ORIGINAL ARTICLE

Role of passive smoking in non-smoking related chronic obstructive pulmonary disease

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Abstract

Objective: To determine the association between passive smoking and chronic obstructive pulmonary disease.

Methods: The cross-sectional study was conducted from October 2015 to March 2016 at the Ojha campus of Dow University Hospital, Karachi, and comprised individuals who denied past or current active smoking and exposure to bio-mass fuel. The subjects were attendants coming with patients, hospital staff, faculty and medical students aged 15-64 years. Each subject undertook pulmonary function tests via spirometer after filling a proforma based on assessment and quantification of the exposure to second-hand smoke and common symptoms of chronic obstructive pulmonary disease. Data was analysed using SPSS20.

Result: Out of 307 subjects, 196(63.84%) were currently exposed to passive smoke either at home or at workplace or at both, and 24(12.24%) of them had chronic obstructive pulmonary disease, diagnosed by means of spirometry. Out of these subjects, 5(20.8%) had stage I, 9(37.5%) had stage II, 8(33.3%) had COPD stage III and 2(8.3%) had stage IV disease. A significant association was found between density of passive smoke inhaled over a period of time and chronic obstructive pulmonary disease ($p<0.05$).

Conclusion: Passive smokers were found to be at risk of chronic obstructive pulmonary disease.

Keywords: Chronic obstructive pulmonary disease, COPD, Passive smoke, Spirometry, Pulmonary function tests, Second-hand smoke. (JPMA 68: 1310; 2018)

Introduction

According to Global Initiative for Chronic Obstructive Lung Disease (GOLD), Chronic Obstructive Pulmonary Disease (COPD) is defined as a common, preventable and treatable disease that is characterised by persistent respiratory symptoms and airflow limitation that is due to airway and/or alveolar abnormalities usually caused by significant exposure to noxious particles or gases. COPD ranks higher in causing mortality and morbidity and is a disease of most concern in this era.[1]

There are multiple risk factors for developing COPD and associations of some factors are already established like cigarette smoking and exposure to biomass fuel, but published data in recent years demonstrates a significant prevalence of COPD among those who never smoke, with varying prevalence across nations.[2] The proportion appears higher in developing countries and gets lower in developed countries.[3] In Asian region, indoor/outdoor air pollution and poor socioeconomic status may play a significant role in the pathogenesis of non-smoking-related COPD.[4] One form of pollution is passive smoke and it is one of the independent aetiological factors for COPD in non-smokers.

According to the World Health Organisation (WHO), involuntary (or passive) smoking is the exposure to second-hand tobacco smoke (SHS) which is a mixture of exhaled mainstream smoke and side-stream smoke released from a smouldering cigarette or other smoking device (cigar, pipe, bidi, etc.) and diluted with ambient air.[5] Second-hand tobacco smoke is also referred to as "environmental" tobacco smoke (ETS). The overall risk depends on the effective dose received over time. ETS can be evaluated either by directly questioning regarding passive exposure to tobacco smoke either at home or at workplace or by using biomarkers in saliva, urine, blood and hair, as an indirect measure of exposure which includes carbon monoxide monitored through breath, nicotine, cotinine and thiocynates.[6]

The workplace is a major source of SHS exposure for many adults. The Occupational Safety and Health Administration (OSHA) and the National Institute for Occupational Safety and Health (NIOSH), federal agencies responsible for health and safety in the workplace, recognise that there are no known safe levels of SHS, and recommend that exposures be

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reduced to the lowest possible levels.[7] Multi-unit housing where smoking is allowed, is a special concern and a subject of research.[8]

SHS causes many of the same diseases as direct smoking, including cardiovascular diseases, lung cancer and respiratory diseases.[9] Between 1964 and 2014, 2.5 million people died from exposure to SHS according to a report from the US Surgeon-General,[10] which also reported that living or working in a place where smoking is permitted increases the non-smokers' risk of developing heart disease by 25-30% and lung cancer by 20-30%. Association between involuntary inhalation of cigarette and increased number of various respiratory diseases, including COPD, either in children or in adults, is now clearly established.[11,12] According to Guangzhou Biobank Cohort Study, conducted in China in 2007, exposure to passive smoking is associated with an increased prevalence of COPD and respiratory symptoms. If this association is causal, 1·9 million excess deaths from COPD among never-smokers could be attributable to passive smoking in the current population in China.[13] A study in the southern district of Karachi in 2009 also showed that respiratory illness was more common in smoker families compared to non-smoker families.[14,15]

The current study was planned to find out casual association of passive smoking with spirometric-diagnosed COPD.

