Original Article

Effect of Smoking Cessation on Vascular Function by Measurement the Flow-Mediated Dilation: A Comparative Study

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Abstract

Smoking predisposes to endothelial dysfunction; however smoking cessation would have a beneficial effect on cardiovascular risk. The objective of this study was to evaluate the effect of smoking cessation on vascular function.

Methods: This is a comparative, prospective, descriptive, and multicenter study, carried out between March 2018 and April 2021 at two Health Services of the National Gendarmerie and at the Laboratory of Physiology and Functional Explorations of the Faculty of Medicine in Dakar, Senegal. The population included 45 consenting adult men, divided into 3 groups: active smokers, weaned smokers, and non-smokers. We assessed vascular function using the Flow Mediated Dilation (FMD) technique, which consisted of measuring the diameter of the humeral artery at rest and then every 30 seconds after its occlusion.

Results: Non-smokers showed better humeral artery dilation at T1 (30 seconds after occlusion release) with a higher mean FMD than the other 2 groups ($p = 0.0007$). In addition, FMD kinetics showed that control subjects as well as weaned subjects had better arterial compliance ($p < 0.05$) compared to active smokers. FMD1 values were positively correlated with smoking cessation duration ($p = 0.0411$; $R2 = 0.3374$).

Conclusion: Our results showed that stopping smoking improves vascular function and thus constitutes a means of preventing cardiovascular diseases, hence the need to promote smoking cessation in Africa and more particularly in Senegal.

Keywords: Smoking cessation, Vascular function, Flow mediated dilation, FMD cardiovascular risk.

Introduction

Smoking is currently a major public health problem and an important cardiovascular risk factor [(1,2)] due to its high morbidity and mortality. In 2020, 22.3% of the world's population used tobacco, including 36.7% of men and 7.8% of women[(3)].

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According to the WHO, 80% of smokers live in low-income countries and more than 10 million deaths per year are attributed to tobacco [4]. In Africa, smoking appears to be the most important risk factor for myocardial infarction in subjects aged under 45 [5,6]. The WHO estimates that 80% of these deaths will occur in low-income countries, particularly on the African continent [7].

In Senegal, the Global Adult Tobacco Survey (GATS) carried out in 2015 by the National Agency for Statistics and Demography (ANSD), found that around half a million (6.0%) of adults are active smokers, i.e. 11.0% of men and 1.2% of women [8].

It is also known that chronic tobacco intoxication is implicated in the occurrence of several pathologies such as cancers and chronic obstructive pulmonary disease (COPD)[9]. Furthermore, it increases the risk of heart attack, stroke, high blood pressure (hypertension), and arteritis [10]. Smoking also leads to impaired vascular function[11]

The mechanisms of tobacco's action on vascular function are not fully elucidated. However, they are probably linked to the presence of carbon monoxide and free radicals which will induce a reduction in the bioavailability and the action of endothelial vasomotor factors but also reduce the anti-fibrinolytic and anti-platelet aggregation effects of NO which can lead to disorders. hemostasis. All these mechanisms will in the long-term lead to an increase in cardiovascular risk [1,11,12].

The consequences of smoking are well recognized and understood, as are the benefits of quitting [9,13–15]. Several studies have reported the beneficial effect of smoking cessation on cardiovascular risk, particularly through the reduction of morbidity and mortality[16,17]. The objective of our study was to compare vascular function by measuring Flow Mediated Dialation in smoking, weaned and non-smoking subjects.

Materials and Methods

This was a multicenter, descriptive, transversal, prospective, and analytical comparative study carried out between March 2018 and April 2021, at the North Garrison Medical Center of Saint Louis, of the Health Service of the National Gendarmerie of the Caserne Samba Diéry Diallo of Dakar and the Laboratory of Physiology and Functional Explorations of the Faculty of Medicine, Pharmacy and Odontology of the Cheikh Anta Diop University of Dakar (FMPO/UCAD).

Population

The study population consisted of 45 adult male volunteers, aged over 18, and active military personnel. They were divided into 3 groups of 15, the smokers (G1), the weaned (G2) and the non-smoking controls (G3). Any subject with a medical-surgical pathology that could influence the results and those who had followed irregular cessation or who had suffered from passive smoking were not included in the study. Participants were informed of the procedures and objectives of the study and provided written consent to participate in the present study.

