HHS Public Access

Author manuscript

Tob Control. Author manuscript; available in PMC 2017 November 01.

Published in final edited form as:

Tob Control. 2016 November ; 25(6): 619–623. doi:10.1136/tobaccocontrol-2015-052506.

Thirdhand smoke contamination in hospital settings: Assessing exposure risk for vulnerable pediatric patients

Thomas F. NORTHRUP, Ph.D.[1], **Amir M. KHAN, M.D.**[2], **Peyton JACOB III, Ph.D.**[3], **Neal L. BENOWITZ, M.D.**[4], **Eunha HOH, Ph.D.**[5], **Melbourne F. HOVELL, Ph.D.**[6], **Georg E. MATT, Ph.D.**[7], and **Angela L. STOTTS, Ph.D.**[8]

Thomas F. NORTHRUP: Thomas.F.Northrup@uth.tmc.edu; Amir M. KHAN: Amir.M.Khan@uth.tmc.edu; Peyton JACOB: Peyton.Jacob@ucsf.edu; Neal L. BENOWITZ: Neal.Benowitz@ucsf.edu; Eunha HOH: EHoh@mail.sdsu.edu; Melbourne F. HOVELL: MHovell@mail.sdsu.edu; Georg E. MATT: GMatt@mail.sdsu.edu; Angela L. STOTTS: Angela.L.Stotts@uth.tmc.edu

[1]Assistant Professor and Coordinator of Behavioral Science Training, Department of Family and Community Medicine, University of Texas Medical School at Houston, 6431 Fannin, JJL 324, Houston, TX 77030

[2]Associate Professor, Department of Pediatrics, University of Texas Medical School at Houston; Medical Director Level III NICU, Children's Memorial Hermann Hospital, 6431 Fannin, MSB 3.236, Houston, TX 77030

[3]Research Chemist, Departments of Medicine and Psychiatry, University of California San Francisco; Division of Clinical Pharmacology, San Francisco General Hospital Medical Center, Box 0482, UCSF, San Francisco, CA 94143-0482

[4]Professor, Departments of Medicine and Bioengineering & Therapeutic Sciences, University of California San Francisco, 1001 Potrero Ave, SFGH 30, San Francisco, CA 94143

[5]Associate Professor and Division Head, Division of Environmental Health, Graduate School of Public Health, San Diego State University, 5500 Campanile Drive, San Diego, CA 92182-4162

[6]Director and Professor, Center for Behavioral Epidemiology and Community Health, Graduate School of Public Health, San Diego State University, 9245 Sky Park Court, Suite 230, Mail Box 230 San Diego, CA 92123–4388

[7]Professor and Chair, Department of Psychology, San Diego State University, 5500 Campanile Drive, San Diego, CA 92182-4611, Office Location: LS-110, Mail Code: 4611

[8]Professor and Director of Research, Department of Family and Community Medicine; Professor, Department of Psychiatry and Behavioral Sciences, University of Texas Medical School at Houston, 6431 Fannin, JJL 324, Houston, TX 77030

Abstract

Correspondence concerning this article should be addressed to Thomas F. Northrup, Ph.D., Department of Family and Community Medicine, University of Texas Medical School at Houston, 6431 Fannin, JJL 328, Houston, TX, 77030, USA, Thomas.F.Northrup@uth.tmc.edu, telephone: (713)500-7590, fax: (713)500-7598.

COMPETING INTERESTS

The authors have no competing interests to declare.

Background—Tobacco has regained the status of the world's number two killer behind heart/vascular disease. Thirdhand smoke (**THS**) residue and particles from secondhand smoke (**SHS**) are a suspected health hazard (e.g., DNA damage) that likely contributes to morbidity and mortality, especially in vulnerable children. THS is easily transported and deposited indoors where it persists and exposes individuals for months, creating potential health consequences in seemingly nicotine-free environments, particularly for vulnerable patients. We collected THS data to estimate infant exposure *in the neonatal ICU (NICU)* after visits from household smokers. Infant exposure to nicotine, potentially from THS, was assessed via assays of infant urine.

Methods—Participants were mothers who smoked and had an infant in the NICU (N=5). Participants provided surface nicotine samples of their fingers, infants' crib/incubator, and hospital-provided furniture. Infant urine was analyzed for cotinine, cotinine's major metabolite: *trans*-3′-hydroxycotinine (3HC), and 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol (NNAL), a metabolite of the nicotine-derived and tobacco-specific carcinogen 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK).

