

Content available at: https://www.ipinnovative.com/open-access-journals

Indian Journal of Clinical Anatomy and Physiology

Journal homepage: https://www.ijcap.org/



Original Research Article

Effects of passive smoking on pulmonary functions of individuals in an urban area

Harsida Gosai¹, Anita Verma²*

¹Dept. of Physiology, Narendra Modi Medical College, Maninagar, Ahmedabad, India



ARTICLE INFO

Article history:
Received 20-10-2023
Accepted 08-11-2023
Available online 20-01-2024

Keywords: Passive smoking Active smoking Lung functions FEF25-75% VC FEV1/FVC%

ABSTRACT

Background: Passive smoking is a world health problem and part of the tobacco epidemic which victimizes mostly adolescents. Epidemiological studies have, essentially, addressed the association between passive smoke exposure and respiratory health in adult population, and increased risk of chronic obstructive pulmonary disease (COPD) & lung cancer among adult nonsmokers. Relatively few studies have been reported on lung function in relation to effects of passive smoking among nonsmoking individuals.

Objective: To investigate the effects of passive smoking on lung functions among individuals in urban region.

Materials and Methods: The present study was composed of smokers, passive smokers and nonsmokers (control), aged between 17 and 25 years. Ninety healthy individuals were placed in three different groups on the basis of questionnaire (Group A: active smoker, Group B: passive smoker, Group C: control). Study was done with the help of spirometer (RMS Helios 401).

Results: $FEF_{25-75\%}$, FEV1/FVC% values were significantly decreased in passive smokers as compared to control individuals (P<0.001, P<0.05).

Conclusions: The present study suggests a significant reduction of lung functions in individuals exposed to passive smoke.

This is an Open Access (OA) journal, and articles are distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

For reprints contact: reprint@ipinnovative.com

1. Introduction

Cigarette smoking has been identified as the single most important source of premature mortality. ^{1–5} Tobacco smoke contains over 4000 chemicals in the forms of particles and gases. ² When burned, cigarettes create more than 7,000 chemicals. At least 69 of these chemicals are known to cause cancer, and many are toxic.

Passive smoking or environmental tobacco smoke (ETS) exposure has been variously described as 'second-hand smoke' or 'involuntary smoking'. ETS comprises the amount of tobacco smoke which is not inhaled by the smoker (sidestream smoke-SS), as well as the portion of

E-mail address: harshi_g100@yahoo.com (A. Verma).

inhaled smoke (mainstream smoke) which is not retained in the smoker's lung, and is exhaled into the environment. In enclosed spaces, smoke accumulates, and the concentration varies with the number of smokers, with the type of smoking, and with the characteristics of the room, especially the ventilation. Although the exposures to active smoke and ETS are not identical, the latter appears to include most tobacco combustion byproducts, including irritants, and carcinogens. ^{6–8}

The most popular Indian smoking product is "bidi" (also spelled as beedi). Bidis are made of crude sun-dried tobacco wrapped in a dried Tendu (Dyospyros melanoxylon) leaf. Another smoking product used in different parts of India is "chillum" or "hooka", which resembles a pipe made of clay. Tobacco is burnt along with molasses and coal, and smoked

²Dept. of Physiology, Smt. NHL Municipal Medical College, Ahmedabad, Gujarat, India

^{*} Corresponding author.

either directly or through a long pipe with smoke passing through a water container. The amount of nicotine and tobacco alkaloids present in the mainstream smoke (MS) of these vernacular tobacco products is likely to be different from those present in the MS of standard cigarettes because of the differences in their design (e.g. water acts as a filter in hooka and no filter exists in most bidis). Smoke causes an increase in the number of pulmonary alveolar macrophages (PAM). Macrophages release a chemical substance that attracts leucocytes in lung. Leucocytes in turn release proteases including elastase which attack elastic tissue in lung. Normally alpha-1 antitrypsin inactivates elastase and other proteases. Alpha-1 antitrypsin is inactivated by oxygen radicals which are released by the leucocytes leading to protease antiprotease imbalance with increased destruction of lung tissue. Exposure to passive smoking is associated with diverse health effects in nonsmokers. These adverse health outcomes include heart disease, lung cancer, asthma, chronic obstructive pulmonary disease (COPD), and upper airway problems. ^{9–16} Few studies have been conducted to study the effect of ETS on pulmonary function in developing countries like India, 17,18 Environmental conditions like overcrowding, poor ventilation in homes and cramped livings conditions, the health effects of ETS exposure may be even more pronounced. Moreover, the effects of ETS exposure on children important aspects, as their respiratory and immune systems are still in developmental stages, possibly putting them at high risk of being affected by passive smoke. Such environment is more common in developing countries like India, because of its large population. In this study we evaluated the effects of exposure to passive smoking among young adults.

