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Jody S. Nicholson and Vida L. Tyc

St. Jude Children's Research Hospital, USA

Shelly Lensing

University of Arkansas for Medical Sciences, USA

Abstract

Children with cancer are at greater risk for the negative consequences of secondhand smoke exposure, making the identification of predictors of exposure critical. The current study investigated the impact of parents' psychosocial variables (perceived stress and vulnerability, self-efficacy), as well as health-related and demographic variables, on children's current exposure levels. Data were from 135 families whose children ($M = 8.6$ years old) lived with a smoker and were being treated for cancer. Self-efficacy was the consistent significant psychosocial predictor of exposure and the time since a child's diagnosis was indicative of lower exposure when limiting the sample to only smoking parents ($n = 95$). Both predictors of exposure have implications on motivation for behavioral change and may be suggestive of a teachable moment. Interventions may profit from tailoring programs to families based on these predictors of exposure, in particular for tobacco-based interventions for parents of medically compromised children, such as children with cancer.

Keywords

Medically compromised children, pediatric cancer, secondhand smoke exposure, self-efficacy

Introduction

Secondhand smoke exposure (SHSe) is a major public health concern affecting premature death and disease in both children and adults who choose not to smoke (United States Department of

Corresponding author:

Jody S. Nicholson, Department of Psychology, St Jude Children's Research Hospital, Memphis, TN 38105, USA.

Email: jody.nicholson@stjude.org

Health and Human Services (USDHHS), 2006). The adverse health effects of SHSe to children are well established and include coughing, asthmatic symptoms, middle ear infections, bronchitis, pneumonia, sudden infant death syndrome, and reduced pulmonary function. In addition to poorer health outcomes, exposure has also been linked to negative outcomes across cognitive and behavioral domains, such as externalizing (hyperactivity and aggression) and internalizing problems (depression), as well as cognitive deficits in memory, numeracy, attention and processing speed, and verbal fluency (Llewellyn et al., 2009; Robinson and Kirkcaldy, 2007; Yolton et al., 2008).

These negative consequences may be even greater for children undergoing treatment for cancer, secondary to diagnostic and treatment related toxicities that may affect their respiratory, pulmonary, and cardiovascular functioning (Benoist et al., 1982; Lipshultz et al., 1991; O'Driscoll et al., 1990; Robinson and Kirkcaldy, 2007). Newly diagnosed children with cancer who live in homes where smoking is permitted are more likely to present with a diagnosis of respiratory and pulmonary symptoms, and are potentially at risk for acute respiratory complications, particularly if they are exposed during treatment when immune-compromised. Additionally, due to restrictions that come with the diagnosis and treatment of cancer, children may not be able to leave the environment in which they are being exposed and consequently may spend more time indoors in the presence of toxic SHSe (Matt et al., 2008; Tyc et al., 2004a, 2008).

In addition to negative health outcomes, SHSe increases the likelihood that children will adopt smoking themselves (Bettcher et al., 2007; Farkas et al., 2000; Song et al., 2009). Children with parents who smoke are more likely to become smokers. In contrast, a smoke-free home and parent cessation has been inversely associated with adolescent smoking initiation (Farkas et al., 1999, 2000). Adoption of smoking can be particularly detrimental to children with cancer given the association between smoking and greater risk for cancer among childhood cancer survivors. In fact, adopting smoking conferred a 20-fold increase in the risk of developing a secondary lung cancer for cancer survivors who had received irradiation and chemotherapy for the treatment of Hodgkin's lymphomas (Nathan et al., 2009). Therefore, parents should be concerned that their smoking habits will not only directly impact their medically compromised children in the short-term via exposure, but that modeling of this behavior may lead to an increased risk for secondary cancers if their children adopt smoking. Despite these concerns, approximately 40 percent of children with cancer are at risk for being exposed to SHS throughout their treatment by parents and other caregivers, and in numerous settings including the home and car (Tyc et al., 2004b)

