Published in final edited form as:

Tob Control. 2016 September ; 26(5): 548–556. doi:10.1136/tobaccocontrol-2016-053119.

When smokers quit: exposure to nicotine and carcinogens persists from thirdhand smoke pollution

Georg E Matt¹, Penelope J E Quintana², Joy M Zakarian³, Eunha Hoh², Melbourne F Hovell², Melinda Mahabee-Gittens⁴, Kayo Watanabe², Kathy Datuin², Cher Vue², and Dale A Chatfield⁵

¹Department of Psychology, San Diego State University, San Diego, California, USA

²San Diego State University Graduate School of Public Health, San Diego, California, USA

³San Diego State University Research Foundation, San Diego, California, USA

⁴Cincinnati Children's Hospital Medical Center, Cincinnati, Ohio, USA

⁵Department of Chemistry, San Diego State University, San Diego, California, USA

Abstract

Background—Over a 6-month period, we examined tobacco smoke pollutants (also known as thirdhand smoke, THS) that remained in the homes of former smokers and the exposure to these pollutants.

Methods—90 smokers completed study measures at baseline (BL). Measures were repeated among verified quitters 1 week (W1), 1 month (M1), 3 months (M3) and 6 months (M6) following cessation. Measures were analysed for THS pollutants on household surfaces, fingers and in dust (ie, nicotine, tobacco-specific nitrosamines) and for urinary markers of exposure (ie, cotinine, 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol (NNAL)).

Results—We observed significant short-term reduction of nicotine on surfaces (BL: 22.2 $\mu\text{g}/\text{m}^2$, W1: 10.8 $\mu\text{g}/\text{m}^2$) and on fingers of non-smoking residents (BL: 29.1 ng/wipe, W1: 9.1 ng/wipe) without further significant changes. Concentrations of nicotine and nicotine-derived nitrosamine ketone (NNK) in dust did not change and remained near BL levels after cessation. Dust nicotine and NNK loadings significantly increased immediately following cessation (nicotine BL: 5.0 $\mu\text{g}/\text{m}^2$, W1: 9.3 $\mu\text{g}/\text{m}^2$; NNK BL: 11.6 ng/m², W1: 36.3 ng/m²) before returning to and remaining

Correspondence to Dr Georg E Matt, Department of Psychology, San Diego State University, San Diego, CA 92182-4611, USA; gmatt@mail.sdsu.edu.

Contributors GEM and PJEQ conceived of and designed the study. PJEQ, EH and JMZ designed field sampling protocols. JMZ and GEM designed the personal interviews. JMZ managed data collection, data entry, cleaning and archiving. EH and DAC designed and supervised laboratory analyses conducted by KW, KD and CV. GEM developed the data analysis plan and conducted the data analyses. MM-G provided input on analysis and interpretation of data. MFH provided feedback on the design of the study and the collection and interpretation of data. All authors contributed to the editing of the manuscript drafts, and GEM prepared the final manuscript. GEM and PJEQ are responsible for the overall content as guarantors.

Collaborators Dr Mark G Myers.

Competing interests None declared.

Ethics approval This study was conducted with the approval of the San Diego State University Institutional Review Board and the Veterans Administration San Diego Healthcare System.

Provenance and peer review Not commissioned; externally peer reviewed.

at near BL levels. Cotinine and NNAL showed significant initial declines (cotinine BL: 4.6 ng/mL, W1: 1.3 ng/mL; NNAL BL: 10.0 pg/mL, W1: 4.2 pg/mL) without further significant changes.

Conclusions—Homes of smokers remained polluted with THS for up to 6 months after cessation. Residents continued to be exposed to THS toxicants that accumulated in settled house dust and on surfaces before smoking cessation. Further research is needed to better understand the consequences of continued THS exposure after cessation and the efforts necessary to remove THS.

INTRODUCTION

Tobacco smoke is a complex mixture of more than 7000 chemical compounds¹ that are released from the smoldering tip of a cigarette between puffs (ie, sidestream smoke) and through the butt of a cigarette during inhalation (ie, mainstream smoke). When the exhaled mainstream smoke mixes with the sidestream smoke, secondhand smoke (SHS) is created. SHS is a combination of particulate and gas-phase chemical compounds that includes 70 known human carcinogens (eg, 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (nicotine-derived nitrosamine ketone, NNK), benzo [a]pyrene)² and has been classified as a toxic air contaminant by the California Air Resources Board.³

Immediately after its creation, SHS begins a process of changes, also known as chemical and physical ageing. SHS is diluted through mixing with the ambient air, and as it spreads to adjacent rooms, hallways and floors of a building, it interacts with the physical environment. SHS compounds absorb to surfaces, settle and accumulate in dust and react with other compounds they encounter. Compared to freshly emitted tobacco smoke, this aged mixture of SHS constituents has changed properties and composition to such an extent that researchers have coined the term thirdhand smoke (THS).⁴ In a demonstration of this process, Sleiman *et al*⁵ monitored fine particulate matter (PM_{2.5}) concentrations and 58 volatile organic compounds (VOCs) in a controlled chamber study for 18 hours after generating SHS. They found that PM_{2.5} concentrations declined by 65% and 98% 2 and 18 hours later, respectively. All volatile and semivolatile amines (including nicotine) except for 3-ethenyl pyridine could no longer be detected in the ambient air after 2 hours, indicating massive adsorption to surfaces consistent with the build-up of a THS reservoir. Significant adsorption was also found for aromatic hydrocarbons (eg, naphthalene, xylene, styrene), chlorinated VOCs and carbonyls (including acrolein, methacrolein).

The small size and mobility of particle and gas-phase compounds allow SHS and THS to penetrate deep into carpet padding and upholstery; infiltrate closed cabinets, closets and drawers and coat every available microsurface of an indoor environment. Among the materials with the most receptive surfaces for THS are many commonly found in indoor environments: carpet fibres, carpet padding, upholstery, mattresses, pillows, blankets, cabinets, doors, clothes, wallpaper, building materials (gypsum board, wood), ceiling tiles, etc.⁶

If SHS is regularly generated over periods of months or years (eg, home or car of daily smokers, a casino, a smoking-permitted hotel room), a significant mass of THS builds up in dust deposits, on surfaces and in objects and materials, creating a large reservoir of THS pollutants.⁷ For instance, smoking 10 high-nicotine cigarettes per day (25 mg/cigarette) over

a 10-year period will release 912 g of nicotine in the home environment, the majority of which will adsorb to indoor surfaces. For reference, the lethal nicotine dose in humans has been estimated to be 6.5–13 mg/kg (oral LD₅₀).⁸