2. Materials and Methods

The study was carried out at department of physiology, Nootan medical College and Research Center, Visnagar. It was a cross-sectional study. The present study was composed of smokers, passive smokers and nonsmokers (control), aged between 17 and 25 years. Only healthy male subjects were included in the present study. No female subjects were included. The subjects selected for present study were recruited from our institution. Ninety healthy individuals were placed in three different groups on the basis of questionnaire (Group A active smoker, Group B passive smoker Group C control). The 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 Helsinki declaration of 1975.

2.1. Study group

- 1. Group A (Actives smokers-male); individuals with history of cigarette smoking more than three year were considered as active smokers
- Group B (Passive smokers-male); Non-smoking male subjects those who lived with at least one smoker or interacted with a smoker at work for at least three years prior to the study.
- 3. Group C (control group male); Non smoking male subjects who were neither active nor passive smokers.

2.2. Inclusion criteria

Healthy individuals in the age group of 17 - 25 years.

2.3. Exclusion criteria

Subjects on medication for Respiratory (RS), Cardiovascular (CVS) and Central nervous system (CNS) disorders. Subjects with past and present history of CVS disorders, diabetes, psychiatric illness. Subjects with H/O drug abusing.

2.4. Test parameters chosen for this study are

- 1. PEF (L/S) (Peak expiratory flow rate)
- 2. FEV1 (L)(Forced expiratory volume in first second of FVC)
- 3. VC (L) (Vital capacity)
- 4. FVC (L) (Forced vital capacity)
- 5. FEVI/FVC (%)
- 6. FEF _{25-75%} (Forced expiratory flow)
- 7. MVV (Maximum voluntary ventilation)

2.5. Study procedure

A detailed clinical history of these subjects are to be taken such as Relevant past history, family history, any drug history, personal history like smoking, alcoholism etc. Study was done with the help of spirometer (RMS Helios 401). The whole procedure was explained to the subject thoroughly. Anthropometric measurements (height, weight, BMI, BSA) were recorded for each subject. Relevant data (name, age, sex, height, weight) was entered into computer program. The subjects were given proper instructions prior to each parameter. All the pulmonary function tests were performed with the subjects in standing position and wearing nose clips. ¹⁹

The subjects were asked to take deep inspiration from outside and then to expire as forcefully and as fast as he can inside the mouthpiece. A nose clip was applied during the entire procedure. Tests were repeated three times and the best matching results were considered for analysis.

2.6. Statistical analysis

The three groups were compared on one way ANOVA with 5% level of significance.

3. Results

Ninety healthy individuals were included in the present study. Table 1 shows mean age, height, weight, body mass index and body surface area of active smokers, passive smokers & control individuals. There was no significant difference in the mean physical parameters like age, height, weight, body mass index and body surface area among active smokers, passive smokers and control individuals. Lung functions (FVC, PEFR, FEV1, VC) of active smokers, passive smokers and control subjects. The values of FVC (P=0.787), PEFR (P=408), FEV1 (P=0.926), and VC were decreased in active smokers and passive smokers as compared to control individuals. Values of VC (vital capacity) were decreased significantly in active smokers (P=0.010) however there were no significant changes in FVC, PEFR, and FEV1values among active smokers and passive smokers as compared to control subjects. Figure 2 shows mean values of MVV among active smokers, passive smokers and control subjects but there is no significant change among these three groups (P=0.269). Figure 3 shows FEV1/FVC% values were significantly lower in active smokers as compared to control individuals (P=0.022). Figure 4 shows FEF25-75% values were highly significant in passive smokers as compared to control individuals.

