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January 22, 2009

Dr Janet M. Woollard, MLA
Chairman,
Education and Health Standing Committee
Western Australian Legislative Assembly

Dear Dr Woollard,

Re: SUPPORT for Tobacco Products Control Amendment Bill 2008

We are writing at this time to support your proposed Tobacco Products Control Amendment Bill 2008, and to inform you of research conducted at Harvard School of Public Health which has relevance to the bill. As former Director of Tobacco Control at Massachusetts Department of Public Health, Dr Connolly has over twenty years of experience in tobacco product regulation and research on tobacco use, public health, and policy. Dr Rees is an Australian citizen with research expertise in secondhand smoke exposure, especially among young people.

Our research that has shown that second hand smoke emissions in cars where people smoke can be dangerous for children. We have found alarmingly high $PM_{2.5}$ levels (over 272 μg/m^3) in private cars in which people smoke. These concentrations were higher than were found in studies of secondhand smoke in (previously) smoky bars and pubs in Massachusetts and western New York state.

The U.S. EPA's Air Quality Index rates 24 hr exposure to $PM_{2.5}$ concentrations above 40 μg/m^3 as "unhealthy for sensitive groups," such as children and the elderly, and above 250 μg/m^3 as "hazardous" for all individuals. Our measurements in private vehicles, obtained from the simulated position of an infant's head in child restrainer seat, highlight the potentially serious threat to children's health presented by secondhand smoke in private cars under normal driving conditions. We concluded that prolonged or repeated exposure to the $PM_{2.5}$ levels observed in cars where people smoke is unsafe for children. A copy of our research paper is attached.

The bill amendment proposes to make smoking in vehicles an offense if a young person is present. Western Australia currently has broad legislative protection from secondhand smoke exposure, a known carcinogen and toxic air contaminant, in public places, including workplaces

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and licensed venues. However, children are more at risk in domestic environments which are not currently covered under state clean indoor air laws. As you know, governments of NSW, South Australia and Tasmania have successfully implemented protections for young people from exposure to secondhand smoke in cars. The current bill amendment is an important step in reducing children's exposure to secondhand smoke and is likely to enhance public health among young people as well as save the government of Western Australia money in costly treatment for secondhand smoke related childhood diseases.

While some basic car safety behaviors, such as compulsory use of seat belts and child restrainers, are widely legislated, we understand there is a concern that legislation affecting private smoking behavior may constitute an unwarranted intrusion on personal privacy. However, research has shown that precisely this sort of legislation would receive popular support in many jurisdictions. Passage of this bill would reinforce the message that there is no safe level of exposure to secondhand smoke, especially for WA's youngest, most vulnerable population.

Yours sincerely,

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Professor of the Practice of Public Health

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Measuring Air Quality to Protect Children from Secondhand Smoke in Cars

Vaughan W. Rees, PhD, Gregory N. Connolly, DMD, MPH

Background: Secondhand tobacco smoke (SHS) is a major, preventable contributor to acute and chronic adverse health outcomes that affect children disproportionately. The predominant source of SHS among children is domestic exposure, and while up to two thirds of U.S. households have car smoking bans, an unacceptable number of children remain vulnerable. To help promote more effective protection through legislation, health communication strategies, or behavioral interventions, data demonstrating the adverse effect of SHS on air quality in cars are needed.

Methods: Secondhand tobacco smoke in a motor vehicle under actual driving conditions was monitored by measuring respirable suspended particles (RSPs) of less than 2.5 microns in diameter, and carbon monoxide. Forty-five driving trials were conducted, using teams of volunteer drivers and smokers recruited from the general community. Three smoking conditions (nonsmoking baseline, active smoking, and immediate post-smoking period, each 5 minutes) were crossed with two ventilation conditions (windows open, closed) in a 3×2 within-sessions factorial design.

Results: The highest mean observed RSP level was 271 $\mu g/m^3$, which is unsafe, particularly for children. Peak RSP levels were considerably higher. RSPs and carbon monoxide increased significantly from baseline after smoking, and these increases were greatest during the closed ventilation condition, compared with open ventilation.

