

Effect of Partial Sleep Deprivation on Lipid Profile in High Fat Diet-Fed Rats in the Presence and Absence of Vitamin C

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Article information	Abstract
<p>Article history: Received: 26 Aug 2011 Accepted: 10 Sep 2012 Available online: 7 Jan 2013 ZJRMS 2013; 15(7): 12-18</p>	<p>Background: The daily stress and shift working cause insomnia. In other hands, fatty food consumption increased this disorder. The aim of present study is evaluation additive effect of partial insomnia and high fatty diet with or without vitamin C on serum lipid profile in rats.</p>
<p>Keywords: Insomnia High fatty diet Vitamin C Lipid profile Rat</p>	<p>Materials and Methods: Fifty six rats in 7 groups (8 rats each group) were conducted for study during 26 days as: 1: normal diet+normal sleep, 2: high fatty diet+normal sleep, 3: normal diet+insomnia, 4: high fatty diet+insomnia, 5: high fatty diet+normal sleep+vitamin C, 6: high fatty diet+insomnia+vitamin C, 7: normal diet+insomnia+vitamin C. The lipid profile was examined at end of study.</p>
<p>*Corresponding author at: Department of Pharmacology, Shahid Chamran University, Ahvaz, Iran E-mail: najafzadeh@scu.ac.ir</p>	<p>Results: Results shown the high fatty diet+insomnia increased triglyceride, LDL, VLDL level and decreased HDL level with comparison to high fatty diet+normal sleep group. But only insomnia did not change serum lipid profile. High fatty diet increased level of cholesterol ($p<0.05$). The normal diet increased body weight but high fatty diet decreased it significantly. Liver weight ratio was elevated by high fatty diet+insomnia. The vitamin C decreased cholesterol and increased HDL level in group of rats which received high fatty diet+insomnia.</p>
	<p>Conclusion: In conclusion, the present study shown the only insomnia did not affect on serum lipid profile while insomnia along with high fatty diet increased lipid high risk factors in blood.</p>

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Introduction

Everyday stresses are today's machinery life features that can cause interfering with normal sleep. Decrease in sleep duration in today's modern societies not only may have a role in causing diabetes mellitus, but also can be involved in the incidence of hypertension [1]. Today's immobile and sedentary form of life along with lack of sleep and depression are considered as the risk factors in type 2 diabetes [2].

Also, working shifts in some jobs, especially in medical-related businesses, transportation systems and factories, with three consecutive shifts have led to that such occupants cannot sleep enough at nights. On the other hand, the increasing tendency of human societies to pre-prepared food diets, which are generally rich in fat, and consequences such as cardiovascular diseases are other problems that have made the situation worse [3].

The relative sleepiness causes the increased risk of diabetes, increased activity of the sympathetic system, decreased satiety hormone or Leptin and increased hunger hormone or Ghrelin [4]. The sleep shortness is associated with increased body mass index, however, the compensatory sleep on weekends as well as on vacations in children leads to reduced risk of overweight and obesity [5].

In a study in Japan, it was determined that the prevalence of insomnia in men is between 17.3 to 22.3% and in women between 20.5 to 21.5%; the dependent factors to insomnia in adults include age, gender, unemployment, poor health status, psychological stress, lack of physical activity, use of sleeping medications, and in youth, the insomnia-related factors include gender, mental health, life style (including not-eating breakfast, sleeping late at night) [6]. Crispim et al. showed that the workers who work at night involve fat metabolism disorders due to chronic insomnia and changes in food the patterns [7].

On the other hand, vitamin C with its antioxidant and protective properties may be able to prevent the complications caused by consuming fat dietary as well as complications due insomnia. Thus, considering the dual problems of partial insomnia as well as increased tendency to consumption of fat diets, the ultimate goal of this study is to study the combined effect of partial sleep deprivation and high fat diet on the profile of blood fats, and also the influence of vitamin C in diet on the complications due to sleep deprivation and high fat diet in rats.

