IUG Journal of Natural Studies
Peer-reviewed Journal of Islamic University-Gaza

ISSN 2409-4587

IUGNS
Vol 27, No 2, 2019, pp 88-95

Received on (06-08-2018) Accepted on (07-06-2019)

The Effect of Cigarette and Hookah Smoking on Lipid Profile Among Adult Males in Rafah Governorate, Palestine

Akram M. Altaher[1], Hosam A. Allhaj-Yousef [1], Sobhi N. Alattar [1], Ali A. Abd-Elnaby [1] and Khalil Z. Almeshal [1]

[1]Department of Medical Sciences University Collage of Science and Technology-Khanyounis, Gaza Strip, Palestine

*** Corresponding author**

e-mail address: a.taher@cst.ps

Abstract

Smoking is an independent risk factor for atherosclerosis & coronary heart disease (CHD). dyslipidemia has been shown to be a significant coronary heart disease risk factor. This study is a case-control, conducted between June to November 2016, and aimed to find out the effect of cigarette smoking and hookah consumption on lipid profiles among the adult population. A total of 200 adults out of them 50 subjects were a non-smoker (controls group) and the other 150 were smokers (cases group), aged between 20-55 years and collected from Rafah Governorate. Personal & demographic data and clinical data situation of the study population were taken by interview questionnaire. Biochemical evaluations were carried out. Collected data were analyzed using SPSS-18. The findings showed that there was a significant difference between the mean ± SD of lipid profile in the controls group and the mean ± SD of lipid profile in seven cases sup-group ($P < 0.05$). Further, there was a statistically significant relationship between the mean ± SD of lipid profiles levels and years of cigarettes smoking among cigarettes smokers group ($p=0.000$). In contrast, among hookah smokers group, a statistical significance relation between the mean ± SD of TC level and years of hookah smoking is reported ($p=0.010$). Cigarette and hookah smokers had appeared with high progressing towards the emergence atherosclerosis and coronary risk factors; because, mostly, they were having high serum TC, TGs, LDL-C, and low serum HDL-C levels as compared to nonsmokers, and these findings agree with most of the previous studies.

Keywords:

Cigarette smoking, Hookah smoking, Lipid profiles, Coronary heart disease; Gaza Strip.

1. Introduction:

Lipids have fundamental and significant functions in biological life, it is acting as steroids hormones and vitamins, providing energy, as structural compounds in bio-membranes in addition to its important role as biological insulator prevents heat loss and allows nerve conduction. Lipid and Lipoprotein Profiles (commonly called Lipid Profiles only are blood test that is used to measure serum total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C) and Triglycerides (TGs) (Altaher, 2016). Cigarette and hookah smoking is associated with increased risk of a variety of medical disorders. Smoking is an important and independent risk factor for atherosclerosis, coronary heart disease (CHD) and peripheral vascular disorders (Macky & crofton,1996). The mechanism by which it increases the

