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Preliminary Exploration of Secondhand Smoke Exposure in Youth with Sickle Cell Disease: Biochemical Verification, Lung Functioning, and Health Care Utilization

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Abstract

Exposure to secondhand smoke (SHSe) can compound an already challenging set of health problems for youth with Sickle Cell Disease (SCD). Exposure to electronic cigarettes may also impact SCD symptomology and respiratory functioning. This study built on these previous findings by a) utilizing two objective measures of SHSe, salivary cotinine and exhaled carbon monoxide and b) examining the association of SHSe with emergency department utilization, physician-reported sickle cell crises, and pulmonary functioning. Caregivers ($N=31$ dyads; $M_{age}=37.6$ years; $SD=8.5$ years) of youth with SCD ($M_{age}=9.0$ years; $SD=4.5$ years) completed self-report measures of tobacco/nicotine use. Youth provided a saliva sample to assess cotinine levels, and performed pulmonary function tests. Dyads provided breath samples for measurement of eCO. A chart review was conducted to obtain medical history and utilization. Majority of youth (88%) were exposed to SHS via salivary cotinine. Interestingly, no significant associations were observed between youth cotinine levels and emergency department utilization, physician-reported sickle cell crises, or pulmonary functioning. Present findings indicate a need to assess for SHSe using objective assessment measures. Future research should investigate which specific factors contribute to increase SHSe/SHVe in youth with SCD.

Keywords

Sickle Cell Disease; Secondhand Smoke Exposure; Pulmonary Functioning; Health Care Utilization

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Sickle Cell Disease (SCD) affects an estimated 90,000 to 100,000 individuals in the US (Lanzkron, Carroll, & Haywood Jr, 2013; Ware, de Montalembert, Tshilolo, & Abboud, 2017; Yawn et al., 2014). Youth with SCD face numerous chronic symptoms and negative health outcomes, including stroke, pulmonary hypertension, acute chest syndrome, and pain associated with vaso-occlusive episodes, known as sickle cell crises (Lanzkron et al., 2013; Ware et al., 2017; Yawn et al., 2014). Recent literature has indicated that exposure to and use of tobacco may exacerbate sickle cell crisis, decrease pulmonary functioning, and increase health care utilization (Cohen et al., 2013; Glassberg, Wang, Cohen, Richardson, & DeBaun, 2012; Sadreameli, Eakin, Robinson, Alade, & Strouse, 2016; West et al., 2003). Several studies demonstrate an association of secondhand smoke exposure (SHSe) with increased rates of pulmonary morbidity and increased health care utilization in youth with SCD. These studies indicated that SHSe may a) double the risk for sickle cell crises (West et al., 2003); b) increase emergency department utilization rates (Glassberg et al., 2012); c) increase airway obstruction and decreases bronchodilator responsiveness (Cohen et al., 2013); and d) increase rates of hospitalization (Sadreameli et al., 2016).

The present pilot study sought to: 1) employ objective measures of SHSe, salivary cotinine and exhaled carbon monoxide (eCO) and to assess SHSe in youth with SCD in a primary care clinic, and 2), assess children's exposure to caregiver use of electronic cigarettes, which produce a visible aerosol containing nicotine and other toxicants (i.e., secondhand 'vapor'; SHVe) (Czogala et al., 2013; Goniewicz et al., 2013; Goniewicz & Lee, 2014). ECs are being utilized by caregivers for a variety of reasons, including smoking reduction and cessation (Garbutt et al., 2015). Currently, few studies have examined the amount of SHV nonusers may be exposed to (Ballbè et al., 2014; Cervellati et al., 2014). Therefore, information assessing SHVe in youth, particularly those with SCD, is needed. To our knowledge, no previous exploration of SHVe has been conducted in youth with SCD ((Cohen et al., 2013; Glassberg et al., 2012; Sadreameli et al., 2016; West et al., 2003)). We assessed if SHSe/SHVe among children with SCD was associated with increased ED visits and increased physician-reported SCD crises, admission for acute chest syndrome, and decreased pulmonary functioning.

Method

Thirty-one youth with SCD (SC Type SS = 45.2%, $M_{age} = 9.0$ years; $SD = 4.5$ years) and their caregivers ($M_{age} = 37.6$ years; $SD = 8.5$ years) completed procedures during scheduled clinic visits at a Midwestern children's hospital. Inclusion criteria were: 1) primary caregiver of a child with SCD; 2) children under 17-years; 3) SCD diagnosis, and 4) English speaking/reading. All families meeting the inclusion criteria were approached, regardless of endorsement of nicotine/tobacco use. If interested, informed consent/child assent, questionnaires, and saliva and breath samples were collected. A medical chart review determined number of ED visits. Each dyad was compensated \$30 for participation. Parent-child dyads ($n = 35$) were approached; 4 dyads declined due to being "too busy" ($n = 2$) or "not interested" ($n = 2$); 31 parent-child dyads completed the study protocol for a participation rate of 89%. See Tables 1 and 2 for caregiver and youth demographics.