Materials and Methods

In this experimental study, the method of developing partial sleep deprivation in animal model was used in rats. In order to do the present study, 56 male rats of Wistar race, weighted average 180-250 g, were prepared from the laboratory animals section of JondiShapur University of Medical Sciences, Ahvaz. The animals were kept exposed to 12-hour light cycle and in a temperature of 20-25°C, and the study was performed on rats for a period of 26 days. The rats were randomly divided into 7 groups, each consisting of 8 rats, and had freely access to food and water. Group 1 (control): Normal diet and normal sleep; Group 2: Fat diet and normal sleep; Group 3: Normal diet and applied sleep deprivation; Group 4: Fat diet and applied sleep deprivation; Group 5: Fat diet, normal sleep and received water-soluble vitamin C (1 g/l); Group 6: Fat diet, applied sleep deprivation and received water-soluble vitamin C; Group 7: Normal diet, applied sleep deprivation and received water-soluble vitamin C.

The weights of all groups were measured once every 5 days. After 26 days, the rats were anesthetized by ether, and blood samples were collected from the rats' hearts and the serum samples were separated. The rats were soft-killed by ether; their livers were removed to measure their weights.

High-fat food preparation method: To produce one kilogram of high-fat diet, 484 g of milled sugar, 10 g of cholesterol, 5 g of colic acid, 250 g of coconut oil with the milled mouse food were mixed together.

The structure and working method of the sleep-deprivation inducing device in rats: This device is composed of a plate that is hanged from its four sides from a desk on a vertical form. This plate is connected to an engine, and the engine is also connected to a timer. The engine vibrates the plate every 5 minutes for 30 seconds horizontally at 150-180 times per minutes. The cages of the rats were placed on this page; hence, the rats could not sleep in any way. The rats could sleep for 1 hour per day (13-14 pm) without disturbance.

Biochemical tests: The biochemical tests included measurements of total cholesterol, triglycerides, HDL-c, LDL-c, VLDL-c that were performed using diagnostic test kit made by Pars Azmoon Company. The VLDL-c values were obtained by calculation. The VLDL cholesterol amount is equal to one-fifth of the triglyceride content [8]. By subtraction the sum of HDL-c and VLDL-

c from the total cholesterol, the LDL cholesterol amount was calculated. Statistical analysis: The SPSS-16 software was used to compare the results of this study. To compare the mean results in different groups, the ANOVA test, and then, the LSD test were used. Also, for comparing the first day weights and the last day weights, ANOVA test with repeated measurement and paired *t* was performed. The significance level was considered as $p < 0.05$, and all the results were presented as Mean \pm SD.

Results

During the 6 weigh-in stages in days 1, 5, 10, 15, 20 and 25 at the test time, the weights changes of the rats showed that while a significant weight gain ($p=0.005$) was observed in control group (normal sleep+normal food), third group (sleep deprivation+normal food) and the seventh group (sleep deprivation+normal food+vitamin C), by giving the high fat diet to other groups with, significant weight loss ($p=0.001$) was seen. In this study, in groups that had received high-fat diet, the ratio of liver weight to their body weight increased significantly compared to the groups that had received a normal diet ($p=0.001$) (Table 1).

In this study, the serum cholesterol concentrations increased significantly in groups that had received high-fat diet compared to the groups that had received a normal diet ($p=0.001$) (Table 1). The serum triglyceride concentrations in group 6 (fat diet+sleep deprivation+vitamin C) had a significant increase compared to all groups except group 4 (fat diet, sleep deprivation) (Table 1). The concentration of serum HDL in group 4 (fat diet+sleep deprivation) (19.12 ± 2.3) had a significant decrease compared to the group 2 (fat diet+normal sleep) (32 ± 2.7) ($p=0.001$) (Table 1). The concentration of serum LDL in group 4 (fat diet+sleep deprivation) (138.4 ± 19.97) had a significant increase compared to the group 2 (fat diet+normal sleep) (101.4 ± 12.45) (Table 1). The concentration of serum VLDL in group 4 (fat diet+sleep deprivation) (25.1 ± 4.68) had a significant increase compared to the group 3 (normal diet+sleep deprivation) (13.1 ± 2.87) ($p=0.02$). But, the concentration of serum VLDL in group 6 (fat diet, sleep deprivation+vitamin C) (29.8 ± 3.3) had a significant increase compared to the group 5 (fat diet+normal sleep+vitamin C) (20.22 ± 2.87) ($p=0.001$) (Table 1).

