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Journal
Nicotine & Tobacco Research, 15(12)

ISSN
1462-2203

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Publication Date
2013-12-01

DOI
10.1093/ntr/ntt096

Peer reviewed

ORIGINAL INVESTIGATION

Levels of Nicotine in Dust From Homes of Smokeless Tobacco Users

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Received March 1, 2013; accepted June 12, 2013

ABSTRACT

Background: Smokeless tobacco products, such as chewing tobacco or moist snuff, contain many of the same constituents as tobacco smoke and are also known to cause cancer; however, little attention has been paid to indirect exposure of children to tobacco constituents via parental smokeless tobacco use.

Methods: As part of the California Childhood Leukemia Study, we collected dust samples from 6 residences occupied by smokeless tobacco users, 6 residences occupied by active smokers, and 20 tobacco-free residences. Children's potential for exposure to tobacco constituents was assessed using nicotine concentrations in vacuum dust measured by gas chromatography-mass spectrometry.

Results: Median nicotine concentrations for residences with smokeless tobacco users were significantly greater than median nicotine concentrations for tobacco-free homes and similar to median nicotine concentrations in homes of active smokers. Using generalized estimating equations derived from a multivariable marginal model to adjust for a history of parental smoking, income, residence construction date, and mother's age and race/ethnicity, we found nicotine levels from homes of smokeless tobacco users to be 21-fold higher than nicotine levels from tobacco-free homes. Based on mass balance equations, we hypothesize that nicotine is transferred to floors in homes of smokeless tobacco users primarily as a constituent of tobacco that is spilled or expectorated.

Conclusions: Based on our findings, we conclude that children living with smokeless tobacco users may be exposed to nicotine and other constituents of tobacco via contact with contaminated dust and household surfaces.

INTRODUCTION

Smokeless tobacco refers to products that contain tobacco as the principal constituent and are used either orally or nasally without combustion (International Agency for Research on Cancer, 2007). The most widely used smokeless tobacco product in the United States is moist snuff followed by loose leaf chewing tobacco (Capehart, 2006). Moist snuff is tobacco that is cured, ground into fine particles, and typically packaged in cans; loose leaf chewing tobacco is cured, stemmed, sweetened, cut into strips, and typically packaged in foil pouches (International Agency for Research on Cancer, 2007). Although the prevalence of adult cigarette smoking in the United States has decreased (Centers for Disease Control and Prevention, 2009), the prevalence of smokeless tobacco use among Americans aged 12 and older has remained constant over the past decade at 3.0%–3.5% (Substance Abuse and Mental Health Services Administration, 2012). Nicotine is a major component of smokeless tobacco products (Djordjevic, Hoffmann, Glynn, & Connolly, 1995).

The use of smokeless tobacco is causally associated with cancers of the oral cavity and pancreas (International Agency for Research on Cancer, 2007). Indeed, several suspected carcinogens are found in smokeless tobacco products, including the nonvolatile tobacco-specific nitrosamines, *N*′-nitrosonornicotine (NNN) and 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK), as well as various volatile nitrosamines, nitrosoamino acids, aldehydes (including formaldehyde), benzo(a)pyrene, nickel, and uranium (International Agency for Research on Cancer, 2007).

There are at least two ways that smokeless tobacco could be released into the residential environment (e.g., the carpet)—tobacco application and expectoration. Users generally place tobacco between their cheek or lip and gums and subsequently expectorate the tobacco-laden saliva (International Agency for Research on Cancer, 2007), thereby contaminating

doi:10.1093/ntr/ntt096
Advance Access publication July 24, 2013

indoor surfaces and carpets. Moreover, semivolatile organic compounds in the indoor environment partition across sources, indoor air, and indoor surfaces (Weschler & Nazaroff, 2008). Thus, some constituents of smokeless tobacco (e.g., nicotine) may be released to indoor air via exhaled breath of the user and subsequently adsorbed on dust particles or surfaces.

Young children spend more time in the home environment, especially near the floor, and are more likely to make hand-to-mouth contact than their adult counterparts (Cohen Hubal et al., 2000). Moreover, young children are less likely than teenagers or adults to use tobacco products (Harrell, Bangdiwala, Deng, Webb, & Bradley, 1998). Thus, compared with adults, young children potentially receive a relatively large portion of their total exposure to hazardous tobacco constituents via the ingestion of settled dust.

