



Burden of Household Smoking Habits on the Occurrence of Respiratory Symptoms among Malaysian Adolescents

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Abstract

Background: This study aimed to assess the burden of household smoking habits on the occurrence of respiratory symptoms among young adolescents in Malaysia.

Methodology: The impact of Secondhand Smoke (SHS) exposures on the occurrence of respiratory symptoms was investigated on 234 adolescents (13-14 years old) residing in two states in Malaysia. Adolescents completed a self-administered questionnaire adapted from Global Youth Tobacco survey that comprised of items on socio-demographic and SHS exposures. Respiratory symptoms were assessed using a standard questionnaire of International Study of Asthma and Allergies in Childhood. Hair samples were collected from each adolescent and were analyzed for nicotine via Gas Chromatography-Mass Spectrophotometry with nitrogen detection method.

Result: The geometric mean of hair nicotine was 0.0184 ± 2.31 ng/mg. The prevalence of current, ever wheeze, nocturnal cough, exercised-induced wheeze and self-reported asthma were 7.69%, 14.1%, 11.5%, 20.5% and 13.7%, respectively. A significant association was found between household smoking status with exercise-induced wheeze (Adjusted Odds Ratio, AOR 1.06; 95%Confidence Interval, CI 0.42-2.66) and nocturnal cough (AOR 14.2; 95%CI 1.60-125). Reported ever wheeze was linked to reported SHS exposure in family's vehicle (AOR 3.27; 95%CI 1.15-9.34). The risk of exercise-induced wheeze was related with postnatal SHS exposure (AOR 2.69; 95%CI 1.13-6.43).

Conclusion: Respiratory symptoms were mostly affected by household smoking habits. Thus, in an attempt to reduce the repercussion of SHS exposure among younger adolescents, effort should be emphasized in encouraging parents to move from indoor to outdoor smoking far from the vicinity of children.

Keywords: Passive smoking, Household smoking habits, Youth, Respiratory health, Hair nicotine

Introduction

Respiratory health is one of the largest public health concerns worldwide. The rising prevalence of respiratory morbidity demonstrated in industrialized regions of the world (1, 2) has suggested the contribution of environmental exposures in this scenario (3). Secondhand Smoke (SHS) has long been linked to ill-health effects and has been known as a respiratory irritant (4, 5).

Globally in 2011, SHS exposure was estimated to be responsible for an estimated 600,000 deaths among

non-smokers population (6). There are firm evidences that SHS exposure can result in wheeze (7), cough (8), impaired lung function (9), lower respiratory illness and asthma (10). Recent reviews clearly addressed the contribution of SHS exposure towards the occurrence of respiratory symptoms among younger adolescents population (11, 12).

As revealed in previous cross-sectional studies, the prevalence of smoking among Malaysian adults was 25% (13, 14). Although the prevalence of smoking

in Malaysia had reduced by 2.8% within 10 years (1996-2006), the decrement is relatively small when compared with other countries that have shown decline between the range of 9% to 25% (15, 16). This figure highlights the fact that SHS exposure among non-smoking population will remain and continuously occur.

Parental smoking habits have been found as the main contributor to SHS exposure among non-smoking household members namely children. Multiple studies have identified SHS exposures to mostly occur at home and have been the most important source of indoor SHS exposure (11, 17-18). In United States, 35% of children (21 million) lived in homes where exposure to SHS occurred on a regular basis where there are residents or visitors who smoke at least a day per week (19). In the context of Malaysia, about 57% of the adolescents were exposed to SHS at home (20). In addition, as revealed by Abidin and colleagues (2011) (14), among 947 Malaysian schoolchildren, more than half of them (54.1%) were living with at least one family member who smokes. Besides the presence of smoker in household, the smoking habit in the family's vehicle worsen the impact of SHS exposure. Majority of smoking parents tends to expose their children to tobacco smoke in cars (21) however; few data are available in Malaysia. In the west, despite the proven repercussion of SHS (9, 11), many parents overlook its dangers and smoke in their vehicles, thus exposing their children to high concentrations of SHS.

This study will provide further investigation regarding the specific burden of household smoking habits on the occurrence of respiratory symptoms among young adolescents in Malaysia.

Material and Methods

Study design and population

This cross-sectional study was conducted in 2013 in two states (Kedah and Melaka) in Malaysia among adolescents aged 13-14 years old. Approximately 18 secondary schools were randomly sampled from a list of secondary schools obtained from Education State Department (22). The students in form one and two (equivalent to eighth and ninth grade in Iran) were chosen as the study populations.

