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Preliminary evidence that high levels of nicotine on children's hands may contribute to overall tobacco smoke exposure

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ABSTRACT

Background Dust and surfaces are important sources of lead and pesticide exposure in young children. The purpose of this pilot study was to investigate if third-hand smoke (THS) pollutants accumulate on the hands of children who live in environments where tobacco is used and if hand nicotine levels are associated with second-hand smoke (SHS), as measured by salivary cotinine.

Methods Participants were parents and children (n=25; age mean (SD)=5.4 (5.3) years) presenting to the emergency department with a potentially SHS-related illness. A convenience sample of participants were recruited at baseline from an ongoing two-group, randomised controlled trial of a SHS reduction and tobacco cessation intervention. Parents were current smokers; thus, all children were at risk of SHS and THS exposure to varying extents. Primary outcome measures, which were assessed in child participants only, were hand nicotine and salivary cotinine. Parents reported sociodemographics and smoking patterns; children's medical records were abstracted for chief complaint, medical history and discharge diagnosis.

Results All children had detectable hand nicotine (range=18.3–690.9 ng/wipe). All but one had detectable cotinine (range=1.2–28.8 ng/mL). Multiple linear regression results showed a significant positive association between hand nicotine and cotinine (p=0.009; semipartial r^2=0.24), independent of child age.

Discussion The higher-than-expected nicotine levels and significant association with cotinine indicate that THS may play a role in the overall exposure of young children to tobacco smoke toxicants and that hand wipes could be a useful marker of overall tobacco smoke pollution and a proxy for exposure.

Trial registration number ClinicalTrials.gov Identifier: NCT02531594

INTRODUCTION

House dust and surfaces are important exposure sources of environmental toxicants (eg, pesticides and lead) in young children because of age-associated behaviours and interactions with their environment (eg, hand-to-mouth behaviours and close physical contact with parents).[1] Research shows the persistent residue from second-hand smoke (SHS) accumulates in dust, in objects, on home surfaces and on smokers' skin and clothes.[2] Commonly referred to as third-hand smoke (THS), non-smokers may be exposed to toxicants (eg, tobacco specific nitrosamine)[3] via inhalation, ingestion and dermal uptake transferred from indoor deposits and parents. Matt *et al*[2 4 5] and Quintana *et al*[6] found that nicotine levels on hands differentiate non-smokers from smokers, and among non-smokers, these are associated with nicotine on home surfaces and in dust. On non-smokers' hands, nicotine can serve as a proxy of THS pollution in an individual's immediate environment, as well as a proximate and immediate cause of nicotine exposure.[4] In addition, cotinine is the major metabolite of nicotine and a biomarker of SHS and THS exposure in non-smokers.[7]

The purpose of this pilot study was to investigate if THS pollutants accumulate on the hands of ill children who live in environments where tobacco is used and to describe any associated clinical characteristics. We compared THS levels on children's hands with those found in other populations and examined the association between THS levels and the biological exposure to tobacco smoke, as measured with salivary cotinine, to determine if the nicotine on children's hands may serve as a proxy measure of overall tobacco smoke (OTS; defined as SHS and THS combined) exposure from children's environments.

METHODS

A convenience sample of participants were recruited at baseline from an ongoing two-group, randomised controlled trial of SHS reduction and tobacco cessation intervention for caregivers who smoke, registered on http://clinicaltrials.gov (NCT02531594). Paediatric patients (n=25) presenting to the emergency department between April and September 2016 of a tertiary care children's hospital in the USA with a potentially SHS-related illness (eg, rhinorrhoea and difficulty breathing) were potentially eligible. All children were at risk of varying smoke exposure. Institutional Review Board parental consent and child assent were obtained. The children provided a saliva sample to obtain cotinine levels. The palm and the volar aspect (ie, palmar surface) of all fingers of their dominant hand were wiped by trained research staff and analysed for nicotine; field blanks were collected to adjust for potential contamination of wipe samples (range 0.41–0.48 ng/wipe).[6] Parents reported sociodemographics, smoking patterns and household characteristics; children's medical records were abstracted for chief complaint, medical history and discharge diagnosis.

