samples were obtained. The samples were centrifuged and the serum was separated. The amounts of serum lipid profiles, including triglyceride, total cholesterol, LDL, and HDL were measured using diagnostic kits and spectrophotometric method and the obtained results were analyzed by ANOVA and Tokay test. During the study, all groups were exposed to the same environmental and light conditions as well as unlimited access to food and water. Statistical comparison of the results obtained in this study showed that there is no significant difference serum LDL and HDL levels in Treatment group. In conclusion it can be said that smoking causes the LDL increase and the HDL decrease which are in turn the risk factors for cardiovascular diseases.

Keywords: Cigarette, Passive Smoke, Lipid Profiles, Diabetic Rats

INTRODUCTION

According to forecasts, in the next 30 years, 10 million people in every year will be added to the addicted people to smoking that 70% of the victims will be from developing countries (Zenzes, 2000). Based on published papers it has been demonstrated that cigarette smoke has s significant effect on mandatory or voluntary companions of a smoker besides him/her (Zenzes, 2000). Although Nicotine has been known as the most toxic component of cigarette, many of the effects of smoking, such as cardiovascular disease, respiratory disease, cancers, especially lung, throat and mouth cancer are not related to the nicotine, but are related to the other toxins and carcinogens in the smoke or extract of tobacco (Ahmadi *et al.,* 2001; Benowitz, 1988).

Two types of smoke produced by burning tobacco:

1) Main Smoke (active smoke): the smoke inhaled by the smoker.

2) Lateral smoke (passive smoke): smoke from burning tobacco, which is inhaled by the people around smokers (these kind of non-smoker inhalers is called passive smokers) and very toxic than the main smoke. The mandatory exposition to cigarette smoke for 30 minutes can harm the non-smoker's heart and increase the risk of heart disease by 30 percent. Lateral cigarette smoke contains 5 times more carbon monoxide and 6 times more nicotine than that smokers inhale because cigarette has a protective filter. The cigarette smoke has over 4000 toxic and mutagenic components such as carbon monoxide, aromatic hydrocarbons, and nicotine (Carvalho *et al.,* 2006). Cigarette smoke contains a range of oxidants and free

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radicals that can increase lipid peroxidation. About 10^{14} free radicals enter the lungs in every inhalation of smoke. Free radicals can directly or indirectly induce oxidative stress in the body (Hecht, 2002). So, the effect of passive cigarette smoke on serum lipid profiles in diabetic rats was investigated. Cigarette smoke is one of the factors in oxidant production and free radicals. It must be noted that adding some aromatic substances in cigarettes has an important role in increased damages caused by free radicals (Dasgupta *et al.,* 2009). Free radicals can increase Oxidized LDL (OX-LDL) via different mechanisms such as lipid peroxidation, inhibition of protein production, and the reduction of adenosine 3-phosphate on the effect of lipid peroxidation, which can in turn be related to the production and secretion of interleukin 1 (IL-1) from macrophages and cytokine secretion. Of course, it must be considered that HDL can have a protective role and inhibit the cholesterol and oxidized LDL damaging effects (Garrido *et al.,* 2000). In one study, a significant increase in total cholesterol and LDL levels in smokers has been reported compared with nonsmokers (Venkatsan *et al.,* 2006). In another study a significant increase in cholesterol and triglycerides and significant HDL reduction in rats receiving nicotine compared with controls have been reported (Valenca *et al.,* 2008).

MATERIALS AND METHODS
In this intervention study, 24 male Wistar rats weighing 200 to 220 g randomly divided into 4 groups of 6 animals: healthy control, diabetic control, smoker treatment and smoker-diabetic treatment groups. The rats were kept in boxes made of polypropylene with dimensions 30 x 30 x 60 cm. In the diabetic control and treatment groups the diabetes was induced with a single dose (65mg / kg) injection of streptozotocin in the IP form. The blood sugar levels of rats were measured using a glucometer after 24 hours; so, the rats with higher glucose levels than 250 mg / dl were considered as diabetic. The healthy treatment and diabetic treatment groups were exposed to the inhalation of passive smoke of a cigarette burned during 1 to 2 minutes for 30 days routinely. After one month, the rats were decapitated and the blood samples were obtained. The samples were centrifuged and the serum was separated. The amounts of serum lipid profiles, including triglyceride, total cholesterol, LDL, and HDL were measured using diagnostic kits and spectrophotometric method and the obtained results were analyzed by ANOVA and Tokay test. During the study, all groups were exposed to the same environmental and light conditions as well as unlimited access to food and water. In the control group, no intervention was applied.

