

Effects of passive smoking on students at College of Applied Medical Sciences, King Saud Bin Abdulaziz University for Health Sciences, Riyadh

Abdullah Alanazi¹,
Farhan Al Enezi²,
Mohammed
Mesfer Alqahtani³,
Turki Faleh Alshammari⁴,
Mumtaz Ahmed Ansari⁵,
Saleh Al-Oraibi⁶,
Shoeb Qureshi⁷

¹Emergency Medical Services Department, ^{2,3,4}Respiratory Therapy Department, ⁵Department of Medicine, ⁶Occupational Therapy Department, ⁷Academic and Research Department, ^{1-4,6,7}College of Applied Medical Sciences, King Saud Bin Abdul-Aziz University for Health Sciences, ⁵King Fahad Security College, Riyadh, Saudi Arabia

Address for correspondence:

Dr. Shoeb Qureshi, College of Applied Medical Sciences, King Saud Bin Abdul-Aziz University, National Guards, P.O. Box 3660, Riyadh - 11481, Saudi Arabia. E-mail: qsab2002@yahoo.co.in

Abstract

Background: Despite the recent campaigns to eliminate smoking, the rates are still increasing world-wide. Exposure to passive smoking (PS) is associated with morbidity and mortality from awful diseases. Although many college students smoke, little is known about their exposure to PS, common places and sources of exposures in Saudi Arabia. **Aim:** The aim of the following study is to identify prevalence and magnitude of PS among college students, exposure time, locations, sources of exposure, investigate the effects and make recommendations. **Materials and Methods:** A cross-sectional study was performed to identify factors associated with PS exposure among students of College of Applied Medical Sciences, Riyadh. **Results:** Out of 61 students included in the study, 91.8% were found exposed to PS. Exposure in Hospitality venues (Estirah) was the most common followed by other areas. Among the sources of exposure, the highest was among friends and the least were parents and guests. The frequency of highest exposure per month was >15 times and the lowest was 10-15 times. Levels of annoyance varied between 18% and 37.7%, respectively. Since the values obtained for different markers in the pulmonary function test are more than the predicted values, the observed spirometry is normal. The percent oxygen saturation in hemoglobin and blood pressure of PS were in normal range. **Conclusion:** Since the properties of mainstream smoke and environmental tobacco smoke are quite different, risk extrapolation from active to PS is uncertain, especially during a short period. Nevertheless, it can be deteriorating during a longer duration, hence; the administrators, policy makers and tobacco control advocates may endorse policies to restrict smoking in shared areas, particularly working environment.

Key words: Biological effects, College of Applied Medical Sciences, exposure, passive smoking, students

INTRODUCTION

Tobacco smoke contains more than 4000 chemical substances including particulates and gases.^[1,2] The reported constituents in the cigarette smoke are nicotine,

polycyclic aromatic hydrocarbons (PAH), nitrated PAH, benzo[a]pyrene, thiocyanate, nitro-lactones, fluoranthene, pyrene, benzo[a]anthracene, chrysene, benzo(g,h,i)perylene, dibenz(a,h)anthracene; aromatic amines.^[3-7] Most of these constituents are toxic, mutagenic and carcinogenic and are perhaps the most significant source of deadly chemical exposure and chemical-mediated diseases, such as; cardiovascular disease, chronic obstructive pulmonary diseases (COPD) and various types of cancers, mainly lung cancer in humans and according to an estimate of WHO, 5.4 million premature deaths are attributable to tobacco smoking throughout the world.^[8-11] The toxic constituents present in tobacco smoke are also present in the environmental tobacco

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smoke (ETS) and become an indirect source to the nonsmokers.^[12]

