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High Cotinine and Healthcare Utilization Disparities Among Low-Income Children

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Abstract

Introduction: This study assesses the associations of child salivary cotinine, parent-reported smoking, and child tobacco smoke exposure with number of child healthcare visits and hospital admissions over a 6-month period. This study also assesses relationships between participant characteristics and child cotinine.

Methods: Longitudinal data were evaluated from a sample of 313 clinically ill children aged 0–9 years who lived with a smoker and presented to a pediatric emergency department or urgent care during 2016–2018. In 2020, cotinine measurements were log-transformed, and Poisson and linear regression were performed.

Results: The majority of children came from low-income homes (66.1%) and had public insurance/self-pay (95.5%). Child cotinine ranged from 0.1 to 332.0 ng/mL (geometric mean=4.8 ng/mL, 95% CI=4.1, 5.5). Poisson regression results indicated each 1-unit increase of log-cotinine was associated with an increase in pediatric emergency department visits over a 6-month period following the baseline visit with an adjusted RR of 1.16 (95% CI=1.01, 1.34). Each 1-unit increase of log-cotinine was associated with an increase in the frequency of hospital admissions over the 6-month period with an adjusted RR of 1.50 (95% CI=1.08, 2.09). No differences were found between parent-reported smoking or child tobacco smoke exposure and healthcare utilization. Linear regression results indicated children who were younger ($\beta = -0.227$, $p=0.049$), white (geometric mean=5.5 ng/mL), had medical history of prematurity (geometric mean=8.1 ng/mL),

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and a winter baseline visit (geometric mean=6.5 ng/mL) had higher cotinine. Children living in apartments (geometric mean=5.5 ng/mL) and multi-unit homes (geometric mean=5.5 ng/mL) had higher cotinine than those in single-family homes (geometric mean=3.6 ng/mL).

Conclusions: Routine biochemical screening could identify children who are in need of intensive tobacco smoke exposure reduction interventions.

INTRODUCTION

Tobacco smoke exposure (TSE) in children is a significant source of preventable morbidity and mortality.[1,2] The national prevalence of this "pediatric disease"[3,4] is about 38%.[5,6] Direct health consequences of child TSE are vast, and knowledge about these costly effects are increasing over time.[1,2] There is strong evidence supporting the relationship between TSE and respiratory symptoms and illnesses.[2] Children with TSE have higher rates of cough, difficulty breathing, wheezing, ear pain, asthma exacerbations,[7,8] tonsillectomies and adenoidectomies, and hospitalizations compared with unexposed children.[1,9]

Although it is known that TSE levels are highest among children who are younger, impoverished, racially/ethnically diverse, and live in rented homes,[5,6] limited information is available on the healthcare burden associated with TSE among this particular population. The majority of work has relied on self-reported healthcare utilization, self-reported TSE, and biochemically measured TSE in children aged ≥3 years; focused on children with specific diagnoses (e.g., asthma); and used a comparison group of unexposed children.[7,10–15] Though this research is beginning to fill an important gap, these relationships have not been biochemically validated or verified with electronic medical record (EMR) documentation among young children. The measurement of cotinine, a widely used nicotine biomarker,[16] and rigorous EMR reviews of racially and ethnically diverse children with varied TSE levels and clinical illnesses would provide an objective approach to assess these associations and identify potential TSE-related disparities.

The objective of this study is to determine whether salivary cotinine, parent-reported smoking, and child TSE are associated with number of healthcare visits and hospital admissions over a 6-month period among children aged 0–9 years with TSE. Children with higher cotinine are hypothesized to have a higher number of healthcare visits and hospital admissions following the baseline visit compared with children with lower cotinine. Children with parents who smoke a higher number of daily cigarettes and are around a higher number of cigarettes smoked per week by all smokers in all locations are posited to have a higher number of healthcare visits and hospital admissions. To identify TSE-related disparities, this study assesses whether cotinine is associated with child characteristics, parent characteristics, and baseline healthcare visit-related characteristics. Children who are younger, non-Hispanic black, and live in apartments or multiunit homes are hypothesized to have higher cotinine than children who are older, non-Hispanic white, and live in single-family homes. Owing to enrolling a homogenous sample of clinically ill children who live with a smoker, it is posited that there will be no differences between cotinine and healthcare visit-related characteristics.

METHODS

Study Sample

From April 2016 to August 2018, a convenience sample was recruited of nonsmoking pediatric patients who presented to a large, Midwestern children's hospital pediatric emergency department (PED) or urgent care (UC). Details about the study protocol are available elsewhere.[17] In brief, pediatric patients were eligible if they were accompanied by a parent who smoked tobacco, presented with a possible TSE-related complaint (e.g., cough) as defined by the U.S. Surgeon General,[1] and were clinically stable as confirmed by a practitioner. Parents were eligible if they were daily cigarette smokers, and were ineligible if they exclusively used smokeless tobacco, e-cigarettes, or were using smoking-cessation medications. If two parents were present and eligible, parents decided which one would be enrolled into the study and only that parent provided data. Parents and their children were recruited and enrolled at baseline. The convenience sample included 313 patients aged 0–9 years who had complete baseline data that included cotinine results, EMR data, and parent assessment data. A hospital IRB approved all procedures including parental consent and EMR data extraction.