Table 1. Mean \pm SD of the ratio of liver weight to the body weight and lipid profile in various groups

	Liver weight to body weight ratio	Total cholesterol	Triglycerides	HDL-c	LDL-c	VLDL-c
Group 1	0.037 \pm 0	78.6 \pm 3.38	89.25 \pm 9.79	25.87 \pm 0.6	34.9 \pm 4	17.85 \pm 1.95
Group 2	0.053 \pm 0.002	152.5 \pm 13.25	95.5 \pm 19.27	32 \pm 2.7	101.4 \pm 12.4	19.1 \pm 3.85
Group 3	0.037 \pm 0.001	80.4 \pm 7.8	65.57 \pm 14.35	27 \pm 1.29	40.3 \pm 5.39	13.1 \pm 2.87
Group 4	0.059 \pm 0.001	182.6 \pm 22.3	125.5 \pm 23.4	19.12 \pm 2.3	138.4 \pm 19.97	25.1 \pm 4.68
Group 5	0.056 \pm 0.001	188.25 \pm 20.6	101.1 \pm 14.39	25.37 \pm 1.6	142.65 \pm 19.8	20.22 \pm 2.87
Group 6	0.059 \pm 0.001	125.37 \pm 13.27	149 \pm 16.9	27.7 \pm 1.7	67.82 \pm 11.99	29.8 \pm 3.3
Group 7	0.036 \pm 0.005	68.25 \pm 3.29	38.75 \pm 4.37	24.75 \pm 0.75	35.75 \pm 3.53	7.75 \pm 0.87

Discussion

In the present study, the normal sleep and partial sleep deprivation as well as vitamin C have had no significant effect on the rats' weights [9, 10]. Increased sleep time in people with short-time sleep may help in preventing the weight gain or facilitating the weight loss. The groups of rats that had received a normal diet, had normal weight gain during the 26 days and in those in groups that had received fat diet, the weight loss was observed. This weight loss can be perhaps related to several factors. The first factor can be the kind and the form of the received food by the rats that was paste-like, since the rodents usually chew relatively hard foods due to their teeth shape, this change in the kind and form of the food might have led to decreased food intake and thus the weight loss. The second factor is that 25% of coconut oil has been used in this diet, which contains medium-chain triglycerides. In a study, Geliebter et al. compared the effect of rations containing medium-chain triglycerides (MCT) and long-chain triglycerides (LCT) on body weight with each other and concluded that diets containing MCT cause the significant weight loss than the diets containing LCT [11].

Consistent with previous studies, Tsuji et al. found similar results [12]. St-Ong et al. examined the MCT-containing oil on the men who were overweight in a study and observed that diets containing MCT result in significant reduction in fat stores compared to the LCT diet [13]. Thirdly, the studies suggest on the other hand that despite that sugar is the main material used in the Desserts and Snacks, if the received total energy is proper and isotonic, it will not cause obesity. The effects of high sucrose diet (60%), high-fat diet (60%) and standard diet (55% starch) were compared with one another on body weight of rats in 2 weeks, and it was observed that weight gain in high-fat diet group was significant compared to the two other groups and the two other groups were not significantly different. They concluded that when the same amount of calories is received, the diet with high sucrose, even containing 60% sucrose, will not lead to obesity in thin rats or weight gain in fat rats [14]. Also, in some other studies, the effects of high fat diets, high sucrose diets and normal diets were compared with separately with one another, but weights of groups had no significant differences [15, 16].

