International Research Journal of BIOLOGICAL SCIENCES

Volume 01 | Issue 01 | 2019

SciRange PUBLICATIONS
www.scirange.com

SciRange

Open Access

International Research Journal of Biological Sciences

Research Article

pISSN: 2663-5968, eISSN: 2663-5976

Tabagism and Risk of Atherogenesis; Comparison of Atherogenic Indices Between Smoking and Non Smoking Populations

[1]Said Nadji, [2]Thamer Zaiter, [3]Aiman Sahra, [3]Cherif Khabbar, [1]Soumia Boudjemaa and [2]Hanane Boukrous

[1]Laboratory of Toxicology, University Hospital of Batna, Route de Tazoult, Batna, Algeria
[2]Laboratory of Biochemistry, University Hospital of Batna, Route de Tazoult, Batna, Algeria
[3]Department of Pharmacy, Faculty of Medicine, University of Batna, Batna, Algeria

ARTICLE INFORMATION

Received: November 29, 2018

Accepted: December 27, 2018

Published: January 31, 2019

Corresponding Author:
Said Nadji,
Laboratory of Toxicology,
University Hospital of Batna,
Route de Tazoult, Batna, Algeria

ABSTRACT

The clinical manifestations of atherosclerosis are one of the most important public health conditions in terms of frequency and smoking is one of its most important risk factors. The aim of this study was to prove the relationship between smoking and atherogenic risk by researching changes in atherogenic indices (CT/HDL-c, LDL-c/HDL-c and CRP) in a population of smokers (n=43) compared to non-smokers (n=17) from the city of Batna. The results show significant increases ($p<0.05$) in atherogenic indices in smokers compared to non-smokers based on different criteria (age, BMI (Body Mass Index), family history and physical activity). The results also show that the duration and frequency of smoking significantly increase the two atherogenic indices Cholesterol/HDL-c and LDL-c/HDL-c. It can be concluded that smoking increases the risk of developing atherosclerosis.

Key words: Tabagism, atherogenesis, atherogenic index, correlation, smokers

INTRODUCTION

Tabagism is one of the most serious threats to public health, indeed, the latest WHO references estimate that more than 1 billion people worldwide are smokers. Beside, smoking was reported to be behind the death of 7 million people each year. About one person died every 6 seconds because of it, which represents one out of every 10 adult deaths[1].

According to an Algerian health ministry report, tobacco smoking addiction, in Algeria represents 15% of the population aged between 15 and 75 years and 9.2% in the age range between 15 and 19 years, which places Algeria among the medium-consumption countries[2].

Tabagism is a factor of risk for many diseases, including different types of cancer (lung, throat, bladder). Sufficient evidence of carcinogenicity in humans has also been found between smoking and cancer of the esophagus, stomach, colon, rectum, liver, pancreas, nasal cavity and cervix, ovary, kidney, ureter and bone marrow[3].

Cardiovascular toxicity of tobacco smoking is possible even for very low levels of consumption and it has a nonlinear dose-response relationship and no threshold of consumption below which smoking is not a factor of risk[4].

Several mechanisms of cardiovascular toxicity are involved:

- Thrombosis, by increasing the aggregation of platelets and arterial spasm, by alteration of endothelium-dependent arterial vasomotricity[5]
- The mechanisms in which smoking contributes to the formation of atherosclerotic plaque (endothelial dysfunction, inflammation, lipid profile modification by increasing total cholesterol, LDL-c and triglycerides with decreasing HDL-c and fats peroxidation)[6,7]
- The hemodynamic effects of smoking are associated with increases in cardiac frequency and blood pressure, by adrenergic nicotine stimulation[6,7]
- Carbon monoxide decreases oxygen carrying capacity due to its high hemoglobin affinity and contributes to the increased ischemia and cardiovascular complications in people with impaired coronary artery function[3,6]

Tabagism is among the risk factors of atherosclerosis, which is characterized by "a variable association of intimal changes in the large and medium-sized arteries, consisting of a focal accumulation of lipids, complex carbohydrates, blood and blood products and fibrous tissues and of calcium deposits, all accompanied by changes in the media"[8].
Several indices are used to estimate the risk for the development of atherosclerotic cardiovascular disease[9,10]:

