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Measurement of nicotine in household dust

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ABSTRACT

An analytical method of measuring nicotine in house dust was optimized and associations among three secondhand smoking exposure markers were evaluated, i.e., nicotine concentrations of both house dust and indoor air, and the self-reported number of cigarettes smoked daily in a household. We obtained seven house dust samples from self-reported nonsmoking homes and 30 samples from smoking homes along with the information on indoor air nicotine concentrations and the number of cigarettes smoked daily from an asthma cohort study conducted by the Johns Hopkins Center for Childhood Asthma in the Urban Environment. House dust nicotine was analyzed by isotope dilution gas chromatography–mass spectrometry (GC/MS). Using our optimized method, the median concentration of nicotine in the dust of self-reported nonsmoking homes was 11.7 ng/mg while that of smoking homes was 43.4 ng/mg. We found a substantially positive association ($r = 0.67$, $P < 0.0001$) between house dust nicotine concentrations and the numbers of cigarettes smoked daily. Optimized analytical methods showed a feasibility to detect nicotine in house dust. Our results indicated that the measurement of nicotine in house dust can be used potentially as a marker of longer term SHS exposure.

1. Introduction

Secondhand smoke (SHS) exposure is assessed by a number of methods, including responses to questionnaires (Gaffney et al., 2003; Klepeis, 1999) and biomonitoring of nicotine or cotinine—one of major metabolites of nicotine—in bio-fluids such as serum, urine or saliva (Pirkle et al., 1996; Willers et al., 1995; Benowitz, 1999). While a questionnaire is often feasible to administer, self-reported exposure status can be limited by inaccurate or biased recall (Persson and Norell, 1989; Wagenknecht et al., 1992). Air nicotine monitoring is also widely used as a SHS exposure assessment tool (Hammond and Leaderer, 1987; Leaderer, 1990; Navas-Acien et al., 2004; Wipfli et al., 2008). However, the estimate of exposure of air nicotine is limited to the duration of the sampling period (Hammond and Leaderer, 1987; Rothberg et al., 1998). Despite high sensitivity and specificity of biomarkers, their reliability as markers of smoke exposure has been questioned due to their short elimination times in body fluids (1–2 days) (Jaakkola and Jaakkola, 1997). To reflect a longer exposure integration period, recent studies have used nicotine concentration in hair or toenails (Wipfli et al., 2008; Al-Delaimy et al., 2002a, b; Al-Delaimy, 2002). However, as some study participants are reluctant to provide hair or toenail samples for cultural or religious reasons, an alternative marker for longer term SHS exposure is needed. An objective environmental measurement that integrates exposure over long period of time would be an additional important tool for SHS exposure assessment.

Nicotine has been used as a marker for vapor-phase constituents of SHS (US EPA, 1992). It can attach to dust particles and surfaces in locations of active smoking, therefore making it possible to detect nicotine in settled house dust or on indoor surfaces of a household where smoking has occurred (Matt et al., 2004). Willers et al. (1995) reported that the nonsmoker could likely inhale nicotine attached to house dust, as well as in its vapor-phase form. In addition, a storage stability test of nicotine in house dust showed that the recovery rate of nicotine after 21 days of storage was still approximately 40% (Chuang et al., 1993). Thus, house dust nicotine possesses the potential to provide longer term SHS exposure information for an indoor environment. House dust also provides a potentially under-recognized exposure route of nicotine and other components of cigarette smoke for children (Matt et al., 2008). It is well recognized that children's hand-to-mouth activity can result in exposure to agents contained in house dust (Matt et al., 2008). According to Pirkle et al. (2006), about

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60% of US children aged from 3 to 11 years, who participated in the National Health and Nutrition Examination Survey (NHANES) 1999–2002, were exposed to SHS at home; this was determined by serum cotinine level (>0.05 ng/ml).

Despite the likelihood that nicotine can be detected in house dust and the potential of house dust nicotine as a longer term SHS exposure marker, only a few studies have evaluated the concentration of nicotine in house dust. One study found a significant correlation ($r=0.65$) between the number of cigarettes smoked daily in a household and concentrations of nicotine in the house dust (Hein et al., 1991). In addition, Willers et al. (2004) reported a strong correlation ($r=0.77$) between urinary cotinine concentrations and nicotine concentrations in the vacuum cleaned house dust samples. However, none of the previous studies evaluated the association among the three markers of tobacco exposure (airborne nicotine, numbers of cigarettes, and dust nicotine). Also the potential for house dust nicotine concentrations to serve as a longer term SHS exposure marker has not been explored in studies looking at health outcomes. In addition, the analytical procedures contained in these earlier publications were not thoroughly described.