Due to the risks and consequences of exposure, community and clinical-based programs have targeted the parents of medically compromised children for smoking cessation and SHSe reduction programming (Emmons et al., 2001; Hovell et al., 1994, 2000, 2002, 2009; Tyc and Throckmorton-Belzer, 2006; Tyc et al., 2008; Wahlgren et al., 1997; Winickoff et al., 2003a, 2003b). These interventions employ theories of behavioral change such as the Health Belief Model and Social Learning Theory, which identify factors that motivate parents to protect their children from SHSe. Psychosocial constructs of particular interest for children with cancer are those which may be impacted as a result of the children's diagnosis and treatment, such as parental stress, self-efficacy, and perception of vulnerability, due to heightened concerns for protecting their child's health (Eiser, 1998; Strecher et al., 1993). A child's health status may prime parents for a 'teachable moment', during which time they may be more motivated for behavioral change (Gehrman and Hovell, 2003; Winickoff et al., 2005).

For these reasons, it is important to investigate parent psychosocial variables which may influence child SHSe and reflect the interpretation and judgment of an event (i.e. Health Belief Model)

and/or expectancies and judgments about outcomes (i.e. Social Learning Theory), especially in the context of the child's cancer (Gehrman and Hovell, 2003; Rosenstock et al., 1988). The current study explored the relationship between parents' perceived vulnerability, self-efficacy and stress, and SHSe outcomes among children with cancer. Specifically, parents' self-reports of their perception of their children's vulnerability to smoke-related health risks, their self-efficacy to control children's SHSe, and their perceived stress were examined as predictors of children's exposure, as measured by urine cotinine and parental self-reports. Because the smoking status of the parent has been shown to influence reported exposure outcomes (Tyc et al., 2009), participating parents who were smokers were analyzed separately, as well as together with the complete sample, which included some non-smokers. We also examined the influences of demographic and medical variables, such as the child's age, time since diagnosis, and diagnosis, for their impact on child SHSe.

Method

Participants

One hundred and thirty-five parents or guardians of children with cancer who lived with at least one adult smoker in the home participated in the study. Families were recruited in the outpatient clinic of a large pediatric oncology hospital. Parents/guardians were eligible for participation regardless of their smoking status. Patients 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 non-smokers. Moreover, patients could not have a poor prognosis, be at high risk for malignancies, in relapse, or have had a medical crisis in the past month. Eligible families were invited to participate in a randomized intervention to reduce SHSe among pediatric cancer patients. Parents were compensated for their participation. Children received gift vouchers for each urine sample provided. Study procedures were reviewed and approved by the Institutional Review Board, Biostatistical Committee, and Clinical Protocol Scientific Review and Monitoring Committee at St Jude Children's Research Hospital. All parents signed informed consent agreements and children (aged ≥ 7 years) provided assent. The data presented here are from the baseline assessment for parents and children who agreed to participate in the SHSe reduction trial.

Procedure

Eligible parents were asked to provide information about their children's SHSe by completing self-report measures, as described below. One parent, deemed the 'target' parent in the project, participated in the study. Parents were eligible regardless of their smoking status – of the 135 parent participants, 95 were smokers. Children provided urine samples for cotinine analyses.

Measures

Demographic and diagnostic variables. The age, gender, race, socioeconomic (Hollingshead, 1975) and marital status of participants were collected through a self-report questionnaire. The number of smokers in the household was also provided via self-report. Information about the child's age, gender, diagnosis, and the time since diagnosis was obtained from medical record reviews.

Psychosocial variables

SHSe self-efficacy. Parents rated their confidence in their ability to maintain a smoke-free environment for their child under 10 different conditions. They were asked to rate their confidence with a 4-point scale from 'not at all confident' to 'very confident.' The first six items were administered to all participants (Cronbach's $\alpha = .82$); the final four were only relevant to smoking individuals as they asked how confident parents felt in their ability to control their smoking behaviors around their children to reduce their exposure (Cronbach's $\alpha = .81$). Items are summed to create a total score with higher scores indicating higher levels of self-efficacy in relation to the ability to control children's SHSe. Past research has shown that mothers who reported low self-efficacy expectations tended to have infants with the highest levels of SHSe (Strecher et al., 1993). The scale has demonstrated good internal validity (Cronbach's $\alpha = .85$) with a sample of mothers with healthy infants and good construct and predictive validity (Strecher et al., 1989, 1993).