Subjects and Methods

The cross-sectional study was conducted from October 2015 to March 2016 at the Ojha Institute of Chest Diseases (OICD), Dow University Hospital, Karachi, and comprised individuals aged 15-64 years who denied past or current active smoking and exposure to biomass fuel. The subjects were attendants coming with patients, hospital staff, faculty and medical students. Approval was obtained from the institutional review board, and the investigating team comprised two doctors from the Medicine department and one highly experienced nurse from the OICD.

A brief history was taken before enrolment of each participant in order to exclude already-diagnosed respiratory or cardiovascular diseases. Those with severe obesity (body mass index [BMI] over 35) and who had current or past exposure to smoking and/or biomass fuel, evaluated by means of history, were also excluded. Non-probability, consecutive sampling was used and the inclusion criterion was strictly followed in order to limit bias and to avoid confounding factors.

After obtaining verbal informed consent, all participants were provided with a proforma to gather information regarding age, gender, educational level, occupation, residential area, level of exposure to passive smoking at home and work by two self-reported measures that are density and duration of exposure, and common respiratory system complaints that is shortness of breath and cough. To establish content validity, team members were asked to narrate the questions and help each participant in filling the proforma. To ascertain the density of exposure per week, simple questions were asked, like how many times in a week a person interacts with a smoking partner who may be his or her spouse, close relative, friend or office coulleage. This session took 10-15 minutes and adequate privacy was provided during this time. After this, participants were invited for pulmonary function test in the OICD out-patient department (OPD) where the established technique of spirometry was used, as directed by the team members under the supervision of chest physicians. The spirometric parameters of interest were Forced Expiratory Volume in 1 second (FEV1), Forced Vital Capacity (FVC) and FEV1/FVC ratio. Each participant was first informed about the procedure and then allowed to proceed. Out of multiple attempts, three readings showing a participant's best efforts were taken and among them, best values were taken for comparison with predicted values. FEV1 normal predicted values, adjusted for age and height of participants, were calculated using European Respiratory Society (ERS) guidelines for spirometry.[16] COPD was diagnosed when FEV1/FVC was less than 70% that was not corrected after bronchodilator challenge, and was staged according to GOLD criteria;[17] stage I when FEV1>80% normal, stage II if FEV1 50-79% normal, stage III if FEV1 30-49% normal, and stage IV if FEV1<30%.

Data was analyzed using SPSS20. Primary outcome variable was detection of COPD. Level of exposure to passive smoking and socioeconomic status was analysed for their influence on developing COPD. Quantitative variables like age, number of smokers in the vicinity and duration of exposure to SHS were expressed as mean ±standard deviation whereas qualitative variables like gender, educational level, childhood exposure to passive smoke, density of exposure to SHS, respiratory symptoms, presence of COPD and its severity were reported in the form of frequencies and percentages. Univariate type of analysis was done using Chi-square test. Respiratory symptoms were scored according to Medical Research

Council criteria.[18] Confidence interval (CI) was set at 95% and p<0.05 was considered statistically significant.

Results

Of the 307 subjects, 214(69.7%) were male and 93(30.3%) were female (Table-1). Overall, 196(63.84%) were currently exposed to passive smoke either at home or at workplace or both. Of them, 158(73.83%) were male and 38(40.86%) were female. Out of 196 exposed subjects, 24(12.24%) had COPD, and, of them, 5(20.8%) had stage I, 9(37.5%) stage II, 8(33.3%) stage III, and 2(8.3%) had COPD stage IV. In terms of gender, 15(62.5%) males and 9(37.5%) females had COPD. Age-wise distribution of COPD suggested it was common in the elderly group (Figure).