- Castelli risk indices (CRI-I = CT/HDL-c) and (CRI-II = LDL-c/HDL-c)
- Atherogenicity coefficient AC = (CT-HDL-c)/HDL-c
- Plasma atherogenicity index (AIP = log TG/HDL-c)
- The ratio Apo B/Apo A1. There are other innovative markers as: C-reactive protein (CRP and CRP ultra-sensitive), Homocysteine, Lipoprotein (a), Adhesion molecules (ICAM-1 and VCAM-1), Interleukin 6 (IL-6), Metalloproteases, Endothelins, Adiponectin, Phospholipase A2

The objective of the present study was to prove the relationship between smoking and atherogenic risk by comparing atherogenic indices (CT / HDL-c, LDL-c / HDL-c and CRP) in a population of smokers and between the populations of smokers and non-smokers from the city of Batna. Several biomarkers are assayed: total cholesterol, triglycerides, LDL-c and HDL-c and then calculate the CT/HDL-c and LDL-c/HDL-c ratios, inflammation marker: CRP (C-Reactive Protein) and marker of smoking carboxy hemoglobin (HbCO) for smokers. Different criteria of comparison are kept as the duration of smoking (more or less than 10 years), the frequency of smoking (lower and higher than 15 cigarettes day^{-1}), the age of people (below and above 30 years old), BMI: inferior and superior than 23 kg m^{-2}, family history of dyslipidemia (with or without) and physical activity (active/sedentary).

MATERIALS AND METHODS

Materials: Sodium heparin tubes, Micropipettes: 100, 200, 500 and 1000 µL, filter paper, funnels and beakers, COBAS INTEGRA 400 plus controller, COBAS 6000 controller c 501 unit, Centrifuge Hettich zentrifugen Universal 320R, Balance: maximum Kern BLT100 100 g, d = 0.01 mg, Water bath, pH meter, UV-Vis spectrophotometer Agilent Technologies Cary 60, Chronometer- Freezer (-20°C), The COBAS INTEGRA assay cassettes.

Methods: The study was conducted on a population of 60 voluntary male people from the city of Batna. This sample was divided into 2 groups, Smoker's group (n = 43) and non-smokers (control) group (n = 17). The exclusion criteria for the study were:

- People with diseases (dyslipidemia, diabetes, hypertension and all cardiovascular diseases, nephropathies, liver diseases, lithiasis, inflammatory syndrome)
- Obesity, Occasional smokers (tobacco consumption in a disturbed manner and not daily)
- People with age lower than 20 years old
- Intake of drugs that may influence lipid balance (statins, beta-blockers, steroids)
- Alcohol consumption

The description of the studied population is grouped in the (Table 1).

Table 1: Statistical description of the smokers and non-smokers populations

Factors	Non-smokers		Smokers	
Size (n)	17		43	
Weight (kg)	70±9.81		70.55±12.87	
Height (m)	1.77±0.05		1.74±0.06	
Age (years)	32±11.73		29.79±9.69	
	<30 years	>30 years	<30 years	>30 years
	13	7	30	10
BMI (kg m^{-2})	22.35±2.73		22.93±3.20	
	<23 kg m^{-2}	>23 kg m^{-2}	<23 kg m^{-2}	>23 kg m^{-2}
	9	8	26	17
Family history of dyslipidemia	41.17%		39.53%	
	Without	With	Without	With
	10	7	26	17
Physical activity (%)	17.64		46.51	
	No	Yes	No	Yes
	14	3	23	20
Duration of smoking	/		12.1±9.59 years	
			<10 years	>10 years
			25	18
Frequency of smoking (cigarette day^{-1})	/		19.79±10.57	
			<15 cig day^{-1}	>15 cig day^{-1}
			15	28

Mean±Standard Deviation

Statistical analysis: The statistical Student (t) test with a confidence level of 95% (statistical significance for $p<0.05$)) was performed to compare between the groups. The results are expressed as mean± relative standard deviation.

RESULTS AND DISCUSSION

Study of the biological parameters of the non-smoking population: The biological parameters of the non-smoking population are given in Table 2, from the results obtained and it can be noted that: The concentrations of CT, TG, LDL-c, CRP and the LDL-c/HDL-c ratio are in the normal range throughout the non-smoking population, while that 88.24% of non-smokers have a moderate risk for HDL-c (0.35 g L^{-1} <HDL-c <0.55 g L^{-1}) and 17.65% are reported to have CT/HDL-c higher than the normal range (CT/HDL-c> 3.5).