A total of 680 adolescents were invited into this study. Initially, envelopes containing parental questionnaire, parental permission form and research information sheets were disseminated to the adolescents. Parents were asked to fill in the parental permission form and those who allowed their children to be involved in the study were required to complete the parental questionnaire. Only 530 (77.9%) (Response rate) of envelopes were returned to the researcher on the next day. In total, 234 (44.2%) eligible adolescents who meet the inclusion criteria of 1) having hair at least 3 cm length and 2) hair was not premed, bleached or dyed in past 3 months were recruited in the study after obtaining parental permission.

Assessment on SHS exposures and respiratory symptoms

Two sets of questionnaires were used in this study in which it comprised of 1) a self-completed questionnaire for adolescent and 2) a household information request questionnaire for parents. A modified-version of Global Youth Tobacco Survey's (GYTS) questionnaire was used to collect SHS exposures information of adolescents while prenatal and postnatal SHS exposure data were obtained from the parental-completed questionnaire. Items on respiratory health were based on the International Study of Asthma and Allergies in Childhood (ISAAC) questionnaire (23). Questionnaire for adolescents was administered in their respective classrooms. In answering the respiratory health items, the paper questionnaire was supplemented with an ISAAC video questionnaire to assist adolescents in giving clearer picture of each symptom.

In this study, the adolescents were also asked to provide hair samples for the measurement of nicotine, a primary metabolite of tobacco that have been widely used in examining SHS exposure levels (24). An average of 30 strands of hair from the vortex posterior of the head was clipped with a clean stainless steel scissors as close as possible to the scalp. The collected strands of hair were then stored in individual polyethylene bag until pending analysis.

Collected hair samples were sent to the National Poison Centre, Penang, Malaysia for analysis with nitrogen detection methods by using Gas Chromatography–Mass Spectrometry (GC-MS). Once in the laboratory, each bag of hair samples was washed twice, using dichloromethane with 15 minutes sonication and were dried overnight at 30°C. Approximately 3cm of collected hair strands for each subject was measured, cut, minced into ~1mm pieces, weighed accurately, and were mixed with 100µl of sodium hydroxide (NaOH) (1M), 50µl of internal standard (IS) and left for overnight digestion at room temperature. The 3cm length of hair approximately represents a previous 3 months exposure to SHS. The following day, 1ml of distilled water was added, briefly mixed and centrifuged. The clear supernatant was recovered and extracted using 0.4mL solvent mixture (methanol:chloroform (1:3)). The extract was centrifuged (5min at 2,500 rpm) and dried over anhydrous sodium sulphate. The organic extract was then transferred into an auto-sampler vial (25).

Statistical data analysis

All data analysis was performed using IBM SPSS Statistics 21.0 (SPSS Inc., Chicago, IL, USA). Descriptive analysis was used to describe socio-demographic characteristics, prevalence of reported respiratory symptoms, SHS exposures and hair nicotine level of adolescents. Hair nicotine concentration below than limit of detection (LOD) (0.04ng/mg) were assigned an imputed value randomly sampled from the left tail of a truncated log normal distribution (14, 26). Multiple logistic regression was performed to estimate the effect of SHS exposures towards the occurrence of respiratory symptoms as presented in AOR and 95%CI. The well-proven independent variables in the previous SHS-related studies, which showed its contribution to the occurrence of respiratory symptoms, were included into the model. The model comprised of two blocks that the first block represented the confounder factors and the related SHS exposure predictors included in the second block. In addition, the correlation within states, schools and adolescents themselves as variance components in the population were

taken into account to be controlled in the model (27, 28).

Result

Socio demographic information and respiratory symptoms

About 60% of the participated adolescents were females and majority of them were of the Malay ethnicity (91%) (Table 1). More than half of the adolescents were of families earning more than RM1000 (67.3%) per month. Overall, the highest parental education was the secondary level (father=65%; mother=68%). The findings of the reported respiratory symptoms were as follows: 14.1% wheezing ever, 7.69% current wheezing, 20.5% exercise-induced wheeze, 11.5% nocturnal cough and 13.7% asthma (Table 1).

SHS exposures

Approximately 146 (62.4%) of the adolescents had smoking family members whereby 97.4% of them lived with at least one smoking family member. About 21.8% of them were exposed to SHS more than 4 days/week in which more than half of adolescents (67.9%) had one-hour exposure or less/day. There were 37.6% of adolescents exposed to SHS at home as shown by home smoking restriction practised (Table 2).