risk of CHD is unknown. Many explanations have been postulated regarding the mechanism by which smoking increases the risk of CHD: The first; the increased carbon monoxide in the blood of smokers may lead to damage the arterial endothelium and accelerate the entry of cholesterol into the wall of the artery. The second, the carboxyhemoglobin formed enhances relative hypoxia in the tissue, including the myocardium. The third, Smoking creates platelets aggregation. Finally, the nicotine absorbed from cigarette or hookah smoke may induce cardiac arrhythmia through its pharmacologic action (Ega & Ega, 2016). Recently it has been suggested that smoking adversely affects the concentration of plasma lipids and lipoprotein levels. However, as devaranavadgi et al., (2012) reported that studies to this point have found out incomplete, inconclusive or conflicting consequences approximately the impact of smoking on the plasma lipids and lipoproteins. It has been estimated that a 1% increase in plasma Lipid concentration is associated with a 2.7% increase of CHD risk (Devaranavadgi, et al., 2012). There may be a dose-response relationship among the number of cigarettes smoked per day and cardiovascular morbidity and mortality (Neki, 2002) . Several studies provide the evidence that tobacco is strongly associated with altering the normal status of the lipid profile (Arslan, et al., 2008). Moreover, several studies reveal significant complications associated with the use of hookah and both respiratory diseases, and lungs cancers (Maziak, 2011), HDL is often lowered and the cardiovascular risk is increased to 1.9 (Shaikh, et al., 2008). The risk of stroke is doubled among hookah consumers (Maziak, 2011)). Another take a look at indicates that a cigarette can launch not more than 10mg of tar, whilst a hookah produces 10 to one hundred times extra tar.The only reality is that hookah releases as more tar in an average of 26 cigarettes (Barnett, et al .,2011). The pack-year is a unit for measuring the amount a person has smoked over a long period of time. it's calculated with the aid of multiplying the number of packs of cigarettes smoked per day by the number of years the person has smoked. For instance, 1 pack-year is equal to smoking 20 cigarettes (1 pack) per day for 1 year, or 40 cigarettes per day for six months, and so on (National Cancer Institute, 2015). Dyslipidemia has been shown to be a significant CHD risk factor. Thus, it seems important to pay attention to lipid abnormalities among cigarette and hookah smokers, in order to reduce the predisposition of cardiovascular disease in this population. Locally, to date, there was no previous studies related to this topic and it's the first time to accomplish this research among the healthy population in Gaza strip. Globally, there were several studies dealt with this subject in many countries like Nigeria, Tunisia, India, and other countries. But, those studies have found out incomplete, inconclusive or conflicting effects regarding the impact of smoking on the plasma lipids and lipoproteins. Therefore, this study aimed to investigate the effect of cigarette smoking and hookah consumption on lipid profiles of the adult population from Palestine.

2. Materials and Methods

2.1 Study Population, Sample Size and Sampling

This study was a case-control, conducted between June to November 2016. The total samples of this study were consisting of 200 adults out of them 50 subjects were non-smokers (controls group) and the other 150 were smokers (cases group) since at least 5 years duration, aged between 20-55 years and collected from Rafah Governorate. The cases group was separated into seven sub-group according to smoking status type (light smoking, intermediate smoking, heavy smoking, hookah & light smoking, hookah & intermediate smoking, hookah& heavy smoking and only hookah smoking). Additionally, the smokers in the study were divided into mild, moderate, and heavy smokers using two different approaches. The first approach was based on pack-years: smokers with<5 pack-years were considered as mild, 5-15 pack-years as moderate, and >15 pack-years as severe smokers. Smoking pack years is a figure that combines smoking duration and smoking intensity (severity), and one smoking pack year is defined as 20 cigarettes smoked daily for 1 year (Masters & Tutt, 2015). The second approach was based only on the numbers of cigarettes smoked per day regardless of the duration component from pack-years: smokers with less than 10 cigarettes per day were considered as mild, 10-19cigarettes per day as moderate, and 20 or more cigarettes per day as severe smokers (Desai et al., 2012). The researchers of this study have taken guidance from a world health organization (WHO) study on waterpipe smoking. From this study a 45-minute session equal around 60 cigarettes (Regulation, 2005). The present study was used a 7.5-minute session (10 cigarette equivalence) as a baseline in the calculator. On the other hand, two kinds of cigarettes were studied; Royal and Manchester. All volunteers with history of diabetes or renal disease or liver disease or any type of cardiovascular disease such CHD & Hypertension were excluded from the study.

2.2 Questionnaire interview

A meeting interview was used for filling in the questionnaire for all volunteers. It consisted of many issues: personal data (name, age, sex, education status and so on…), smoking data (the type of smoking, kind of cigarettes and so on..)

2.3 Blood sampling and biochemical analysis

Under aseptic technique, about 4 ml of fasting (14-16 hours) venous blood sample was collected from each subject in a plain tube (without anticoagulation) and samples were allowed to clot and the serum was centrifuged at room temperature to 4000 round/minute for 10 minutes. Serum was stored at -18°C until analyzed. Serum was used to determine TC, TGs, and HDL-C levels. TC, TG and HDL-C were measured by spectrophotomer using suitable clinical Kit in the clinical chemistry laboratory of university college of science and technology in Khanyounies, whereas LDL-C was calculated using Friedewald formula: [LDL-C = TC – (HDL-C) – TG/5(mg/dl)] (Fried Wald's et al., 1972).