Study of the biological parameters of the smoking population: The results of the study of the biological parameters of the smoker population are given in Table 3 and it can be seen that: 2.32% of smokers are at moderate risk for the parameters: CT and TG (CT: 2-2.39 g L^{-1}, TG: 1.5-1.99 g L^{-1}). All smokers have an HDL-c concentration lower than the normal range (HDL-c <0.55 g L^{-1}), 60.46% have a moderate risk (0.35-0.55 g L^{-1}) and 39.54% are at high risk (HDL-c <0.35 g L^{-1}). The 34.88% of smokers have a concentration of LDL-c higher than the normal range (< 1 g L^{-1}), 23.25% have a slightly elevated concentration (1-1.29 g L^{-1}) and 11.62% have a moderate risk (1.3-1.59 g L^{-1}). All smokers have CRP levels within the normal range (CRP<5 mg L^{-1}) and 58.14% have CT/HDL-c > 3.5, while that 27.91% have LDL-c/HDL-c> 3 (above the normal range). HbCO% ranges from 2.21-12.26% (mean: 5.61%), resulting that the whole smoking population have an HbCO above the normal range (HbCO> 2%).

Study of biological parameters according to the impact of smoking: Significant difference (Student test) was noted between the smoking and non-smoking populations for HDL-c, LDL-c, CRP, HbCO, CT/HDL-c and LDL-c/HDL-c ratios while that the values of CT and TG levels showed no statistical significant difference between the two groups, values of LDL-c, CRP, CT/HDL-c, LDL-c/HDL-c, HbCO are significantly higher, on the other hand, HDL-c was significantly lower in the smoking population (Table 4).

Table 2: Biological parameters in the non-smoking population

	CT (g L^{-1})	TG (g L^{-1})	HDL-c (g L^{-1})	LDL-c (g L^{-1})	CRP (mg L^{-1})	CT/HDL-c	LDL-c/HDL-c
Mean	1.36	0.75	0.46	0.63	0.50	3.02	1.41
Std dev	0.22	0.22	0.07	0.19	0.25	0.62	0.48
Median	1.37	0.72	0.46	0.67	0.45	3.10	1.39
Minimum	0.88	0.34	0.36	0.28	0.18	1.87	0.59
Maximum	1.75	1.21	0.65	0.89	1.10	4.33	2.47

CT: Total Cholesterol, TG: Triglycerides, HDL-c: High-Density Lipoprotein Cholesterol, LDL-c: Low-Density Lipoprotein Cholesterol, CRP: C-Reactive Protein

Table 3: Biological parameters in the smoking population

	CT (g L^{-1})	TG (g L^{-1})	HDL-c (g L^{-1})	LDL-c (g L^{-1})	CRP (mg L^{-1})	CT/HDL-c	LDL-c/HDL-c
Mean	1.43	0.83	0.38	0.91	1.19	3.92	2.50
Std dev	0.30	0.30	0.06	0.24	1.02	1.33	0.99
Median	1.34	0.76	0.38	0.83	0.77	3.72	2.26
Minimum	1.02	0.43	0.30	0.60	0.14	2.04	1.15
Maximum	2.08	1.54	0.53	1.48	3.60	6.93	4.93

CT: Total Cholesterol, TG: Triglycerides, HDL-c: High-Density Lipoprotein Cholesterol, LDL-c: Low-Density Lipoprotein Cholesterol, CRP: C-Reactive Protein

Table 4: Comparison of biological parameters between smoking and non-smoking populations

	Mean (n=17) non-smokers	Mean (n=43) smokers	p (t-test)	Percentage of pathological values: non-smokers	Percentage of pathological values: smokers
CT (g L^{-1})	1.36±0.22	1.43±0.30	0.16	0%	Moderate risk: 2.32%
TG (g L^{-1})	0.75±0.22	0.83±0.30	0.12	0%	Moderate risk: 2.32%
HDL-c (g L^{-1})	0.46±0.07	0.38±0.06	0.0004	Moderate risk: 88.24%	Moderate risk: 60.46% High risk: 39.54%
LDL-c (g L^{-1})	0.63±0.19	0.91±0.24	0.00001	0%	Moderate risk: 23.25% High risk: 11.62%
CRP (mg L^{-1})	0.50±0.25	1.19±1.02	0.00006	0%	0%
CT/HDL-c	3.02±0.62	3.92±1.33	0.0003	17.65%	58.14%
LDL-c/HDL-c	1.41±0.48	2.50±0.99	1.88×10^{-7}	0%	27.91%
HbCO (%)	2±0	5.61±2.38	6.22×10^{-13}	0%	100%

