

## ORIGINAL ARTICLE

## SELF-REPORTED EXPOSURE TO SECOND-HAND TOBACCO SMOKE AND LUNG FUNCTION IN UNDERGRADUATE MALE MEDICAL STUDENTS IN PAKISTAN

Huma Saeed Khan, Naveen Siddique Sheikh, Maria Muhammad Ayub\*, Mehak Tariq, Sidra Zahid, Farida Hafeez

Department of Physiology, CMH Lahore Medical College and Institute of Dentistry,

\*Department of Counselling and Psychological Services, Lahore University of Management Sciences, Lahore, Pakistan

**Background:** Passive smoking, where an individual inhales tobacco smoke, has been associated with many health issues from asthma to cancer and is attributed to affecting pulmonary function tests like the peak expiratory flow rate of individuals. Keeping this in mind, the study was conducted to compare the peak expiratory flow rate of passive smokers with that of non-smokers. **Methods:** A cross-sectional comparative study was conducted from 2017 to 2019 in which 184 male undergraduate medical students enrolled in the study at the time of admission for each successive year after informed consent. Participants were recruited by non probability consecutive sampling technique and divided into two groups based on status of passive smoking, passive smokers (n=97) and non-smokers (n=87). Their height, weight, waist to hip ratio, and peak expiratory flow rate were recorded. The recorded data was analysed on SPSS-26. **Results:** The current study showed that 52.7% of the study participants were passive smokers and 47.3% were non-smokers. A significant difference ( $p<0.01$ ) of peak expiratory flow rate was seen between non-smokers and passive smokers on the Mann-Whitney U Test. As observed by mean ranks, nonsmokers had a higher peak expiratory flow rate (109.86 L/min) than passive smokers (76.93 L/min) ( $p=0.01$ ). Spearman's Rank Correlation Coefficient revealed a positive significant relationship between peak expiratory flow rate and height in passive smokers ( $\rho=0.21$ ,  $p=0.04$ ). **Conclusion:** The peak expiratory flow rate of passive smokers is less than that of non-smokers and there is a positive significant relationship between height and peak expiratory flow rate.

**Keywords:** environmental tobacco smoke, passive smoking, peak expiratory flow rate, lung function test, second-hand smoke

Pak J Physiol 2021;17(3):32-5

### INTRODUCTION

Passive smoking has been attributed to 1% mortality occurring worldwide.<sup>1</sup> It is a modifiable risk factor that is associated with a wide variety of preventable diseases.<sup>2</sup> Passive smoking, i.e., environmental tobacco smoke (ETS) or second-hand smoke (SHS), occurs involuntarily where an individual inhales the mainstream smoke that is expired by the smoker or the sidestream smoke that is diluted in the ambient air, released from burning tobacco.<sup>3</sup> In 2015, 4% of deaths resulting from chronic obstructive pulmonary disease (COPD) were attributed to SHS while 3% of deaths due to stroke were linked to SHS.<sup>4</sup>

Passive smoking not only causes adverse effects to an individual's health but also negatively impacts the economy in terms of production losses due to unavailable workforce and healthcare expenditures, these amounted to US\$ 1436 billion in 2012.<sup>5</sup> In 2018, the economic burden of smoking related illnesses has been attributed to 192 billion rupees (equal to 1.3 billion dollars) in Pakistan.<sup>6</sup>

Second-hand smoke contains around 4,000-4,700 chemicals comprising of hazardous amines, nicotine, hydrocarbons, noxious particles, and metals among others<sup>7</sup>, many of which are reported to

be carcinogenic. These chemicals are irritants that cause stimulation of submucosal irritant receptors which consequently lead to an increment in airway resistance through vagally mediated smooth muscle constriction, impair ciliary movement, thereby decreasing mucociliary clearance, inhibit the function of alveolar macrophages<sup>8</sup>, and cause mucus-secreting glands to undergo hyperplasia and hypertrophy.<sup>9</sup> They cause proteolytic enzymes to be released from polymorphonuclear leucocytes due to antiprotease inhibition thereby playing a significant role in lung parenchymal destruction and COPD pathogenesis.<sup>10</sup>

Passive smoking has even been suggested by a few studies to increase the risk of non-smokers developing heart disease by 25-30%.<sup>11</sup> Sudden infant death syndrome in children and infants, certain cancers, respiratory illnesses such as COPD, and cardiovascular diseases such as coronary artery diseases in adults are linked to passive smoking.<sup>12</sup>