Passive smoking (PS) or second hand smoke (SHS) or ETS is defined as the inhalation of tobacco smoke by nonsmokers against their will or as being the involuntary exposure to tobacco smoke.^[13] Ishimine^[14] have reported that PS consists of 15% mainstream smoke and 85% side stream smoke. Mainstream smoke is the smoke discharged by expiration after being filtered through the smoker's lungs, while side stream smoke is the smoke that goes directly into the air from a burning cigarette. Passive smoke contains >50 chemicals identified as known and/or probable human carcinogens and many toxic and irritant agents.^[15] Globally, 40% of children, 33% of male nonsmokers and 35% of female nonsmokers were exposed to PS.^[16] Studies have also reported that nonsmokers with PS exposure have a 2.1 times greater risk of developing lung cancer compared with those without PS exposure.^[17] Evidence linking PS to adverse health outcomes (respiratory diseases, cardiovascular effects and lung cancer) has accumulated over the past two decades.^[15,18] Furthermore, exposure to PS is shown as one of the causes for early deaths.^[19]

Literature reports document many researches on exposure to PS from developed countries. However; despite of a different life-style and cultural back ground, there is a paucity of literature on exposure to PS among Saudis, especially the students. Unmindful of the impact of PS many of the PS suffer serious deteriorating effects on their health. Hence, it was found imperative to focus the effects of PS among the students of College of Applied Medical Sciences (CAMS) and highlight the possible deterrent steps that can be taken.

MATERIALS AND METHODS

A cross-sectional study was performed to identify factors associated with PS exposure among college students in Saudi Arabia. Data was collected from the students of CAMS at King Saud Bin Abdul-Aziz University for Health Sciences in Riyadh, Saudi Arabia.

Study design

The data from non-smokers was collected by a questionnaire described by Jaakkola and Jaakkola,^[20] which included locations, sources, frequency of exposure to PS, besides, annoyance due to smoke. Medical tests were conducted to measure breath, saturation of the hemoglobin and blood pressure. The parameters used for medical tests are (1) spirometry, which is the most common to conduct the pulmonary function test (PFT), specifically the amount (volume) and/or speed (flow) of air that can be inhaled and exhaled. The method of Cooper and Mitchell^[21]

was used to assess the lung function test by spirometry (2) pulse oximeter to monitor the saturation of a patient's hemoglobin. The method devised by Jørgensen *et al.*,^[22] and Brand *et al.*,^[23] were used. (3) Sphygmometer was used to measure the blood pressure.^[24]

Data collection

Questionnaires were distributed to the nonsmokers in CAMS after obtaining their consent. The respondents completed the questionnaires and submitted to the concerned researchers.

RESULTS

Data from 61 students were included in this study. We found about 56 students (91.8%) out of 61 were exposed to PS during the last 2 months of questionnaire administration. Exposure in Estirah was the most common place reported by 67.2%, followed by exposure in the University Campus (39.3%), coffee shop (29.5%), public area (26.2), car (21.3%) and home (14.8%) [Figure 1]. Among the sources of exposure, the highest was among friends (89%) followed by brothers (16%), teachers (13%), parents (11%), guests and other persons (9%, each category) [Figure 2]. The frequency of exposure per month showed the highest was >15 times (32.8%) followed by 1-5 times (29.3%), 5-10 times (17.2%), 10-15 times (15.5%) [Table 1]. The levels of annoyance varied between 18, 44.3 and 37.7 (low, average and maximum levels, respectively [Table 2]. The results obtained in the PFT showed normal spirometry [Table 3]. The results were computed by comparing the predicted values for forced expiratory volume in 1st s/forced vital capacity (FEV1/FVC) (<70%) and FEV1 (50-80%) which were less than the values of different markers. Monitoring of saturation of patient's hemoglobin revealed, majority had normal range of saturation (97-100%) [Figure 3] and measurement of blood pressure demonstrated normal systolic and diastolic blood pressure in almost all the students [Figure 4].