2.4 Data analysis

Data obtained were analyzed using the Statistical Package of Social Sciences (SPSS) system (V. 18.0). Descriptive statistics, Chi-Square test, t-test & ANOVA test were applied. A significant result means that the P-value for the hypothesis tests was less than 0.05. The confidence intervals (CI) was reported as 95%.

3. Results

3.1 General characteristics of the study subjects

Table 1 General characteristics of the study subjects

Variables	Frequency (%)
Categories of study populations	
Controls	50 (25.0%)
Light smoking	20 (10.0%)
Intermediate smoking	20 (10.0%)
Heavy smoking	20 (10.0%)
Hookah & light smoking	15 (7.5%)
Hookah & intermediate smoking	15 (7.5%)
Hookah& heavy smoking	15 (7.5%)
Only hookah smoking	45 (22.5%)
Total	**200 (100.0%)**
Coffee drinking status	
No	9 (9.5%)
yes	181 (90.5%)
Total	**200 (100.0%)**
Kinds of cigarettes smoked by smokers group	
Royal	98 (97.3%)
Manchester	7 (6.7%)
Total	**105 (100.0%)**
Years of cigarettes smoking	
5-12Years	23 (22.0%)
13-17years	37 (35.0%)
14-40years	45 (63.0%)
Total	**105 (100.0%)**
Years of hookah smoking	
5-11Years	29 (49.0%)
12-16years	27 (28.0%)
16-35years	34 (33.0%)
Total	**90 (100.0%)**

The finding showed that the mean ± standard deviation (SD) of age among the smokers group was 39.56±8.50 years whereas, the mean ± SD of age was 36.38±10.17 among controls group. However, the T-Test statistical analysis showed that there is no statistically significant difference between the study subjects

with respect to the mean ± SD of age in years (P=0.089). The study population was categorized in to two groups: cases (smokers) group and the control group. The cases (smokers) group was categorized into seven sub-groups; light smoking, intermediate smoking, and so on. (table 1). Regarding the coffee drinking, in this research It is found that 181 (90.5%) of subjects were used to drink coffee daily whereas, 19 (9.5%) of them were used to drink coffee daily (table 1). Furthermore, about 97.0% of cigarettes smokers were smoking Royal whereas 6.7% of them were smoking Manchester. In addition, most of the cigarette smokers (63.0%) were smoking since at least 14 years (table 1). On the other hand, most of the hookah smokers (49.0%) were smoking since 5-11 years duration.

3.2 Comparison of lipid profiles among the study groups

Table 2 compares the mean ± SD of TC, TGs, HDL-C & LDL-C among groups of the study. However, after using ANOVA Test; there were a highly statistically differences between the mean ± SD of lipid profile levels among the study groups (P=0.000).

Table 3 illustrates the multiple comparisons of lipid profiles between the controls group and case sub-groups. However, after applying of Post-Hoc Test; there was also significant difference between the mean ± SD of lipid profile in control group and the mean ± SD of lipid profile in the seven cases sub-group ($P < 0.05$)

3.3 Relationship of lipid profiles levels with independent variables that suspected to be related

Table 4 demonstrates the relationship of lipid profiles with kind of cigarettes smoked. However, there is no statistically difference between the mean ± SD of lipid profiles among cigarette smokers with respect to the kind of cigarettes ($P \geq 0.05$).