Hair nicotine level ranged from less than LOD to 0.161ng/mg. A cut off point of more than 0.2ng/mg was used to identify active smoking (29-31). Thus, all participated adolescents were considered as non-smokers. The geometric mean of hair nicotine was 0.0184 ± 2.31 ng/mg.

Effect of SHS exposure on the occurrence of respiratory symptoms

From the results of the multi-level logistic regression, household smoking was found to be associated with the symptoms of exercise-induced wheeze (AOR 1.06; 95%CI, 0.42-2.66) and nocturnal cough (AOR 14.2; 95%CI, 1.60-125) (Table 3).

Adolescents who lived with smoking family members were more likely to report exercise-induced

wheeze (1.06-fold risk) and nocturnal cough (14-fold risk) compared to those without smoking family members. SHS exposure in family's vehicle was a significant factor, which contributed to the reported symptom of wheezing ever (AOR 3.27; 95%CI, 1.15-9.34).

This study found that home smoking restriction was linked with a protective effect for adolescents to report the exercise-induced wheeze (AOR 0.33; 95%CI, 0.14-0.77). In addition, postnatal SHS exposure was shown to be associated with the reported exercise-induced wheeze (AOR 2.69; 95%CI 1.13-6.43).

The history of parental asthma was the only variable, which showed a significant association with self-reported asthma among adolescents. The adolescents who have an asthmatic parent tend to be more likely to report asthma (AOR 3.17; 95%CI 1.08-9.36) compared with other adolescents.

Gender was shown to be associated with reported ever, current wheezing and nocturnal cough. Female adolescents were more likely to report ever wheezing (AOR 3.78; 95%CI 1.27-11.2), current wheezing (AOR 8.42; 95%CI 1.42-49.9) and nocturnal cough (AOR 4.66; 95%CI 1.30-16.7) compared to male adolescents.

Table 1: Socio-demographic characteristics of adolescents (n= 234)

Variables	n	%
Socio-demographic characteristics		
Gender		
Male	94	40.2
Female	140	59.8
Ethnicity		
Malay	213	91.0
Non-Malay	21	8.97
Household income		
<RM1000*	99	42.3
≥RM1000	134	57.3
Missing	1	0.43
Father's education level		
Secondary	153	65.4
Tertiary	69	29.5
Missing	12	5.12
Mother's education level		
Secondary	160	68.4
Tertiary	64	27.4
Missing	10	4.27
Respiratory symptoms		
Wheezing ever		
Yes	33	14.1
Current wheezing		
Yes	18	7.69
Exercise-induced wheeze		
Yes	48	20.5
Nocturnal cough		
Yes	27	11.5
Self-reported asthma		
Yes	32	13.7

*RM1000 = 307.50 USD

Table 2: SHS exposure of adolescents (n=234)

SHS exposure	n	%
Smoking family member		
No	86	36.8
Yes	146	62.4
Missing	2	0.86
No. of smoking family members		
≤1 person	228	97.4
>1 person	4	1.71
Missing	2	0.85
Daily SHS exposure (day/week)		
≤4 days	178	76.1
>4 days	51	21.8
Missing	5	2.14
SHS exposure hour (hour/day)		
≤1 hour	159	67.9
>1 hours	68	29.1
Missing	7	2.99
Smoking in family's vehicle		
No	182	77.8
Yes	50	21.4
Missing	2	0.86
Home-smoking restriction		
No	88	37.6
Yes	143	61.1
Missing	3	1.28
Prenatal SHS exposure		
No	177	75.6
Yes	56	23.9
Missing	1	0.43
Postnatal SHS exposure		
No	142	60.7
Yes	86	36.8
Missing	6	2.57

Discussion

This study aimed to assess the burden of household smoking habits on the occurrence of respiratory symptoms among 234 young adolescents in Malaysia.

This study addressed several significant findings. Primarily, living with smoking family members was linked to the likelihood to report exercise-induced wheeze and nocturnal cough. Secondly, SHS exposure in the family's vehicle was linked with the increased risk of adolescents to report wheezing ever. Thirdly, the home-smoking restriction was shown to be a protective factor for adolescents to report exercise-induced wheeze. Also, having the SHS exposure during early of life

was shown to be one of the predictors for reporting the exercise-induced wheezing during the adolescent ages.

Many SHS-related studies conducted have focused only on the effect of SHS exposures on respiratory symptoms without involving any objective measurement of biomarkers (7, 10, 32). Thus, the data on hair nicotine level have added to the body of current knowledge in confirming the SHS exposure of the adolescents. Hair nicotine level has been used and validated as a gold standard in assessing SHS exposure among children and adults (33-35).

