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Research Article

A CROSS SECTIONAL STUDY OF VO2 MAX ON PASSIVE SMOKERS IN CHENNAI CITY

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ABSTRACT

Background: Passive smoking increases the risk of occurrence of cardiovascular diseases. One of the parameters used to estimate cardiopulmonary functional capacity is the maximum consumption of oxygen (VO2 max). VO2 max reflects the maximum capacity of absorption, transportation and consumption of oxygen (O2). It is the most influential parameter of a person's physical conditioning and is an independent and objective measure for the prognosis of cardiovascular disease. The effects of passive smoking on cardiorespiratory parameter such as VO2 max were rarely addressed. Thus purpose of present study is to study VO2 max on passive smokers in urban population.
Objective: To evaluate the effects of passive smoking on VO2 max among passive smokers
Materials and Methods: Ninety male subjects aged between 17 and 25 years were placed in three different groups (Passive smokers, active smokers and nonsmokers) on the basis of questionnaire. Weight and height was measured. Maximal oxygen consumption (VO2max) was estimated using Bruce treadmill test.
Results: VO2 max values were decreased significantly in active smokers when compared with passive smokers and control subjects. In present study VO2 max level in active smokers and passive smokers were decreased when compare to control subjects ($p<0.0001$). There was an inverse correlation between Body mass index (BMI) and VO2 max of passive smokers ($p=0.06$) and positive correlations between lean body mass, fat percent and VO2 max ($p=0.60$) ($p=0.38$), But these correlations were not statistically significant.
Conclusion: VO2 max was significantly decreased in active smokers and passive smokers compared to non smokers. It is, therefore, concluded that the VO2 max is an important determinant of the ability of an individual to carry on extended work and is the best indicator of physical fitness.

INTRODUCTION

Cigarette smoking is the most common type of tobacco use and it affects multiple organ systems. The problem has been documented especially in cardiorespiratory function (1,2). The main ingredient causing the adverse health effects of cigarette smoking is the nicotine in the tobacco smoke, which increases heart rate, blood pressure, impairs ventilatory functions, and constricts ventilator pathways and blood vessels (3-9). Nicotine addiction does not cause changes in the behavior or functioning of a smoker; however, it adversely affects the general health status of people within their environment. Passive smoking (so called ETS - Environmental Tobacco Smoke), which means accompanying smokers negatively influences the health of passive smokers. Environmental conditions like overcrowding, poor ventilation in homes and cramped livings conditions, the health effects of ETS exposure may be even more pronounced. Such environment is more common in developing countries like india, because of its large population. Tobacco smoke and its by-products affect the respiratory tract and lungs of adolescents, who either actively smoke or are exposed to secondhand smoke (SHS) produced by their parents, relatives, and/or friends (10). Active smoking (11,12) and passive smoking are associated with multiple adverse respiratory health outcomes including higher rates of asthma, infections of the upper and lower respiratory tract, and reduced lung function. Adolescents may be especially a risk because their lungs are still developing and active (13,14) passive smoking exposure is considered to be health hazards to their respiratory health as that of active smoking(15).

Passive smoking increases the risk of occurrence of cardiovascular diseases. Cardiorespiratory impairment increases morbidity and is an independent predictor of all-cause mortality (16). One of the parameters used to estimate cardiopulmonary functional capacity is the maximum consumption of oxygen (VO2 max) (17,18). VO2 max reflects the maximum capacity of absorption, transportation and consumption of oxygen (O2). It is the most influential parameter of a person's physical conditioning and is an independent and objective measure for the prognosis of

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cardiovascular disease (18,19). Tobacco continues to be the second major cause of death in the world. By 2030, if current trends continue smoking will kill >9 million people annually (20). Recently, cardio respiratory ailments are increasing in the younger generation. Physical activity is known to improve physical fitness and to reduce morbidity & mortality from numerous chronic conditions (21) Inactivity is one of major risk factors for coronary artery disease, at par with smoking, passive smoking. Impaired cardiovascular and respiratory functions are associated with increased mortality and morbidity (22-25). The effects of passive smoking on cardiorespiratory parameter such as VO2 max were rarely addressed. Some studies were done to evaluate the exposure of passive smoking but they did not show significant cardiopulmonary impairment (26). Thus purpose of present study is to study VO2 max on passive smokers in urban population. Objectives of the present study were to estimate VO2 in passive smokers and to compare VO2 max obtained from active smokers and control subjects.