DISCUSSION

Direct smoking is a recognized source of exposure to toxic, mutagenic and carcinogenic chemicals and is

Table 1: Frequency of exposure to PS

Exposure frequency	Frequency	Percentage
1-5 times/month	17	29.3
5-10 times/month	10	17.2
10-15 times/month	9	15.5
More than 15 times/month	19	32.8
Other	3	5.2
Total	58	100.0

PS: Passive smoking

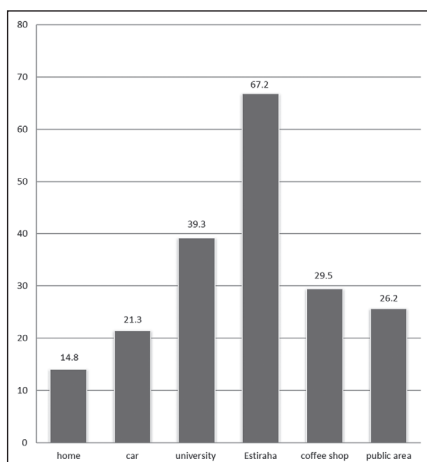


Figure 1: Most common places of exposure to passive smoking

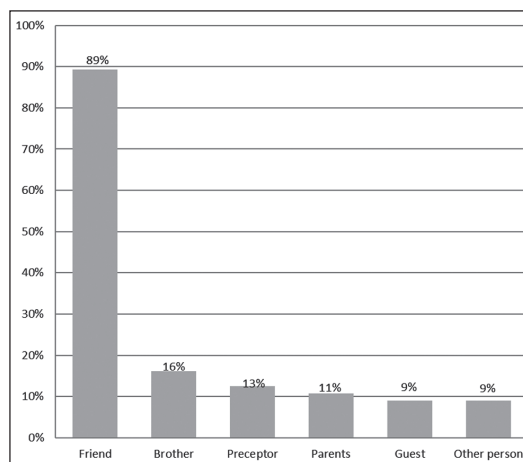


Figure 2: Common source of exposure to passive smoking

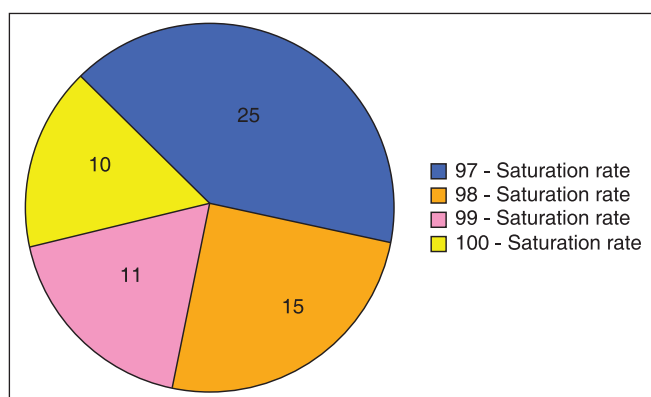


Figure 3: Oxygen saturation (%) in hemoglobin of PS

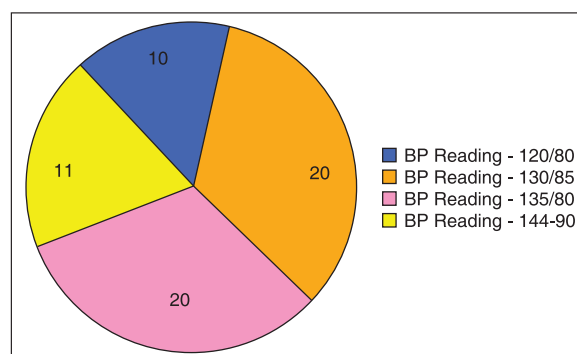


Figure 4: Sphygmometer readings

renowned primary etiology for several diseases, including cardiovascular disease, COPD and various types of cancers, mainly lung cancer in humans. The toxic ingredients present in tobacco smoke are also present in the ETS and become an indirect source to the nonsmokers.^[12] Literature reports suggest PS contains chemicals identified as known human carcinogens, toxicants and irritants.^[15] Evidence linking PS to adverse health outcomes (respiratory diseases, cardiovascular effects and lung cancer) has accumulated over the past two decades.^[15,18]

Nevertheless, results of chemical analysis, animal experiments and human studies are reviewed, criticized and found not to support claims of an association between PS and direct smoking. It is argued that the data on constituents of ETS (nicotine, carbon monoxide, benzo[a] pyrene and carbon disulfide) indicate that the levels of these substances arising from ETS are far below their respective permissible exposure limits.^[25] Macdonald^[26] in his study has reported that there is no link between PS and lung cancer. Furthermore, the author has shown that people who are married to work with and grew with smokers are found to have no threat of the effects of PS. Thus the reports