Table 3: Multiple logistic regression of SHS exposure associated with respiratory symptoms ⁴

	B(S.E)	Wheezing ever			Wheezing current			Exercise-induced wheeze			Nocturnal cough			Self-reported asthma		
		Wald	OR (95% CI)		B(S.E)	Wald	OR (95% CI)	B(S.E)	Wald	OR (95% CI)	B(S.E)	Wald	OR (95% CI)	B(S.E)	Wald	OR (95% CI)
Gender (^a Male)																
Female	1.33 (0.56)	5.71	3.78 (1.27-11.2)*	2.13 (0.91)	5.49	8.42 (1.42-49.9)*	0.72 (0.42)	3.02	2.06 (0.91-4.64)	1.54 (0.65)	5.60	4.66 (1.30-16.7)*	0.30 (0.47)	0.42	1.36 (0.54-3.41)	
Ethnicity (^a Non Malay)																
Malay	-1.52 (1.17)	1.68	0.22 (0.02-2.18)	-0.79 (1.27)	0.39	0.46 (0.04-5.45)	-0.09 (0.67)	0.02	0.92 (0.25-3.38)	-1.14 (1.23)	0.88	0.32 (0.03-3.49)	0.15 (0.73)	0.04	1.16 (0.28-4.80)	
Smoking status of family members (^a No)																
Yes	0.91 (0.66)	1.92	2.49 (0.69-9.00)	1.99 (1.16)	2.96	7.29 (0.76-70.1)	1.29 (0.49)	6.93	3.63 (1.39-9.50)*	2.65 (1.11)	5.68	14.2 (1.60-125.)*	0.46 (0.55)	0.68	1.58 (0.54-4.65)	
Daily SHS exposure (^a ≤4 days)																
>4 days	-0.83 (0.55)	2.24	0.44 (0.15-1.29)	-0.79 (0.69)	1.32	0.45 (0.12-1.75)	0.06 (0.47)	0.01	1.06 (0.42-2.66)	-0.45 (0.60)	0.57	0.64 (0.20-2.07)	-0.41 (0.56)	0.53	0.66 (0.22-2.01)	
SHS exposure hours (^a ≤1 hours)																
>1 hours	0.70 (0.50)	1.99	2.01 (0.76-5.32)	0.12 (0.64)	0.03	1.13 (0.32-3.94)	-0.88 (0.45)	3.79	0.41 (0.17-1.01)	0.44 (0.55)	0.65	1.56 (0.53-4.56)	-0.22 (0.52)	0.18	0.80 (0.29-2.22)	
Smoking in family's vehicle (^a No)																
Yes	1.19 (0.54)	4.92	3.27 (1.15-9.34)*	1.33 (0.68)	3.86	3.76(1.00-14.1)	0.57 (0.47)	1.46	1.77 (0.70-4.49)	0.79 (0.60)	1.72	2.20 (0.68-7.14)	0.85 (0.55)	2.38	2.33 (0.80-6.80)	
Home smoking restriction (Yes)																
No	0.14 (0.51)	0.08	1.15 (0.43-3.09)	0.08 (0.69)	0.01	1.08 (0.28-4.17)	-1.12 (0.44)	6.60	0.33 (0.14-0.77)*	-0.95 (0.63)	2.30	0.39 (0.12-1.32)	-0.93 (0.52)	3.23	0.40 (0.14-1.09)	
Hair nicotine level (ng/mg)																
	0.29 (0.63)	0.21	1.33 (0.39-4.55)	0.89 (0.94)	0.90	2.44 (0.39-15.4)	1.05 (0.57)	3.44	2.87 (0.94-8.72)	-0.92 (0.64)	2.09	0.40 (0.12-1.38)	1.13 (0.71)	2.56	3.10 (0.78-12.3)	
Parental asthma(^a No)																
Yes	0.23 (0.66)	0.12	1.26 (0.35-4.56)	0.01 (0.85)	0.00	1.01 (0.19-5.27)	-0.30 (0.60)	0.25	0.74 (0.23-2.41)	-1.24 (0.92)	1.80	0.29 (0.05-1.77)	1.16 (0.55)	4.38	3.17 (1.08-9.36)*	
Prenatal SHS exposure (^a No)																
Yes	0.21 (0.59)	0.12	1.23 (0.38-3.93)	0.94(0.77)	1.50	2.57 (0.57-11.6)	-0.95(0.51)	3.51	0.39 (0.14-1.05)	0.99 (0.66)	2.23	2.68 (0.73-9.78)	0.06 (0.58)	0.01	1.06 (0.34-3.31)	
Postnatal SHS exposure (^a No)																
Yes	0.23 (0.57)	0.16	1.25 (0.41-3.80)	0.11 (0.77)	0.02	1.11 (0.24-5.07)	0.99 (0.45)	4.96	2.69 (1.13-6.43)*	0.28 (0.66)	0.18	1.33 (0.36-4.87)	0.37 (0.54)	0.47	1.40 (0.50-4.18)	
Classification rate		85.6 (18.8/96.8)			91.9 (11.1/99.0)			80.6 (21.7/96.0)			88.7 (26.9/96.9)			86.9 (6.9/99.0)		
Cox & Snell R Square-		0.171-0.305			0.153-0.355			0.141-0.221			0.202-0.393			0.085-0.158		
Nagelkerke R Square																