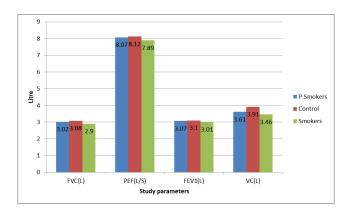


Figure 1: Pulmonary function tests (FVC, PEF, FEV1 & VC) among Active smokers (A. Smokers), Passive smokers (P. Smokers) & control subjects

4. Discussion

Nowadays, smoking is considered to be one of the unhealthiest of human behaviors and the most hazardous of addictions. Recently, considerable attention has been focused on the harmful effect of "passive" smoking, confirming ETS as a serious and substantial public health

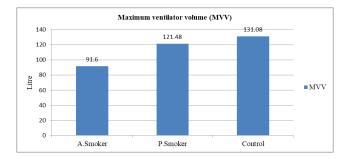


Figure 2: Pulmonary function test (MVV) among active smokers (A. Smokers), passive smokers (P. smokers) & control subjects

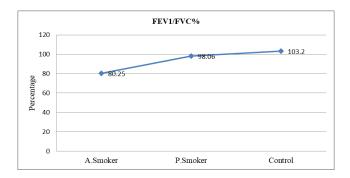


Figure 3: Pulmonary function test (FEV1/FVC %) among Active smokers (A.Smokers), Passive smokers (P. Smokers) & control subjects

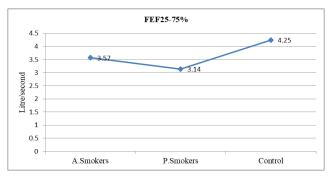


Figure 4: Pulmonary function test (FEF25-75%) among Active smokers (A.Smokers), Passive smokers (P.Smokers) & control subject

problem, with particular impact on respiratory health of children. ^{20,21} Some studies have also demonstrated this effect in adults. ²² Exposure to passive smoke, contributes to the occurrence of diseases of the lower and higher airways, asthma, wheeze, lung lesions detectable in computer tomography, as well as lung function impairment. ^{23–25} In the case of passive smokers, the risk of the occurrence of chronic obstructive pulmonary disease is significantly higher. Worldwide, 40% children, 33% male non-smokers and 35% female nonsmokers were exposed to ETS in 2004. This exposure was estimated to have caused 603,000 deaths,

·		3
A.Smoker Mean ± S.D.*	P.Smoker Mean ± S.D.*	Control Mean ± S.D.*
24.10 ± 10.11	23.19 ± 12.09	23.25 ± 11.08
1.70 ± 0.19	1.71 ± 0.22	1.71 ± 0.12
59.5 ± 9.20	61.4 ± 12.06	60 ± 10.02
21.7 ± 2.21	21 ± 3.02	21.5 ± 2.10
1.70 ± 0.09	1.72 ± 0.02	1.70 ± 0.06
	24.10 ± 10.11 1.70 ± 0.19 59.5 ± 9.20 21.7 ± 2.21	$24.10 \pm 10.11 \qquad 23.19 \pm 12.09$ $1.70 \pm 0.19 \qquad 1.71 \pm 0.22$ $59.5 \pm 9.20 \qquad 61.4 \pm 12.06$ $21.7 \pm 2.21 \qquad 21 \pm 3.02$

Table 1: Physical characters of active smokers (A. Smokers), passive smokers (P. Smokers) and control subjects

which was about 1% of worldwide mortality By 2020, this disease is probably going to be the top third cause of mortality. ^{26,27}

FEF₂₅₋₇₅ indicates flow rates in small airways, i.e. airways with internal diameters less than 2 mm, which is reduced in both restrictive and obstructive diseases.²⁸ The present study showed significant reduction of FEF₂₅₋₇₅ values in passive smokers (P=0.001). It is in agreement with previous studies 29,30 showing that FEF₂₅₋₇₅ values were significantly decreased in passive smokers. White et al"31 reported small airways dysfunction with a 14% reduction in FEF₂₅₋₇₅ in non-smokers exposed to environmental tobacco smoke; Kauffman et al³² studied the effect of exposure to cigarette smoke at home on lung function in French men and showed a 6% fall in FEF₂₅₋₇₅, Masi et al' 33 found adverse effects of passive smoking on FEF₂₅₋₇₅ in young men (aged 15- 35). Our results were in agreement the findings of White et al., Kauffman et al., Masi et al. Another study done by Casale et al, 34 who investigated the effects of passive smoking on lung function of children aged 6-11 years old, and found the FEF₂₅₋₇₅ to be significantly reduced in study group.