Table 2: Annoyance level in response to PS

Annoyance variance	Frequency	Percentage
Not at all annoying	11	18.0
Somewhat annoying	27	44.3
Very annoying	23	37.7
Total	61	100

PS: Passive smoking

Table 3: Effect of PS determined by the PFT

Markers of PFT	Exposure frequency per month	Mean (%)
FEV1	1-5	90.75
	5-10	81.80
	10-15	78.75
	>15	89.83
	Mean	85.28
FVC	1-5	89.08
	5-10	82.40
	10-15	81.75
	>15	85.33
	Mean	84.64
Ratio between FEV1 and FVC	1-5	101.81
	5-10	99.85
	10-15	95.74
	>15	105.43
	Mean	100.71

Predicted values for FEV₁/FVC is <70% and FEV₁ is 50-80%. Since the values in the table for the different markers are more than the predicted values, the observed spirometry is normal. FEV₁: Forced expiratory volume in 1st s, FVC: Forced vital capacity, PS: Passive smoking, PFT: Pulmonary function test

appearing in the literature on possible similarity between passive and direct smoking are controversial. This became one of the bases for the conduct of present study.

This investigation was conducted by administering questionnaire which involved some of the very crucial parameters. The question relating to the places of exposure, majority of the PS were positive about Estirah followed by university campus, coffee shop, public area, car and home. These results are in agreement with the study of Trotter *et al.*,^[27] who found majority of the Victorians were exposed to ETS at home and work place. Cigarette smoking is reported to be intertwined in American cultural landscape besides; it is found rampant in social gatherings, coffee breaks, seminars, restaurants, boardroom, and bed room and in interactions during social gatherings. Furthermore, people also smoke during traveling by car, train and airplane.^[15,28] A study by Hitchman *et al.*,^[29] also confirmed that smoking in cars produces high levels of tobacco smoke pollution. Household, work place and social settings were described as the places of lifetime exposure to PS.^[30] Wolfson *et al.*,^[31] showed that the overall campus smoking rate was positively associated with reported exposure in cars, at home or in someone's room. Cummings *et al.*,^[32] also showed home and work place to be the most vulnerable places for exposure. López *et al.*,^[33] have reported that outdoor (terraces, areas of hospitality venues) SHS concentrations are usually lower than indoor concentrations, yet some studies have shown that outdoor SHS levels could be comparable to indoor levels under specific conditions. Regarding the sources of exposure, majority of the PS opted for friends, while the other sources including brothers, cousins, teachers, parents, guests and other persons were much less. This observation is in corroboration with the findings of Cummings *et al.*,^[32] who found friends and immediate family members were the most vulnerable source of exposure. The study of Reynolds *et al.*,^[34] also found maximum ETS exposures at home, followed by work place and other social settings, which subsequently changed to the peak as the spousal smoking and followed by work place and the household. In a study on control of adolescent smoking in seven European countries, Wold *et al.*,^[35] showed that students are exposed to teachers smoking during school hours. Another question was about the frequency of exposure to know how often the PS was exposed (1-5 times/month to >15 times/month), the results showed >15 times/month to be the highest followed by 1-5 times/month. Different health outcomes are linked to different time periods of exposure. The respiratory symptoms are related to repeated high peak exposure levels, while lung cancer may require cumulative exposure over long time periods. Exposure to ETS may cause acute exacerbation of asthma or development of lung cancer detected 10-20 years later.^[36] On the contrary, the duration and the exposure times in the present study

are very less and correspond with the intensity of effects observed.