⁴Model controlled for clustering effects in hierarchical analysis (state-classroom-students)/ *Significant at p value P<0.05 /^a Reference group

The lack of objective measures in assessing respiratory symptoms is one of the limitations in this study. Notwithstanding this limitation, this study relied on both the validated ISAAC written and video questionnaire (23) as it is a well-established tool in the measurement of the respiratory symptoms of children and adolescents. The household smoking status was shown to be associated with reported exercise-induced wheeze and nocturnal cough among adolescents. Adolescents who lived with smoking family members would encounter about 1.06-fold and 14-fold of risk to report a symptom of exercise-induced wheeze and nocturnal cough, respectively compared to those living with non-smoking family members. However, this finding was in contrast with the earlier study (36) in relation to reported nocturnal cough in which household smoking habit was not found to have a significant influence on the occurrence of the symptom. This result therefore needs to be interpreted with caution. The significant association found might be due to over estimation of multiple logistic regression result, which was affected by a large range of confidence interval shown for the AOR (37).

This study also revealed that the SHS exposure in the family's vehicle was statistically associated with the higher probability for reported wheezing ever among adolescents. The risk of reporting wheezing ever would be 3-fold for adolescents who were exposed to SHS in the family's vehicle compared to those without the exposure. Adolescents who were exposed to SHS in such enclosed spaces tend to have higher SHS exposure due to the small volume of area in which particles can easily become concentrated within the limited space. Previous studies have addressed that children who were subjected to SHS in small spaces such as cars would encounter 27 times the amount of toxins than usual (38). Even though the exposure might occur intermittently, exposure for a long period will adversely affect the respiratory system of exposed adolescents. There is a lack of public awareness campaign or health promotion advocating smoking bans in vehicles in Malaysia, but for other western countries, smoking bans in vehicles with small children have been implemented (39).

Home-smoking restriction was found to be a protective factor in reducing the risk of adolescents to report the exercise-induced wheeze. The practice of smoke-free home policy appeared to be a very important factor in minimizing adolescents' exposure to SHS. In fact, the current study have found that majority of parents (85%) who have strictly enforced a home-smoking restriction had also enforced smoke-free car policy (data not shown) and this was also revealed earlier (21). This suggests that parents who practised the smoke-free policy in vehicles and home have recognized and were aware of the main source and the health consequences of SHS exposure, which might be encountered by their children.

This study was able to demonstrate a significant association of postnatal SHS exposure on the occurrence of exercise-induced wheeze whereas prenatal exposure was not correlated with any reported respiratory symptom. Adolescent who experienced postnatal SHS exposure were over 3-fold more likely to report the symptoms than those without the exposure. This finding was in line with Larsson and colleagues (40) in which the exposure to SHS during childhood was linked with reported asthma and contributed to smoking likelihood in their adulthood (41).

In assessing the impact of SHS exposures on the likelihood of adolescents to report asthma, only parental asthma was shown to be associated with adolescents' self-reported asthma. This finding addressed that asthmatic heredity might be important factor in rising risk of asthma cases among adolescents (42-44). In addition, this study also highlighted that gender has become one of the predictors for the occurrence of few respiratory symptoms. Female was found to have higher risk to report current wheezing and nocturnal cough compared to male. Higher level of awareness and sensitiveness on a disease status among girls and women might explain the association as revealed in previous study (45) and these reasons make female more willing to report and discuss an experienced health symptom than male counterpart (46). The geometric mean of hair nicotine level was 0.0184 ± 2.31 ng/mg within the range of less than LOD to 0.160 ng/mg. Further analysis was per-