STATISTICAL ANALYSES

We calculated geometric means (GeoMs), CIs, medians (Mdn) and IQRs. We conducted linear

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regression analyses to examine associations between salivary cotinine and hand nicotine (both log-transformed), sociodemographics, smoking behaviours, household characteristics and clinical findings. The alpha level of significance was set at 0.05 (two-tailed).

RESULTS

See table 1 for descriptive information about the sample. All but one child had detectable cotinine (Level of Detection, LOD=1.0 ng/mL) with the range of 1.2–28.8 ng/mL (GeoM=5.2 ng/mL, 95% CI 95% 3.5 to 7.6, Mdn=5.3 ng/mL, IQR=2.3–9.1). The cotinine value below LOD was recoded to 0.50 ng/mL (ie, one-half the LOD) and included in all analyses. All children had detectable nicotine on their hands (LOD=0.05 ng nicotine), with the range of 18.26–690.94 ng/wipe (GeoM=86.4 ng/wipe, 95% CI (61.0 to 122.2), Mdn=91.6 ng/wipe, IQR=57.2–121.6).

To be eligible for the study, children had to have an SHS-related complaint. Over half (52%) had a medical history or discharge diagnosis of asthma or bronchiolitis. Of note, the child with the highest nicotine level was 2 years old and had a chief complaint of difficulty breathing and a discharge diagnosis of asthma. There were no statistically significant associations between sociodemographics, smoking behaviour and household or clinical characteristics. Bivariate associations showed a positive correlation between cotinine and hand nicotine (r=0.473, p=0.017). We found a negative association between cotinine and age (r=−0.334, p=0.103) and no association between hand nicotine and age (r=0.047, p=0.823). When combined in a multiple linear regression model (R^2=0.35, F (2, 22)=5.93, p=0.009), hand nicotine showed an independent significant positive association with cotinine (p=0.009; semipartial r^2=0.24), and age showed a marginally significant independent negative association with cotinine (p=0.0502; semipartial r^2=0.13). There was no significant moderator effect of age. In combination, these findings show that, independent of age, higher levels of hand nicotine were associated with higher levels of cotinine. Moreover, independent of hand nicotine, younger children had higher levels of cotinine. Reported smoking behaviour was not correlated with nicotine or cotinine.

Table 1 Participant characteristics

Characteristic	
Age of child (years)	
25th–50th–75th percentiles	0.8–2.1–9.9
Race of child, n (%)	
Black, non-Hispanic	18 (75.0)
White, non-Hispanic	5 (20.8)
Other, non-Hispanic	1 (4.2)
Sex of child, n (%)	
Male	13 (52.0)
Insurance type, n (%)	
Public/self-pay	22 (88.0)
Private	3 (12.0)
Income level, n (%)	
<US$14 999	16 (66.7)
Number of smokers, n (%)	
1 smoker	18 (72.0)
Number of cigarettes/day, n (%)	
<10	15 (60.0)
Daily electronic cigarette use, n (%)	
Yes	1 (4.0)
Housing, n (%)	
Single-family	13 (52.0)
Multiunit or apartment	12 (48.0)
Cleaning frequency, n (%)	
Daily	19 (76.0)
Chief complaints, n (%)	
Cough or difficulty breathing	12 (48.0)
Past medical history, n (%)	
Asthma	8 (32.0)
Asthma or bronchiolitis	10 (40.0)
Discharge diagnosis, n (%)	
Asthma or bronchiolitis	8 (32.0)
Past medical history or discharge diagnosis, n (%)	
Asthma or bronchiolitis	13 (52.0)

DISCUSSION

Our findings indicate that children carry tobacco smoke toxicants on their hands, even when nobody around them is smoking. Thus, nicotine and other THS compounds on children's hands may contribute to OTS, independent of SHS. Moreover, nicotine on children's hands may serve as a proxy of tobacco smoke pollution in their immediate environment. We found that non-smoking children whose parents smoke had higher-than-expected levels of nicotine on their hands. Specifically, the GeoM of 86.4 ng/wipe was over three times higher than the levels of 25.6 ng/wipe in previous research on non-smoking adults living with active smokers.[4] In other research in which finger wipes of adults were analysed, we have reported GeoMs of 5.2 ng/wipe among non-smokers moving into an apartment formerly occupied by a smoker[5] and 630–650 ng/wipe among active smokers.[2 5] Because nicotine is specific to tobacco, its presence on children's hands may serve as a proxy of tobacco smoke pollution in their immediate environment. This finding suggests that OTS may occur through a combination of SHS and THS exposure via multiple pathways; thus, a comprehensive assessment of OTS needs to include assessments of both SHS and THS exposure. Hand wipes may also be useful in the clinical setting, as these may provide a relatively unobtrusive test of OTS pollution and exposure. However, more research is needed to assess the reliability and validity of such a test and if hand wipe analyses can be conducted in a cost-effective and timely enough manner that would improve clinical care of patients exposed to smoke.

