ORIGINAL ARTICLE

COMPARISON OF PEAK EXPIRATORY FLOW RATE AND LIPID PROFILE IN ASYMPTOMATIC SMOKERS AND NON-SMOKERS

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Background: Tobacco is the major risk factor for chronic obstructive airway disease (COAD), other pulmonary diseases, cancer, cardiovascular and cerebrovascular diseases. The objective of study was to determine the mean Peak Expiratory Flow Rate (PEFR) and serum lipid profile in apparently healthy male smokers and non-smokers. **Methods:** This cross-sectional study was conducted in Ayub Teaching Hospital, Abbottabad from 15th December, 2009 to 15th June, 2010. Apparently healthy smokers and non-smokers from population coming to Hospital as attendants of the patients or as employees of the hospital were inducted in the study. PEFR and lipid profile of all the subjects was accessed. **Results:** There were total of 300 male subjects, 150 smokers and 150 non-smokers. The mean age of study subjects was 26.60±5.5 years. The mean PEFR of smokers was 450.62l/min and that of non-smokers was 494.81 L/min, the difference being statistically significant (*p*-value <0.05).The mean total cholesterol of smokers is 5.30±0.86 mmol/l and it was 3.84±0.54 mmol/l in non-smokers. Mean serum Triacyl Glycerols (TAGs) and Low Density Lipoproteins (LDL) cholesterol of smokers was 2.04±0.38 and 3.5±0.83 mmol/l whereas it was 1.44±0.52 and 2.02±0.66mmol/l in non-smokers. Mean High Density Lipo-protein (HDL) of smokers was 0.86±0.30mmol/l and of non-smokers is 1.20±0.41mmo/l. There was statistically significant difference between serum lipid profile of smokers and non-smokers ($p<0.05$). the mean serum Total Cholesterol (TC), TAGs and LDL were significantly higher in smokers as compared to non-smokers. However HDL was significantly lower in smokers in comparison to non-smokers. **Conclusion:** There was statistically significant difference between PEFR of smokers and non-smokers. Higher and significant mean values of TC, TAG and LDL-C was observed in smokers as compared to non-smokers.

Keywords: Peak Expiratory Flow Rate, smoker, lipid profile, Triacyl Glycerol, Low Density Lipoproteins

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INTRODUCTION

Tobacco is the leading preventable cause of morbidity and mortality around the globe. It is a major risk factor for COAD and other pulmonary diseases, malignancies, cardiovascular and cerebrovascular diseases. Each year, 4.9 million people die due to cigarette smoking and about one in ten deaths among adults are attributed to smoking worldwide.[1] The death toll is expected to rise to ten millions per year by 2020 if the smoking trend prevails.[2] Over all prevalence in Pakistan is 28.6% among males with highest among men aged 40–49 years (40.9%).[3]

PEFR is a frequently used effective tool for the assessment of respiratory functions and air flow obstruction,[4] introduced by Hadron in 1942 and accepted as spirometric index in 1949.[6] It is an effort dependent parameter and is defined as the largest expiratory flow rate achieved with a maximally forced effort from a position of maximal inspiration expressed in L/min.[5] It reflects the changes with air flow calibre. The narrow the airways lower will be the PEFR. It is used to assess the severity of air flow obstruction, in monitoring the response to the treatment and in diagnosing poor control of obstructive air way diseases.[6] The lung function tests of cigarette smokers shows accelerated decline over the period of as compared to non-smokers, and significant improvement in PEFR is shown in the younger population after smoking cessation.[7]

Smoking is also a strong predictor of atherosclerosis, and predisposes to several clinical atherosclerotic syndromes like stable angina, acute coronary syndrome (ACS), sudden death and coma. There is a direct relationship between cardiovascular morbidity and mortality and extent of smoking.

Both active and passive smoking increases adverse cardiovascular events. Active smokers have 80% increased risk of CAD, while passive smokers 30% increased risk. Although the exact mechanism involved is not yet clear but cigarette smoke increases inflammation and thrombosis through alteration in lipid profile.[8] Total cholesterol, triacylglycerol (TAG) and low density lipoproteins (LDL) are raised in smokers in comparison to non-smokers. Whereas a fall in anti-

atherogenic cholesterol, i.e., high density lipoprotein (HDL) has been reported.[9] There is high prevalence of dyslipidaemia in smokers, high TAG (46.1% vs 29.9%) and low HDL cholesterol (42.2% vs 30.4%).[10]

This effect on lipid profile is mediated through the release of catecholamine by the nicotine by enhancing lipolysis, increase in lipoprotein lipase activity and decrease in estrogen.[11] Owing to its insidious nature, the injurious effects of smoking may not evident for a considerable period of time.