The growing increase of obesity is often associated with high consumption of food fats [17, 18]. The rodents similar to humans prefer high-fat diet [19, 20], especially rats that will gain weight with access to high-fat diet [21]. Fu et al. studied the effects of high fat and high sucrose diets (containing 16.2% fat and 15% sucrose) as well as the chronic stress effects on the rats for 12 weeks and found that the weights of groups received high-fat diet were more than the control groups, but the weight gain was not statistically significant. While the weights of groups, which had received fat diets and also the chronic stress was applied to them, had decreased significantly [22]. In our study, the sleep deprivation had no effect on body weight. Many studies have been conducted

regarding the effect of sleep deprivation on body weight. Koban and Stewart, with excluding the rats from sleep for 10 to 12 days observed that the sleep deprivation leads to increased food intake and weight loss [23]. With sleep deprivation for 5 days in rats, it was observed their food consumption increased and their weights decreased [24]. Barf et al. found in a study conducted on sleep disorders that sleep restriction as well as disturbance in sleep can cause reduced body weight without influencing on food intake [25].

In contrast with previous studies, Watanabe et al., in a one-year study conducted on men, observed that people with shorter-time sleep have a greater weight gain [26]. It was also observed that sub-chronic sleep deprivation (4 days) will cause weight loss compared to the first day and the control group, and chronic sleep deprivation (21 days) leads to increased weight compared to the first day and the control group [27]. It was shown in several widespread reviews that one reason for obesity may be the reduced amount of sleep in people, because sleep deprivation can destroy the structure of hormones regulating glucose metabolism and the appetite [28, 29]. In the present study, vitamin C had no effect on body weight, while vitamin C has caused changes in body weight in other studies. Schorah et al. observed that 2 months of taking vitamin C in elderly will cause slight but significant increase in mean body weight [30]. On the other hand, it was reported that consumption of 13200 mg/kg of vitamin C in Hindi guinea pigs for 5 weeks will lead in weight loss [31].

In the present study, in groups that had received high-fat diet, increased ratio of liver weight to body weight was observed, and sleep deprivation, alone, had no effect on the ratio of liver weight to body weight; while, sleep deprivation with high-fat diet caused significant increase in liver weight to body weight ratio compared to the group with normal sleep and having high-fat diet. Vitamin C had no significant effect on the ratio of liver weight to body weight. Consistent with this study, numerous studies suggest that high-fat diet consumption leads to increased liver weight in rats [32]. With a high fat diet for 8 weeks in rats, a significant increase in liver weight was observed [33]. Also in consumption of high fat diet for 8 weeks, similar results were obtained. While it was found in another study that giving high-fat diets (containing 15% soybean oil) for 2 weeks to the rats, the liver weight will not change significantly [34]. Not much information is available on sleep deprivation and vitamin C effects on liver weight. In this study, receiving high-fat diet increased the serum cholesterol level, and sleep deprivation made no change in serum cholesterol levels. Taking extra vitamin C levels in the diet caused reduced cholesterol levels only in the group receiving fat diet simultaneous with sleep deprivation.

In line with the results of this research, the effect of a variety of stresses on fat metabolism was examined in 22 hours a day for 4 consecutive days in rats and it was observed that sleep deprivation sleep as a kind of stress did not any changes in total cholesterol levels [35]. Also,

by inducing sub-chronic (4 days) and chronic (21 days) sleep deprivation on blood parameters related to cardiovascular diseases in the rats, it was reported that the cholesterol levels has had no significant changes in sub-chronic and chronic sleep deprivation [27]. Antunes et al. in comparing the effects of sleep deprivation in male rats and in healthy and ovariectomized female rats on blood parameters related to cardiovascular diseases showed that the cholesterol levels in sleep-deprived male rats compared to control male rats had no significant change, and in sleep-deprived female rats (Strauss phase and de-Strauss phase) a significant reduction in blood cholesterol levels compared to the sleep-deprived male rats was observed [36].