Passive smoking induces pulmonary damage slowly, but unfortunately it may not show symptoms until pulmonary functions are compromised. In Pakistan, the prevalence of passive smokers has been estimated to be 69.1% as compared to 63% in Bangladesh.<sup>13</sup>

Peak Expiratory Flow Rate (PEFR) is defined as the maximum flow rate in forced expiration beginning from full inspiration, measured in liters per minute, taking place within the initial 200 ms of expiration.<sup>14</sup> PEFR varies with sex, age, and anthropometric indices such as height, weight, waist and hip circumference, waist/hip ratio, and body mass index (BMI). Race, ethnicity, and the individual's lifestyle such as smoking also influence PEFR.<sup>15</sup>

PEFR is useful to assess and monitor respiratory conditions such as restrictive and obstructive lung diseases.<sup>16</sup> It is an easy and non-invasive test that gives a rough estimate of the degree of disease extent. PEFR can be used to monitor lung capacity in the early stages of smoke exposure and hence can be used as a reliable tool to prevent lung damage in smokers.<sup>17</sup>

This study was designed with an objective to compare the PEFR in healthy male individuals who were exposed to passive smoke with that of non-smokers.

## SUBJECTS AND METHODS

This cross-sectional comparative study was conducted at CMH Lahore Medical College and Institute of Dentistry from 2017 to 2019 to evaluate and compare the PEFR of passive smokers with nonsmokers. The Ethical Review Board's approval was obtained (case# 433/ERC/CMHLMC).

All male students inducted into 1<sup>st</sup> Year MBBS for three consecutive years (2017, 2018, and 2019) were requested to participate in the study voluntarily at the time of admission in MBBS. A non-probability consecutive sampling technique was used. Sample size of 184 was calculated using the Raosoft sample size calculator, keeping 5% margin of error, 95% confidence interval and 350 as the estimated population size with 50% response distribution.

Participants were interviewed after their informed consent. Boys who had a history of respiratory illness or who had recently suffered a respiratory infection were excluded from the study. Smokers were also excluded from the study. The inducted boys were then divided into two groups: Passive smokers (n=97) and nonsmokers (n=87). Self-reported exposure to passive smoking was defined as being in the same room as a smoker for a minimum of one hour per day for 12 consecutive months or more.<sup>18</sup>

Height was recorded to the nearest centimeter (Cm) with the subject standing, and head in the Frankfurt imaginary plane. Weight was recorded in kilograms (Kg) in usual light clothing. Body mass index (BMI) was calculated using the

formula  $\text{weight in Kg}/(\text{height in meters})^2$ . Waist circumference and hip circumference were measured to the nearest centimeter and waist to hip ratio was calculated. Boys who were obese based upon BMI were excluded from the study.

PEFR was measured using the Wright's peak flow meter by adhering to standard guidelines.<sup>19</sup> Each subject was first explained the technique of performing the lung function test and then was demonstrated as how to perform PEFR before the actual measurement was recorded. Each subject was asked to perform the test thrice in the standing position and the highest value was taken as final.

Data was recorded on a predesigned proforma, and analysed using SPSS-20. Data was explored for normality by using the Shapiro Wilk Test, and accordingly non parametric tests were chosen for inferential statistics. Mann-Whitney U Test and Spearman's Rank Correlation Coefficient were applied to the quantitative parameters of the data, and  $p < 0.05$  was considered to be statistically significant.

## RESULTS

A total of 184 male students participated in the study. The mean age of the study participants was  $18.65 \pm 0.88$  years. Among the study participants, 52.7% were passive smokers and 47.3% were non-smokers. The median peak expiratory flow rate was 450 (IQR 370–550) L/min. The median height of the participants was 173 (IQR 169–177) Cm and weight was 71 (IQR 62–81) Kg. Median BMI was 23.99 (IQR 20.79–36.83). Participants had a median waist and hip circumference of 86 (IQR 81–91) Cm and 96 (IQR 92–104) Cm respectively, and the median waist to hip ratio of the sample was found to be 0.89 (IQR 0.85–0.91). The results of the anthropometric values and PEFR of all study participants are summarized in Table-1.

