RESEARCH ARTICLE

Electronic Health Record Classification of Tobacco Smoke Exposure and Cotinine Levels in Hospitalized Pediatric Patients

E. Melinda Mahabee-Gittens, MD, MS,[a,b] Ashley L. Merianos, PhD,[c] Judith S. Gordon, PhD,[d] Lara Stone, MA,[a] Olga Semenova, BS,[a] Georg E. Matt, PhD[e]

ABSTRACT

OBJECTIVES: Documentation of children's tobacco smoke exposure (TSE) in the electronic health record (EHR) can have important implications for clinical care. However, it may not be accurate if it is not based on biochemical assessment, the most reliable method of verifying TSE. Our objectives were to compare the accuracy of EHR classification of TSE with cotinine verification and to explore parent and child variables associated with biochemically verified TSE.

METHODS: Participants were 171 hospitalized pediatric patients (ages 0–17 years; mean age 5.1 [SD 3.7] years) who had EHR documentation of TSE and measured salivary cotinine. Children with cotinine levels >1 ng/mL were classified as having biochemical verification of TSE. Parents reported sociodemographic characteristics, and children's EHRs were abstracted for TSE status, past medical history, and diagnoses. We conducted χ^2 tests to assess the agreement between EHR classification of TSE status and cotinine levels. Then, we assessed the relationship between sociodemographic and clinical variables and cotinine using crude and adjusted logistic regression models.

RESULTS: Overall, 71% (121 of 171) of EHR classifications were correct on the basis of cotinine levels. Specificity analyses showed that 77% (53 of 69) were correctly identified as exposed to tobacco smoke. Sensitivity analyses showed that 67% (68 of 102) were correctly identified as unexposed. The negative predictive value was 0.61 (53 of 87); 39% (34 of 87) were misclassified as unexposed. The positive predictive value was 0.81 (68 of 84); 19% (16 of 84) were misclassified as exposed.

CONCLUSIONS: Almost 40% of children were misclassified in the EHR as unexposed to tobacco smoke. Biochemical verification should be used as part of universal TSE screening during pediatric hospitalizations.

[a]Division of Emergency Medicine, Cincinnati Children's Hospital Medical Center, Cincinnati, Ohio; [b]College of Medicine and [c]School of Human Services, University of Cincinnati, Cincinnati, Ohio; [d]College of Nursing, The University of Arizona, Tucson, Arizona; and [e]Department of Psychology, San Diego State University, San Diego, California

www.hospitalpediatrics.org
DOI:https://doi.org/10.1542/hpeds.2018-0247

Address correspondence to E. Melinda Mahabee-Gittens, MD, MS, Division of Emergency Medicine, Cincinnati Children's Hospital Medical Center, 3333 Burnet Ave, MLC 2008, Cincinnati, OH 45229. E-mail: melinda.mahabee-gittens@cchmc.org

HOSPITAL PEDIATRICS (ISSN Numbers: Print, 2154-1663; Online, 2154-1671).

FINANCIAL DISCLOSURE: The authors have indicated they have no financial relationships relevant to this article to disclose.

FUNDING: Funded by the National Cancer Institute (National Institutes of Health; 3R21CA184337S1) and Institutional Clinical and Translational Science Award (National Institutes of Health and National Center for Advancing Translational Sciences; grant 8UL1TR000077). Funded by the National Institutes of Health (NIH).

POTENTIAL CONFLICT OF INTEREST: The authors have indicated they have no potential conflicts of interest to disclose.

Dr Mahabee-Gittens conceptualized and designed the study and drafted the initial manuscript; Drs Merianos, Gordon, and Matt assisted in the analysis and interpretation of data for the work, provided content expertise, and edited revisions of the manuscript; Ms Stone and Ms Semenova assisted in the acquisition of the data, data entry, and data cleaning; and all authors approved the final manuscript as submitted.