Table 2 The comparative distribution of lipid profiles among the study groups

Description	N	Mean	Std. Deviation	ANOVA Test F	ANOVA Test P-Value
Total cholesterol					
Control	50	153.8800	12.36790		
Light smoking	20	180.3000	13.75768		
Intermediate smoking	20	197.4500	17.22750		
Heavy smoking	20	208.1500	14.88650		
Hookah & Light smoking	15	202.0000	10.70380	61.850	**0.000****
Hookah & Intermediate smoking	15	212.5333	13.88662		
Hookah& Heavy smoking	15	224.5333	19.32750		
Only hookah smoking	45	200.9333	19.85905		
Triglycerides					
Control	50	110.2000	15.34501		
Light smoking	20	155.4500	20.25749		
Intermediate smoking	20	168.4500	10.94231		
Heavy smoking	20	185.8500	20.18409	95.729	**0.000****
Hookah & Light smoking	15	197.3333	16.60321		
Hookah & Intermediate smoking	15	192.5333	18.46180		
Hookah& Heavy smoking	15	207.4667	18.65808		
Only hookah smoking	45	181.8444	22.55091		
HDL-C					
Control	50	49.7600	4.02320		
Light smoking	20	45.5000	5.63355		
Intermediate smoking	20	40.6500	3.58762		
Heavy smoking	20	35.6500	3.11659	57.661	**0.000****
Hookah & Light smoking	15	36.6667	2.55417		

	Hookah & Intermediate smoking	15	35.8667	2.19957		
	Hookah& Heavy smoking	15	34.2000	2.42605		
	Only hookah smoking	45	39.9556	4.43107		
LDL-C						
	Control	50	81.8140	12.33515		
	Light smoking	20	103.8600	14.70240		
	Intermediate smoking	20	123.1100	17.86422		
	Heavy smoking	20	135.8300	14.26225		
	Hookah & Light smoking	15	125.8733	9.44718	48.977	**0.000****
	Hookah & Intermediate smoking	15	142.4267	30.59504		
	Hookah& Heavy smoking	15	148.8400	17.93165		
	Only hookah smoking	45	124.6267	19.43495		
	Total No.	**200**				

P < 0.05: Significant, * Statistically significant, ** Highly statistically significant

Table 5 reveals the relationship of lipid profiles with years of cigarettes and hookah smoking. thus, there is a statistically significant relationship between the mean ± SD of lipid profiles levels and years of cigarettes smoking among cigarettes smokers group (p=0.000). In addition, there was no statistical relation between the mean ± SD of TGs, HDL-C & LDL-C levels and years of hookah smoking among hookah smokers group (P ≥ 0.05). In contrast, among the same group, a statistical significant relation between the mean ± SD of TC level and years of hookah smoking is reported (p=0.010) (table 5). Table 6 describes the relation of lipid profiles with coffee drinking status. However, there is a highly statistically difference between the mean ± SD of lipid profiles among the study population according to the coffee drinking status (P=0.000).

Table 3 Comparison of lipid profiles between controls group and case sub- groups

Dependent Variable	**(I) Stud group (control)**	**(J) Study group (cases)**	**Mean Difference (I-J)**	**Sig.**
Total cholesterol				
		Light smoking	-26.42000-*	**0.000****
		Intermediate smoking	-43.57000-*	**0.000****
		Heavy smoking	-54.27000-*	**0.000****
	Control	Hookah & Light smoking	-48.12000-*	**0.000****
		Hookah & Intermediate smoking	-58.65333-*	**0.000****
		Hookah& Heavy smoking	-70.65333-*	**0.000****
		Only hookah smoking	-47.05333-*	**0.000****
Triglycerides				
		Light smoking	-45.25000-*	**0.000****
		Intermediate smoking	-58.25000-*	**0.000****
		Heavy smoking	-75.65000-*	**0.000****
	Control	Hookah & Light smoking	-87.13333-*	**0.000****
		Hookah & Intermediate smoking	-82.33333-*	**0.000****
		Hookah& Heavy smoking	-97.26667-*	**0.000****
		Only hookah smoking	-71.64444-*	**0.000****
HDL-C				
		Light smoking	4.26000*	**0.021**
		Intermediate smoking	9.11000*	**0.000****
		Heavy smoking	14.11000*	**0.000****
	Control	Hookah & Light smoking	13.09333*	**0.000****
		Hookah & Intermediate smoking	13.89333*	**0.000****
		Hookah& Heavy smoking	15.56000*	**0.000****
		Only hookah smoking	9.80444*	**0.000****
LDL-C				
		Light smoking	-22.04600-*	**0.002**

