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Smoking Restrictions in the Homes of Children With Cancer

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Abstract

Objectives: To examine smoking restrictions in households of children with cancer and their effect on biological measures of children's secondhand smoke exposure (SHSe).

Methods: A sample of 135 parents of nonsmoking children with cancer who lived with a smoker completed structured interviews.

Results: Approximately 43% of families prohibited smoking in the home. Children living in homes that prohibited smoking had median cotinine levels that were 71% and 52% lower than did those from homes with no and partial restrictions. **Conclusions:** Parents should be directed to completely ban all smoking from the home and car to best protect their children from SHSe.

Keywords

secondhand smoke exposure; household smoking bans; pediatric cancer

Exposure to secondhand smoke (SHS) is harmful to children's health and is known to increase morbidity and mortality risks in infants, children, and adult nonsmokers.¹ SHS exposure (SHSe) has been linked to cardiovascular and respiratory disease, increased severity of asthma, increased risk for sudden infant death syndrome,^{2,3} and otitis media⁴; and the risk of complication increases with higher levels of exposure.⁵

Children with cancer may be especially vulnerable to these health risks secondary to disease and treatment-related toxicities that may affect their respiratory, pulmonary, and cardiovascular functioning.⁶⁻⁸ Children newly diagnosed with cancer who reside in smoking households are more likely to present with a history of respiratory and pulmonary problems.⁹ Continued SHSe may exacerbate the pediatric cancer patient's risk for developing second malignancies.¹⁰⁻¹² Youngsters who are exposed to SHS are also more

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Conflict of Interest Statement

The authors declare that they have no competing interests.

Human Subjects Statement

The study was approved by the institutional review board. All human subjects who participated in this study gave approval (via informed consent) prior to participation.

likely to initiate smoking than are those who are not exposed,¹³⁻¹⁶ and adoption of smoking habits can be particularly detrimental to this medically at-risk population.

Policies and regulations that prohibit smoking in public places have largely protected adults from SHSe¹⁷ but do not adequately protect children from exposure to tobacco toxins in their own homes and vehicles, where they spend the majority of their time. In the United States, more than one third of children and adolescents live in homes where residents and visitors smoke regularly, with the majority of SHSe resulting from parental smoking.^{18,19} Despite their increased health risks, children with cancer are as likely to live with smokers as healthy children are.⁹ Notably, the household is the primary source of exposure to SHS for infants and children.²⁰ In 2009, 22.8% of nonsmoking US middle and high school students reported exposure in the car within the past 7 days.²¹

Home smoking restrictions (ie, limiting or banning cigarette smoking in the home) have been associated with reduced SHSe among healthy children and children with asthma as quantified by parental report, nicotine dosimeters, and urine cotinine assays.²²⁻²⁶ Studies have documented that children living in homes with total smoking bans had lower cotinine levels than did children in homes with partial bans or no smoking restrictions.²²⁻²⁷ Adult smokers who live in homes with strict home smoking restrictions are more likely to decrease their daily cigarette consumption,²⁸ have greater motivation to quit or make a quit attempt,²⁹ and have longer periods of sustained abstinence and successful cessation.^{29,30}

The adoption of home smoking bans is influenced by the interaction of individual variables, characteristics of the proximal family and social environments, and the overall environmental contexts in which individuals are embedded.³¹ Demographically, the prevalence of home smoking restrictions is lower among rural, low-income, and low SES households.^{22,23,28,32} However, bans are more likely if the household includes children^{22,23,28,33,34} or a nonsmoking adult,^{22,23,32-34} who often plays a critical role in the establishment of home smoking restrictions. Having a child with a medical condition, such as asthma, residing in the home further increases the likelihood of strict smoking bans.²⁶ The number of smokers in the household is inversely associated with the level of household smoking restrictions.^{28,30,32}

This is the first study to document the prevalence of smoking restrictions in the households of children who live with smokers and are undergoing treatment for cancer. It also evaluates the effects of home smoking restrictions on reported levels of parent cigarette consumption as well as parent-reported measures and biological assays of child SHSe. Child-related medical variables, as well as family and environmental factors,³⁵ often associated with the adoption of home smoking rules, will be examined in families of children with cancer.

METHODS

Participants

Participants were 135 parents or guardians of a child with cancer who lived with at least one adult smoker who smoked in the home or car at the time of recruitment. Parents/guardians were eligible for participation regardless of their own smoking status. Parents were not required to be the identified smoker in the household to participate in the study. Children were eligible for this study if they were younger than 18 years of age, were receiving active treatment for cancer, were at least 30 days post-diagnosis, and were nonsmokers. Parents of children in medical crisis, who had relapsed, had disease recurrence within the past month, or whose disease had progressed, were not recruited for the study. Recruitment took place in the outpatient clinic of a large pediatric oncology hospital between 2002 and 2008. Eligible families were invited to participate in a randomized intervention trial to reduce SHSe among

children with cancer. Parents were compensated for their participation, and children received gift vouchers for providing a urine sample. The data presented represent the baseline data for parents and children who agreed to participate in the trial.³⁶

Procedures

Eligible parents were asked to provide smoking-related information and estimates of their child's SHSe by completing structured interviews. The parent providing this information was designated as the target parent. Children provided urine samples for cotinine analyses. Informed consent was obtained from all parent participants, and children (aged 7 years) provided assent. The study was approved by the institutional review board.