Measures

Trained research staff obtained saliva samples from children during the baseline visit, which was analyzed to assess cotinine using liquid chromatography tandem mass spectrometry. These measurements detect low TSE levels with high sensitivity and specificity, at an assay level of quantification of 0.1 ng/mL.[18] All samples had detectable cotinine with 2 at the level of quantification.

Cotinine >1.0 ng/mL is a commonly used cut point to classify children with biochemically confirmed TSE.[19–21] A sensitivity analysis was performed to assess whether child, parent, or visit characteristics differed based on cotinine indicative of TSE (>1 ng/mL) versus no/minimal cotinine (≤1 ng/mL). A total of 84.7% had cotinine >1.0 ng/mL (n=265). Sensitivity analyses showed that cotinine groups differed based on insurance and housing type. Children with cotinine <1 ng/mL had significantly higher percentages of commercial insurance (14.6%, p<0.001) and living in a single-family home (60.4%, p<0.008) than children with cotinine ≤1 ng/mL (2.6% and 36.6%, respectively).

Child EMR data and parent assessment data were assessed for sociodemographic information that included child sex, age, race (white, black, other), ethnicity, insurance type (commercial or public/self-pay), and housing type (single-family home, multi-unit home, apartment). Given previously identified TSE patterns by age group,[20] age-specific categories (i.e., 0–1, 2–4, and 5–9 years) were assessed. Other races included Asian, multiple races, and other race not listed. Data were extracted from EMRs on TSE-related past medical history (PMH) and surgical history. TSE-related PMH included respiratory conditions (asthma, bronchiolitis, and pneumonia), otitis media, and prematurity. TSE-related surgical history included tonsillectomy and adenoidectomy.

Parents reported their sex, daily number of cigarettes smoked, and number of cigarettes smoked around their child by all smokers (e.g., other parent) in all locations (e.g., home, car) in the past week.

Data were extracted specific to the baseline visit including season, year, treatment location (PED or UC), triage level, TSE-related chief complaint, disposition, and primary discharge diagnosis. PED triage level ranged from 2 (high acuity) to 3–5 (moderate to low acuity); chief complaints included cough, congestion, difficulty breathing, and wheezing; disposition included discharge to home and admitted to the hospital; and discharge diagnosis included asthma, bronchiolitis, pneumonia, upper respiratory infections/croup, otitis media, conjunctivitis, and non-TSE-related diagnoses.

Longitudinal data were abstracted from children's EMRs on the number of revisits to the PED/UC within 30 days of discharge from the baseline visit. This is a commonly used timeframe to assess potentially preventable revisits.[22,23] Data were extracted on the total number of PED visits, UC visits, and inpatient hospital admissions over the 6-month period following children's baseline visits. The 6-month timeframe varied for each child because they were followed prospectively from their baseline PED/UC visit.

Statistical Analysis

Data were analyzed in 2020, and log-transformed cotinine values were used to address skewed distributions. Linear regression models were built to explore the relationships between child and parent characteristics and log-cotinine. Spearman correlations were used to assess associations between parent-reported smoking and child TSE and log-cotinine. Linear regression was conducted to assess the associations between baseline visit: season, year, treatment site, triage level, chief complaint, disposition, and discharge diagnosis and log-cotinine. Four Poisson regression models were tested to assess associations between log-cotinine and the healthcare utilization outcomes, while controlling for child sociodemographics, PMH, parent sex, and baseline visit season and year. As a sensitivity analysis, zero-inflated Poisson regression was used to model excess zeros and possible overdispersion due to the nature of the visit data, and the results were very similar to the Poisson model results. The type I error was set at 0.05 (2-tailed).

RESULTS

Child mean age was 3.2 (SD=2.8) years. Most parents (86.6%) were female and 47.9% of children were female (Table 1). The majority of children were black (56.3%), of non-Hispanic origin (97.4%), and lived in single-family homes (40.3%) or apartments (36.7%). Two thirds (66.1%, n=207) of children had an income ≤$15,000 and 95.5% had public insurance or were self-pay. Most children were treated in the UC (73.5%) and were discharged to home (92.6%). About 14% of children had a respiratory-related PMH of asthma, bronchiolitis, or pneumonia; 44.4% had a chief complaint of cough or congestion and 24.0% reported difficulty breathing or wheezing. The top two diagnoses were upper respiratory infection/croup (45.1%) and otitis media (18.8%) (Table 2). Cotinine levels ranged from 0.1 to 332.0 ng/mL (geometric mean [GeoM]=4.8 ng/mL, 95% CI=4.1, 5.5; median=5.1 ng/mL, IQR=1.8–12.4).