RESULTS AND DISCUSSION
Results
The mean values of cholesterol (mg/dl) in the groups: Average levels of cholesterol were measured in different groups, and the results were compared using ANOVA test at a possibility level of 95% and a significant level of 0.05 were compared and the results are shown in Table 1

Table 1: Comparison of cholesterol (mg/dl) in understudied groups

Group	Mean±SE	SD	Sig (P value)
Smoker-diabetic treatment	83.63±5.54	15.69	
Smoker treatment	79.00± 5.13	12.57	0.40
Diabetic control	87.50±7.65	18.75	
Healthy control	66.5±4.50	6.36	

Table 2: Comparison of triglyceride (mg/dl) in understudied groups

Group	Mean±SE	SD	Sig (P value)
Smoker-diabetic treatment	63.38±7.52	21.28	
Smoker treatment	63.50±6.28	15.39	
Diabetic control	63.50±7.98	19.55	0.55
Healthy control	43.00±3.00	4.24	

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According to the obtained results from this study, there was no significant statistical difference in cholesterol among the groups (P>0.05). Also, the highest and lowest levels of cholesterol were in diabetic-control and healthy-control groups, respectively.

The mean values of triglyceride (mg/dl) in the groups: Average levels of triglyceride were measured in different groups, and the results were compared using ANOVA test at a possibility level of 95% and a significant level of 0.05 were compared and the results are shown in Table 2.

According to the obtained results from this study, there was no significant statistical difference in triglyceride among the groups (P>0.05). Also, the highest level of triglyceride was in smoker-treatment and diabetic-control and the lowest level of triglyceride was in the healthy-control group.

The mean values of HDL (mg/dl) in the groups: Average levels of HDL were measured in different groups, and the results were compared using ANOVA test at a possibility level of 95% and a significant level of 0.05 were compared and the results are shown in Table 3

Table 3: Comparison of HDL (mg/dl) in understudied groups

Group	Mean±SE	SD	Sig (P value)
Smoker-diabetic treatment	28.61±2.25[a]	6.37	
Smoker treatment	26.73±0.99 [a]	2.44	
Diabetic control	40.83±4.38[b]	10.73	0.01
Healthy control	43.25±2.85[b]	4.03	

the different letters show the dignificant statistical difference.

According to the obtained results from this study, there was a significant statistical difference in HDL among the groups (P<0.05). Also, the highest and lowest levels of HDL were in healthy-control and smoker-treatment groups, respectively.

The mean values of LDL (mg/dl) in the groups: Average levels of LDL were measured in different groups, and the results were compared using ANOVA test at a possibility level of 95% and a significant level of 0.05 were compared and the results are shown in Table 4

Table 4: Comparison of LDL (mg/dl) in understudied groups

Group	Mean±SE	SD	Sig (P value)
Smoker-diabetic treatment	42.33±5.71[b]	16.17	
Smoker treatment	39.56±4.03[a]	9.89	
Diabetic control	33.96±3.30[ab]	8.08	0.01
Healthy control	14.65±1.05[a]	1.48	

the different letters show the dignificant statistical difference.

According to the obtained results from this study, there was a significant statistical difference in LDL among the groups (P<0.05). Also, the highest and lowest levels of LDL were in the smoker- diabetic-treatment and healthy-control groups, respectively.

Discussion

In a study conducted on the students of Ankara University (22 smokers and 22 nonsmokers), significant differences were observed between the two groups in levels of total cholesterol, HDL and LDL, which are inconsistent with the results of the present study, but is consistent with the results of this study in LDL and HDL (Erguder *et al.,* 2009).

In the study conducted by Antoniades there was a significant increase in total cholesterol, serum triglycerides, and LDL and a significant decrease in HDL in smokers compared with the control group, which is consistent with the results of this study in LDL and HDL (Holloway *et al.,* 2005).

In this study the significant increase in LDL as well as a significant decrease in HDL of smokers was proved, which was in contrast with the results obtained by Ergunder *et al.,* (2009).

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Tai ES and colleagues showed that total cholesterol levels did not differ between smokers and non-smokers, which is consistent with the results of this study (Tai and Tan, 2004).

In a study in which the rats were exposed daily to smoke from burning tobacco for a month, there was a significant increase (p <0.01) in serum total cholesterol and triglyceride levels in smokers compared with the control group which is completely in contrast with the results of the present study (Hakki *et al.,* 2001). Previous studies indicate that tobacco consumption causes a decrease in serum HDL, which is consistent with the results of this study (Yang and Liu, 2004). In conclusion, it can be said that passive tobacco smoke causes the increased serum LDL and decreased HDL, which are considered the risk factors for cardiovascular disease.

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