Measures

Parent cigarette consumption—Parents reported the number of cigarettes consumed by all persons who smoked in the home and car over the past 7 days. Responses were used to calculate the all-source, 7-day total cigarette consumption, a measure that has been used and validated in prior studies.³⁷⁻³⁹

Parent-reported child SHSe—Parents were asked to report on the number of cigarettes to which the child was exposed by all persons who smoked in the home and car for the previous 7 days. Exposure was defined as the number of cigarettes smoked in the same room as the child or in the car when the child was present. Responses were used to calculate the all-source, 7-day total parent-reported child SHSe. This measure has been used extensively in other pediatric populations,³⁶⁻³⁸ and acceptable test-retest reliability and validity of parent reports of exposure in children with cancer are reported elsewhere.⁴⁰

Urine cotinine assays—Cotinine is a metabolite of nicotine and a reliable biomarker of recent SHS exposure.³⁸ All samples were obtained from patients in the hospital clinic setting and stored in polyethylene Cryo tubes that were frozen in a standard laboratory freezer at -80 degrees Centigrade. Batched samples were packed in dry ice and shipped to the mass spectrometry laboratories at San Diego State University for analyses. Samples were analyzed for cotinine levels using high-performance liquid chromatography and tandem mass spectrometry methods that are sensitive to low levels of SHSe.⁴¹

Household smoking rules—Household smoking rules were assessed using the following question: "What are the rules, if any, about smoking in your home?" The response options included (1) people can't smoke in the home (total/complete ban); (2) people can only smoke in certain rooms of the house (partial ban); and (3) there are no rules about smoking at home (no ban). Home smoking restrictions have been similarly assessed in other studies.^{30,42} The number of smokers residing in the home was also recorded.

Parental perceived vulnerability—Parents responded to a question that asked about the likelihood that their child would develop serious health problems if exposed to SHS.

Provider communication—Parents were asked how often their child's health care provider communicated with them about smoking.

Stage of change—Target parents who smoked were classified into the precontemplation, contemplation, or preparation stage^{43,44} with respect to intention to quit. For purposes of analyses, precontemplators were combined with contemplators due to similarities in addiction level and history of quit attempts, as done in prior studies.³⁰

Statistical Methods

Descriptive statistics for smoking and exposure outcomes were calculated for the entire sample and according to level of home smoking restrictions. Univariate comparisons were made using the nonparametric Kruskal-Wallis test and the chi-square test or exact chi-square test. Ordinary least squares (OLS) regression models examined the impact of smoking ban on parent report of the child's 7-day SHSe and cotinine levels. Given the skewed distribution of these variables, data were natural log transformed so that more robust results would be obtained as in prior studies.^{38,39,45} One was added to SHSe values prior to transformation given some zero values. Model results were back-transformed by exponentiation, so that presented estimates represent the fold-change in geometric means. Variables significant at $\alpha = .15$ in single-variable models were entered into multivariate models; variables remaining significant at the $\alpha = .15$ level were retained. A logistic model examining variables associated with home smoking restrictions (full ban vs partial/no ban) was also similarly investigated.

RESULTS

Participant Characteristics

Table 1 presents the demographic, medical, and psychosocial characteristics of the study sample according to level of home smoking restrictions and overall. Seventy percent ($N = 95$) of the parents/guardians participating in the trial were self-reported smokers. Close to 23% of smoking parents/guardians in our study intended to quit in the next 30 days and had made a quit attempt in the previous year (preparation stage). Roughly 68% of parents perceived their child to be at risk for developing serious health problems if exposed to SHS. Sixty percent of all parents reported that their child's health care provider never talked with them about smoking. This lack of communication did not differ by the smoking status of the target parent, with 57% of smokers reporting that their child's provider did not talk with them about smoking as compared to 68% of nonsmokers. The majority of our sample had one (48%) or 2 (41%) smokers living in the household. Only one family had 4 smokers. No demographic, child medical, and parent psychosocial variables were significantly associated with level of home smoking restrictions (all p 's $> .05$).

Household Smoking Restrictions

A full smoking ban (ie, no smoking allowed) was reported to be in effect in 58 (43%) of the 135 households; 49 (36.3%) had partial smoking restrictions (smoking allowed in specified rooms); and 28 (21%) had no smoking restrictions in the home (smoking allowed) at baseline. There were no significant differences in the number of smokers who resided in the homes of families with full or partial smoking restrictions compared to those with no smoking restrictions ($p > .10$). Almost 49% of homes with full or partial smoking restrictions had 2-4 smokers living in the home as compared to 46% of those with no smoking restrictions. Sixty-two percent of families with full home smoking bans reported exposing their child in the car as compared to 69% and 75% of families with partial or no smoking restrictions at home, respectively.