Linear regression results with log-cotinine as the outcome indicated there was a negative association between child age and log-cotinine (β= −0.227, p=0.049). Children aged 2–4 years (GeoM=3.7 ng/mL, p=0.045) had lower cotinine than children aged 0–1 years (GeoM=5.6 ng/mL) (Table 1). Children of other race (GeoM=2.6 ng/mL, p=0.02) had lower cotinine than white children (GeoM=5.5 ng/mL). Children living in apartments (GeoM=5.5 ng/mL, p=0.009) and multi-unit homes (GeoM=5.5 ng/mL, p=0.009) had higher cotinine than children living in single-family homes (GeoM=3.6 ng/mL). Children with PMH of prematurity (GeoM=8.1 ng/mL, p=0.04) had higher cotinine than those without this PMH (GeoM=4.5 ng/mL).

Children with a baseline visit during winter (GeoM=6.5 ng/mL, p=0.04) had higher cotinine than children with a visit during summer (GeoM=4.3 ng/mL) (Table 2). No other differences were found based on other visit characteristics and log-cotinine.

Mean daily cigarettes smoked was 10.5 (SD=6.3) cigarettes by parents (range=2–30) and 10.0 (SD=21.4) cigarettes per week by all smokers in all locations (range=0–224) (Table 1) with an average of 2.4 (SD=1.3) smokers around the child in the past week (range=l-7). The number of daily cigarettes smoked by parents (ρ=0.11, p=0.007) and weekly cigarettes smoked around the child by all smokers in all locations (ρ=0.08, p=0.008) were positively correlated with child log-cotinine.

Overall mean of revisits was 0.2 (SE=0.02) with 15.7% (n=49) having a past 30-day revisit. Mean past 6-month PED visits and UC visits were 0.4 (SE=0.05) and 0.6 (SE=0.05) with 26.4% (n=83) and 39.5% (n=124) having at least one PED or UC visit, respectively. Mean past 6-month hospital admissions were 0.1 (SE=0.02) with 5.5% (n=17) having an admission.

Using log-cotinine and controlling for the covariates, Poisson results indicated that each 1-unit increase of log-cotinine was associated with an increase in past 6-month PED visits following the baseline visit with an adjusted RR of 1.16 (95% CI=1.01, 1.34, p=0.04) (Table 3). Concerning hospitalizations, each 1-unit increase of log-cotinine was associated with an increase in past 6-month hospital admissions following the baseline visit with an adjusted RR of 1.50 (95% CI=1.08, 2.09, p=0.02). No differences were found between number of daily cigarettes smoked by parents or past-week cigarettes smoked around the child by all smokers in all locations and healthcare visits and hospitalizations.

DISCUSSION

This study assessed associations between cotinine, parent-reported smoking behavior and child TSE, and the number of past 6-month healthcare visits and hospital admissions among clinically ill, racially/ethnically diverse children who lived with a parental smoker. As hypothesized, child cotinine during the baseline visit was associated with a higher number of PED visits and hospital admissions. For each 1-unit increase of log-cotinine, children were at a 16% excess risk for PED visits and 50% excess risk for hospital admissions over the 6-month period. Although parent smoking behavior and child TSE were positively correlated with child cotinine, these parent-reported measures did not differ based on healthcare

utilization outcomes. Nevertheless, these findings build upon prior national research that indicated children with high serum cotinine (>3 ng/mL) were 3.5 times more likely to seek ED-based healthcare, and were nearly three times more likely to have an overnight hospital stay than children with no/minimal cotinine (<0.05 ng/mL).[9] Specific to TSE-related diagnoses, research also reported that bronchiolitic children who had EMR-documented TSE were more likely to be admitted to the hospital from the PED than unexposed bronchiolitic children.[24] Pediatric TSE control efforts initiated in the PED/UC[25] and inpatient settings[26,27] are deemed feasible, and could reduce the morbidity and healthcare costs associated with exposure.

This study was conducted at a large, urban PED/UC, which serves as a "safety net" that provides care for disadvantaged children with TSE-related diagnoses.[28] TSE rates are generally higher among economically disadvantaged groups,[5,6] and thus it is important to note that 95.5% of children had public insurance or were self-pay, a proxy of low SES. The GeoM of child cotinine was 4.8 ng/mL with levels ranging up to 332.0 ng/mL, which translates to levels well above the cut point to distinguish smokers and nonsmokers (cut off range=3.0–15.0 ng/mL).[16] These results underscore the importance of following national recommendations to universally screen for TSE during all pediatric healthcare visits with special efforts to inquire about TSE during visits that may be caused or exacerbated by exposure.[4]

Potential TSE-related disparities were identified with high cotinine concentrations (all GeoM ≥5.5 ng/mL) among children living with a smoker who were aged 0–1 years, white, and lived in multi-unit homes or apartments. Findings on child TSE characteristics are similar to, and expand upon, trends from National Health and Nutrition Examination Survey studies.[5,6] These studies report high cotinine among younger children aged ≥3 years and those who lived in rented homes. Concerning home type, research reports that children who live in multi-unit homes or apartments have higher cotinine levels than children who live in detached homes.[29] Major progress has been made in voluntary and legal smoking restrictions over the past 20 years.[30] However, there is room for improvement as the implementation of voluntary smoke-free rules is present in about 6 of 10 households in which there is at least 1 child and 1 smoking occupant[31] Clean indoor air policies at the U.S. state or local levels only cover about 1 of every 2 individuals.[2] In 2017, a final rule instituted smoke-free housing in U.S. public housing living units[32] which may reduce exposure and improve indoor air quality among children living in multi-unit homes over time. Prior research indicated that this public health legislation contributed to a decrease in ED visits among asthmatic children.[33] Thus, smoke-free rules should be strongly advocated for from the voluntary household to the policy levels.