Overall, 295(96%) were aware of the hazards associated with passive smoke, and 12(4%) persons, who were primarily uneducated also, claimed that it was not dangerous as it is not directly inhaled in the same amount compared to that inhaled by the smoker. Besides, 190(62%) subjects were passively exposed to tobacco smoke in their homes during their childhood, and 148(78%) of them had current exposure to SHS also. Of the 24 subjects diagnosed with COPD, 21(87.5) belonged to this category. The number of smokers in the surroundings of a passive smoker (p=0.386) and ventilation status (p=0.150) were not statistically significant factors in relation to COPD. A positive correlation was found between density of smoke inhaled and duration of its exposure with COPD (p<0.05) (Table-2). Among the 24 subjects diagnosed with COPD, 22(91.7%) complained of cough; 11(50%) had occasional cough with cold or chest infection, 7(31.8%) had cough for a few days in a month, and 4(18.8%) had cough for most days in a week. No one complained of daily severe cough. Besides, out of 24 COPD patients, 16(66.7%) complained of shortness of breath; 9(56.2%) noticed it during exercise, 3(18.7%) when walking upstairs, 3(18.7%) while walking on level ground, and 1(6.25%) at rest.

Table-1: Characteristics of participants.

Description	N (%)	Mean ± SD
Gender		
Male	214 (69.7)	
Female	93 (30.3)	
Childhood Exposure	190 (49.2)	
Current Exposure	196 (50.8)	
Density of Current Exposure		
Daily	85 (43.4)	
> 2 times/week	80 (40.8)	
< 2 times/week	31 (15.8)	
Age in years		2.15 ± 1.11
15-24	103	
24-34	109	
35-44	52	
45-54	31	
55-64	12	
Educational level		2.97 ± 1.05
None	38	
Primary	47	
Secondary	121	
Graduation	88	
Post-Graduation	13	
Numbers of smokers in surrounding		1.83 ± 0.81
One	84	
Two	61	
More than two	51	
Duration of exposure		2.15 ± 0.74
< 2 years	42	
2-5 years	83	
> 5 years	71	

SD: Standard deviation.

Table-2: Significant Risk Factors for chronic obstructive pulmonary disease (COPD).

Variables	COPD		p-value
	Yes (%)	No (%)	
Total Nos. of participants	24 (7.8)	283 (92.2)	
Childhood Exposure			
Present	21 (87.5)	169 (59.7)	< 0.0001
Absent	3 (12.5)	114 (40.3)	
Current Exposure			
Present	24 (100)	172 (60.8)	< 0.0001
Absent	0 (0.0)	111 (39.2)	
Density of Exposure			
Daily	20 (83.3)	65 (37.8)	
> 2 times/week	4 (16.7)	76 (44.2)	< 0.0001
< 2 times/week	0 (0.0)	31 (18)	
Duration of Exposure			
< 2 Years	1 (4.2)	41 (23.8)	
2-5 years	5 (20.8)	78 (45.3)	< 0.0001
> 5 years	18 (75.0)	53 (30.8)	
Smokers in Surrounding			
1	8 (33.3)	76 (44.2)	
2	7 (29.2)	54 (31.4)	0.368
>2	9 (37.5)	42 (24.2)	
Adequate Ventilation			
Yes	24 (100)	158 (91.9)	0.150
No	0 (0.0)	14 (8.1)	