Table 2 shows the final logistic regression model predicting full household smoking bans vs partial/no smoking ban combined. Younger age of target parent ($p = .003$) and younger patient age ($p = .001$) were associated with an increased odds of a full smoking ban being implemented in the home. There was a trend for target parents who were married to be 2.2 times more likely to establish full smoking bans compared to parents who had never been married ($p = .064$). Time since diagnosis also met the model inclusion criteria, but was not significant. The final model explained 22% of the variance in the distribution of full home smoking bans.

Parent-Reported Cigarette Consumption and Child SHSe

The median total number of cigarettes smoked by all sources in the home and car over a 7-day period and related reported exposure and cotinine levels according to level of household smoking restrictions are presented in Table 3. Significant differences were reported in cigarette consumption by smokers across households with different smoking rules ($p < .001$). No significant differences were found in the number of cigarettes smoked in the car according to level of home smoking restrictions ($p > .10$). Significant differences were found in cotinine levels obtained from children residing in homes with different smoking restrictions ($p = .002$).

The final multiple variable regression models predicting all-source 7-day total reported exposure and child cotinine levels are shown in Table 4. Compared to households with no ban, those with full smoking bans had a 90% reduction in geometric mean 7-day total exposure ($p < .001$). Those with partial restrictions had a 76% reduction as compared to those with no bans ($p < .001$). Geometric mean exposure was 1.9 times higher for children whose target parent was white as compared to those whose parents were not white ($p = .045$). Children with leukemia had a 47% lower exposure as compared to those with solid or CNS tumors ($p = .019$). Level of home smoking restrictions, parent race, and child diagnosis accounted for 30% of the variation in 7-day total reported exposure.

Households with full smoking bans were associated with a 71% lower level in children's geometric mean cotinine values compared to no bans ($p < .001$) and a 52% lower level compared to partial restrictions ($p = .002$), whereas partial restrictions were nonsignificantly associated with a 33% lower level compared to no smoking restrictions ($p = .147$). Greater parent perceptions of health risks from SHSe ($p = .002$) were associated with lower cotinine levels. There was a 55% lower-level geometric mean cotinine for children from high SES families compared to those from low or middle SES families ($p < .001$). Children whose target parent was married ($p = .010$) or divorced/separated ($p = .038$) had lower cotinine levels as compared to those who were never married. Children whose target parents were smokers had geometric mean cotinine levels that were 2.03 times higher than were those from children whose target parents were not smokers ($p = .003$). Target parent age and race also met model inclusion criteria, but were not significant. The predictors in Table 4 accounted for 40% of the variance in children's cotinine levels. Although the number of smokers in the home was significantly associated with cotinine, it was not significant in the multivariate model after adjusting for smoking status of the target parents, which was highly associated with number of smokers.

DISCUSSION

The results of our study show that a significant proportion of children undergoing treatment for cancer are exposed to SHS in their households. Only 43% of families in our study imposed strict smoking rules within the home, similar to results reported for families of children with asthma.^{25,26,46} Parents often used incomplete procedures to protect their child from SHSe. These included not smoking in areas where the child sleeps or spends significant time or restricting smoking to a single room in the house not necessarily off limits to the child. Children's exposure to smoke in the car was also relatively common in our sample, and those living in households with full home smoking restrictions were often exposed in the car. Car exposure is concerning because it is highly concentrated and may reach unhealthy levels that are more toxic than SHSe in the home setting.⁴⁷ Of note is that during the time period when the study data were collected (2000-2008), there was an increase in the number of states that had enacted comprehensive smoke-free laws in public places,^{17,21} resulting in better population-level protection from SHSe. However, many children continue to be exposed in domestic settings, which depend on adoption of voluntary

smoking restrictions by their families. The finding that many children with cancer live in homes and travel in cars that are not smoke-free is even more disturbing against a background of improved public policy regarding tobacco control.

Full household smoking bans were significantly associated with lower cigarette consumption by smokers in the home as well as lower child SHSe as based on parent report and quantifiable biological cotinine assays. These associations demonstrate construct validity for reported measures, even if not exact estimates of total exposure, as previously reported.^{38,40} Significantly lower cotinine levels were observed in children from homes with a full ban on smoking when compared to those obtained from children living in homes with less restrictive smoking conditions. However, more restrictive household smoking rules did not completely eliminate children's SHSe. This may be due to other sources of exposure not related to the home, such as car exposure, but may be also due to insufficient time since initiation of such rules to see more complete protection from SHSe.