As part of the California Childhood Leukemia Study, we collected dust samples and compared nicotine concentrations from residences occupied by smokeless tobacco users, residences occupied by active smokers, and tobacco-free residences. Using mass balance equations, we tried to deduce the mechanism of nicotine transfer from smokeless tobacco products to settled dust. Based on our results, we assess the potential for exposure to tobacco constituents for a child sharing a residence with a smokeless tobacco user.

METHODS

Study Population

The California Childhood Leukemia Study is a case–control study of childhood leukemia conducted in the San Francisco Bay area and California Central Valley that seeks to identify genetic and environmental risk factors for childhood leukemia. Case and control subjects that were enrolled in the study from December 1999 to November 2007 were eligible for initial dust collection if they were 0–7 years old. Subsequently, in 2010, a subset of the subjects that participated in the initial dust collection and still lived in the same residence was eligible for a second dust collection. Among 629 subjects who participated in the initial dust collection, 225 were eligible for a second dust collection and 204 participated. Of the 204 participating residences, six were occupied by a smokeless tobacco user. We analyzed two dust samples for nicotine from each of five of these residences occupied by a smokeless tobacco user but were only able to analyze nicotine in one dust sample from the remaining residence. For comparison, we analyzed two dust samples for nicotine from each of six randomly selected residences occupied by an active smoker and 20 randomly selected tobacco-free residences. We obtained written informed consent from the participating families in accordance with the institutional review boards' requirements at the University of California, Berkeley.

Collection of Vacuum Dust

During the first round of dust sampling (2002–2007), we obtained vacuum cleaner dust and administered a questionnaire during an in-home visit. During the second round of dust sampling (2010), we interviewed subjects via telephone and instructed them to mail their vacuum cleaner bags (or the contents of their vacuum cleaner canisters) to the study center in prepaid parcels. The median interval between paired sample collections was 4.7 years (range: 2.9–8.2 years). We stored dust samples in the dark at or below 4°C prior to chemical analysis. We previously analyzed the dust samples from the first round of dust collection for nicotine (Whitehead et al., 2009); however, for consistency, the dust samples from the first round of dust collection were re-extracted and reanalyzed together with samples from the second round of dust collection.

Laboratory Analysis of Nicotine

We homogenized and fractionated each dust sample using a mechanical shaker equipped with a 100-mesh sieve to obtain dust particles smaller than 150 µm. Prior to each extraction, hexane, butanol, toluene, and water were each passed through a benzenesulfonic acid silica-gel cartridge (United Chemical Technologies), and glassware was heated in a muffle furnace at 500°C for 3 hr to remove background nicotine contamination. The liquid–liquid extraction protocol used for this analysis was adapted from Jacob, Wu, Yu, and Benowitz (2000). To a 0.1-g portion of dust in a centrifuge tube was added an internal standard (40 ng of methyl-d_3-nicotine) and a 2-ml aqueous solution containing 45% potassium carbonate and 5% edetic acid, followed by sonication for 10 min. Subsequently, 5 ml of 70:30 toluene:butanol was added, the sample was vortex-mixed for 5 min, the organic and aqueous phases were separated by centrifugation at 3,000 rpm for 5 min, and the tube was placed in a dry ice-acetone bath to rapidly freeze the aqueous layer. The organic layer was then transferred to a tube containing 0.5 ml of 1 M sulfuric acid, and the sample was vortex-mixed, centrifuged, and frozen again. After discarding the organic layer, the aqueous phase was washed with 5 ml of 2:1 ethyl acetate:toluene and again vortex-mixed, centrifuged, and frozen. After discarding the organic layer, the remaining aqueous layer was neutralized with 0.5 ml of an aqueous solution containing 50% potassium carbonate and subsequently 200 µl of 90:10 toluene:butanol was added. Finally, after vortex-mixing, centrifuging, and freezing, the organic phase was transferred to 300-µl silanized autosampler vials and spiked with an injection standard (80 ng of d_{10}-pyrene). A 2-µl injection was analyzed for nicotine using gas chromatography-mass spectrometry in the multiple-ion detection mode (ions used for quantitation: m/z = 162 for nicotine, m/z = 165 for methyl-d_3-nicotine, and m/z = 212 for d_{10}-pyrene). The gas chromatograph was equipped with an ultra-inert splitless injection liner with glass wool and an ultra-inert column (DB-5MS-UI, 30 m, 0.25-mm i.d., 0.25-µm film) from Agilent Technologies, and the chromatographic separation was programmed from 40°C to 150°C at 50°C per min and then from 150°C to 320°C at 10°C per min with a 1-min initial hold time. We analyzed a five-point calibration curve (range 50–200,000 ng/ml) at the beginning and the end of all analyses and a single point standard with each sample batch.