On determination of the level of annoyance, only a limited number of PS showed tolerance to the smoke, while majority revealed that the smoke annoys them either to some extent or to extreme. Literature reports suggest that the negative impact of ETS is its odor, which is unpleasant for many PS. The smell is related mainly to vapor phase which develops intolerance in many people. In addition to the smell the smoke has been found to be allergic, irritant to eyes and nose and causes headache and cough to some of the PS.^[37,38]

The values obtained for different markers in the PFT are found more than the predicted values for FEV1/FVC (<70%) and FEV1 (50-80%), the observed spirometry is normal showing no indication of any respiratory disease. Our results are not in agreement with reports available in the literature. Exposure to tobacco smoke at home is known to cause airway inflammation and altered cytokine regulation and has been identified as a cause of premature death and disease in nonsmokers.^[29] Exposure to ETS has been linked to a broad array of diseases, including asthma, COPD, Cardiovascular disease and cancer.^[39] Exposure to ETS, at work place, during the period June to December, adversely affects pulmonary function in adults.^[40] Rizzi *et al.*,^[41] in their study have reported exposure to ETS is associated with lung function impairment. Although adverse effects of PS are biologically plausible, it remains controversial, whether ETS exposure is linked with chronic respiratory symptoms and occurrence of COPD, including asthma.^[42] For the purpose of risk evaluation, PS is often regarded as low-dose cigarette smoking. Nevertheless, since the physical, chemical and biological properties of mainstream smoke, inhaled by the smoker and ETS, which is breathed by the passive smoker, are quite different, risk extrapolation from active smoking to PS is uncertain.^[25]

Determined by the pulse oximeter, the percent oxygen saturation in hemoglobin of PS was in the normal range. The seasoned smokers are known to have lower blood oxygenation levels, as the smoke inhaled damages the biological mechanisms needed to carry oxygen through the blood stream.^[43] The normal range obtained in the present study of PS is ascribed to a very short duration of exposure where the smoke was inhaled was much diluted.^[25] The blood pressure of the PS was recorded normal. Blood pressure profile is relevant to the disturbances in the heart rate variability which is one pathway by which the second hand smoke and air pollutants affect cardiovascular morbidity and mortality.^[44] Felber Dietrich *et al.*,^[45] in their study have reported exposure to ETS influence the rate variability and is a predictor of increased cardiac risk. Taken

together, the observed negative effects of PS observed in the present study are attributed to short duration of exposure with a limited frequency.

CONCLUSION

Since the properties of mainstream smoke and ETS are quite different, risk extrapolation from active to PS is uncertain, especially during a short period. Nevertheless, it can be deteriorating during a longer duration, hence; the administrators, policy makers and tobacco control advocates may endorse policies to restrict smoking in shared areas, particularly working environment.

SUGGESTIONS

1. In view of the health risks of many of chemical substances present in the tobacco smoke, studies are needed to determine biomarkers of exposure to ETS constituents in body fluids, in addition to saliva, urine and blood^[2] of people who actively or passively are exposed to the toxic compounds. Active researches into discovery of these methodologies are required.
2. Quantitative Structure Toxicity Relationship may be included in the battery of tests to identify chemicals in the ETS with a high probability of being toxic, mutagenic and/or carcinogenic before undertaking the biological toxicity assays.
3. The use of an innovative tobacco-substitute and smoke dilution to reduce toxicant impact in cigarette smoke will diminish toxicity burden on the PS. Hence, more reviews of literature on the subject and promotion of experimental cigarettes have to become part of the literature.

