

Time Course Effects of Cigarette Smoking on Peak Expiratory Flow Rate among Youths within the Age of 18-25 years

^{1*}Azekhumen, G.N. and ^{1,2}Edeha, D.O.

¹Department of Physiology, School of Basic Medical Sciences, College of Medical Sciences, University of Benin, Benin City, Nigeria.

²Department of Physiology, Faculty of Basic Medical Sciences, College of Medical Sciences, Benson Idahosa University, Benin City, Nigeria.

***Corresponding author**

Email: gloria.azekhumen@uniben.edu

Abstract

Background/Objective: There is increased prevalence of cigarette smoking, especially among youths, and this has become a leading cause of premature morbidity and mortality worldwide, through the development and subsequent complications of respiratory diseases. The aim of this study was to understand how cigarette smoking affects the peak expiratory flow rate (PEFR) among young adults. **Materials and Methods:** Sixty students selected at random (18-25 years) volunteered for this study and were divided into Group A: control (non-smokers), Group B: young adults that have been smoking daily for 0-6 months (acute smokers) and Group C: young adults that have been smoking daily for 6 months above (chronic smokers). Anthropometric values were collected, blood pressure values and Body mass index were measured. Subjects blew into a standard spirometer three times, at resting and standing position, and the PEFR was recorded at the end of the third blow. **Results and conclusion:** There was a significant decrease in the PEFR of the smokers (both acute and chronic) regardless of the time they started smoking compared to the PEFR of non-smokers (control). The study demonstrates that cigarette smoking, regardless of the duration (acute or chronic), significantly reduces Peak Expiratory Flow Rate (PEFR) among young adults. This indicates that smoking adversely affects lung function, with potential implications for respiratory health even in individuals who have smoked for a short period. Chronic smoking exacerbates the decline in PEFR, highlighting the progressive harm caused by prolonged exposure to cigarette smoke.

Keywords: *Cigarettes, Smoking, Youth, Peak Expiratory Flow Rate, Spirometry*

Introduction

Tobacco smoking consists of drawing into the mouth, and usually the lungs, smoke from burning tobacco (1), which is usually found in cigarettes. Cigarette smoking carries major health risk, and it's a worldwide epidemic and the most prevalent cause of many diseases leading to increased morbidity and mortality across the globe (2). Nicotine induces pleasure and reduces stress and anxiety. Smokers use it to modulate levels of arousal and to control mood. Also, smoking improves concentration, reaction time, and performance of certain tasks. The basis of nicotine addiction is a combination of positive reinforcements, including enhancement of mood and avoidance of

withdrawal symptoms (3). Cigarettes are made from dried leaves of the tobacco plants. The chemical composition of the tobacco varies widely with different site due to the diversity of climatic condition (4). The peak expiratory flow rate is a measurement of the maximum airflow achieved when forcefully exhaling air after taking a deep breath (5), it is expressed in liters per minute, and tests assessing PEFR reveal alterations in the caliber of the airways (6). Spirometry is a physiological test that measures the ability to inhale and exhale air relative to time. Spirometry is a diagnostic test of several common respiratory disorders such as asthma and chronic obstructive pulmonary disease (COPD). The main

results of spirometry are forced vital capacity (FVC), forced expiratory volume exhaled in the first second (FEV1), and the FEV1/FVC ratio (7).

While the long-term effects of smoking on lung function are well-documented, limited research exists on the time-dependent impact of smoking, specifically the differences between acute and chronic smoking on Peak Expiratory Flow Rate (PEFR). Understanding how the duration of smoking influences lung function over time is essential for identifying early declines in respiratory health and for designing time-sensitive interventions. This study provides critical insights into the progression of lung function impairment from short-term (acute) to long-term (chronic) smoking.

Materials and Methods

Materials

Spirometer, 60 students (selected at random both male and female), weighing scale and stadiometer.