Protocol

A complete clinical analysis comprising 2 parts was carried out, firstly an interrogation to collect, among other things, socio-demographic data, and medical history (diabetes, hypertension, dyslipidemia, etc.). The interview also allowed us to determine the notions of active smoking and the number of pack-years (P/A), smoking cessation and the duration of withdrawal as well as the absence of regular active and passive smoking. Secondly, a physical examination was employed to measure anthropometric (age, height, and body weight); and cardiovascular (systolic and diastolic blood pressure) parameters. Blood samples were realized for biological (lipid and glycemic profile) parameters.

The body mass index (BMI) of the subjects was calculated using Quetelet equation, BMI (kg / m^2). The systolic and diastolic arterial pressures (SAP/DAP) measured for every participant on both arms, with an electronic sphygmomanometer, after a 10-minute rest in a seated or lying position on a horizontal plane and the mean arterial pressures (MAP) was evaluated by MAP = (SAP + 2 DAP) / 3. Arterial hypertension was defined according to the New York Heart Association: systolic blood pressure greater than 140 mm Hg and/or diastolic blood pressure greater than 90 mm Hg. Mean SAP, DAP and MAP for each group were also determined.

The biochemical parameters consisted of measuring the fasting blood sugar, C-reactive protein (CRP) and determining the lipid profile of the participants.

All measurements (anthropometrics, biochemical, and cardiovascular parameters) were performed after eight hours of fasting during the morning of the experimental day (i.e., 8:00 h). For vascular function, we used hemodynamic parameters such as Flow medicated dilation or dilation mediated (FMD).

This involved measuring the diameters of the humeral artery and calculating the flow-mediated dilation which consists of compressing the artery for 5 minutes then measuring the diameter of the vessel after the compression is lifted.

The diameters of the humeral artery were measured at different times:

- Before compression: T0
- After compression: T1 = just after lifting the compression; then at T2=2 min; T3 = 5 min; T4 = 10 min; T5 = 15 min; and T6 = 30 min after lifting of the compression

To carry out this work, we used as instruments a cuff of a manual sphygmomanometer to perform the compression of the humeral artery and an echodoppler device (Vascular Doppler Sonotrax 8 MHz) to measure its diameter.

The FMDs at the different times of the ultrasound examination were calculated using the following formula: FMDx = [(Diameter at Tx – Diameter at T0) / Diameter at T0] *100 (18).

Statistical analyzes of data

Data were collected and analyzed in an Excel spreadsheet by determining means and standard deviations. The statistical tests used for the quantitative variables were the analysis of variances (ANOVA) or of covariances (ANCOVA) for the comparison of the groups. Analyzes with the Newman-keuls software were also carried out to determine the differences between the groups. The significance threshold is set at a P-value < 0.05. The Chi-square test was used for the analysis of the qualitative variables with the Pearson coefficient for the determination of the P-values. The correlation curves made it possible to study the interdependence relationships in the evolution of 2 given variables. Significance rates were determined by the Pearson and Spearman tests.

Ethical considerations

Our work was carried out with the authorization of those responsible for the structures and the data was collected confidentially and anonymously.

Results

Anthropometric parameters

The average age between the three groups was comparable. Indeed, the average age was 37.7, 38.2 and 38.8 years for smokers (group 1), withdrawal (group 2) and controls (group 3) respectively. The average weight of the 3 groups was respectively 70.5 kg (G1), 71.7 kg (G2), and 75.9 kg (G3). The average height in G1(smokers), G2 (withdrawals), G3 (controls) was 176.4 cm, 177.7 cm, and 179.5 cm, respectively.

Also, the average BMI of the subjects was almost identical in the 3 groups with 22.3, 22.6, and 23.6 kg/m^2 for G1, G2 and G3 respectively. However, the percentage of overweight individuals was higher in G3 with 33.3% against 6.6% for G1 and G2 p < 0.001, OR = 0.1528; $_{95\%}$CI: 0.063-0.366 (table I).

Table I: Average anthropometric parameters of the 3 groups

Parameters	Group 1	Group 2	Group 3	P-value
Age (years)	37.7 ± 6.7	38.2 ± 5.9	38.8 ± 6.6	0.8843
Weight (Kg)	70.5 ± 4.8	71.7 ± 7.7	75.9 ± 6.2	0.0841
Height (cm)	176.4 ± 4.9	177.7 ± 5.3	179.3 ± 4.1	0.4531
BMI (Kg/m2)	22.3 ± 1.5	22.6 ± 2.3	23.6 ± 2.4	0.3847
Overweight (%)	6.6	6.6	33.3	< 0.001

Group 1: tobacco users; Group 2: weaned; Group 3: controls; BMI: body mass index

Cardiovascular parameters

A history of diabetes, hypertension, dyslipidemia, or other medical pathologies was not found in individuals from the 3 different groups. Examination of the cardiovascular systems of the subjects in the 3 groups showed no detectable clinical abnormalities.

