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# BASIC CARDIORESPIRATORY STATUS OF ADULT MALE SMOKERS AND NON SMOKERS OF VILLAGE PATASHIMUL, JHARGRAM DISTRICT OF WEST BENGAL, INDIA: A COMPARATIVE STUDY

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## ABSTRACT

Smoking is a deadly addiction. Smoking affects our lungs and cardiovascular system adversely. Non smokers are known to have better cardiorespiratory status. Nicotine in cigarette causes cardiac and pulmonary ailments. Even Chronic Obstructive Pulmonary Disease (COPD) is also known to cause due to smoking. Peak expiratory flow rate ( PEFR) is the maximum flow rate generated during a forceful exhalation , starting from full lung inflation. PEFR primarily reflects large airway flow and depends on the voluntary effort and muscular strength of the patient. Peak Expiratory Flow Rate (PEFR) helps us to understand the condition of the lungs. We have conducted the study on adult male population of village Patashimul, Jhargrm District of West Bengal, India, West Bengal, india. Our studies reveal better cardiorespiratory status of non smokers compared to smokers.

**KEYWORDS:** Addiction, smokers, non smokers, Peak Expiratory Flow Rate, Chronic Obstructive Pulmonary Disease.

## INTRODUCTION

Smoking is recognized as one of the major causes of death in developed and developing countries. Fortunately, smoking is preventable. Studies reveal that smoking has mild to severe adverse effects on the respiratory system. Significant changes in respiratory function tests of smokers have been observed compared to that of non smokers.[1] Lungs are the prime respiratory organs. The respiratory passage is from the nose to the inside of the lungs where there are alveoli. Exchange of gasses occurs at those alveoli which are well vuscularised with capillaries. The walls of these alveoli and the capillaries are very thin.[3] The gasses which are soluble get absorbed into the blood through the walls of the alveoli. During smoking, certain soluble gaseous materials get absorbed in the blood. The particulate matters present in cigarette get deposited on the walls of the alveoli.[2] Gradually with continued and long term exposure, the toxins from inhaled smoke of cigarette damages and breaks the walls of the alveoli. This leads to larger, damaged, fused, inefficient alveoli which actually lose their ability to exchange gasses. The air sacs thus lose their bounce, elasticity and thus the normal physiological process of absorbing oxygen and expelling carbon dioxide gets difficult.[3,4] Those toxicants and poisonous gasses may stay trapped partially in the lungs and stimulate signals for onset of pulmonary pathological conditions like COPD.[4]

Smoking Cigarette is a very common addiction. Specially youth and teen agers get addicted to smoking easily and in most of the cases this addiction continues life long. In majority of cases, if not all various pathological complications arise whose underlying cause is smoking addiction. Some of those conditions turn to be even life threatening. More than 80% of smokers wish to quit smoking at some point of life.[5,6] Surprisingly only 33% people actually attempt to quit smoking. Only very few people among who want to quit smoking becomes successful. In 70-80% people who attempt to quit smoking, the addiction relapses.[5-7] Unfortunately smoking addiction is increasing around the world. Body weight is known to be correlated to smoking. Body Mass Index (BMI) and Body Surface area (BSA) are common anthroplogical parameters for ascertaining body weight status. Increased BMI is found to be associated with cardiovascular risk.[8] There are controversial reports regarding weight loss of smokers. Studies reveal that light smokers tend to have lower BMI and lower body weight whereas heavy smokers are found to have higer

BMI.[9] Low body weight in smokers is often said to be due to increased metabolism by nicotine. Whereas high BMI in heavy smoker has a different story. Smoking has been reported to increase insulin resistance and cause central fat deposition leading to increased BMI and obesity in heavy smokers. All these culminate to severe metabolic disorders in smokers and are the underlying causes of several pathological conditions observed in smokers. Smokers are much prone to diseases like diabetes and cardiovascular disorders.[9,10] BSA is correlated to BMI and thus is correlated to body weight and cardiovascular risk factor. Body surface area thus being correlated to BMI is also effected and alters in smokers compared to non smokers.

