ISSN (Online): 2393-915X; (Print): 2454-7379 IJCMR

ORIGINAL RESEARCH

A Study of absolute Eosinophil Count and Peak Expiratory Flow Rate in Smokers and Non Smokers in Nandyal, AP

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ABSTRACT

Introduction: Smoking largely affects the PEFR and absolute eosinophil count.Thestudy was aimed to assess the effects of smoking on PEFR and absolute eosinophil count (AEC).

Material and Methods: Total of 82 young male adults between the ages of 16 to 25 years were selected for the study. After collecting the required data, peak expiratory flow rate and absolute eosinophil count were determined.Student t test and Pearson's correlation were used.

Results: The mean eosinophil count and PEFR was significantly higher in smokers compared to non-smokers ($p < 0.05$). The PEFR was higher in subjects who were performing regular exercise, while absolute eosinophil count showed a very slight negative correlation ($r = - 0.04$, $p > 0.05$) with exercise rate. The correlation between PEFR and absolute eosinophil count was not statistically significant.

Conclusion: The absolute eosinophil count and PEFR were elevated in both smokers and non-smokers. The raise of PEFR in smokers might contribute to the effect of regular exercise in these subjects.

Keywords: Cigarette smoking, Peak expiratory flow rate, absolute eosinophil count

How to cite this article: R. D. Gaikwad, Z. Naveen Kumar, M. Lavanya. A study of absolute eosinophil count and peak expiratory flow rate in smokers and non smokers in Nandyal, AP. International Journal of Contemporary Medical Research 2015;2(5):1318-1321.

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Source of Support: Nil

Conflict of Interest: None

INTRODUCTION

WHO reports that about 5 million people die each year across the globe due to cigarette smoking.[1] In United States, 440000 premature deaths are attributed to cigarette smoking.[2] The death toll is progressively increasing and unless current smoking trends are reversed, this figure is expected to rise to 10 million deaths per year by the 2020 or early 2030, with 70% of those deaths occurring in the developing countries.[3] Cigarette is the leading known risk factor for the development of chronic obstructive pulmonary disease and 50% of smokers develop clinically significant airflow obstruction.[4] The lung functions of cigarette smokers showed accelerated decline when com-pared with the non-smokers.[5] Earlier detection of air-flow obstruction and smoking cessation may result in significant health gain.[6] Elevated eosinophil blood count has generally been associated with indications of an allergic reaction.Previous study has shown that blood eosinophils were found to be substantially elevated in smokers.[7] Peak expiratory flow rate is a good parameter for detecting patients with COPD and tests of PEFR reflect changes in airways caliber. Airflow obstruction in cigarette smokers is often diagnosed relatively late. Earlier detection of air-flow obstruction and smoking cessation may result in significant health gain.[8] If a cigarette smoker stops smoking, peak expiratory flow rate improves with the passage of time. So the objective of this study was to investigate the effect of smoking on PEFR and circulating eosinophils in males.

Materials and Methods

82 young male subjects between16 to 25 years ofage were selected for the study. Subjects were the students from surrounding areas of Nandyal, Kurnool District, Andhra Pradesh. Institutional ethical committee approval was taken before the start of the research and all subjects who participated in the study were given a consent form to sign before the experiment was carried. The recordings were collected and completed in five months duration.

Criteria for the study group consist of (1) subjects within the age of 16 to 25 years, (2) for smokers, a minimum of two years of smoking and a maximum history of seven years of smoking expected, and (3) the control group of non-smokers were of individuals who had never smoked tobacco even once in their lifetime.Subjects with history and signs ofatopy, asthma or other diseases, were excluded from this study. Volunteers were subjected to a detailed clinical examination with general questioning on information relevant to the experiment such as age, race, sex, with a history of non-smoking or smoking. The remaining questioning included history of respiratory disorders, history of exercise, with details such as frequency, type and duration if any were done. History of drug abuse were also noted. For smokers,

Physiological Variables		Smoking Volunteers	Non-Smoking Volunteers	P value
Mean age (years)±SD		20.7±2.8	21.0±1.4	NS
Sex (%)	Male	54.9%	45.1%	NS
Mean BMI (kg/m^2)		23.4±4.1	22.11±3.7	> 0.05
Exercise (no. of times/month)		0.33±0.40	0.24±0.30	> 0.05
Heart Rate (beats/min)		81.6±9.3	79.4±9.8	> 0.05
Blood Pressure (mmHg)	SBP	119.8±10.8	116.1±13.8	> 0.05
	DBP	75.1±8.6	70.7±11.4	< 0.05
*NS: Not significant.				