The physical and chemical differences between SHS and THS have a profound impact on human exposure opportunities and pathways. SHS exposure is limited to inhalation of compounds while tobacco is being smoked and, depending on ventilation conditions, may last for up to 2 to 3 hours after a cigarette has been smoked.⁹ In contrast, THS exposure continues after SHS ends and, depending on the depth of the reservoirs, may last for months after the last cigarette has been extinguished. Since THS accumulates on surfaces, in objects and in dust, THS exposure may take place through ingestion, through dermal transfer via skin contact with polluted objects (eg, clothes) and through inhalation of suspended house dust. Hand and skin wipes have been used in studies of other pollutants, such as pesticides, to measure the potential for dermal exposure.¹⁰¹¹ Nicotine on the hands of non-smokers can serve as a proxy of the pollution of surfaces in an individual's immediate environment as well as a proximate and immediate cause of exposure to nicotine.¹²¹³

As THS accumulates on surfaces and becomes embedded in materials and objects of an indoor environment of a smoker, chemical and physical ageing continues over weeks and months. Nicotine is the most abundant compound in THS and undergoes secondary reactions that have resulted in the creation of more hazardous products in chamber studies of THS ageing. For instance, nicotine reacts with ozone and forms potentially harmful ultrafine particles through oxidative ageing.¹⁴ Nicotine on surfaces reacts with nitrous acid (ie, a common ambient oxidant and emitted from gas stoves) and forms secondary compounds including several carcinogenic tobacco-specific nitrosamines (TSNAs), some of which are not present in freshly emitted SHS.¹⁴ However, the extent to which these laboratory findings can be demonstrated under real-world conditions is an important question and one addressed in this study.

The present study was designed to examine the ageing and persistence of THS pollutants in homes of smokers who successfully quit smoking. Studying homes of successful quitters allowed us to measure how THS pollutants and exposure change under natural conditions of human habitation and in the absence of new tobacco smoke emissions. Studying THS under real-world conditions is important because different lifestyles, cleaning habits, home furnishing, interior designs, building conditions and building locations may affect the chemical ageing of THS pollutants and exposure. These settings provide important tests for findings and hypotheses generated under controlled laboratory conditions and can in return generate new hypotheses to be tested under more controlled conditions. Finally, examining THS pollution and exposure in homes of quitters draws attention to the possibility that quitting smoking may not immediately and completely end the exposure to tobacco smoke toxicants. Specifically, examination of homes of quitters allows the exploration of how and when levels of THS pollution and exposure to tobacco smoke toxicants change after successful cessation.

METHODS

Study design

This study relied on a repeated measures design, examining THS pollution in participants' homes and THS exposure in non-smoking cohabitants before and 1 week, 1 month, 3 months and 6 months following smoking cessation. Ninety smokers initially completed baseline (BL) interviews, and house dust and surface wipe samples were collected in the primary smoking location at home. Eligible non-smoking residents provided urine and finger wipe samples.

Participants

Recruitment—Following Institutional Review Board approval at San Diego State University and the Veterans Administration (VA) San Diego Healthcare System, participants were recruited through advertisements in local print (N=80) and electronic news media (N=4), referrals from friends, relatives or coworkers (N=3) and the VA San Diego Pharmacy Telephone Smoking Cessation Clinic (N=3).

Eligibility—Smokers were eligible to participate at BL if they were age 18 or older, spoke English, had set a date to quit smoking and were enrolled in a cessation programme or were receiving help from a medical professional to quit. We recruited participants only if they were the only smoker living in their home; had lived in their home for at least 6 months and planned to live there for the next 6 months; reported smoking a minimum of seven cigarettes, cigars or tobacco pipe bowls per week indoors at home during the week prior to the BL measure and for at least 5 of the past 6 months and planned to have a ban on smoking inside their home after they quit. If there was a non-smoking resident who lived in the home, the youngest individual willing to participate (and not breastfeeding) was selected as the target non-smoker from whom urine and finger wipe samples were collected. Adult participants (smokers and non-smokers) signed informed consent forms, and non-smoking participants aged 6–17 signed assent forms following parental permission. Parents or legal guardians gave consent for children under age 6 to participate.

After BL, participants were eligible for subsequent study measures if they reported that they had not smoked and no one had smoked inside their home since their quit date. To verify this, exhaled carbon monoxide (CO) measures were obtained from former smokers at each post-BL measurement visit. Those who had an expired breath CO concentration of 5 ppm or higher were asked to provide a saliva sample for on-site testing for cotinine levels using a NicAlert test strip (TestCountry, San Diego, California, USA; sensitivity 99%; specificity 96%).¹⁵ A value >0 on a 5-point scale disqualified them from the study due to presumed continued smoking.

Two homes were excluded from analyses because the non-smoking cohabitants showed cotinine and 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol (NNAL) levels indicative of tobacco use, one of whom was a non-smoker but lifelong user of chewing tobacco. See table 1 for characteristics of participants and their homes at BL (N=88) and for the subset of successful quitters 3 months later (N = 13).

Measures

Pairs of research assistants visited participants' homes to conduct in-person interviews and to collect environmental and biological samples. Interviews were conducted with the eligible smoker; and questions about SHS exposure of non-smokers were asked of the non-smokers if they were adults. Dust and surface wipe samples were collected in the primary smoking location (most often the living room).

Personal interviews—At each interview, participants reported their smoking and the target non-smoker's SHS exposure on typical work and non-work days (or week and weekend days if participants did not work outside the home) during the past 7 days, including exposure from other residents and visitors, and outside of the home including in the car. We defined SHS exposure as the number of cigarettes smoked while the non-smoker was in the same indoor room or car. We computed non-smokers' weekly exposure to cigarettes in the home and used the variable 'total exposure' as a measure of exposure to all cigarettes in the home, car and elsewhere. These measures have shown acceptable test–retest reliability and validity in relation to cotinine and nicotine assays in past studies.¹⁶¹⁷

Nicotine on living room and bedroom surfaces—Prescreened cotton rounds (100% cotton facial wipes) were wetted with 1.5 mL of 0.1% ascorbic acid and wiped over a 100 cm² area, typically a wooden door unlikely to be frequently cleaned.¹⁸ Samples were stored at –20°C in the dark until analysis by liquid chromatography-tandem mass spectrometry (LC-MS/MS) using electrospray ionisation (ESI) on a Thermo-Finnigan TSQ Quantum Mass Spectrometer. A description of the analytic methods for the measurement of nicotine on surfaces is provided in the online supplementary material. Nicotine was quantified against the deuterated internal standard, nicotine-d₄ (CDN Isotopes, Pointe-Claire, Quebec, Canada). Nicotine levels were reported as micrograms of nicotine per square metre of surface (µg/m²).