Methods

After approval from the state ministry of health Questionnaires were distributed at random among several students of the University of Benin and 60 students were carefully selected considering the age of 18-25. The students that participated in this project were given consent form which was signed by them. The exclusion criteria were those that drink alcohol, have a family history of lung diseases or asthma or had recurrent bronchitis since childhood or damage to their respiratory system. The anthropometric such as height, weight were recorded the height nearest to 0.1 and the weight nearest to 0.1 with minimal clothing were recorded using the height and weight scale. Their blood pressure, pulse rate and body mass index (BMI) which were derived from the weight and height of the student measured by the scale was recorded. The entire students were examined properly to avoid cases of an already existing heart or lungs disease. At resting and standing position the students selected blew into a standard spirometer three times and the highest PEFR value (in litres per millilitre)

were recorded in percentages of the predicted normal value of PEFR for young adult Nigerian males and females (18 – 30 years) resident in Benin City as determined by Ebomoyi and Iyawe below (8).

Males: 573.18 ± 15.73 L/Min

Females: 537.18 ± 53.05 L/Min

Study Population

The study was conducted on 60 healthy young adults who were selected at random (18-25 years), and classified into three (3) groups: Group A: 20 life-long non-smoker subjects with age, sex and BMI matched with cases (control). Group B: 20 smokers that started smoking daily within the duration 0 to 3 months (acute smokers). Group C: 20 smokers that smoked at least a pack of cigarettes daily for more than 3 months and above (chronic smoker).

Inclusion Criteria

Healthy male subjects in age group of 18-25 years, that are Non-Smokers and healthy male subjects in the age group of 18-25 years that are smokers.

Exclusion Criteria

Individuals with history of cardiovascular diseases, history of respiratory issues like asthma, cough, and individuals older than 25 years and younger than 18 years.

Ethical Consideration

Approval and Clearance for this study was obtained from the ethics and research committee of the University of Benin.

Method of Collection of Data:

Health Status, Duration of smoking and Number of Cigarettes smoked daily was obtained by comprehensive questionnaire.

Age and Sex was also included in the questionnaire.

Weight was recorded using a portable weighing machine.

Height was recorded using a wall stadiometer.

A sphygmomanometer was used to record the blood pressure.

A handheld spirometer was used in recording the Forced Vital Capacity.

Statistical Analysis

All results were expressed as

mean±standard error of mean (SEM) and analysed for statistical significance by Analysis of Variance (ANOVA) Post-Hoc Test. $p < 0.05$ was considered significant.

Results and Discussion

In this study, it is shown that the effect of smoking statistically increased amongst the smokers (chronic and acute). For the BMI, it was observed during the course of this research that age and sex affects PEFR result because of the period of time compared to the acute smokers. Most of the acute smokers were of younger age but the chronic smokers who having been smoking for longer had a higher chance of the PEFR affected. For the pulse rate, there was an increase amongst chronic smokers when compared to the acute smokers and non-smokers. Smokers are primarily addicted to nicotine (9) the level of nicotine in the blood is known to stimulate the nervous system to increase heart rate. Earlier findings indicate that individuals who smoke regularly experience elevated pulse rates and blood pressure in contrast to those who do not smoke, suggesting heightened sympathetic activity (10). Research has demonstrated that smoking cigarettes leads to an immediate surge in plasma catecholamines, cardiac norepinephrine overflow and subsequently, an increase in both blood pressure and heart rate, along with heightened sympathetic outflow (11). Furthermore, chronic smokers experience a long term effect of smoking such as a persistent activation of the sympathetic nervous system, leading to higher heart rate compared to acute smokers and non-smokers (11). Fig III showed that there was a significant decrease in peak expiratory flow rate in acute and chronic smokers compared with control respectively, this is in accordance with the study carried out by Gurung (12) which PEFR was lower among smokers compared to the non- smokers with a difference that was statistically significant.