When compared by the Mann-Whitney U Test, it was noted that the difference in PEFR between non-smokers and passive smokers is significant ( $p < 0.01$ ). Participants who were nonsmokers had a higher peak expiratory flow rate than passive smokers as suggested by their mean ranks. Non-significant differences were observed on other anthropometric parameters between the two groups (Table-2).

Spearman's Rank Correlation Coefficient was applied to the quantitative parameters of the data. Table-3 shows the result of Spearman's Rank Correlation Coefficient where a positive significant relationship was observed between peak expiratory flow rate and height of the subjects.

**Table-1: Anthropometric variables and peak expiratory flow rate (PEFR) reported with their median value, lower quartile (LQ), and upper quartile (UQ) for all study participants (n=184)**

Variables	Median	LQ	UQ
PEFR (L/min)	450	370	550
Age (Years)	19	18	19
Height (Cm)	173	169	177
Weight (Kg)	71	62	81
BMI (Kg/Cm <sup>2</sup> )	23.99	20.79	36.83
Waist Circumference (Cm)	86	81	91
Hip Circumference (Cm)	96	92	104
Waist to Hip Ratio	0.89	0.85	0.91

**Table-2: Mann-Whitney U Test for PEFR with anthropometric parameters between non-smokers and passive smokers (n=184) (Mean Ranks)**

Variables	Non Smokers (n=87)	Passive Smokers (n=97)	p
PEFR (L/min)	109.86	76.93	0.01*
Age (Years)	95.65	89.68	0.42
Height (Cm)	84.18	99.96	0.05
Weight (Kg)	88.46	96.12	0.33
BMI (Kg/Cm <sup>2</sup> )	90.37	94.41	0.61
Waist Circumference (Cm)	89.16	95.49	0.42
Hip Circumference (Cm)	88.45	96.13	0.33
Waist to Hip Ratio	96.00	89.36	0.40

\*Significant

**Table-3: Spearman's rank correlation coefficient between PEFR with anthropometric parameters for non smokers and passive smokers**

Variables	Age		Height		Weight		WC		HC		WHR		BMI	
	rho	p	rho	p	rho	p	rho	p	rho	p	rho	p	rho	p
Non Smokers	0.09	0.42	0.20	0.06	0.12	0.27	0.00	0.99	0.04	0.72	-0.12	0.27	0.03	0.81
Passive Smokers	0.17	0.10	0.21	0.04*	0.10	0.35	0.19	0.06	0.10	0.31	0.19	0.06	0.05	0.65

\*Significant

## DISCUSSION

Passive smoking is attributed to cause a decrease in pulmonary functions, as shown by previous researches. In our study, there was a significant difference between the PEFR of passive smokers and non-smokers inducted in the first-year MBBS. The results of our study are in line with the findings reported by an Iraqi researcher whose study demonstrated a statistically significant difference in PEFR between young adult passive smokers and nonsmokers.<sup>20</sup> Similar findings have been reported from a study previously done in Lahore which evaluated differences in PEFR between adult passive smokers and nonsmokers to be statistically significant, with nonsmokers having a lower PEFR than their passive smoking counterparts.<sup>21</sup>

The key variables that affect PEFR are the strength of respiratory muscles producing a contraction that results in expiration, lung compliance, airway competency, and resistance offered to airflow.<sup>22</sup> Gender, age, height, weight, and body surface area have shown a significant correlation for PEFR in previous studies.<sup>23</sup> The results of our study are also in line with the findings reported by Indonesian researchers, however, their study population comprised of a younger age group between the ages of 10–13 years.<sup>22</sup> This shows that the effect of passive smoking is the same on lung function regardless of the age group affected.

Considering gender has been reported to affect PEFR, we conducted the study in only males to rule out gender differences and evaluate the correlates within the same gender for various variables. Our study highlighted a significant positive correlation between height and PEFR, where taller male subjects had a higher PEFR value, a finding which is supported by previously conducted studies.<sup>24</sup> With a height

increment, there is an increment in the chest girth, and the thoracic area hence total surface area of the lungs increases. Because taller people have a larger surface area for air exchange than shorter people, a greater amount of air can circulate in and out, resulting in an increase in vital capacity and consequently PEFR in taller people. This is probably because taller subjects have a greater chest volume and with an increment in height the effort by the expiratory muscles and the growth of the airway passages also increases thus increasing PEFR with height as evidenced in previous literature.<sup>25</sup> Previous studies have reported that in males, not only does an increase in height causes an increase in PEFR but a weight increment also causes PEFR to increase, this however, does not support our research findings.<sup>26</sup> A study done in Nigeria reported a significant correlation between height and PEFR of men subjected to passive smoking, this finding resonates with the findings of our study which showed similar correlation between the two indices, however the study also showed a correlation between PEFR and other anthropometric indices such as weight, which does not support our study findings of nonsignificance between weight and PEFR.<sup>27</sup>

