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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

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ABSTRACT: ***Purpose:*** *Hazard of smoking tobacco is believed to be minimized by smoking hubble-bubble (HB) instead of cigarettes. Our aims were to evaluate and compare the effect of smoking on antioxidant status, lipid profile and cardiopulmonary quality in cigarette and HB smokers*. ***Methods:*** *68 male sedentary smokers and nonsmokers having a good health participated in this study. We consider cigarette smokers; all subjects who consumed greater than or equal to 10 pack-years (PA). In fact, hookah smoker subjects, those having consumption greater than or equal to 5-year Hookah (YH). The subjects were divided into three equal groups. Cigarette smokers group, n = 23 (CS), a hookah smokers group, n = 22 (HS) and another non-smokers group, n = 23 (NS). The subjects were invited to undergo spirometry and exercise testing on speedwalk. Blood samples were collected at fasting for lipid profile determination and antioxidant status.* ***Results:*** *for all values explored of spirometry, the statistical analysis showed no difference between the two smoke methods. Our biochemical analysis showed no significant difference in plasma TG, HDL-C and reports HDL-C/TG and TC / HDL-C between CS and HS groups. The HDL-C plasma concentration of NS group was significantly higher than both CS and HS groups. Values are respectively 1.12 ± 0.12 (mmol.l-1) 0.99 ± 0.04 (mmol.l-1) and 0.97 ± 0.05 (mmol.l-1). The MDA concentrations and α-tocopherol are almost similar in subjects of both smoke methods (CS and HS). The MDA average concentration was 1.387 ± 0.095 (µmol.l-1) in CS, 1.363 ± 0111 (µmol.l-1) in HS and 1.154 ± 0.17 (µmol.l-1) in NS. For CS, SOD is significantly higher than HS and NS groups (1651.3 ± 87.2 (U /gHg) Vs 1545.1 ± 105.9 (U /ghg) Vs 1432.1 ± 171.2 (U /gHg) respectively). No difference between these two smoke methods concerning GR. HS and CS have a similar SBP and resting HR and significantly higher than those of non-smokers group (p <0.001). For HS group, concerning VO2max, the MAS and CI, statistical analysis showed a significant difference compared to CS subjects (p <0.01, p <0.001 and p <0.001 respectively).* ***Conclusions:*** *This study reinforces the evidence that the hookah use is associated with exposure to toxic substances and produces the same effects as cigarettes. Given the harmful nature of hookah smoke, impact on human health may be similar or even worse than cigarette smoking, it is recommended that men who have a habit of hookah smoking as an alternative to cigarette smoking tobacco should be informed about the potential adverse effects of their habit on cardiorespiratory quality and metabolic levels to stop it.*

Keywords: *cigarettes smokers; hookah smokers; antioxidant status; lipid profile; cardiopulmonary.*

I. INTRODUCTION

The use of hookah is quite widespread and socially accepted by men and little by little by women. Indeed, since the 90s, the popularity of this smoke type is in increase. To United States, 10-20% of some populations of young adults are hookah users [1]. This popularity may be explained, at least in part, to the perception that hookah is less toxic than cigarettes [2-3-4]. However, pregnant Lebanese women replace the cigarette by hookah thinking that it acts for the well-being of their baby. [5]

In a study conducted in Saudi Arabia, 49.7% of respondents reported that hookah is less harmful than cigarettes, 60.5% believe that harmful substances were purified by filtration of water and 67 8% of them also believe that hookah is not addictive [6]. Contrary to this perception, recent studies indicate that smoking hookah causes harmful effects for health and contains many toxic substances, such as nicotine [7-8] and carbon monoxide [9] that, according to Barnett TE et al [10], take 40 puffs of hookah is equivalent to smoking two packs of 20 cigarettes.

These effects are more marked in cardiovascular and respiratory systems, and also throughout the body. Studies reveal significant complications associated with the use of hookah: respiratory diseases and lungs cancers [2-11], HDL is often lowered and the cardiovascular risk is increased to 1.9 [12]. The risk of stroke is doubled among hookah consumers [13]. Another study suggests that a cigarette can release not more than 10mg of tar, while a hookah produces 10 to 100 times more tar. The only certainty is that hookah releases as much tar in an average of 26 cigarettes [10].