Perceived Vulnerability (PV) to general health and tobacco-related problems. A 14-item measure was administered to assess parent's perception of their child's current vulnerability to smoking-related health risks: 'I am worried about my child's exposure to second-hand smoke because he/she is being treated for cancer', and the future: 'Later health problems can be prevented if my child is not exposed to second-hand smoke'. Items were ranked on a 5-point Likert scale from 'strongly agree' to 'strongly disagree' and summed so higher scores indicated higher perceptions of vulnerability. The current measure was adapted from a shorter 8-item, 5-point scale version that was specifically developed for assessing perceived vulnerability to general health and tobacco-related problems while being treated for cancer (Tyc et al., 2006, 2009). Adequate internal consistency (Cronbach's $\alpha = .73-.74$) was established during prior versions of the measure (Tyc et al., 2003, 2006). Good internal consistency was established for the current study (Cronbach's $\alpha = .87$).

Perceived Stress Scale (PSS). A 14-item self-report measure of perceived stress measured one's degree to which situations were perceived as unpredictable, uncontrollable, and overwhelming. Seven positive and seven negative questions were measured from 'never' to 'very often' on a 5-point Likert-type scale. In its development, the scale had adequate reliability and validity and was correlated with social anxiety, life-event scores, and depressive and physical symptomatology, but was differentially predictive of depressive symptomatology (Cohen et al., 1983). Internal reliability was .82 for the current study.

Exposure outcome variables

Parent-reported child SHSe. Parents were asked to report on the number of cigarettes to which the child was exposed by all persons living in the home, including themselves, for the previous seven days. Exposure was defined as smoking that occurred in the same room or car in the presence of the child on each of the last seven days. These estimates were summed and divided by seven for an average daily measure of parent-reported child SHSe from all sources. Acceptable test-retest reliability and validity of parent reports of exposure in relation to cotinine assays in children with cancer and other diseases are reported elsewhere (Hovell et al., 2002; Tyc et al., 2009).

Urine cotinine assays. Urine samples were collected from children and analyzed for cotinine levels. Cotinine is a metabolite of nicotine and a reliable biomarker of recent SHS exposure (Matt et al., 1999) and has been used previously in studies with medically compromised children such as children with cancer and asthma (McIntosh et al., 1994; Tyc et al., 2009; Wakefield et al.,

2002). Obtained samples were frozen in a standard freezer with tubes labeled with a randomly assigned identification number for laboratory use. Batched samples were packed in dry ice and shipped to the mass spectrometry laboratories at San Diego State University, San Diego, California for analyses of cotinine levels. All samples were analyzed by a high performance liquid chromatography and tandem mass spectrometry method that is sensitive to low levels of SHS exposure (Bernert et al., 1997). The reported minimum level of detection was .1 ng/ml.

Statistical methods. Descriptive statistics (e.g. means, standard deviations, percentages) were computed for demographic, psychological, and exposure variables, and Spearman's rank correlations were calculated between psychological and exposure variables. For the total sample, which included both nonsmoking and smoking parents, and the smoking parent sample, multiple linear regression models were performed separately for children's average daily exposure from all sources over seven days and children's urine cotinine assays. The independent variable selection process involved entering into the model variables that were univariately significant at $p < .20$ and retaining variables that remained significant at $p < .20$. Due to the skewed distributions of exposure variables, the data were natural log transformed. Model results have been back-transformed by exponentiation to the original scale, so that presented estimates represent the fold-change in geometric mean exposure. Data were analyzed using SAS 9.1.

Results

Descriptive statistics

Table 1 provides the demographic and medical characteristics of child and parent participants. Children were predominately Caucasian and diagnosed with leukemia/lymphoma. Seventy percent of target parents were smokers. Table 2 reports summary statistics and correlations for psychosocial and exposure outcome variables: self-efficacy (general questions), self-efficacy (administered to smoking-parents only), perceived vulnerability, stress, children's urine cotinine, and parent's reports of all-source exposure. There were moderate correlations of urine cotinine and average daily all-source exposure with the self-efficacy subscales. Parental psychosocial variables were not associated with demographic and treatment characteristics, although stress was significantly and negatively correlated with parental age ($p < .05$) and SES ($p < .01$).