Analytical Precision and Sensitivity

We performed analyses in batches of 12, consisting of seven single samples, one method blank, one duplicate sample pair (i.e., two 200 mg portions of fine dust taken from the same vacuum cleaner) and one duplicate quality control sample pair (i.e., two 200 mg portions of fine dust taken from a pool of nicotine-fortified dust collected from a representative vacuum

cleaner). The coefficient of variation of nicotine concentrations in 10 pairs of duplicate samples was 9.7%. Based on the observed nicotine content of 12.0 ± 5.4 ng ($M \pm SD$) in 10 method blanks, we estimated a limit of detection (LOD) of 270 ng/g dust, equivalent to a signal-to-noise ratio of five. We did not correct nicotine concentrations in samples for the nicotine content in blanks.

Questionnaires

Parents who participated in the dust collection initially responded to a structured in-home interview designed to ascertain information relevant to childhood leukemia, including (but not limited to) parental race/ethnicity and age, household annual income, and exposure to cigarette smoke. The initial interviews were conducted from 2001 to 2007, on average 6 months prior to the first round of dust collection (2002–2007). Subsequently, at the time of the second dust collection in 2010, participating households completed a second questionnaire by telephone designed to ascertain information about sources of residential chemical exposures and residential characteristics, such as the construction date, type, and square footage.

During both interviews (2001–2007 and 2010), respondents (primarily mothers, i.e., 99%) were asked to report current and past household smoking habits. Specifically, during the initial questionnaire (2001–2007), respondents were asked to report the history of active smoking for each parent at various times (i.e., lifetime, before, during, and after the index pregnancy, at the time of the interview). In addition, respondents were asked to report the history of passive smoking exposures in the home, at work/childcare, in the car, and in public/social settings for the mother, father, and child at various times. During the second interview (2010), respondents were asked to characterize household smoking habits during the past year and the history of household smoking since moving into their current home by reporting whether anyone had regularly smoked cigarettes, pipes, or cigars inside the home and whether any resident had regularly smoked outside the home (e.g., on the deck, in the yard, in the car, or at work). During the second questionnaire (2010), respondents were also asked whether anyone used smokeless tobacco products such as dipping or chewing tobacco in the home once a week or more during the last 12 months.

Statistical Analysis

Based on questionnaire responses from the interviews conducted in 2010, we stratified households into residences occupied by smokeless tobacco users (i.e., regular use at home during the previous year), residences occupied by active smokers (i.e., regular smoking by a resident inside [$N = 1$] or outside [$N = 5$] of the home during the previous year), and tobacco-free residences (i.e., no smokeless tobacco use at home during the previous year and no active smoking by a resident since the family moved into the home). We compared nicotine concentrations between tobacco-use categories using the Wilcoxon two-sample Z-test. We also tested whether observed differences in logged nicotine concentrations by tobacco-use category remained significant after adjustment for a history of parental smoking at prior residences, household annual income, residence construction date, mother's age at dust collection, and mother's race/ethnicity using generalized estimating equations derived from a multivariable marginal model. These contextual variables were previously associated with nicotine concentrations in dust from California Childhood Leukemia Study (CCLS) homes (Whitehead et al., 2009). Demographic descriptors of mothers and fathers were mostly concordant within a household, so we used the more complete data describing mothers to characterize the demographics of the households. For Z-tests and regression models, observations below the LOD were assigned a value of $\mathrm{LOD}/\sqrt{2}$.