The current study has a few limitations. The role of other attributes like socioeconomic status, malnutrition, years of exposure to second-hand smoke, and childhood smoke exposure history in non-smokers was not explored. A larger sample size taken at random and evaluating other lung function tests such as FVC and FEV<sub>1</sub> could help improve future researches. The active metabolite of nicotine, cotinine may be measured in passive smokers. Furthermore, evaluating PEFR corrected for forced expiratory pressure could shed more light on the disparities in PEFR results between the two groups.

## CONCLUSIONS

First-year MBBS male students who are exposed to passive smoking have lesser PEFR values as compared to their non-smoking non-exposed class fellows. This study highlights the need to create awareness about the detrimental effects of passive smoking on an individual's health and the need for regulation of its control to curb the passive smoking issue.

## REFERENCES

- Torres S, Merino C, Paton B, Correig X, Ramirez N. Biomarkers of exposure to secondhand and thirdhand Tobacco smoke: Recent advances and future perspectives. *Int J Environ Res Public Health* 2018;15(12):2693.
- Dede C, Cinar N. Environmental tobacco smoke and children's health. *Iran J Pediatr* 2016;26(5):e5935.
- Buran MN, Samet JM. Secondhand tobacco smoke. In: Lippmann M, Leikauf GD, editors. *Environmental Toxicants*. 1<sup>st</sup> ed. Wiley; 2020.p. 911–26. Available from: <https://onlinelibrary.wiley.com/doi/10.1002/9781119438922.ch24>
- WHO. Preventing noncommunicable diseases (NCDs) by reducing environmental risk factors. Geneva: World Health Organization; 2017. Available from: <https://apps.who.int/iris/bitstream/handle/10665/258796/WHO-FWC-EPE-17.01-eng.pdf> (last accessed on 29 November 2020)
- Goodchild M, Nargis N, D'Espaignet ET. Global economic cost of smoking-attributable diseases. *Tob Control* 2018;27:58–64.
- Saqib MAN, Malik A, Rafique I, Raza FA, Obaidullah, Sajjad SF, et al. Economic burden of smoking attributed illnesses in Pakistan. *medRxiv* 2020; Available at: <https://www.medrxiv.org/content/10.1101/2020.06.15.20131425v1> [cited 2021 Sep 17]
- Raghuvver G, White DA, Hayman LL, Woo JG, Villafane J, Celermajer D, et al. Cardiovascular Consequences of Childhood Secondhand Tobacco Smoke Exposure: Prevailing Evidence, Burden, and Racial and Socioeconomic Disparities: A Scientific Statement from the American Heart Association. *Circulation* 2016;134:336–59.
- Ween MP, Whittall JJ, Hamon R, Reynolds PN, Hodge SJ. Phagocytosis and Inflammation: Exploring the effects of the components of E-cigarette vapor on macrophages. *Physiol Rep* 2017;5(16):e13370.
- Harkema JR, Eldridge EA, Freeland A, Jackson-Humbles D, Lewandowski RA, Wagner JG, et al. Pathogenesis and Persistence of Increased Epithelial Mucosubstances in the Nasal Airways of Rats and Mice Episodically Exposed to Ethylene. *Toxicol Pathol* 2020;48(7):875–86.
- Strzelak A, Ratajczak A, Adamiec A, Feleszko W. Tobacco smoke induces and alters immune responses in the lung triggering inflammation, allergy, asthma and other lung diseases: A mechanistic review. *Int J Environ Res Public Health* 2018;15(5):1033.
- Lee W, Hwang SH, Choi H, Kim H. The association between smoking or passive smoking and cardiovascular diseases using a Bayesian hierarchical model: based on the 2008-2013 Korea Community Health Survey. *Epidemiol Health* 2017;39:e2017026.
- Permitasari NPAL, Satibi S, Kristina SA. National burden of cancers attributable to secondhand smoking in Indonesia. *Asian Pac J Cancer Prev* 2018;19:1951–5.
