

Intervention to reduce secondhand smoke exposure among children with cancer: a controlled trial

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Abstract

Objective: This randomized controlled trial tested the efficacy of parent-based behavioral counseling for reducing secondhand smoke exposure (SHSe) among children with cancer. It also examined predictors of smoking and SHSe outcomes.

Methods: Participants were 135 parents or guardians of nonsmoking children with cancer, <18 years, at least 30 days postdiagnosis, and living with at least one adult smoker. Parents were randomized to either a standard care control group or an intervention consisting of six counseling sessions delivered over 3 months. Parent-reported smoking and child SHSe levels were obtained at baseline, 3, 6, 9, and 12 months. Children provided urine samples for cotinine analyses.

Results: Reductions in parent-reported smoking and exposure were observed in both the intervention and control conditions. There was a significantly greater reduction in parent-reported smoking and child SHSe at 3 months for the intervention group compared with the control group. Child SHSe was significantly lower at 12 months relative to baseline in both groups. Children's cotinine levels did not show significant change over time in either group. Exposure outcomes were influenced by the number of smokers at home, smoking status of the parent participating in the trial, and the child's environment (home versus hospital) the day before the assessment.

Conclusions: Children's SHSe can be reduced by advising parents to protect their child from SHSe, combined with routine reporting of their child's exposure and cotinine testing, when delivered in the context of the pediatric cancer setting. More intensive interventions may be required to achieve greater reductions in SHSe.

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Introduction

Secondhand smoke exposure (SHSe) is carcinogenic, linked to respiratory and cardiovascular diseases, and represents a leading preventable cause of child morbidity and mortality [1]. The adverse health effects of SHSe among children include increased risk for respiratory illness, ear infections, bronchitis, pneumonia, asthma, and reduced pulmonary function [2–6], and the risk of complication increases with higher levels of exposure [7]. 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 [8–10]. Newly diagnosed children with cancer who are exposed to smoke in their homes are more likely to present with a history of respiratory and pulmonary symptoms [11] and are potentially at risk for acute respiratory complications, particularly if immunocompromised and exposed during treatment. In addition to disease outcomes, youngsters who are exposed to secondhand smoke (SHS) are also more likely to initiate smoking than those who are not exposed [12]. Adoption of smoking habits can be particularly detrimental to survivors of childhood cancer who are already at risk for smoking-related diseases secondary to their disease and treatment [8–10].

Despite recent tobacco control efforts and community policies aimed at reducing SHSe in public places [13], children continue to be exposed to tobacco toxins in their own homes and cars. In the United States, more than one-third of children and adolescents live in homes where residents and visitors smoke regularly [14–17]. Youth exposure to SHS while traveling in the car is also frequent [18,19] and may be 23 times more toxic than SHSe in the home because of the enclosed space [20]. Despite their compromised health status, children with cancer are at risk of being regularly exposed to tobacco smoke throughout their treatment from multiple sources and in numerous settings. It has been reported that between 40% and 46% of newly diagnosed children with cancer live in smoking households that typically include at least one parent smoker [21]. At home, over half of these youngsters are directly exposed to someone's cigarettes smoked in their presence with an even greater percentage of them frequently exposed in the family vehicle.

Interventions that reduce children's SHSe have yielded mixed success [22]. The most successful trials have tested intensive, individualized, parent-based counseling approaches [22]. Reduction in children's exposure to cigarettes [23–25], decreased cotinine levels [26], and decreased air nicotine [27] have been reported in children with asthma and

respiratory problems as well as in healthy children. This study was the first to test the efficacy of a parent-based behavioral counseling intervention to reduce SHSe among children undergoing treatment for cancer. Child and parent sociodemographic characteristics and clinical variables were examined as predictors of parent-reported cigarette consumption and child SHSe as well as child biological cotinine outcomes.