MVV (L/min) is a dynamic and scientific method for measurements of respiration muscles capacity and indeed, is a test for evaluation of human respiration system. The MVV was formerly called the maximum breathing capacity(MBC) is the largest volume of gas that can be moved in and out of the lungs in one minute by voluntary effort. The normal MVV is 125-170 l/min. 35 In the present study, the MVV was decreased when compare to control group but it was not statistically significant. In the present study, VC was decreased in passive smokers and smokers, but it did not reach the level of statistical significance in passive smokers but it was significantly decreased in active smokers. It is in agreement with Gupta D et al 18 who couldn't able to find out significant changes in VC among passive smokers.

FEV1 is an important part of evaluating chronic obstructive pulmonary disease (COPD) and monitoring progression of the condition. Some studies show that the FEV1 values of children were significantly reduced by parental smoking. The more recent findings reported by Merghani and Saeed the students (9–14 years old) in Khartoum, Sudan, and found the FEV1 and FVC to be significantly lower in the passive

smoke exposed group than the nonsmoker control group. Our study does not show significant difference in FEV1 & FVC values. Kentner et al. 38 carried out an investigation involving 1,351 white collar workers. He couldn't able to find out significance difference in FVC. Our findings were in agreement with Kentner et al. Another study from Mohammad-Reza Masjedi et al shows significant reduction of FEV1 and FVC values in male passive smoker. ³⁹ Results of previous studies regarding FEV1 are more consistent, with a majority showing a lower FEV in children exposed to parental smoking. 40 In the present study we did not find any significant changes in FEV1 & FVC parameters in passive smokers but it shows lower values in passive smokers however it did not show statistical significance. It might be due to nature of exposure of passive smoking, many individuals in our study were exposed by passive smoking in open air. This might not produce changes in FEV1 & FVC. The FEV1/FVC ratio, also called Tiffeneau-Pinelli index, is a calculated ratio used in the diagnosis of obstructive and restrictive lung disease. 41,42 It represents the proportion of a person's vital capacity that they are able to expire in the first second of forced expiration (FEV1) to the full, forced vital capacity (FVC). According to recent studies, even 1 hour of passive smoking exposure can induce a significant decrease in FEV1 and FEV1/FVC ratio along with cytokine releases. 43 The present study showed significant changes in FEV1/FVC ratio (P=0.02). It is in agreement with Meenakshi Kalyan et al. 44

Smoking in an enclosed area increases the concentration of respirable particles such as nicotine, polyacrylic hydrocarbons, carbon monoxide, acrolein and nitrogen dioxide. The impact of smoking on indoor air quality depends on the number of smokers, the intensity of smoking, the size of the indoor space, the rate of exchange of the air of the indoor space with the outdoor air, and the use of air cleaning devices. In our study less number of people have exposure on passive smoking in enclosed area. There are some limitations that may influence the interpretation of our cross-sectional results. We were unable to determine the extent of indoor air pollution in the homes of the subjects. In addition, we lacked information about status of air pollution in their living area, and we couldn't able to get proper details regarding ventilation, population in their places. Some reports have linked cooking energy sources to impairment of ventilatory function, 45-47 whereas

^{*}S.D. = Standard Deviation

others have not found such an effect. 48 It needs further detailed analysis.

5. Conclusion

The present study suggests a significant reduction of lung functions in individuals exposed to passive smoke. Passive smoke exposure is an independent risk factor for developing COPD and passive smokers are at similar risk like active smokers.

6. Source of Funding

None.

7. Conflict of Interest

None.