formed (data not shown) and there was no significant difference found between adolescents living with and without household smoking in contrary to the findings of other studies (47, 48). Compared with other studies (49, 50), the hair nicotine level in this study was low. The detection of low hair nicotine level among the adolescents might be due to the ability of adolescents to show avoidance behaviour from smoking parents compared with younger children who tend to be closer with their parents. In addition, the intermittent exposure of adolescents to SHS while in public places caused the exposure to occur at low level and this might explain the lower hair nicotine level detected among adolescents compared with other studies' population. Several studies found evidence of a dose-response relationship between increasing hair nicotine level and related health outcomes (51, 52). As reported earlier (52), the increment of hair nicotine level was associated with low birth weight and preterm delivery. However, this study was unable to depict the dose-response relationship as no significant association were found between hair nicotine levels and any reported respiratory symptoms among adolescents. Under reporting of the respiratory symptoms among the adolescents may as well occur due to recall bias and this might weaken the possibility of association with hair nicotine level to be depicted.

Conclusion

This study was able to link SHS exposures with reported respiratory symptoms among adolescents. Overall, the finding showed that parental or household smoking habits played a vital role in predicting their children's risk towards respiratory symptoms specifically in their adolescents' ages. This study highlights the problem of adolescents who were exposed to SHS due to smoking habits of their parents. Thus, in an attempt to reduce the repercussion of SHS exposure among younger adolescents, efforts should be emphasized in encouraging parents to move from indoor to outdoor smoking far from the vicinity of children.

Ethical considerations

Ethical issues such as plagiarisms, informed consent, misconduct, data fabrication and/or falsification, double publication and/or submission, redundancy, et cetera have been completely observed by the authors.

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References

1. Beasley R (1998). Worldwide variation in prevalence of symptoms of asthma, allergic rhinoconjunctivitis, and atopic eczema: ISAAC. *Lancet*, 351(9111): 1225-1232.
2. Lee YL, Hwang BF (2007). Time trend of asthma prevalence among school children in Taiwan: ISAAC phase I and III surveys. *Pediatr Allergy Immunol*, 18(3): 188-195.
3. Committee on the Assessment of Asthma and Indoor Air (2000). Clearing the Air: Asthma and Indoor Exposures, National Academy of Sciences. Washington, DC. Available from: aspe.hhs.gov/sp/asthma/appxh.pdf
4. International Agency for Research on Cancer Tobacco (IARC) (2004). Tobacco smoke and involuntary smoking. IARC monographs on the evaluation of carcinogenic risks to humans/World Health Organization, International Agency for Research on Cancer, Volume 83. Lyon, France. Available from: mono-