The prevalence of tobacco smoke exposure (TSE) in US children is estimated to be as high as 38%.[1] Given the many clinical, developmental, and behavioral risks of TSE to children,[2] clear guidelines exhort pediatricians to routinely screen for and document child TSE in the electronic health record (EHR) at all pediatric clinical encounters.[3–5] Accurate assessment of the TSE status of hospitalized patients is an important step in increasing clinicians' rates of counseling parents about TSE reduction strategies that would improve children's health.[3,4,6] Almost 100% of nonfederal acute care hospitals have certified EHR systems,[7] and clinicians consider the EHR a source of high-quality data that is accurate and precise.[8,9] Clinicians frequently rely on documentation in the EHR to classify TSE exposure among hospitalized pediatric patients. However, this documentation may not accurately reflect children's current TSE status because EHR documentation of children's TSE often relies on parental self-report at the time of the pediatric health care visit.[10,11] Biochemical assessment has been shown to be the most reliable method of verifying TSE. The preferred biomarker to verify TSE is cotinine, the major proximate metabolite of nicotine, which has a half-life of ∼16 to 18 hours in children. The presence of cotinine in a child's saliva, urine, or blood represents recent TSE.[12] Several studies have reported discrepancies between cotinine measurements and parental self-reports.[10,13–15] Given the concern about misclassification and the morbidity associated with child TSE, studies have assessed the prevalence of TSE using routine assessment with cotinine.[16–18] These studies demonstrate positive cotinine screening results in children with no reported TSE, suggesting the need for a universal approach to biochemical TSE screening. Other investigators recommend that screening should be directed to parents whose children have respiratory illnesses.[11,19,20] Our primary objectives in this study were to compare the accuracy of EHR classification of TSE status with cotinine verification among hospitalized pediatric patients and to examine associations between sociodemographic and clinical factors and biochemically verified TSE.

METHODS

A convenience sample of 0- to 17-year-old patients who were hospitalized on a general medical unit between December 2016 and June 2017 in a US children's hospital and had EHR documentation of positive or negative TSE status were recruited during day hours by study staff. TSE status was assessed by a physician or nurse with 1 of 2 prompts: "tobacco smoke exposure" or "smokers in the home." Children were classified as having a positive TSE status if either answer was "yes" ($n = 100$) or a negative TSE status if both answers were "no" ($n = 99$). Children were excluded if they had a tracheostomy ($n = 2$) or were smokers ($n = 10$). Caregiver consent on all children and child assent on children age 11 and older were obtained. Saliva samples were obtained from all children at the time of study enrollment; the mean collection time was 18 hours and 43 minutes (SD 0.68 hours) after admission. Saliva was tested for cotinine by Salimetrics LLC[21] using enzyme-linked immunosorbent assay (ELISA) techniques; the level of detection was 0.15 ng/mL. Previous research has classified children with salivary cotinine values >1.0 ng/mL as having positive biochemical verification of TSE.[22–24] We assessed the robustness of using the >1.0 ng/mL cut point by building the same models and using different cotinine cut points (ie, >0.70, >0.85, and >1.25 ng/mL), and overall, our results showed consistent findings (see footnote in Table 2). Thus, we have classified children with cotinine levels of >1.0 ng/mL as having positive biochemical verification of TSE, hereafter classified as positive cotinine levels.

Of the 199 participants, 171 had sufficient saliva volume to have cotinine measured and were included in the study. Caregivers reported sociodemographic characteristics, and children's EHRs were abstracted for the following: past medical history (PMH) and *International Classification of Diseases, 10th Revision* discharge diagnosis (specifically asthma, bronchiolitis, and pneumonia), number of emergency department (ED) and urgent care (UC) visits, hospitalizations for a 6-month period before and after the index hospitalization, and number of revisits (ie, ED, UC, or hospitalizations) in a 30-day period. This study was approved by our hospital's institutional review board.

ANALYSES

We conducted χ^2 tests to assess the agreement between the proportions of children with positive or negative EHR classification of TSE status and biochemical verification of exposure or no exposure among all patients and by patients within age categories. Then, we assessed the relationship between sociodemographic and clinical variables and cotinine levels by building a series of crude and adjusted logistic regression models while controlling for age, race, and ethnicity. We also evaluated which demographic characteristics were associated with insurance status and assessed whether insurance status accounted for the variance independent of age. R version 3.3.0[25] was used for all statistical analyses, and associations were considered significant at $\alpha < 0.05$.