Table-1: Physiological variables in smokers and non-smokers

Subjects	Mean PEFR ±SD (L/min)	Mean Eosinophil Count ±SD (cells/mm^3)
Smokers	514.7±89.9	249.7±179.2
Non-smokers	474.9±103.9	139.5±110.5
Smokers and Non-Smokers	493.3±99.1	190.5±155.5

Table-2: Showing mean PEFR and Absolute Eosinophil Count

Subjects		Mean Exercise Rate±SD (no. of times per month)	Mean PEFR (l/min)	Mean Eosinophil Count (cells/mm^3)
Male	Smoker	0.43±0.42	550.9	236.3
	Non-smoker	0.30±0.35	547.8	113.0

Table-3: Mean exercise rate and the association with mean PEFR and eosinophil count

additional questioning was done regarding the number of years of smoking and the quantity of cigarettes consumed in a day.

The blood was diluted 10 times in the white blood cell (WBC) pipette, using eosinophil solution, which lyses the red blood cells and leucocytes other than eosinophils so that it can be counted easily in Neubauer counting chamber.[9]

A mini Wright peak flow meter was used. The meter was set to zero and subjects blew into the device three times, while standing straight, with the device held horizontal to the mouth, without wearing a nose clip. After proper rest, subjects were asked to take a deep breath and exhale as forcefully as possible in one single blow into the instrument. Subjects were observed carefully in order to assure the correct technique was done during the blowing. After each blow, the meter was always reset to zero before the next reading was taken. The highest of the three readings obtained was taken as the final PEFR for each subject.[10]

STATISTICAL ANALYSIS

Data was analyzed using SPSS; version 14 for windows (SPSS Inc.,2005). Comparisons between groups were performed with Student's t-test. Pearson correlation analysis was conducted to assess associations between variables. Values of P<0.05 were considered as statistically significant.

RESULTS

The present study evaluated the effect of smoking on absolute eosinophil count and PEFR, and also to establish a relationship between absolute eosinophil count and PEFR inmale smokers and non- smokers.

In table 1, it was observed that smokers and non smokers showed comparable values with respect to the physiological data. Although the overall mean value for smokers were noticeably slightly higher compared to that of non-smokers, the mean value for BMI, exercise frequency, heart rate and systolic blood pressure, were not statistically significant ($p > 0.05$). While the mean DBP on the other hand showed a significant difference ($p < 0.05$).

From table 2, it was noted that the mean PEFRin smokers and non-smokers were statistically significant ($p < 0.05$). The smoker subjects showed an elevated PEFR valuewhen compared to the non-smoking subjects. Even there was a high mean eosinophil count in smokers compared to non-smokers, which was statistically significant ($p<0.05$).

With reference to table 3, the mean PEFR observed highest in smoker volunteers. There was also a highest value of mean exercise rate observed in the same group of male smoker volunteers. The mean absolute eosinophil count was observed lowest in non-smoking male volunteers that showed a relatively frequent rate of exercise as well.

As shown in Figure 1, there was a significant linear relationship between the PEFR and the exercise frequency ($p < 0.05$) that gave a moderately positive correlation coefficient ($r = 0.36$) between the two variables.

With regard to figure 2, there was a very slight negative correlation coefficient ($r = - 0.04$) between the rate of exercise in a month and absolute eosinophil count**.**This results were not significant.

There wasno apparent association between the PEFR and the number of years smoking. Although,there was a moderately positive correlation coefficient ($r = 0.40$) seen in the absolute eosinophil count with number of years smoking. Absolute eosinophil count increased significantly ($p < 0.05$) with increased years of smoking.

In figure 3, points plotted appeared to be randomly distributed with almost no signfiacntrelationship between absolute

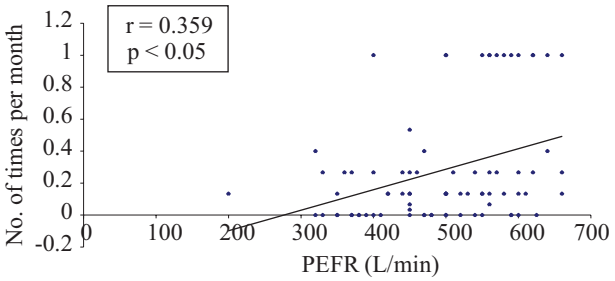

Figure-1: Graph showing the rate of exercise in a month and its association with PEFR

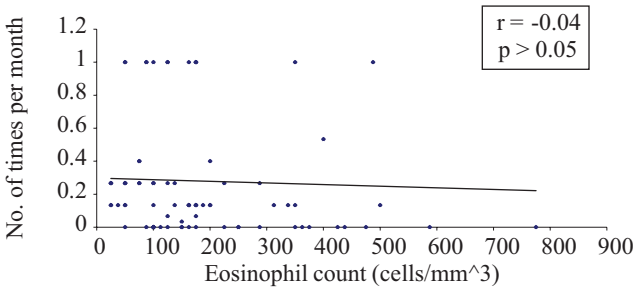

Figure-2: Graph showing the rate of exercise in a month and its association with absolute eosinophil count.