As hypothesized, there were no differences between cotinine and most PED/UC-related visit characteristics potentially owing to enrolling only clinically ill children of parental smokers. The higher cotinine levels observed during the winter months may be due to increased time spent indoors with smokers compared with the summer months. Notably, children who had PMH of prematurity (8%) had a significantly higher cotinine level of 8.1 ng/mL. Although information on prenatal TSE was unavailable, preterm birth is a well-known consequence of TSE[1] and adversely affects specific organ development including the lungs.[34] This is

concerning as respiratory complications including increased risk of wheezing, bronchopulmonary dysplasia, asthma, and respiratory diseases can be observed in the newborn period and beyond in children born prematurely.[35,36] This finding, in combination with children aged 0–1 years having the highest cotinine levels, emphasizes the need for immediate postnatal TSE efforts. For example, neonatal intensive care unit hospitalization for prematurity may be an opportune time to offer interventions to mothers and other family members who smoke. Prior research indicates that TSE reduction interventions with mothers of young infants within 6 months of post-neonatal intensive care unit discharge are promising. These mothers had greater readiness to protect their infant from TSE with reduced biochemically measured TSE among infants and decreased postpartum smoking rates among mothers.[37] Robust TSE interventions are warranted starting during infancy to decrease the many deleterious and preventable consequences of TSE on children and their families, especially among disadvantaged groups. For example, PED/UC settings may consider implementing the evidence-based 5 A V[38] by screening and counseling parents and families, and providing resources for parents and family members to facilitate tobacco cessation and child TSE reduction. Further, as cotinine assessment, and not parent-reported TSE, was associated with healthcare utilization, routine cotinine assessment may help target children who are most at risk.

Limitations

This longitudinal study has several limitations including the convenience sampling design employed at one large children's hospital PED/UC. Therefore, causality could not be assessed and generalizability may be limited. Total healthcare visits and hospital admissions inclusive of all diagnoses over a 6-month period were assessed with detectable cotinine-based differences but other potential contributing factors were not assessed (e.g., primary care access). EMR data were used for baseline visit-related characteristics and past 6-month healthcare utilization, which has several limitations including potential for missing data and data errors but also has several benefits that outweigh related barriers.[39] Benefits of using EMR data include increasing data quality by reducing parent-reported recall and report biases, for example. These data have been found to have high diagnostic accuracy between physician pediatric diagnoses and EMR-extracted diagnoses.[40] Future studies should assess whether these findings remain consistent using longer periods (e.g., 12 months), consider assessing healthcare utilization for TSE-related diagnoses only, and use reported data in addition to EMR data. Cotinine has an average half-life of about 16 hours, and levels could have decreased if children were not exposed recently.[16] However, all parents were daily smokers so enrolled children were exposed to either secondhand or thirdhand smoke to varying extents within the past week. Parent report on the length of their smoking history was not collected, but data were collected on parents' daily cigarette smoking consumption in addition to cumulative past week child TSE. Additionally, data were not collected on the mother's smoking status while pregnant with the sampled child, but prematurity was one of the PMH accounted for in analyses, which can be caused by prenatal TSE.[1] It is important to note the racial/ethnic difference between white children and those of other races should be interpreted with caution given the genetic variant differences in metabolism of cotinine.[41] Child PMH of respiratory conditions, otitis media, and prematurity accounted for baseline health status in all healthcare utilization analyses.

CONCLUSIONS

This study provides objective evidence that higher biochemically validated TSE is associated with higher healthcare utilization among clinically ill, racially and ethnically diverse children who live with a smoker. Although parent report of child TSE is important to screen for during children's PED/UC visits, no differences were found based on parent-reported daily smoking behavior or cumulative child TSE, and healthcare utilization. Thus, routine biochemical screening for TSE could identify all children and families who are in need of more intensive TSE reduction interventions, which can in turn alleviate the child health burden associated with parental smoking. Specifically, routine cotinine tests would detect TSE and values would alert healthcare providers that TSE needs to be addressed. This screening could be conducted in a similar fashion to other laboratory tests that show high values (e.g., HbAlc) that could be harbingers of adverse morbidity and preventable healthcare visits. Routine cotinine screening for child TSE could positively affect their patient care and potentially reduce future healthcare visits.