METHODOLOGY

The cross sectional study was carried out at department of physiology, ESIC Medical College & PGIMSR, Chennai. The present study was composed of smokers, passive smokers and nonsmokers (control), aged between 17 and 25 years. Only male subjects of our college staff and relatives of patients attending various outpatient departments, who volunteered for the study and satisfied the inclusion criteria, were recruited. No female subjects were included. Ninety male subjects were placed in three different groups on the basis of questionnaire (Group A active smoker, Group B passive smoker Group C control). Subjects on medication for lung disorder, cardiovascular disorder and central nervous system disorders, diabetes, psychiatric illness were excluded from the study. The present study was approved by the Institutional ethics committee (Approval No: 16-03/07/2015) and an informed consent was taken from all the subjects after explaining the test procedures and the goal of the study. Experiments were done in accordance with revised Helsinki Declaration of 2000. The weight was taken on a weighing scale with standard minimum clothing to the nearest 0.5kg. Height was measured on a vertical scale with the heel, buttocks and occiput against the wall and the head in the Frankfurt plane to the nearest 0.5cm. Resting heart rate was measured with a digital handheld pulse oximeter (BPL Smart Oxy fingertip pulse Oximeter, India). Participants rested at least 15 minutes before examination. An aneroid sphygmomanometer was used to measure systolic arterial pressure (SAP) and diastolic arterial pressure (DAP). Maximal oxygen consumption (VO2 max) was estimated using Bruce treadmill test. It is a reliable and widely used method for estimation of VO2 max using predicted equations. Treadmill cardiopulmonary exercise testing was performed using the Bruce protocol (27). The protocol was stopped once the subject completed all the stages or complained of exhaustion and could not continue the study. The experiments were performed at a room temperature varying from 27-29°C with the relative humidity varying between 60-70°c.

Statistical analysis

Statistical analysis was performed with SPSS (version 17.0). Results are expressed as mean ± SD, and the three groups were compared on one way ANOVA with 5% level of significance. The relation between BMI, fat percent and lean body mass with maximal oxygen consumption (VO2 max) was done using Karl Pearson's correlation with level of significance was considered statistically significant if $p < 0.05$.

RESULTS

Age matched subjects were selected on the basis of inclusion and exclusion criteria. Anthropometric characteristics of active smokers, passive smokers and control subjects were studied thoroughly (fig 1, 2 & 3). VO2 max expressed in l/min. mean and standard deviations of maximum oxygen uptake capacity and its related parameters at their level of significance are listed in table 1. ANOVA was performed to study VO2 max values among active smokers, passive smokers and control subjects. Significant difference were observed between these groups ($P<0.0001$). VO2 max values were decreased significantly in active smokers when compared with passive smokers and control subjects.

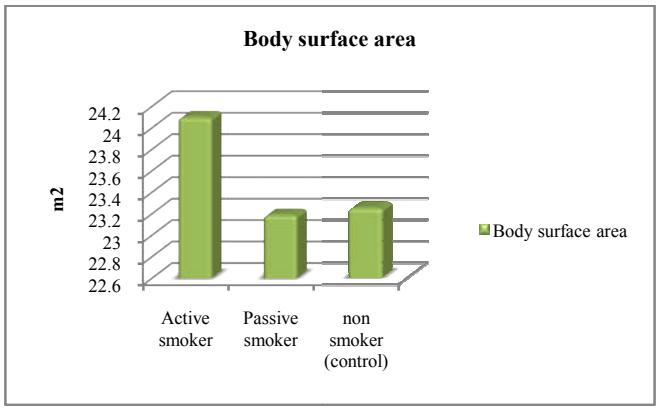

Figure 1 Body surface area of active smoker, passive smoker and control subjects (non smoker)