Depending on the measured toxicant, a session of hookah produces on average the equivalent of 1-50 cigarettes [1]. The combustion temperature reaches 450 ° with the hookah, while it culminates 850 ° with cigarette. Inhaled volumes per puff are 1 to 2 (L) for a hookah consumer , against 0.003-0.005 (L) for a cigarette smoker, that is to say, a subject inhales 1L of cigarettes smokes , while hookah smoker inhales from 60 to 90 L per session of hookah [13]. The Caroline O's study et al [14] showed that expired CO after consumption of a hookah is 4.5 times higher compared to cigarettes. The hookah is more toxic than cigarettes. [15]. In other studies, the nicotine amount in a hookah session is equivalent to the consumption of 10-20 cigarettes. The plasma's nicotine amount peaked after 5 minutes with a cigarette and after 30 minutes with a hookah [14].

Cigarettes consumption and hookahs presents risks of addiction, illness and even death and it seems important to assess, through this study, the dangers of smoking by measuring cardio-vascular, respiratory lipid and oxidative stress in these two smoke methods. We wanted to bring to the knowledge the harms of hookah consumption compared to cigarettes with our Tunisian sedentary adults.

II. METHODS

2.1. Subjects: 68 male sedentary smokers and nonsmokers having good health participated in this study. Their mean values of age, height and weight were respectively 44.7 ± 4.5 years, 174.3 ± 2.3 cm, 71.3 ± 2.7 kg. After receiving a complete verbal description of protocol, risks and benefits of the study, the subjects provided written consent to an experimental protocol approved by the Researsh Ethics Committee of the Faculty Medicine's, from University of Sfax in Tunisia.

Cigarette and hookah smoking subjects have been recruited on the basis of the number of cigarette and hookah per day and career period. We consider cigarette smokers; all subjects who consumed greater than or equal to 10 pack-years (PA) and an average score of tobacco dependence of 4.33 ±1.67 measured by the Fagerström Nicotine Dependence Scale [16].

In the absence of specific international assessment, we quantified the use of hookah, as in the Kiter study et al [17], year hookah (YH) and in kg of cumulative tobacco. The tobacco used for hookah weighs between 10 and 25 g. [18] In fact, hookah smoker subjects, those having a consumption greater than or equal to 5-year Hookah (YH) [19] or 45,625 kg of cumulative tobacco.

The subjects were divided into three equal groups. Cigarette smokers group, n = 23 (CS), a hookah smokers group, n = 22 (HS) and another non-smokers group, n = 23 (NS).

2.2. Materials and measured parameters

The subjects of the three groups have been subjected to a test session and chemical and metabolic analysis. This session includes: An anthropometric review and body composition, A biochemical analysis, Pulmonary function review (RFE), A stress test on a treadmill. All these measures have been performed by the same examiner to avoid methodological uncertainties.

2.2. 1. Anthropometric measures

The subjects mass was measured with an impedancemeter (TANITA Model TBF 350) in kilograms and standing height (m) was measured with a stadiometer fixed. BMI is calculated for each subject using the following formula: BMI = weight / Size2 (kg.m-2).

2.2. 2. Cardiovascular parameters measures

The systolic and diastolic blood pressures were measured in the right arm by an electronic sphygmomanometer (OMRON 70 - CP) with digital display.

2.2. 3. Exercise testing

Measures of VO2 max and the recovery ability post-exercise were examined at the triangular test with speedwalk (COSMED Pulmonan-Function Equipment 37 Via dei Piani di monte Savello I-00040 Rome ITALY). This dynamic test and maximum, until fatigue, consists in increasing the speed of 1km /h every 2 min, after warm up for 5 min with a speed of 6km / h. Heart rate and VO2 during the test and recovery were measured using an analyzer (version 1.2 PRO Fit mate COSMED).

2.2. 4. Biochemical Measures

Analyses were performed in the laboratory of Pharmacology, Faculty of Medicine of Sfax. Smokers were instructed to refrain from smoking for the one hour period prior to reporting to the lab suggested by Dietrich et al [20].