Multivariate models. Tables 3 and 4 present model results for the total sample ($n = 135$) and smoking parent sample ($n = 95$). Of the variables pertaining to family demographics and medical characteristics of the child (see Table 1), time since diagnosis, parent and patient age, SES, marital status, and target parent smoking status met model inclusion criteria and were retained in one or more models. Psychosocial predictors of perceived stress and perceived vulnerability did not meet the variable inclusion criteria for many of the models. However, self-efficacy remained a significant variable across models predicting child SHSe.

Full sample. Self-efficacy was significantly associated with child urine cotinine levels after adjusting for parent's age, SES, marital status, and smoking status ($p < .001$; Table 3). An 11 percent reduction in geometric mean cotinine level was evident for each unit increase in self-efficacy ($\beta = .89$; 95% CI = .85–.94). When assessing demographic variables, urine cotinine was 2.8 times

Table 1. Child and parent demographic characteristics for total study sample ($n = 135$)

Child variables	
Age in years – M (SD)	8.6 (5.2)
Range	.4 – 17.7
Gender	47.4% Female/52.6% Male
Race	
Caucasian	75.6%
African-American	20.0%
Other ¹	4.4%
Diagnosis	
CNS	7.4%
Leukemia/lymphoma	65.2%
Solid tumor	27.4%
Months since diagnosis – M (SD)	7.0 (9.9)
Range	1.0–59.5
Parent variables	
Age in years – M (SD)	34.7 (8.8)
Range	19.6–61.2
Gender	83.0% Female/17.0% Male
Marital status	
Married	57.8%
Divorced/separated	9.6%
Never married	32.6%
SES ²	
Low	49.6%
Middle	24.4%
High	25.9%
Target parent status	
Smoker	70.3%
Non-smoker	29.6%
Smokers in home ³	
0, 1	51.9%
2 or more	48.1%

Notes: ¹White with Hispanic origin ($n = 2$), Asian ($n = 1$), and more than one race ($n = 3$). ²Hollingshead score of 4 or 5 is Low, 3 is Middle, and 1 or 2 is High. ³Five children lived in nonsmoking primary residences but were in homes where there was regular smoke exposure.

higher for children whose target parent was a smoker as compared to those whose target parent was a non-smoker. There were also observed differences in cotinine levels according to marital status and SES with a 47 percent reduction for married parents as compared to singles and a 51 percent reduction for high SES families as compared to low SES ones. A respectable amount of variance in urine cotinine was accounted for in the model ($R^2 = .35$).

Self-efficacy remained a significant predictor in the model predicting reported average daily all-source exposure after adjusting for parent's age and smoking status ($p < .01$; Table 3) with every unit in self-efficacy conferring an 18 percent reduction in all-source exposure ($\beta = .82$; 95% CI = .77–.87). The only significant demographic variable was parental age; for every 10 year increase in parental age there was a 26 percent reduction in geometric mean reported exposure. The model explained 26 percent of the variance in reported all-source exposure ($R^2 = .26$).

Table 2. Spearman’s rank correlations and descriptive statistics for parental psychological and child exposure variables for total study sample

	1	2	3	4	5	6
	Efficacy total	Efficacy smoke	Vulnerability	Stress	Urine cotinine (ng/ml)	All-source exposure (cigarettes/day)
1	—	.66**	.17	-.21*	-.27**	-.46**
2	—	—	.30**	-.36**	-.41**	-.45**
3	—	—	—	-.19*	-.17	-.10
4	—	—	—	—	.16	.19*
5	—	—	—	—	—	.51**
6	—	—	—	—	—	—
N	135	93	135	134	134	135
M	18.8	13.4	57.9	26.1	3.9 ¹	1.6 ¹
SD	4.2	2.8	7.1	7.5	—	—

Notes: ¹Geometric means presented since exposure measures were logged transformed given their skewed distribution. The interquartile range was 1.5–11.3 for urine cotinine and .3–6.9 for average daily all-source exposure. *0.01 < p < 0.05; **p < 0.01

Table 3. Psychosocial, treatment related, and demographic predictors of SHSe in the full sample