No significant difference was found in the cotinine levels of children residing in homes where smoking was permitted vs those where smoking was partially restricted. Although parent-reported exposure outcomes suggest some benefit for measures that fall short of a full smoking ban (partial ban) relative to no bans, they afford children less protection from SHS, as measured by urinary cotinine. Additionally, partial smoking restrictions are unlikely to offer protection from exposure to high levels of tobacco toxins that are deposited onto household surfaces and that can persist well beyond the period of active smoking.^{27,37,48-50} Dust and surface contaminants in homes and cars and their re-emission into the air can be a source of carcinogenic third-hand smoke exposure (TSHS) for children.⁴⁹⁻⁵¹

Younger parents, and those living with younger children, were more likely to establish smoke-free homes, as demonstrated in previous studies.⁵²⁻⁵⁴ Younger parents may more readily adopt smoke-free homes because they are more likely to have young children in the household whose health is a primary concern. Parents without the support of a partner may have fewer financial resources and less available backup adult supervision of children in the home. Tobacco education programs should, therefore, consider the barriers experienced by families with limited support in order to promote the adoption of smoking restrictions in homes not likely to have them.

The results from this study should be considered within the context of its limitations. First, estimates of homes with smoking restrictions were based on self-reports and not validated by an objective measure; however, a number of studies have supported the validity of such self-reported data in population surveys.^{55,56} It is possible that the hospital setting and collection of information during the child's cancer treatment influenced the degree of accurate disclosure by parents about their household smoking rules and child's exposure. Second, the duration of the home bans was not assessed. Further evaluation of the process of and reasons for adopting home smoking restrictions in households of children with cancer, particularly as they relate to the timing of the child's diagnosis, is warranted. The lower levels of reported exposure among children diagnosed with leukemia in our sample suggest a potential biological, treatment-related, or psychosocial mechanism that should be explored.

Additionally, the collection of children's urine samples and parent reports of exposure were intended to capture exposure during the same one week prior to assessment. Urine samples were obtained from the child at the time the parent completed the exposure reports for the past 7 days. However, only a single urine sample was obtained at the time of assessment, providing accurate estimates of only recent exposure (2-3 days) due to the short half-life of cotinine. Reliance on a single cotinine sample does not preclude the possibility that estimates of exposure were artificially inflated or reduced if the timing of the urine samples

reflected episodic high- or low-level exposure events. For example, children being treated at a smoke-free hospital environment prior to urine sample collection may have had less opportunity for exposure and lower cotinine levels than those of children exposed in other settings prior to their clinic visit. Future studies should require more frequent cotinine measurements to resolve these temporal sampling problems and obtain representative levels of exposure for this patient population.⁵⁷

Of clinical importance is the finding that the majority of parents in our sample had never discussed their smoking habits or their child's SHSe with their child's health care provider. This is consistent with low rates of pediatrician tobacco screening and counseling provided across medical settings.⁵⁸⁻⁶⁰ Advice and counseling about tobacco control may be one of the most cost-effective means of reducing tobacco use or SHSe.⁶¹ Typically, providers ask about smoking and advise smokers to quit. However, child exposure is not fully protected by one parent's quitting, as most smokers have friends and family members who smoke. Providers should be trained and encouraged to direct parents to establish complete tobacco bans in their homes and cars, even if they remain smokers.³¹ Exposure to hospital settings that promote tighter tobacco control may further encourage personal home smoking bans.

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REFERENCES

1. United States Department of Health and Human Services (USDHHS). The Health Consequences of Involuntary Exposure to Tobacco Smoke: A Report of the Surgeon General. United States Department of Health and Human Services, CDC; Atlanta, GA: 2006.
2. Etzel RA. Environmental tobacco smoke. *Immunol Allergy Clin North Am.* 1994; 14:621–633.
3. United States Environmental Protection Agency. Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders. Environmental Protection Agency; Washington, DC: 1993.
4. Cook DG, Strachan DP. Health effects of passive smoking- 10: summary of effects of parental smoking on the respiratory health of children and implications for research. *Thorax.* 1999; 54:357–366. [PubMed: 10092699]
5. DiFranza JR, Lew RA. Morbidity and mortality in children associated with the use of tobacco products by other people. *Pediatrics.* 1996; 97:560–568. [PubMed: 8632946]
6. Benoist MR, Lemerle J, Jean R, et al. Effects of pulmonary function of whole lung irradiation for Wilm's tumour in children. *Thorax.* 1982; 37:175–180. [PubMed: 6285541]
7. Lipshultz SE, Colan SD, Gelber RD, et al. Late cardiac effects of doxorubicin therapy for Acute Lymphoblastic Leukemia in childhood. *N Engl J Med.* 1991; 324:808–815. [PubMed: 1997853]
8. O'Driscoll BR, Hasleton PS, Taylor PM, et al. Active lung fibrosis up to 17 years after chemotherapy with carmustine (BCNU) in childhood. *N Engl J Med.* 1990; 323:378–382. [PubMed: 2370889]
9. Tyc VL, Throckmorton-Belzer L, Klosky JL, et al. Smoking among parents of pediatric cancer patients and children's exposure to environmental tobacco smoke. *J Child Health Care.* 2004; 8:288–300. [PubMed: 15507466]
10. Meisler L. Late effects of childhood cancer therapy. *Curr Probl Pediatr Adolesc Health Care.* 1993; 23:102–131.
11. Neglia JP, Meadows AT, Robison LL, et al. Second neoplasms after Acute Lymphoblastic Leukemia in childhood. *N Engl J Med.* 1991; 325:1330–1336. [PubMed: 1922234]
12. Robison LL, Mertens A. Second tumors after treatment of childhood malignancies. *Hematol Oncol Clin North Am.* 1993; 7:401–415. [PubMed: 8468273]
13. Becklake MR, Ghezzo H, Ernst P. Childhood predictors of smoking in adolescence: a follow-up study of Montreal schoolchildren. *CMAJ.* 2005; 173:377–379. [PubMed: 16103510]