The mean systolic blood pressure (SBP) of G1 was 125.1 ± 6.5 mmHg and 80.5 ± 7.2 mmHg for the mean diastolic blood pressure (DBP). The mean SBP and DBP of G2 were 118.5 ± 6.6 mmHg and 76.1 ± 5mmHg respectively. For G3, mean SBP was 118.5 ± 3.5 mmHg and mean DBP was 76.7 ± 4.4 mmHg. The mean blood pressure of the 3 groups was respectively, 102.8 ± 6.1 mmHg, 97.3 ± 5 mmHg, and 97.6 ± 3.1 mmHg. (Table II).

Table II: Average cardiovascular parameters of the 3 groups

Parameters	Group 1	Group 2	Group 3	P-value
SBP (mmHg)	125.1 ± 6.5	118.5 ± 6.6	118.8 ± 3.5	0.0293
DBP (mmHg)	80.5 ± 7.2	76.1 ± 5.0	76.7 ± 4.4	0.1770
MAP (mmHg)	102.8 ± 6.1	97.3 ±5.0	97.6 ± 3.1	0.0332

Group 1: tobacco users; Group 2: weaned; Group 3: controls; SBP-DBP: systolic and diastolic blood pressure; MAP: average arterial pressure

Biochemical parameters

Furthermore, the fasting blood sugar levels and lipid profiles of individuals in the 3 groups returned normal. The CRP measurements came back negative for the subjects in the 3 different groups.

Hemodynamic parameters

Measurement of humeral artery diameter (HAD)

Concerning the diameter of the humeral artery, before compression, the mean diameter of the humeral artery (HAD0) was 4.22 ± 0.3 mm, 4.45 ± 0.3 mm, and 5.22 ± 0.6 mm respectively for G1 (smokers), G2 (weaned) G3 (controls). After lifting the compression (T1), the HAD1 were 4.78 ± 0.4 mm for G1, 4.91 ± 0.3 mm for G2 and 6.66 0.8 mm for G3.

At T2, the mean diameter of the humeral artery (HAD2) of G1 measured 4.57 ± 0.3mm, 4.70 ± 0.4mm for G2 and 5.73 ± 0.7mm for G3. The results obtained at T3 were 4.44 ± 0.3 mm, 4.53 ± 0.4 mm, and 5.15 ± 0.6 mm for G1, G2 and G3 respectively. The HAD4 were 4.29 ± 0.3 mm, 4.48 ± 0.4 mm, and 5.15 ± 0.6 mm for groups G1, G2 and G3 respectively.

At T5, the HAD5 had given 4.24 ± 0.3 mm in tobacco users (G1), 4.42 ± 0.3 mm in weaned people (G2) and 5.15 ± 0.6 mm in controls (G3). At the last ultrasound time (T6), the HAD6 of G1 was 4.24 ± 0.3 mm, G2 4.43 ± 0.3 mm and G3 5.15 ± 0.6 mm (Table III).

Table III: Average diameters of the humeral artery (HAD) of the 3 groups at the different ultrasound times

Parameters (%)	Group 1	Group 2	Group 3	P-value
HAD0	4.22 ± 0.3	4.45 ± 0.3	5.22 ± 0.6	0.0004
HAD1	4.78 ± 0.4	4.91 ± 0.3	6.66 ± 0.8	< 0.0001
HAD2	4.57 ± 0.3	4.70 ± 0.4	5.73 ± 0.7	0.0002
HAD3	4.45 ± 0.3	4.53 ± 0.4	5.15 ± 0.6	0.0257
HAD4	4.29 ± 0.3	4.48 ± 0.4	5.15 ± 0.6	0.0045
HAD5	4.24 ±0.3	4.42 ± 0.3	5.15 ± 0.6	0.0014
HAD6	4.24 ± 0.3	4.43 ± 0.3	5.15 ± 0.6	0.0013

Kinetics of Flow Mediated Dilation (FMD)

The results at the different times of the ultrasound examination of the kinetics of the FMD of the 3 groups are represented in Figure 1. The average FMD at T1 was for G1 13.51%; G2 10.59% and G3 was 28.15%.

At T2, smokers (G1) had an average FMD equal to -4.25%; the weaned (G2) -0.92% and the controls (G3) at 10.17%. The average FMD at T3 of G1, G2 and G3 were 5.78%, 1.93% and -1.41%, respectively.

Group 1 had a mean FMD of -4.82% at T4; group 2 by 0.56% and group 3 by -1.41%. At T5 the average FMD was 0.6% for group 1, -6.49% and -1.41 for groups 2 and 3 respectively. The mean FMD at T6 was for G1 0.6%; G2 -0.39% and G3 -1.41%.