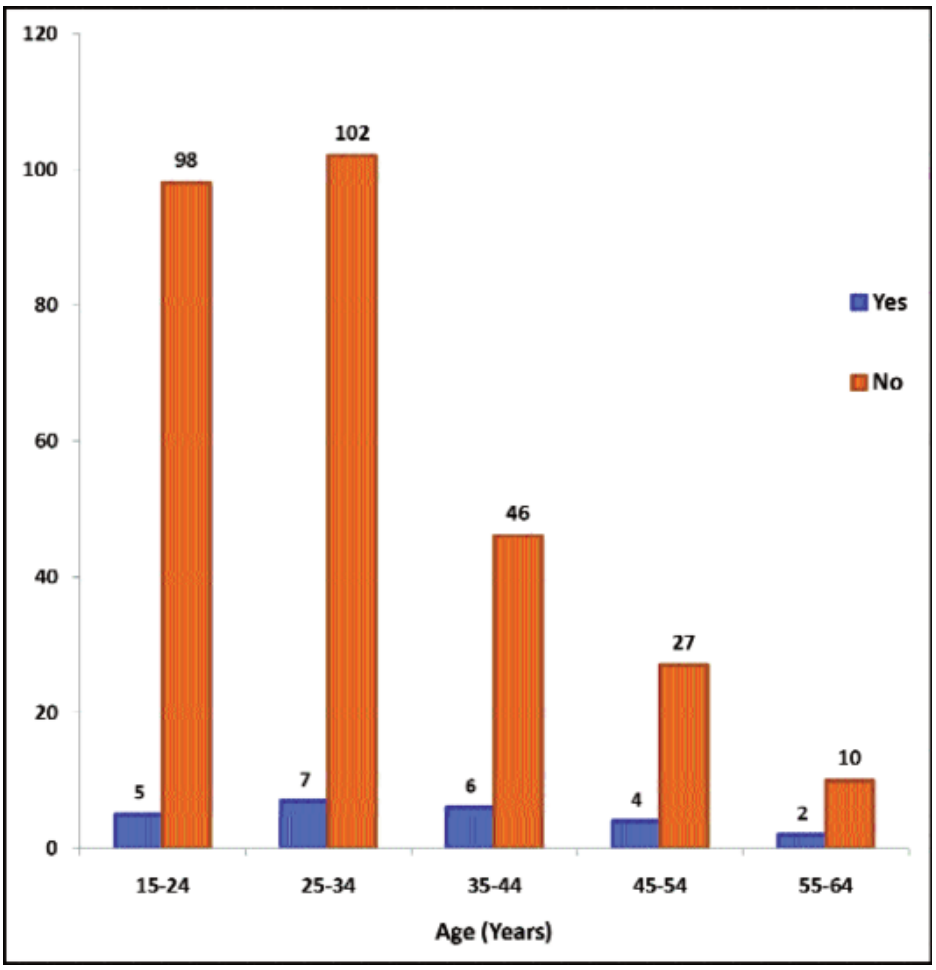

Figure: Age-wise frequency of chronic obstructive pulmonary disease (COPD).

Discussion

The main outcome of the current study is that 63.84% study population was exposed to environmental tobacco smoke and 12.24% of this exposed population had undiagnosed COPD. Males were more exposed than females but the prevalence of COPD was slightly higher in females compared to the male population. Statistically significant factors contributing to COPD in passively exposed individuals were found to be childhood exposure, current domestic and workplace exposure and density of smoke inhaled passively over a period of years.

The actual burden of passive smoking is very difficult to estimate as a single smoker can expose many people to the harmful effects of his smoke. Worldwide, 40% children, 33% male non-smokers and 35% female non-smokers were exposed to SHS in 2004. This exposure was estimated to have caused 603,000 deaths, which was about 1% of worldwide mortality.[19,20] According to Global Adult Tobacco Survey (GATS) 2014, exposure to second-hand smoke in Pakistan was seen in 86% and this exposure was 76% each in a restaurant and on public transportation.[21] In a multi-centric, cross-sectional study from three of Pakistan's major cities, Islamabad, Rawalpindi and Karachi, SHS exposure was assessed by measuring mean concentrations of particulate matter ≤2.5 microns in diameter (PM 2.5), a sensitive indicator of SHS levels, in hospitality venues such as cafés, restaurants and food courts. The United States Environmental Protection Agency (EPA) has defined standards for safe exposure to PM limiting average annual atmospheric exposure to 15 μg/m^3 environments,[22] with PM levels of >250 μg/m being considered 'hazardous' for health. It was found that the overall mean PM 2.5 value for the visited venues was 846 μg/m^3.[23] This finding correlates with high exposure rate found in our study. There is no risk-free level of exposure to SHS and even short-term exposure can potentially increase the risk of severe diseases.