Control	Intermediate smoking	-41.29600-*	**0.000****
	Heavy smoking	-54.01600-*	**0.000****
	Hookah & Light smoking	-44.05933-*	**0.000****
	Hookah & Intermediate smoking	-60.61267-*	**0.000****
	Hookah& Heavy smoking	-67.02600-*	**0.000****
	Only hookah smoking	-42.81267-*	**0.000****

Table 4 Relationship of lipid profiles with kind of cigarettes smoked

Dependent Variable		Kind of cigarettes	No.	Mean	Std. Dev.	t	P-Value
Total cholesterol	x	Royal	53	194.3019	19.81897	1.121	**0.267**
		Manchester	7	202.8571	8.78310		
Triglycerides	x	Royal	53	168.7736	22.40076	1.139	**0.259**
		Manchester	7	178.5714	8.48247		
HDL-C	x	Royal	53	40.8113	6.05124	0.770	**0.444**
		Manchester	7	39.0000	3.60555		
LDL-C	x	Royal	53	119.9811	21.14442	0.998	**0.322**
		Manchester	7	128.1429	11.08976		

Table 5 Relationship of lipid profiles with years of cigarettes and hookah smoking

Correlations		Cholesterol	Triglycerides	HDL-C	LDL-C
Years of cigarettes smoking					
	Pearson Correlation	0.450**	0.416**	0.368**	0.358**
	Sig. (2-tailed)	**0.000****	**0.000****	**0.000****	**0.000****
	No.	60	60	60	60
Years of hookah smoking					
	Pearson Correlation	0.269*	0.172	0.112	0.161
	Sig. (2-tailed)	**0.010***	**0.106**	**0.292**	**0.128**
	No.	45	45	45	45

Correlation is significant at the 0.05 level (2-tailed).

Table 6 Relationship of lipid profiles with coffee drinking status

Dependent Variable	Coffee drinking		No.	Mean	Std. Dev.	t	P-Value
Total cholesterol	x	No	19	151.1579	11.10187	7.158	**0.000****
		Yes	181	194.2983	25.97605		
Triglycerides	x	No	19	105.1053	18.07052	8.138	**0.000****
		Yes	181	170.4475	34.44825		
HDL-C	x	No	19	51.6842	3.71263	7.780	**0.000****
		Yes	181	40.5580	6.10766		
LDL-C	x	No	19	78.3579	10.04713	6.772	**0.000****
		Yes	181	120.0177	26.56513		

P < 0.05: Significant, * Statistically significant, ** Highly statistically significant

4. Discussion

The present study has shown that cigarette smoking and hookah consumption increases serum TC, TGs, LDL-C, and decreases serum HDL-C levels in smokers as compared to nonsmokers. The present findings agree with many earlier reports (Kong, et al., 2001; Zhu, et al., 2011& Devaranavadgi, 2012). In details; the mean value of serum TC in cigarette smokers and hookah consumers is significantly higher (P<0.05) as compared to nonsmokers. Similar findings were found in the study done by Trupti, et al. (2014) where they showed that there is an increased level of serum TC in smokers. The present study also showed a significant increase (P<0.05) in serum TGs in cigarette smokers and hookah consumers as compared to non-smokers (Table 3). A similar finding was observed by Trupti, et al. (2014) where they reported that mean serum TGs was significantly higher in smokers than in non-smokers (P<0.001). The current study also reported that the