Thus, the kinetics of the FMD of the 3 groups showed that that of the smokers (G1) was less regular than that of the controls (G3) which was more regular with stability from T3. Furthermore, the kinetics of the weaned (G2) were closer to those of the controls.

It should be noted that the higher the FMD, the greater the artery benefits from good endothelial function. A rapid decrease, over time, in FMD values also shows good vascular compliance.

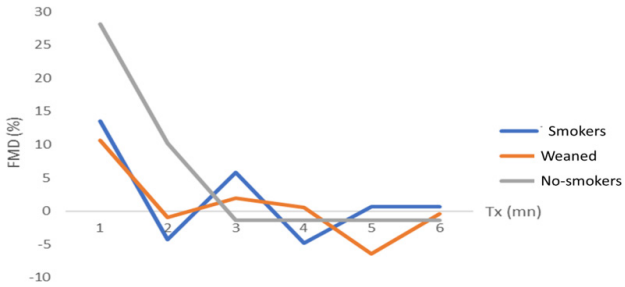
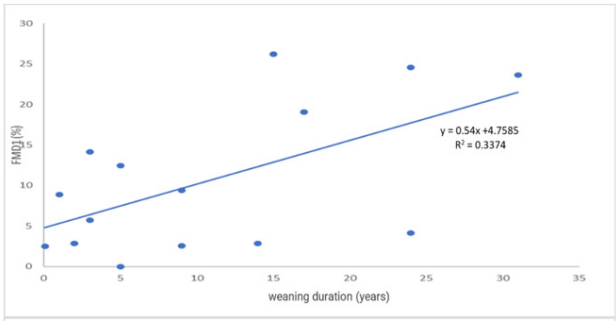


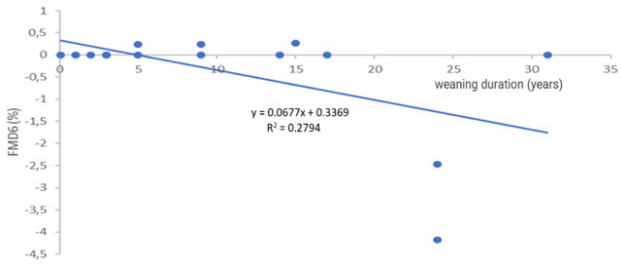
Figure 1: Kinetics of FMD 3 groups at different times of the ultrasound examination

The links between the FMD values and the different parameters were determined using the correlation curves.

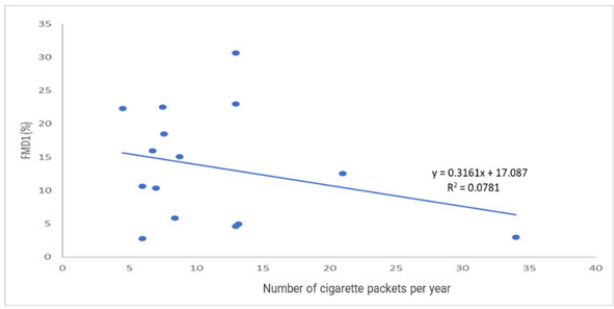
The correlation curve between FMD1 of weaned subjects and the duration of their withdrawal showed a positive trend because FMD1 had higher values when the duration of withdrawal was longer (p = 0.0411) (Figure 2a).



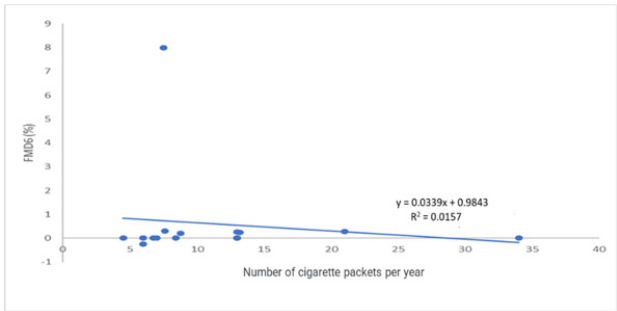
The correlation curve between FMD6 (at 30 min after compression) of G2 and the duration of weaning showed a negative trend, the mean FMD6 values were lower with more prolonged weaning. This shows that the more weaning is prolonged, the faster the return to resting diameter is (p = 0.0428) (figure 2b).



The correlation curve between the FMD1 of tobacco users and their number of packs/years showed a negative trend, the greater the number of P/A, the lower the FMD1 values were (p = 0.02) (figure 3a).