14. Belanger M, O'Loughlin J, Okoli CTC, et al. Nicotine dependence symptoms among youth never smokers exposed to secondhand tobacco smoke. *Addict Behav.* 2008; 33:1557–1563. [PubMed: 18760878]
15. Centers for Disease Control and Prevention (CDC). Exposure to Secondhand Smoke Among Students Aged 13-15 Years -- Worldwide, 2000-2007. *Morbidity and Mortality Weekly Report.* 2007; 56:497–500. [PubMed: 17522587]
16. Seo DC, Torabi MR, Weaver AE. Factors influencing openness to future smoking among nonsmoking adolescents. *J Sch Health.* 2008; 78:328–336. [PubMed: 18489466]
17. Marano C, Schober SE, Brody DJ, Zhang C. Secondhand tobacco smoke exposure among children and adolescents: United States, 2003-2006. *Pediatrics.* 2009; 124:1299–1305. [PubMed: 19841116]
18. American Legacy Foundation. Secondhand Smoke: Youth Exposure and Adult Attitudes-results from Three National Surveys. American Legacy Foundation; Washington, DC: 2005. (Rep. No. 14)
19. Centers for Disease Control and Prevention (CDC). Statespecific prevalence of smoke-free home rules -- United States, 1992-2003. *Morbidity and Mortality Weekly.* 2007; 56:501–504.
20. Pirkle JL, Flegal KM, Bernert JT, et al. Exposure of the US population to environmental tobacco smoke: the Third National Health and Nutrition Examination Survey, 1988 to 1991. *JAMA.* 1996; 275:1233–1240. [PubMed: 8601954]
21. King BA, Dube SR, Tynan MA. Secondhand smoke exposure in cars among middle and high school students—U.S., 2000-2009. *Pediatrics.* 2012; 129:446–452. [PubMed: 22311992]
22. 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; 1:153–162. [PubMed: 11072396]
23. Pizacani BA, Martin DP, Stark MJ, et al. Household smoking bans: which households have them and do they work? *Prev Med.* 2003; 36:99–107. [PubMed: 12473430]
24. Spencer N, Blackburn C, Bonas S, et al. Parent reported home smoking bans and toddler (18-30 month) smoke exposure: a cross-sectional survey. *Arch Dis Child.* 2005; 90:670–674. [PubMed: 15970606]
25. Wakefield M, Banham D, Martin J, et al. Restrictions on smoking at home and urinary cotinine levels among children with asthma. *Prev Med.* 2000; 19:188–192.
26. Wamboldt FS, Balkissoon RC, Rankin AE, et al. Correlates of household smoking bans in low-income families of children with and without asthma. *Fam Process.* 2008; 47:81–94. [PubMed: 18411831]
27. Matt GE, Quintana PJE, Hovell MF, et al. Households contaminated by environmental tobacco smoke: sources of infant exposures. *Tob Control.* 2004; 13:29–37. [PubMed: 14985592]
28. Kegler MC, Malcoe LH. Smoking restrictions in the home and car among rural Native American and white families with young children. *Prev Med.* 2002; 35:334–342. [PubMed: 12453710]
29. Farkas AJ, Gilpin EA, Distefan JM, Pierce JP. The effects of household and workplace smoking restrictions on quitting behaviours. *Tob Control.* 1999; 8:261–265. [PubMed: 10599569]
30. Pizacani BA, Martin DP, Stark MJ, et al. A prospective study of household smoking bans and subsequent cessation related behaviour: the role of stage of change. *Tob Control.* 2004; 13:23–28. [PubMed: 14985591]
31. Hovell MF, Hughes SC. The behavioral ecology of secondhand smoke exposure: a pathway to complete tobacco control. *Nicotine Tob Res.* 2009; 11:1254–1264. [PubMed: 19776346]
32. Okah FA, Choi W, Okuyemi KS, Ahluwalia JS. Effect of children on home smoking restrictions by inner-city smokers. *Pediatrics.* 2002; 109:244–249. [PubMed: 11826202]
33. Borland R, Yong HH, Cummings KM, et al. Determinants and consequences of smoke-free homes: findings from the International Tobacco Control (ITC) Four Country Survey. *Tob Control.* 2006; 15:iii42–iii50. [PubMed: 16754946]
34. Pyle SA, Haddock CK, Hymowitz N, et al. Family rules about exposure to environmental tobacco smoke. *Fam Systems Health.* 2005; 23:3–16.
35. Hovell, MF.; Wahlgren, DR.; Adams, M. The logical and empirical basis for the behavioral ecological model. In: DiClemente, RJ.; Crosby, RA.; Kegler, M., editors. *Emerging Theories in Health Promotion Practice and Research.* 2nd edition. Jossey-Bass, Inc; San Francisco, CA: 2009.