CT: Total Cholesterol, TG: Triglycerides, HDL-c: High-Density Lipoprotein Cholesterol, LDL-c: Low-Density Lipoprotein Cholesterol, CRP: C-Reactive Protein, HbCO: Carboxyhemoglobin

Concordance was found with similar studies for LDL-c and HDL-c levels, but found a significant difference for CT and TG between smokers and non-smokers[11,12]. Other studies found similar results for CT and TG[13,14]. These results show a significant increase in CT/HDL-c and LDL-c/HDL-c ratios in smokers compared to non-smokers, these results are in agreement with other studies. The obtained results show a significant increase in CRP between smokers and non-smokers. It could be concluded that tabacco smoking has a negative impact on the lipid profile the CRP and the atherogenic indices, which indeed increases the atherogenic risk.

Study of biological parameters within the population of smokers:

Influence of the duration of tabagism: A significant difference was showed for CT, TG, HDL-c, LDL-c, CT/HDL-c and LDL-c/HDL-c ratios, while that, no statistical significant difference noted for the parameters CRP and HbCO. The CT, TG, LDL-c, CT/HDL-c and LDL-c/HDL-c levels are significantly higher within the population that has more than 10 years of tobacco smoking, which has a negative impact on the lipid profile and rises consequently the atherogenic risk, on the other hand, the HDL-c level was significantly higher within the population of smokers with less of 10 years of tobacco (Table 5). Beside, strong positive linear correlation was found between the duration of smoking and CT/HDL-c ratio ($r = 0.867$) and the LDL-c/HDL-c ratio ($r = 0.894$). The obtained results corroborate similar studies[15,16].

Influence of the frequency of smoking: Statistical significant difference for the parameters CT, TG, HDL-c, LDL-c, CT/HDL-c and LDL-c/HDL-c and HbCO and was not significant for CRP between the groups of smokers that consume more than 15 cigarettes per day and those who take than 15 per day. The levels of CT, TG, LDL-c, CT/HDL-c, LDL-c/HDL-c and HbCO are significantly higher for the first group whereas, the HDL-c levels are significantly lower (Table 5).

Study of the influence of the age: Within the population of smokers which are older than 30 years old and those younger than 30 years old, significant difference was observed for the parameters CT, TG, HDL-c, LDL-c, CT/HDL-c, LDL-c/HDL-c and HBCO and not significant for CRP.

A comparison between smokers and non smokers populations at the same age ranges are carried out, with significant difference was noted for HDL-c, LDL-c, CT/HDL-c, LDL-c/HDL-c, CRP and HbCO between the populations of smokers and non-smokers under the age of 30.

Samely, the levels of CT, TG, LDL-c, CT/HDL-c, LDL-c/HDL-c, CRP and HbCO are significantly higher for the smokers who are older than 30 years comparing to non smokers at the same age range, while that HDL-c levels are lower (Table 6).

Table 5: Biological parameters according to duration and the frequency of the tobacco smoking

	Mean (n=25) duration <10 years	Mean (n=18) duration >10 years	p (t-test)	Mean (n=15) <15 cig day^{-1}	Mean (n=28) >15 cig day^{-1}	p (t-test)
CT (g L^{-1})	1.23±0.16	1.72±0.18	8.15×10^{-11}	1.30±0.26	1.50±0.29	0.01
TG (g L^{-1})	0.62±0.15	1.12±0.21	6.27×10^{-10}	0.70±0.26	0.90±0.30	0.01
HDL-c (g L^{-1})	0.42±0.05	0.33±0.02	4.90×10^{-10}	0.41±0.06	0.37±0.05	0.03
LDL-c (g L^{-1})	0.74±0.12	1.14±0.14	2.09×10^{-9}	0.80±0.20	0.97±0.25	0.009
CRP (mg L^{-1})	1.12±1.00	1.28±1.06	0.31	1.12±1.08	1.23±1.00	0.37
CT/HDL-c	2.98±0.65	5.23±0.79	1.25×10^{-11}	3.35±1.15	4.23±1.33	0.01
LDL-c/HDL-c	1.80±0.44	3.47±0.66	3.01×10^{-10}	2.00±0.80	2.74±1.02	0.01
HbCO (%)	5.62±2.05	5.61±1.93	0.49	4.22±1.37	6.36±2.48	0.0003