mean value of serum HDL-C was significantly low (P < 0.001) and the mean value of serum LDL-C was significantly high (P < 0.001) in all groups of cigarette smokers and hookah consumers. The present study findings are similar to the finding of Trupti, et al. (2014) who found that mean HDL-C was significantly lower and LDL-C was significantly higher among smokers as compared to control. On the other hand, analyzing the results of the current study with regard to the duration of smoking revealed that there was a significant increase (P<0.05) in the level of serum cholesterol with regard to an increase in duration of cigarette smoking and hookah consumption. Furthermore, the present study observed that there was a significant increase (P<0.05) in the level of serum TGs with regard to an increase in duration of cigarette smoking, in contrast, the relation with duration of hookah consumption was not statistically significant. Moreover, the findings of present study observed that there is a significant decrease (P<0.05) in the level of serum HDL-C and increase in the level of serum LDL-C with regard to an increase in duration of cigarette smoking, in contrast the relation with duration of hookah consumption was not statistically significant. The P values obtained with regards to all fractions of the serum lipid profile are paralleled increase in mild to heavy smokers (Table 3). In contrast to our findings, one study reported that there is significant decrease in HDL-C, but there was no change in total cholesterol and triglycerides in cigarette smokers as compared to non-smokers. The above findings, except for a decrease in HDL-C are contradictory to our findings (Devaranavadgi, 2012). In a separate context, all studies that included biochemical measures, specified that hookah smoking involves inhaling several liters of smoke and contains many other toxic substances (Koubaa, et al., 2013). The rise in blood lipid levels in smokers may be through catecholamine and Adenyl cyclase axis induced tissue lipolysis as suggested in the chart (Devaranavadgi, 2012).

Smoking
↓
Absorption of nicotine into the body
↓
Secretion of catecholamines, cortisol and growth hormones
↓
Activation of adenyl cyclase in adipose tissue
↓
Lipolysis of stored TG and release of FFA into plasma
↓
Release of FFA from adipose tissue TG into plasma bound to albumin
↓
Increased Hepatic synthesis of TG, VLDL-C
↓
Increased Plasma TG, VLDL-C
↓
↓ HDL-Cholesterol

About of the effect of coffee drinking on lipid profiles levels, the present findings reported that coffee drinking increases serum TC, TGs, LDL-C, and decreases serum HDL-C levels among the study population (table 6). The present results agree with many earlier reports(Kong, et al., 2001; Zhu, et al., 2011 & Devaranavadgi, 2012). Observational studies have identified a positive association between coffee drinking and higher levels of serum cholesterol (Jee, et al., 2001).

5. Conclusions:

Cigarette and hookah smokers have appeared with high progressing towards atherosclerosis and coronary risk factors; because, mostly, they were having higher value of serum TC, TGs, LDL-C, and low serum HDL-C levels as compared to nonsmokers, and this finding agrees with most of the previous studies. In addition, there is a significant increase in the level of serum TC, TG, LDL-C and decrease HDL-C with regard to an increase in duration and intensity of cigarette smoking. Moreover, there is a significant increase in the level of serum TC with regard to an increase in the duration of hookah consumption.

References:

Altaher, A. M. R., Alewaity, S. S., & Abu-Touima, J. A. (2016). Lipid Profiles Levels of Type One Diabetics Compared to Controls in Gaza Strip. American Journal of Biomedical and Life Sciences, 4(4), 61-68. Retrieved from: https://www.researchgate.net/publication/305531499_Lipid_Profiles_Levels_of_Type_One_Diabetics_Compared_to_Controls_in_Gaza_Strip

Arslan, E., Yakar, T., & Yavaşoğlu, İ. (2008). The effect of smoking on mean platelet volume and lipid profile in young male subjects. *The Anatolian Journal of Cardiology*, *8*(6), 422-425. Retrieved from: http://anakarder.com/jvi.aspx?pdir=anatoljcardiol&plng=eng&un=AJC-24582&look4=

Barnett, T. E., Curbow, B. A., Soule Jr, E. K., Tomar, S. L., & Thombs, D. L. (2011). Carbon monoxide levels among patrons of hookah cafes. American journal of preventive medicine, 40(3), 324-328. Retrieved from: https://www.sciencedirect.com/science/article/pii/S0749379710007014

Devaranavadgi, B. B., Aski, B. S., Kashinath, R. T., & Hundekari, I. A. (2012). Effect of cigarette smoking on blood lipids–A study in Belgaum, Northern Karnataka, India. Global Journal of Medical Research, 12(6), 57-60. Retrieved from: https://medicalresearchjournal.org/index.php/GJMR/article/view/283

Desai, K. T., Gharat, V. V., Nayak, S. N., Patel, P. B., & Bansal, R. (2012). Tobacco smoking patterns, awareness and expenditure: a cross-sectional overview from Surat City, India. *Tobacco Control and Public Health in Eastern Europe*, *2*(1), 25-32. Retrieved from: http://journals.uran.ua/tcphee/article/view/4890