Smoking adversely effects the respiratory system of individuals. Frequency and duration of smoking are related to the intensity of adverse effects of respiratory status. Smoking is known to cause irritation and damage to the respiratory pathways. Smoking causes breathlessness and effects reduced lung function.[11]

Thus all together, smokers seem to have a worse cardio-respiratory status compared to nonsmokers of the same age group which we have investigated in this study.

## METHODS AND MATERIALS

### Selection of subjects

We conducted the study on 20 adult male human subjects of village Patashimul, Jhargrm District of West Bengal, India who are non smokers and 20 adult male human subjects of the same region of the district of Jahrgram, West Bengal who are smokers. We selected healthy adult male subjects of age group 20 - 30 years for the study. Each parameter was measured three times and mean of those were used for statistical analysis. Subjects with some chronic diseases like diabetes, pulmonary disorder or subjects with reported cardiac ailments were excluded from the study. Also subjects who were on any kind of regular medication were not included in the study in order to ensure homogeneity of sample groups.

We measured certain basic parameters by using anthropometric instruments, weighing machine and peak flow meter to assess BMI, BSA & respiratory peak flow of our subjects.

### Measurement of Anthropometric variables

### Height, Weight and Body mass index (BMI)

Body mass index has been used as a simple anthropometric index used for assessing obesity in an individual which also reflects the current nutritional status of an individual and potential cardiovascular risk status of the individual corresponding to his obesity condition.[12]

### Body surface area (BSA)

Body surface area of the subjects were measured by using the formula of Dubois & Dubois

$A = w^{0.425} * H^{0.725} * 71.84$

Where A (cm2) = Body surface area.
W = Weight in kg.
H = Height in cm.
71.84 is constant

### Body mass index (BMI)

Body mass index has been used as a simple anthropometric index which also reflects the current nutritional status of an individual. BMI was calculated by weight and height measurements using the following formula

**BMI= Weight (kg)/Height ($m^2$)**

### Measurements of Respiratory Peak Flow

Respiratory peak flow of subjects was measured using respiratory peak flow meter [Fig.1]. The subject was asked to take deep breath The meter was placed in the mouth and lips where closed around the mouth piece. Lips formed tight seal around the mouth piece. As hard and fast as possible air was blown out.Number next to indicator was noted. This is the peak flow measure.The indicator was moved to zero, the steps were repeated and two more peak flow measures were taken for each subject.