REFERENCES

1. Merghani TH, Saeed A, Alawad A. Changes in plasma IL4, TNF α and CRP in response to regular passive smoking at home among healthy school children in Khartoum, Sudan. *Afr Health Sci* 2012;12:41-7.
2. Narkowicz S, Polkowska Z, Marć M, Simeonov V, Namieśnik J. Determination of thiocyanate (biomarkers of ETS) and other inorganic ions in human nasal discharge samples using ion chromatography. *Ecotoxicol Environ Saf* 2013;96:131-8.
3. Lewtas J. Air pollution combustion emissions: Characterization of causative agents and mechanisms associated with cancer, reproductive, and cardiovascular effects. *Mutat Res* 2007; 636:95-133.
4. Neal MS, Zhu J, Foster WG. Quantification of benzo[a]pyrene and other PAHs in the serum and follicular fluid of smokers versus non-smokers. *Reprod Toxicol* 2008;25:100-6.
5. Leone A, Landini L, Leone A. What is tobacco smoke? Sociocultural dimensions of the association with cardiovascular risk. *Curr Pharm Des* 2010;16:2510-7.
6. Lodovici M, Akpan V, Caldini S, Akanju B, Dolara P. DNA solution(R) in cigarette filters reduces polycyclic aromatic hydrocarbon (PAH) levels in mainstream tobacco smoke. *Food Chem Toxicol* 2007;45:1752-6.
7. Riedel K, Scherer G, Engl J, Hagedorn HW, Tricker AR. Determination of three carcinogenic aromatic amines in urine of smokers and nonsmokers. *J Anal Toxicol* 2006;30:187-95.
8. Ezzati M, Lopez AD. Estimates of global mortality attributable to smoking in 2000. *Lancet* 2003;362:847-52.
9. Fowles J, Dybing E. Application of toxicological risk assessment principles to the chemical constituents of cigarette smoke. *Tob Control* 2003;12:424-30.
10. The Health Consequences of Smoking: A Report of the Surgeon General. Atlanta, GA, USA: U.S. Department of Health and Human Services; 2004. p. 1-910.
11. WHO Report on the Global Tobacco Epidemic: The MPOWER Package. Geneva, Switzerland: WHO; 2008. p. 1-329.
12. Talhout R, Schulz T, Florek E, van Benthem J, Wester P, Opperhuizen A. Hazardous compounds in tobacco smoke. *Int J Environ Res Public Health* 2011;8:613-28.
13. Palazzi DL. Etiologies of fever of unknown origin in children. *UpToDate*; 2012. p. 1-21. Available from: <http://www.uptodate.com/contents/etiologies-of-fever-unknown-origin-in-children?vsie>.
14. Ishimine P. Fever without source in children 0 to 36 months of age. *Pediatr Clin North Am* 2006;53:167-94.
15. U.S. Department of Health and Human Services. The Health Consequences of Involuntary Exposure to Tobacco Smoke: A Report of the Surgeon General. Rockville, MD: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, Coordinating Center for Health Promotion, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health; 2006.
16. Oberg 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.
17. Demir F, Sekreter O. Knowledge, attitudes and misconceptions of primary care physicians regarding fever in children: A cross sectional study. *Ital J Pediatr* 2012;38:40.
18. IARC Working Group on the Evaluation of Carcinogenic Risks to Humans. Tobacco smoke and involuntary smoking. *IARC Monogr Eval Carcinog Risks Hum* 2004;83:1-1438.
19. Wipfli H, Avila-Tang E, Navas-Acien A, Kim S, Onicescu G, Yuan J, *et al.* Secondhand smoke exposure among women and children: Evidence from 31 countries. *Am J Public Health* 2008;98:672-9.
20. Jaakkola MS, Jaakkola JJ. Assessment of exposure to environmental tobacco smoke. *Eur Respir J* 1997;10:2384-97.
21. Cooper K, Mitchell P. Procedure for the assessment of lung function with spirometry. *Nurs Times* 2003;99:57-8.
22. Jørgensen JS, Schmid ER, König V, Faisst K, Huch A, Huch R. Limitations of forehead pulse oximetry. *J Clin Monit* 1995;11:253-6.
23. Brand TM, Brand ME, Jay GD. Enamel nail polish does not interfere with pulse oximetry among normoxic volunteers. *J Clin Monit Comput* 2002;17:93-6.
24. Booth J. A short history of blood pressure measurement. *Proc R Soc Med* 1977;70:793-9.
25. Aviado DM. Cardiovascular disease and occupational exposure to environmental tobacco smoke. *Am Ind Hyg Assoc J* 1996;57:285-94.
26. Macdonald V. Passive smoking doesn't cause cancer. *UK Sunday Telegraph* [13 November 2002]. <http://www.freerepublic.com/focus/fr/788186/posts>
27. Trotter L, Mullins R, Freeman J. Key findings of the 1998 and 1999 population surveys. In: Trotter L, Letcher T, editors. *Quit Evaluation Studies No. 10. Victorian Smoking and Health Program*. Melbourne: Centre for Behavioral Research in Cancer; 2000.
28. Brandt AM. Blow some my way: Passive smoking, risk and American culture. In: Lock S, Reynolds L, Tansey EM, editors. *Ashes to Ashes: The History of Smoking and Health*. Amsterdam, Atlanta, GA: Rodopi; 1998. p. 164-91.
29. Hitchman SC, Guignard R, Nagelhout GE, Mons U, Beck F, van den Putte B, *et al.* Predictors of car smoking rules among smokers in France, Germany and the Netherlands. *Eur J Public Health* 2012;22 Suppl 1:17-22.
30. Lu Y, Wang SS, Reynolds P, Chang ET, Ma H, Sullivan-Halley J, *et al.* Cigarette smoking, passive smoking, and non-Hodgkin lymphoma