Nicotine on index finger—A wipe sample of the non-smoker's dominant hand index finger was taken at each home visit.¹² Wipes were prepared and processed as above. Nicotine levels were reported in nanograms of nicotine per finger wipe (ng/wipe).

Dust nicotine and TSNAs in living room and bedroom—Dust samples were collected from a 1 mx1 m area (or from a larger area if needed to collect ~1 cm of dust in a collection bottle) with a high-volume-small surface-sampler (HVS4, CS3, Venice, Florida, USA) cyclone vacuum into methanol-washed amber bottles. Samples were transported, cooled and weighed, and large debris (such as pet hairs) was removed with tweezers. Next, samples were methanol-washed, sieved through a 150 µm stainless steel mesh, weighed again and then stored at –20°C until analysis using LC-MS/MS. A description of the analytic methods for the measurement of dust nicotine and TSNAs is provided in the online supplementary material. Nicotine levels are reported in micrograms of nicotine per gram of dust collected (µg/g; ie, concentration) and in micrograms of nicotine per square metre vacuumed (µg/m²; loading). Levels of the TSNAs (NNK, 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone; NNN, 3-(1-nitroso-2-pyrrolidinyl)pyridine; NAT, 1,2,3,6-tetrahydro-1-nitroso-2,3'-bipyridine; NAB, 3-(1-nitroso-2-piperidinyl)pyridine) are reported in nanograms

per gram of dust collected (ng/g; ie, concentration) and in nanograms per square metre vacuumed (ng/m²; loading).

Urinary cotinine and NNAL concentration—At each home visit, a urine sample was collected from the non-smoker. Samples were frozen at −20°C until analysis for cotinine concentration by LC-MS/MS using ESI. Our methods have been previously described.¹³ Urine samples were analysed for NNAL by LC-MS/MS at the Division of Clinical Pharmacology and Experimental Therapeutics, University of California, San Francisco.¹⁹ Cotinine and NNAL concentrations were reported in nanograms of cotinine and picograms of NNAL per millilitre of urine (ng/mL; pg/mL).

Limits of quantitation (LOQ) were ~0.1 µg nicotine/m² for wipe samples, 0.01 µg nicotine/g dust, 0.30 ng NNK and NAB/g dust, 1.25 ng NNN and NAT/g dust. The detection limit for urine cotinine was ~0.05 ng/mL.

Statistical analyses

To control for non-normal distributions and heterogeneous error variances, we subjected response variables to logarithmic transformation and report geometric means and their CIs instead of arithmetic means. Stata V.14 statistical software was employed for analyses (StataCorp, Stata statistical software: Release V.14, College Station, Texas, USA: Stata Corporation, 2015). The repeated-measures data were analysed using Stata's random-effects regression models for panel data: xtreg for uncensored and xtlogit for censored response variables. We compared BL to follow-up measures in models with the following covariates: square footage of home, proportion of floor area carpeted, cleaning practices and age, gender and number of residents. These covariates did not improve model fit or affect parameter estimates for any of the models. The type I error rate was set at $\alpha=0.05$, two tailed. Since the amount of dust collected varied between assessments, we included sieved dust weight as a covariate in all models examining change in dust nicotine and TSNA loading. These models did not change our conclusions, and we report and interpret the unadjusted geometric means for nicotine and TSNA loadings. Tables of the sieved dust weights and the dust weight-adjusted means are reported in the online supplementary tables S1 and S2.

RESULTS

Smoking behaviour at BL and quit rates

Prior to smoking cessation, participants reported smoking an average total of 95 cigarettes per week (95% CI (83.8 to 108.5)). An average of 50 cigarettes per week (95% CI (41.3 to 59.6)) were smoked indoors at home by participants and their visitors. Of the 88 smokers with a designated quit date at the BL interview, 35 (40%) were successful quitters at the week 1 follow-up visit that took place (median=9 days) after the quit date. At the month 1 visit (median=35 days after quit date), 18 participants (20%) maintained successful abstinence. At the month 3 visit (median=97 days), successful quitters decreased to 13 (15%), and at month 6 (median = 184 quit days), 8 participants (9%) had maintained successful smoking cessation.

Changes in THS pollution following smoking cessation

Table 2 shows nicotine levels on surfaces, on fingers of non-smokers and in dust for cohorts of successful quitters at BL before cessation and at 1 week, 1 month, 3 months and 6 months after cessation. Table 3 shows corresponding levels of the four TSNA's measured in dust at the same time points. To capitalise on the largest sample size for each follow-up examination, the statistical analysis of changes compared BL levels to each postcessation follow-up for each successful quitter cohort to that point. Nicotine and TSNA levels based on all available samples collected at each of the time points are presented in online supplementary table S3.

Short-term changes in THS pollution: BL to 1 week postcessation—The week 1 follow-up measure took place ~14 days (median) after the BL and 9 days (median) after the quit date. Using random-effects regression models comparing THS levels at BL and week 1 postcessation, we found statistically significant declines in surface nicotine levels (22.2 vs 10.8 $\mu\text{g}/\text{m}^2$; $z = -2.7$, $p = 0.007$) and finger nicotine levels (29.1 vs 9.1 ng/wipe, $z = -2.10$, $p = 0.036$). We found a significant increase in dust nicotine loading (5.0 vs 9.3 $\mu\text{g}/\text{m}^2$; $z = 2.09$, $p = 0.036$) and no significant change in dust nicotine concentration (10.4 vs 10.5 $\mu\text{g}/\text{g}$; $z = -0.25$, $p = 0.803$).

For TSNA's in dust, NNK showed the same pattern as dust nicotine levels; that is, we observed a statistically significant increase in dust NNK loading (8.4 vs 19.9 ng/ m^2 ; $z = 2.14$, $p = 0.032$), a non-significant change in dust NNK concentration (7.9 vs 11.2 $\mu\text{g}/\text{g}$; $z = 0.8$, $p = 0.421$) and no changes in NAT, NAB or NNN loading or concentrations.

Long-term change in THS pollutants: BL to 1 and 6 months postcessation—From month 1 to 6 after smoking cessation, surface nicotine levels continued to be significantly lower than BL ($p < 0.05$). After week 1, however, there were no further significant declines. Among the participants who remained abstinent at month 1, surface nicotine was at 27% of BL levels (15.8 vs 4.3 $\mu\text{g}/\text{m}^2$). Among the participants who remained abstinent at month 3, surface nicotine was at 23% of their BL levels (11.2 vs 2.6 $\mu\text{g}/\text{m}^2$), and for month 6 quitters, the mean level was at 35% of BL levels (9.2 vs 3.2 $\mu\text{g}/\text{m}^2$).