References

- 1. Fielding JE. Smoking: health effects and control (1). *N Engl J Med*. 1985;313(8):491–8.
- Tobacco smoking: IARC monographs on the evaluation of the carcinogenic risk of chemicals to humans. International Agency for Research on Cancer; 1986.
- 3. Doll R, Gray R, Peto R, Wheatley K. Tobacco related diseases. *J Smok Relat Dis.* 1990;1:3–13.
- International Agency for Research on Cancer. Cancer: causes, occurrence, and control. Lyon: IARC Scientific Publications; 1990.
- Schelling TC. Addictive drugs: the cigarette experience. Science. 1992;255:430–3.
- Mason RJ, Buist AS, Fisher EB, Merchant JA, Samet JM, Welsh CH. Cigarette smoking and health. Am Rev Respir Dis. 1985;132:1133–6.
- O'Neill IK, Brunnemann KD, Dodet B, Hoffmann D. Environmental carcinogens: methods of analysis and exposure measurement - passive smoking. *IARC Sci Publ.* 1987;9(81):1–372.
- Maclure M, Katz RB, Bryant MS, Skipper PL, Tannenbaum SR. Elevated blood levels of carcinogens in passive smokers. Am J Public Health. 1989;79(10):1381–4.
- Eisner MD, Balmes J, Katz PP, Trupin L, Yelin EH, Blanc PD. Lifetime environmental tobacco smoke exposure and the risk of chronic obstructive pulmonary disease. *Environ Health*. 2005;4(1):7.
- Kawachi I, Colditz GA, Speizer FE, Manson JE, Stampfer MJ, Willett WC, et al. A prospective study of passive smoking and coronary heart disease. *Circulation*. 1997;95(10):2374–9.
- Lam T, Ho L, Hedley A, Adab P, Fielding R, McGhee S, et al. Secondhand smoke and respiratory ill health in current smokers. *Tob Control*. 2005;14(5):307–14.
- Taylor AE, Johnson DC, Kazemi H. Environmental tobacco smoke and cardiovascular disease. A position paper from the Council on Cardiopulmonary and Critical Care. Circulation. 1992;86(2):699– 702
- 13. Eisner MD, Wang Y, Haight TJ, Balmes J, Hammond SK, Tager IB, et al. Secondhand smoke exposure, pulmonary function, and cardiovascular mortality. *Ann Epidemiol*. 2007;17(5):364–73.
- Eisner MD, Klein J, Hammond SK, Koren G, Lactao G, Iribarren C. Directly measured second hand smoke exposure and asthma health outcomes. *Thorax*. 2005;60(10):814–21.
- Pitsavos C, Panagiotakos D, Chrysohoou C, Skoumas J, Tzioumis K, Stefanadis C, et al. Association between exposure to environmental tobacco smoke and the development of acute coronary syndromes: the cardio 2000 case-control study. *Tob Control*. 2002;11(3):220–5.
- Pitsavos C, Panagiotakos DB, Chrysohoou C. Association between passive cigarette smoking and the risk of developing acute coronary

- syndromes: the cardio 2000 study. Heart Vessels. 2002;16:127–130.
- Ğupta D, Aggarwal AN, Kumar Ř, Jindal DK. Prevalence of bronchial asthma and association with environmental tobacco smoke exposure in adolescent school children in Chandigarh, North India. *J Asthma*. 2001;38(6):501–7.
- 18. Gupta D, Aggarwal AN, Jindal SK. Pulmonary effects of passive smoking: the Indian experience. *Tob Induc Dis.* 2002;1(2):129–36.
- Standardization of spirometry, 1994. Update. American Thorasic Society. Am J Respir Crit Care Med. 1994;152(3):1107–36.
- Zlotkowska R, Zejda JE. Fetal and postnatal exposure to tobacco smoke and respiratory health in children. Eur J Epidemiol. 2005;20(8):719–27.
- Skorge TD, Eagan TM, Eide GE, Gulsvik A, Bakke PS. The adult incidence of asthma and respiratory symptoms by passive smoking in uterus or in childhood. Am J Respir Crit Care Med. 2005;172(1):61–6.
- Carey IM, Cook DG, Strachnan DP. The effects of environmental tobacco smoke exposure on lung function in a longitudinal study of British adults. *Epidemiology*. 1999;10:319–26.
- Wang L, Pinkerton KE. Detrimental effects of tobacco smoke exposure during development on postnatal lung function and asthma. *Birth Defects Res C Embryo Today*. 2008;84(1):54–60.
- Butz AM, Halterman JS, Bellin M, Tsoukleris M, Donithan M, Kub J, et al. Factors associated with second-hand smoke exposure in young inner-city children with asthma. *J Asthma*. 2011;48(5):449–57.
- Burgess JA, Matheson MC, Gurrin LC, Byrnes GB, Adams KS, Wharton CL, et al. Factors influencing asthma remission: a longitudinal study from childhood to middle age. *Thorax*. 2011;66(6):508–13.
- Tanaka K, Miyake Y. Association between prenatal and postnatal tobacco smoke exposure and allergies in young children. *J Asthma*. 2011;48(5):458–63.
- Burgess JA, Matheson MC, Gurrin LC, Byrnes GB, Adams KS, Wharton CL, et al. Factors influencing asthma remission: a longitudinal study from childhood to middle age. *Thorax*. 2011;66(6):508–13.
- Cotes JE. Lung functions. 3rd ed. Oxford: Blackwell Scientific Publications; 1975. p. 68–108.
- Chen Y, Li WX. The effect of passive smoking on children's pulmonary function in Shanghai. Am J Public Health. 1986;76(5):515–8.
- Bek K, Tomac N, Delibas A, Tuna F, Tezic HT, Sungur M. The effect of passive smoking on pulmonary function during childhood. *Postgrad Med J.* 1999;75(884):339–41.
- White JR, Froeb HF. Small-airways dysfunction in nonsmokers chronically exposed to tobacco smoke. N Engl J Med. 1980;302(13):720–3.
- Kauffman F, Tessier JF, Oriol P. Adult passive smoking in the home environment: a risk factor for chronic airflow limitation. Am J Epidemiol. 1983;117(3):269–80.
- Masi JA, Hanley JA, Ernst P, Becklake MR. Environmental exposure to tobacco smoke and lung function in young adults. *Am Rev Respir Dis*. 1988;138(2):296–9.
- Casale R, Colantonio D, Cialente M, Colorizio V, Barnabei R, Pasqualetti P. Impaired pulmonary function in school children exposed to passive smoking: detection by questionnaire and urinary cotinine levels. *Respiration*. 1991;58(3-4):198–203.
- and KMR. Comparative study of peak expiratory flow rate and maximum voluntary ventilation between smokers and nonsmokers. *Natl J Med Res*. 2012;2(2):191–3.
- Fielding JE, Phenow KJ. Health effects of involuntary smoking. N Engl J Med. 1988;319:1452–60.
- Merghani TH, Saeed AM. The relationship between regular secondhand smoke exposure at home and indictors of lung function in healthy school boys in Khartoum. *Tob Control*. 2013;22(5):315–8.
- Kentner M, Triebig G, Weltle D. The influence of passive smoking on pulmonary function: a study of 1,351 office workers. *Prev Med*. 1984;13(6):656–69.
- Masjedi M, Kazemi H, Johnson DC. Effects of passive smoking on the pulmonary function of adults. *Thorax*. 1990;45(1):27–31.