Conclusions: Private passenger cars are a domestic environment with the potential to yield unsafe levels of SHS contaminants. These data may assist policymakers and health advocates to promote protective strategies to ensure smoke-free domestic environments for children.
(Am J Prev Med 2006;31(5):363–368) © 2006 American Journal of Preventive Medicine

Introduction

Secondhand tobacco smoke (SHS) is a toxic air contaminant that contributes to multiple, preventable adverse health outcomes. Among adults, SHS exposure is associated with cardiovascular disease, cancers, and respiratory and reproductive problems.[1–3] Children exposed to SHS show greater likelihood of lower respiratory infections,[4–6] sudden infant death syndrome,[7,8] ear infections,[9] and severity of asthma symptoms.[10,11] Children may be more vulnerable to SHS-induced respiratory diseases due to smaller airways and greater oxygen demand and, hence, higher respiratory rates, as well as less-mature immune systems. Accordingly, SHS is regarded as a major pediatric problem.[7] The estimated prevalence of SHS exposure among children varies according to the source of exposure, age of the child, and family smoking behavior. Recent estimates based on national surveys indicate that 35% to 43% of children live in homes where secondhand smoke is present.[12–14] While secondhand smoke exposure in cars is less well investigated, New York state middle school students reported being present in a car with a smoker an average of 1.2 days per week, while high school students reported an average of 1.5 days per week.[15] These frequency estimates were significantly higher among students from smoking households.

Reducing exposure to SHS in the United States has been achieved through evolving strategies that include legislation, public policy, and health communications. Despite recent successes in protecting adults, there is a lack of legislative protection from SHS arising from domestic sources for children. The predominant cause of SHS among children is domestic exposure,[16,17] and most existing protection arises from voluntary individual or family-based smoking restrictions in the home or car. Smoking bans in the home have been shown to contribute to lower urinary cotinine in children.[18] Some three in four homes in California have indoor smoking bans and two thirds of households have car smoking bans.[19,20] Despite the relative benefit of such

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0749-3797/06/$–see front matter

doi:10.1016/j.amepre.2006.07.021

personal bans, a vast number of children in the United States continue to be exposed to SHS, especially among low-income families, which are less likely to impose domestic bans on smoking.[21]

Secondhand smoke monitoring has been used to document unsafe levels of contaminants, and to focus attention on health risks.[22] Findings have more recently been used to enhance support for clean indoor air laws or to confirm the benefits of such laws.[23–25] Air quality monitoring of environments in which children are present has focused on exposure in the home.[26,27] Although high emission concentrations have been found in simulated exposure to SHS in vehicles,[28–30] measures of SHS from passenger cars in a real driving situation have not been published. To help promote protection through legislation, health communication strategies, or behavioral interventions, such data are needed.

This study aimed to simulate children's exposure to secondhand smoke in a motor vehicle by measuring carbon dioxide and respirable suspended particles (RSPs) of less than 2.5 microns in diameter, under actual driving conditions.

Methods

Apparatus

Tobacco smoke concentration was measured by a TSI SidePak AM510 Personal Aerosol Monitor, using a 0.32 calibration factor. This device uses a laser photometer that samples airborne particles in real time. The device was fitted with an impactor to detect RSPs with a mass median aerodynamic diameter of <2.5 microns. The SidePak was zero-calibrated prior to each sampling session and set so that data were averaged and logged over 1-minute intervals. The air flow rate was set at 1.7 L/min. Carbon monoxide was monitored using a TSI Q-Trak Plus Indoor Air Quality Monitor. This instrument also provides real-time data collection and storage and was set to log data at 1-minute intervals.

The SidePak and Q-Trak were housed in a modified infant car restrainer seat and positioned in the rear passenger seat opposite the driver's side of the vehicle. Also located in the seat was a digital clock to ensure standardization of measurement periods for each session. A 2-foot length of clear, nonreactive tubing was attached to the air intake inlet on the SidePak, and the open end of the tube was positioned at the simulated head position of an infant in the car seat.

Three cars were used to conduct data-collection sessions: a 1991 Honda Civic sedan, a 1986 Toyota Tercel, and a 2005 Honda Civic sedan. The internal volumes of these vehicles, according to the manufacturers, were 2152 L, 2588 L, and 2293 L, respectively. All vehicles were owned and driven by smokers, and each had a history of being used for smoking.