Methods

Participants

Participants were 135 parents or guardians of children receiving treatment for cancer who lived with at least one adult smoker and were exposed to SHS in the home or car setting, per parent report at the time of recruitment. Nonsmoking patients (<18 years), at least 30 days post-diagnosis, were recruited along with their families. Patients/families were excluded if they had a high risk prognosis or had a medical or family social crisis precluding participation. Eligible families were initially identified from

medical records and further screened in person to confirm their eligibility. Figure 1 outlines the number of families enrolled through completion of 12-month measures.

Procedure

Design

Families were randomized (Figure 1) to either an intervention or a standard control group using a stratified, blocked randomization scheme with strata being child’s age (≤5, 6–12, 13–17 years), race (White, non-White), and smoking status of the participating parent (smoker, nonsmoker). Parents/guardians were eligible for participation regardless of their smoking status. Families were followed longitudinally, and parent-reported and child biological measures were obtained five times over 12 months (baseline, 3, 6, 9, and 12 months). Parents provided information about cigarettes they or others smoked and their child’s SHSe by completing structured interviews. The parent who accompanied the child to the hospital for clinical visits and participated in the study was designated as the ‘target’ parent. Parents were compensated for completion of study

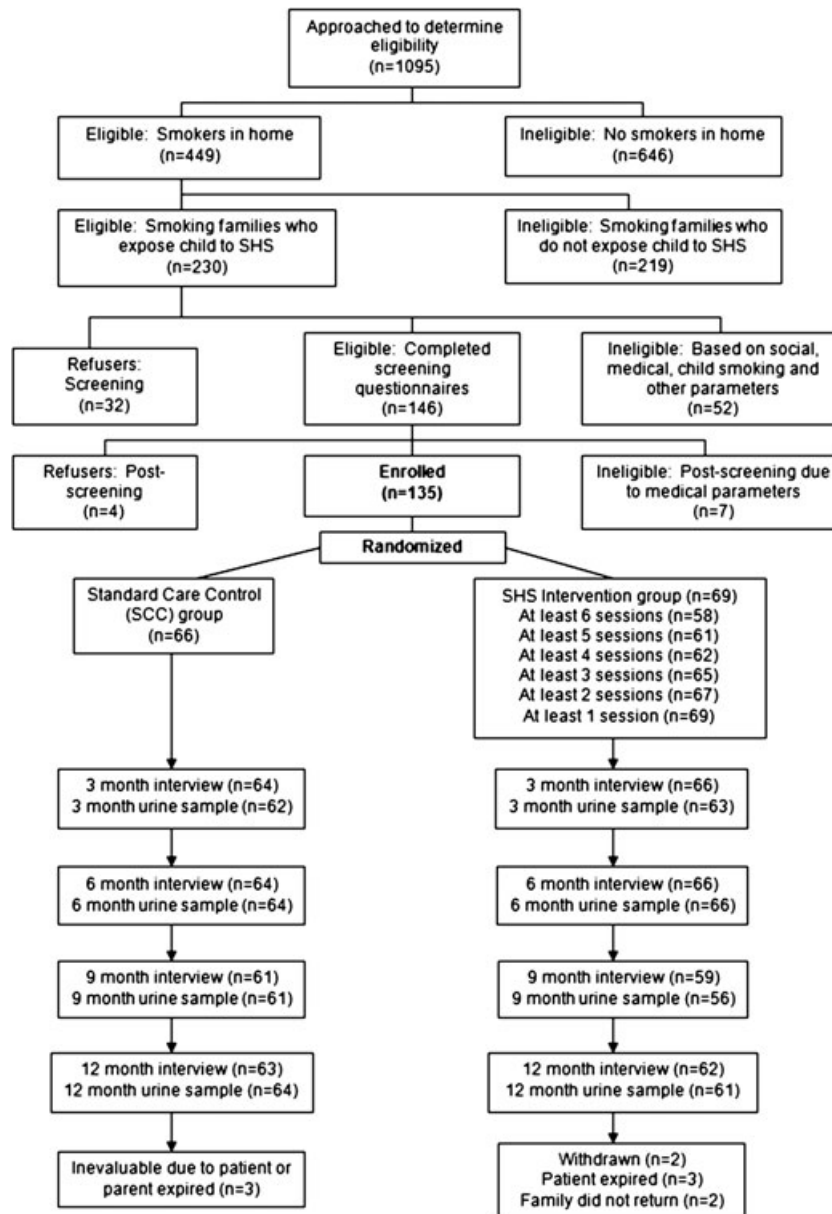


Figure 1. Consort: flow of participants through trial

questionnaires and participation in counseling sessions. Children received gift vouchers for each urine sample provided. Study procedures were reviewed and approved by the Institutional Review Board. All parents signed informed consent agreements and children (aged >7 years) provided assent.

Intervention procedures

Parents in the intervention group received a multicomponent behavioral program delivered by trained counselors over 3 months. Counselors were fully informed about each patient's diagnosis, medical status, and treatment-related complications to enable them to deliver the counseling in the context of the child's ongoing cancer treatment. Counseling consisted of three individual, face-to-face, biweekly 1-h sessions followed by three 25-min telephone sessions for a total of six individual contacts with their counselor. Parents also received letters from their child's physician at the start and end of the counseling phase to acknowledge their participation and progress.