Finger nicotine—Finger nicotine levels showed a decline similar to surface nicotine. Finger wipes at the month 1 follow-up were at 40% of the cohort's BL levels (25.6 vs 10.2 ng/wipe). At the 3-month follow-up, wipe values were at 25% of their BL levels (21.1 vs 5.2 ng/wipe). However, among month 6 quitters, we observed a decline to 6% of their BL levels (49.4 vs 2.9 ng/wipe).

Nicotine concentrations and loadings in settled house dust—Nicotine concentrations and loadings in settled house dust among the successful quitters changed little after week 1. For month 1 quitters, the mean nicotine concentration was almost identical to that at BL (5.3 vs 5.2 $\mu\text{g}/\text{g}$, $p > 0.30$). For month 3 quitters, the mean nicotine concentration declined to 51% of BL levels (5.5 vs 2.8 $\mu\text{g}/\text{g}$, $p > 0.10$), and for month 6 quitters, nicotine concentrations were at 79% of their BL levels (4.8 vs 3.8 $\mu\text{g}/\text{g}$, $p > 0.30$). Similar patterns held for nicotine loadings of settled house dust. Dust from successful

quitters at month 1 (3.0 $\mu\text{g}/\text{m}^2$), month 3 (2.1 $\mu\text{g}/\text{m}^2$) and month 6 (2.2 $\mu\text{g}/\text{m}^2$) showed almost equivalent levels and changed little compared to their respective BL levels (2.8, 1.6 and 1.4 $\mu\text{g}/\text{m}^2$).

Similar to dust nicotine, we observed no significant changes in NNK concentration or loading in dust 1 month after smoking cessation. Compared to BL, mean levels of NNK concentration (8.7 vs 9.9 pg/g) and NNK loading (11.6 vs 13.2 pg/m^2) showed statistically non-significant increases (all $p>0.30$). Limited sample sizes prevented statistical tests for changes in NNK for month 3 and 6 and for NNN, NAB and NAT for week 1 and later cohorts. All available measures of NAB at month 6 ($N=2$) and of NAT at month 1 ($N=4$) and month 3 ($N=3$) were below LOQ.

Changes in THS exposure following smoking cessation

Table 4 shows urinary cotinine and NNAL levels and reported exposure for cohorts of successful quitters at BL before cessation and at time points up to 6 months following successful cessation. Table 4 also reports cotinine and NNAL levels for the subset of participants who reported no SHS exposure outside the home after cessation. Cotinine and NNAL levels based on all available urine samples collected at each of the time points are presented in online supplementary table S4.

Short-term change in exposure to THS pollutants: BL to 1 week postcessation

—Random-effects regression models comparing exposure levels at BL and week 1 postcessation revealed 39% and 37% reduction in cotinine and NNAL in the non-smokers sharing the home, respectively. For the cohort of week 1 quitters, the levels of cotinine declined from 9.9 to 6.0 ng/mL , and the levels of NNAL decreased from 10.7 to 6.2 ng/mL . Neither difference was statistically significant (cotinine: $z = 1.07$, $p = 0.29$; NNAL: $z = 1.66$, $p = 0.10$). Cotinine and NNAL levels for participants without any reported SHS exposure (BL—month 1) were of similar magnitude to those based on all participants.

Long-term change in THS exposure: BL to 1–6 months postcessation—

Beginning at month 1 after smoking cessation, urine cotinine levels were significantly lower than those at BL (all $p<0.05$), with levels between 1.3 and 2.7 ng/mL . These levels were 28–59% of the corresponding BL levels of the non-smokers in the month 1, 3 and 6 quitter cohorts. Cotinine levels for participants without any reported SHS exposure (BL—month 1) were similar to those based on all participants.

Compared to nicotine exposure, NNK exposure declined more gradually. In the month 1 quitter cohort, there was no change from week 1 to month 1 (6.7 vs 6.7 pg/mL). It was not until 3 months after smoking cessation that NNAL levels declined significantly compared to BL (11.0 vs 3.2 pg/mL , $p = 0.001$), and this significant reduction was observed at month 6 as well (2.7 pg/mL , $p = 0.014$). NNAL levels for participants without any reported SHS exposure (BL—month 1) were similar to those based on all participants.

DISCUSSION

This is the first study to examine THS pollutants in homes of smokers after smoking cessation and to investigate exposure to tobacco smoke toxicants among non-smokers living in homes of successful quitters. We found that long after successful cessation, homes of smokers showed measurable levels of nicotine in dust and on surfaces and of NNK in dust. While surface nicotine levels showed significant initial declines, followed by a more stable level compared to BL, dust nicotine and NNK levels remained mostly unchanged. This is consistent with homes of smokers presenting deep nicotine reservoirs that contribute to continued pollution of house dust with nicotine and with TSNA through secondary reaction long after successful cessation. Decreases in nicotine levels on non-smokers' fingers matched those on surfaces, suggesting that surfaces may be the source of nicotine on non-smokers' fingers. THS exposure of non-smokers living with former smokers (ie, urinary cotinine and NNAL) showed similar patterns as surface nicotine; that is, initial decline followed by stable levels of exposure above those found in non-smokers without exposure to SHS or THS.

While the present study did not collect comparison data in non-smokers' homes, we offer for reference the THS levels from our previous studies of private homes of non-smokers with smoking bans who were recruited using similar strategies, were of similar sociodemographic background and lived in similar size homes in similar neighbourhoods.¹³¹⁸ In these past studies, geometric mean levels of surface nicotine ranged from 0 to 1.6 $\mu\text{g}/\text{m}^2$, and finger nicotine levels ranged from 0.5 to 1.4 $\mu\text{g}/\text{m}^2$. Three to 6 months after cessation, surface nicotine levels in homes of former smokers were 2–3 times higher and finger nicotine levels were 2–20 times higher than in homes of non-smokers with smoking bans. Dust nicotine concentrations were 100 times higher than levels found in private homes occupied by never smokers with smoking bans for more than 1 year who had not visited any smokers over the past month (3.8 vs <0.03 $\mu\text{g}/\text{m}^2$).¹⁸ They were, however, similar to levels found in another study of homes of past non-smokers where no one had smoked for the past 6 months (3.1–3.6 $\mu\text{g}/\text{m}^2$).¹³ Urine cotinine levels 3–6 months after cessation were still 8–14 times higher than those found in non-smokers living in homes with smoking bans.¹³

The elimination half-life of cotinine in adults is ~ 16–20 hours.²⁰²¹ In the absence of additional exposure to nicotine after smoking cessation, BL levels of 10 ng/mL of cotinine should have declined to <0.1 ng/mL 1 week after last exposure and be near the limit of detection in the urine of non-smokers. However, cotinine levels in non-smokers remained above 1.5 ng/mL 3–6 months after cessation. This suggests that residents of homes of former smokers continue to be exposed to nicotine and other tobacco smoke toxicants after smoking cessation. Although some participants reported occasional exposure to SHS outside the home, this exposure cannot fully account for the observed cotinine levels. In combination with the elevated levels of nicotine on fingers of non-smokers, this makes nicotine deposits in settled house dust and on surfaces a plausible additional exposure source.