Study Design

A 3 (smoking phase) × (ventilation) within-sessions factorial design was employed. This enabled a comparison between nonsmoking baseline and smoking conditions within the same session, thus enabling better control of ambient environmental conditions. The three smoking conditions were nonsmoking baseline, active smoking, and immediate post-smoking period. The two ventilation conditions were employed to represent likely real-life driving conditions. Ventilation "open" required all four windows to be lowered half way, or approximately 25 cm. This arrangement allowed for a high degree of air flow. Ventilation "closed" required only the smoker's side window to be lowered 5 cm. It is plausible that this setting is often used by smokers under inclement weather conditions, such as rain or cold. The chosen ventilation conditions therefore reflected a range of driver ventilation settings that were easily replicable.

Air quality measurements were sampled in 1-minute blocks and averaged over the whole 5-minute sampling period.

Procedure

Three teams, consisting of two volunteers each, were recruited from the general community to drive a vehicle in city traffic while generating secondhand smoke under controlled conditions. Each team comprised a nominated driver and smoker. Hence, the individual unit of measurement was the smoking trial, rather than research participant.

Forty-five driving "trials" were conducted. Data were collected from within the vehicle while each driving team followed a standard route designed to encounter light urban traffic. The route comprised a section of a major arterial road in the city of Boston, Massachusetts, which allows continuous traffic flow without impediment by traffic signals. The posted speed limit on this route is 40 mph, and the study drivers were required to maintain speed between 30 and 40 mph. Drivers performed three, 23-mile circuits of this roadway, which took an average of 57 minutes to complete.

In each driving trial, measurements were obtained with the open-windows condition first, followed by the closed-windows condition. This order was employed so that the rate of dissipation of secondhand smoke in a vehicle with windows closed could be monitored (data not reported here). After an approximate 10-minute stabilization period in which drivers negotiated local traffic to reach the designated roadway, a 5-minute baseline measurement period was started. Immediately following this, the person designated as smoker lit a single cigarette and smoked ad lib for 5 minutes (active smoking period), after which time the unused portion of the cigarette was extinguished. Air monitoring then continued for 5 minutes (post-smoking period). A 15-minute washout period was then performed to reduce or eliminate the secondhand smoke, by driving with windows open. After this, the car windows were placed in the ventilation-closed setting, and the procedure was repeated, beginning with a new 5-minute baseline. The procedure is summarized in Table 1.

Car air conditioning and heating and/or ventilation systems, including internal air recycling, were not used during the driving trials so as to reduce additional sources of variability in internal air quality. The study was conducted during the summer months when the ambient air temperature allowed drivers to operate comfortably under both open- and closed-windows conditions.

Institutional review board approval for the study was granted on March 15, 2005 (Federal Assurance ID

Table 1. Summary of the study procedure

Time (minutes)	Action	Ventilation
0–10	Driver travel to study route	Open
10	Commence open-phase monitoring	
10–15	Baseline	Open
15–20	Smoke one cigarette	Open
20–25	Post-smoking monitoring	Open
25	Cease monitoring	
25–40	Washout period	Open
40	Commence closed-phase monitoring	
40–45	Baseline	Closed
45–50	Smoke one cigarette	Closed
50–55	Post-smoking monitoring	Closed
55	Cease monitoring	

FWA00002642). The study was conducted and data analyzed in 2005 and 2006.

Data Analysis

The main outcome variables were RSPs of 2.5 micron diameter or less (particulate matter [$PM_{2.5}$] and carbon monoxide (CO, parts per million [ppm]) concentrations, under conditions of open and closed ventilation. Data were averaged over 1-minute blocks to create a mean $PM_{2.5}$ measure within each measurement period.

Two-way within-groups analyses of variance (ANOVA) were employed to test main effects and interactions of the experimental factors. Factors employed were smoking phase (baseline, smoking, post-smoking) and ventilation (windows open or closed). Pearson's product-moment correlations for paired samples, using pairwise deletion of missing cases, were used to determine whether changes in RSPs and CO from their baseline values covaried similarly.