36. Tyc, VL.; Huang, Q.; Nicholson, J., et al. Intervention to reduce secondhand smoke exposure (SHSe) among children with cancer: a controlled trial. *Psycho-Oncology*. 2012. Available at: <http://onlinelibrary.wiley.com/doi/10.1002/pon.3117/abstract>. Accessed August 29, 2012
37. Matt GE, Bernert JT, Hovell MF. Measuring secondhand smoke exposure in children: an ecological measurement approach. *J Pediatr Psychol*. 2008; 33:156–175. [PubMed: 18079169]
38. Matt GE, Hovell MF, Zakarian JM, et al. Measuring secondhand smoke exposure in babies: the reliability and validity of mother reports in a sample of low-income families. *Health Psychol*. 2000; 19:232–241. [PubMed: 10868767]
39. Matt GE, Wahlgren DR, Hovell MF, et al. Measuring environmental tobacco smoke exposure in infants and young children through urine cotinine and memory-based parental reports: empirical findings and discussion. *Tob Control*. 1999; 8:282–289. [PubMed: 10599573]
40. Tyc VL, Lensing S, Vukadinovich CM, Hovell MF. Can parents of children with cancer accurately report their child's passive smoking exposure? *Nicotine Tob Res*. 2009; 11:1289–1295. [PubMed: 19696308]
41. Bernert JT, Turner WE, Pirkle JL, et al. Development and validation of sensitive method for determination of serum cotinine in smokers and nonsmokers by liquid chromatography atmospheric pressure ionization tandem mass spectrometry. *Clin Chem*. 1997; 43:2281–2291. [PubMed: 9439445]
42. Norman GJ, Ribisl KM, Howard-Pitney B, et al. The relationship between home smoking bans and exposure to state tobacco control efforts and smoking behaviors. *Am J Health Promot*. 2000; 15:81–88. [PubMed: 11194699]
43. DiClemente CC, Prochaska JO, Fairhurst SK, et al. The process of smoking cessation: an analysis of precontemplation, contemplation, and preparation stages of change. *J Consult Clin Psychol*. 1991; 59:295–304. [PubMed: 2030191]
44. Prochaska JO, Velicer WF. The transtheoretical model of health behavior change. *Am J Health Promot*. 1997; 12:38–48. [PubMed: 10170434]
45. Hovell MF, Zakarian JM, Wahlgren DR, et al. Reported measures of environmental tobacco smoke exposure: trials and tribulations. *Tob Control*. 2000; 9:iii22–iii28. [PubMed: 10982901]
46. Berman BA, Wong GC, Bastani R, et al. Household smoking behavior and ETS exposure among children with asthma in low-income, minority households. *Addict Behav*. 2003; 28:111–128. [PubMed: 12507531]
47. Ontario Medical Association. *Exposure to Secondhand Smoke: Are We Protecting Our Kids?*. Ontario Medical Association; Toronto, Canada: 2005.
48. Hovell MF, Zakarian JM, Matt GE, et al. Counseling to reduce children's secondhand smoke exposure and help parents quit smoking: a controlled trial. *Nicotine Tob Res*. 2009; 11:1383–1394. [PubMed: 19875762]
49. Singer BC, Hodgson AT, Geuvarra KS, et al. Gas-phase organics in environmental tobacco smoke: 1-effects of smoking rate, ventilation and furnishing level on emission factors. *Environ Sci Technol*. 2002; 36:846–853. [PubMed: 11918006]
50. Sleiman M, Gundel LA, Pankow JF, et al. Formation of carcinogens indoors by surface-mediated reactions of nicotine with nitrous acid, leading to potential thirdhand smoke hazards. *Proc Natl Acad Sci*. 2010; 107:6576–6581. [PubMed: 20142504]
51. Singer BC, Hodgson AT, Nazaroff WW. Gas-phase organics in environmental tobacco smoke: 2---exposure relevant emission factors and indirect exposures from habitual smoking. *Atmos Environ*. 2003; 37:5551–5561.
52. Berg CJ, Cox LS, Nazir N, et al. Correlates of home smoking restrictions among rural smokers. *Nicotine Tob Res*. 2006; 8:353–360. [PubMed: 16801293]
53. Mannino DM, Caraballo R, Benowitz N, Repace J. Predictors of cotinine levels in U.S. children: data from the third National Health and Nutrition Examination Survey. *Chest*. 2001; 120:1–13. [PubMed: 11451802]
54. Okah FA, Okuyemi KS, McCarter KS, et al. Predicting adoption of home smoking restriction by inner-city black smokers. *Arch Pediatr Adolesc Med*. 2003; 157:1202–1205. [PubMed: 14662576]
55. Gilpin EA, Pierce JP, Cavin SW, et al. Estimates of population smoking prevalence: self- vs. proxy reports of smoking status. *Am J Public Health*. 1994; 84:1576–1579. [PubMed: 7943473]