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REFERENCES

1. HHS. The health consequences of involuntary exposure to tobacco smoke: a report of the Surgeon General. Atlanta, GA: HHS, CDC, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health; 2006.
2. HHS. The health consequences of smoking—50 years of progress. A report of the Surgeon General. Atlanta, GA: HHS, CDC, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health; 2014.
3. American Academy of Pediatrics. Policy statement - tobacco use: a pediatric disease. Pediatrics. 2009;124(5): 1474–1487. 10.1542/peds.2009-2114. [PubMed: 19841108]
4. Farber HJ, Groner J, Walley S, Nelson K, Section on Tobacco Control. Protecting children from tobacco, nicotine, and tobacco smoke. Pediatrics. 2015;136(5):el439–el467. 10.1542/peds.2015-3110.
5. Merianos AL, Jandarov RA, Choi K, Mahabee-Gittens EM. Tobacco smoke exposure disparities persist in U.S. children: NHANES 1999–2014. Prev Med. 2019;123:138–142. 10.1016/j.ypmed.2019.03.028. [PubMed: 30902698]
6. Tsai J, Homa DM, Gentzke AS, et al. Exposure to secondhand smoke among nonsmokers--United States, 1988–2014. MMWR. 2018;67(48): 1342–1346. 10.15585/mmwr.mm6748a3. [PubMed: 30521502]
7. Wang Z, May SM, Charoenlap S, et al. Effects of secondhand smoke exposure on asthma morbidity and health care utilization in children: a systematic review and meta-analysis. Ann Allergy Asthma Immunol. 2015; 115 (5): 396–401.e2. 10.1016/j.anai.2015.08.005. [PubMed: 26411971]
8. Merianos AL, Jandarov RA, Mahabee-Gittens EM. Association of secondhand smoke exposure with asthma symptoms, medication use, and healthcare utilization among asthmatic adolescents. J Asthma. 2019;56(4):369–379. 10.1080/02770903.2018.1463379. [PubMed: 29641269]
9. Merianos A, Jandarov R, Mahabee-Gittens E. Secondhand smoke exposure and pediatric healthcare visits and hospitalizations. Am J Prev Med. 2017;53(4):441–448. 10.1016/j.amepre.2017.03.020. [PubMed: 28532658]

10. Merianos AL, Odar Stough C, Nabors LA, Mahabee-Gittens EM. Tobacco smoke exposure and health-care utilization among children in the United States. Am J Health Promot. 2018;32(1): 123–130. 10.1177/0890117116686885. [PubMed: 29214835]
11. Merianos AL, Jandarov RA, Mahabee-Gittens EM. Adolescent tobacco smoke exposure, respiratory symptoms, and emergency department use. Pediatrics. 2018; 142(3):e20180266 10.1542/peds.2018-0266. [PubMed: 30082449]
12. Jin Y, Seiber EE, Ferketich AK. Second hand smoke and asthma: what are the effects on healthcare utilization among children? Prev Med. 2013;57(2): 125–128. 10.1016/j.ypmed.2013.05.003. [PubMed: 23701846]
13. Yao T, Sung H, Wang Y, Lightwood J, Max W. Healthcare costs of secondhand smoke exposure at home for U.S. children. Am J Prev Med. 2019;56(2):281–287. 10.1016/j.amepre.2018.08.013. [PubMed: 30553690]
14. Rodriguez-Martinez CE, Sossa-Briceno MP, Castro-Rodriguez JA. Predictors of hospitalization for asthma in children: results of a 1-year prospective study: predictors of hospitalization for asthma. Pediatr Pulmonol. 2014;49(11):1058–1064. 10.1002/ppul.22936. [PubMed: 24376022]
15. Hill SC, Liang L. Smoking in the home and children's health. Tob Control. 2008;17(l):32–37. 10.1136/tc.2007.020990. [PubMed: 18218804]
16. Benowitz NL, Bernert JT, Foulds J, et al. Biochemical verification of tobacco use and abstinence: 2019 update. Nicotine Tobacco Res. 2020;22(7): 1086–1097. 10.1093/ntr/ntz132.
17. Mahabee-Gittens EM, Ammerman RT, Khoury JC, et al. Healthy families: study protocol for a randomized controlled trial of screening, brief intervention, and referral to treatment intervention for caregivers to reduce secondhand smoke exposure among pediatric emergency department patients. BMC Public Health. 2017;17:374–387. 10.1186/s12889-017-4278-8. [PubMed: 28464887]
18. Mahabee-Gittens EM, Mazzella MJ, Doucette JT, et al. Comparison of liquid chromatography mass spectrometry and enzyme-linked immunosorbent assay methods to measure salivary cotinine levels in ill children. Int J Environ Res Public Health. 2020;17(4):1157 10.3390/ijerph17041157.
19. Mahabee-Gittens EM, Merianos AL, Fulkerson PC, Stone L, Matt GE. The association of environmental tobacco smoke exposure and inflammatory markers in hospitalized children. Int J Environ Res Public Health. 2019;16(23):4625 10.3390/ijerph16234625.
20. Mahabee-Gittens EM, Merianos AL, Hoh E, Quintana PJ, Matt GE. Nicotine on children's hands: limited protection of smoking bans and initial clinical findings. Tob Use Insights. 2019;12:1–6. 10.1177/1179173x18823493.
21. Butz AM, Bollinger ME, Halterman JS, et al. Factors associated with second-hand smoke exposure in young inner-city children with asthma. J Asthma. 2011;48(5):449–457. 10.3109/02770903.2011.576742. [PubMed: 21545248]
22. Hao S, Jin B, Shin AY, et al. Risk prediction of emergency department revisit 30 days post discharge: a prospective study. PLoS One. 2014;9(ll):el12944 10.1371/journal.pone.0112944.
23. Bardach NS, Vittinghoff E, Asteria-Penaloza R, et al. Measuring hospital quality using pediatric readmission and revisit rates. Pediatrics. 2013;132(3):429–436. 10.1542/peds.2012-3527. [PubMed: 23979094]
24. Merianos AL, Dixon CA, Mahabee-Gittens EM. Secondhand smoke exposure, illness severity, and resource utilization in pediatric emergency department patients with respiratory illnesses. J Asthma. 2017;54(8):798–806. 10.1080/02770903.2016.1265127. [PubMed: 27929695]
25. Mahabee-Gittens EM, Merianos AL, Dexheimer JW, et al. Utilization of a clinical decision support tool to reduce child tobacco smoke exposure in the urgent care setting. Pediatr Emerg Care. In press. Online October 19, 2018 10.1097/pec.0000000000001646.
26. Kruse GR, Rigotti NA. Routine screening of hospital patients for secondhand tobacco smoke exposure: a feasibility study. Prev Med. 2014;69:141–145. 10.1016/j.ypmed.2014.09.017. [PubMed: 25284258]
27. Jenssen BP, Shelov ED, Bonafide CP, Bernstein SL, Fiks AG, Bryant-Stephens T. Clinical decision support tool for parental tobacco treatment in hospitalized children. Appl Clin Inform. 2016;7(2):399–411. 10.4338/aci-2015-12-ra-0169. [PubMed: 27437049]