CT: Total Cholesterol, TG: Triglycerides, HDL-c: High-Density Lipoprotein Cholesterol, LDL-c: Low-Density Lipoprotein Cholesterol, CRP: C-Reactive Protein, HbCO: Carboxyhemoglobin

Table 6: The influence of the age for the populations of smokers and non-smokers

	Mean (n=10) Non-smokers (<30 years)	Mean (n=30) smokers (<30 years)	p (t-test)	Mean (n=7) non-smokers (>30 years)	Mean (n=13) smokers (>30 years)	Percentage pathological levels in smokers
CT (g L^{-1})	1.27±0.23	1.29±0.20	0.39	1.49±0.12	1.75±0.23	Moderate risk: 7.69%
TG (g L^{-1})	0.66±0.21	0.67±0.15	0.41	0.88±0.19	1.20±0.19	Moderate risk: 7.69%
HDL-c (g L^{-1})	0.45±0.06	0.41±0.05	0.03	0.47±0.09	0.33±0.02	High risk: 92.31%
LDL-c (g L^{-1})	0.59±0.18	0.79±0.15	0.004	0.69±0.19	1.17±0.20	Moderate risk: 38.46%
CRP (mg L^{-1})	0.53±0.28	1.08±0.96	0.003	0.47±0.21	1.44±1.14	0%
CT/HDL-c	2.86±0.55	3.29±0.91	0.04	3.26±0.67	5.39±0.92	100%
LDL-c/HDL-c	1.34±0.43	2.02±0.62	0.0004	1.50±0.56	3.62±0.76	76.92%
HbCO (%)	2±0	5.53±2.57	1.35×10^{-8}	2±0	5.80±1.94	100%

CT: Total Cholesterol, TG: Triglycerides, HDL-c: High-Density Lipoprotein Cholesterol, LDL-c: Low-Density Lipoprotein Cholesterol, CRP: C-Reactive Protein, HbCO: Carboxyhemoglobin

Table 7: The influence of the BMI in the populations of smokers and non-smokers

	Mean non-smokers (n=9) BMI <23 kg m^{-2}	Mean smokers (n=26) BMI <23 kg m^{-2}	p (t-test)	Mean non-smokers (n=8) BMI >23 kg m^{-2}	Mean smokers (n=17) BMI >23 kg m^{-2}	p (t-test)
CT (g L^{-1})	1.36±0.24	1.41±0.31	0.32	1.36±0.21	1.46±0.29	0.16
TG (g L^{-1})	0.75±0.18	0.80±0.31	0.27	0.75±0.28	0.88±0.29	0.14
HDL-c (g L^{-1})	0.47±0.08	0.39±0.06	0.006	0.44±0.07	0.37±0.06	0.01
LDL-c (g L^{-1})	0.55±0.22	0.89±0.25	0.0005	0.73±0.08	0.93±0.23	0.002
CRP (mg L^{-1})	0.55±0.29	0.81±0.54	0.04	0.45±0.20	1.78±1.28	0.0002
CT/HDL-c	2.93±0.57	3.82±1.36	0.005	3.14±0.69	4.07±1.29	0.01
LDL-c/HDL-c	1.15±0.41	2.43±1.02	4.26×10^{-6}	1.69±0.39	2.06±0.97	0.001
HbCO (%)	2±0	5.64±2.54	1.81×10^{-6}	2±0	5.57±2.18	2.34×10^{-6}

CT: Total Cholesterol, TG: Triglycerides, HDL-c: High-Density Lipoprotein Cholesterol, LDL-c: Low-Density Lipoprotein Cholesterol, CRP: C-Reactive Protein, HbCO: Carboxyhemoglobin

Study of biological parameters according to BMI: For the population of smokers, the statistical study showed that the difference was significant between those with BMI above and below 23 kg m^{-2} for CRP and was not significant for CT, TG, HDL-c, LDL-c, CT/HDL-c and LDL-c/HDL-c and HbCO. A similar study by Shamai *et al.*[17] found similarity for LDL-c result and discordance for HDL-c and TG results, furthermore, another study by Raju *et al.*[18] found no significant difference in CT, TG and HDL-c, which is in agreement with the obtained results.

CRP levels were significantly higher in the smoker population above 23 kg m^{-2} compared to those below 23 kg m^{-2} and a low positive linear correlation was found between BMI and CRP (r = 0.479). Several studies concluded that CRP is strongly associated with BMI[19,20]. From the obtained results, it may be said that the atherogenic risk in smokers is not influenced by BMI.