	Urine cotinine (n = 134)		All-source exposure (n = 135)	
	Beta ¹	95% CI ¹	Beta ¹	95% CI ¹
Parent’s age (per 10 years)	.84	.66–1.06	.74*	.55–1.00
SES ² : High vs low	.49*	.30–.82	—	—
Middle vs low	1.16	.69–1.93	—	—
Marital: Married vs single	.53*	.33–.84	—	—
Divorced vs single	.53	.25–1.13	—	—
Smoking status: Smoker vs non-smoker	2.78**	1.76–4.38	1.75	.98–3.11
Efficacy ³ (per unit)	.89**	.85–.94	.82**	.77–.87

Notes: ¹Due to the skewed distributions of exposure variables, data were natural log transformed for analyses. The results above have been back-transformed (exponentiated), so that the results represent fold-change in geometric means. ²Hollingshead score of 4 or 5 is Low, 3 is Middle, and 1 or 2 is High. ³Efficacy was measured by six items given to all participants, including nonsmokers. *0.01 < p < 0.05; **p < 0.01.

Smoking-only sample. Self-efficacy continued to remain the only significant psychosocial predictor for models predicting urine cotinine and average daily all-source exposure when restricted to the sample of 95 smoking parents (both p < .01; Table 4). Although perceived stress did meet model inclusion criteria, it was not statistically significant for either model (p > .10). For the model predicting children’s urine cotinine in the sample of smoking parents, each unit increase in self-efficacy was associated with a 16 percent reduction in the geometric mean child cotinine level after adjusting for other variables (β = .84; 95% CI = .76–.91). Time since diagnosis was a significant predictor in the model; children who were less than six months from diagnosis had a 44 percent reduction in geometric mean urine cotinine as compared to those who were six months or more from diagnosis. Parents’ age was also associated with children’s urine cotinine levels. For each 10 year increase in parents’ age, there was a 35 percent reduction in geometric mean exposure.

Table 4. Psychosocial, treatment related, and demographic predictors of SHSe in the smoking target parent sample

	Urine cotinine (n = 91)		All-source exposure (n = 92)	
	Beta ¹	95% CI ¹	Beta ¹	95% CI ¹
Child's age (per year)	1.06	1.00–1.13	—	—
Months from diagnosis: <6 vs. ≥ 6	.56*	.34–.94	.57	.29–1.10
Parent's age (per 10 years)	.65*	.44–.95	—	—
Smoking Efficacy (per unit)	.84**	.76–.91	.78**	.70–.88
Perceived Stress (per unit)	1.03	.99–1.06	1.04	.99–1.09

Notes: ¹Due to the skewed distributions of exposure variables, data were natural log transformed for analyses. The results above have been back-transformed (exponentiated), so that the results represent fold-change in geometric means. ²Efficacy was comprised of four items specific to the smoker's ability to limit their smoking around their children. *0.01 < p < 0.05; **p < 0.01.

Similarly, for the model predicting all-source exposure, each unit increase in self-efficacy demonstrated a reduction of 22 percent in exposure ($\beta = .78$; 95% CI = .70–.88). For the subset of smoking parents, the models explained 30 percent of the variation in urine cotinine and 26 percent of the variation in all-source exposure.

Discussion

This is the first study to investigate the relationship of parent psychosocial variables and SHSe outcomes among children with cancer. Self-efficacy was the strongest psychological predictor of children's SHSe, as measured by parent report and urine cotinine levels for both the full sample and when limiting the sample to smoking parents. Perceived stress and perceived vulnerability were not statistically associated with outcomes. Even after adjusting for other treatment and demographic factors, self-efficacy remained highly significant. The only other factor that was significant at the $\alpha = 0.01$ level was the smoking status of the target parent. The current findings are not surprising given self-efficacy is the most theoretically grounded construct in health behavior change that was included in the current study (e.g. Health Belief Model and Bandura's Social Learning Theory). This replicates prior research linking parental self-efficacy and children's exposure (Streicher et al., 1993), and suggests that self-efficacy may be an important psychosocial construct when considering children's SHSe and, subsequently, could be an integral tool when designing interventions to reduce children's exposure.