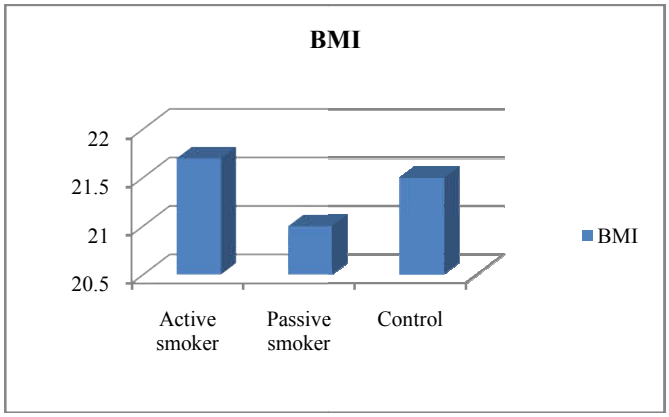

Figure 2 Body mass index (BMI) on active smokers, passive smokers and control subjects (non smoker)

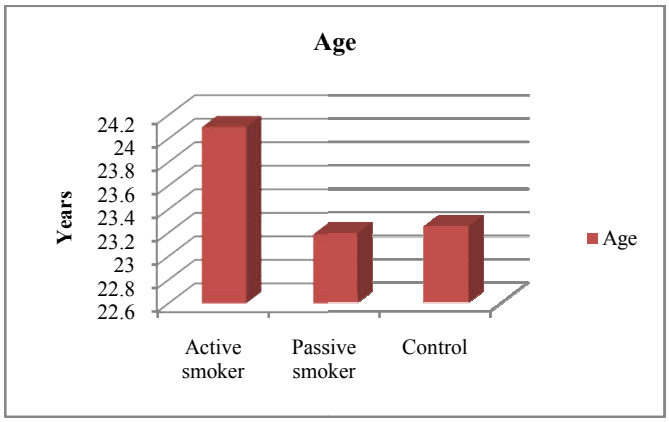

Figure 3 Mean age of study participants

In present study VO2 max level in active smokers and passive smokers were decreased when compare to control subjects. There were no significant difference in resting heart rate, resting systolic blood pressure, resting diastolic blood pressure, lean body mass and fat % between passive smokers, active smokers and control smokers (Table 1).

Table 1 study parameters on active smoker, passive smoker and control subjects

Variables	A.Smoker Mean ± S.D	P.Smoker Mean ± S.D	Control Mean ± S.D	P Value
VO2 Max	40.26± 1.44	42.70 ± 0.16	45.24± 0.65	<0.0001*
Heart rate (resting)	71.96±1.03	71.63±0.49	71.56±0.56	0.08517
SBP (resting)	120.66±1.32	120.73±1.92	119.8±1.68	0.05857
DBP (resting)	76.26±0.73	76.56±1.13	76.76±0.62	0.08321
Lean body mass (Kg)	50.96±0.76	51.10±0.54	50.9±0.66	0.49667
Fat %	19.03±0.43	18.85±0.45	18.77±0.38	0.06605

S.D Standard deviation * statistically significant

Fat % was higher in active smokers when compare to control group but it was not statistically significant. But on the other hand, the present study shows inverse correlation was exist between the VO2 max and Body Mass Index (BMI) of passive smokers and it was not statistically significant (p=0.0677) (Figure 4). Figure 5 showed positive correlation between fat % and VO2 max of passive smokers but it was not statistically significant (p=0.38). Figure 6 showed positive correlation between LBM and VO2 max of passive smokers but it was also not statistically significant (p=0.60). Although technically a positive correlations, the relations between these variables were weak.