Comparing groups of smokers and non-smokers with BMI less than 23 kg m^{-2}, found a significant statistical difference for HDL-c, LDL-c, CRP, HbCO, CT/HDL-c and LDL-c/. The HDL-c and was not significant for CT and TG. Moreover, in smokers and non-smokers above 23 kg m^{-2}, the statistical study showed that the difference was significant for HDL-c, LDL-c, CRP, HbCO, CT/HDL-c and LDL-c/HDL-c ratios and was not significant for CT and TG. It may be concluded that the atherogenic risk is increased among smokers compared to non-smokers having similar BMI (Table7).

Study of biological parameters according to family history of dyslipidemia: In the smoking population, statistical study showed that the difference was significant for CT, TG, LDL-c, CT/HDL-c and LDL-c/HDL-c ratios (p=0.01) and was not significant for HDL-c and CRP levels (0.08 and 0.85, respectively) between smokers with and without family history of dyslipidemia, in fact, the group of smokers without family history of dyslipidemia shows higher levels of the abovementioned significant parameters. The obtained results can be explained by the clear difference between the two groups of smokers with and without family history according to:

- **Age:** Without family history of dyslipidemia (32.58±3.8 years); with family history of dyslipidemia (25.52±3.7 years)
- **The duration of smoking:** Without family history of dyslipidemia (21.92±10.5 years); with without family history of dyslipidemia (16.53±10.57 years)
- **The frequency of smoking:** Without family history of dyslipidemia (14.80±3.55 cig day^{-1}); with family history of dyslipidemia (8.1±3.6 cig day^{-1})

In smokers and non-smokers with no family history of dyslipidemia, the statistical comparison showed that the difference was significant for CT, TG, HDL-c, LDL-c, CRP and CT/HDL-c and LDL-c/HDL ratios (p=0.01, 0.02, 0.001, 0.00008, 0.0002, 0.0006 and 0.00001, respectively), indeed, CT, TG, LDL-c, CRP, CT/HDL-c, LDL-c/HDL-c levels are significantly higher and HDL-c was significantly lower in the smokers without family history of dyslipidemia comparing to the non-smoking population with no family history of dyslipidemia.

On the other hand, in smokers and non-smokers with a family history of dyslipidemia, LDL-c, CRP, CT/HDL-c and LDL-c/HDL-c levels are significantly higher and the HDL-c level was significantly lower in the population of smokers with family history of dyslipidemia (with 29.41% at a high risk),comparing to the non-smoking population with a family history.

Study of biological parameters according to physical activity: Within smokers, the statistical study showed that the difference was not significant for CT, TG, HDL-c, LDL-c, CRP and CT/HDL-c and LDL-c/HDL-c and HbCO (p=0.31, 0.38, 0.08, 0.31, 0.31, 0.24, 0.26, 0.09) between active smokers and non-active smokers, a similar study[21] corroborate the obtained results in this study, the reduced size of the sample may be an explanation.

Comparison of non-active smokers and non-smokers, the statistical study showed that the difference was significant for HDL-c, LDL-c, CRP, HbCO and CT/HDL-c and LDL-c/HDL-c ($p=0.0005$, 0.0001, 0.004, 0.002, 6.35×10^{-6}, 3.93×10^{-8}), whereas, it was not significant for CT and TG (0.25, 0.23), the levels of LDL-c, CRP, HbCO, CT/HDL-c and LDL-c/HDL-c are significantly higher and the HDL-c level was significantly lower for the population of sedentary smokers.

Within the active population, the comparison of smokers and non-smokers group showed that the parameters: LDL-c, CRP, HbCO, CT/HDL-c and LDL-c/HDL-c levels are significantly higher in the active smoker's population.

CONCLUSION

The clinical manifestations of atherosclerosis are one of the common important public health problems due to its prevalence. Atherosclerosis is a multifactorial disease that becomes symptomatic only after several years of insidious evolution. Smoking, among the most important risk factor of the disease, due to its effects on the lipid profile and inflammatory markers. The present study consists in determining the levels of CT, TG, HDL-c, LDL-c, CRP and HbCO and calculating the ratios CT/HDL-c and LDL-c/HDL-c in the studied populations. Atherogenic indices (CT/HDL-c, LDL-c/HDL-c and CRP) are compared between populations of smokers and non-smokers in the city of Batna (Algeria) and have established a relationship between tobacco smoking and atherosclerosis. It can be concluded that smoking increases the risk of the development of atherosclerosis.