- graphs.iarc.fr/ENG/Monographs/vol83/mo-no83-1.pdf
- Dong GH, Cao Y, Ding HL, Ma YN, Jin J, Zhao YD, He QC (2007). Effects of environmental tobacco smoke on respiratory health of boys and girls from kindergarten: results from 15 districts of northern China. *Indoor Air*, 17(6): 475-483.
 - Eriksen M, Mackay J, Ross H. The Tobacco Atlas. Fourth Ed. Atlanta, GA: American Cancer Society; New York, NY: World Lung Foundation; 2012. Available from: www.TobaccoAtlas.org. <http://www.tobaccoatlas.org/more#sthash.uhMQIMG.dpuf>
 - Constant C, Sampaio I, Negreiro F, Aguiar P, Silva A, Salgueiro M, Bandeira T (2011). Environmental tobacco smoke (ETS) exposure and respiratory morbidity in school age children. *Revista Portuguesa de Pneumologia (English Edition)*, 17(1): 20-26.
 - Ho SY, Lam TH, Chung SF, Lam TP (2007). Cross-sectional and prospective associations between passive smoking and respiratory symptoms at the workplace. *Ann Epidemiol*, 17(2): 126-131.
 - US Department of Health and Human Services. (2006). The health consequences of involuntary exposure to tobacco smoke: a report of the Surgeon General. Atlanta, GA: US 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. GA, USA. Available from: www.surgeongeneral.gov/library/reports/secondhand-smoke/fullreport.pdf
 - He QQ, Wong TW, Du L, Jiang ZQ, Yu TI, Qiu H, Wu JG (2011). Environmental tobacco smoke exposure and chinese schoolchildren's respiratory health: A prospective cohort study. *American Prev Med*, 41(5): 487-493.
 - Gergen PJ. (2001). Environmental tobacco smoke as a risk factor for respiratory disease in children. *Resp Physiol*, 128(1): 39-46.
 - Cook DG, Strachan DP (1997). Health effects of passive smoking. 3. Parental smoking and prevalence of respiratory symptoms and asthma in school age children. *Thorax*, 52(12): 1081-1094.
 - Rampal L, Rampal S, Azhar MZ, Sherina MS, Mohamad T, Ramlee R, and Ahmad J (2008). A national study on the prevalence and factors associated with smoking among Malaysians aged 18 years and above. *Malays J Med Health Sc*: 4:41-53.
 - Abidin EZ, Semple S, Omar A, Rahman HA, Turner SW, Ayres JG (2011). A survey of schoolchildren's exposure to secondhand smoke in Malaysia. *BMC Public Health*, 11(1): 634.
 - California Department of Public Health (CDPH): *California Tobacco Control Update*. (2009). *California Tobacco Control Program Web site*. California, USA. Available from: <http://www.cdph.ca.gov/programs/tobacco/Documents/CTCUpdate2009.pdf>
 - Levy DT, Benjakul S, Ross H, Ritthiphakdee B (2008). The role of tobacco control policies in reducing smoking and deaths in a middle income nation: results from the Thailand SimSmoke simulation model. *Tob Control*, 17(1): 53-59.
 - Pizacani BA, Stark MJ, Koepsell TD, Thompson B, Diehr P (2002). Household smoking bans: which households have them and do they work? *Prev Med*, 36(1): 99-107.
 - Timothy R. Jordon J, Joseph A, Dake SS (2005). Adolescent exposure to and perceptions of environmental tobacco smoke. *J Sch Health*, 75(5): 178-186.
 - Schuster MA, Franke T, Pham CB (2002). Smoking patterns of household members and visitors in homes with children in the United States. *Arch Pediat Adol Med*, 156(11): 1094-1100.
 - Group T. G. C. (2006). A cross country comparison of exposure to secondhand smoke among youth. *Tob Control*, 15(2): ii4-ii19.
 - Nabi-Burza E, Regan S, Drehmer J, Ossip D, Rigotti N, Hipple B, Dempsey J, Hall N, Friebely J, Weiley V (2012). Parents smoking in their cars with children present. *Pediatrics*, 130(6): e1471-e1478.
 - Educational Research Application System (ERAS). (2012). Ministry of Education, Malaysia. <http://eras.moe.gov.my/eras/PanduanM.aspx>
 - Asher M, Keil U, Anderson H, Beasley R, Crane J, Martinez F, Mitchell EA, Pearce N, Sibbald B, Stewart AW (1995). International Study of