It is important to know the alteration in PEFR and serum lipid profile in apparently healthy male population due to smoking and to compare it with non-smokers in our set up, with a view to educate them about the harmful effects of smoking.

MATERIAL AND METHODS

This cross-sectional study was conducted in department of Medicine, Ayub Medical College, Abbottabad, from 15th December 2009 to 15th June 2010, after taking ethical approval from institution's ethical committee. A total of 300 subjects, 150 smokers and 150 non-smokers were enrolled in this study by consecutive (non-probability) sampling technique.

A person who had been smoking cigarettes daily, irrespective of numbers for the last one year or more was taken as smoker. Asymptomatic males determined through history and clinical examination, visiting ATH Abbottabad as attendants or working as employees of the hospital. All subjects, with symptoms of COAD, asthma, or any respiratory complaint, subjects with diabetes and/or hypertension, clinically hypothyroid subjects and those with xanthomas or xanthelasmas, cases with symptoms of any overt disease, subjects with a Body Mass Index of more than 25, industrial workers and road labourers working on asphalt were excluded from the study.

Informed consent was taken from the subjects. Data was collected using structured pro forma. A detailed history and thorough clinical examination of all subjects was performed. A fasting blood sugar level and a urinalysis was done on all subjects. Fasting Lipid Profile including estimation of total cholesterol (TC), triglycerides (TG) and high density lipoprotein cholesterol (HDL-C) in mmol/l. Low density lipoprotein cholesterol (LDL-C) was calculated according to Friedwald formula was done on venous sample drawn through strict aseptic technique for every subject. Peak expiratory flow rate of each subject was measured using mini-Wright peak expiratory flow meter. An average of three readings was measured in litres/min.

Data was analysed by SPSS version 14. Numeric variables like age, pack years, PEFR, total cholesterol, TAG, LDL, HDL were described in terms of mean and standard deviation. Categorical variables like education, occupation and income were determined as frequencies and percentages and *p*-value <0.05 was considered as significant.

RESULTS

A total of 300 subjects included in the study were divided into two equal groups, i.e., smokers and non-smokers, so that each group contained 150 subjects. All the subjects were males aged 18–40 years with a mean age of subjects was 26.60±5.5 years. Distribution of age in both groups tabulated in table-1.

Out of 300 subjects 51 (17%) were unemployed. Majority of the subjects were either professional 104 (34.7%) or salaried 76 (25.3%). Business, manual work and vocational are 32 (10.7%), 29 (9.7%) and 8 (2.7%) respectively. Out of 51 unemployed subjects 37 (72.54%) were non-smokers. Thirty two percent of the smokers were professional and 56 (37%) of the non-smokers were professionals.

The mean PEFR of smokers was 450.62 L/min and that of non-smokers was 494.81 L/min. The difference being statistically significant with a *p*-value of <0.05 (Table-2).

Mean PEFR was maximum between age group 26-35 years both in smokers and non-smokers groups, i.e., 463.77 L/min (n=61) and 507.52 L/min (n=60) and was minimum at 36 years and above, i.e., 405.45l/min (n=22) for smokers and 465.50l/min for non-smokers. In age group 18-25 years mean PEFR of smokers was 453.48±83.49l/min (n=67) and of non-smokers was 487.30±81.62 (n=86). There was statistically significant difference between PEFR of smokers and non-smokers in each age group (*p*-value <0.05) (Table-3).

The mean PEFR of smokers who had smoked for more than 21 years was minimum, i.e., 415±18.60 L/min (n=8) and it was 461.16±76.67 L/min, 470.81±81.15 L/min (n=36), and 417.78±71.42 L/min (n=36) for smokers smoking for 1–5 years, 6–10 years and 11–20 years respectively.

A statistically significant difference between mean PEFR of non-smokers and smokers was observed. Similarly significant difference was observed in smokers having smoked for 11–20 years. When compared to those having smoked 1–5 years and 6–10 years. However no significant difference was established as far as other groups of smoking based on years are concerned.