REFERENCES

1. World Health Organization, 2018. Smoking. https://www.who.int/fr/news-room/fact-sheets/detail/tobacco.
2. Keita B., 2014. World Health Organization. http://www.afro.who.int/fr/news/journee-mondiale-sans-tabac-setif-ville-sans-tabac.
3. Alain, V. and B. Alain, 2005. Toxicologie. 2nd Edn., Lavoisier, Paris, pp: 853-873.
4. Centers for Disease Control and Prevention, 2018. Health Effects of Cigarette Smoking. National Center for Chronic Disease Prevention and Health Promotion. https://www.cdc.gov/tobacco/data_statistics/fact_sheets/health_effects/effects_cig_smoking/.
5. Centers for Disease Control and Prevention, 2018. Heart Disease and Stroke. https://www.cdc.gov/tobacco/basic_information/health_effects/heart_disease/.
6. Alexander, G., M. Tristan, R. Aldo and M. Enrico, 2016. Tobacco and arteritis. Archives of the mals of the heart and the Vaissaux, pp: 18-24.
7. Catherine, V.B., 1997. Tobacco use and cardiovascular risk. The Internal Med. Rev., 18: 702-708.
8. Isabelle, B., 2010. Pathology Data in Vascular Pathology: Vascular Medicine Treatise, Elsevier Masson, Paris, pp: 5-28.
9. Ranjit, P.M., R.B. Pothineni and G. Guntuku, 2015. Estimation of lipid profile and assessment of cardiovascular risk in smokers by using new atherogenic indices. Asian J. Pharm. Clin. Res., 8: 247-250.
10. Dobiasova, M., 2006. AIP-atherogenic index of plasma as a significant predictor of cardiovascular risk: From research to practice. Vnitr Lek., 52: 64-71.
11. Haj Mouhamed, D., A. Ezzaher, F. Neffati, L. Gaha, W. Douki and M.F. Najjar, 2013. Association between cigarette smoking and dyslipidemia. Immunoanalyse Biol. Specialisee, 28: 195-200.
12. Kavita, G.P., M.G. Nanavati and R.N. Gonsai, 2014. A comparative study of lipid profile among smokers and non smokers. Int. J. Res. Med., 3: 76-81.
13. Ole, D.M., 1988. Lipid effects of smoking. Am. Heart J., 115: 272-275.
14. Nesje, L.A. and D.M. Ole, 1985. Plasma HDL cholesterol and the subclasses HDL2 and HDL3 insmokers and non-smokers. Artery, 13: 7-18.
15. Deodurg, P.M. and Srikanth, 2014. Effect of cigarette smoking on lipid profile. J. Biomed. Pharm. Res., 3 (3).
16. Hassan, E.E., 2013. Effect of cigarette smoking on Lipid profile in male at collage of police and low Khartoum, Sudan. Asian J. Biomed. Pharm. Sci., 3: 28-31.
17. Shamai, L., E. Lurix, M. Shen, G.M. Novaro and S. Szomstein *et al*., 2011. Association of body mass index and lipid profiles: Evaluation of a broad spectrum of body mass index patients including the morbidly obese. Obes. Surg., 21: 42-47.
18. Raju, S., B.D. Singh, N.S. Verma, S. Tiwari and J. Narayan *et al*., 2015. Study of body mass index (BMI) and lipid profile of blood donors of north Indian population: A cross sectional study. Int. J. Biomed. Res., 6: 425-428.
19. Nicholas, J.T., G.N. Borge, M.H. Roger, Z. Jeppe and M.F. Tim *et al*., 2011. C-reactive protein levels and body mass index: Elucidating direction of causation through reciprocal Mendelian randomization. Int. J. Obes. (Lond)., 35: 300-308.
20. Marjolein, V., M.B. Lex, M.M. Geraldine, H.W. Mark and B.H. Tamara, 1999. Elevated C-reactive protein levels in overweight and obese adults. JAMA, 282: 2131-2135.
21. John, S., P. Christof, B.P. Demosthenes, C. Christina and Z. Akis *et al*., 2003. Physical activity, high density lipoprotein cholesterol and other lipids levels, in men and women from the ATTICA study. Lipids Health Dis., 2: 3.