The purpose of this paper is to describe an isotope dilution gas chromatography–mass spectrometry (GC/MS) analytical method for detecting nicotine in house dust. Using our optimized method, we measured house dust nicotine levels in Baltimore homes. We also evaluated associations among self-reported numbers of cigarette smoked daily, indoor airborne nicotine concentrations, and dust nicotine concentrations.

2. Materials and methods

2.1. Dust nicotine analytical method optimization

We developed and optimized a method for the analysis of nicotine in house dust using an isotope dilution GC/MS based on methods developed for hair nicotine analysis (Kintz, 1992). Prior to extraction, we sieved house dust with a methanol-washed 150 μm mesh sieve to remove hair and other large pieces of debris from the dust samples. Since the minimum mass of dust needed to provide a sufficient mass of nicotine to be analyzed is not known, we evaluated the impact of dust mass on analytical sensitivity using two house dust samples collected from two households: one with a low and one with a high self-reported SHS exposure level. The dust masses tested ranged from 20 to 200 mg.

Nicotine was extracted from house dust using 3.5 ml of diethyl ether, 1.5 ml of 1 M NaOH, and 70 μl of 1.0 μg/ml internal standard (Nicotinine-d3, Sigma) which were added to each sample in a centrifuge tube. Sample tubes were then placed in a horizontal shaker. To identify the optimum extraction time, the effect of extraction shaking time on nicotine extraction efficiency was evaluated. We prepared four spiked samples of house dust at a concentration of 2 ng/mg for each time point (10, 20, 30, 60, 90, and 120 min).

Following extraction, we centrifuged the samples at $2000\times g$ for 10 min; next, the organic phase was transferred to a clean evaporation tube. To prevent volatilization of nicotine, 17.5 μl of octanol was added to the tube. After repeating the extraction steps, we evaporated the combined organic phase in the tubes in a water bath at 50 °C and dissolved the remaining nicotine in 52.5 μl of methanol. The final solution was then transferred to GC autosampler vials.

Nicotine analysis was conducted using an isotope dilution GC/MS (GC-17/MS-QP5000, Shimadzu) in a selected ion monitoring (SIM) and splitless mode. Nicotine was separated by a capillary column (30 m × 0.25 mm i.d., 0.25 μm film thickness; Rtx-624, Restek, Bellefonte, PA). The GC oven temperature was maintained at 70 °C for 1 minute and then ramped up to 280 °C at a rate of 25 °C/min; it was then maintained at that level for 1 min. The temperatures of injection port and GC interface was 250 and 280 °C and ionizing voltage was 70 eV. In the SIM mode, we used m/z (162), 133 for nicotine and m/z (87), 165, 136 for nicotine-d3 detection (quantitative ions are indicated in parenthesis).

Calibration standards were prepared by diluting stock solution of nicotine (Sigma) in the mixture of methanol and octanol (3/1, v/v). The amount of internal standard spiked in calibration standards and dust samples was 70 ng. We evaluated the linearity of the calibration line using a simple linear regression.

We conducted a bias and precision test of the nicotine measurement using known nonsmoking house dust samples spiked with nicotine (0.4 and 2 ng/mg, $n=5$ per each level). Bias was evaluated by calculating recovery (%), while precision was determined as a relative standard deviation (%) from the five replicate analyses. We calculated a limit of detection (LOD) by multiplying a standard deviation obtained from eight method blank samples (containing the lowest level of nicotine calibration standard in clean nonsmoking house dust samples then extracted using methods described as above) and the Student's t-value appropriate for a 99% confidence level with $n-1$ degrees of freedom. Approximately, 50% of the dust samples from our sample batch underwent duplicate analyses as an ongoing evaluation of reproducibility.