Smoking in front of others is a harmful practice. Infact, the prevalence and hazards of passive smoking are more lethal as its burden is shared by all groups in a community, including infants, pregnant women, innocent children and all non-smokers. Passive smokers are at risk of acquiring all those illnesses to which active smokers are exposed, like ischaemic heart disease, stroke, lower respiratory infections, asthma and lung cancer. A study conducted on rural community in Islamabad showed that the risk of having respiratory illness is higher in females when the husband is a smoker and the risk increases four times if the husband smokes at home.[24] Pregnant women exposed to SHS are 23 percent more likely to experience stillbirth and 13 percent more likely to have children with congenital malformations, in addition to their children's increased risks for low birth weight and Sudden Infant Death Syndrome.[25] Such Infants are also prone to developing respiratory diseases. According to WHO

recommendations 2013,[26] healthcare providers should engage directly with household members to promote reduction of exposure of SHS to pregnant women and offer smoking cessation support. Children are more susceptible to acquire diseases related to ETS exposure.[27] According to the Global School-based Student Health Survey (2006-13) and the China Global Tobacco Youth Survey (2013),[28] the overall prevalence of SHS exposure was 55·9%. Parental tobacco use, as reported by the young adolescents, especially maternal use, was associated with tobacco use in young adolescents. SHS is thus, not only hazardous for children, but also promote smoking habits among them.

According to a study done in Bangladesh,[29] 55% of households had at least one regular smoker and in 30% of households, smoking occurred in the presence of children, exposing nearly 40% of children to SHS. That study also mentioned about lack of awareness about the harms associated with SHS. A number of studies have been done assessing the awareness about hazards of passive smoking.[30] According to a cross-sectional survey to assess awareness and attitudes towards passive smoking, conducted among consenting working adults from urban areas in Malaysia in 2014,[31] 90 per cent adults agreed that cigarette smoke is harmful to those around the smoker and that children are more vulnerable to passive smoking than do adults though the practices are poor. In most parts of the world, there exists a discrepancy between thoughts and practices regarding passive smoking. Similarly in our study, 295(96.1%) people were aware of the hazards associated with passive smoking. Majority of people, despite having knowledge and awareness about smoking and its effect on passive smokers, still practised smoking in front of their family members and colleagues. There is actually a need to spread awareness among smokers and encourage them to stop this practice for the sake of health of their family members and themselves, and to advise passive smokers to protect themselves from this environmental smoke.

Although smoking in closed spaces seems to be more hazardous than smoking in an open and well-ventilated environment, as concentration of smoke present in a particular environment relates to its noxious effects, but there is no established evidence to support this. This is because conventional air cleaning systems can remove large particles. But not the smaller particles or the gases found in SHS and current heating, ventilating and air-conditioning systems alone do not control SHS exposure. As suggested by our results, there was no significant association between the status of ventilation and the existence of COPD. In the light of available scientific evidence on ventilation, in 2007 WHO made the recommendation to protect workers and the public from exposure to SHS by applying 100% smoke-free environments.[32] This is the only operative policy to reduce exposure to tobacco smoke in indoor environments. Ventilation cannot reduce exposure to a harmless level of risk.

The respiratory system is the portal of entry for SHS and one of the key systems at risk for damage by SHS. Several studies established causal relationship between passive smoking and obstructive lung disease,[33-35] some also documented decline in pulmonary functions in relation to passive smoke exposure.[36] In a cross-sectional analysis of data from 3 years of the Health Survey for England, marked dose-response relationships were observed between passive smoking exposure and respiratory symptoms, but the most marked effects were on the development of clinically significant COPD, where the risk among never-smokers was doubled if exposure exceeded 20 h/week.[37] In another study conducted on females in Syria, passive smoking was associated with FEV1/FVC<70% after bronchodilators, which confirms the role of passive smoking in airway obstruction.[38]

Though we enrolled our subjects from different age groups, residence and educational levels, by involving attendants of patients coming in the OPD, the single-centre nature is our study's limitation. Moreover, other respiratory, including bronchial asthma, and cardiovascular disorders which can influence spirometric interpretations were excluded on the basis of history rather than definitive screening investigations. Quantification of exposure to passive smoking was also subject-dependent rather than through objective assessment.

Conclusion

Passive smoke exposure is an independent risk factor for developing COPD and passive smokers are at similar risk like active smokers. There is a need to screen them also, particularly if they are exposed on a regular basis for a certain period of time. Spirometry should be introduced for screening purposes for all passive smokers.