RESULTS

The mean age of children in this study was 5.1 years (SD 3.7; range 0.04–17), 53.2% were boys, and 62% had public insurance or were self-pay. Of the sample, 74.9% were white, 18.7% were African American, and 96.5% were non-Hispanic. There were no differences based on child age, sex, race, ethnicity, insurance type, or TSE status

TABLE 1 TSE Classifications Based on EHR Documentation and Cotinine Levels Among Hospitalized Pediatric Patients

EHR classification of TSE	Cotinine TSE Classification		Total (%)
	Negative,[a] *n* (%)	Positive,[b] *n* (%)	
Negative TSE status	53 (31)	34 (20)	87 (51)
Positive TSE status	16 (9)	68 (40)	84 (49)
Total	69 (40)	102 (60)	171 (100)

[a] Negative cotinine level = cotinine ≤1.0 ng/mL.
[b] Positive cotinine level = cotinine >1.0 ng/mL.

TABLE 2 Characteristics Associated With Negative and Positive TSE Classification Based on Cotinine Levels Among Hospitalized Pediatric Patients

Item	*N* (%)	Negative[a] (*N* = 69)	Positive[b] (*N* = 102)	Univariate Analysis		Multivariable Analysis	
		n (%)	*n* (%)	OR	95% CI	aOR[c]	95% CI
Age, y							
0–1	68 (39.8)	16 (23.5)	52 (76.5)	5.63***	2.41–13.14	6.02***	2.53–14.31
2–4	27 (15.8)	9 (33.3)	18 (66.7)	3.47*	1.25–9.63	3.65*	1.27–10.51
5–9	35 (20.5)	18 (51.4)	17 (48.6)	1.64	0.65–4.10	1.60	0.63–4.09
10–17	41 (24.0)	26 (63.4)	15 (36.6)	Ref	Ref	Ref	Ref
Race							
White	128 (74.9)	54 (42.2)	74 (57.8)	Ref	Ref	Ref	Ref
African American	32 (18.7)	12 (37.5)	20 (62.5)	1.22	0.55–2.70	1.17	0.50–2.74
Other	11 (6.4)	3 (27.3)	8 (72.7)	1.95	0.49–7.68	3.54	0.77–16.25
Ethnicity							
Non-Hispanic	164 (96.5)	66 (40.2)	98 (59.8)	Ref	Ref	Ref	Ref
Hispanic	6 (3.5)	3 (50.0)	3 (50.0)	0.67	0.13–3.44	0.50	0.09–2.84
Sex							
Male	91 (53.2)	37 (40.7)	54 (59.3)	0.97	0.53–1.80	0.96	0.49–1.90
Female	80 (46.8)	32 (40.0)	48 (60.0)	Ref	Ref	Ref	Ref
Insurance type							
Public or self-pay	106 (62.0)	23 (21.7)	83 (78.3)	8.74***	4.31–17.71	8.33***	3.83–18.12
Private	65 (38.0)	46 (70.8)	19 (29.2)	Ref	Ref	Ref	Ref
PMH of asthma							
No	114 (66.7)	43 (37.7)	71 (62.3)	Ref	Ref	Ref	Ref
Yes	57 (33.3)	26 (45.6)	31 (54.4)	0.72	0.38–1.38	1.24	0.56–2.75
PMH of bronchiolitis							
No	132 (77.2)	59 (44.7)	73 (55.3)	Ref	Ref	Ref	Ref
Yes	39 (22.8)	10 (25.6)	29 (74.4)	2.34*	1.06–5.20	1.75	0.74–4.14
PMH of pneumonia							
No	157 (91.8)	62 (39.5)	95 (60.5)	Ref	Ref	Ref	Ref
Yes	14 (8.2)	7 (50.0)	7 (50.0)	0.65	0.22–1.95	1.13	0.35–3.71
Asthma diagnosis							
No	141 (82.5)	58 (41.1)	83 (58.9)	Ref	Ref	Ref	Ref
Yes	30 (17.5)	11 (36.7)	19 (63.3)	1.21	0.53–2.73	2.45	0.95–6.27
Bronchiolitis diagnosis							
No	134 (78.4)	62 (46.3)	72 (53.7)	Ref	Ref	Ref	Ref
Yes	37 (21.6)	7 (18.9)	30 (81.1)	3.69**	1.52–8.99	1.94	0.62–6.07
Asthma or bronchiolitis diagnosis[c]							
No	104 (60.8)	51 (49.0)	53 (51.0)	Ref	Ref	Ref	Ref
Yes	67 (39.2)	18 (26.9)	49 (73.1)	2.62**	1.35–5.08	2.22*	1.08–4.57
Pneumonia diagnosis							
No	143 (83.6)	54 (37.8)	89 (62.2)	Ref	Ref	Ref	Ref
Yes	28 (16.4)	15 (53.6)	13 (46.4)	0.53	0.23–1.19	0.60	0.24–1.51
Total ED or UC visits							
0	66 (38.6)	32 (48.5)	34 (51.5)	Ref	Ref	Ref	Ref
≥1	105 (61.4)	37 (35.2)	68 (64.8)	1.73	0.92–3.24	1.23	0.62–2.44
ED visits							