The intervention was based on previous behavioral trials [23–25,28,29] and included behavioral contracting for reducing children's exposure, self-monitoring, problem solving, and social reinforcement for successes. Sessions were designed to gradually shape participants' behavior to remove children from sources of exposure (e.g., their own smoking and smoking by family members and/or friends). Goal achievement resulted in prompting to do more. Parents were provided with literature about SHS-related health risks in children and for stress management. The study did not involve formal cessation counseling. Counselors invited and encouraged all family members to participate in the counseling sessions.

Standard care control group

Parents in the standard care control (SCC) group were asked about their smoking behaviors in the presence of their child and advised about the adverse health problems for children exposed to SHS. Parents were briefly advised to remove their child from sources of exposure and to protect their child from SHSe. This group received all study measures but did not receive SHSe counseling from the study counselors.

Measures

Parent-reported smoking

Parents reported the number of cigarettes smoked by all persons in the home and car over the past 7 days. Responses were used to calculate the all-source, 7-day total smoking as validated in prior studies [30–32].

Parent-reported child secondhand smoke exposure

Parents were asked to report on the number of cigarettes to which the child was exposed by all smoking persons in the home and car for the previous 7 days. Exposure was defined as the number of cigarettes smoked (even one puff) 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. Acceptable test-retest reliability and validity of parent

reports of exposure in children with cancer in relation to cotinine assays are reported elsewhere [33].

Urine cotinine assays

Cotinine is a metabolite of nicotine and a reliable biomarker of recent SHS exposure [30]. All urine samples were obtained from patients in the hospital clinic setting and frozen. Batched samples were packed in dry ice and shipped to the mass spectrometry laboratories at San Diego State University for analysis of cotinine levels. All samples were analyzed using methods that are sensitive to low levels of SHSe [30,31]. Level of detection was less than 0.05 ng/ml.

Parent satisfaction survey

Parents in the intervention group were asked to complete a satisfaction survey that asked about their experience with counseling and requested their feedback about the intervention sessions.

Statistical analyses

Analyses were based on the intention-to-treat rule with 135 families analyzed as randomized ($n = 69$ in the intervention group and $n = 66$ in the SCC group). Descriptive statistics, including means, standard deviations, percentages, and frequencies were reported for selected variables. Given that the distribution of the study outcome measures was highly skewed, geometric means and corresponding 95% confidence intervals were estimated for the intervention and control groups at each time point. *t*-tests were used to examine group and time-point comparisons. Cotinine levels were analyzed after logarithmic transformation.