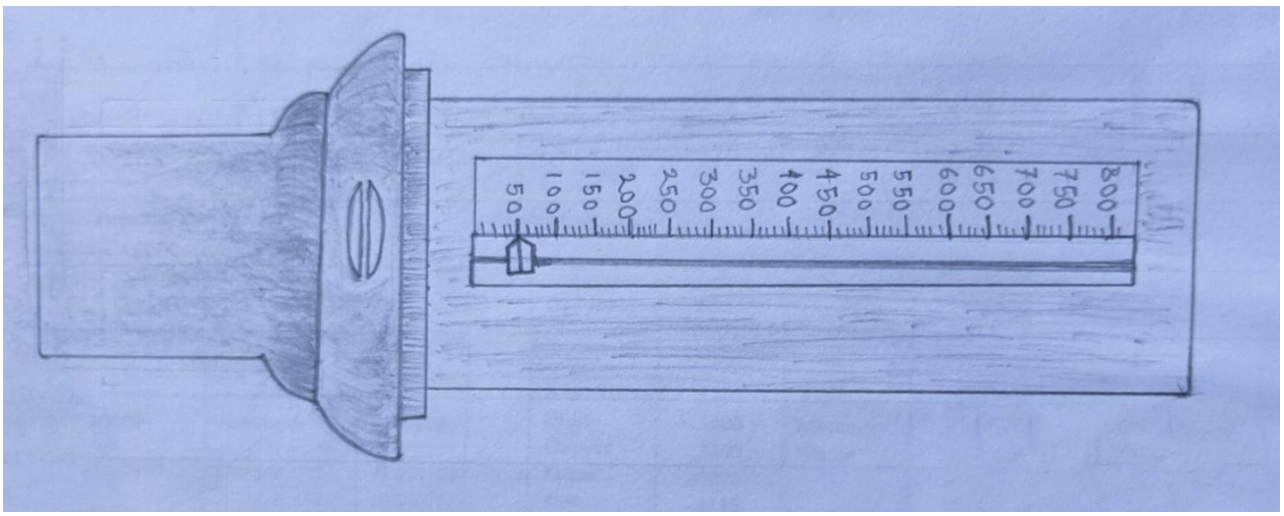


**Fig. 1: Peak Flow Meter.**

**Statistical evaluation**

Each parameter was measured at least three times in each individual. Data are presented as Mean± S.E.M. Significance of difference of mean values of different parameters between the sample groups were analyzed using One Way Analysis (ANOVA). Statistical test were performed using Microcal origin version 7.0 for windows.

## RESULTS

**Body Mass Index (BMI) & Body Surface Area (BSA)**

Body Mass Index (BMI) & Body Surface Area (BSA) of adult male smokers & non smokers [age: 20 years to 30 years] of village Patashimul, Jhargrm District of West Bengal, India were evaluated using their individual body weight and height.

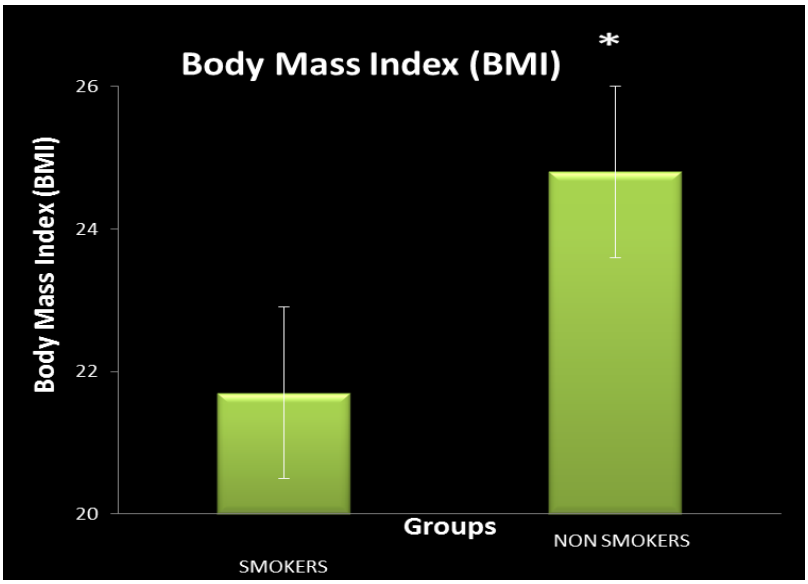


**Figure 2: Body Mass Index (BMI) of smokers and non smokers adult male subjects of village Patashimul, Jhargrm District of West Bengal, India.**
**Values are Mean ± SEM; *P<0.01 Vs Control**

Our study reveals that BMI of nonsmokers is significantly higher than that of smokers (fig.2.). The differences observed are primarily due to differences in the habit & addiction of smoking and impact of nicotine on their metabolism.