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Conflict of Interest: None.

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References

1. Changing the burden of copd mortality. Int J Chron Obstruct Pulmon Dis. 2006; 1:219-33.
2. Salvi SS, Barnes PJ. Chronic obstructive pulmonary disease in non-

smokers. Lancet.2009; 374: 733-43.
3. Cerveri I, Accordini S, Verlato G. European Community Respiratory Health Survey (ECRHS) Study Group. Variations in the prevalence across countries of chronic bronchitis and smoking habits in young adults. Eur Respir J.2001; 18:85-9.
4. Yin P, Zhang M, Li YC. Prevalence of COPD and its association with socioeconomic status in China: findings from China Chronic Disease Risk Factor Surveillance 2007. BMC Public Health.2011;11:586-93.
5. WHO | Second-hand tobacco smoke. Who.int. [Online] 2018 [Cited 2018 March 21]. Available from URL: http://www.who.int/tobacco/research/secondhand_smoke/en/
6. Passive smoking En.wikipedia.org. [Online] 2018 [Cited 2018 June 11]. Available from URL: https://en.wikipedia.org/wiki/Passive_smoking
7. Ayres JG, Semple S, MacCalman L, Dempsey S, Hilton S, Hurley JF, et al. Bar workers' health and environmental tobacco smoke exposure (BHETSE): symptomatic improvement in bar staff following smoke-free legislation in Scotland. Occup Environ Med. 2009; 66:339-46.
8. Comstock GW, Meyer MB, Helsing KJ, Tockman MS. Respiratory effects of household exposures to tobacco smoke and gas cooking. Am J Epidemiol. 2008; 168:810-5.
9. Fischer F, Kraemer A. Meta-analysis of the association between second-hand smoke exposure and Ischemic heart diseases, COPD and stroke. BMC Public Health. 2015; 15:1202.
10. CDC - Fact Sheet - Health Effects of Secondhand Smoke - Smoking & Tobacco Use .Smoking and Tobacco Use. [Online] 2018 [Cited 2018 March 14]. Available from: URL: https://www.cdc.gov/tobacco/data_statistics/fact_sheets/secondhand_smoke/health_effects/index.htm
11. Zhang J, Lin XF, Bai C. Comparison of clinical features between non-smokers with COPD and smokers with COPD: a retrospective observational study. Int J Chron Obstruct Pulmon Dis. 2014; 9:57-63.
12. Svanes C, Omenaas E, Jarvis D. Parental smoking in childhood and adult obstructive lung disease: results from the European Community Respiratory Health Survey. Thorax. 2004; 59:295-302.
13. Yin P, Jiang CQ, Cheng KK, Lam TH, Lam KH, Miller MR, et al. Passive smoking exposure and risk of COPD among adults in China: the Guangzhou Biobank Cohort Study. Lancet. 2007; 370: 751-7.
14. Habibullah S, Soomro IB. Effect of smoking on the health of family members in District South Karachi. Pak J Med Res. 2009; 48:44-7.
15. Mal R, Rizvi N, Rathi S. Prevalence of Smokers among Air-Conditioned Coaches, Pakistan. J Pak Med Assoc. 2001; 51:405-6.
16. Biomedical Instrumentation| Digital Textbook Library. Tankonyvtar.hu. [Online] 2018 [Cited 2018 March 23]. Available from: URL: http://www.tankonyvtar.hu/en/tartalom/tamop412A.
17. Global Initiative for Chronic Obstructive Lung Disease. Pocket guide to COPD Diagnosis Management and Prevention. Amazon: Global Initiative for Chronic Obstructive Lung Disease; 2016.
18. Stenton C. The MRC breathlessness scale. Occup Med. 2008; 58:226-27.
19. Öberg M, Jaakkola MS, Woodward A, Peruga A, Prüss-Ustün A. Worldwide burden of disease from exposure to second-hand smoke: a retrospective analysis of data from 192 countries. Lancet. 2011;377:139-46.