Linear mixed-effect models (LMM)/generalized linear mixed-effect models (GLMM) were employed using the SAS Procedure PROC Mixed/Nlmixed (SAS, Cary, NC) to address inpatient correlations with respect to repeated measurements over time. To analyze the intervention effect, the LMM/GLMM examined group differences for smoking and exposure outcomes at baseline and across the 12-month study period. In this model, the variables of time (baseline, 3, 6, 9, and 12 months), group (intervention versus control), and the interaction of time by group were included as covariates. Because the greatest reduction for parent-reported smoking and child exposure variables was observed in both groups at 3 months, a spline (discontinuity of slope) was also introduced into the model at the 3-month time point [34]. Outcomes were examined before and after the 3-month time point to test for treatment effects and effects through follow-up. As no such pattern was observed for cotinine outcomes, time was included as a continuous variable without a spline. Akaike's information criterion was used for covariance structure selection with compound symmetry assumption. For each outcome measure, covariates were selected from the following list: sociodemographic (parent and child age, race, gender; parent marital status, and family socioeconomic status), child clinical (diagnosis and time from diagnosis), smoking-related covariates (number of smokers in the home and smoking status of the target parent), the child's location/setting on the day prior to urine collection (hospital campus versus home or other residence), number of counseling sessions received (coded as 0 sessions for the

control group), and all two-way interactions between these variables. All factors significant at level $\alpha=0.15$ in the univariate analyses were investigated in the multivariate model. The final model presented included the factors that were significant at level $\alpha=0.05$.

Results

Participant characteristics

Table 1 presents the sociodemographic and child medical characteristics of the 135 families by intervention and control groups and overall. There were no significant group differences on demographic and child medical variables at baseline except for child gender. Male patients were more likely to be included in the intervention group.

Intervention effects

Parent-reported smoking and child secondhand smoke exposure

Geometric means and corresponding 95% CI values for parent-reported cigarettes smoked and child SHSe by all sources in the child's home and car over a 7-day period for the intervention and control groups at each assessment point are presented in Table 2. There were no significant

group differences for cigarettes smoked and child SHSe at baseline. After adjusting for sociodemographic and child medical covariates, results of GLMM modeling indicated a significant reduction in reported smoking and child SHSe from baseline to 3 months in both the control and intervention groups (Table 3). A significantly steeper decline (greater change in slope) was observed for the intervention group ($p < 0.05$). A 65.8% reduction from baseline to 3 months in parent-reported child SHSe levels was observed for the intervention group compared with a 32.8% reduction for the control group ($p < 0.05$). From 3 to 12 months, the slope (rate of change) for cigarettes smoked and child SHSe slightly increased for the intervention group ($p < 0.05$). For the control group, a slightly decreased slope was observed after 3 months ($p < 0.05$). The group difference for average cigarettes smoked and child SHSe was not significantly different at the 12-month follow-up ($p > 0.05$). However, child SHSe, the target of our intervention, was significantly lower at 12 months relative to baseline in both the intervention and control groups ($p < 0.05$). Figure 2 demonstrates these results for reported child SHSe.

Cotinine

The geometric means and 95% CI for cotinine outcomes for the intervention and control groups at baseline and

Table 1. Demographic and smoking-related characteristics for study sample by group