In comparison to cotinine, NNAL has a significantly longer half-life (18–45 days).²²²³ This partly explains why NNAL levels 1 month after cessation are still about 60% of BL levels. The slow metabolism of NNK, however, cannot fully explain why 3 and 6 months after

smoking cessation NNAL levels in non-smokers were still 54–72% of those found 1 week after cessation. On the basis of elimination half-life, the expected level at 3 and 6 months would be ~1 and <0.2 pg/mL, respectively. However, the observed levels were 3.1 and 2.7 pg/mL, respectively. This suggests that non-smokers continue to be exposed to the carcinogen NNK after smoking cessation. While we cannot rule out the occasional SHS exposure, NNK deposits in settled house dust are a plausible additional exposure source.

While cotinine and NNAL levels associated with THS exposure are relatively low compared to active smokers and SHS exposed persons, their levels among non-smokers should all be below the LOQ in a completely tobacco-free environment where no tobacco is used. The pervasiveness and persistence of tobacco toxicants in indoor environments (ie, THS) as well as in outdoor environments (eg, landfill leachates, groundwater and outdoor air) raise important questions about the cumulative impact of low-level chronic exposure.^{24–30}

The study findings show that successful smoking cessation, while very important for immediate and long-term health benefits, does not immediately and completely eliminate exposure of the former smoker or non-smoking cohabitants to tobacco smoke toxicants. Homes of smokers remained polluted with nicotine and TSNA in dust and on surfaces, and residents continued to be exposed for at least 6 months after smoking cessation. Our findings show that although the THS pollutants in dust and surface reservoirs declined, they did not reach levels that would be found in the homes of long-time non-smokers with smoking bans.^{131831–33} Since there is no risk-free level of exposure to tobacco smoke carcinogens,³⁴ the continued exposure to THS after quitting limits the full benefits of cessation that would be expected after eliminating all exposure to tobacco smoke toxicants.

The persistence of THS pollutants in settled house dust and on surfaces presents a potential risk after smoking cessation, especially for infants and young children in the home, and poses interesting questions for future research. It is well documented that successful smoking cessation is often difficult to achieve even after multiple attempts. Consistent with the relapse observed in the present study, 80–90% of quitters overall relapse and begin smoking again within the first 3 months of cessation.³⁵³⁶ The main reasons for such poor cessation outcomes are the addictive properties of nicotine, strong cravings and withdrawal symptoms and conditioned cues for smoking (eg, odours, paraphernalia, situations).³⁷ Findings from this study raise the possibility that relapse may be mediated or moderated by the continued low-level exposure to nicotine and odorant compounds of THS in a smoker's home that serve as triggers and cues. Chemical exposure to tobacco constituents found in THS may contribute to relapse due to physiological processes associated with cravings. If this is indeed the case, removing THS pollutants from the home environment after smoking cessation might lead to more prolonged and sustained cessation outcomes.

The persistence and pervasiveness of THS pollutants also raise the question of what remediation efforts might be necessary to achieve levels of background THS found in the homes of lifelong non-smokers with smoking bans. Owing to the physical and chemical properties of tobacco smoke constituents, full remediation might include washing, removal, or replacement of carpets, upholstery, drapes, clothes, blankets, mattresses, etc, extensive vacuuming to remove accumulated dust deposits and washing of all surfaces (cabinets,

furniture, doors, walls, ceilings) to remove surface deposits. In homes of long-term heavy smokers, this could even require replacement of such THS reservoirs as gypsum board (drywall) and ceiling tiles. Obviously, this would be very expensive and could create a significant burden to smokers trying to quit. However, the evidence provided by this study warrants future research to better understand if continued THS exposure following cessation affects cessation and health outcomes. The persistence and pervasiveness of THS pollution and the involuntary and unnoticed exposure to THS suggest that we may have to rethink the meaning of ‘smoke-free’, to include THS toxicants that are embedded in indoor environments where they leave a lasting legacy of earlier tobacco use.

Limitations

The observational nature of this study design and the ethical standards of research on humans did not allow experimental control over smoking cessation and relapse. Although we enrolled 90 smokers at BL, attrition due to relapse reduced sample sizes at each follow-up assessment. This reduced the statistical power to detect changes between BL and postcessation measures. The pattern of attrition suggested that smokers with higher levels of surface and dust nicotine in their homes were more likely to drop out than smokers with lower THS levels. Since our statistical analyses focused on cohorts of successful quitters with complete data at each assessment, the observed changes were not affected by missing data due to attrition. This study compared THS pollution and exposure levels among former smokers and non-smoking residents to historical data on non-smokers from our previous studies. Although these studies used similar recruitment procedures, analytic laboratory methods, and were conducted in similar communities, it is possible that the participants in the present study and the non-smoking participants in previous studies differed on factors other than their tobacco addiction. As we noted in the ‘Results’ section, some non-smokers reported that they were exposed to SHS outside the home after the smoker quit. It is also possible that they may have been unknowingly exposed to SHS and THS outside the home. Some of the non-smoking residents were children, and their parents provided proxy reports for exposure. This could have resulted in questionable reports of SHS for some of the non-smokers.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgements

The authors thank Dr Mark G Myers for his assistance with recruiting participants from the San Diego Veterans Administration Pharmacy Telephone Smoking Cessation Clinic.

Funding This research was supported by funds from the California Tobacco-Related Disease Research Program of the University of California, grant number 19CA-0164.

REFERENCES

1. Rodgman A, Perfetti TA. The chemical components of tobacco and tobacco smoke. CRC Press, Taylor & Francis Group, 2009.

