GLOBAL JOURNAL OF BIO-SCIENCE AND BIOTECHNOLOGY

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EFFECT OF CIGARETTE SMOKING ON PEAK EXPIRATORY FLOW RATE

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ABSTRACT
Tobacco smoking in India has been increasing alarmingly. Smoking is a known risk factor for chronic obstructive pulmonary disease (COPD), cardiovascular diseases and certain cancers, especially, the lung cancer. Carbon monoxide from the smoke and nicotine both put a strain on the heart by making it work faster. They also increase your risk of blood clots. Other chemicals in cigarette smoke damage the lining of your coronary arteries, leading to furring of the arteries. To examine whether PEFR differs between cigarette smokers compared to non-smokers and also to estimate the intensity of cigarette smoking on PEFR. PEFR was recorded in cigarette smokers (n=53) as well as in non-smokers (n=71) using Wright's mini Peak Flow Meter. PEFR is decreased in cigarette smokers compared to non-smokers and the magnitude of decline was higher in elderly individuals. The intensity of cigarette smoking (pack-years) emerged as the main variable to influence airway obstruction in smokers that caused greater reduction in PEFR.

KEY WORDS: Smoking, Peak expiratory flow rate.

INTRODUCTION

India is the second largest consumer of tobacco products and third largest producer of tobacco in the world. The adult population of smokers in India is about 84.8 million and is almost equal to the population of Vietnam or Germany. The death toll from tobacco use is projected to rise from 5.4 million in 2004 to 8.3 million in 2030[1].Tobacco smoking is a major risk factor for cardiovascular disease, chronic obstructive pulmonary disease and some cancers and the morbidity and mortality with tobacco use is entirely preventable[2]. The prevalence of tobacco smoking in Indian males is much higher (24%) than females (3%) according to Global Adult Tobacco Survey (GATS) India 2009-10 Report [1]. It is interesting to note that though the prevalence of cigarette smoking in rural areas is lower than in urban areas, the number of cigarette smokers in rural areas is higher than in urban areas. Further, the number of smokers of any kind of smoking tobacco product in rural areas is higher than in urban areas[2]. Nicotine is the addictive drug in tobacco smoke that causes smokers to continue to smoke. Addicted smokers need enough nicotine over a day to 'feel normal'– to satisfy cravings or control their mood. How much nicotine a smoker needs determines how much smoke they are likely to inhale, no matter what type of cigarette they smoke. Along with nicotine, smokers inhale about 7,000 other chemicals in cigarette smoke. Many of these chemicals come from burning tobacco leaf. Some of these compounds are chemically active and trigger profound and damaging changes in the body. Tobacco smoke contains over 60 known cancer-causing chemicals. Smoking harms nearly every organ in the body, causing many diseases and reducing health in general. Further, a quarter of smokers develop chronic obstructive pulmonary disease[3] and is the fourth commonest cause of death worldwide[4]. COPD is characterized by airflow limitation that is not fully reversible[5,6]. Air flow limitation may be due to inflammation [5-7] or due to increase in the thickness of the wall[9]. PEFR is a useful parameter to monitor airway obstruction, assess its severity and variation and evaluate the effects of treatment[10]. Earlier studies have reported that the PEFR is an effort dependent parameter emerging from large airways[11-12] and it does not detect small airways obstruction[13]. Further, there are inconsistent findings which show that smoking affects medium and large airways [14-15]. Others have reported that smoking affects both small and large airways [16-17]. Several studies have reported that PEFR was significantly lower in smokers than in non-smokers [18-22] and some studies found maximum reduction in PEFR was in bidi smokers than cigarette smokers [21]. The primary objective of the study was to investigate whether PEFR differs between cigarette smokers compared to non-smokers and the second objective was to estimate the intensity of cigarette smoking on PEFR.

MATERIALS & METHODS

The study was conducted at Geetanjali Medical College and hospital, Udaipur, Rajasthan during a period of May 2014 to September 2014. Subjects were recruited from villages of Udaipur District in Rajasthan. History of smoking, occupation and nutritional status was obtained from all the smokers. This entire study was conducted in male subjects. About 53 smokers were taken for the study with age range about 20-40 years and 71 healthy non-smokers of same age group served as controls. Cigarette smoking group was further subdivided into following four categories based on intensity of cigarette smoking which is expressed as pack years; 1-50 pack-years (I group, n=16), 51-100 pack-years (II group, n=15), 101-150 pack-years

(III group, n=13) and 151-230 pack-years (n=9). This sub-grouping was necessitated to understand the effect of the intensity of cigarette smoking on PEFR and the average number of packs of cigarettes smoked per day multiplied by the total number of years of smoking which is usually expressed as pack-years.

Inclusion criteria

- Apparently healthy smokers and non-smokers from the same village.

Exclusion criteria

- Women.
- Patients with known hypertension, asthma, COPD and disorders that affect air flow.
- Individuals having mechanical obstruction preventing the performance of the test were also excluded.
- Patients having oral lesions or any other abnormalities that prevent the performance of the test.

Subjects were invited to the research lab at an appointed time. The entire procedures involved in the study were explained. After explaining the purpose of the study and familiarizing to all the research techniques, a written informed consent were obtained from the participants. Subject's body weight to the nearest kilograms was measured using the weighing machine. Height was measured to the nearest 1cm with the subject standing by side of the wall mounted stadiometer in bare foot with chin raised up. PEFR was measured with the Wright's mini Peak Flow Meter[23].Three attempts were made from each participant with a gap of 2 minutes between each effort and the mean value obtained was taken as the data for the subject. For uniformity, the data was collected by the same investigator throughout the study.