We used mass balance equations to deduce the likely mechanism of nicotine transfer from smokeless tobacco products to settled dust. As described in the Supplementary Material, we made crude estimates of the mass fraction of nicotine in settled dust attributable to each of two mechanisms—nicotine spilled directly onto the floor as a constituent of smokeless tobacco and nicotine volatilized from the mouth of a smokeless tobacco user with subsequent contamination of the indoor air and settled dust.

RESULTS

Table 1 shows the smoking histories for the six households with a smokeless tobacco user. None of the households with a smokeless tobacco user reported any history of smoking (by the parents or others) in the index residence. Some of the households with a smokeless tobacco user reported a history of parental smoking at a prior residence; however, smoking ceased in these families at least 6 years prior to the first dust collection. Each family occupied the index residence for at least 1 year prior to the initial dust collection and for at least 4 years prior to the second dust collection.

Characteristics of the study households are shown by tobacco-use category in the Supplementary Table S1. A significantly larger proportion of households with a smokeless tobacco user had a lifetime history of parental smoking compared with tobacco-free homes (83% vs. 30%, $p = .02$). Otherwise, there were no statistically significant differences in the characteristics of the households with a smokeless tobacco user compared with tobacco-free homes (i.e., household annual income, residence construction date and type, mother's age, and mother's race/ethnicity were similar in both groups).

Figure 1 shows individual nicotine concentrations in vacuum dust by tobacco-use category, and Table 2 shows corresponding summary statistics. Nicotine was found at concentrations above the LOD in each dust sample collected from a residence with a smokeless tobacco user or an active smoker; nicotine was also found at concentrations above the LOD in 37 of 40 dust samples collected from tobacco-free residences. Median nicotine concentrations for residences with a smokeless tobacco user were significantly greater than median nicotine concentrations for tobacco-free homes during both sampling rounds (Wilcoxon two-sample Z-test, two-sided $p < .01$). Likewise, median nicotine concentrations were significantly greater in residences of active smokers compared with tobacco-free homes during both sampling rounds ($p < .01$). Median nicotine concentrations for residences with a smokeless tobacco user did not differ significantly from median nicotine concentrations for residences of active smokers during either sampling round. The maximum nicotine concentration (370,000 ng/g) was found in a dust

Table 1. Smoking Histories for Households With a Smokeless Tobacco User

Home	Any smoking at current residence	Active parental smoking before occupying current residence: Lifetime history of active parental smoking? Mother	Lifetime history of active parental smoking? Father	Quit date reported? Mother	Quit date reported? Father	Minimum number of years without active smoking prior to round 1[a] Mother	Minimum number of years without active smoking prior to round 1[a] Father	History of passive smoking exposures for household	Years at residence prior to dust collection: Round 1	Years at residence prior to dust collection: Round 2	Nicotine concentration, ng/g: Round 1	Nicotine concentration, ng/g: Round 2
1	Never	No	No	NA	NA	NA	NA	Father exposed to smoking at work and in public	5.7	9.4	29,225	-
2	Never	No	Yes	NA	Yes	NA	20.5	None	7.9	10.8	2,200	3,261
3	Never	Yes	No	No	NA	6.8	NA	None	4.0	7.0	9,176	3,034
4	Never	Yes	Yes	No	No	8.5	8.5	Mother exposed to smoking at work	2.4	8.1	371,844	33,110
5	Never	Yes	Yes	No	No	7.3	7.3	Father exposed to smoking at work and in public	3.4	8.4	14,302	18,001
6	Never	Yes	Yes	Yes	No	6.6	6.8	Father exposed to smoking at work; mother, father, and child exposed to smoking in public	1.0	4.0	39,171	40,628

Note. NA = not applicable.

[a]Each subject without a quit date reported not smoking at each time interval specified during the initial interview, and it is assumed that these subjects stopped smoking prior to the first specified time interval (i.e., at least 3 months prior to the child's conception).