2. IARC Working Group on the Evaluation of Carcinogenic Risks to Humans. Review of human carcinogens. International Agency for Research on Cancer, WHO, 2012.
3. State of California Air Resource Board. Proposed Identification of Environmental Tobacco Smoke as a Toxic Air Contaminant: California Environmental Protection Agency, Air Resources Board, Office of Environmental Health Hazard Assessment; 2005 <http://www.arb.ca.gov/regact/ets2006/app3exe.pdf>
4. Matt GE, Quintana PJ, Destailats H, et al. Thirdhand tobacco smoke: emerging evidence and arguments for a multidisciplinary research agenda. *Environ Health Perspect* 2011;119:1218–26. [PubMed: 21628107]
5. Sleiman M, Logue JM, Luo W, et al. Inhalable constituents of thirdhand tobacco smoke: chemical characterization and health impact considerations. *Environ Sci Technol* 2014;48:13093–101. [PubMed: 25317906]
6. Van Loy MD, Riley WJ, Daisey JM, et al. Dynamic behavior of semivolatile organic compounds in indoor air. 2. Nicotine and phenanthrene with carpet and wallboard. *Environ Sci Technol* 2001;35:560–7. [PubMed: 11351729]
7. Daisey JM. Tracers for assessing exposure to environmental tobacco smoke: what are they tracing? *Environ Health Perspect* 1999;107(Suppl 2):319–27. [PubMed: 10350517]
8. Mayer B How much nicotine kills a human? Tracing back the generally accepted lethal dose to dubious self-experiments in the nineteenth century. *Arch Toxicol* 2014;88:5–7. [PubMed: 24091634]
9. Martins-Green M, Adhami N, Frankos M, et al. Cigarette smoke toxins deposited on surfaces: implications for human health. *PLoS One* 2014;9:e86391.
10. Morgan MK, Sheldon LS, Croghan CW, et al. Exposures of preschool children to chlorpyrifos and its degradation product 3,5,6-trichloro-2-pyridinol in their everyday environments. *J Expo Anal Environ Epidemiol* 2005;15:297–309. [PubMed: 15367928]
11. Wilson NK, Chuang JC, Lyu C, et al. Aggregate exposures of nine preschool children to persistent organic pollutants at day care and at home. *J Expo Anal Environ Epidemiol* 2003;13:187–202. [PubMed: 12743613]
12. Quintana PJ, Matt GE, Chatfield D, et al. Wipe sampling for nicotine as a marker of thirdhand tobacco smoke contamination on surfaces in homes, cars, and hotels. *Nicotine Tob Res* 2013;15:1555–63. [PubMed: 23460657]
13. Matt GE, Quintana PJ, Zakarian JM, et al. When smokers move out and non-smokers move in: residential thirdhand smoke pollution and exposure. *Tob Control* 2011;20:e1.
14. Sleiman M, Destailats H, Smith JD, et al. Secondary organic aerosol formation from ozone-initiated reactions with nicotine and secondhand tobacco smoke. *Atmos Environ* 2010;44:4191–8.
15. Montalto NJ, Wells WO. Validation of self-reported smoking status using saliva cotinine: a rapid semiquantitative dipstick method. *Cancer Epidemiol Biomarkers Prev* 2007;16:1858–62. [PubMed: 17855706]
16. Matt GE, Wahlgren DR, Hovell MF, et al. Measuring environmental tobacco smoke exposure in infants and young children through urine cotinine and memory-based parental reports: empirical findings and discussion. *Tob Control* 1999;8:282–9. [PubMed: 10599573]
17. Matt GE, Hovell MF, Zakarian JM, et al. Measuring secondhand smoke exposure in babies: the reliability and validity of mother reports in a sample of low-income families. *Health Psychol* 2000;19:232–41. [PubMed: 10868767]
18. Matt GE, Quintana PJ, Hovell MF, et al. Households contaminated by environmental tobacco smoke: sources of infant exposures. *Tob Control* 2004;13:29–37. [PubMed: 14985592]
19. Jacob P III, Havel C, Lee DH, et al. Subpicogram per milliliter determination of the tobacco-specific carcinogen metabolite 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol in human urine using liquid chromatography-tandem mass spectrometry. *Anal Chem* 2008;80:8115–21. [PubMed: 18841944]
20. Benowitz NL. Cotinine as a biomarker of environmental tobacco smoke exposure. *Epidemiol Rev* 1996;18:188–204. [PubMed: 9021312]
21. Benowitz NL. Biomarkers of environmental tobacco smoke exposure. *Environ Health Perspect* 1999;107(Suppl 2):349–55.

22. Goniewicz ML, Havel CM, Peng MW, et al. Elimination kinetics of the tobacco-specific biomarker and lung carcinogen 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol. *Cancer Epidemiol Biomarkers Prev* 2009;18:3421–5. [PubMed: 19959691]
23. Hecht SS, Carmella SG, Chen M, et al. Quantitation of urinary metabolites of a tobacco-specific lung carcinogen after smoking cessation. *Cancer Res* 1999;59:590–6. [PubMed: 9973205]
24. Kaufman P, Zhang B, Bondy SJ, et al. Not just ‘a few wisps’: real-time measurement of tobacco smoke at entrances to office buildings. *Tob Control* 2011;20:212–18. [PubMed: 21177666]
25. Mackul’ak T, Birošová L, Grabic R, et al. National monitoring of nicotine use in Czech and Slovak Republic based on wastewater analysis. *Environ Sci Pollut Res Int* 2015;22:14000–6. [PubMed: 25956519]
26. Buerge IJ, Kahle M, Buser HR, et al. Nicotine derivatives in wastewater and surface waters: application as chemical markers for domestic wastewater. *Environ Sci Technol* 2008;42:6354–60. [PubMed: 18800501]
27. Buszka PM, Yeskis DJ, Kolpin DW, et al. Waste-indicator and pharmaceutical compounds in landfill-leachate-affected ground water near Elkhart, Indiana, 2000–2002. *Bull Environ Contam Toxicol* 2009;82:653–9. [PubMed: 19290448]
28. Masoner JR, Kolpin DW, Furlong ET, et al. Landfill leachate as a mirror of today’s disposable society: pharmaceuticals and other contaminants of emerging concern in final leachate from landfills in the conterminous United States. *Environ Toxicol Chem* 2016;35:906–18. [PubMed: 26562222]
29. Masoner JR, Kolpin DW, Furlong ET, et al. Contaminants of emerging concern in fresh leachate from landfills in the conterminous United States. *Environ Sci Process Impacts* 2014;16:2335–54. [PubMed: 25111596]
30. Farren NJ, Ramírez N, Lee JD, et al. Estimated exposure risks from carcinogenic nitrosamines in urban airborne particulate matter. *Environ Sci Technol* 2015;49:9648–56. [PubMed: 26207531]
31. Matt GE, Quintana PJ, Fortmann AL, et al. Thirdhand smoke and exposure in California hotels: non-smoking rooms fail to protect non-smoking hotel guests from tobacco smoke exposure. *Tob Control* 2014;23:264–72. [PubMed: 23669058]
32. Thomas JL, Hecht SS, Luo X, et al. Thirdhand tobacco smoke: a tobacco-specific lung carcinogen on surfaces in smokers’ homes. *Nicotine Tob Res* 2014;16:26–32. [PubMed: 23892827]
33. Whitehead TP, Havel C, Metayer C, et al. Tobacco alkaloids and tobacco-specific nitrosamines in dust from homes of smokeless tobacco users, active smokers, and non-tobacco users. *Chem Res Toxicol* 2015;28:1007–14. [PubMed: 25794360]
34. U.S. Department of Health and Human Services. The health consequences of involuntary exposure to tobacco smoke: a report of the surgeon general. Atlanta (GA): US Department of Health and Human Services, Centers for Disease Control and Prevention, Coordinating Center for Health Promotion, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health, 2006.
35. Gilpin EA, Pierce JP, Farkas AJ. Duration of smoking abstinence and success in quitting. *J Natl Cancer Inst* 1997;89:572–6. [PubMed: 9106646]
36. Marti J Successful smoking cessation and duration of abstinence—an analysis of socioeconomic determinants. *Int J Environ Res Public Health* 2010;7:2789–99. [PubMed: 20717538]
37. Winkler MH, Weyers P, Mucha RF, et al. Conditioned cues for smoking elicit preparatory responses in healthy smokers. *Psychopharmacology (Berl)* 2011;213:781–9. [PubMed: 20953588]