- Rafique I, Nadeem Saqib MA, Bashir F, Naz S, Naz S. comparison of tobacco consumption among adults in SAARC countries (Pakistan, India and Bangladesh). *J Pak Med Assoc* 2018;68(Suppl 2)(5):S2–6.
- Dobra R, Equi A. How to use peak expiratory flow rate. *Arch Dis Child Educ Pract Ed* 2018;103:158–62.
- Mukherjee S, Banerjee G, Mahapatra ABS. Peak expiratory flow rate changes with relevant variables in a population of eastern India. *Indian J Physiol Pharmacol* 2018; 62(3):372–9.
- Archana D, pawankalyan P, reddy S, Sharma V. To Determine PEFR in Children Aged 8 to 12 years in Nellore District: IOSR J Dent Med Sci 2016;15(6):30–36.
- Sawant GV, Kubde SR, Kokiwar PR. Effect of smoking on PEFR: a comparative study among smokers and non smokers in an urban slum community of Hyderabad, India. *Int J Community Med Public Health* 2016;3(1):246–50.
- MR, Clarkson P, Robinson J, McCredie R, Donald A, Deanfield JE. Passive smoking and impaired endothelium-dependent arterial dilatation in healthy young adults. *N Engl J Med* 1996;334(3):150–4.
- Atta K, Zia S, Jabeen F. Impact of anthropometric parameters on peak expiratory flow rate. *J Aziz Fatimah Med Dent Col* 2019;1:63–6.
- Abdulrahman WF. Effect of smoking on peak expiratory flow rate in Tikrit University. *Med J Tikrit* 2011;17(1):11–8.
- Hussain G, Zafar S, Chaudhary AA, Chaudhary ZA, Ahmad MZ. Comparative Study of Peak Expiratory Flow Rate in Cigarette Smokers and Non-Smokers of Lahore District. *Ann King Ed Med Univ* 2007;13(4):255–9.
- Saxena Y, Purwar B, Upmanyu R. Adiposity: determinant of peak expiratory flow rate in young Indian adults male. *Ind J Chest Dis Allied Sci* 2011;53:29–33.
- Srivastava S, Agarwal D. Correlates of peak expiratory flow rate and deriving the prediction equation in school going children of Lucknow. *Int J Com Med Public Health* 2017;4(11):3550–3.
- Skladanowski M, Jarosz P, Mackiewicz B. Variations of Peak Expiratory Flow Rate Associated with Various Factors Among Healthy Adults in a City Setting. *Pol J Public Health* 2016;126:91–4.
- Kaur H, Singh J, Makkar M, Singh K, Garg R. Variations in the peak expiratory flow rate with various factors in a population of healthy women of the malwa region of Punjab, India. *J Clin Diagn Res* 2013;7(6):1000–3.
- Dharamshii HA, Faraz A, Ashraf E, Alam SS, Ali A, Shakeel O, et al. Variation of PEFR with height, weight and waist-hip ratio in medical students. *Int Arch Med* 2015;8(84):1–6.
- Elebute EA, Femi-Pearse D. Peak flow rate in Nigeria: anthropometric determinants and usefulness in assessment of ventilatory function. *Thorax* 1971;26:597–601.

## Address for Correspondence:

**Dr Naveen Siddique Sheikh**, Department of Physiology, CMH-Lahore Medical College and Institute of Dentistry, Lahore, Pakistan. **Cell:** +92-321-4153436

**Email:** naveen.s.sheikh@gmail.com

Received: 5 Jan 2021

Reviewed: 17 Sep 2021

Accepted: 18 Sep 2021

## Contribution of Authors:

**HSK:** Conceptualisation, visualization, data curation, methodology design, project administration, reviewing and editing draft, final approval

**NSS:** Data curation, methodology design, original draft writing, reviewing and editing draft, reference cross-checking

**MMA:** Methodology design, formal statistical analysis of data on SPSS, result write-up for manuscript

**MT:** Data curation, methodology design, reference cross-checking

**SZ:** Data curation, methodology design, reference cross-checking

**FH:** Methodology design, project administration, Final approval

**Funding:** No funding sources

**Conflict of interest:** None declared

**Ethical approval:** The study was approved by the IRB