- Asthma and Allergies in Childhood (ISAAC): rationale and methods. *Eur Respir J*, 8(3): 483-491.
24. Chetiyankornkul T, Toriba A, Kizu R, Kimura K, Hayakawa K (2004). Hair analysis of nicotine and cotinine for evaluating tobacco smoke exposure by liquid chromatography mass spectrometry. *Biomed Chromatogr*, 18(9): 655-661.
 25. Man CN, Ismail S, Harn GL, Lajis R, Awang R (2009). Determination of hair nicotine by gas chromatography mass spectrometry. *J Chromatogr B*, 877(3): 339-342.
 26. Akhtar PC, Currie DB, Currie CE, Haw SJ (2007). Changes in child exposure to environmental tobacco smoke (CHETS) study after implementation of smoke-free legislation in Scotland: national cross sectional survey. *BMJ*, 335(7619): 545.
 27. Lam T, Chung S, Wong C, Hedley A (1998). Respiratory symptoms due to active and passive smoking in junior secondary school students in Hong Kong. *Int J Epidemiol*, 27(1): 41-48.
 28. Goldstein H (1995). *Multilevel Statistical Models*, 2nd Edn. London: Edward Arnold. pp. 25-26.
 29. Avila-Tang E, Al-Delaimy WK, Ashley DL, Benowitz N, Bernert JT, Kim S, Samet JM, Hecht SS (2013). Assessing secondhand smoke using biological markers. *Tob Control*, 22(3): 164-171.
 30. Benowitz NL, Hukkanen J, Jacob III P (2009). Nicotine chemistry, metabolism, kinetics and biomarkers *Nicotine Psychopharmacology* Springer. pp. 29-60.
 31. Hecht SS (2004). Carcinogen derived biomarkers: applications in studies of human exposure to second-hand tobacco smoke. *Tob Control*, 13(suppl 1): i48-i56.
 32. Al-Delaimy W, Fraser T, Woodward A (2001). Nicotine in hair of bar and restaurant workers. *New Zeal Med J*, 114(1127): 80-83.
 33. Martin-Pujol A, Fernandez E, Schiaffino A, Moncada A, Ariza C, Blanch C, Martinez-Sanchez, Jose M (2013). Tobacco smoking, exposure to second-hand smoke, and asthma and wheezing in schoolchildren: a cross-sectional study. *Acta Paediatr*, 102(7): e305-e309.
 34. Al-Delaimy W (2002). Hair as a biomarker for exposure to tobacco smoke. *Tob Control*, 11(3): 176-182.
 35. Woodruff SI, Conway TL, Edwards CC, Hovell MF (2003). Acceptability and validity of hair collection from Latino children to assess exposure to environmental tobacco smoke. *Nicotine Tob Res*, 5(3): 375-385.
 36. Vlaski E, Stavric K, Seckova L, Kimovska M, Isjanovska R (2011). Do household tobacco smoking habits influence asthma, rhinitis and eczema among 13&14 year-old adolescents? *Allergol Immunopath*, 39(1): 39-44.
 37. James RC, Savitri A (2005). Statistical analysis of survey data In: *Household Sample Surveys in Developing and Transition Countries*. Brogan D. United Nations Publications pp. 8.
 38. Farkas AJ, Gilpin EA, Distefan JM, Pierce JP (1999). The effects of household and workplace smoking restrictions on quitting behaviours. *Tob Control*, 8(3): 261-265.
 39. Karen B (2014). Smoke-free vehicles when children are present: tobacco control policy & legal resource center. New Jersey, US. Available from: www.njgasp.org/f_SF%20cars,kids,%20info,%20arguments.pdf.
 40. Larsson ML, Frisk M, Hallström J, Kiviloog J, Lundbäck B (2001). Environmental tobacco smoke exposure during childhood is associated with increased prevalence of asthma in adults. *CHEST Journal*, 120(3): 711-717.
 41. Carlsen KH, Carlsen KCL (2008). Respiratory effects of tobacco smoking on infants and young children. *Paediatr Respir Rev*, 9(1): 11-20.
 42. Zlotkowska R, Zejda JE (2005). Fetal and post-natal exposure to tobacco smoke and respiratory health in children. *Eur J Epidemiol*, 20(8): 719-727.
 43. Gilliland FD, Li YF, Dubeau L, Berhane K, Avol E, McConnell R, Gauderman WJ, Peters JM (2002). Effects of glutathione S-transferase M1, maternal smoking during pregnancy, and environmental tobacco smoke on asthma and wheezing in children. *Am J Resp Crit Care*, 166(4): 457-463.
 44. Ober C, Yao TC (1990). The genetics of asthma and allergic disease: a 21st century perspective. *Immunol Rev*, 242(1): 10-30.
 45. Wool CA, Barsky AJ (1994). Do women somatize more than men?: Gender differences in somatization. *Psychosomatics*, 35(5): 445-452.
 46. Tollefsen E, Langhammer A, Romundstad PL, Bjermer L, Johnsen R, Holmen TL (2007). Fe-

- male gender is associated with higher incidence and more stable respiratory symptoms during adolescence. *Resp Med*, 101(5): 896-902.
47. Al-Delaimy WK, Crane J, Woodward A (2001). Passive smoking in children: effect of avoidance strategies at home as measured by hair nicotine levels. *Arch Environ Health: An International Journal*, 56(2): 117-122.
 48. Kalinic N, Skender L, Karacic V, Brcic I, Vadjic V (2003). Passive exposure to tobacco smoke: hair nicotine levels in preschool children. *B Environ Contam Toxicol*, 71(1): 0001-0005.
 49. Al-Delaimy W, Crane J, Woodward A (2000). Questionnaire and hair measurement of exposure to tobacco smoke. *J Expo Anal Env Epidemiol*, 10(4): 378-384.
 50. Al-Delaimy W, Fraser T, Woodward A (2001). Nicotine in hair of bar and restaurant workers. *New Zeal Med J*, 114(1127): 80-83.
 51. Jaakkola M, Jaakkola J (2006). Impact of smoke-free workplace legislation on exposures and health: possibilities for prevention. *Eur Respir J*, 28(2): 397-408.
 52. Jaakkola J, Jaakkola N, Zahlsen K (2001). Fetal growth and length of gestation in relation to prenatal exposure to environmental tobacco smoke assessed by hair nicotine concentration. *Environ Health Persp*, 109(6): 557.