The mean PEFR among the participants of smokers group was minimum for those smokers who had smoked >20 cigarettes/day, i.e., 440.48±58.02 L/min (n=23), while it was 451.08±83.21 L/min (n=59) for those who smoked 1–9 cigarettes per day and 453.65+/-68 L/min (n=68) for those who smoked 10–20 cigarettes per day. There was statistically significant difference between the PEFR of non-smokers and smokers irrespective of the number of cigarette smoked

per day in the smokers group. However there was no significant difference between mean PEFR of smokers smoking any number of cigarette smoked per day (p>0.05).

The mean PEFR of smokers with pack years less than one was 462.82±81.10l/min, 459.13±81.38l/min for 1–10 pack years and was 423.52±58.65l/min for >10 pack years of cigarette smoking. There was no statistically significant difference between the PEFR of smokers with pack years <1 when compared with those having pack years 1–10 and >10.

The odds ratio was calculated for the two groups, i.e., Smokers and non-smokers with PEFR <400 L/min. The odds ratio was 2.53 (95% Confidence interval, 1.28–4.98) which is statistically significant. Smokers were 2½ times more liable to have PEFR <400 L/min.

Odds ratio for Smokers group (smoking <10 cigarettes/day and those smoking >10 cigarettes/day) and PEFR <400l/min is 1.11 (95% confidence interval, 0.50–2.46) which is not significant statistically.

The mean total cholesterol of non-smokers was 3.84±0.54 mmol/l (n=150) while TGs, HDL and LDL were 1.44±0.52 mmol/l, 1.2±0.41 mmol/l and 2.02±0.66 mmol/l respectively. Non-smokers had mean total cholesterol 5.3±0.86 mmol/l (n=150). The mean TGs, LDL and HDL were 2.04±0.38 mmol/l, 3.54±0.83 mmol/l and 0.86±0.30 mmol/l respectively.

There was statistically significant difference between serum lipid profile of smokers and non-smokers (p<0.05). The mean serum TC, TGs and LDL were significantly higher in smokers as compare to non-smokers. However HDL was significantly lower in smokers in comparison to non-smokers (Table-4).

The mean serum total cholesterol of smokers smoking 1–9 cigarette per day was 4.71±0.6 mmol/l (n=59) whereas it was 5.56±0.77 mmol/l (n=68) and 6.09±0.64 mmol/l (n=23) for those having smoked 10–0 and >20 cigarette per day respectively. The mean serum TGs of smokers smoking 1–9, 10–20 and >20 cigarette per day were 1.8±0.36 mmol/l, 2.1±0.34mmol/l and 2.2±0.35 mmol/l respectively. As far as serum HDL is concerned, it was 0.96±0.36 mmol/l for smokers smoking 1–9 cigarette per day and was 0.78±0.36 mmol/l for smokers smoking >20 cigarettes per day.

When mean serum total cholesterol of non-smokers was compared with that of smokers there was statistically significant difference irrespective of the number of the cigarettes smoked per day. The mean serum cholesterol in smokers smoking 1–9 cig per day and those smoking 10–20 or >20 cig per day was significantly different statistically.

The mean serum TGs of non-smokers and smokers smoking 1–9 cigarette per day was significantly different (p<0.05) and so were those having smoked >10 cigarettes per day. No statistically significant difference in serum TGs was observed in smokers who had smoked 10–20 cigarettes per day and >20 cigarette per day. However there was significant difference between those smoking 1–9 cigarette per day and more than >10 cigarette per day.

The mean serum LDL between non-smokers and smokers smoking any no. of cigarette per day was statistically significant (p <0.05).

The mean serum LDL of smokers when compared within the group based upon number of cigarette smoked per day was found to be statistically significant. The mean difference was significant at the 0.05 level. When mean serum HDL of non-smokers was compared with smokers smoking 1–9, 10–20 or >20 cigarette per day statistically significant difference was observed (p<0.05).

No statistically significant difference was seen between mean HDL of smokers smoking 1–9 and >10 cigarette per day. Similarly no significant association was established between mean serum HDL of smokers smoking 10–20 cigarette per day or >20 cigarette per day. Smokers with pack years <1 had mean serum TC of 4.5±0.53 mmol/l and for those having pack years >10.01 had mean TC of 6.16±0.69 mmol/l. also mean serum TGs and serum LDL for the smokers in the former group was 1.75±0.31 mmol/l (n=40) and 2.80±0.54 (n=40) and for the latter group was 2.3±0.36 mmol/l and 4.39±0.74 mmol/l. However HDL was 0.97±0.38 mmol/l for smokers having <1 pack years of smoking and 0.72±0.30 mmol/l for >10.01 pack years of smoking.