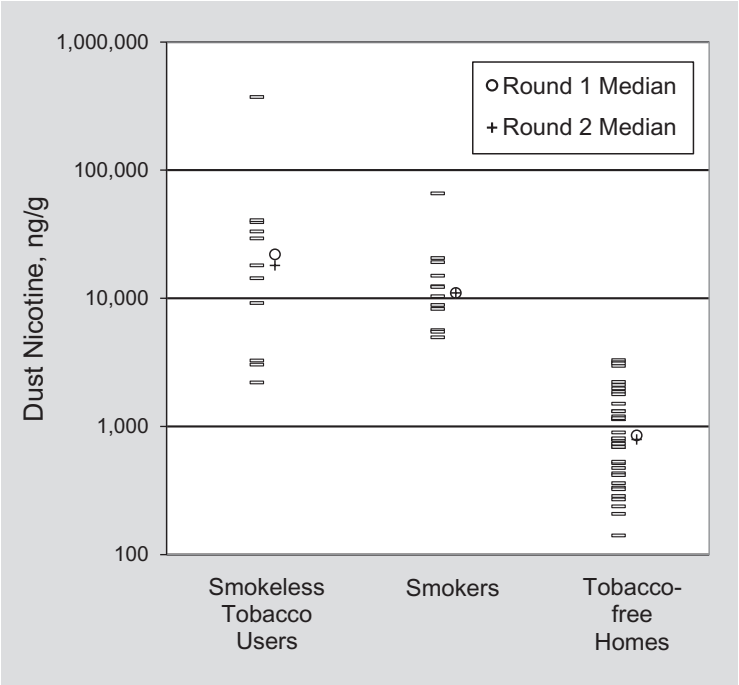

Figure 1. Nicotine concentrations (ng/g) in vacuum dust samples collected from residences in the California Childhood Leukemia Study during the first (2002–2007) and second (2010) sampling rounds, by tobacco-use category (vertical axis is shown on a logarithmic scale).

Table 2. Summary Statistics of Nicotine Concentrations (ng/g) in Vacuum Dust Collected From Residences in the California Childhood Leukemia Study During the First (2002–2007) and Second (2010) Sampling Rounds, by Tobacco-Use Category

Tobacco-use category[a]	*N*	Percent detected	Median	Minimum	Maximum	*SD*
Smokeless tobacco users						
First round	6	100	22,000[b]	2,200	370,000	140,000
Second round	5	100	18,000[c]	3,000	41,000	17,000
Active smokers						
First round	6	100	11,000[b]	5,000	21,000	6,300
Second round	6	100	11,000[c]	5,500	66,000	23,000
Tobacco-free homes						
First round	20	95	850	<LOD[d]	3,300	970
Second round	20	90	790	<LOD[d]	3,100	740

Note. [a]Tobacco use at index residence.
[b]Significantly greater than median nicotine concentration of dust samples collected during first sampling round from tobacco-free homes, using the Wilcoxon two-sample *Z*-test, two-sided $p < .01$.
[c]Significantly greater than median nicotine concentration of dust samples collected during second sampling round from tobacco-free homes, using the Wilcoxon two-sample *Z*-test, two-sided $p < .01$.
[d]Limit of detection (LOD) = 270 ng/g for 100-mg dust sample.

sample collected from the home of a smokeless tobacco user. The maximum nicotine concentrations in residences of active smokers (21,000 and 66,000 ng/g) were found in the household that reported indoor smoking.

Table 2 shows that the median nicotine concentrations for each tobacco-use category varied little between sampling rounds and that the relative order of the median nicotine concentrations between the three groups did not change from the first to second sampling rounds. Differences in nicotine concentrations within households between the first and second sampling rounds were not explained by reported smoking habits, which did not change between rounds. Of the three tobacco-use categories, dust samples from households with a smokeless tobacco user demonstrated the greatest variability in nicotine concentrations.

Table 3 shows results from the regression models of logged nicotine concentrations. In the unadjusted model, nicotine levels were estimated to be 18-fold higher in residences with a

Table 3. Results From Generalized Estimating Equations Derived From the Marginal Models; Proportional Change[a] (95% CI[b]) in Nicotine Concentrations in Vacuum Dust Collected From Residences in the California Childhood Leukemia Study (2002–2010) per Unit Change in Each Covariate