28. Merianos AL, Dixon CA, Mahabee-Gittens EM. Tobacco smoke exposure-related illnesses among pediatric emergency department patients. J Pediatr Health Care. 2017;31(2): 161–166. 10.1016/j.pedhc.2016.07.001. [PubMed: 27542562]
29. Wilson KM, Klein JD, Blumkin AK, Gottlieb M, Winickoff JP. Tobacco-smoke exposure in children who live in multiunit housing. Pediatrics. 2011;127(l):85–92. 10.1542/peds.2010-2046. [PubMed: 21149434]
30. McMillen R, Wilson K, Tanski S, Klein JD, Winickoff JP. Adult attitudes and practices regarding smoking restrictions and child tobacco smoke exposure: 2000 to 2015. Pediatrics. 2018;141(suppl 1):S21–S29. 10.1542/peds.2017-1026f. [PubMed: 29292303]
31. King BA, Patel R, Babb SD, Hartman AM, Freeman A. National and state prevalence of smoke-free rules in homes with and without children and smokers: two decades of progress. Prev Med. 2016;82:51–58. 10.1016/j.ypmed.2015.11.010. [PubMed: 26601642]
32. U.S. Department of Housing and Urban Development. Instituting smoke-free public housing, https://www.hud.gov/sites/documents/smokefreephfinalrule.pdf. Updated 2016 Accessed February 12, 2020.
33. Ciaccio CE, Gurley-Calvez T, Shireman TI. Indoor tobacco legislation is associated with fewer emergency department visits for asthma exacerbation in children. Arm Allergy Asthma Immunol. 2016;117(6):641–645. 10.1016/j.anai.2016.10.005.
34. Wagijo M, Sheikh A, Duijts F, Been JY. Reducing tobacco smoking and smoke exposure to prevent preterm birth and its complications. Paediatr Respir Rev. 2017;22:3–10. 10.1016/j.prrv.2015.09.002. [PubMed: 26482273]
35. Islam JY, Keller RE, Aschner JF, Hartert TV, Moore PE. Understanding the short- and long-term respiratory outcomes of prematurity and bronchopulmonary dysplasia. Am J Respir Crit Care Med. 2015; 192(2): 134–156. 10.1164/rccm.201412-2142pp. [PubMed: 26038806]
36. Vanker A, Gie RP, Zar HJ. The association between environmental tobacco smoke exposure and childhood respiratory disease: a review. Expert Rev Respir Med. 2017;11(8):661—673. 10.1080/17476348.2017.1338949. [PubMed: 28580865]
37. Stotts AL, Northrup TF, Green C, et al. Reducing tobacco smoke exposure in high-risk infants: a randomized, controlled trial. J Pediatr. 2020;218:35–41.el. 10.1016/j.jpeds.2019.10.070. [PubMed: 31870605]
38. The Clinical Practice Guideline Treating Tobacco Use and Dependence 2008 Update Panel, Liaisons, and Staff. A Clinical Practice Guideline for Treating Tobacco Use and Dependence: 2008 update: A U.S. public health service report. Am J Prev Med. 2008;35(2): 158–176. 10.1016/j.amepre.2008.04.009. [PubMed: 18617085]
39. Kruse CS, Stein A, Thomas H, Kaur H. The use of electronic health records to support population health: a systematic review of the literature. J Med Syst. 2018;42(11): 1–16. 10.1007/s10916-018-1075-6.
40. Liang H, Tsui BY, Ni H, et al. Evaluation and accurate diagnoses of pediatric diseases using artificial intelligence. Nat Med. 2019;25(3):433–438. 10.1038/s41591-018-0335-9. [PubMed: 30742121]
41. Murphy SE. Nicotine metabolism and smoking: ethnic differences in the role of P450 2A6. Chem Res Toxicol. 2017;30(1):410–419. 10.1021/acs.chemrestox.6b00387. [PubMed: 28092945]

Table 1.