Venous blood samples (ante-cubital vein) were performed in dry tubes under basal conditions (8 am morning). After centrifugation, the sera were frozen at -80 ° C until analysis. Total cholesterol (TC), triglycerides (TG), and high-density lipoprotein cholesterol (HDL-C) were measured in all subjects after 12 hours fasting and 9 hours sleep using standardized techniques described by Wegge JK [21].

Low-density lipoprotein cholesterol (LDL-C) was calculated as described by the Friedewald formula [22]: [LDL = TC - HDL - (TG /2, 18)].

Plasma concentrations of SOD, GPx and the SAT were measured by spectrophotometrically using a spectrophotometer type DU-640 (Beckman Instruments, Inc.., California. United States) and the dosage kits of SAT and anti-oxidant enzymes (SOD and GPx) were learned from Randox laboratories.

2.2. 5. Urinary cotinine measures

Urine samples collected at the end of the day in sterile vial were kept in the laboratory at -20 ◦ C until analysis. Free urinary cotinine, a major catabolite of nicotine, was measured by HPLC-UV according to a consensus protocol [23].

2.2. 5. Carbon monoxide measures

The rate measuring of exhaled CO is well correlated with CO bound to hemoglobin. Its half-life is approximately 6 hours. The measurement of exhaled CO can assess the level of tobacco intoxication.

2.2. 6. Respiratory parameters measures: Respiratory Functional Exploration (RFE).

The subjects must have stopped smoking at least for an hour. It should be quiet and at rest. It must not have been violent effort for at least 30 minutes.

The subject is seated and the nose is blocked by a pliers. He is asked to blow the fastest and strongest possible through a mouthpiece connected to a spirometer (MIR Spirobank G USB Spirometer Roma-Italy serial No. A23-048 00 503) connected to computer to measure capacity and lung volumes, which are then compared with theoretical standards namely: The Peak Expiratory Flow (PEF), the Expiratory Volume in one second (FEV), the Forced Vital Capacity (FVC) and FEV / FVC or Tiffeneau index. Repeat the measurement at least three times to max change of 200ml.

2.3. Statistical analysis

All statistical tests were performed using STATISTICA Software (StatSoft, France). Analysis of variance (ANOVA) was applied. Fischer LSD post hoc test was performed where appropriate. Differences between cigarette smokers and hookah smokers were analyzed using non-paired Student's t-test. Statistical significance was set at $P<0.05$. All values are expressed as mean ± SD.

III. RESULTS

We reported in (Table.1) respiratory parameters changes in percentage of predicted values of our entire population. Compared to non-smokers group, ANOVA showed significant differences for all measured parameters, except for Tiffeneau index , our study revealed no significant difference in the three groups ($p = 0,362$). Well for all values explored, the statistical analysis showed no difference between the two modes of smoke. (Table1). The application of post-hoc LSD test showed that hookah smokers have significantly lower FVC than non-smokers. FVC Values of CS and HS groups were respectively 95.5 ± 4.5% and 93.1 ± 7.9%, lower than that of the NS group (100.5 ± 5.8%).

Regarding the PEF, the two groups cigarette smokers and hookah are homogeneous on the one hand, and heterogeneous with non-smokers, on the other hand ($p <0.001$). Values were respectively 102.5 ± 6.7%; 101 ± 4.3% and 110.3 ± 5.2%. Non-smokers subjects justified also FEV values significantly higher than both groups CS and HS (103 ±5 for the NS group and 94.1 ± 6.5; 95.3 ± 6.6 for CS and HS groups respectively). LSD Post-hoc test showed that cigarette smokers subjects and hookah smokers subjects, have FEF25-75 and FEF 50 values, significantly lower ($p <0.01$) compared to the non-smokers group. The results of the study failed to demonstrate a significant difference in these variables between the CS and the HS group.

Table1. Respiratory parameters changes of three groups: NS, CS and HS.