Andersen et al. in studying the effect of sleep deprivation on blood parameters related to the cardiovascular system diseases in older mice concluded that the sleep deprivation had no significant effect on blood cholesterol levels in young rats (3 months old) and aged rats (22 months old) [37]. Adamkova et al. by studying the effect of sleep deprivation on 18-65 years old men and women proved that the short sleep had no impact on blood total cholesterol levels [38]. Similar to the results obtained in this study, it has been shown in many studies that with consumption of high fat or hypercholesterolemic diet in laboratory animals, the blood cholesterol levels significantly increase [39-41].

Fu et al., with inducing stress on rats and the simultaneous consuming of high-fat and high-sucrose diets (containing 16.2% fat and 15% sucrose) for 12 weeks observed significant increased serum cholesterol levels [22]. In this study, vitamin C only in one of the groups that had received high-fat diet caused significant reduced cholesterol levels and had no effect on the rest. Perhaps, higher doses should be used for more effectiveness of vitamin C. In review of vitamin C and selenium effects on the inhibition of cholesterol oxidation in rats, it was observed that no change occurred in serum cholesterol levels in the group receiving high-fat diet and the group receiving fat diet associated with vitamin C (100 mg/kg) and selenium (200 mg/kg), and the amount of cholesterol oxidation products was doubled in the high fat diet group compared to the control group and when the antioxidants were added to the diet, the amount of products caused by cholesterol oxidation was as the same in the control group [42]. Also, Abdollahzadeh et al. observed a significant reduction in serum total cholesterol levels following the prescribing of 250 mg of vitamin C supplementation in hemodialysis patients for 12 weeks [43].

In the present study, each of the cases of inducing sleep deprivation and fat diet intake did not alone cause changes in serum triglyceride levels, while combined effects of sleep deprivation and fat diet consumption caused increased serum triglycerides levels, which may be due to that the short-chain fatty acids found in coconut oil are metabolized more rapidly in the liver and converted into liver total, and thus leads to decreased plasma triglycerides. On the other hand, vitamin C had no significant effect on serum triglycerides levels. Unlike the

results of this study, in many studies; the sleep deprivation has caused a decrease in blood triglycerides levels. Andersen et al. also observed in the study of the effect of stress types on lipid metabolism in rats that sleep deprivation as a type of stress as caused a significant decrease in blood triglycerides levels [35]. Also, Antunes et al. in comparing the effects of sleep deprivation on male rats, healthy and ovariectomized female rats on blood parameters related to cardiovascular diseases showed that the triglyceride levels in male rats, healthy female rats (estrous and pre-estrous stages) and ovariectomized female rats decreased significantly compared to their corresponding control groups [36].

Perry et al. by inducing sub-chronic (4 days) and chronic (21 days) sleep deprivation examined its effect on blood parameters related to the cardiovascular diseases in rats and it was found that the triglycerides levels in sub chronic and chronic sleep deprivation reduced significantly [27]. Andersen et al. in study the effect of sleep deprivation on blood parameters related to cardiovascular diseases in older rats concluded that sleep deprivation causes a decrease in blood triglycerides levels in young rats (3 months old) and aged rats (22 months old) compared to their control groups [37]. In this study, high fat diet consumption caused no significant change in triglyceride levels. Fu et al. by inducing stress on mice coincided with giving them high-fat and high-sucrose diet (containing 16.2% fat and 15% sucrose) for 12 weeks did not observe significant differences in serum triglyceride levels [22].