Participant Characteristics and Salivary Cotinine Concentrations Among Children 0–9 Years Old

Characteristic		Log-cotinine concentration		
	n (%)[a]	GeoM (95 % CI)	Median (IQR)	*p*-value[b]
Child characteristics				
Child age, years				
0–1	177 (56.5)	5.6 (4.5, 6.9)	5.7 (2.2–15.6)	ref
2–4	59 (18.9)	3.7 (2.8, 5.0)	4.6 (1.4–7.8)	**0.045**
5–9	77 (24.6)	4.0 (3.0, 5.3)	3.9 (1.8–84)	0.07
Child sex				
Male	163 (52.1)	5.5 (4.5, 6.7)	6.0 (2.3–13.3)	ref
Female	150 (47.9)	4.1 (3.3, 5.2)	4.1 (1.7–10.6)	0.07
Child race				
White	112 (36.8)	5.5 (4.1,7.4)	5.6 (1.8–14.1)	ref
Black	171 (56.3)	4.7 (3.9,57)	5.1 (2.2–11.5)	0.35
Other	21 (6.9)	2.6 (17,3.9)	2.3 (14–5.5)	**0.02**
Child ethnicity				
Non-Hispanic	305 (97.4)	4.8 (4.2, 5.7)	5.1 (1.8–12.8)	ref
Hispanic	5 (1.6)	3.4 (1.4, 8.2)	2.8 (1.6–6.4)	0.55
Unknown	3 (1.0)	2.6 (0.4, 16.8)	6.0 (3.2–6.8)	0.45
Child insurance type				
Commercial	14 (4.5)	3.6 (14, 11.2)	3.25 (0.4–31.2)	ref
Public	299 (95.5)	4.8 (4.2, 5.6)	5.1 (1.9–11.5)	0.41
Housing type				
Single-family	126 (40.3)	3.6 (2.8,47)	4.0 (1.1–10.5)	ref
Multi-unit home	72 (23.0)	57 (4.3,7.6)	4.9 (2.3–16.0)	**0.03**
Apartment	115 (36.7)	57 (4.6, 7.2)	6.2 (2.4–14.2)	**0.009**
Child PMH of respiratory condition[c]				
No	269 (85.9)	4.8 (44,57)	5.1 (1.8–13.4)	ref
Yes	44 (14.1)	4.4 (34,6.2)	5.1 (2.8–9.3)	0.69
Child PMH of otitis media				
No	292 (93.3)	4.9 (4.2, 5.7)	5.2 (1.9–12.5)	ref
Yes	21 (6.7)	3.4 (17,6.6)	3.3 (14–87)	0.23
Child PMH of prematurity				
No	287 (91.7)	4.5 (3.9, 5.3)	4.9 (1.8–11.3)	ref
Yes	26 (8.3)	8.1 (4.6, 14.2)	10.0 (3.0–15.8)	**0.04**
Child surgical history of tonsillectomy/adenoidectomy				
No	298 (95.2)	4.9 (4.2, 5.7)	5.2 (1.9–13.0)	ref
Yes	15 (4.8)	3.0 (1.6, 5.8)	3.0 (1.7–84)	0.19
Parent characteristic				
Parent sex				

Characteristic		Log-cotinine concentration		
	***n* (%)**[a]	**GeoM (95 % CI)**	**Median (IQR)**	***p*-value**[b]
Male	42 (13.4)	4.4 (2.9, 6.7)	5.2 (1.5–10.2)	ref
Female	271 (86.6)	4.8 (44,5.7)	5.1 (1.9–12.9)	0.67
Parent-reported smoking behavior and child TSE				
No. cigarettes/day by parent, (*n*=293), M (SD)	10.5 (6.3)			**0.007**[d]
No. cigarettes/week around child by all smokers (*n*=285), M (SD)	10.0 (21.4)	-	-	**0.008**[d]

Notes: N=313 unless noted. Missing data excluded.

[a]Percent refers to valid column percent unless otherwise noted.

[b]*p*-values refer to linear regression results, and boldface indicates statistical significance (*p*<0.05) unless otherwise noted.

[c]Respiratory conditions include asthma, bronchiolitis, and pneumonia.

[d]*p*-values refer to Spearman correlation results, and boldface indicates statistical significance (*p*<0.05).

PED, pediatric emergency department; UC, urgent care; M, mean; GeoM, geometric mean; PMH, past medical history; No., number.

Table 2.