What this paper adds

- ▶ Thirdhand smoke (THS) is created when tobacco smoke pollutants remain on surfaces and in dust, and are re-emitted and resuspended back into the air or react with oxidants and other compounds in the environment to yield secondary pollutants.
- ▶ Indoor environments in which tobacco is regularly smoked become reservoirs of THS that store and gradually release pollutants over time, potentially leading to the involuntary exposure of non-smokers through inhalation, ingestion and dermal transfer long after smoking has taken place.
- ▶ This is the first study to examine THS pollutants in homes of smokers after smoking cessation and to investigate exposure to THS among non-smokers living in homes of successful quitters.
- ▶ When smokers quit and maintained abstinence, THS in dust and on surfaces persisted and non-smokers continued to be exposed.
- ▶ Six months after cessation, homes of former smokers continued to show elevated levels of nicotine in dust and on surfaces and of the carcinogen, 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone in dust.

Table 1

Characteristics of participating smokers, non-smoker residents and residences at baseline

Smoker participants (N=88)	
Mean age (years)	49
Gender (% female)	51
Race/ethnicity (%)	
African-American/Black	48
Caucasian/White	32
Latino/Hispanic	10
Multiracial and other	10
Education (%)	
Less than HS degree	10
HS/GED	21
Some college	59
Bachelor's degree or higher	10
Employment (%)	
Not employed	69
Working part-time	14
Working full time	11
Annual income (US\$1000)	
25th–50th–75th centile	11–18–30
Total smoking (cigs/week)	
25th–50th–75th centile	60–93–140
Non-smoker participants living with smoker (N=50)	
Age	
Mean age (years)	35
% <18 years	34
Gender (% female)	52
Race/ethnicity (%)	
African-American/Black	46
Caucasian/White	23
Latino/Hispanic	12
Multiracial and other	19
Reported exposure (prequit)	
At home (cigs/week)	
25th–50th–75th centile	0–8–35
Overall (cigs/week)	
25th–50th–75th centile	1–29–50
Residences (N=88)	
Surface area (sq ft)	

Smoker participants (N=88)	
25th–50th–75th centile	371–504–692
Area carpeted (%)	
25th–50th–75th centile	0–65–75
Number of occupants	
25th–50th–75th centile	1 –2–2
Mean % children (<18 years)	12
Years in residence	
25th–50th–75th centile	1 –2–6

GED, General Educational Development Diploma; HS, High School Diploma.

Geometric means and 95% CIs (in parentheses) of nicotine on surfaces, in dust and on fingers in homes of smokers at baseline and of successful quitters after smoking cessation

Table 2

	N	Baseline prequit	W1 postquit (Mdn=9d)	M1 postquit (Mdn=35d)	M3 postquit (Mdn=97d)	M6 postquit (Mdn=184d)
Surface nicotine loading (µg/m ²)						
Baseline	45	31.2 (16.2 to 59.0)				
Week 1 quitters	35	22.2 (11.1 to 43.6)	10.8** (4.9 to 22.9)			
Month 1 quitters	17	15.8 (5.6 to 41.1)	5.6** (1.5 to 16.6)	4.3** (1.4 to 10.5)		
Month 3 quitters	11	11.2 (2.6 to 40.3)	5.4** (0.6 to 24.6)	3.2** (0.3 to 12.8)	2.6** (0.3 to 10.0)	
Month 6 quitters	7	9.2 (2.5 to 28.6)	3.1 [†] (0.4 to 10.8)	2.7 [†] (0.3 to 9.3)	1.5* (0.2 to 4.6)	3.2 [†] (0 to 4.6)
Finger nicotine loading—non-smoker residents (ng/wipe)						
Baseline	25	25.6 (13.2 to 48.9)				
All week 1 quitters	16	29.1 (12.2 to 67.8)	9.1* (3.8 to 20.2)			
All month 1 quitters	9	25.6 (6.0 to 100.6)	9.8 (5.2 to 17.6)	10.2 (4.7 to 21.2)		
All month 3 quitters	8	21.1 (5.0 to 97.1)	9.8 (4.7 to 19.5)	10.4 (4.2 to 24.2)	5.2* (0.9 to 19.9)	
All month 6 quitters	6	49.4 (17.0 to 141.6)	7.5** (4.1 to 17.6)	11.5* (3.0 to 37.7)	9.1* (1.5 to 39.6)	2.9** (0 to 46.1)
Dust nicotine concentration (µg/g)						
All baseline smokers	65	17.4 (12.5 to 24.0)				
All week 1 quitters	22	10.4 (5.6 to 18.7)	10.5 (5.6 to 19.0)			
All month 1 quitters	9	5.3 (2.2 to 11.5)	6.2 (2.5 to 13.8)	5.2 (2.0 to 11.9)		
All month 3 quitters	7	5.5 (2.2 to 12.2)	5.6 (1.5 to 16.5)	4.0 (1.0 to 11.3)	2.8 (0.9 to 6.7)	
All month 6 quitters	5	4.8 (2.3 to 9.2)	5.2 (1.1 to 17.1)	5.0 (0.5 to 23.3)	3.6 (0.6 to 11.8)	3.8 (0 to 27.6)
Dust nicotine loading (µg/m ²)						
All baseline smokers	65	9.3 (5.9 to 14.4)				
All week 1 quitters	22	5.0 (3.3 to 9.8)	9.3* (4.1 to 19.9)			
All month 1 quitters	9	2.8 (0.4 to 9.7)	4.8* (1.0 to 15.5)	3.0 (0.3 to 11.7)		
All month 3 quitters	7	1.6 (0.4 to 3.8)	3.3* (0.8 to 9.2)	1.8 (0.2 to 5.6)	2.1 (0.1 to 7.9)	
All month 6 quitters	5	1.4 (0.1 to 4.5)	2.9* (0 to 14.7)	1.8 (0 to 9.3)	1.8 (0 to 8.6)	2.2 (0 to 10.5)