Inconsistent with this study, it has been shown in many studies that with high fat or hypercholesterolemic diet consumption in laboratory animals, the blood triglyceride levels increase significantly [39-41]. The effects of high fat and high-sucrose diet on triglyceride changes were separately reviewed and was expressed that the diet rich in sucrose causes the specific increased triglyceride by triglyceride production and reduced triglycerides removal. While high-fat diet causes a mild increase in triglyceride levels through reducing its removal [14]. Ryu et al. came to the same conclusion in a similar survey [15]. While Cole et al. in review of the effect high-fat diet on lipoproteins in rats showed that adding 2% cholesterol and 5% fat to the diet for 19-21 days caused significant decreased serum triglycerides levels [44].

In line with this research results, it was observed in reviewing the effect of vitamin C and selenium on the inhibition of cholesterol oxidation that in the group receiving the high fat diet and the group receiving high-fat diet associated with vitamin C (100 mg/kg) and Selenium (200 mg/kg), no changes occur in serum triglycerides levels [42]. Also, Abdollahzadeh et al. did not observe any significant differences in serum triglyceride levels following the prescribing vitamin C supplementation in hemodialysis patients for 12 weeks [43]. While Ness et al. observed that vitamin C has an indirect relationship with triglyceride in women [45]. Also, Afkhami and Shojaeddin concluded that a daily intake of 1000 mg of vitamin C causes reduced levels of serum triglycerides [46].

In the present study, fatty food intake caused increased serum HDL, and inducing sleep deprivation combined with consumption of fatty food caused decreased serum HDL. Vitamin C also caused increased serum HDL in the group with sleep deprivation coupled with consumption of fatty diet, while this vitamin lowered serum HDL in the group with fat diet associated with normal sleep. In this study, sleep deprivation alone caused no significant change on serum HDL levels. Similarly, Perry et al. by inducing sub-chronic (4 days) and chronic (21 days) sleep deprivation and studying its effect on blood parameters related to the cardiovascular diseases in rats reported that the HDL levels did not change significantly in sub-chronic and chronic sleep deprivation [27]. While Andersen et al. studied the effect of stress types for 22 hours a day for 4 consecutive days on lipid metabolism in the rats and found that sleep deprivation as a type of stress significantly increased the HDL levels [35].

Consistent with this research results, it has been shown in many studies that with consumption of high-fat or hypercholesterolemic diet in laboratory animals, the blood HDL levels significantly will increase [39-41]. It was stated in a report that diets containing polyunsaturated single-chain fatty acids cause reduction in ratio of total cholesterol to blood HDL levels [47]. Ryu et al. reviewed the effects of high-fat and high-sucrose diet for 4 weeks separately on HDL changes and concluded that high-sucrose diet and even normal diet have caused significant increased HDL levels compared to the high-fat diet, which may be related to increased binding proteins to HDL in the liver cells [15]. As the simultaneous effects of sleep deprivation and high fat diet caused reduced HDL levels in this study, Fu et al. also by inducing stress on mice coincided with giving them high-fat and high-sucrose diets (containing 16.2% fat and 15% sucrose) for 12 weeks, observed significant reduction in serum HDL levels [22].

Regarding the vitamin C effect on HDL levels, different results were obtained in this study. Many of the studies have been also followed by different results. Abdollahzadeh et al. did not observe any significant differences in serum HDL levels in hemodialysis patients following the prescribing 250 mg of vitamin C supplementation for 12 weeks, but the HDL to LDL ratio significantly increased [43]. Ness et al. observed that taking vitamin C has direct correlation with HDL in women [45]. In the present study, consumption of fatty foods resulted in increased serum LDL, and sleep deprivation alone had no effect on LDL levels; however, inducing sleep deprivation combined with high fat diet compared to normal sleep associated with high-fat diet caused significant increased serum LDL levels. Consumption of vitamin C also caused a decrease in LDL levels in all groups except the group with normal sleep associated with consumption of fatty foods.