Past studies involving health behavior change in smoking research have indicated self-efficacy is an important correlate to individuals' motivation to change as a result of health concerns (Boardman et al., 2005; McBride et al., 2003; Rosenstock et al., 1988). Consequently, research focused broadly on health behavior change has proposed that participants' self-efficacy be enhanced prior to intervention in order to be more cost-effective and ensure successful completion of programming (Hevey et al., 1998; Hovell et al., 2002; Tyc et al., 2009). Furthermore, self-efficacy has been found to be an influencing factor on motivation to quit smoking (Boardman et al., 2005), and even if cessation is not an immediate outcome, individuals demonstrating higher self-efficacy are more likely to move towards quitting (Warnecke et al., 2001). Consequently, prior successful interventions have been designed to increase self-efficacy in order to elicit changes in smoking households (Emmons et al., 2001), and past studies on health behavior change have linked decreases in

self-efficacy with program non-adherence and drop-out (Gwaltney et al., 2009; Lechner and DeVries, 1995).

Demographic characteristics were also important in explaining children's SHSe. Demographic covariates were consistent with past findings which suggest a link between smoking and poverty and single mother status (Emmons et al., 1994; Stuber et al., 2008). This highlights the potential importance of considering the social context in which families reside when working with parents to change their children's SHS exposure (Emmons et al., 2001). Assisting low-income and single-mother families to reduce their children's exposure may necessitate different intervention approaches.

The finding that children more recently diagnosed were exposed to less cigarette smoke among the sample limited to only smoking target parents has important implications for tobacco-control efforts with parents of children under treatment for cancer. This suggests that there may be a teachable moment during the early stages of treatment when families may be more sensitive to the health consequences of their smoking for their children and more likely to make health behavior changes (Gehrman and Hovell, 2003). McBride and colleagues (2003) outlined a teachable moment heuristic suggesting that whether an event is significant enough to warrant change in a health behavior is likely impacted by cognitive, emotional, demographic, or health related variables (McBride et al., 2003). The child's cancer diagnosis, medical care, and clinical treatment setting may, therefore, serve as powerful motivators for parents who aim to reduce their smoking in the child's presence.

Clinical implications

The current study suggests children of parents who report high self-efficacy and whose children are closer to diagnosis are exposed to less SHS. The relationship between lower exposure, more recent diagnoses, and higher perceived self-efficacy to control their children's SHSe may have implications for parents' motivation for behavioral change; parents with high self-efficacy and those whose children were more recently diagnosed may be primed for a teachable moment. Parents of children beginning treatment for cancer could be targeted for smoking cessation and exposure reduction interventions because they may be particularly receptive to programming. In a similar manner, measures of self-efficacy could be used as a screener by clinicians to identify parents who may be more receptive to smoking programs and who may already be taking steps towards reducing their child's tobacco-related health risks. Individuals who score lower on this measure may benefit from first undergoing an intervention to improve their self-efficacy before participating in an SHSe reduction trial (Strecher et al., 1993). Past empirical findings, combined with theory, suggest that the assessment of parental self-efficacy may help clinicians determine where to begin with parents in order to improve the success of SHSe reduction interventions.

Limitations and future directions

The current study is a first step in contributing empirical data on parental psychosocial predictors of SHSe for children with pediatric cancer. Future research could investigate other psychosocial variables that could be influential contributors of child SHSe. Moreover, perceived stress remained a predictor in the model for the smoking parents, though it was non-significant. Because it is well established that stress influences smoking habits (Gehrman and Hovell, 2003; Perkins and Groner, 2006; Steptoe et al., 1996), perceived stress should also be considered when predicting SHSe and

may play a more critical role in families of other pediatric populations. Further examination of the impact of time since diagnosis on parents' motivation is warranted to more closely examine the optimal time to implement tobacco-control efforts with families of children with cancer.

Limitations from the current study include the lack of a comparison group which limits the extrapolation of the findings beyond parents of children currently under treatment for pediatric cancer. Furthermore, because of the unique population, measures had to be constructed specifically for the study, although good reliability of these measures was obtained. As the intervention from which this baseline data was drawn is completed, further analyses will investigate the role of self-efficacy and time since diagnosis on intervention effectiveness.

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