2.2. House dust analysis

We randomly selected house dust samples ($n=30$) from a subset of smoking homes enrolled in the Baltimore Indoor Environment Study of Asthma in Kids (BIESAK), a cohort study of childhood asthma conducted by the Johns Hopkins Center for Childhood Asthma in the Urban Environment (CCAUE). As a part of the BIESAK study, settled dust samples were collected for allergen analysis; airborne nicotine was measured, and participants reported the number of cigarettes smoked in their homes. More detailed methods used in this study have been previously reported (Diette et al., 2007). Using a handheld Redi-Vac vacuum cleaner containing vacuum filter cones, a composite dust sample was collected from the asthmatic child's bed, bedding and the floor close to the bed (Wood et al., 2001). Large debris was removed from the samples using a 150 μm sieve. Archived aliquots of dust samples were used in this analysis. Using the same procedure, we collected seven dust samples from self-reported nonsmoking homes. At the same time, airborne nicotine sampling was conducted over a 72-h period in the bedrooms using passive samplers. Analysis was conducted using GC equipped with a nitrogen–phosphorus detector (GC-NPD) (Hammond and Leaderer, 1987; Navas-Acien et al., 2004).

We classified the smoking status of each household using self-reported smoking behavior included in the baseline questionnaire; participants were asked for the total number of cigarettes smoked per day by household members. Those who reported smoking no cigarettes were identified as living in "nonsmoking homes" and those who reported smoking one or more cigarettes were labeled as living in "smoking homes".

2.3. Statistical analysis

We performed statistical analyses using SAS (version 9.1; SAS Institute Inc., Cary, NC). The extraction efficiency at different time points was compared using the Wilcoxon rank-sum test. Using the Spearman correlation and a simple regression analyses, we evaluated associations among house dust nicotine concentrations, air nicotine concentrations, and the number of cigarettes smoked daily. After assessing the results of the Shapiro–Wilks normality test for distribution of house dust nicotine concentration, we conducted with a simple regression analysis with a log-transformation of house dust nicotine concentrations as a dependent variable.

3. Results and discussion

3.1. Optimization of analytical method

Our analysis of the nicotine extraction efficiency over time (Fig. 1) showed that nicotine concentrations peaked at an extraction duration of 30 min. Nicotine concentration at 30 min did not differ significantly from measures made at 20, 60, 90, and 120 min. However, we chose 30 min as the optimal time point, since median recovery was highest at that time.

The calibration curve showed a strong relationship between instrument response and known amount of nicotine calibration standard samples. Coefficients of determination (R^2) were 0.99 or higher, and a limit of detection was 0.02 ng/mg. Recoveries from house dust samples at two different concentrations (i.e., 0.4 and 2 ng/mg, $n=5$ per level) were 110.6% and 94.7% with less than 20% variation. This optimized method showed a feasibility for measuring nicotine in household dust.

Fig. 2 summarizes the effect of the mass of house dust extracted on the mass of nicotine detected. A strong linearity between the amount of house dust used for extraction and the amount of nicotine detected in the dust samples was obtained from two-parallel experiments: coefficients of determination (R^2) were 0.91 or higher with the mass range from 10 to 200 mg. Based on these results, we chose 50 mg as the target mass of house dust to be used to assess nicotine content since our method could

provide sufficient sensitivity for this small amount of dust even when little smoking occurs in the household. Fifty milligrams also represent a mass of dust that can be readily collected from the surface of any floor and can be easily handled and weighed.

3.2. Concentrations of nicotine in house dust from Baltimore households

Using the optimized analytical conditions described above, we analyzed 37 Baltimore house dust samples. Nicotine was detected in all dust samples obtained from the bedrooms of the children. The median nicotine concentration from the samples was 37 ng/mg, with a range of 4.6–300 ng/mg. The slope between the duplicate and primary analysis was 1.06 ($P<0.0001$) with a determination coefficient of 0.95 ($n=19$), indicating that our optimized method has a high level of analytical precision. The median concentration of nicotine in the dust of self-reported nonsmoking homes ($n=7$) was 11.7 ng/mg, while that of smoking homes ($n=30$) was approximately four times (43.4 ng/mg) higher ($P<0.001$). The range of house dust nicotine concentrations from self-reported nonsmoking homes was 4.6–26.5 ng/mg, while that from self-reported smoking homes was 11.5–300 ng/mg.

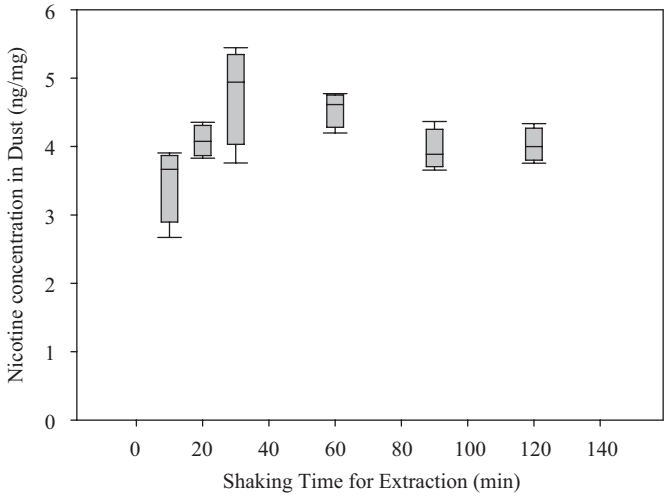

Fig. 1. Distribution of dust nicotine concentrations by extraction shaking time. Boxes represent median (50th) and inter quartile values (25th/75th) per each time. Whiskers represent median and 5th/95th percentile values.