In addition, the correlation curve between FMD6 of tobacco users and the number of packs/years also showed a negative trend (p = 0.04) (figure 3b).



Finally, the correlation curve between the FMD and the mean arterial pressure of the subjects in the 3 different groups showed a negative trend because the FMD values were less significant at higher baseline blood pressure values (p = 0.0266).

Discussion

In this comparative study, the age difference between the 3 groups studied was not significant. Likewise, the analysis of anthropometric parameters,

notably BMI, did not show a significant difference between the 3 groups.

Age and BMI being important cardiovascular risk factors, the quasi-homogeneity of the anthropometric parameters in the 3 groups limited the impact of the latter on the results of the study. The study population was made up of soldiers with relatively comparable on regular physical and sporting activity. This allowed us to better discern the specific effect of smoking and withdrawal on vascular function.

In addition, individuals in the 3 groups did not have any cardiovascular or metabolic diseases or other medical conditions that could interfere with the results.

However, when comparing the mean blood pressure values, those of smokers were higher than those of controls, with a statistically significant difference ($p < 0.05$). This finding has been reported in the literature because smoking (nicotine) can transiently modify the regulation of blood pressure through a rapid effect on the autonomic nervous system. It also accelerates arterial aging, implicated in chronic hypertension, responsible for increased stiffness [10]. Thus, smokers could be at greater risk of developing cardiovascular diseases. If hypertension is the primary risk factor for stroke, tobacco has a more powerful impact on coronary heart disease, aortic aneurysm, or even obliterating arteriopathy of the lower limbs [6,12].

Furthermore, our results showed that non-smoking controls had better vascular compliance ($p < 0.0004$) and therefore better reactive vasodilation with average FMD1 higher than those of weaned people and smokers. This demonstrated better dilation of the vascular wall of controls after compression of the humeral artery following the release by the endothelium of vasodilator factors such as NO. It is known that tobacco influences arterial vasomotor function by causing an arterial spasm which suddenly reduces the diameter of the artery and blood flow [19,20]. This mechanism is triggered by hypoxia caused by CO which has a greater affinity with hemoglobin than oxygen, which causes cellular ischemia [21–23]. In our study, this could explain the fact that the mean arterial pressure was higher and that the baseline humeral arterial diameter was less dilated in smokers and former smokers compared to controls.

It has been reported in the literature that flow-mediated dilation was significantly lower in smokers (6.26%, 95%CI 5.58-6.94) compared to never smokers (8.68%, 95% CI 7.92-9.44), ($P < 0.0001$) [1].

Furthermore, the results showed that chronic tobacco consumption was correlated with more impaired vascular function. In fact, in the group of smokers, the higher the number of cigarette packs per year, the lower the value of Flow Mediated Dilation. Thus, the vascular response in smokers was less significant, less rapid, and less regular due to the alteration of the structure of the vessels. This was recently confirmed (2020) by a study which explained the probable role of oxidative stress in vascular dysfunction in tobacco users [22].

Also, it was noted that the humeral artery was better dilated depending on the duration of the subjects' withdrawal. Long-term withdrawal improved vessel wall compliance and vascular function in the arteries of weaned subjects. The beneficial effects of smoking cessation on the blood vessels have also been mentioned in the literature; it prevents early vascular accidents in primary prevention and reduces recurrences by 30 to 50% in secondary prevention [2,7,17,24].

Quitting smoking not only brings significant and immediate health benefits, but also reduces most of the associated risks within a few years of quitting [25–27]. Even people who drop out later in life benefit from it. For example, among smokers who quit at age 65, men gain on average two years of life and women three [15]. Quitting smoking is associated with a 36% reduction in risk of all-cause mortality among people with cardiovascular disease [13,28,29].

Conclusion

Our results demonstrated that tobacco use has detrimental effects on vascular function, which increases cardiovascular risk. On the other hand, they showed that stopping smoking improves vascular function and thus constitutes a means of preventing cardiovascular diseases. However, our study has limitations, notably the small study population but also, we were not able to assess the biodiversity of the NO. Additional scientific work is therefore necessary to better study the effect of smoking cessation

by evaluating the biodiversity of nitric oxide in smokers compared to quit subjects. But also follow a larger cohort over several years to better study the relationship between smoking cessation and the beneficial effect on vascular function.

Although Senegal has signed the WHO Framework Convention on Tobacco Control (FCTC) and has implemented legislative measures to reduce tobacco use, the promotion of smoking cessation remains an essential parameter in cardiovascular disease prevention strategies.

Conflicts of interest: The authors declare that they have no conflict of interest.

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