M1, month 1 ; M3, month 3; M6, month 6; W1, week 1

p<0.01

**

p<0.05

*

p<0.10

+

Two-tailed pre-post comparison:

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

Geometric means and 95% CIs (in brackets) of tobacco-specific nitrosamines in dust in homes of smokers at baseline and of successful quitters after smoking cessation

Table 3

	N	Baseline prequit	W1 postquit (Mdn=9d)	M1 postquit (Mdn=35d)
Dust NNK concentration (ng/g)				
All baseline smokers	22	8.9 (4.5 to 16.8)		
All week 1 quitters	13	7.9 (2.9 to 19.3)	11.2 (5.0 to 23.8)	
All month 1 quitters	4	8.7 (1.9 to 31.7)	13.8 (1.5 to 86.6)	9.9 (2.3 to 35.4)
Dust NNK loading (ng/m ²)				
All baseline smokers	22	14.5 (6.4 to 31.5)		
All week 1 quitters	13	8.4 (2.8 to 22.4)	19.9* (6.3 to 58.9)	
All month 1 quitters	4	11.6 (2.2 to 49.4)	36.3* (0.3 to 1067.6)	13.2 (1.6 to 76.7)
Dust NNN concentration (ng/g)				
All baseline smokers	17	2.5 (1.1 to 4.9)		
All week 1 quitters	7	3.1 (0.7 to 9.0)	1.5 (0 to 6.81)	
Dust NNN loading (ng/m ²)				
All baseline smokers	17	2.5 (1.1 to 4.9)		
All week 1 quitters	7	3.1 (0.7 to 9.0)	1.5 (0 to 6.81)	
Dust NAT concentration (ng/g)				
All baseline smokers	17	2.0 (0.1 to 5.1)		
All week 1 quitters	7	1.8 (0.3 to 5.0)	3.4 (0.3 to 13.1)	
Dust NAT loading (ng/m ²)				
All baseline smokers	17	3.5 (0.8 to 10.1)		
All week 1 quitters	7	2.9 (0.3 to 10.7)	8.4 (0.4 to 61.1)	
Dust NAB concentration (ng/g)				
All baseline smokers	17	0.9 (0.5 to 1.3)		
All week 1 quitters	7	0.8 (0.3 to 1.3)	<LOQ	
Dust NAB loading (ng/m ²)				
All baseline smokers	17	2.2 (1.4 to 3.3)		
All week 1 quitters	7	1.4 (0.5 to 1.3)	<LOQ	

Two-tailed pre-post comparison:

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

0.05
*

LOQ, Limit of quantitation; M1, month 1; NAB, N-nitrosoanabasine (3-(1-nitroso-2-piperidinyl)pyridine); NAT, N-nitrosoanatabine (1,2,3,6-tetrahydro-1-nitroso-2,3'-bipyridine); NNK, nicotine-derived nitrosamine ketone (4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone); NNN, N'-nitrososonornicotine (3-(1-nitroso-2-pyrrolidinyl)pyridine); W1, week 1.

Geometric means and 95% CIs (in brackets) for reported exposure and biomarkers of tobacco smoke exposure among non-smoking residents in homes of smokers at baseline and of successful quitters after smoking cessation

Table 4

	N	Baseline prequit	W1 postquit (Mdn=9d)	M1 postquit (Mdn=35d)	M3 postquit (Mdn=97d)	M6 postquit (Mdn=184d)
Reported SHS exposure (cigarettes/past week)						
At home		7.9 (3.7 to 12.3)	0	0	0	0
Total						
All week 1 quitters	17	17.6 (6.6 to 44.5)	1.6 ^{**} (0.4 to 3.8)			
All month 1 quitters	9	19.4 (3.8 to 85.3)	1.7 ^{**} (0.1 to 2.9)	2.0 ^{**} (0 to 8.6)		
All month 3 quitters	8	15.1 (0.4 to 17.6)	1.1 ^{**} (0 to 4.1)	1.0 ^{**} (0 to 3.9)	1.4 ^{**} (0.1 to 4.7)	
All month 6 quitters	6	12.9 (0.5 to 129.6)	1.1 ^{**} (0 to 6.4)	0.8 ^{**} (0 to 4.1)	1.2 ^{**} (0 to 4.9)	1.3 ^{**} (0 to 9.3)
Urinary cotinine (ng/mL)						
All week 1 quitters	17	9.9 (2.6 to 32.5)	6.0 (1.2 to 20.8)			
All month 1 quitters	9	3.7 (0.8 to 11.1)	1.4 ^{**} (0.5 to 2.7)	1.5 ^{**} (0.5 to 3.4)		
All month 3 quitters	8	3.9 (0.6 to 13.6)	1.3 ^{**} (0.4 to 2.8)	1.7 ^{**} (0.5 to 4.0)	1.7 ^{**} (0.4 to 4.2)	
All month 6 quitters	6	4.6 (0.1 to 25.8)	1.3 ^{**} (0.1 to 3.9)	1.6 ^{**} (0.1 to 5.6)	1.8 [*] (0.1 to 6.6)	2.7 (0 to 6.3)
Urinary cotinine (ng/mL) if no reported exposure postquit						
All week 1 quitters	9	6.5 (0.5 to 36.6)	3.9 (0.3 to 16.8)			
All month 1 quitters	5	5.2 (0 to 47.0)	1.9 (0.2 to 5.6)	1.8 (0 to 8.2)		
Urinary NNAL (pg/mL)						
All week 1 quitters	9	10.7 (4.7 to 24.2)	6.7 ⁺ (2.5 to 15.9)			
All month 1 quitters	9	10.7 (4.7 to 24.2)	6.7 ⁺ (2.5 to 15.9)	6.7 ⁺ (3.5 to 12.2)		
All month 3 quitters	8	11.0 (4.2 to 26.7)	5.9 ⁺ (1.9 to 15.5)	6.9 (3.3 to 13.7)	3.2 ^{**} (1.1 to 7.4)	
All month 6 quitters	6	10.0 (2.9 to 29.9)	4.2 ⁺ (0.7 to 14.9)	6.0 (1.9 to 15.7)	3.1 [*] (0.5 to 10.3)	2.7 [*] (0.5 to 8.1)
Urinary NNAL (pg/mL) if no reported exposure postquit						
All week 1 quitters	5	14.6 (5.3 to 37.9)	6.7 (1.7 to 21.0)			
All month 1 quitters	5	14.6 (5.3 to 37.9)	6.7 (1.7 to 21.0)	8.1 (2.5 to 17.7)		

Two-tailed pre-post comparison:

⁺p<0.10