Despite the potential public health relevance, there have been only a few previous reports of nicotine concentration in house dust samples. Hein et al. and Wellers et al. reported concentrations for 72 and 23 houses in Denmark (Hein et al., 1991) and Sweden (Willers et al., 2004), respectively. Matt et al. (2004) measured nicotine in house dust samples collected in San Diego County, CA, USA. Our median nicotine concentration from nonsmoking homes was 1.5 and 2.6 times lower than that of nonsmoking homes in two European countries reported by Hein et al. (18 ng/mg, $n=38$) and Willer et al. (31 ng/mg, $n=8$) while that of our smoking homes was six and three times lower than what Hein et al. (242 ng/mg, $n=34$) and Willer et al. (121 ng/mg, $n=15$) reported for smoking homes, respectively. We found about 17 and 8 times as much nicotine in our Baltimore house dust, compared to house dust collected in San Diego County nonsmoking homes (0.7 ng/mg, $n=56$) and smoking homes (5.4 ng/mg, $n=70$), respectively. Because the population characteristics and the dust sample collection protocols varied among the studies, it is difficult to ascribe too much meaning to these observed differences.

3.3. Association among three SHS exposure markers

To evaluate associations among three secondhand smoking exposure markers—self-reported number of cigarettes smoked per day, house dust nicotine concentrations, and indoor air nicotine concentrations—we first examined correlation coefficients among the three markers. According to the Spearman correlation analysis, house dust nicotine concentrations were positively associated ($r=0.67$, $P<0.001$) with the number of cigarettes smoked. An evaluation of the change of house dust nicotine concentrations associated with each cigarette smoked was also conducted using a simple regression analysis with log-transformed house dust nicotine concentration as a dependent

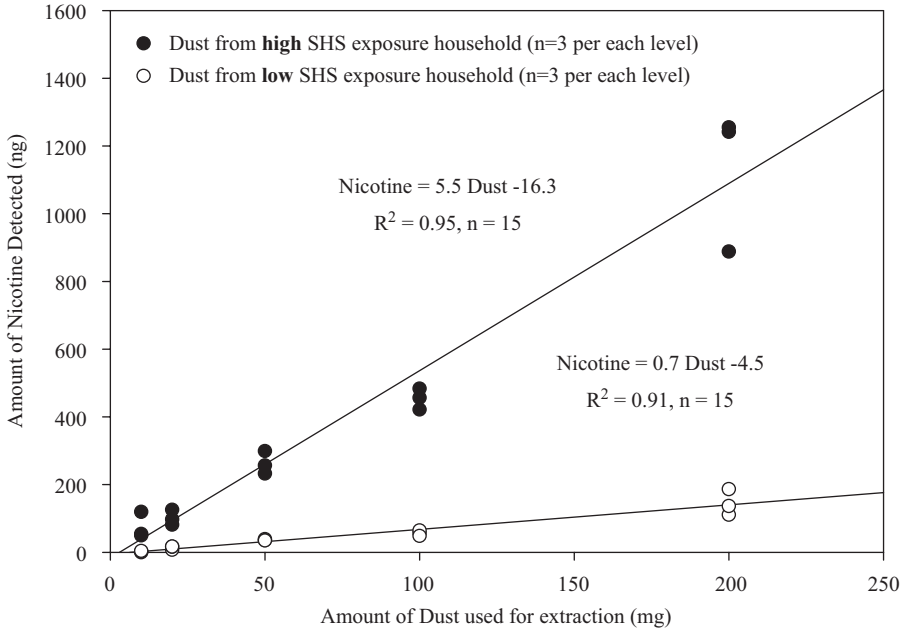

Fig. 2. Relationship between the mass of dust analyzed and amount of nicotine detected.