between participants who did (*n* = 171) and did not (*n* = 28) have saliva analyzed for cotinine. A total of 102 (59.6%) children had positive cotinine levels. Table 1 presents the cross-tabulations of EHR classification of TSE status and positive or negative cotinine levels. Overall, 71% (121 of 171) of EHR classifications were correct given the 1.0 ng/mL cutoff of salivary cotinine indicating exposure. Specificity was 0.77 (53 of 69) and sensitivity was 0.67 (68 of 102). Of the 102 patients with positive cotinine levels, 68 (67%) were correctly identified as TSE case patients in the EHR. Of the 69 patients with negative cotinine levels, 53 (77%) were correctly identified as unexposed in the EHR. The negative predictive value was 0.61 (53 of 87); 61% were confirmed unexposed with negative cotinine levels, and 39% (34 of 87) were misclassified as unexposed. The positive predictive value was 0.81 (68 of 84); 19% (16 of 84) were misclassified as exposed.

Positive cotinine levels indicative of TSE decreased as child age increased, with 76.5% of 0- to 1-year-olds having positive cotinine levels, followed by 2- to 4-year-olds (66.7%), 5- to 9-year-olds (48.6%), and 10- to 17-year-olds (36.6%; Table 2). Because younger children had higher levels of cotinine overall, we examined if the false-negative results were primarily in younger children. Although 52.9% (*n* = 36) of children 0 to 1 year old were classified as positive TSE status in the EHR, 76.5% (*n* = 52) of children in this age group had positive cotinine levels. We found similar discrepancies in children 2 to 4 years old; 37.0% (*n* = 10) had positive TSE status in the EHR, but 66.7% (*n* = 18) had positive cotinine levels. In children 5 to 9 years old, 40.0% (*n* = 14) had positive TSE status in the EHR, but 48.6% (*n* = 17) had positive cotinine levels. However, in older children age 10 to 17 years, 58.5% (*n* = 24) had positive TSE status in the EHR, but 36.6% (*n* = 15) had positive cotinine levels.

We examined if there were associations between sociodemographic characteristics of participants or clinical factors and positive cotinine levels (Table 2). Univariate logistic regression models indicated that 0- to 1-year-olds (odds ratio [OR] 5.63; 95%

TABLE 2 Continued

Item	*N* (%)	Negative[a] (*N* = 69)	Positive[b] (*N* = 102)	Univariate Analysis		Multivariable Analysis	
		n (%)	*n* (%)	OR	95% CI	aOR[c]	95% CI
0	80 (46.8)	41 (51.2)	39 (48.8)	Ref	Ref	Ref	Ref
≥1	91 (53.2)	28 (30.8)	63 (69.2)	2.37**	1.27–4.42	1.85	0.93–3.69
UC visits							
0	122 (71.3)	50 (41.0)	72 (59.0)	Ref	Ref	Ref	Ref
≥1	49 (28.7)	19 38.8)	30 (61.2)	1.10	0.56–2.16	0.86	0.41–1.83
Revisits in 30 d							
0	139 (81.3)	58 (41.7)	81 (58.3)	Ref	Ref	Ref	Ref
≥1	32 (18.7)	11 (34.4)	21 (65.6)	1.37	0.61–3.05	1.07	0.44–2.56
Hospitalizations							
0 visits	142 (83.0)	59 (41.5)	83 (58.5)	Ref	Ref	Ref	Ref
≥1 visit	29 (17.0)	10 (34.5)	19 (65.5)	1.35	0.59–3.11	0.87	0.34–2.21
Inpatient LOS, d							
0	43 (25.1)	17 (39.5)	26 (60.5)	Ref	Ref	Ref	Ref
≥1	128 (74.9)	52 (40.6)	76 (59.4)	0.96	0.47–1.94	0.80	0.37–1.73