Table-1: Distribution of age groups in smokers and non-smokers

Age groups (years)	Smokers		Non-smokers	
	Frequency	%age	Frequency	%age
18–25	67	44.7	86	57.3
26–35	61	40.7	60	40.0
36 and above	22	14.7	4	2.7
Total	150	100	150	100

Table-2: Mean PEFR in smokers and non-smokers

Study Group	N	Mean PEFR (L/min)	SD	p-value
Smokers	150	450.62	77.20	<.005
Non-Smokers	150	494.81	81.98	
Total	300	472.71	81.98	

Table-3: Mean PEFR in Various Age Groups

Age Group	Study Group	N	Mean PEFR (l/min)	SD
18–25 years	Smokers	67	453.48	83.49
	Non-Smokers	86	487.30	81.62
26–35 years	Smokers	61	463.77	75.74
	Non-Smokers	60	507.52	83.82
36 & Above	Smokers	22	405.45	36.34
	Non-Smokers	4	465.50	29.28

(p-value <0.05)

Table-4: Lipid Profile in Smokers and non-Smokers

Variable	Study Group	Mean	SD	T	df	*p*-value
Serum Total Cholesterol	Smokers	5.31	0.86	17.573	298	.000*
	Non-Smokers	3.85	0.54			
Serum TGs	Smokers	2.04	0.39	11.187	298	.000*
	Non-Smokers	1.45	0.53			
Serum HDL	Smokers	0.87	0.31	-7.840	298	.000*
	Non-Smokers	1.20	0.41			
Serum LDL	Smokers	3.55	0.83	17.586	298	.000*
	Non-Smokers	2.02	0.67			

Table-5: Serum Lipid Profile by Age groups

Age Group (Years)		Mean±SD (mmol/l)			
		TC	TG	HDL	LDL
18-25	Smoker (n=67)	4.86 ±0.71	1.89±0.36	0.93±0.32	3.13±0.63
	Non Smoker (n= 86)	3.77±0.55	1.43±0.53	1.18±0.52	2.05±0.64
26–35	Smoker (n=61)	5.45±0.74	2.07±0.30	0.87±0.29	3.66±0.73
	Non Smoker (n=60)	3.91±0.43	1.50±0.50	1.22±0.40	1.95±0.60
36 & Above	Smoker (n=22)	6.26±0.74	2.40±0.42	0.65±0.14	4.50±0.77
	Non Smoker(n=4)	4.27±1.33	0.80±0.14	1.12±0.17	2.41±1.72
Total	Smoker (n=150)	5.30±0.86	2.04±0.38	0.86±0.30	3.55±0.83
	Non Smoker (n=150)	3.84±0.54	1.44±0.52	1.20±0.41	2.02±0.66

DISCUSSION

The hazardous effects of smoking are insidious in onset and clinical manifestations of various smoking related diseases occur years after indulgence into this habit. However the biochemical and physiological effects are evident before the occurrence of an adverse clinical event. Cigarette smoking significantly affects the PEFR and serum lipid profile.[8] The lung function of smokers show accelerated decline over the period of time and there is high prevalence of dyslipidaemia in smokers. The present study aimed to find out the alteration in PEFR and serum lipid profile in apparently healthy young male smokers and non-smokers hypothesizing that non-smokers have higher PEFR and HDL levels than smokers while LDL, TC and TG are lower in non-smokers as compared to smokers. PEFR and lipid profile of 300 male subjects aged 18–40 (150 smokers and 150 non-smokers) were determined in our study. We observed significantly higher values of PEFR in non-smokers as compared to smokers. Serum lipid profile in two study groups were also significantly different, i.e., smokers had higher mean TC, TG and LDL and lower HDL levels than smokers.