Parent or child variable	All (N = 135)		Intervention (n = 69)		Control (n = 66)	
	N	%	n	%	n	%
Child gender						
Male	71	52.6	44	63.8	27	40.9
Female	64	47.4	25	36.2	39	59.1
Child race ^a						
White	102	75.6	52	75.4	50	75.8
Non-White	33	24.4	17	24.6	16	24.2
Child diagnosis						
CNS	10	7.4	4	5.8	6	9.1
Leukemia/lymphoma	88	65.2	43	62.3	45	68.2
Solid tumor	37	27.4	22	31.9	15	22.7
Parent gender						
Male	23	17.0	10	14.5	13	19.7
Female	112	83.0	59	85.5	53	80.3
Target parent race ^a						
White	108	80.0	55	79.7	53	80.3
Non-White	27	20.0	14	20.3	13	19.7
Parent SES ^b						
Low	67	49.6	34	49.3	33	50.0
Middle	33	24.4	21	30.4	12	18.2
High	35	26.0	14	20.3	21	31.8
Parent marital status						
Married	78	57.8	36	52.2	42	63.6
Not married	57	42.2	33	47.8	24	36.4
Target parent smoking status						
Smoker	95	70.4	49	71.0	46	69.7
Nonsmoker	40	29.6	20	29.0	20	30.3
Smokers in home ^c						
0 or 1	70	51.9	38	55.1	32	48.5
≥2	65	48.1	31	44.9	34	51.5
	Mean	SD	Mean	SD	Mean	SD
Child age (years)	8.6	5.2	8.9	5.2	8.4	5.1
Parent age (years)	34.7	8.8	34.5	8.4	35.0	9.2
Time since diagnosis (years)	0.58	0.83	0.58	0.83	0.59	0.83

CNS, central nervous system; SES, socioeconomic status.

^a100 White and not of Hispanic origin and two Whites of Hispanic origin. Remaining patients were 27 Black except for one Asian and three of more than one race.

^bSES measured using Hollingshead index [44].

^cOnly one family had four smokers. Five children lived in nonsmoking primary residences but were in homes where there was regular smoke exposure.

Table 2. Parent-reported smoking and child secondhand smoke exposure outcomes at baseline and follow-up

Variable	Baseline	3 months	6 months	9 months	12 months
Geometric mean ^a (95% CI)					
Reported smoking(cigarettes per week)					
Intervention	30.6 (20.0, 46.4)	8.5 (4.7, 14.7)	7.6 (4.3, 13.0)	7.2 (3.9, 12.8)	11.5 (6.7, 19.3)
Control	25.3 (15.1, 41.9)	15.2 (8.6, 26.4)	15.6 (8.5, 27.8)	12.0 (6.6, 21.4)	13.2 (7.3, 23.3)
Reported exposure(cigarettes per week)					
Intervention	13.0 (8.4, 19.8)	2.9 (1.7, 4.7)	3.3 (1.8, 5.5)	3.3 (1.8, 5.5)	4.0 (2.4, 6.4)
Control	9.5 (5.7, 15.4)	6.1 (3.6, 10.1)	5.8 (3.4, 9.5)	4.1 (2.2, 7.1)	4.5 (2.5, 7.7)
Child cotinine levels(ng/ml)					
Intervention	3.9 (2.9, 5.3)	3.9 (2.8, 5.5)	3.8 (2.5, 5.7)	5.3 (3.7, 7.5)	4.0 (2.9, 5.6)
Control	3.7 (2.4, 5.5)	3.5 (2.3, 5.3)	3.1 (2.1, 4.6)	3.6 (2.4, 5.4)	3.9 (2.6, 6.0)

^aPrior to and after calculating the geometric mean, 1 was added and then subtracted to the cigarettes smoked or exposed because there were 0 exposures. This method [45] has been confirmed by simulation.