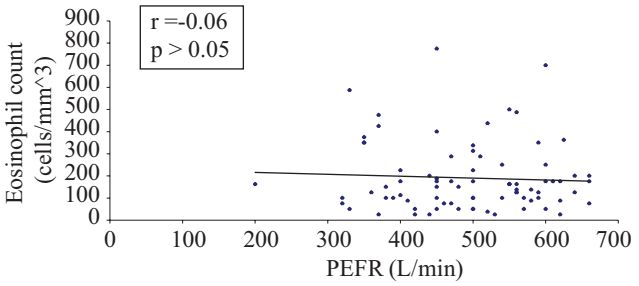

Figure-3: Graph shows absolute eosinophil count against PEFR in smoking and non-smoking volunteers

eosinophil count and the PEFR. But there was a slight negative correlation coefficient (r = - 0.06) observed, indicating there were a small number of individuals that showed low PEFR at increased absolute eosinophil count.

DISCUSSION

Smoking causes decrease in lung function,[11-15] butonlya minority of smokers develops severe respiratoryimpairment.[16] Reasons for this difference in susceptibility are not fully understood. Blood eosinophilcount is elevated in nonatopic smokers compared to nonsmokers.[17,18] A simple test to measure how quickly air can be forced out from the lungs is peak expiratory flow rate. Narrowing of the airways reduces the ability to move air in and out of the lungs, which lowers the PEFR.[19] The present study evaluated the effect of smoking on absolute eosinophil count and PEFR, the relationship between absolute eosinophil count and PEFR in male smokers and non- smokers.

Our study noted that the smoker subjects showed a high mean PEFR compared to non-smoking subjects. This was not consistent with the expected finding of lower PEFR in smokers compared to that of non-smokers. Smokers have reduced lung size, both the airways and the actual capacity.[11-15] The total capacity of smokers' lungs is reduced, therefore having a lower PEFR than the non-smokers. This higher PEFR seen in smokers could be speculated due to the increased rate of exercise seen in male smoking volunteers. The mean PEFR was observed highest in male smoker volunteers. The exercise frequency was relatively consistent in the past 6 months in the smoker volunteers.

Previous research has demonstrated that inspiratory muscle training improves performance in highly trained rowers (Cycling Performance Tips, 2004).[23] Moreover, increasing exercise frequency in the long term appeared to have improved PEFR in smokers and non-smokers.

There was a high mean eosinophil count in smokers compared to non-smokers. This was consistent with previous studies done by Ulrik, C.S (1998)[20] O'Connor (2004)[21] and Sunyer(2004)[22], which showed that blood eosinophils were found to be substantially elevated in smokers and high blood eosinophil count was related to lung defense.

There was no apparent association between the PEFR and the number of years smoking so that the rate of exercise had altered the PEFR in several male smoking individuals. Exercise training increased PEFR in smoking and non-smoking individuals by increasing the efficiency of the lung capacity. Absolute eosinophil count increased significantly ($p < 0.05$) with increased years of smoking. There was a slight negative correlation coefficient (r = - 0.06) observed between eosinophil count and PEFR, indicating there were a small number of individuals that showed low PEFR at increased absolute eosinophil count. This relationship was consistent with isolated effects of smoking, whereby smoking causes elevated absolute eosinophil count (Ulrik, 1998, Sunyer, 2004, and O'Connor, 2004)[20-22] and reduced PEFR.[24] The association between the two variables was not especially distinctive due to the exercise factor that contributed to the increased PEFR in many smokers, the fact that these volunteers were a young sample of subjects (16-25 years) and the study was on short-term smokers (2-7 years).

CONCLUSION

The mean absolute eosinophil count and PEFR were significantly higher in male smokers compared to non-smokers. The PEFR was elevated with increase in exerciserate, whereas absolute eosinophil count showed aweak negative correlation with exercise rate.We speculate that the exercise rate had altered the PEFR in smoking individuals. The relationship between PEFR and Absoulte eosinophil count in this study were weak and the study population was small. Our results could be due to chance but, therefore, be overlooked. To establish the proposed relationships, these should be reinvestigated in a large prospective study. Besides, the nicotine and tar content plays a significant role on blood pressure, heart rate and PEFR should be considered. The brand of cig-

arette used by subjects should be taken into account.

ACKNOWLEDGEMENT

We sincerely thank all subjects who gave consent and participated in this study.