However, there was a significant difference in the PEFR of acute smokers when compared to chronic smokers, as the chronic smokers where seen to have a significant reduction in their PEFR. This may have been caused by persistent inflammation of the airways caused by long term smoking and leading to thickening of the bronchial walls, increased mucus production and narrowing of the small airways (13). The effects of these processes is a limitation of airflow and a rapid drop in PEFR compared to acute smokers who may only experience transient airway irritation which may not cause significant changes to PEFR values (13). This assertion is supported by Tashkin et al., who also stated that chronic smoking leads to the destruction of the alveolar walls, resulting in a decreased elasticity and lung compliance. But acute smokers may not have developed the extensive alveolar wall damage which decreases lung compliance and as such may still be able to produce PEFR values that are greater than those by chronic smokers (14). Overall, the PEFR reduction in smokers may be due to various biological effects on lung tissues and airway cells, these effects encompass DNA damage, bronchoconstriction linked to elevated thromboxane levels, and the progression of conditions like emphysema and COPD (15).

Conclusion

In conclusion this study showed smokers have significant reduction in PEFR due to airway obstruction caused by smoking than the non-smokers. Smokers should be given enough education and information of what harm they are causing to themselves and also the constituent the constituent contained in what is being smoked attacks the functionality of their airways.

Table 1: Comparing the mean values of PEFR and some blood pressure indices on smokers and non-smokers.

Parameters	Non-smokers	Acute smokers	Chronic smokers
SBP (mmHg)	121.3± 1.80	121.8±3.10	120.6±2.92
DBP (mmHg)	74.90±2.10	79.60±1.62	74.80±2.07
Pulse rate (bpm)	73.75±1.42	71.30±1.70	80.60±2.13
PEFR (%)	60.20±1.33	53.10±1.18	38.00±3.06
BMI (kg/m ²)	21.21±0.57	23.19±0.44	23.09±0.63

P < 0.05 indicates significant difference with respect to control.