Parameters	Means±SD			ANOVA
	NS (n=23)	CS (n=23)	HS (n=22)	
FVC (%)	100,5±5,8	95,5±4,5	93,1±7,9**	F(2;33) = 4,54 ; p = 0,018
FEV1(%)	103,3±5	94,1±6,5***	95,3±6,6**	F(2;33) = 8,43 ; p < 0,001
PEF (%)	110,3±5,2	102,5±6,7**	101±4,3***	F(2;33) = 10,42 ; p < 0,001
FEV1/FVC (TI)	1,03±0,07	0,99±0,06	1,03±0,11	F(2;33) = 1,05 ; p = 0,362
FEF 25-75 (%)	103,3±10,1	94,9±5**	93,9±4,4**	F(2;33) = 6,55 ; p = 0,004
FEF 50 (%)	99,8±4,5	94,7±2,6**	93±3,8***	F(2;33) = 10,8 ; p < 0,001

Legend: NS, non-smokers; CS, cigarette smokers; HS, hookah smokers; FVC, forced vital capacity; FEV1, expiratory volume in one second; PEF, peak expiratory flow; TI, Tiffeneau index; *, **, ***: Significant difference compared with non-smokers at p < 0,05 , p< 0,01 , p < 0,001 respectively ; #, ##, ## #: Significant difference compared with smoking cigarettes at p <0.05, p <0.01, p <0.001, respectively.

According to Table 2, our biochemical analysis showed no significant difference in plasma TG, HDL-C and reports HDL-C/TG and TC / HDL-C between CS and HS groups. Similarly, we recorded in these groups increased report HDL-C/TG and decreased report TC / HDL-C by the NS group compared to HS and CS groups (p <0.001). In addition, the HDL-C plasma concentration of NS group was significantly higher than both CS and HS groups. Values are respectively 1.12 ± 0.12 (mmol.l-1) 0.99 ± 0.04 (mmol.l-1) and 0.97 ± 0.05 (mmol.l-1). According to ANOVA, the TC plasma concentration is statistically similar among subjects in the three groups. In applying the LSD Post-hoc test, we recorded only one difference (p <0.05) in the CS group compared to the HS group (table 2). Finally, subjects in NS groups, CS and HS showed similar values LDL-C (p = 0.079).

Table2. Plasma lipid changes of three groups: NS, CS, and HS.

Parameters	Means±SD			ANOVA
	NS (n=23)	CS (n=23)	HS (n=22)	
HDL-C (mmol.l^{-1})	1,12±0,12	0,99±0,04***	0,97±0,05***	F(2;33) = 12,19 ; p < 0,001
LDL-C (mmol.l^{-1})	2,89±0,22	2,9±0,1	2,75±0,17	F(2;33) = 2,75 ; p = 0,079
TG (mmol.l^{-1})	0,9±0,2	1,28±0,22***	1,38±0,32***	F(2;33) = 12,51 ; p < 0,001
TC (mmol.l^{-1})	4,42±0,12	4,48±0,09	4,36±0,11#	F(2;33) = 3,73 ; p = 0,035
HDL-C/TG	1,29±0,32	0,8±0,15***	0,74±0,15***	F(2;33) = 22,45 ; p < 0,001
TC/HDL-C	4±0,44	4,52±0,18***	4,49±0,22***	F(2;33) = 11,09 ; p < 0,001

Legend: HDL-C, height density lipoprotein cholesterol; LDL-C, low density lipoprotein cholesterol; TC, Total cholesterol ;TG, triglyceride; * , ** , *** : Significant difference compared with non-smokers at p< 0,05 , p< 0,01 , p < 0,001 respectively ; #, ##, ## #: Significant difference compared with smoking cigarettes at p<0.05, p <0.01, p <0.001, respectively.

Table 3 shows that there was no significant difference in TAS, GR, SOD, Glutathione peroxidase (GPx) and α-tocopherol between the CS subjects and HS, except the SOD concentration of CS group is greater than that of HS.

Concerning Malondialdehyde (MDA) and α-tocopherol, the ANOVA showed a statistically significant difference (p <0.001) of smoker groups compared to NS. For cigarette smokers, by applying the post hoc test, the analysis showed that the concentration of these two variables is almost similar in subjects of both smoke modes (CS and HS).