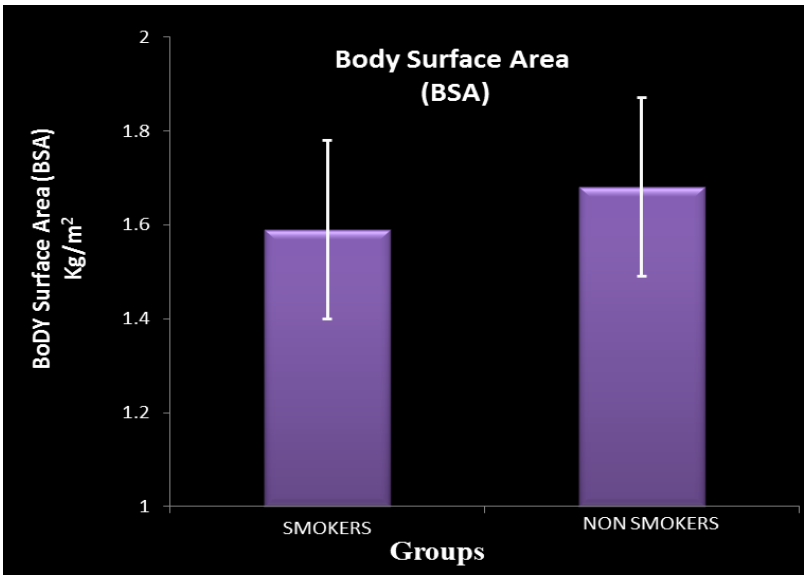


**Figure 3. Body Surface Area (BSA) of smokers and non smokers adult male subjects of village Patashimul, Jhargrm District of West Bengal, India.**
**Values are Mean ± SEM; *P<0.01 Vs Control**

No statistically significant difference is observed in Body surface Area (BSA) of smokers and non smokers (fig.2.).

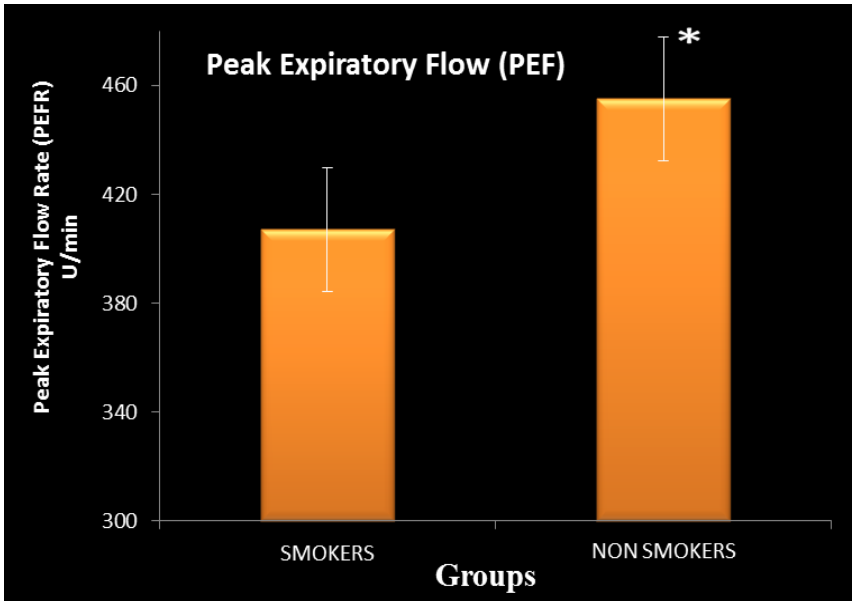


**Figure 4: Peak expiratory Flow rate (PFR) of smokers and non smokers adult male subjects of village Patashimul, Jhargrm District of West Bengal, India.**
**Values are Mean ± SEM;**

No statistically significant difference is observed in Body surface area (BSA) of smokers and non smokers (fig.3.).

We observed a significant difference between the PFR of smokers and non smokers of village Patashimul, Jhargrm District of West Bengal, we considered for our study. It was observed that PEF of non smokers is significantly higher than that of smokers (fig.4.). This observation reveals a better pulmonary condition of non smokers compared to that of smokers.