Results

Respirable Suspended Particles

Mean (and standard error) real-time plots of RSPs under open- and closed-ventilation conditions are presented in Figure 1. These show substantial increases from baseline in airborne smoke particles during the smoking period. As expected, this increase was greater during the closed-ventilation condition, compared with open ventilation. Dissipation of airborne particles occurred rapidly after the active smoking period ended. The highest peak 1-minute values that comprised each 5-minute measurement period mean are presented as dashed lines in Figure 1. These values demonstrate higher RSP concentrations than the overall 5-minute mean.

A two-way ANOVA revealed significantly higher RSPs under the closed-ventilation condition than the comparison open-ventilation condition ($F[1,44]=29.03$, $p=0.001$). Similarly, there was a highly significant main effect of smoking phase ($F[2,88]=25.76$, $p<0.001$), indicating an overall change in average RSP levels across the baseline, smoking, and post-smoking phases. The ventilation—phase interaction also was significant ($F[2,88]=14.60$, $p<0.001$).

Carbon Monoxide

Mean (and standard error) CO levels are presented in Figure 2. Data from two driving trials were lost due to equipment malfunction, resulting in available CO data from 43 driving trials for analysis. Data were analyzed using two-way ANOVA for the main effects of smoking phase and ventilation. There was a significant main effect of ventilation ($F[1,42]=24.12$, $p<0.001$), with CO higher through the closed condition. There was a significant overall change in CO arising from smoking phase ($F[2,84]=16.90$, $p<0.001$), with the significant ventilation—phase interaction ($F[2,84]=13.74$, $p<0.001$) indicating that CO level differed among the measurement phases according to ventilation condition.

A post-hoc analysis was used to explore the nature of the significant interaction between smoking phase and ventilation on this measure. To determine whether the opened and closed levels of ventilation were each influencing CO levels under the smoking-phase conditions, an analysis of simple effects[31] was applied. Under the closed-ventilation condition, CO levels increased significantly across the three smoking phases ($F[2,84]=18.38$,

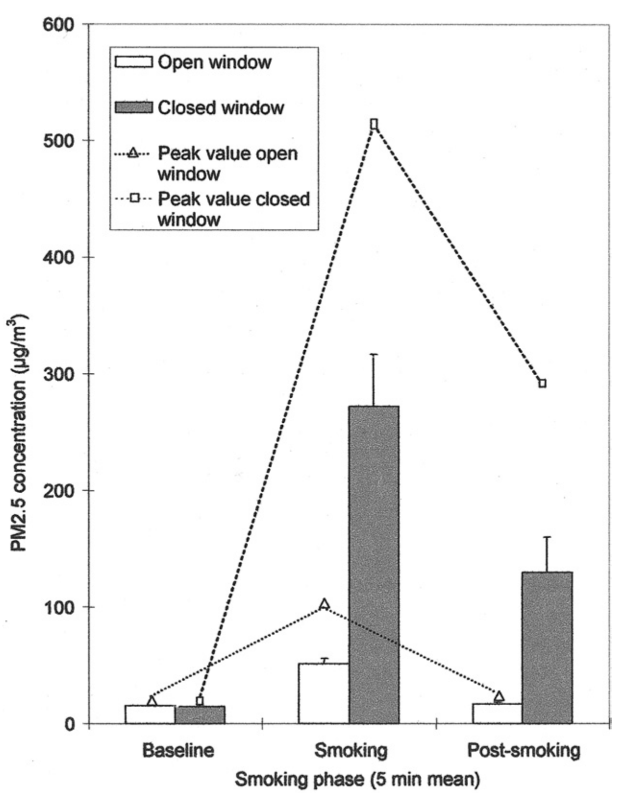

Figure 1. Mean RSP values for open- and closed-ventilation conditions during three phases of measurement (peak values shown in dashed lines).
PM, particulate matter; RSP, respirable suspended particles.