The MDA average concentration was 1.387 ± 0.095 (µmol.l-1) in CS, 1.363 ± 0111 (µmol.l-1) in HS and 1.154 ± 0.17 (µmol.l-1) in NS. Regarding tobacco effect on superoxide dismutase concentrations, we observed a statistically significant difference (p = 0.001). For CS group, this concentration is significantly higher than HS and NS groups (1651.3 ± 87.2 (U /gHg) Vs 1545.1 ± 105.9 (U /ghg) Vs 1432.1 ± 171.2 (U /gHg) respectively). For glutathione reductase plasma concentrations, ANOVA showed lower values in smoking subjects, which differ significantly from the values of NS subjects. The post hoc test application showed no difference between these two smoke methods.

Table3. Antioxidants plasma changes of three groups: NS, CS, and HS.

Parameters	Means±SD			ANOVA
	NS (n=23)	CS (n=23)	HS (n=22)	
GPX (U/gHg)	33,84±5,07	38,84±4,31**	39,12±2,6**	F(2;33) = 5,6 ; p = 0,008
SOD (U/gHg)	1432,1±171,2	1651,3±87,2***	1545,1±105,9*#	F(2;33) = 8,1 ; p = 0,001
MDA (µmol.l^{-1})	1,154±0,17	1,387±0,095***	1,363±0,111***	F(2;33) = 10,15 ; p < 0,001
GR (U/gHg)	10,46±2,01	8,3±1,55**	8,21±1,6**	F(2;33) = 5,43 ; p = 0,009
TAS (U/gHg)	1,41±0,02	1,68±0,01**	1,53±0,02*	F(2;33) = 5,38 ; p = 0,008
α-tocophérol (µmol.l^{-1})	5,78±0,95	4,24±0,88***	4,19±0,88***	F(2;33) = 12,42 ; p < 0,001

Legend: GPx, Glutathione peroxidase; SOD, superoxide dismutase; MDA, malondialdehyde; GR, Glutathione reductase; TAS, Total antioxidant status; * , ** , *** : Significant difference compared with non-smokers at P< 0,05 , p< 0,01 , p < 0,001 respectively ; #, ##, ## #: Significant difference compared with smoking cigarettes at p <0.05, p <0.01, p <0.001, respectively.

The Post-Hoc LSD test allowed us to conclude that the two groups HS and CS have a similar SBP and resting HR and significantly higher than those of non-smokers group (p <0.001). Similarly, we recorded in these groups similar values of DBP for all subjects (p <0.05). LSD Post-hoc test application showed no significant difference in the DBP values of our population (table 4).

For HS group, concerning VO2max, the MAS and CI, statistical analysis showed a significant difference compared to CS subjects (p <0.01, p <0.001 and p <0.001 respectively). Similarly for MAS, we recorded, significant differences between smoker groups and non-smokers (p <0.01 CS Vs NS and p <0.05 HS Vs NS). Best recovery for HS subjects then of NS and finally of CS which differs alone of NS subjects (p <0.05).

Table4. Cardiovascular parameters changes of three groups: NS, CS and HS.

Parameters	Means±SD			ANOVA
	NS (n=23)	CS (n=23)	HS (n=22)	
Resting HR (beats.m^{-1})	78±4	91±2***	93±4***	F(2;33) = 66,52 ; p < 0,001
Systolic.BP(mmHg)	131±3	138±3***	141±4***	F(2;33) = 27,91 ; p < 0,001
Diastolic.BP(mmHg)	85±6	87±5	86±4	F(2;33) = 0,48 ; p = 0,62
Urinary cotinine (µg.ml^{-1})	0.035±0.012	4.314±1.082***	4.439±0.94***	F(2;33) = 73,29 ; p < 0,001
MAS (km.h^{-1})	10,5±0,9	11,6±0,7**	9,9±0,6*###	F(2;33) = 15,15 ; p < 0,001
VO2max (ml. Kg^{-1}. Min^{-1})	37,5±1,6	38,9±2,5	36,6±1,2##	F(2;33) = 4,79 ; p = 0,015
Recovery index(RI)	15,8±0,7	14,8±1*	16,5±1,4###	F(2;33) = 7,16 ; p = 0,003

Legend: Resting. HR, resting hears rat; Systolic.BP, systolic blood pressure; Diastolic.BP, diastolic blood pressure; VO2max, maximum oxygen uptake; MAS, maximal aerobic speed.