56. Pierce, JP.; Gilpin, EA.; Emery, SL., et al. Tobacco Control in California: Who's Winning the War? An Evaluation of the Tobacco Control Program, 1989-1996. University of California, San Diego; La Jolla, CA: 1998.
57. Matt GE, Hovell MF, Quintana PJE, et al. The variability of urinary cotinine levels in young children: implications for measuring ETS exposure. *Nicotine Tob Res.* 2007; 9:83–92. [PubMed: 17365739]
58. Collins BN, Levin KP, Bryant-Stephens T. Pediatricians' practices and attitudes about environmental tobacco smoke and parental smoking. *Pediatrics.* 2007; 150:547–552.
59. Winickoff JP, McMillen RC, Carroll BC, et al. Addressing parental smoking in pediatrics and family practice: a national survey of parents. *Pediatrics.* 2003; 112:1146–1151. [PubMed: 14595060]
60. Winickoff JP, Tanski SE, McMillen RC, et al. Child health care clinicians' use of medications to help parents quit smoking: a national parent survey. *Pediatrics.* 2005; 115:1013–1017. [PubMed: 15805379]
61. Williams GC, Williams SA, Korn RJJ. Secondhand smoke (SHS) deserves more than secondhand attention: modifying the 5As model to include counseling to eliminate exposure. *Fam Systems Health.* 2005; 23:266–277.

Table 1

Family Demographic and Smoking-Related Characteristics According to Level of Smoking Restrictions in the Home

Characteristic	All N = 135	Full N = 58	Partial N = 49	None N = 28	p-value ^a
Child's Age (in years)	7.6	6.4	9.3	10.2	.285
Median (range)	(0.4-17.7)	(0.4-17.1)	(0.9-17.3)	(1.0-17.7)	
Child's Gender	71 (52.6)	29 (50.0)	30 (61.2)	12 (42.9)	.261
Male – N (%)					
White Child's Race ^b – N (%)	102 (75.6)	45 (77.6)	35 (71.4)	22 (78.6)	.698
Child's Diagnosis					
Leukemia/lymphoma	88 (65.2)	37 (63.8)	31 (63.3)	20 (71.4)	.834
Solid tumor	37 (27.4)	17 (29.3)	13 (26.5)	7 (25.0)	
CNS	10 (7.4)	4 (6.9)	5 (10.2)	1 (3.6)	
Months From Diagnosis	3.0	2.4	2.8	3.7	.299
Median (range)	(1.0-59.5)	(1.0-59.5)	(1.0-37.0)	(1.5-25.1)	
Target Parent Age (in years)	33.9	33.2	35.9	32.4	.771
Median (range)	(19.6-61.2)	(22.3-56.9)	(19.9-54.0)	(19.6-61.2)	
Target Parent Gender	23 (17.0)	10 (17.2)	6 (12.2)	7 (25.0)	.358
Male – N (%)					
White Target Parent's Race ^b – N (%)	108 (80.0)	46 (79.3)	39 (79.6)	23 (82.1)	.950
SES – N (%)					
High	35 (25.9)	14 (24.1)	15 (30.6)	6 (21.4)	
Middle	33 (24.4)	16 (27.6)	14 (28.6)	3 (10.7)	.192
Low	67 (49.6)	28 (48.3)	20 (40.8)	19 (67.9)	
Marital Status					
Married	78 (57.8)	39 (67.2)	24 (49.0)	15 (53.6)	
Divorce/separated	13 (9.6)	7 (12.1)	3 (6.1)	3 (10.7)	.110
Never married	44 (32.6)	12 (20.7)	22 (44.9)	10 (35.7)	
# Smokers ^c – N (%)					
0	5 (3.7)	3 (5.2)	2 (4.1)	0 (0.0)	.869
1	65 (48.2)	27 (46.6)	23 (46.9)	15 (53.6)	
2	56 (41.5)	24 (41.4)	21 (42.9)	11 (39.3)	
3-4	9 (6.7)	4 (6.9)	3 (6.1)	2 (7.1)	
Smoking Target Parent – N (%)	95 (70.4)	43 (74.1)	34 (69.4)	18 (64.3)	.633
Perceived vulnerability					
Serious health problems (very likely/likely) – N (%)	92 (68.2)	38 (65.5)	32 (65.3)	22 (78.6)	.413
Provider Communication – N (%)					
Many times	16 (11.9)	4 (6.9)	6 (12.2)	6 (21.4)	.082
Sometimes	38 (28.2)	17 (29.3)	10 (20.4)	11 (39.3)	
Never	81 (60.0)	37 (66.8)	33 (67.4)	11 (39.3)	
Stages of Change ^d – N (%)					

Characteristic	All N = 135	Full N = 58	Partial N = 49	None N = 28	p-value ^a
Preparation	21 (22.8)	8 (19.1)	8 (25.0)	5 (27.8)	.876
Contemplation	43 (46.7)	22 (52.4)	14 (43.8)	7 (38.9)	
Precontemplation	28 (30.4)	12 (28.6)	10 (31.3)	6 (33.3)	

Note.