Ega, J. K., & Ega, L. K. (2016). Journal of Chemical and Pharmaceutical Research, 2016, 8 (2): 513-525. *Journal of Chemical and Pharmaceutical Research*, *8*(2), 513-525. Retrieved from; https://www.researchgate.net/publication/296419616_Comparative_study_of_lipid_profile_in_young_smokers_and_non_smokers

Friedewald, W. T., Levy, R. I., & Fredrickson, D. S. (1972). Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. Clinical chemistry, 18(6), 499-502. Retrieved from: http://clinchem.aaccjnls.org/content/18/6/499.short

Koubaa, A., Trabelsi, H., Masmoudi, L., Triki, M., Sahnoun, Z., Zeghal, K., & Hakim, A. (2013). Water pipe tobacco smoking and cigarette smoking: comparative analysis of the smoking effects on antioxidant status, lipid profile and cardiopulmonary quality in sedentary smokers Tunisian. *Int J Invent Pharmaceut Sci*, *2*, 51-7. Retrieved from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5907743/

Kong, C., Nimmo, L., Elatrozy, T., Anyaoku, V., Hughes, C., Robinson, S., ... & Elkeles, R. S. (2001). Smoking is associated with increased hepatic lipase activity, insulin resistance, dyslipidaemia and early atherosclerosis in Type 2 diabetes. *Atherosclerosis*, *156*(2), 373-378. Retrieved from: https://www.sciencedirect.com/science/article/pii/S002191500000664X

Mackay, J., & Crofton, J. (1996). Tobacco and the developing world. *British Medical Bulletin*, *52*(1), 206-221. Retrieved from: https://academic.oup.com/bmb/article/52/1/206/284538

Masters, N & Tuttt, C. (2015). Smoking. Pack years. Retrieved from: http://www.smokingpackyears.com

Maziak, W. (2011). The global epidemic of waterpipe smoking. *Addictive behaviors*, *36*(1-2), 1-5. Retrieved from: https://www.sciencedirect.com/science/article/abs/pii/S0306460310002662

Neki, N. S. (2002). Lipid profile in chronic smokers–A clinical study. *Journal, Indian Academy of Clinical Medicine*, *3*(1), 51-4. Retrieved from: medind.nic.in/jac/t02/i1/jact02i1p51.pdf

National.Cancer.Institute.(2015)."definition.of.pack.year".http://www.cancer.gov/dictionary?CdrID=306510

Shaikh, R. B., Vijayaraghavan, N., Sulaiman, A. S., Kazi, S., & Shafi, M. S. (2008). The acute effects of waterpipe smoking on the cardiovascular and respiratory systems. *Journal of preventive medicine and hygiene*, *49*(3). Retrieved from: http://www.jpmh.org/index.php/jpmh/article/view/124

Trupti RR, Ramakrishna M R, Desai R D, Taklikar R& Sreekantha. (2014). Comparative study of effect of lipid profile in smokers and non smokers of age group of 40-50 years" . ISSN (o):2321–7251. Retrieved from: http://cutt.us/5ZrCn

Regulation, T. P. (2005). Waterpipe Tobacco Smoking: Health Effects, Research Needs and Recommended Actions by Regulators.

ZHU, Y., ZHANG, M., HOU, X., LU, J., PENG, L., GU, H., ... & JIA, W. (2011). Cigarette smoking increases risk for incident metabolic syndrome in Chinese men—Shanghai diabetes study. *Biomedical and Environmental Sciences*, *24*(5), 475. Retrieved from: www.besjournal.com/Articles/pastIssues/2011/No5/201111/P020111103377239434808.pdf

Jee, S. H., He, J., Appel, L. J., Whelton, P. K., Suh, I. I., & Klag, M. J. (2001). Coffee consumption and serum lipids: a meta-analysis of randomized controlled clinical trials. *American journal of epidemiology*, *153*(4), 353-362. Retrieved from: https://academic.oup.com/aje/article/153/4/353/129046