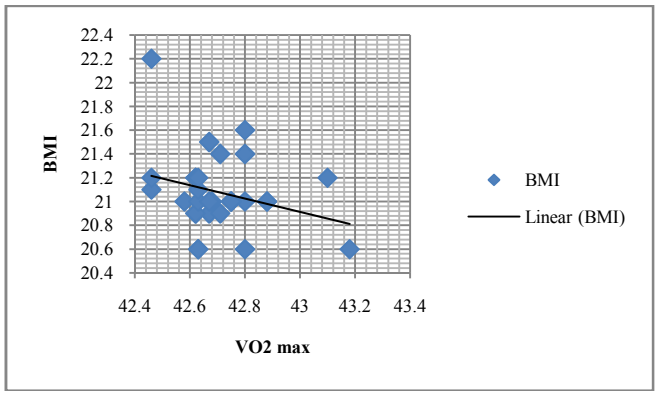

Figure 4 correlation between BMI and VO2 Max in passive smokers

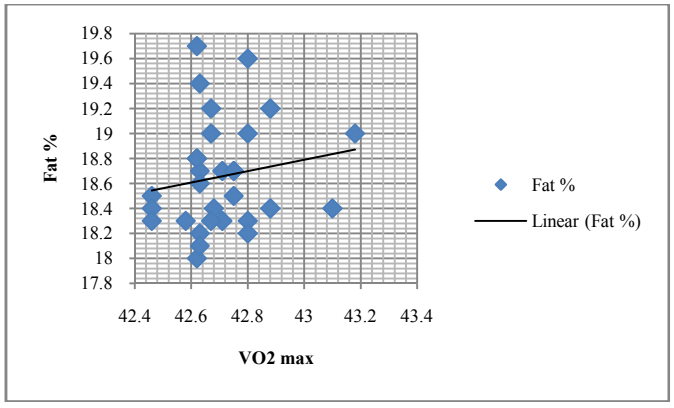

Figure 5 correlation between Fat % and VO2 max in passive smokers

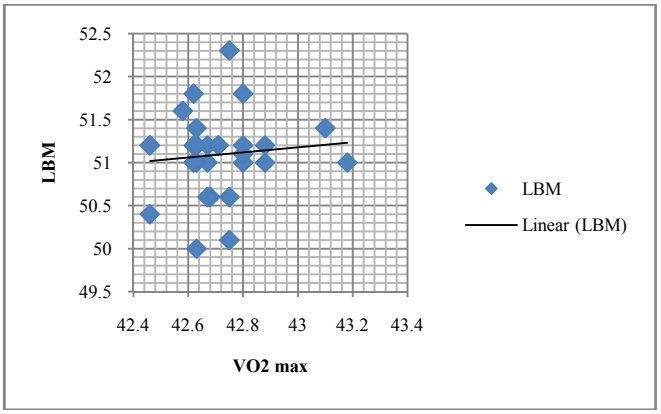

Figure 6 correlation between LBM and VO2 max in passive smokers

Table 2 Correlation between BMI, LBM, Fat% and VO2 max on passive smokers

Parameters	Correlation coefficient(r)	p value
BMI	- 0.33	0.06 (NS)
LBM	0.09	0.60 (NS)
Fat %	0.16	0.38 (NS)

NS: Not significant

DISCUSSION

All active smokers had been smoking for over 10 years. Among the passive smokers, most individuals were exposed to cigarette smoke for over an hour per day many of them had experienced this exposure for over four years. Damage caused by smoking also affects non-smokers who are exposed to cigarette smoke at home, during travelling with smokers, at work, in leisure environments, at school and in other enclosed public areas (28). Passive smoking has been considered a significant risk factor for the development of cardiopulmonary dysfunctions (29, 30). Flouris *et al*. (31) evaluated cardiopulmonary and immunological responses during physical activity training after cigarette smoke exposure. Seventeen individuals of both genders were exposed to cigarette smoke in a controlled environment with a concentration of carbon monoxide similar to what is found in restaurants and bars. These individuals were monitored during and after the completion of moderate physical activities. The authors found cardiopulmonary (increased RQ) and immunological (increased interleukins) changes occur immediately after exposure to cigarette smoke. Several studies have reported a correlation between VO2 max and smoking. Ren *et al*. (32) studied 79 flight attendants exposed to cigarette smoke inside aircraft cabins for over five years reported that passive smoking was linked to systemic arterial hypertension but did not cause hemodynamic, pulmonary or systemic consequences. In our study, there was evidence of significant differences in VO2 max results between passive smokers and non-smokers. The lower VO2 max in passive smokers and active smokers illustrates either a decreased stroke volume or decreased arteriovenous oxygen difference. Another probable reason for the poor VO2 max in smokers and passive smokers is suggested by increased terminal airway obstruction. A decrease in terminal air flow rates due to narrowing of peripheral airways has been reported in smokers (Walter *et al*., 1979) (33). Peripheral airway obstruction is known to cause ventilation perfusion imbalance and abnormalities in the gas exchange resulting in arterial desaturation (Anthonisen *et al*., 1968) (34). Table 1 shows values among passive smokers, active smokers and control subjects. VO2 max results were