Model coefficients	Unadjusted model	Adjusted model[c]
Smokeless tobacco users versus tobacco-free homes	18 (7.6, 42)*	21 (11, 39)*
Active smoker homes versus tobacco-free homes	14 (9.1, 22)*	16 (9.2, 27)*
Lifetime history of parental smoking versus none		1.17 (0.74, 1.8)
Household annual income ≥$75,000 versus <$75,000		0.62 (0.41, 0.94)*
Residence construction date, per 10-year increment		0.92 (0.86, 0.99)*
Mother's age, per 5-year increment		0.57 (0.46, 0.70)*
Mother is non-Hispanic, White versus Hispanic or Asian		2.3 (1.5, 3.5)*

Note. [a]Proportional change = exp(β_1); where β_1 is the coefficient from the multivariable model of logged nicotine concentrations.
[b]95% confidence intervals (CI) were calculated using robust standard errors.
[c]Adjusted model includes contextual factors that impact nicotine concentrations: history of parental smoking in prior residences, household annual income, residence construction date, mother's age, and mother's race/ethnicity.
*Factor had significant effect on nicotine concentrations in model, $p < .05$.

smokeless tobacco user compared with tobacco-free homes. Likewise, in the unadjusted model, nicotine levels were estimated to be 14-fold higher in homes of active smokers compared with tobacco-free homes. After adjustment for a history of parental smoking at prior residences, household annual income, residence construction date, and mother's age and race/ethnicity, nicotine levels from homes with smokeless tobacco users and active smokers remained elevated in comparison with nicotine levels found in tobacco-free homes (21-fold and 16-fold higher, respectively).

Households with non-Hispanic, white mothers had significantly higher nicotine concentrations in vacuum dust than households with Hispanic or Asian mothers (2.3-fold higher). Nicotine concentrations were significantly lower in vacuum dust collected from more affluent households (38% lower for households with annual income ≥$75,000 vs. <$75,000), from more recently constructed residences (8% decrease per 10-year increment in residence construction date), and from residences with older mothers (43% decrease per 5-year increment in mother's age). Tobacco-free homes and homes with a smokeless tobacco user that reported a history of parental smoking at previous homes did not have significantly higher nicotine concentrations in vacuum dust than lifetime nonsmokers.

DISCUSSION

We found that nicotine concentrations in vacuum dust collected from homes of smokeless tobacco users were significantly higher than levels from tobacco-free homes and comparable with levels in homes of active smokers. To our knowledge, this analysis represents the first attempt to characterize nicotine levels in dust from the homes of smokeless tobacco users. Importantly, our findings raise new concerns about the safety of smokeless tobacco use in homes with young children.

The range of nicotine concentrations in vacuum dust collected from tobacco-free homes in our study (range: <270 to 3,300 ng/g) was similar to the range of nicotine concentrations in floor dust collected with a high-volume surface sampler from the living rooms of other California homes of nonsmokers (95% *CI*: 1,100–4,000 ng/g; Matt et al., 2011). Nicotine concentrations in vacuum dust collected from homes of active smokers in our study (median of 11,000 ng/g for both sampling rounds) were somewhat lower than recently reported nicotine concentrations in floor dust collected using a high-volume small surface sampler from living rooms of other California homes of active smokers (*M*; 95% CI: 40,000; 30,000–52,000 ng/g; Matt et al., 2011). However, in our analysis, most of the households that were classified as homes of active smokers reported exclusively smoking outside of the home (i.e., only one of six households of active smokers reported indoor smoking during the year prior to the second dust collection), whereas each household of an active smoker in the study by Matt et al. (2011) reported smoking inside the home. Not surprisingly, the one household that reported smoking indoors in our study had higher nicotine concentrations (i.e., 20,000 and 66,000 ng/g) than the five households that reported exclusively smoking outside of the home. Nicotine concentrations in vacuum dust collected from the homes of smokeless tobacco users in our study (medians of 22,000 and 18,000 ng/g for sampling rounds 1 and 2, respectively) were slightly lower than levels in homes of active smokers and much higher than the levels in homes of nonsmokers from the study by Matt et al. (2011).