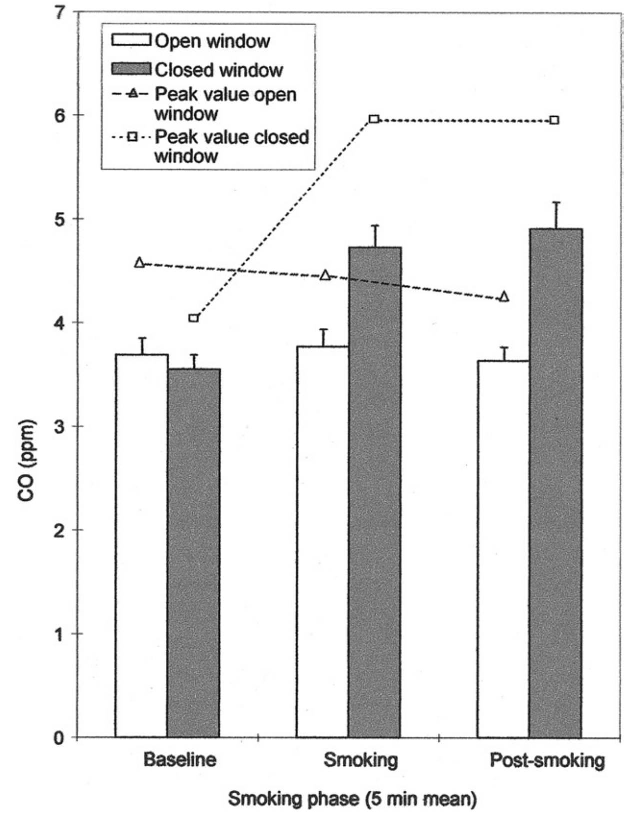

Figure 2. Mean CO values for open- and closed-ventilation conditions during three phases of measurement (peak values shown in dashed lines).
CO, carbon monoxide.

$p<0.001$). However, CO levels did not change across the open-ventilation condition ($F[2,84]<1.00$, $p=0.54$), suggesting that the significant interaction between ventilation and smoking phase was determined only by the increase in CO across the closed-ventilation condition.

Correlations Between Variables

Pearson's product-moment correlations for paired samples were used to explore potential covariations in increases in RSPs and CO from their respective baselines. For each variable, baseline data were subtracted from the smoking and post-smoking phases. Significant correlations were observed between mean RSP and CO concentrations during the smoking ($r_{45}=0.699$, $p<0.001$) and post-smoking ($r_{45}=0.310$, $p=0.043$) phases under closed-ventilation conditions. Correlations between the two outcome variables under the open-ventilation condition were not significant ($p<0.068$).

Discussion and Conclusions

These data reveal alarming RSP levels generated from smoking a single cigarette for only 5 minutes in a private car. RSP concentrations were significantly higher than baseline during the smoking and post-smoking measurement periods. As expected, RSP levels were higher under the closed-windows condition than with windows open. The observed interaction between ventilation and smoking phase suggests that greater increases in mean RSP level following smoking were found under the closed-ventilation condition, which further underscores the role of (external) ventilation on air quality.

While smoking, mean RSP concentrations of 272 $\mu g/m^3$ (closed) and 51 $\mu g/m^3$ (open) were attained, with even higher peak levels observed briefly (505 $\mu g/m^3$ closed, and 104 $\mu g/m^3$ open). These RSP levels can be better contextualized when considered against recent studies of bars that allow smoking, using similar methodology. A mean $PM_{2.5}$ concentration of 206 $\mu g/m^3$ was found among 27 bars in eastern Massachusetts,[25] while a mean concentration of 412 $\mu g/m^3$was observed in 14 bars in western New York state.[24] Health standards that would enable adequate characterization of risk from SHS-generated RSPs, based on present data, are not available. However, the U.S. Environmental Protection Agency's air quality index[32,33] rates 24-hour exposure to $PM_{2.5}$ concentrations of >40 $\mu g/m^3$ as "unhealthy for sensitive groups," such as children and the elderly, and >250 $\mu g/m^3$ as "hazardous" for all individuals. The current RSP measurements, obtained from the simulated position of an infant's head in a child-restraint seat, highlight the potentially serious threat to children's health presented by secondhand smoke in private cars under normal driving conditions.