significantly reduced in passive smokers when compare to control group (P<0.0001). Our study also shows mean level of VO2 max was significantly decreased when compare to control group. These findings are in agreement results of earlier investigations (35). Thorsten *et al* (36) shows significant negative correlation between maximal oxygen uptake and cigarette smoking which is in agreement with our study. This concurs with the results of McDonough, Kusumi & Bruce (37) and of Cooper, Gey & Bottenberg (38). This indicates that the main difference between smokers and non-smokers may be located in the muscle tissue itself, although another possible mechanism may be the action of carbon monoxide (CO), with a decrease in the oxygen transporting capacity to the mitochondria in the muscle tissue (39). Past reports shows increased heart rate among smokers but there were no proper research statement to defend these reports (40-47). Heart rate response to exercise stimulus may not be influenced by nicotine inhalation. This statement needs detailed clarification with proper research findings. Resting heart rate is considered as one of ideal parameter for assessment of cardiovascular functions. Our study did not show significant changes in resting heart rate values in response to smoking and non smoking activities. This is in agreement with the findings of Dressendorfer *et al* (48), who also reported no difference in heart rate values among smoking and non smoking boys of 16-18 years old. Our results also in agreement with the findings of Blackburn (49) and co-workers who reported that in a group of young men there was no difference in heart rate between smokers and non-smokers at rest. Unverdorben *et al*. (50) observed higher heart rate (HR) and resting SBP (systolic blood pressure) in active smokers than in non-smokers. In this study, there were no significant differences among groups in resting HR or resting SBP, although the active smoker group had slightly lower averages in SBP and slightly higher averages in resting HR. Laukkanen *et al*. (51) evaluated determinants of cardiorespiratory capacity in men aged 42 to 60 years and found an inverse correlation between smoking and VO2 max. Louie (52) conducted a running test in 2 teenagers smokers or non-smokers and showed that, even in young individuals, smoking was associated with a significant reduction of cardiopulmonary activity and exercise tolerance; this effect remained even at light smoking levels. Lean Body mass (LBM) and VO2 max is given in table 2. Though there was a positive correlation between LBM and VO2 max in passive smoker group it was not statistically significant. low lean body mass and muscle mass in smokers may reflects individuals enhanced susceptibility to smoking related problems such as carcinogenesis and chronic obstructive pulmonary diseases (53). In our present study LBM did not show significant changes compared to control group. This is due to smokers in our study does not have long history of smoking habit and many of them were young adults. There were no significant findings on fat % in smokers and passive smokers because many people in our study are young adults. Since our results found positive correlation with week variables between VO2 max and LBM, fat % it augments to the observations that LBM & fat % must be taken into consideration when estimating VO2 max in different ethnic group with different type of cigarette smoking exposure in active and passive smoking groups in detailed manner.

CONCLUSION

VO2 max was significantly decreased in active smokers and passive smokers compared to non smokers. More studies on the effects of passive smoking on VO2 max are needed to be conducted in future. It is, therefore, concluded that the VO2 max is an important determinant of the ability of an individual to carry on extended work and is the best indicator of physical fitness.

Conflicts of interest

There are no conflicts of interest.

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