After adjustment for the covariates, patients with TSE using the >0.70 cut point were more likely to be 0- to 1-y-olds, have public or self-pay insurance, have an asthma diagnosis, have a respiratory-related diagnosis of asthma and/or bronchiolitis, have ≥1 ED visit, and have caregiver report of TSE. After adjustment for the covariates, patients with TSE using the >0.85 cut point were more likely to be 0- to 1-y-olds, have public or self-pay insurance, and have caregiver report of TSE. After adjustment for the covariates, patients with TSE using the >1.25 cut point were more likely to be 0- to 1-y-olds and 2- to 4-y-olds, have public or self-pay insurance, have an asthma diagnosis, and have caregiver report of TSE. aOR; adjusted odds ratio; LOS, length of stay; ref, referent.

[a] Negative classification = cotinine ≤1.0 ng/mL.

[b] Positive classification = cotinine >1.0 ng/mL.

[c] Adjusted logistic regression models controlling for age, race, and ethnicity.

*** $P < .001$; ** $P < .01$; * $P < .05$.

confidence interval [CI] 2.41–13.14; $P < .001$) and 2- to 4-year-olds (OR 3.47; 95% CI 1.25–9.63; $P = .02$) were more likely to have positive cotinine levels than 10- to 17-year-olds. Patients with public insurance or who were self-pay were 8.7 times (95% CI 4.31–17.71; $P < .001$) more likely to have positive cotinine levels than patients with private insurance. A total of 57.3% of children with a PMH of asthma or bronchiolitis had positive cotinine levels. Those with a PMH of bronchiolitis (OR 2.34; 95% CI 1.06–5.20; $P = .04$), a bronchiolitis diagnosis (OR 3.69; 95% CI 1.52–8.99; $P = .004$), or asthma and/or bronchiolitis diagnosis (OR 2.62; 95% CI 1.35–5.08; $P = .004$) were at increased risk of having positive cotinine levels compared with those without these PMH or discharge diagnoses. Patients who had ≥1 ED visit (excluding an ED visit associated with the index hospitalization) within the past 6 months were 2.4 times (95% CI 1.27–4.42; $P = .007$) more likely to have positive cotinine levels compared with those with no ED visits.

Age, insurance type, and asthma and/or bronchiolitis diagnosis remained statistically significant in the adjusted models (Table 2). While assessing whether insurance status accounted for the variance independent of age, we found that patients with public insurance or self-pay were more likely to be 0 to 1 year old ($P = .02$), be African American ($P = .02$), be of other race ($P = .05$), or have positive TSE status in the EHR ($P < .001$).

DISCUSSION

This study tested the accuracy of EHR documentation of hospitalized pediatric patients' TSE status using cotinine verification. In our study, we found a lack of concordance with EHR classification of TSE status and biochemically verified TSE. Specifically, we found that 39% of the children who were thought not to be exposed to tobacco smoke on the basis of their EHR were actually exposed on the basis of positive cotinine levels. Conversely, we found that 19% of children who had positive TSE documented in the EHR were not exposed on the basis of negative cotinine levels. Our findings were similar to those of other studies in different settings. A study of children with asthma recruited from health departments and asthma clinics[26] found poor agreement across all clinics between EHR documentation and parent report, although agreement was higher within asthma specialty clinics (κ 0.410 compared with 0.205). In 1 study of infants and young children in a primary care clinic, reported TSE based on 1 question about exposure to passive TSE was 13% compared with 55% with cotinine-verified exposure.[16] In another study of children hospitalized with asthma, parents were asked 3 questions about potential TSE, and 65% reported no exposure, yet 70% of children had positive salivary cotinine levels.[13]

Factors that may lead to discrepancies we found in the EHR compared with cotinine results include a lack of standardized methods to assess TSE via parent reports, the use of an outdated TSE status, underreporting of child TSE, and parents' lack of awareness of all TSE sources.[10,13,27,28] The latter possibility is important to consider given the many sources of potential TSE in children's environments of which parents may be unaware.[29,30] We were unable to assess different TSE sources (eg, electronic cigarettes and multiunit housing) and locations of exposure (eg, home, day care, cars, and public areas) because we did not collect these data. Future research should examine these factors among hospitalized pediatric patients to provide further insight into reporting discrepancies. Furthermore, our finding that 19% of children with positive TSE status were actually unexposed on the basis of negative cotinine levels may have been due to the timing of saliva collection. Some saliva samples were obtained later during the hospitalization, which may have resulted in decreased cotinine levels because cotinine has a half-life of 16 to 18 hours.[12] However, the mean collection time was ~19 hours with little variation; thus, the cotinine levels of the children in this study

population may have been higher if obtained earlier in the hospitalization.