CNS = central nervous system; SES, socioeconomic status

^a Kruskal-Wallis test, chi-square or exact chi-square test

^b White and not of Hispanic origin. Remaining patients were black except for 2 whites of Hispanic origin, one Asian, and 3 of more than one race. Remaining parents were black except for one, who was Asian.

^c Only one family had 4 smokers. Five children lived in nonsmoking homes, but were in family situations where there was regular smoke exposure.

^d Only captured for smoking target parents (N = 95). Three of these had missing responses. Refers to stage of parental intentions to quit smoking.

Table 2Predictors of Full Smoking Restrictions in the Home (N = 135) ^a

Effect ^b	Odds Ratio	95% Confidence Intervals		p-value
Married	2.23	0.95	5.53	.064
Divorced/Separated	2.89	0.75	11.08	.123
Target Parent Age: < 25 years	9.09	2.13	33.33	.003
Patient Age (in years)	0.86	0.79	0.94	.001
Time Since Diagnosis: < 6 months	2.00	0.84	4.77	.116

Note.

Pseudo R-square = .22

^a Outcome is full home restrictions vs partial/no home smoking restrictions combined.^b Reference categories are never married, parent age ≥ 25 years, and ≥ 6 months from diagnosis. Patient age was analyzed as a continuous variable.

Table 3

Median (IQR) Reported All-Source Smoking and Exposure Levels and Cotinine Estimates of Exposure Over 7 Days According to Level of Smoking Restrictions in the Home

All Source	All N = 135	Full N = 58	Partial N = 49	None N = 28	p-value ^a
Home					
Smoking	18 (0-102)	0 (0-0)	48.5 (5.5-140.5)	75 (45-210)	< .001
Exposure	0 (0-36)	0 (0-0)	2 (0-48)	47.5 (13.5-123.5)	< .001
Car					
Smoking	7 (0-23)	8 (0-23)	4 (0-19)	10 (0.5-25)	.391
Exposure	3 (0-14)	3 (0-12)	3 (0-11)	7.5 (0.5-18)	.288
Total					
Smoking	42 (7-120)	10 (2-40)	49.5 (22.5-145.5)	102 (59-216)	< .001
Exposure	13 (2-48)	3.5 (0-15)	21 (2-61)	58.5 (24.5-132.5)	< .001
Cotinine (ng/ml)	3.3 (1.5-11.3)	2.1 (1.2-5.4)	6.6 (1.5-15.2)	8.7 (2.8-17.4)	.002

Note.

IQR Interquartile range; ng/ml, nanograms per milliliter

Smoking exposure was missing for 2 families.

^aNonparametric Kruskal-Wallis test

Table 4

Predictors of 7-Day All-Source Reported Exposure and Cotinine

Effect	B ^a	95% Confidence Intervals		p-value
Reported Exposure (N = 135) ^b				
Full ban	0.10	0.05	0.19	< .001
Partial ban	0.24	0.12	0.48	< .001
White target parent's race	1.90	1.02	3.57	.045
Leukemia diagnosis	0.53	0.31	0.90	.019
R-square=0.30				
Cotinine (N = 133) ^c				
Full ban	0.29	0.19	0.55	< .001
Partial ban	0.67	0.39	1.15	.147
Patient age (in years)	0.96	0.92	1.00	.059
White target parent's race	1.48	0.89	2.58	.126
High SES	0.45	0.25	0.64	< .001
Married	0.53	0.33	0.85	.010
Divorced/separated	0.46	0.22	0.96	.038
Smoking target parent	2.03	1.29	3.20	.003
High perceived vulnerability	0.49	0.32	0.77	.002
R-square = .40				

Note.

SES = socioeconomic status

The p-values for Full vs Partial ban for all source exposure and cotinine were both $p = .002$. Results for comparisons with No ban (reference category) are shown above.

^aExposure on natural log scale was analyzed, and then results were back-transformed. Therefore, confidence intervals not containing one indicate significant results. One was added to 7-day exposure prior to transformation to address zero exposure values.

^bReference categories are No ban for Ban, Nonwhite for Race, and Solid or central nervous system tumors for Diagnosis.

^cReference categories are No ban for Ban, Male for Gender, low SES for SES, Never married for Married and Divorced/separated, Nonsmoking for Target parent status, and Low perceived vulnerability (score 1-3) for Perceived vulnerability. Patient age was a continuous variable.