Measures

Child and Caregiver Variables:

Exhaled Carbon Monoxide (eCO): Caregivers and children provided breath samples for assessment of eCO as a measure of SHSe. Caregiver eCO was used to confirm smoking status; eCO<10 ppm indicated confirmed smoking abstinence (Jarvis, Russell, & Saloojee, 1980; Sato et al., 2003) (Deveci, Deveci, Açık, & Ozan, 2004).

Child Variables

Spirometry.—To obtain an estimate of pulmonary functioning, children over the age of six ($N = 24$) completed 5 trials of forced spirometry. Spirometric indices were automatically recorded for the “best” test (Barreiro & Perillo, 2004).

Salivary Cotinine.—Children provided saliva for assessment of cotinine levels, an objective biomarker of SHSe (Benowitz, Hukkanen, & Jacob III, 2009). All samples were processed utilizing Salimetrics® specifications.

Caregiver Variables

Current and previous nicotine/tobacco use was assessed via self-report surveys. Caregivers reported past 30-day and previous or ever-use of tobacco. Caregivers who denied current cigarette use, but indicated past use were categorized as “former smokers”; caregivers who reported current cigarette use were categorized as current smokers. Caregivers who answered “no” to both questions were categorized as “never smokers.” To assess household SHSe or SHVe, caregivers reported how many individuals in the household smoked and/or used an EC. A household SHS/SHVe variable was created by dichotomizing exposure as “yes” or “no.”

Data analytic plan

Descriptive statistics were conducted on demographic variables and child/caregiver variables, including smoking status (i.e., child cotinine, eCO), child’s medical history (e.g., number of acute chest and SCD pain crises), child pulmonary functioning, and healthcare utilization. Correlation analyses and chi-square tests of independence were conducted to assess relationships between child cotinine, child ED utilization, number of sickle cell crises, and number of acute chest syndrome, caregiver smoking status, and household SHSe/SHVe. Nonparametric statistics were utilized to assess caregiver smoking status (i.e., “never,” or “current/ever”) and household exposure via SHS/SHV on health-related child outcomes (i.e., ED utilization, sickle cell crisis, acute chest syndrome).

Results

All medical information and biochemical data are reported in Table 2. Child Spirometry. Lung function results were in the average range ($FEV1\ 80\%–120\% = 90.9\%$; $FEV1/FCV > 70\% = 54.5\%$) for all youth, regardless of SC-type or SHSe. Caregivers and child eCO levels were consistent with nonsmokers. No child values were above 10 ppm and only two caregivers were above 10 ppm.

Child Salivary Cotinine: SHSe/SHVe.—Due to centrifuge malfunction, four samples were unable to be processed. Of the remaining 27 samples, 24 (88%) indicated child SHSe ($M_{cotinine} = .92$, $SD = 1.28$, $range = 0.00$ ng/mL – 5.47 ng/mL). Mean cotinine values for children of caregivers who reported personal use and/or household SHSe/SHVe values ranged from 0 ng/ml to 5.5 ng/ml ($N = 9$; $M_{cotinine} = 1.36$ ng/mL, $SD = 1.87$ ng/mL). The mean cotinine values for children of caregivers who indicated no SHSe/SHVe ranged from 0 ng/ml to 1.07 ng/ml ($N = 18$; $M_{cotinine} = 0.63$ ng/mL, $SD = 0.38$ ng/mL). See Table 2 for child cotinine values and figure 1 for mean cotinine values across child age.

Child Variables and Caregiver Smoking Status.—Significant correlations were observed for child cotinine and caregiver smoking status ($r = .48$). Caregiver smoking status was correlated with household nicotine/tobacco use ($r = .69$). The number of sickle cell crises was correlated with ED utilization ($r = .86$) and number of acute chest syndrome ($r = .69$). No significant association was observed between caregiver smoking status and sickle cell crisis $X^2(1, N = 31) = .06$, $p = .58$, $phi = -.16$, ED visits $X^2(1, N = 31) = .17$, $p = .68$, $phi = .16$ or acute chest events $X^2(1, N = 31) = .01$, $p = .98$, $phi = .14$ in the past 30 days or the past 1 year (sickle cell crisis $X^2(1, N = 31) = .12$, $p = .73$, $phi = -.14$; ED visits $X^2(1, N = 31) = .01$, $p = .93$, $phi = .09$; acute chest events $X^2(1, N = 31) = <.001$, $p = 1.00$, $phi = .08$). Similarly, no significant interaction was observed for household SHSe/SHVe and sickle cell crisis $X^2(1, N = 31) = 1.93$, $p = .13$, $phi = .34$, ED visits $X^2(1, N = 31) = .91$, $p = .23$, $phi = .25$, or acute chest events $X^2(1, N = 31) = .00$, $p = .99$, $phi = .04$.