PED/UC-Related Visit Characteristics and Salivary Cotinine Concentrations among Children 0–9 Years Old

		Log-cotinine concentration		
PED visit characteristic	***n* (%)[a]**	**GeoM (95% CI)**	**Median (IQR)**	***p*-value[b]**
Baseline visit season				
Summer (June–August)	87 (27.8)	4.3 (3.2, 5.6)	4.8 (1.7–13.5)	ref
Fall (September–November)	61 (19.5)	4.2 (3,5.8)	4.8 (1.8–10.8)	0.95
Winter (December–February)	87 (27.8)	6.5 (4.7, 8.9)	6.2 (2.6–19.5)	0.04
Spring (March–May)	78 (24.9)	4.2 (3.2, 5.5)	5.2 (1.83–8.9)	0.94
Baseline visit year				
2016	102 (32.6)	4.8 (3.8, 6.2)	5.4 (2.3–11.5)	ref
2017	134 (42.8)	4.7 (3.7, 6)	5.0 (1.7–11.4)	0.87
2018	77 (24.6)	4.9 (3.6, 6.5)	5.1 (1.8–13.9)	0.97
Arrival location				
Urgent care	230 (73.5)	4.9 (44,5.8)	5.4 (1.9–13.2)	ref
Emergency department	83 (26.5)	4.5 (3.2, 6.2)	4.7 (1.6–10.3)	0.61
Triage level				
Low to moderate acuity (3–5)	86 (84.3)	4.6 (3.4, 6.4)	5.0 (1.5–12.5)	ref
Eligh acuity (2)	16 (15.7)	5.0 (2.8, 9.0)	4.0 (2.5–10.2)	0.83
Chief complaint of cough/congestion				
No	174 (55.6)	4.5 (3.7, 5.6)	4.9 (1.8–11.1)	ref
Yes	139 (44.4)	5.1 (44,6.4)	5.5 (1.8–13.7)	0.43
Chief complaint of difficulty breathing/wheezing				
No	238 (76.0)	4.5 (3.8, 5.3)	5.2 (1.7–11.3)	ref
Yes	75 (24.0)	5.8 (4.2, 8.0)	4.9 (2.95–13.8)	0.17
Disposition				
Discharge to home	289 (92.6)	4.8 (44,5.6)	5.1 (1.8–13)	ref
Admit	23 (7.4)	4.8 (2.9, 7.7)	4.9 (2.5–9.3)	0.99
Primary discharge diagnosis				
Non-TSE related	50 (16.0)	4.3 (2.8, 6.7)	3.9 (1.2–11.5)	ref
Asthma	20 (6.4)	3.5 (2.2, 5.6)	3.6 (2.3–5.7)	0.55
Bronchiolitis	28 (8.9)	7.5 (4.5, 12.5)	6.3 (3.2–15.3)	0.09
Pneumonia	4 (1.3)	7.6 (1.4,42.4)	13.1 (5.1–24.1)	0.42
URI/Croup	141 (45.1)	4.4 (3.6, 5.5)	5.1 (1.7–9.6)	0.93
Otitis media	59 (18.8)	5.0 (3.5, 7.1)	5.6 (2.0–13.4)	0.59
Conjunctivitis	11 (3.5)	6.8 (2.7, 17.4)	7.3 (3.3–18.0)	0.32

Notes: N=313.

[a]Percent refers to valid column percent.

[b]*p*-values refer to linear regression results.

PED, pediatric emergency department; UC, urgent care; GeoM, geometric mean; TSE, tobacco smoke exposure; URI, upper respiratory infection.

Table 3.

Cotinine and Parent-Reported TSE and Healthcare Visits Over 6-Months Children 0–9 Years Old

Variable	Total revisits, past 30 days			Total PED visits			Total UC visits			Total hospital admissions		
	Mean (SE)	ARR (95% CI)	*p*-value[a]	Mean (SE)	ARR (95% CI)	*p*-value[a]	Mean (SE)	ARR (95% CI)	*p*-value[a]	Mean (SE)	ARR (95% CI)	*p*-value[a]
Log-cotinine (N=313)	0.2 (0.02)	1.05 (0.85, 1.30)	0.65	0.4 (0.05)	1.16 (1.01, 1.34)	**0.04**	0.6 (0.05)	1.05 (0.94, 1.19)	0.41	0.1 (0.02)	1.50 (1.08, 2.09)	0.02
No. cigarettes/day by parents (*n*=293)	10.5 (0.37)	0.97 (0.92, 1.02)	0.24	10.5 (0.37)	0.98 (0.95, 1.02)	0.32	10.5 (0.37)	0.98 (0.95, 1.01)	0.21	10.5 (0.37)	0.95 (0.87, 1.03)	0.19
No. cigarettes/week around child by all smokers (*n*=285)	10.0 (1.26)	0.99 (0.79, 1.25)	0.96	10.0 (1.26)	0.92 (0.79, 1.07)	0.26	10.0 (1.26)	1.06 (0.94, 1.19)	0.33	10.0 (1.26)	0.84 (0.59, 1.20)	0.35

[a]Poisson regression results adjusted tor child age, sex, race, ethnicity, insurance type, and PMtt, parental smoker sex, and visit season and year. Boldface indicates statistical significance ($p<0.05$).

PED, pediatric emergency department; UC, urgent care; ARR, adjusted relative risk; No., number.