## DISCUSSION AND CONCLUSION

It is now an well established and scary fact that smoking is not only injurious to health but also is a genuine threat to life. To add on the fatality, addiction to cigarette smoking is very difficult to quit and sadly enough, addiction to smoking reverts back even after months of abstaince. We observed in our studies that smokers have a lower level of BMI, BSA and PFR compared to those of nonsmokers of the same age group and place. Studies reveal that BMI has relationship with cardovascular health status. It is known that post menopausal women with increased BMI have tendencies of developing cardiovascular disorders.[12] Involvement of other factors responsible for such variation in BMI and physiological response to smoking may not be over ruled.[13] Both BMI an BSA are considered determining parameters for obesity and health status.[14] Nicotine is known to play some role and have impact on BMI and BSA in smokers. The exact mechanism cannot be stated but several studies show that nicotine effects various signaling pathways in our body and thus adversely effects several physiological processes. Continuous exposure to nicotine is reported to cause serious pathological condition including diabetes, cancer etc., Nicotine is also reported to activate certain neurons in brain indirectly regulating some physiological processes in addicts.[15] Recent studies reveal that smoking cigarette and inflammatory response causes increased expression of angiotensin converting enzyme 2 (ACE2), the SARS-CoV-2 receptor, in lungs of rodent and human. It is also reported that ACE2 is an interferon-stimulated gene in lung cells, thus it is suggested that SARS-CoV-2 infections can enhance ACE2 levels and facilitate viral dissemination by virtue of positive feedback mechanism.[16] This explains increased risk of smokers to SARS-CoV-2 infections compared to non smokers.[16]

As already mentioned, we have found a reduced peak expiratory flow rate in smokers compared to that in non smokers. The peak flow number tells us how well air is moving through the air ways in our lungs. Peak expiratory flow rate (PEFR) is the maximum flow rate generated during a forceful exhalation, starting from full lung inflation. PEFR primarily reflects large airway flow and depends on the voluntary effort and muscular strength of the patient. Peak flow meter is used to detect pathological conditions associated with the respiratory system. Some of those are, asthma, chronic obstructive pulmonary disease (COPD), a transplanted lung that is not working properly etc., Reduced PFR in smokers clearly reflects a pulmonary insufficiency.

Tobacco smoking is known to trigger inflammatory pathway in the lungs an leads to allergy, asthma etc., Exposure to tobacco smoke causes enhanced mucosal inflammation, increased expression of inflammatory cytokines namely interleukin (IL)-8, IL-6 and tumor necrosis factor α ([TNF]-α). Tobacco smoke is also known to effect epithelial cells directly and causes increase in permeability, mucus overproduction, impaired mucociliary clearance, increased release of

proinflammatory cytokines and chemokines. Increased macrophages and neutrophils get engaged (fig.5.).[17]

The cytokines transforming growth factor β, interleukin (IL-) 10, IL-27, and IL-35 are key coordinators of immune regulation in pulmonary tissue.[18]

In COPD patients increased plasma total TGF-β1 levels were observed. It reflected a feedback regulation by which smoking-induced chronic inflammation promoted high TGF-β1 production.[19] Interleukin (IL)-6, IL-23 and TGF-β activate STAT3 and thus induce Th17 differentiation. Whereas, differentiation of Treg depends on the presence of IL-12 and TGF-β to activate STAT5.[19] The importance of Treg cells in COPD progression is reported but how this Treg cell is involved in COPD exacerbation is not known.[20] It is known that anti-inflammatory cytokines like TGF-β and IL-10 are released by Treg cells. Those Treg cells are known for their ability to inhibit autoimmunity by suppressing inflammation. Studies reveal that a decrease in number of those Treg cells was associated with an increase of Il-17 in the airways of smokers and this lead to obstructions of the respiratory pathway (fig.5).

We also observed a marked increase in the peak flow rate of non-smokers compared to smoker indicating a better pulmonary condition of nonsmokers than that of smokers. It is also revealed from our studies that smoking leads to increased obesity indirctly increasing cardiovascular risk and on the other hand, decreased PFR in smokers reflect pulmonary insufficiency and increased risk of pulmonary disorders in smokers.