According to this study, Perry et al. with inducing sub-chronic (4 days) and chronic (21 days) sleep deprivation and studying its effect on blood parameters related to the cardiovascular diseases in rats reported that the LDL levels will not change significantly in sub-chronic sleep

deprivation [27]. While Andersen et al. studied the effects of stress types on lipid metabolism for 22 hours per day for 4 consecutive days and reported that sleep deprivation as a type of stress causes significant increase in serum LDL levels [35]. Antunes et al. in comparing the effects of sleep deprivation in male rats, and in healthy and ovariectomized female rats on blood parameters related to the cardiovascular diseases showed that in sleep deprivation, the serum LDL levels in male rats and in ovariectomized female rats showed a significant increase compared to the control group [36].

Similar to the present study, it has been shown in numerous studies that with high fat or hypercholesterolemic diet consumption in laboratory animals, the blood LDL levels significantly increase [39-41]. Fu et al. with inducing stress on rats and simultaneous high-fat and high-sucrose diet consumption (containing 16.2% fat and 15% sucrose) for 12 weeks observed significant increased serum LDL levels [22]. Ryu et al. reviewed the effects of high-fat and high-sucrose diet for 4 weeks on LDL changes in rats separately and concluded that high-fat and high-sucrose diets can cause significant increased LDL levels compared to normal diet [15].

In this study, with vitamin C consumption a significant reduction was seen in serum LDL levels of all groups except the group with fat diet associated with normal sleep. In reviewing the effect of Vitamin C on blood lipids, it was observed that daily consumption of 100-200 mg/kg BW of vitamin C for 30 days will cause reduced serum LDL levels [45]. Also, Abdollahzadeh et al. following the administration of 250 mg vitamin C supplementation in hemodialysis patients for 12 weeks observed significant reduced serum LDL levels [43]. Afkhami and Shojaeddin also concluded that a daily intake of vitamin C as 1000 mg in individuals leads to reduced serum LDL levels [35]. Ness et al. found that Vitamin C has no relationship with serum LDL [45].

In the present study, each of the cases of inducing sleep deprivation and fatty food intake did not alone cause changes in serum levels of VLDL, and consumption of fatty foods associated with sleep deprivation caused increased serum VLDL levels. On the other hand, vitamin C had no significant effect on serum VLDL levels. Unlike this study, Anderson et al. studied the effect if types of stress on lipid metabolism for 22 hours per day for 4 consecutive days in rats and reported that sleep deprivation as a type of stress causes a significant decrease in serum VLDL levels [35]. Perry et al. by inducing sub-chronic (4 days) and chronic (21 days) sleep deprivation and studying its effect on blood parameters related to the cardiovascular disease reported that VLDL levels will reduce significantly in sub-chronic and chronic sleep deprivation [27]. Andersen et al. reported that sleep deprivation causes a decrease in blood VLDL levels in rats [37]. While in this study, vitamin C did not cause a significant change on serum VLDL levels, it was observed in studying the effect of vitamin C on blood lipids that daily consumption of 100-200 mg/kg vitamin C for 30 days causes decreased serum VLDL levels [48].

The present study showed that sleep deprivation alone had little effect on serum lipid profile, whereas sleep deprivation with high-fat diet increased the risk factors of blood lipids. Vitamin C has also no specific effect on blood lipid profile except for LDL. However, it is suggested that other involved factors, including oxidative stress indicators and intervening factors in cardiovascular diseases will be reviewed in a similar study.

Acknowledgements

We appreciate the Chamran University Research Deputy, Ahvaz that helped in supporting us in providing the expenditures of this research by the No: 8958757. Also,

we thank the officials of Physiology Research Center of JondiShapur Medical Sciences University that helped us in performing this study.

Authors' Contributions

All authors had equal role in design, work, statistical analysis and manuscript writing.

Conflict of Interest

The authors declare no conflict of interest.

Funding/Support

Shahid Chamran University, Ahvaz.

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Please cite this article as: Najafzade H, Gharibnaseri M, Shahriyari A, Aliabad H. Effect of partial sleep deprivation on lipid profile in high fat diet-fed rats in the presence and absence of vitamin C. *Zahedan J Res Med Sci (ZJRMS)* 2013; 15(7): 12-18.