A significant increase in CO also was observed following smoking under the closed-ventilation condition. Like the RSP measure, CO levels were higher during the closed-ventilation condition, and showed an increase in ppm level from baseline. However, this increase was not observed under the open-ventilation condition, which maintained ppm levels close to baseline. The failure to observe an increase in CO during the open-ventilation phase may also explain the absence of a correlation between changes in CO and RSPs under the open-ventilation condition. The reason for the lack of an increase in CO under the open-ventilation condition is unclear. This very light gas-phase compound might be more easily removed or diluted by air flow than heavier particles such as RSPs. While the maximum observed levels (peak CO=6 ppm) were not high enough to cause short-term health risks, this observation underscores the fact that secondhand smoke presents potentially dangerous contaminants beyond fine particle (RSP) exposure. CO is a poisonous gas, which may cause coma and death in large amounts, but among infants is known to induce lethargy and loss of alertness even in small quantities.[34]

Some caution should be used in the interpretation of these findings. The present observations were made under the relatively arbitrary ventilation conditions of windows open and closed. Because secondhand smoke measures were significantly higher during the closed-windows condition, it might be implied that open windows or adequate ventilation in a moving vehicle help to maintain SHS exposure at an acceptable level. The significant main effects of smoking phase on RSPs and CO, together with interactions between ventilation and smoking phase, indicate that smoking under both open- and closed-ventilation conditions resulted in increased SHS, although the increase was greater under the closed condition. Further research is required to understand whether drivers manipulate ventilation to reduce SHS, either by use of open windows or internal vehicle ventilation systems. To increase control of potential sources of variation, this study did not attempt to manipulate internal ventilation, such as fresh-air intake or air conditioning. Until such research is conducted, it should be concluded that smoking in cars under typical driver and traffic conditions provides potentially unsafe secondhand smoke exposure.

Respirable suspended particles and CO can also arise from other sources, most notably traffic emissions. Both are by-products of the combustion of fossil fuels, and are present in vehicle exhaust. Therefore, both types of contaminant could arise from leakage of exhaust into the interior of the vehicle or from ambient traffic conditions. While this source cannot be ruled out conclusively, the observation of low baseline levels of RSPs and CO, even under the closed-ventilation condition, suggests that vehicle emissions played a negligible role in the observed increases in air contaminants. Furthermore, RSPs and CO covaried in a predictable manner according to ventilation and smoking phase, suggesting that they were attributable to the smoking source.

Legislation banning smoking in cars with young children present was adopted recently in Arkansas (Act 13, 2006) and Louisiana (Act 838, 2006). As of this writing, smoking bans in cars with children has been introduced, but not yet finally passed, in the states of California (AB1569-AB2997-04), Georgia (HB1138-05), Michigan (HB5085-05; HB5407-05), New Jersey (A2717-05), New York (A175-05), Pennsylvania (HB1963-06), and Vermont (H324-05). Calls have been made for similar legislation in Australia.[35–37] While some basic car-safety behaviors, such as the compulsory use of seat belts and child restraints, are widely legislated, there is a concern that legislation affecting private smoking behavior may constitute an unwarranted intrusion on personal privacy. Roberts et al.[36] have shown that precisely this sort of legislation could receive popular support in some jurisdictions. Effective alternative strategies also are available, including smoking-cessation interventions,[38,39] behavioral interventions that do not require quitting,[40] and health communication campaigns (e.g., Car and Home: Smoke Free Zone,[41] Don't Pass Gas[42]).

Studies of air quality have shown that indoor domestic environments can be a source of dangerous secondhand smoke contaminants for children.[26,27] Private passenger cars can now be included as another domestic environment with the potential to yield high levels of SHS contaminants under normal conditions of use. Prolonged or repeated exposure to the RSP levels observed in the present study is unsafe for children. Air quality measurement in smoking environments, together with communication of the health risks of SHS, have been used to promote smoke-free environments. Data such as those reported here should be used by policymakers and health advocates to promote protective strategies, including legislation, personal health behavior interventions, and broad-ranging health communications, to ensure smoke-free domestic environments for children.