- Cook DG, Strachnan DP, Carey IM. Parental smoking and spirometric indices in children. *Thorax*. 1998;53(10):884–93.
- 41. Swanney MP, Ruppel G, Enright PL, Pedersen OF, Crapo RO, Miller MR, et al. Using the lower limit of normal for the FEV1/FVC ratio reduces the misclassification of airway obstruction. *Thorax*. 2008;63(12):1046–51.
- 42. Sahebjami H, Gartside PS. Pulmonary function in obese subjects with a normal FEV1/FVC ratio. *Chest*. 1996;110(6):1425–9.
- Allwright S, Paul G, Greiner B, Mullally BJ, Pursell L, Kelly A. Legislation for smoke-free workplaces and health of bar workers in Ireland: before and after study. *BMJ*. 2005;331(7525):1117.
- 44. Kalyan M, Kanitkar SA, Gupta A, Sharma D, Singh S. Effect of passive smoking as a risk factor for chronic obstructive pulmonary disease in normal healthy women. *Int J Res Med Sci.* 2016;4(11):4970–3.
- Samet JM, Marbury MC, Spengler JD. Health effects and sources of indoor air pollution, part I (state of the art). Am Rev Respir Dis. 1987;136:1486–508.
- Tager IB. "Passive smoking" and respiratory health in childrensophistry or cause for concern? Am Rev Respir Dis. 1986;133(6):959– 61

- Burchfiel CM, Higgins MN, Keller JB, Howatt WF, Butler WJ, Higgins ITT. Passive smoking in childhood. Respiratory conditions and pulmonary function in Tecumseh, Michigan. Am Rev Respir Dis. 1986;133:966–73.
- Comstock GW, Meyer MB, Helsing KJ, Tockman MS. Respiratory effects of household exposures to tobacco smoke and gas cooking. Am Rev Respir Dis. 1981;124:143–8.

Author biography

Harsida Gosai, PhD Scholar, Tutor

Anita Verma, PhD Guide, Professor and HOD

Cite this article: Gosai H, Verma A. Effects of passive smoking on pulmonary functions of individuals in an urban area. *Indian J Clin Anat Physiol* 2023;10(4):225-230.