REFERENCE

1. World Health Organization. MPOWER: A Policy Package to Reverse the Tobacco Epidemic. Geneva: WHO; 2011.
2. Fellows JL. Annual smoking attributable mortality, years of potential life lost and economic costs-United States, 2002; 51: 300-303.
3. WHO-A global status report. Anonymous. Tobacco or health: World Health Organization Geneva. 1997; 115-117.
4. Lundback B, Lindberg A, Lindstrom M, Ronmark E, Jonsson AC, Jonsson E et al. Not 15 but 50% of smokers develop COPD? Report from the Obstructive Lung Disease in Northern Sweden Studies.Obstructive Lung Disease in Northern Sweden Studies. Respir Med. 2003;97:115-22.
5. Peter KJ. Chronic obstructive pulmonary disease. Pa-thology of COPD. In: Respiratory Medicine. Gibson JG Saunders Elsevier Science Ltd. 3rd ed, 2003; 2: 1141.
6. Geijer RM, Sachs AP, Hoes AW, Salome PL, Lammers JW, Verheij TJ. Prevalence of undetected persistent airflow obstruction in male smokers 40-65 yearsold.Fam-Pract. 2005;22: 485-489.
7. Taylor RG, Gross E, Joyce H, Holland F, Pride NB. Smoking, allergy, and the differential white blood cell count. Thorax 1985;40:9-16
8. Hussain G., Zafar S., Ch A.A, Ch Z.A., Ahamad M.Z. Comparative Study of Peak Expiratory Flow Rate in Cigarette Smokers and Non-Smokers of Lahore District. Annals 2007;13:255-259.
9. Dacie, J.V. and Lewis, S.M. Differential Leucocyte Count. In Practical Haematology, 7th edition: London: Churchill Livingstone. 1991;70-71.
10. Halgate ST and Frew A. Respiratory Disease. In: Clinical Medicine 5th Ed. PK, MC. Ed. Edinburgh W.B Saunders Co. 2002;849.
11. Andersen AE, Hernandes JA, Holmes WL, Fotaker AG. Pulmonary emphysema: prevalence, severity, and anatomicalpatterns in macrosections, with respect to smoking habits. Arch Environ Health 1966;12:569–577.
12. Petty TL, Ryan SF, Mitchell RS. Cigarette smoking and the lungs: relation to postmortem evidence of emphysema,chronic bronchitis and black lung pigmentation. Arch Environ Health 1967;14:172–177.
13. Auerbach O, Hammond EC, Garfinkel L, Benante C. Relation of smoking and age to emphysema: whole lungsection study. N Engl J Med 1972;286:853–857.
14. Niewoehner DE, Kleinerman J, Rice DB. Pathologicchanges in the peripheral airways of young cigarettesmokers. N Engl J Med 1974;291:755–758.
15. Auerbach O, Garfinkel L, Hammond EC. Relation ofsmoking and age to findings in the lung parenchyma: amicroscopic study. Chest 1974;65:29–35.
16. Janoff A, Brook S, Pryor WA, Bengali ZH(eds). NHLBIWorkshop Summary. Effects of tobacco smoke componentson cellular and biochemical processes in thelungs. Am Rev Respir Dis 1987;136:1058–1064.
17. Burrows B, Hasan FM, Barbee RA, HalonenM,Lebowitz MD. Epidemiologic observations on eosinophilsand its relation to respiratory disorders.AmRevRespir Dis 1980;122:709–719.
18. Kauffmann F, Neukirch F, Korobaeff M, Marne MJ,-Claude JR, Lellouch J. Eosinophils, smoking and lungfunction. An epidemiologic survey among 912 working men. Am Rev Respir Dis 1986;134:1172–1175.
19. Diner, Brenner, and Camargo. Inaccuracy of "Personal Best" Peak expiratory flow rate reported by inner city patients with acute asthma.AcadEmerg Med. 2000;7:469.
20. Ulrik, CS. Eosinophil and Pulmonary Function: An Epidemiologic Study of Asolescents and Young Adults. Ann. Allergy Asthma Immunol., 1998;80:487-493.
21. O'Connor GT., Sparrow, D. and Weiss, S.T. The Role of Allergy and Nonspecific Airway Hyperresponsiveness in the Pathogenesis of Chronic Obstructive Pulmonary Disease.Am Rev Respir Dis. 1989;140:225-52
22. Sunyer, J., Springer, G., Jamieson, B., Conover, C., Detels, R., Rinaldo, C., Margolick, J. and Munoz, A. Effects of Asthma on Cell Components in Peripheral Blood Among Smokers and Non-smokers. JIMA.2004;112:156-159.
23. Cysling Performance Tips (2004). Breathing for Highly Trained Athletes. http://www.cptips.com/respmus.htm Date accessed: 25 June 2013.
24. Crompton, G. K., Haslett, C., Chilvers, E. R. (1999). Diseases of the respiratory system. In C. Haslett, E.R. Chilvers, J.A.A. Hunter & N.A. Boon (eds.) Davidson's Principles and Practice of Medicine (18th ed.) Edinburgh: Churchill Livingstone.