Table 3. Model estimates: parent-reported smoking, child SHSe, and cotinine

Variable	Estimate	Std error	p-value
<i>Cigarettes smoked</i>			
Intercept	4.133	0.635	<0.001
Intervention and time effect			
Time	-0.048	0.006	<0.001
Spline ^a	0.017	0.007	0.025
Intervention ^b	0.022	0.297	0.940
Time*intervention ^c	-0.151	0.009	<0.001
Spline*intervention ^c	0.210	0.012	<0.001
Predictive covariates			
Two or more smokers in home ^b	0.424	0.022	<0.001
Race (White) ^b	0.688	0.347	0.050
Parent age	-0.033	0.017	0.051
<i>Child SHSe</i>			
Intercept	2.079	0.264	<0.001
Intervention and time effect			
Time	-0.154	0.009	<0.001
Spline ^a	0.131	0.012	<0.001
Intervention ^b	0.332	0.337	0.326
Time*intervention ^c	-0.138	0.015	<0.001
Spline*intervention ^c	0.181	0.019	<0.001
Predictive covariates			
Two or more smokers in home ^b	0.228	0.034	<0.001
Race (White) ^b	0.919	0.398	0.022
<i>Log cotinine</i>			
Intercept	1.829	0.215	<0.001
Intervention and time effect			
Time	0.014	0.008	0.076
Intervention ^b	0.126	0.197	0.523
Predictive covariates			
Two or more smokers in home ^b	0.310	0.125	0.017
Smoking status of target parent (smoker) ^b	0.732	0.223	0.001
Location (on campus) ^d	-0.237	0.056	<0.001

SHSe, secondhand smoke exposure; Std Error, standard error; LMM/GLMM, linear mixed-effect models/generalized linear mixed-effect models.

^aIn the employed LMM/GLMM, a spline was used to indicate the slope change before and after the 3-month time points. The spline variable was constructed in the following way: if time < 3 months, then spline = 0; if time > 3 months, then spline = time - 3 [34].

^bFor the variables 'intervention', 'two or more smokers in home', 'race (White)', and 'smoking status of target parent (smoker)', the reference groups are 'SCC (standard care control)', '0 or 1 smoker', 'non-White', and 'non-smoker', respectively.

^cThe terms 'time*intervention' and 'spline*intervention' were used to indicate the interaction between time and/or spline and the intervention, respectively.

^d'Location (on campus)' indicates the setting where the child spent the majority of time on the day preceding the urine collection (on the hospital campus versus home or other residence).

follow-up time points are presented in Table 2. There were no significant group differences in cotinine levels at baseline. Children's cotinine levels did not show a significant

change over time in either the intervention or control group. No significant group differences or time by group interactions were observed.

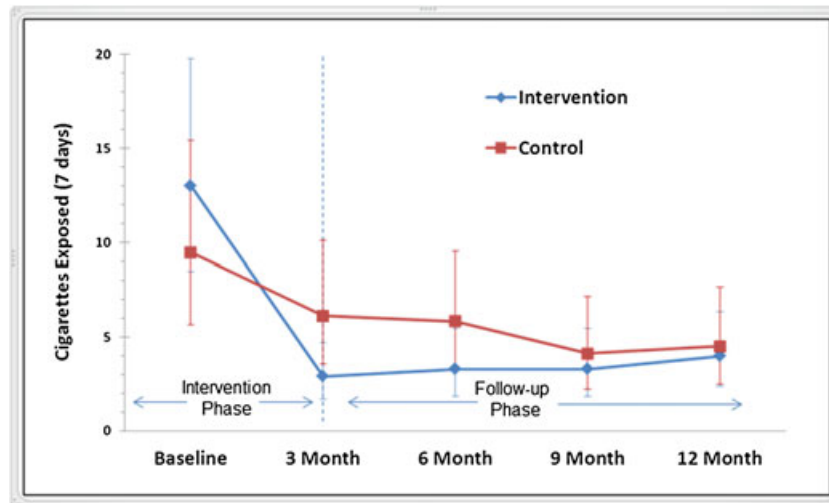


Figure 2. Geometric mean of parent-report child secondhand smoke exposure at baseline and follow-up by group

Predictors of parent-reported smoking and child secondhand smoke exposure

In the model to predict parent-reported smoking from all sources in the past 7 days (Table 3), more than one smoker in the household was significantly associated with a greater number of cigarettes smoked. Parents of white children smoked a marginally greater number of cigarettes, whereas older parents smoked fewer cigarettes. Higher levels of child SHSe were reported among white families and were significantly associated with the presence of more than one household smoker. More than one smoker in the home and a target parent who smoked were significant predictors of higher average child cotinine levels (Table 3). Children who spent the majority of time on the hospital campus (versus other settings) on the day prior to urine sample collection had significantly lower cotinine levels.