IV. DISCUSSION

In our study, with 68 participants, compared to cigarette smoke, hookah consumption was also associated with a high concentration of TG of report HDL-C/TG and TC / HDL-C and lowered concentrations of HDL -C. These results are also consistent with several other studies [24-25].Thus, there is now overwhelming evidence that, like cigarettes, hookah consumption involves a significant increase in triglyceride and LDL-C [25].In addition, all studies that have included biochemical measures, specify that hookah smoking involves inhaling several liters of smoke and the smoke is known to contain many other toxic substances [26-27].

Regarding the effects on respiratory parameters, the results presented here suggest that cigarette and hookah can produce the same effect profiles. The study of Al-Fayez et al.[28], showed that for both hookah and cigarette smokers, FVC mean value was significantly decreased compared with the FEV. And contrary to our results, the risk was higher for the hookah. This discrepancy may be partly explained by the diversity of protocols (age, sex) as well as career period and the smoke quantity cumulative for each subject.

However, for both cigarettes and hookah, a significant reduction in capacity and measured volumes was observed (Table 1).

Our findings support the conclusions of Raad D et al [29]. This is explained by the smoke amount inhaled by the two smoking modes and increasing effects of nicotine. However, we observed concentrations

peak of urinary cotinine similar for CS and HS. These results are also consistent with several other findings [7-9]. The results similarity observed for cigarette and hookah, suggests a similar risk of alteration and inflammation in the airways by these two smoke methods.

Light of the findings set, we find that non-smokers have a much better antioxidant capacity than cigarette smokers or hookah. This can be explained by the fact that the inhaled smoke is associated with increased oxidative stress and changes in antioxidant [30-31]. In fact, smoke induces deterioration of antioxidant protection system in order to maintain harmful effects of oxidative stress [31-32].

The results obtained revealed that the smoke, whatsoever cigarette or hookah leads to increased TAS concentrations compared to non-smoking subjects. Our results showed that SOD concentrations were significantly higher in smokers than non-smokers. These findings are even more visible in cigarette smokers than hookah. Regarding the tobacco effect on MDA concentrations, we observed an increase in lipid peroxidation products in both groups, which leads us to suggest the presence of oxidative stress in both smoking groups.

In addition, smoking cigarettes or hookah induces almost the same increase of GR concentrations and GPx, in contrast to non-smokers subjects. A vitamin E deficiency leads to increased oxidative stress [33-34]. Based on the α-tocopherol results, we observed a decrease in concentrations of this parameter in both smoker groups, with higher values in non-smoking group. This fat-soluble vitamin is effective against lipid peroxidation and is one of the main fat-soluble antioxidants [35-36].

Our study results confirm the hypothesis that increased oxidative stress due to tobacco consumption, be it cigarettes or hookah, may be related to a decrease in the efficiency of antioxidant system. Through proposed measures to our subjects, some parameters of cardio-respiratory capacity have been illustrated and therefore represent our main results. These are characterized by a high resting HR, as well as systolic and diastolic BP, and this for both smoking groups, be it CS or HS. Our finding is consistent with Caroline advances [14].

Concerning VO2 max, VMA and RI of all our subjects, we found that in the HS group, these variables are lower than those in the CS group. This is due that hookah smoke is more harmful than cigarettes. Conclusions were discussed by Jabbour S et al and Mohammad Y et al [37-38], which found, in hookah smokers, impaired cardiorespiratory function and presence of oxidative stress that would result in a reduced level of physical activity [12-39].

V. CONCLUSION

In conclusion, this study reinforces the evidence that the hookah use is associated with exposure to toxic substances and produces, some, of the same effects as cigarettes. These results should be used to resolve misunderstandings concerning exposure to toxic substances and the risks associated with the hookah use.

Given the harmful nature of hookah smoke, impact on human health may be similar or even worse than cigarette smoking, it is recommended that men who have a habit of hookah smoking as an alternative to cigarette smoking tobacco should be informed about the potential adverse effects of their habit on cardiorespiratory quality and metabolic levels to stop it. Such results can be incorporated into prevention interventions that could help deter more adolescents and young adults to test a smoking method almost deadly certainly.

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Conflict of interests: None

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