Nicotine can be tracked inside a home on a smoker's contaminated clothes, shoes, or skin, or it can enter a home when tobacco smoke infiltrates doors or windows (Matt et al., 2004). It follows that we were able to detect nicotine in nearly every dust sample, including 37 of 40 samples from homes without tobacco users. In a previous analysis of the CCLS population, we reported that contextual factors, such as past smoking, household annual income, residence construction date, parental age, and (to a lesser extent) mother's race/ethnicity, were determinants of background nicotine contamination in settled dust (Whitehead et al., 2009). In this present analysis, we confirmed that some of these factors were related to nicotine concentrations using a multivariable model, but adjusting for these covariates did not explain the observed difference in nicotine levels between homes of smokeless tobacco users and tobacco-free homes.

There are at least two ways that smokeless tobacco could be released into the residential environment (e.g., the carpet): (a) a user might spill smokeless tobacco directly onto the floor or (b) nicotine might volatilize to the indoor air from the mouth of a smokeless tobacco user and subsequently contaminate the settled dust on the floor. In the former case, the mass of nicotine

spilled on the floor depends on the amount of tobacco that is consumed in the home (a readily estimated quantity) and the proportion of consumed tobacco that is accidentally released (an unknown and difficult to estimate quantity). Using mass balance equations (see Supplementary Material for details), we estimate that the concentration of nicotine in carpet dust associated with spilled tobacco could be on the order of ~500 to 5,000,000 ng/g. In contrast, we estimate that the nicotine concentration in carpet dust associated with the volatilization of nicotine to indoor air from the moist snuff held in a user's mouth and subsequent deposition of nicotine from indoor air to settled dust could be expected to be lower: on the order of ~1 to 5,000 ng/g. Based on these estimates, we suggest that tobacco spillage was probably the predominant mechanism for nicotine transfer to settled dust in most of the smokeless tobacco-user homes in our analysis and that nicotine volatilization and deposition likely played a minor role. Thus, we expect carcinogenic constituents of smokeless tobacco, such as the nonvolatile tobacco-specific *N*-nitrosamines, NNN, and NNK, may also be present at high levels in dust from residences of smokeless tobacco users.

Because young children spend more time in the home environment, spend more time near the floor, and are more likely to make hand-to-mouth contact than their adult counterparts (Cohen Hubal et al., 2000), elevated nicotine concentrations in settled dust are of particular concern in households with young children. Because semivolatile tobacco constituents in settled dust persist in the indoor environment (Matt et al., 2011) and are readily transported throughout the indoor environment (Hoh et al., 2012), children are likely exposed to these compounds at times when, and in rooms where, tobacco is not used (e.g., in a child's bedroom). Moreover, as semivolatile organic compounds (e.g., nicotine) partition readily between settled dust and other surfaces (Weschler & Nazaroff, 2012), exposures to tobacco constituents via dermal contact with household surfaces may also be a concern for children in homes with elevated levels of nicotine in dust.

The health risks for a child exposed to residential tobacco contamination without coincident exposure to tobacco smoke are not known. Matt et al. (2011) reported that children living in apartments of nonsmokers with elevated dust-nicotine concentrations (10,900 and 11,000 ng/g for living rooms and bedrooms, respectively) had significantly higher urinary cotinine levels than children living in apartments of nonsmokers with lower dust-nicotine concentrations. Likewise, da Silva et al. (2012) reported that nonsmoking farmers exposed to high levels of tobacco constituents via dermal contact with tobacco leaves had elevated cotinine levels and increased markers of DNA damage such as micronuclei, nuclear buds, and binucleated cells using the buccal micronucleus cytome assay. Sleiman et al. (2010) showed that residual nicotine sorbed to indoor surfaces can react with ambient nitrous acid to form carcinogenic tobacco-specific nitrosamines (e.g., NNK), which suggests that children living with smokeless tobacco users may be exposed to these carcinogens. The potential adverse health impacts of exposure to tobacco constituents via dust and household surfaces for young children living with smokeless tobacco users warrant further investigation.

There is interest in using the concentration of nicotine in settled dust as an unbiased measure of a child's exposure to tobacco smoke in epidemiological studies. Because investigators have shown that nicotine concentrations in dust are correlated with cigarette consumption reported by parents (Whitehead et al., 2009) and with children's urinary cotinine levels (Willers, Hein, & Jansson, 2004), dust nicotine may be a suitable indicator of household smoking. However, our results indicate that both smoking and the use of smokeless tobacco products can result in elevated nicotine concentrations in vacuum dust. Therefore, epidemiologists who seek to estimate children's exposure to tobacco smoke using dust-nicotine levels may misclassify children living in homes with smokeless tobacco users.