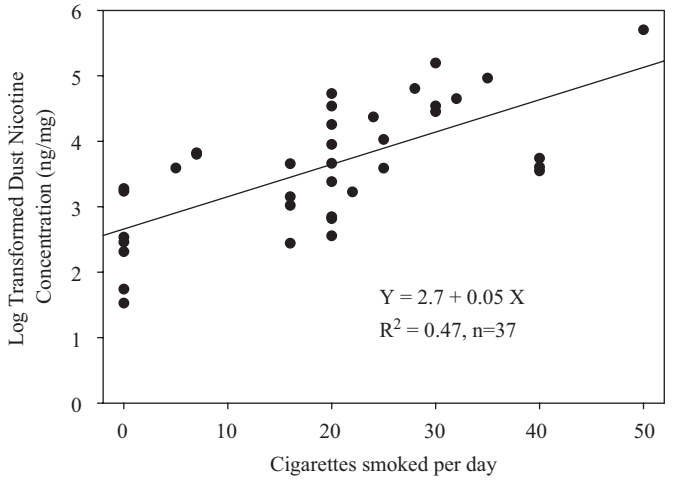

Fig. 3. Relationship between house dust concentrations and self-reported number of cigarettes smoked per day in a household.

variable. A unit increase in the number of cigarettes smoked per day in a household was associated with an estimated increase of 5% (95% CI = 3.1–6.7) house dust nicotine concentrations in our study (Fig. 3). In contrast to this strong positive association, there was no significant association between the number of cigarettes smoked and indoor air nicotine concentrations ($P = 0.38$). Also no significant association was evident between indoor air nicotine concentrations and dust nicotine concentrations ($P = 0.24$).

One explanation for these results is that airborne nicotine results represent a "snap-shot" in time and may not capture typical smoking behaviors. Air nicotine samples that were collected at one 72-h time point might not represent a "typical" smoking activity pattern. Other factors may explain the lack of correlation between house dust nicotine and air nicotine concentrations or between air nicotine and the number of cigarettes smoked. Differing human activity patterns, such as degree of ventilation (open doors and windows) or vacuuming, can affect the strengths of the associations among air nicotine concentration, dust nicotine, and self-reported number of cigarettes smoked per day. Missing information about the number of cigarettes smoked per day indoors versus outdoors, rather than overall number of cigarettes smoked, might also contribute to the differences of associations. Finally, a potential time gap between questionnaire administration and air sample collection might also be a factor in reducing the degree of association.

In this paper, we presented a validated method for assessing nicotine in house dust. This method was optimized in terms of the mass of house dust needed for analysis, and the extraction time using an isotope dilution method. A thoroughly described analytical method has not been previously published. Compared with earlier studies (Hein et al., 1991; Willers et al., 2004) except for Matt et al., 2004, another advantage of our study is that we used a standardized dust sample collection procedure. The bedroom samples were collected with established methods (Wood et al., 2001); vacuuming a 1 m^2 area near and underneath a child's bed for 2 min combined with another 2 min from the mattress and bedding. Then we screened out dust larger than 150 μm using a sieve in the laboratory. Only a few (e.g., Matt et al., 2004) of the earlier studies used size segregated dust samples for dust nicotine measurement. Our limit of detection (0.02 ng/mg) was comparable to that of CDC method (0.03 ng/mg) mentioned in Matt et al. (2004).

In addition, this study showed that SHS can contaminate not only indoor air but also dust in asthmatic children's bedrooms, indicating that children may be at risk of exposure to the toxic components of cigarette smoke through multiple pathways: inhalation of contaminated air, inhalation and ingestion of contaminated bedroom dust, and dermal contact with the dust (Matt et al., 2004, 2008). Given the strong correlation between dust nicotine and reported smoking behavior, and the lack of correlation with airborne nicotine concentrations, these results suggest that nicotine concentration in house dust may be a suitable longer term SHS exposure marker.

Thus, dust nicotine may be a better marker of "typical" smoking behaviors than a short-term (3-day snapshot in this case) air sample. Finding a longer term marker is particularly critical in studies of SHS exposure because health outcomes of SHS are often latent (Eisner et al., 2005; CDC, 2007). Results of a recent meta-analysis strongly support that children's exposure to SHS can be a risk factor for childhood asthma (Vork et al., 2007). Therefore, measurement of nicotine in house dust, in combination with other evaluation methods including questionnaire, biomarkers, and air nicotine monitoring, could contribute to the prevention of childhood asthma by more accurately evaluating the smoking status of the homes of asthmatic children. These approaches should provide more reliable data for epidemiologists and policy makers.

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