Intervention adherence and satisfaction

Eighty-four percent of parents randomly assigned to the intervention group successfully completed all six counseling sessions. Almost 90% completed at least four of the sessions. Feedback from 88.4% (61/69) of parents who participated in our intervention and completed a satisfaction survey was generally positive in terms of the number and content of sessions and the sensitivity of the counseling approach. Almost 92% of parents reported they acquired information regarding the health effects of SHSe and learned specific strategies to reduce their child's SHSe. Approximately 93% of parents reported that the number of session contacts was 'just right'. No concerns were raised regarding the timing of our intervention delivery; almost 97% reported that the information was provided at the appropriate time during the child's treatment, whereas two parents wanted earlier access to this information.

Discussion

This was the first study to test an intervention to reduce SHSe among children undergoing treatment for cancer. In this trial, parent-reported measures indicated significantly greater reduction in children's exposure to cigarette smoke in the intervention group compared with the control group

at 3 months (after completion of the intervention phase). Differential patterns of reduction in SHSe for the two groups indicated that the control families showed a slower and more gradual decline in SHSe over the study period, whereas the intervention group showed a significant initial counseling effect with slight increases in exposure after the first 3 months (during the follow-up phase). Although reported child SHSe levels for families who received the intervention were significantly lower at 12 months relative to baseline, results suggest that maintenance of low levels of exposure and/or greater reduction of SHSe will likely require counseling of greater duration or booster sessions that occur during the follow-up phase.

In this study, reductions in parent-reported exposure were observed in both the intervention and control conditions. The decrease in parent-reported exposure for the intervention and control groups may be partially accounted for by reactivity of standard measurement procedures recognized in prior exposure studies [23,26]. Asking parents to report on their child's exposure, while also sampling their child's urine, may have contributed to the observed reductions in exposure that may not be specifically attributed to behavioral counseling alone. Participants in the control group also received a minimal intervention consisting of brief advice about protecting the child from sources of SHSe. This advice, combined with routine reporting procedures and cotinine testing, may account for observed reductions in exposure observed for the control group. This approach was employed as an ethical minimum for our control participants, which also served to enhance recruitment and cohort retention. Because the trial was conducted in the clinical setting, it is also likely that the setting took on discriminative properties that served to remind parents, in both conditions, of the dangers of SHSe and prompt greater attention to their smoking behaviors around their child. This follows behavioral ecological theory [35], where the child's diagnosis of disease, medical treatment, and clinical setting serve as motivating operations that promote change in parental smoking behaviors in their child's presence.

It is important to note that a variety of patterns in cotinine concentrations have been reported across SHSe studies [23,26,28,36,37]. We did not demonstrate significant differences in cotinine levels between the intervention

and control groups over time. Our findings are similar to those of Greenberg and colleagues [37] who demonstrated decreased exposure levels based on parent reports but no significant reductions in urine cotinine levels. This finding should not diminish the significance of the study. Although cotinine is an endpoint of interest for evaluating health risk, the more proximal targeted behavior (i.e., fewer cigarettes smoked in the child's presence) that is relevant for testing the efficacy of our behavioral intervention was significantly changed. Reliance on cotinine, by itself, for evaluating the effects of the intervention may result in faulty conclusions.