Our results indicate that children age 4 years and younger had higher rates of biochemically validated TSE compared with children age 5 to 9 years and 10 to 17 years. This may be because younger children have decreased opportunities to leave environments where cigarettes are smoked compared with older children. In contrast to other research that reported higher TSE rates in non-Hispanic African American children,[13,16,31] we did not find higher TSE rates in this population. However, consistent with other studies, we found that those with TSE were >8 times more likely to have public or self-pay insurance (a proxy for low income).[13,31,32] Similar to previous studies,[19,20,33] we found that children with a diagnosis of asthma or bronchiolitis were twice as likely to have TSE. However, if PMH of asthma or bronchiolitis alone were used to determine who should be screened for TSE, then 57.3% of exposed children would have been missed. Thus, these findings suggest that screening should be considered in all hospitalized children regardless of their sociodemographic background, PMH, or current diagnoses. Additionally, the high prevalence of TSE in this population is consistent with our previous work in patients in the pediatric ED,[34,35] and these high prevalence rates provide further impetus to provide parental cessation interventions in the pediatric hospital setting. Previous research of parental smokers in the hospital setting indicates that parents are interested in receiving counseling and that after brief counseling, self-reported quitting and TSE behavior changes are encouraging.[36–39] More research is needed to further develop and test the efficacy of these interventions.

Limitations of this study include the use of a convenience sample of participants, which limits generalizability. We assessed cotinine in saliva using the ELISA method, which is not as sensitive or specific as other methods, such as liquid chromatography mass spectrometry[40]; however, the use of ELISA to measure cotinine does have good sensitivity and specificity.[41] The screening questions that were used in the EHRs were nonspecific and did not account for many factors that affect children's TSE levels, such as the type of tobacco or nicotine product and the locations, amount, and frequency of exposure. Future research and quality-improvement projects should expand the TSE screening questions to account for other factors affecting exposure and address tobacco or nicotine product use of any form by parents.

CONCLUSIONS

Our findings indicate that it is not adequate to rely on EHR classification or parental reports of exposure. Almost 40% of participants in the current study were misclassified as nonexposed to tobacco smoke. We found that a high proportion of hospitalized children with both respiratory and nonrespiratory PMHs were exposed to tobacco smoke. Given the significant health benefits of a smoke-free environment for hospitalized children, the reliance on EHR classification to determine TSE status may result in missed opportunities for reducing TSE in this vulnerable population. Our findings provide support for the recommendation to adopt a universal approach to TSE-related screening. The regular collection of cotinine assays would greatly reduce the underreporting biases that are prevalent during pediatric health care visits and improve the accuracy of EHR TSE documentation.[13,18,33] If this is not feasible because of costs or other concerns, incorporating standardized and expanded TSE screening and counseling into pediatric hospital settings, regardless of the child's diagnosis, could significantly decrease TSE and related morbidity in children.[4]