20. Lamprecht B, McBurnie MA, Vollmer WM. COPD in never smokers. Results from the Population-based Burden of Obstructive Lung Disease Study. Chest. 2011; 139:752-63.
21. Saqib MAN, Rafique I, Qureshi H, Munir MA, Bashir R, Arif BW, et al. Burden of Tobacco in Pakistan - Findings from Global Adult Tobacco Survey 2014. Nicotine Tob Res. 2017.
22. What are the Air Quality Standards for PM? | Air Quality Planning Unit | New England | US EPA [Internet]. Www3.epa.gov.[Online] 2018 [Cited 21 August 25]. Available from: URL: https://www3.epa.gov/region1/airquality/pm-aq-standards.html
23. Zaidi SM, Moin O, Khan JA. Second-hand smoke in indoor hospitality venues in Pakistan Int J Tuberc Lung Dis. 2011; 15:972-7.
24. Hammad M, Atta K, Manzoor M, Tariq M, Saeed Z, Masood Z, et al. Association of passive smoking with respiratory symptoms and clinical correlates, among married women, in a rural community in Islamabad. J Pak Med Assoc. 2010; 60:601-4.
25. Hofhuis W, de Jongste JC, Merkus PJ. Adverse health effects of prenatal and postnatal tobacco smoke exposure on children. Arch Dis Child. 2003; 88: 1086-90.
26. WHO recommendations for the prevention and management of tobacco use and second-hand smoke exposure in pregnancy. World Health Organization. [Online] 2018 [Cited 2018 Dec 18]. Available from: URL: http://www.who.int/tobacco/publications/pregnancy/guidelinestobaccosmokeexposure/en/.
27. Treyster Z, Gitterman B. Second hand smoke exposure in children: environmental factors, physiological effects, and interventions within pediatrics. Rev Environ Health. 2011; 26:187-95.
28. Xi B, Liang Y, Liu Y, Yan Y, Zhao M, Ma C, et al. Tobacco use and second-hand smoke exposure in young adolescents aged 12-15 years: data from 68 low-income and middle-income countries. Lancet Glob Health. 2016; 4:e795-e805.
29. Zafar Ullah AN, Huque R, Akter S, Nasreen S, Akter H, Thomson H, et al. Children's exposure to second-hand smoke at home in Bangladesh: a community survey. BMJ Open. 2013; 3:e003059.
30. O?berg M, Jaakkola M, Pru?ss-U?stu?n A, Schweizer C, Woodward A. Second-hand smoke. Geneva: World Health Organization; 2010.
31. Jia XO, Kai XT, Cai LT, Sivalal SA, Amudha KJ. Passive Smoking: Perceptions and Practices among Urban Working Adults. Inter J Collabo Res Internal Med Public Health. 2014; 6.
32. WHO | Only 100% smoke-free environments adequately protect from dangers of second-hand smoke. Who.int. [Online]2018 [cited 2017 Sep 29]. Available from: URL: http://www.who.int/mediacentre/news/releases/2007/pr26/en/
33. Pelicaric D, Petanjek BB, Gluncic TJ, Bogdan M. COPD among non-smokers. Eur Respir J. 2012; 40:P3999.
34. Role of passive smoking on COPD risk in non-smokers. Lancet. 2007; 370:716-17.
35. Zeng G, Sun B, Zhong N. Non-smoking-related chronic obstructive pulmonary disease: a neglected entity? Respirology. 2012; 17:908-12.
36. Khan A, Shabbir K, Ansari JK, Zia N. Comparison of forced expiratory volume in one second (FEV1) among asymptomatic smokers and non-smokers. J Pak Med Assoc. 2010; 60:209-13.
37. Jordan RE, Cheng KK, Miller MR, Adab P. Passive smoking and chronic obstructive pulmonary disease: cross-sectional analysis of data from the Health Survey for England.BMJ Open. 2011; 1:e000153.
38. Yousser M, Rafea S, Bassam AZ, Nikolai K, Jean B, Basim D. Impact of active and passive smoking as risk factors for asthma and COPD in women presenting to primary care in Syria: first report by the WHO-GARD survey group. Int. 2013; 8: 473-82 Int J Chron Obstruct Pulmon Dis. 2013; 8:473-82.