We gratefully acknowledge the support of the Flight Attendant Medical Research Institute (FAMRI), which provided funding under a Distinguished Professorship awarded to GNC. We also thank Dr. Carrie Carpenter for expert advice on this project, Jonathan Nadel for assistance with data collection, and Colin Benjamin Jackson for assistance with manuscript preparation.

No financial conflict of interest was reported by the authors of this article.

References

1. U.S. Department of Health and Human Services. Reducing tobacco use: a report of the Surgeon General. Atlanta GA: Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, 2000.
2. U.S. Environmental Protection Agency. Respiratory health effects of passive smoking: lung cancer and other disorders, vol. 4. Washington DC: U.S. Department of Health and Human Services, U.S. Environmental Protection Agency, 1993.
3. California Environmental Protection Agency. Health effects of exposure to environmental tobacco smoke: final report. Sacramento CA: Office of Environmental Health Hazard Assessment, 1997.
4. Gurkan F, Kiral A, Dagli E, Karakoc F. The effect of passive smoking on the development of respiratory syncytial virus bronchiolitis. Eur J Epidemiol 2000;16:465–8.
5. Strachan DP, Cook DG. Health effects of passive smoking. 1. Parental smoking and lower respiratory illness in infancy and early childhood. Thorax 1997;52:905–14.
6. Jedrychowski W, Flak E. Maternal smoking during pregnancy and postnatal exposure to environmental tobacco smoke as predisposition factors to acute respiratory infections. Environ Health Perspect 1997;105:302–6.
7. Klonoff-Cohen HS, Edelstein SL, Lefkowitz ES, et al. The effect of passive smoking and tobacco exposure through breast milk on sudden infant death syndrome. JAMA 1995;273:795–8.
8. Mitchell EA, Ford RP, Stewart AW, et al. Smoking and the sudden infant death syndrome. Pediatrics 1993;91:893–6.
9. Adair-Bischoff CE, Sauve RS. Environmental tobacco smoke and middle ear disease in pre-school age children. Arch Pediatr Adolesc Med 1998;152:127–33.
10. Mannino DM, Homa DM, Redd SC. Involuntary smoking and asthma severity in children: data from the Third National Health and Nutrition Examination Survey. Chest 2002;122:409–15.
11. Jaakkola JJ, Nafstad P, Magnus P. Environmental tobacco smoke, parental atopy, and childhood asthma. Environ Health Perspect 2001;109:579–82.
12. Schuster, MA, Franke T, Pham CB. Smoking patterns of household members and visitors in homes with children in the United States. Arch Pediatr Adolesc Med 2002;156:1095–100.