Discussion

Our findings highlight the variability of different assessment techniques (e.g., caregiver report vs. eCO vs. child cotinine). Similar to past research (Sadreameli et al., 2016), the majority of our sample demonstrated SHSe or SHVe, identified via salivary cotinine. Cotinine analyses indicated that 24 of the 27 participants (88%) were exposed, in some capacity, to SHS/SHV. Interestingly, no child eCO values were elevated and only two caregivers (both self-identified as current smokers) were elevated, highlighting the variability of two different measures of SHSe and the benefit of utilizing salivary cotinine to measure SHSe/SHVe among children.

Several findings in the present study are not consistent with literature. Specifically, no associations were observed between SHSe/SHVe and health care utilization, pulmonary functioning, or increased disease severity (Cohen et al., 2013; Glassberg et al., 2012; Sadreameli et al., 2016; West et al., 2003). This could be due to the limited sample size or missing information about health events in medical records (e.g., received care at another medical facility/ED). Possibly, families did not report acute health events, which then went undocumented in their records.

The present study highlights some important considerations. First, caregiver-reported SHSe/SHVe were poor indicators of actual SHSe/SHVe, with the majority of these reports incorrectly indicating no exposure. Twenty-four of 27 children in the present study showed evidence of SHSe/SHVe as measured by salivary cotinine. Nine caregivers endorsed exposure via cigarettes or ECs. Additionally, children with the highest cotinine values were

aged 10 years and younger, consistent with estimates that 4 out of 10 U.S. children aged 3–11 years experience SHSe (Homa et al., 2015). The high prevalence of nicotine/tobacco exposure may also be a result of the limited tobacco control policies within the state in which the study was conducted given that stronger policies or greater coverage of state-level smoke-free policies are associated with a lower prevalence of adult smoking (Dinno & Glantz, 2009; Eriksen, Serrum, & Bruusgaard, 1996; Wakefield et al., 2008; Wilson et al., 2012). Consistent with the current sample, tobacco use trends in the US indicate significant SHSe/SHVe for children from lower socioeconomic households (Hiscock, Bauld, Amos, Fidler, & Munafo, 2012). The current results are limited in generalizability due to the small sample size ($n = 31$) and cross-sectional design. The small sample size and the high prevalence of SHSe/SHVe prevented the utilization of inferential statistics for some outcome variables. Additionally, SHSe may not be attributed to a primary caregiver per se, but may be SHSe/SHVe from other sources.

Conclusion

Future studies need to evaluate SHSe/SHVe in youth with SCD. Given the limitations of SHSe/SHVe information via eCO and self-, parent-, and physician-report, future studies should utilize biochemical verification, salivary or serum cotinine, to assess exposure and trends among health-related outcomes including pulmonary functioning, ED utilization, and sickle cell crises.

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Note. All values are reported in ng/mL.

Figure 1.
Child Cotinine Values by Child Age.

Table 1

Caregiver/Child Demographics

Variable	Percent
Caregiver Age (years; <i>M</i> (<i>SD</i>))	37.6 (8.5)
Child Age (years; <i>M</i> (<i>SD</i>))	9.0 (4.5)
Relationship to Child	
Mother	87%
Child Gender	
Male	42%
Female	58%
Caregiver/Child Ethnicity	
African American	90%
White	3%
Middle Eastern	7%
Child Sickle Cell Type	
SS	45%
SC	42%
S/Beta 0 Thalassemia	3%
HgbS/HgbD	3%
HgbS/Beta+Thalassemia	3%
Unspecified	3%
Annual Family Income	
0 – 20,000	33%
21,000 – 40,000	33%
41,000 – 50,000	17%
60,000 or Greater	17%
Education Level	
High school graduate	45%
College graduate	32%
Master level graduate	7%
Undisclosed	16%
Marital Status	
Married	47%
Separated/Divorced	17%
Never Married	37%
Primary Insurance	
Private	7%
Employer Provided	33%
Medicare	7%
Medicaid	41%

Variable	Percent
No Insurance	11%

Note. Percent of participants reported, unless otherwise noted.

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Table 2

Medical Information and Biochemical Data

Variables	<i>M (SD)</i>
Caregiver Exhaled CO (ppm)	3.9 (4.5)
Child Exhaled CO (ppm)	4.4 (2.7)
Child Diagnosis of Asthma (% yes)	37% (n=10)
Child Cotinine (ng/mL)	92 (1.28)
Number of Emergency Department Visits	
30 days	3 (.7)
365 days	1.5 (2.5)
Number of Sickle Cell Crises	
30 days	2 (.5)
365 days	1.0 (2.1)
Number of Acute Chest Syndrome Occurrences	
30 days	1 (.3)

Note. CO = Carbon Monoxide; ppm = parts per million; ng/mL = Nanogram/milliliter; means (standard deviation), unless otherwise noted. The range for nonsmoking levels of eCO are values under 10 ppm. The range for nonsmoking adolescent cotinine values are: Non-hispanic whites 3 ng/mL; non-hispanic blacks 3 ng/mL; Mexican-Americans 1 ng/mL.