REFERENCES

1. Tsai J, Homa DM, Gentzke AS, et al. Exposure to secondhand smoke among nonsmokers - United States, 1988-2014. *MMWR Morb Mortal Wkly Rep*. 2018; 67(48):1342–1346
2. US Department of Health and Human Services. *The Health Consequences of Smoking—50 Years of Progress: A Report of the Surgeon General*. Atlanta, GA: US Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health; 2014
3. Committee on Environmental Health; Committee on Substance Abuse; Committee on Adolescence; Committee on Native American Child. From the American Academy of Pediatrics: policy statement–tobacco use: a pediatric disease [published correction appears in *Pediatrics*. 2010;125(4):861]. *Pediatrics*. 2009;124(5):1474–1487
4. Farber HJ, Walley SC, Groner JA, Nelson KE; Section on Tobacco Control. Clinical practice policy to protect children from tobacco, nicotine, and tobacco smoke. *Pediatrics*. 2015;136(5):1008–1017
5. Fiore MC, Jaén CR, Baker TB, et al. *Treating Tobacco Use and Dependence: 2008 Update—Clinical Practice Guideline*. Rockville, MD: US Department of Human Services; 2008
6. Jenssen BP, Wilson KM. Tobacco control and treatment for the pediatric clinician: practice, policy, and research updates. *Acad Pediatr*. 2017;17(3):233–242
7. Office of the National Coordinator for Health Information Technology. Non-federal acute care hospital electronic health record adoption. 2017. Available at: https://dashboard.healthit.gov/quickstats/pages/FIG-Hospital-EHR-Adoption.php. Accessed October 15, 2018
8. 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):214
9. Poon EG, Wright A, Simon SR, et al. Relationship between use of electronic health record features and health care quality: results of a statewide survey. *Med Care*. 2010;48(3):203–209
10. Wilson KM, Wesgate SC, Best D, Blumkin AK, Klein JD. Admission screening for secondhand tobacco smoke exposure. *Hosp Pediatr*. 2012;2(1):26–33
11. Andrews AL, Shirley N, Ojukwu E, Robinson M, Torok M, Wilson KM. Is secondhand smoke exposure associated with increased exacerbation severity among children hospitalized for asthma? *Hosp Pediatr*. 2015;5(5):249–255

12. Benowitz NL, Hukkanen J, Jacob P III. Nicotine chemistry, metabolism, kinetics and biomarkers. *Handb Exp Pharmacol*. 2009;(192):29–60

13. Howrylak JA, Spanier AJ, Huang B, et al. Cotinine in children admitted for asthma and readmission. *Pediatrics*. 2014; 133(2). Available at: www.pediatrics.org/cgi/content/full/133/2/e355

14. Spanier AJ, Kahn RS, Xu Y, Hornung R, Lanphear BP. Comparison of biomarkers and parent report of tobacco exposure to predict wheeze. *J Pediatr*. 2011;159(5): 776–782

15. Connor Gorber S, Schofield-Hurwitz S, Hardt J, Levasseur G, Tremblay M. The accuracy of self-reported smoking: a systematic review of the relationship between self-reported and cotinine-assessed smoking status. *Nicotine Tob Res*. 2009;11(1):12–24

16. Dempsey DA, Meyers MJ, Oh SS, et al. Determination of tobacco smoke exposure by plasma cotinine levels in infants and children attending urban public hospital clinics [published correction appears in *JAMA Pediatr*. 2014;168(8):779]. *Arch Pediatr Adolesc Med*. 2012;166(9):851–856

17. Joseph A, Spector L, Wickham K, et al. Biomarker evidence of tobacco smoke exposure in children participating in lead screening. *Am J Public Health*. 2013; 103(12):e54–e59

18. Benowitz NL, Jain S, Dempsey DA, Nardone N, Helen GS, Jacob P III. Urine cotinine screening detects nearly ubiquitous tobacco smoke exposure in urban adolescents. *Nicotine Tob Res*. 2017;19(9):1048–1054

19. Snodgrass AM, Tan PT, Soh SE, et al; GUSTO Study Group. Tobacco smoke exposure and respiratory morbidity in young children. *Tob Control*. 2016;25(e2):e75–e82

20. Ahn A, Edwards KM, Grijalva CG, et al. Secondhand smoke exposure and illness severity among children hospitalized with pneumonia. *J Pediatr*. 2015;167(4): 869–874.e1

21. Salimetrics, LLC. *High Sensitivity Salivary Cotinine Quantitative Enzyme Immunoassay Kit*. State College, PA: Salimetrics, LLC; 2015

22. Butz AM, Halterman JS, Bellin M, et al. Factors associated with second-hand smoke exposure in young inner-city children with asthma. *J Asthma*. 2011; 48(5):449–457

23. Kumar R, Curtis LM, Khiani S, et al. A community-based study of tobacco smoke exposure among inner-city children with asthma in Chicago. *J Allergy Clin Immunol*. 2008;122(4):754–759.e1

24. Butz AM, Tsoukleris M, Elizabeth Bollinger M, et al. Association between second hand smoke (SHS) exposure and caregiver stress in children with poorly controlled asthma. *J Asthma*. 2018:1–12

25. R Core Team. *R: A Language and Environment for Statistical Computing*. Vienna, Austria: R Foundation for Statistical Computing; 2013