All children had potentially SHS-related complaints, and many had a discharge diagnosis or history of asthma or bronchiolitis. While causal associations cannot be made between hand nicotine levels and clinical findings, these results warrant further examination, given that recent work demonstrates the association of THS exposure and respiratory symptoms in exposed children.[8] Although unexamined, there is concern that THS may be more toxic than SHS, given that THS includes novel pollutants not found in SHS, has multiple exposure routes and has a much longer duration of exposure.[9–12]

The positive partial association between nicotine and cotinine indicates that, independent of age, hand nicotine can serve as a marker of OTS. The exposure may be through SHS or THS that accumulated in dust, on objects and on surfaces of the children's environments. The negative partial association between age and cotinine indicates that, independent of

nicotine, younger children show higher exposure to tobacco smoke. This finding supports the hypothesis that younger children interact with their environment in ways that increase the uptake of THS pollutants. This may be because younger children receive higher doses of THS (ie, more pica behaviour) or higher doses of SHS (ie, limited opportunities to leave smoking areas).

Since hand nicotine may have its origins in SHS and THS and because of its significant association with systemic biological exposure to tobacco smoke pollutants as measured by cotinine, our findings support the use of hand nicotine as a marker of OTS pollution in children's environments. Because SHS and THS are composed of constituents that cause many different adverse health effects,[10–13] these findings support additional epidemiological and experimental studies.

This study has limitations, including the inability to measure SHS independent of THS pollution or to distinguish how THS and SHS independently contribute to OTS exposure. Thus, hand nicotine may be a direct consequence and proxy of SHS exposure or contracted through interaction with THS pollutants in a child's environment without SHS exposure.[14 15] In either case, nicotine on a child's hands is prima facie evidence of OTS in the child's environment and a source of THS exposure. However, independent measures of SHS and THS exposure are needed. Furthermore, the results from this small sample size warrant replications in larger samples taken from similar and different child populations to assess if our findings can be confirmed or may be moderated by characteristics of the child (eg, age) or his/her environment (eg, THS reservoirs at home). Another limitation is that we may have found higher-than-expected nicotine levels, because we wiped the insides of children's entire hands (ie, palm and volar aspect of fingers). Our ongoing research now includes hand surface area measurements so that we can compare new findings with our previous work.[16]

Limitations notwithstanding, the presence of nicotine on children's hands and the association between hand nicotine and salivary cotinine makes it plausible that OTS in children is not limited to airborne pollutants (ie, SHS) but should include THS pollutants in children's entire physical environment. Future work should differentiate how much SHS and THS contribute to OTS, how THS and SHS exposure affect child health and which types of prevention interventions are needed to better protect children from OTS.

What this paper adds

- This is the first study to show that children of smokers carry tobacco smoke toxicants on their hands, even when nobody around them is smoking at the time.
- Children had high levels of hand nicotine, higher than that of non-smokers in our previous work.
- The significant association between hand nicotine and salivary cotinine suggests that hand nicotine may contribute to overall tobacco smoke exposure, independent of SHS, and can serve as a marker of overall tobacco smoke exposure.

Contributors EMMG conceived the study, overall study aims and study design and wrote the first draft of the paper. GEM guided the sample and data collection protocol and developed the specific study aims. GEM and ALM conducted the statistical analysis. All authors made critical comments and revised drafts of the paper. All authors read and approved the final manuscript.

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Competing interests None declared.

Patient consent Our institutional review board (IRB) has approved the study, and we have IRB-approved participant consent and child assent forms that have been signed by participants.

Ethics approval Cincinnati Children's Hospital Medical Center IRB.

Provenance and peer review Not commissioned; externally peer reviewed.

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