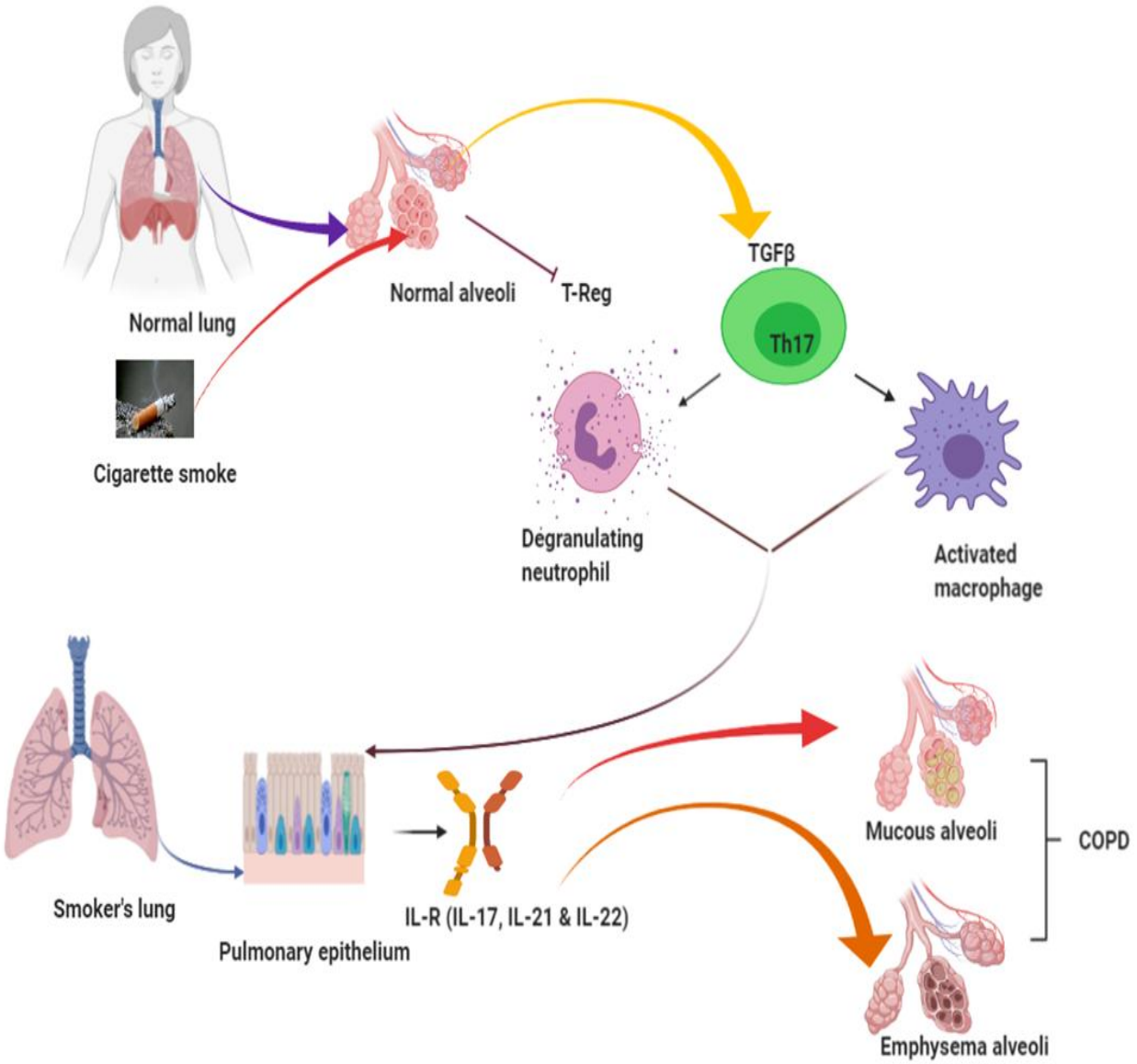


**Figure 5: Mechanism of action of effects of smoking on pulmonary pathophysiology.**

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