26. Harrington KF, Haven KM, Nuño VL, Magruder T, Bailey WC, Gerald LB. Parent report and electronic medical record agreement on asthma education provided and children's tobacco smoke exposure. *J Asthma*. 2013;50(9):968–974

27. Stuber J, Galea S, Link BG. Smoking and the emergence of a stigmatized social status. *Soc Sci Med*. 2008;67(3):420–430

28. Stuber J, Galea S. Who conceals their smoking status from their health care provider? *Nicotine Tob Res*. 2009;11(3): 303–307

29. Jacob P III, Benowitz NL, Destaillats H, et al. Thirdhand smoke: new evidence, challenges, and future directions. *Chem Res Toxicol*. 2017;30(1):270–294

30. Matt GE, Quintana PJ, Hovell MF, et al. Households contaminated by environmental tobacco smoke: sources of infant exposures. *Tob Control*. 2004;13(1):29–37

31. Homa DM, Neff LJ, King BA, et al; Centers for Disease Control and Prevention. Vital signs: disparities in nonsmokers' exposure to secondhand smoke–United States, 1999-2012. *MMWR Morb Mortal Wkly Rep*. 2015;64(4):103–108

32. Shenassa ED, Rossen LM, Cohen J, Morello-Frosch R, Payne-Sturges DC. Income inequality and US children's secondhand smoke exposure: distinct associations by race-ethnicity. *Nicotine Tob Res*. 2017;19(11):1292–1299

33. Butz A, Bellin MH, Bollinger ME, et al. Salivary cotinine measurement for all children with persistent asthma: spit matters. *Ann Allergy Asthma Immunol*. 2016;116(5):463–465

34. Mahabee-Gittens EM, Gordon JS. Missed opportunities to intervene with caregivers of young children highly exposed to secondhand tobacco smoke [published correction appears in *Prev Med*. 2016;84: 97]. *Prev Med*. 2014;69:304–305

35. Mahabee-Gittens EM, Merianos AL, Matt GE. Preliminary evidence that high levels of nicotine on children's hands may contribute to overall tobacco smoke exposure. *Tob Control*. 2018;27(2):217–219

36. Behbod B, Sharma M, Baxi R, Roseby R, Webster P. Family and carer smoking control programmes for reducing children's exposure to environmental tobacco smoke. *Cochrane Database Syst Rev*. 2018;1:CD001746

37. Ralston S, Roohi M. A randomized, controlled trial of smoking cessation counseling provided during child hospitalization for respiratory illness. *Pediatr Pulmonol*. 2008;43(6):561–566

38. Ralston S, Grohman C, Word D, Williams J. A randomized trial of a brief intervention to promote smoking cessation for parents during child hospitalization. *Pediatr Pulmonol*. 2013; 48(6):608–613

39. Walley SC, Chime C, Powell J, Walker K, Burczyk-Brown J, Funkhouser E. A brief inpatient intervention using a short video to promote reduction of child tobacco smoke exposure. *Hosp Pediatr*. 2015;5(10):534–541

40. Avila-Tang E, Elf JL, Cummings KM, et al. Assessing secondhand smoke exposure with reported measures. *Tob Control*. 2013;22(3):156–163

41. Avila-Tang E, Al-Delaimy WK, Ashley DL, et al. Assessing secondhand smoke using biological markers. *Tob Control*. 2013;22(3):164–171

Electronic Health Record Classification of Tobacco Smoke Exposure and Cotinine Levels in Hospitalized Pediatric Patients

E. Melinda Mahabee-Gittens, Ashley L. Merianos, Judith S. Gordon, Lara Stone, Olga Semenova and Georg E. Matt

Hospital Pediatrics 2019;9;659
DOI: 10.1542/hpeds.2018-0247 originally published online August 26, 2019;

Hospital Pediatrics®

AN OFFICIAL JOURNAL OF THE AMERICAN ACADEMY OF PEDIATRICS

Electronic Health Record Classification of Tobacco Smoke Exposure and Cotinine Levels in Hospitalized Pediatric Patients

E. Melinda Mahabee-Gittens, Ashley L. Merianos, Judith S. Gordon, Lara Stone, Olga Semenova and Georg E. Matt

Hospital Pediatrics 2019;9;659
DOI: 10.1542/hpeds.2018-0247 originally published online August 26, 2019;