- risk: Evidence from the California Teachers Study. *Am J Epidemiol* 2011;174:563-73.
31. Wolfson M, McCoy TP, Sutfin EL. College students' exposure to secondhand smoke. *Nicotine Tob Res* 2009;11:977-84.
 32. Cummings KM, Markello SJ, Mahoney M, Bhargava AK, McElroy PD, Marshall JR. Measurement of current exposure to environmental tobacco smoke. *Arch Environ Health* 1990;45:74-9.
 33. López MJ, Fernández E, Gorini G, Moshammer H, Polanska K, Clancy L, *et al.* Exposure to secondhand smoke in terraces and other outdoor areas of hospitality venues in eight European countries. *PLoS One* 2012;7:e42130.
 34. Reynolds P, Goldberg DE, Hurley S, California Teachers Study Steering Committee. Prevalence and patterns of environmental tobacco smoke exposures among California teachers. *Am J Health Promot* 2004;18:358-65.
 35. Wold B, Torsheim T, Currie C, Roberts C. National and school policies on restrictions of teacher smoking: A multilevel analysis of student exposure to teacher smoking in seven European countries. *Health Educ Res* 2004;19:217-26.
 36. Lebowitz MD. Evaluation of peak expiratory flow variability in an adolescent population sample. *Am J Respir Crit Care Med* 1995;151 3 Pt 1:919-20.
 37. Speer F. Tobacco and the nonsmoker. A study of subjective symptoms. *Arch Environ Health* 1968;16:443-6.
 38. Byrd JC, Shapiro RS, Schiedermayer DL. Passive smoking: A review of medical and legal issues. *Am J Public Health* 1989;79:209-15.
 39. 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;4:7.
 40. Jung YR, Youn CH, Ko HJ. Effects of passive smoking on pulmonary function in adults. *Korean J Health Promot* 2011;11:115-21.
 41. Rizzi M, Sergi M, Andreoli A, Pecis M, Bruschi C, Fanfulla F. Environmental tobacco smoke may induce early lung damage in healthy male adolescents. *Chest* 2004;125:1387-93.
 42. Trédaniel J, Boffetta P, Saracci R, Hirsch A. Exposure to environmental tobacco smoke and adult non-neoplastic respiratory diseases. *Eur Respir J* 1994;7:173-85.
 43. Witting MD, Scharf SM. Diagnostic room-air pulse oximetry: Effects of smoking, race, and sex. *Am J Emerg Med* 2008;26:131-6.
 44. Probst-Hensch NM, Imboden M, Felber Dietrich D, Barthélemy JC, Ackermann-Liebrich U, Berger W, *et al.* Glutathione S-transferase polymorphisms, passive smoking, obesity, and heart rate variability in nonsmokers. *Environ Health Perspect* 2008;116:1494-9.
 45. Felber Dietrich D, Schwartz J, Schindler C, Gaspoz JM, Barthélemy JC, Tschopp JM, *et al.* Effects of passive smoking on heart rate variability, heart rate and blood pressure: An observational study. *Int J Epidemiol* 2007;36:834-40.

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