The mean PEFR of non-smokers male is 494.81±81.98l/ min (n=150) which was slightly higher than the mean PEFR of non-smoker males in a study carried out by Inayatullah *et al* at Multan,[12] which is 475±73l/min. similarly it was 424±86l/min (26-30 years of age) as documented by Hussain *et al* in Lahore.[13] And Memon *et al* reported non-smokers PEFR of 399±136l/min (mean age 37 years).[14] The difference in the altitude of the three places might have contributed to this difference, as Lenggenhager has reported that resistance to airway flow was reduced as barometric pressure decreases.[15] The mean PEFR in Indian non-smokers males aged 25 years was 448l/min in a study conducted by Dikshit *at el*.[4] However for non-smokers Chinese males aged 15–70 years, it was 514±59l/min which was higher than PEFR of our non-smoking population but age group in this study was much wider than ours, apart from racial and geographical difference.[16] For European and Americans non-smokers males (mean age 25 years) PEFR was 541l/min and 504l/min respectively. This difference from present study might be due to the physical characteristics, racial and geographical difference in study populations.[4]

A significant difference between mean PEFR of smokers and non-smokers in all age groups was observed in our study. This is in accordance with the findings of Vaidya *et al*,[17] Georgewill *et al*[18]. the mean PEFR of our smokers was 450.62l/min which was significantly lower than non-smokers at the mean age of 26 years. A finding consistence with that of Hussain *et al*[13], Qureshi *et al*[19] and Lyawe *et al*[20] suggesting, significantly reduced lung function in smokers. In contrary to this, Chaterjee *et al*[21] did not observe any statistically significant difference between the mean PEFR in smokers and non-smokers in the age group 20–45 years. However he observed statistically significant difference between PEFR of these two groups after that age group, i.e., 45 years. Duration of smoking was found to be significantly related to mean PEFR, i.e., smokers smoking for 11–20 years had significantly lower PEFR as compared to those smoking for either 1–5 years or 6–10 years. Similar observation regarding duration of smoking was found by Ukoli *et al*.[22]

A pack years of smoking in our study had no effect on PEFR of smokers totally in contrast to the one made by Boezen *et al.*[23] Heaviness of cigarette smoking. i.e., no. of cigarette sticks per day did not significantly affect the lung function (PEFR) in smokers in our study same as documented by Ukoli *et al.*[22]

The mean serum TC in smokers was 5.3±0.83mmol/l which was significantly higher than

that of non-smokers, i.e., 3.84±0.54 mml/l. this observation is consistent with the finding of Akberi *et al* and Sirhindi *et al*.[8,9] Higher TC are associated with CHD. A dose dependent increase in TC was also observed among smokers that were in accordance to the observation made by Abassi *et al*.[11] But it was in contrast with finding of Mammas *et al*[24] which did not show any statistically significant difference between the mean TC of smokers and non-smokers, neither the duration of smoking was correlated with it. However significant relationship was shown between the level of smoking and progressively unfavou8rable TC levels. Racial or dietary factors might have made a contribution towards such a discrepancy.

Smoking was related to lower HDL levels in our study and subjects smoking 11–20 or more cigarettes had significant lower HDL levels to those who smoked 1–10 cigarettes per day. Similar result was documented by Neki NS.[25] No significant difference between smokers and controls with regard to HDL cholesterol was observed by Venkateshan.[26] However the number of subjects in the study was limited that might had contributed towards the normal HDL levels in smokers. Cigarette smoking had been found to lower the concentration of anti-atherogenic HDL levels as demonstrated by Masulli *et al*[10] and Arsalan *et al*.[27] A significant increase in mean serum LDL and mean TG levels were observed in smokers in comparison to non-smokers in our study. Similar results were obtained in studies carried out in Lahore and Karachi.[8–10]

A study carried out by Venkatesan *et al*[26] and Arsalan *et al*[27] showed increases LDL levels in smokers (p <0.05), however the serum TGs in smokers was not significantly raised compared to controls, limited numbers of subjects explained to be the one of the reason for that difference in the results. The mean serum LDL was significantly higher in subjects smoking >20 cigarettes per day as compared to those smoking 1–9 or 10–20 cigarette per day showing a dose dependant relationship. A finding that was consistent with Chun Wu[28] and Neki NS.[25] In contrary to above Tan *et al* interestingly found that LDL levels were normal in smokers.[29]

CONCLUSION

Non-smokers had significantly higher PEFR as compared to smokers. The PEFR for both smokers and non-smokers was in the age group 26–35 years. There is statistically significant difference in the PEFR of smokers and non-smokers in each and every studied age group. The mean TC, TG and LDL of non-smokers were significantly lower whereas serum HDL was higher in non-smokers in comparison to smokers.

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