13. Pirkle JL, Flegal KM, Bernert JT, Brody DJ, Etzel RA, Maurer KR. Exposure of the U.S. population to environmental tobacco smoke: the Third National Health and Nutrition Examination Survey, 1988–1991. JAMA 1996;275:1233–40.
14. Gergen PJ, Fowler JA, Maurer KR, Davis WW, Overpeck MD. The burden of environmental tobacco smoke exposure on the respiratory health of children 2 months through 5 years of age in the United States: Third National Health and Nutrition Examination Survey, 1988–1994. Pediatrics1998;101. Available at: www.pediatrics.org/cgi/content/full/101/2/e8.
15. New York State Department of Health. Second annual independent evaluation of New York's tobacco control program: final report. Albany: New York State Department of Health, 2005.
16. Emmons KM, Abrams DB, Marshall RJ, et al. Exposure to environmental tobacco smoke in naturalistic settings. Am J Public Health 1992;82:24–8.
17. Thaqi A, Franke K, Merkel G, Wichmann HE, Heinrich J. Biomarkers of exposure to passive smoking of school children: frequency and determinants. Indoor Air 2005;15:302–10.
18. Wakefield M, Banham D, Martin J, Ruffin R, McCaul K, Badcock N. Restrictions on smoking at home and urinary cotinine levels among children with asthma. Am J Prev Med 2000;19:188–92.
19. Norman GJ, Ribisl KM, Howard-Pitney B, Howard KA. Smoking bans in the home and car: do those who really need them have them? Prev Med 1999;29:581–9.
20. Gilpin EA, White MM, Farkas AJ, Pierce JP. Home smoking restrictions: which smokers have them and how they are associated with smoking behavior. Nicotine Tob Res 1999;1:153–62.
21. Halterman JS, Fagnano M, Conn KM, Szilagyi PG. Do parents of urban children with persistent asthma ban smoking in their homes and cars? Ambul Pediatr 2006;6:115–9.
22. Repace JL, Lowrey AH. Indoor air pollution, tobacco smoke, and public health. Science 1980;208:464–72.
23. Nardini S, Cagnin R, Invernizzi G, Ruprecht A, Boffi R, Formentini S. Indoor particulate matter measurement as a tool in the process of the implementation of smoke-free hospitals. Monaldi Arch Chest Dis 2004;61:183–92.
24. Travers M, Cummings M, Hyland A. Indoor air quality in hospitality venues before and after implementation of a clean indoor air law, western New York, 2003. MMWR Morb Mortal Wkly Rev Weekly 2004;53:1038–41.
25. Connolly GN, Carpenter C, Alpert HR, Skeer, M, Travers M. Evaluation of the Massachusetts smoke-free workplace law 2005. Cambridge MA: Harvard School of Public Health, 2005 (Tobacco Control Research and Training Program Research Report 02-05).
26. Matt GE, Hovell MF, Zakarian JM, Bernert JT, Pirkle JL, Hammond SK. Measuring secondhand smoke exposure in babies: the reliability and validity of mother reports in a sample of low-income families. Health Psychol 2000;19:232–41.
27. Matt GE, Quintana PJ, Hovell MF, et al. Households contaminated by environmental tobacco smoke: sources of infant exposures. Tob Control 2004;13:29–37.
28. Ott W, Langan L, Switzer P. A time series model for cigarette smoking activity patterns: model validation for carbon monoxide and respirable particles in a chamber and an automobile. J Exp Anal Environ Epidemiol 1992;2:175–200.
29. Park JH, Spengler JD, Yoon D, Dumyahn T, Lee K, Ozkaynak H. Measurement of air exchange rate of stationary vehicles and estimation of in-vehicle exposure. J Exp Anal Environ Epidemiol 1998;8:65–78.
30. Zhou R, Li S, Zhou Y, Haug A. Comparison of environmental tobacco smoke concentrations and mutagenicity for several indoor environments. Mutat Res 2000;465:191–200.
31. Keppel G. Design and analysis: a researcher's handbook. Englewood Cliffs, NJ: Prentice-Hall, 1991.
32. U.S. Environmental Protection Agency. Air quality guide for particle pollution. Available at: http://cfpub.epa.gov/airnow/index.cfm?action=static.aqguidepart. Accessed May 17, 2006.
33. U.S. Environmental Protection Agency. Air quality criteria for particulate matter, vols. I and II. Research Triangle Park NC: U.S. Environmental Protection Agency, 2005.
34. Crocker PJ, Walker JS. Pediatric carbon monoxide toxicity. J Emerg Med 1985;3:443–8.
35. Bauman A, Chen XC, Chapman S. Protecting children in cars from tobacco smoke. BMJ 1995;311:1164.
36. Roberts LM, Wakefield M, Reynolds CS. Children's exposure to environmental tobacco smoke in private vehicles. Med J Aust 1996;165:350.
37. www.democrats.org.au/news/index.htm?press_id=4358.
38. Emmons KM, Hammond SK, Abrams DB. Smoking at home: the impact of smoking cessation on nonsmokers' exposure to environmental tobacco smoke. Health Psychol 1994;13:516–20.
39. Emmons KM, Hammond SK, Fava JL, Velicer WF, Evans JL, Monroe AD. A randomized trial to reduce passive smoke exposure in low-income households with young children. Pediatrics 2001;108:18–24.
40. Gehrman CA, Hovell M. Protecting children from environmental tobacco smoke (ETS) exposure: a critical review. Nicotine Tob Res 2003;5:289–301.
41. Yiow L, Hutchinson, I, Oakes, W. Car and home: smoke free zone. A report on the Environmental Tobacco Smoke and Children Project 2001–2005. Sydney: Cancer Council NSW, 2005.
42. American Legacy Foundation. Don't pass gas, 2006. Available at: www.dontpassgas.org.