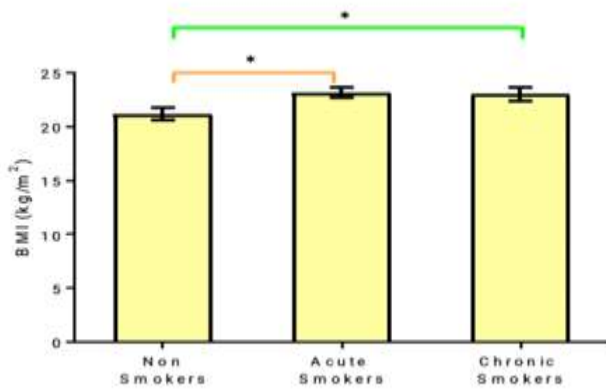


Fig I: shows body mass index in smoking and non-smoking individuals

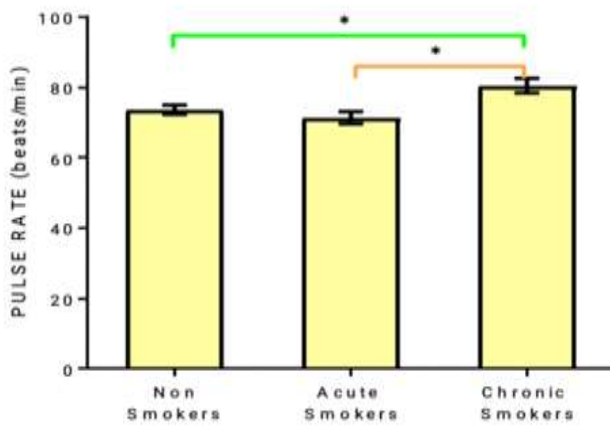


Fig II: shows pulse rate in smoking and non-smoking individuals

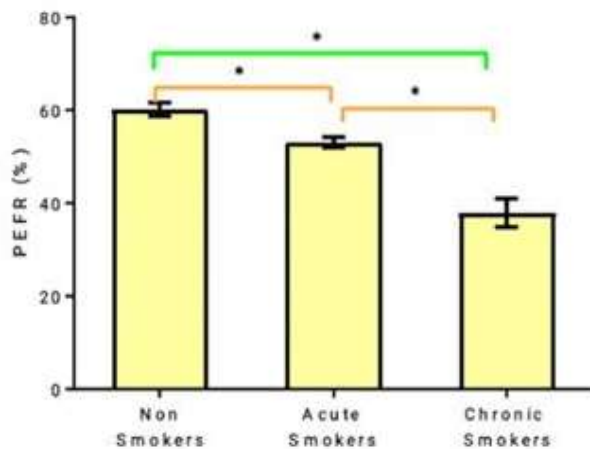


Fig III: shows peak expiratory flow rate in smoking and non-smoking individuals

Table 1: Showing the mean values of the weight of adult Wistar rats.

Parameters	Group 1	Group 2	Group 3	Group 4	Group 5
Initial body weight (g)	131.5± 3.47	149.0±0.82	129.5±1.43	148.5±1.02	147.5±1.02
Final body weight (g)	158.0±0.41	175.5±1.84	145.5±0.61	165.0±2.86	169.0±2.04
Liver weight (g)	6.300±0.10	7.400±0.20 ^x	5.950±0.050	7.250±0.55 ^x	7.100±0.40 ^x
Kidney weight (g)	0.80±0.00	1.050±0.15	0.90±0.10	0.95±0.05	0.90±0.10

P < 0.05 indicates significant difference

Table 2: Liver Function Test (Enzymes) in Both Treated and Untreated Rats

Parameters	Group 1	Group 2	Group 3	Group 4	Group 5
AST	28.00±1.62	32.00±0.82	28.00±1.23	32.50±0.25 ^x	34.50±0.25 ^x
ALT	21.50±0.21	18.50±0.61 ^x	23.00±2.85	22.50±2.65	22.50±2.25
ALP	32.50±0.61	28.50±0.20 ^x	30.00±2.45	24.00±1.23 ^x	27.00±1.23 ^x
Total bilirubin (mg/dl)	0.250±0.02	0.3000±0.04	0.2500±0.02	0.2500±0.02	0.3000±0.04
Conjugated bilirubin	0.150±0.02	0.150±0.02	0.150±0.01	0.10±0.00	0.150±0.03
Total protein (mg/dl)	4.950±0.14	4.050±0.06 ^x	4.450±0.14 ^x	4.850±0.10	4.850±0.06
Albumin (mg/dl)	2.900±0.04	2.550±0.06 ^x	2.850±0.02	3.000±0.04	2.850±0.06
Globulin (mg/dl)	2.050±0.18	1.900±0.12	1.600±0.12 ^x	1.850±0.06	1.900±0.08

^xindicates significance; *P* < 0.05 indicates significant difference

Table 3: Renal Test (Electrolyte and Urea/ Creatinine) in Both Treated and Untreated

Parameters	Group 1	Group 2	Group 3	Group 4	Group 5
Urea (mg/dl)	27.00±1.22	19.50±0.61 ^x	25.50±0.61	31.50±0.20 ^x	31.00±0.41 ^x
Sodium ion (Mmol/L)	139.5±0.61	143.0±0.41	142.0±2.04	141.0±0.41	139.5±0.21
Potassium ion (Mmol/L)	4.300±0.23	4.700±0.16	4.650±0.23	4.950±0.10 ^x	4.900±0.04 ^x
Bicarbonate ion (Mmol/L)	18.50±0.61	19.50±0.20	17.50±0.20	18.50±0.20	19.50±0.20
Chloride ion (Mmol/L)	103.0±0.40	103.0±0.40	103.5±1.02	102.5±0.20	102.0±0.81
Creatinine (mg/dl)	0.750±0.02	0.750±0.02	0.750±0.02	0.800±0.41	0.95±0.02 ^x

P < 0.05 (One-way ANOVA)

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