The principal limitation of our analysis is our ability to characterize nicotine concentrations in a small number of homes with a smokeless tobacco user ($N = 6$). Moreover, while we were able to confirm our findings using two independent dust samples from each household, we only identified residences with a smokeless tobacco user at the time of the second dust collection, and we assumed, but did not verify, that smokeless tobacco was also used in these homes at the time of the first dust collection. Because our analysis included only a small number of households, it will be important to replicate our findings in other populations.

Because of the limited number of households studied, we did not attempt to measure the impact that exposure to residential tobacco contamination in the absence of coincident exposure to tobacco smoke could have on children's health. Additionally, we assumed that nicotine would be an appropriate surrogate for the presence of other tobacco constituents, but we did not measure potentially carcinogenic species, such as NNN or NNK, in the dust samples and do not know how strongly correlated nicotine is with NNN and NNK in these dust samples. Moreover, we assume that nicotine concentrations measured in dust collected from household vacuum cleaners are representative of children's exposure to tobacco constituents; however, vacuum samples may be misleading in homes where residents have used a vacuum to clean an accidental smokeless tobacco spill. We did not ask study participants to describe their vacuum cleaner usage with sufficient detail to evaluate this possibility, and we did not use indicators of children's biological intake of tobacco constituents, such as urinary cotinine, to validate our findings. Future studies should measure carcinogenic constituents of tobacco in environmental samples from additional homes occupied by smokeless tobacco users, characterize exposure to tobacco constituents for children living in these homes using biological samples, and identify whether the health of these children is affected by exposure to tobacco constituents.

Using dust samples collected from homes participating in the California Childhood Leukemia Study, we demonstrated that nicotine concentrations in dust from residences occupied exclusively by smokeless tobacco users were elevated above background nicotine levels found in tobacco-free residences and comparable with levels in homes occupied by active smokers. We hypothesize that nicotine is transferred to floor surfaces in homes of smokeless tobacco users primarily as a constituent of tobacco that is spilled during application or expectoration. We anticipate that other constituents of tobacco may also be present at elevated levels in dust from homes of smokeless tobacco users, and we expect that young children will be exposed to these hazardous chemicals via ingestion of contaminated dust and dermal contact with contaminated dust and household surfaces. The potential health impact that results from a child's exposure to residential smokeless tobacco contamination warrants further investigation.

SUPPLEMENTARY MATERIAL

Supplementary Material and Table S1 can be found online at http://www.ntr.oxfordjournals.org

FUNDING

This work was supported by the National Institute of Environmental Health Sciences (R01ES009137, R01ES015899, and P42ES0470518). The NIEHS had no role in the study design; in the collection, analysis, or interpretation of data; in the writing of the manuscript; or in the decision to submit the paper for publication.

DISCLAIMER

The ideas and opinions expressed herein are those of the authors and do not necessarily reflect the official position of the California Department of Toxic Substances Control or those of the National Institute of Environmental Health Sciences.

DECLARATION OF INTERESTS

None declared.

ACKNOWLEDGMENTS

We thank Dr. Peyton Jacob, Mr. Christopher Havel, and Ms. Lisa Yu (University of California, San Francisco) for their advice in conducting nicotine analyses. We thank the families for their participation. We also thank the clinical investigators at the following collaborating hospitals for help in recruiting patients: University of California Davis Medical Center (Dr. Jonathan Ducore), University of California San Francisco (Drs. Mignon Loh and Katherine Matthay), Children's Hospital of Central California (Dr. Vonda Crouse), Lucile Packard Children's Hospital (Dr. Gary Dahl), Children's Hospital Oakland (Dr. James Feusner), Kaiser Permanente Oakland (Drs. Daniel Kronish and Stacy Month), Kaiser Permanente Roseville (Drs. Kent Jolly and Vincent Kiley), Kaiser Permanente Santa Clara (Drs. Carolyn Russo, Denah Taggart, and Alan Wong), and Kaiser Permanente San Francisco (Dr. Kenneth Leung). Finally, we acknowledge the entire California Childhood Leukemia Study staff for their effort and dedication.

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