RESULTS

Results show a significant variation in the age and PEFR ($p<0.001$) among non-smokers and cigarette smokers [Table-1]. P-Value less than 0.05 were considered as significant.

TABLE 1: Comparison of Age and PEFR among non smokers and smokers

	Non Smoker (n=71) [mean ± sd]	Smoker (n=53) [mean ± sd]	P value Significance
Age(yrs)	26.42± 5.61	27.85±5.73	0.001
PEFR (lpm) (liters per minute)	513.43±87.58	409.79±90.31	0.001

Pearson correlation analysis shows that there was a negative and strong correlation between cigarette–years of smoking and PEFR ($r = -.830$, $p<0.01$). [Table 2].

TABLE 2: Correlation between cigarette yrs and PEFR

	Pearson corelation	Significance
Ciggarate yrs Vs PEFR	-0.830	0.001

We have shown the comparison of means among four groups of cigarette smokers (based on pack-years) with respect to age, cigarette–years and PEFR. The ANOVA shows that the intensity of cigarette smoking was high in elder age group (189.2 cigarette years) than in the younger age group (30.61 cigarette years) and this difference was statistically significant ($p<0.01$). This was resulted in greater fall in the PEFR ($p<0.001$) with increasing age and the number of cigarette usage [Table 3].

TABLE 3: Comparison of four subgroups of cigarette smokers based on cigarette (packe) years

	Group 1(n=16) 1-50 cigarette/yrs	Group 1(n=15) 51-100 cigarette/yrs	Group 1(n=13) 101-150 cigarette/yrs	Group 1(n=9) 151-230 cigarette/yrs	P- value
Age(yrs)	22.82± 3.27	26.66 ± 3.59	28.90 ± 4.10	36.22± 2.81	0.001
Cigarette(in yrs)	30.61 ± 10.47	73.80 ± 16.51	127.27± 9.66	189.22± 27.50	0.001
PEFR(lpm)	494.70 ±79.22	443.33 ± 45.14	350.90 ± 32.38	300 ± 46.90	0.001

DISCUSSION

In the present study, we found that PEFR was decreased significantly in both cigarette and cigar smokers compared to non-smokers and our findings are in agreement with the findings of others [18-22]. One possible reason for the decrease in PEFR could be inflammation which is common and constant pathological finding in cigarette smokers [7]. Earlier studies have reported that airway flow limitation occurs due to bronchial constriction caused by mediators of inflammation[8]. Inflammation either directly or by increasing smooth muscle tone, indirectly, may cause airway fibrosis[5]. All these changes promote wall thickness leading to airway narrowing and flow limitation [5, 9]. In addition, inflammation causes destruction of the alveolar walls attached to the airway contributing further to airflow limitation by deforming and narrowing the airway lumen[5]. Pearson correlation analysis [Table-2-4] shows that there exist a strong negative correlation between intensity of cigarette/cigar smoking and PEFR *i.e.* the greater the intensity of cigarette/cigar smoking, lesser the PEFR value. However, negative correlation was highest in cigarette smoking compared to cigar smoking and this difference may be attributed to small sample size for cigar smoking group. In spite of difference in the magnitude of decline in PEFR, the negative correlation suggests that both types of tobacco smoking adversely affect the lung function.

Another important finding in this study was that reduction in PEFR was proportional to the increase number of cigarette–years and this finding particularly is evident in

older age group compared to younger age group [Table-3]. This suggests that age would have further aggravated the extent of decline in lung function besides severity of cigarette smoking. Invariably, age has been affecting the PEFR aside from cigarette/ cigar smoking and PEFR was decreased both in cigarette and cigar smokers with advancing age. Earlier, we reported that both obstruction to the air flow and senile degenerative changes decrease the PEFR in agricultural workers [23]. Though it was not our objective to study the effect of cigarette and cigar smoking on PEFR in agricultural workers, the participants in the present study were from that background. This fact might have influenced our present results because earlier one study reported that older people who work on smaller agricultural farms have the higher risk of distal airway obstruction[30]. Agricultural dusts, fumes, and gases can increase the airflow resistance[31] and organophosphate insecticides[32] may trigger bronchospasm in agricultural workers. Airway narrowing caused by inflammation, edema, or smooth-muscle hyper reactivity results in acute and reversible decreases in airflow[33]. Further, previous studies have shown that the senile degenerative changes in the lungs such as loss of respiratory muscle strength and stiffness of joint movements are probably the most important factors reducing lung function with advancing age in agricultural workers. These factors limit ventilatory functions and thus cause a reduction in the total lung capacity and PEFR[34,35]. The loss of elastic recoiling which limits the ventilatory function with advancing age may also be the reason for declining of lung function[34]. As age advances there is an oxidative damage that results in increased production of elastases which degrade elastic recoiling of the lung [36]. With age, the thorax is compressed and calcification of costal cartilage increases the severe kyphosis leading to loss of chest wall compliance and reduced diaphragmatic efficiency[37]. Overall, our findings are consistent with others that the intensity of cigarette smoking (pack-years) emerged as the main variable to influence airway obstruction in smokers [30].

LIMITATIONS & CONCLUSIONS

Despite of certain limitations like small sample size, self reporting, and the study population only from one rural village, our present findings suggest that both cigarette and cigar smoking have their deleterious effects on lung function causing reduction in PEFR with advancing age and intensity of smoking. This study adds pertinent information about severity of cigarette smoking in general and cigar smoking in particular from the study area. But large scale studies are required from all other regions of the Rajasthan, India, for the extrapolation of present results to entire population of the state to make appropriate policy decisions.

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