The finding that cotinine levels did not change over the course of the trial in the context of reduced parent-reported exposure may have several possible explanations. First, a reporting bias on the part of the parent respondents should be considered. It is possible that the hospital setting and delivery of the intervention during the child's treatment could influence the degree of accurate disclosure by parents about their smoking and child's SHSe. Another potential reporting bias was that the smoking status of the target parent may have influenced the accuracy of the reported exposure. Smoking parents tend to provide more accurate estimates of child SHSe than nonsmoking parents [32,33] because they are often the sources of the child's exposure. Additionally, parents may not be able to accurately report on exposure that is not directly observed. Lastly, the definition of reported 'exposure' for our trial, and many others, required that the child be present in the room or car when a cigarette was smoked. Yet, children may be in close proximity to a room where smoking occurs, or they may enter a room soon after cigarettes are smoked, thus exposing them to SHS contaminants. Therefore, our parent-reported measures were not as inclusive in measuring all sources of SHSe, as was cotinine.

On a related note, it is likely that residual exposure could account for the failure to obtain significant reductions in children's cotinine levels, even when parents reported reduced smoking in the home or other environments or smoked when the child was not at home. It is now well known that children are at particular risk to thirdhand smoke exposure (THSe) through contamination of home surfaces by volatile SHSe components that can be off-gassed into the air and affect cotinine levels [38–40]. It is likely that the homes of families in our study may have been contaminated for some time, suggesting that children may have been exposed to THSe, which was not separately measured in this study. The fact that families in our study were not required to completely ban smoking in their homes and cars and were permitted to implement less restrictive options to protect their children from SHSe (e.g., restricting smoking to certain rooms of the home, smoking in the home when the child is not present) was not sufficient to offset the risk from THS. Complete elimination of smoking in the home and/or car for an extended period would be necessary to result in substantive reductions in both SHS and THS exposures that are measured by the child's cotinine levels. Our study and the counseling interventions conducted to date have not sufficiently focused on this goal.

The considerable variability in cotinine levels for children in our sample, as noted in prior studies [41], may reflect differences in opportunity for exposure and account for the lack of a differential reduction in cotinine levels

between the intervention and control groups. Because of treatment-related schedules and the distance some families travel to receive treatment, patients were not consistently in their primary residence or in the same location/setting in the 24 hours prior to urine sample collection. Therefore, children spending more time on the smoke-free hospital campus setting prior to urine sample collection likely had less opportunity for exposure, as reflected in lower cotinine levels.

The collection of urine samples and parent reports in this study were intended to capture exposure during the same 1-week period prior to assessment. Urine samples were obtained at the time the parent completed the exposure reports for the past 7 days. However, only a single urine sample was obtained at each assessment point, providing accurate estimates of only recent exposure (2–3 days) because of the short half-life of cotinine [30]. With a single urine sample, it is also possible that our estimates of exposure were artificially biased if the timing of the urine samples reflected episodic high or low level exposure events. More frequent and targeted cotinine measurements, preferably in the child's home environment, may be necessary to obtain more representative levels of exposure for this mobile patient population [39]. Travel, time away from home, and treatment schedules may present methodological challenges for cotinine measurement among other pediatric populations, in addition to cancer, who often cannot be treated locally.

An important observation from this trial was that only 16% of families assigned to the intervention group did not complete all six counseling sessions, and the intervention was delivered without high rates of withdrawal at follow-up. These findings, combined with the positive feedback from parents, support their willingness to participate in multisession tobacco-based counseling during their child's cancer treatment. However, families of children with cancer may require even more powerful clinical interventions, which also include decontamination procedures to reduce THSe [42,43], to ensure larger reductions in SHSe as measured by both parent report and cotinine levels. A logical next step would be an intervention trial that requires complete home and car smoking bans with longer follow-up intervals to capture reductions in home contamination that contribute to child cotinine measurements. Our finding that more smokers in the home was associated with higher SHSe and child cotinine levels highlights the need to engage multiple family members, particularly parent smokers, in the counseling process. Repeated advice by health care providers to prohibit smoking in all environments inhabited by children is critical. An ongoing dialogue about smoking and exposure between the clinical team and families at each clinical contact may be the most ecological way to reduce SHSe for children with cancer.

Acknowledgements

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Conflicts of interest

The authors declare that they have no conflicts of interest.

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