Results—Incubators/cribs and other furniture had detectable surface nicotine. Detectable levels of cotinine, 3HC, and NNAL were found in the infants' urine.

Discussion—THS appears to be ubiquitous, even in closely guarded healthcare settings. Future research will address potential health consequences and THS-reduction policies. Ultimately hospital policies and interventions to reduce THS transport and exposure may prove necessary, especially for immunocompromised children.

Keywords

Thirdhand smoke; THS; environmental tobacco smoke; neonatal intensive care unit; NICU

1. INTRODUCTION

Thirdhand smoke (**THS**) results from secondhand smoke (**SHS**) and is a distinct public health hazard.[1,2,3] THS-related harm (compared to SHS) has been predicted at 5%-60%,[4] and THS has been related to cardiovascular and lung disease (e.g., via inflammatory cytokines, implicated in diseases like asthma)[5] and hindered respiratory development in animal models.[6] In-vitro studies have reported DNA damage[7] and impaired wound healing.[8]

THS is difficult to remove,[9,10,11] can persist for ≥18 months,[12] reacts with extant compounds, forming new toxicants and carcinogens,[13,14,15,16] and is reemitted slowly over long time periods well after smoking has ceased.[2,3,11,17] Further, smoking outdoors does not fully protect homes/residents from SHS/THS,[17,18,19] as THS dispersal (e.g., smokers' clothes) and exposure routes (e.g., dermal absorption) are numerous.[10,16,20,21]

Studies find that non-smokers occupying homes vacated by smokers (or staying in non-smoking hotel rooms) had elevated finger nicotine, urine cotinine, and THS-related carcinogens.[10,22] These findings are concerning for premature, low-birth-weight infants exposed to THS. Approximately 50% of infants born <1500 grams will be ventilated in the neonatal ICU (**NICU**) and 22% will develop bronchopulmonary dysplasia (BPD).[23]

Ventilation is life-saving but leads to long-term damage (decreased lung volume)[24] and BPD is associated with increased risk for pneumonia, asthma, repeated hospitalizations, neurodevelopmental problems, and death.[23] Over a quarter of NICU infants are discharged to a home with ≥1 smoker,[18] making this a sizable population at risk for potential THS-related harm. Despite non-smoking policies, SHS levels in hospitals are detectable,[25] as 25–60% of hospitalized smokers and visitors step outside to smoke and then reenter[26,27]. Healthcare provider smoking may contribute as well, as data from 2007 showed 2.3% of physicians, 10.7% of registered nurses, and 19.2% of respiratory therapists smoke or live with a smoker.[28]

This pilot was undertaken to determine whether detectable THS levels (surface nicotine) could be found inside the NICU after smokers visit, which is important as microbes found on NICU surfaces have later been found in premature infants' intestines.[29] Infant-nicotine exposure, potentially from THS, was assessed via infant urine samples. It is plausible that THS/SHS exposure on discharge from the NICU may contribute to infant morbidity and mortality (e.g.,SIDS)[30,31,32]. This study was designed to provide proof of exposure at birth for vulnerable babies.

2. METHODS

Smoking mothers with an infant (N=5) admitted to a NICU, participating in a study to reduce SHS exposure in their homes, were recruited. Research associates obtained IRB-compliant consent. Participants provided a THS (surface nicotine) wipe of their index finger, infants' incubator/crib, and a hospital-provided chair/couch (furniture; **see** Table **Note**) inside infants' NICU rooms. Participants consented to infant urine collection and answered smoking-behavior, breastfeeding, and visitation questions.

THS-surface-nicotine wipes were collected with standardized procedures;[10,17,22,33]. Briefly, a 10cm × 10cm template was taped to the arm of the couch or chair and a screened cotton wipe, doused with a distilled-water and 1%-ascorbic-acid solution was used to wipe inside the template. For cribs, the top railing was measured and wiped. Wipe values were standardized and reported in micrograms per meter squared ($\mu g/m^2$), except for finger wipes and "blanks" (i.e., no sample is taken but all other procedures are followed), which are reported in nanograms (ng/wipe). Surface nicotine was quantified using established methods.[34] The limit-of-detection (**LOD**) for surface nicotine is 0.1 $\mu g/m^2$.[33] Participant 3′s infant's room was sampled twice.

Urine samples (collected on the day of wipe collection) were analyzed for cotinine (nicotine's primary metabolite), 3′-hydroxycotinine (cotinine's primary metabolite; **3HC**), and 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol (NNAL; metabolite of the nicotine-derived and tobacco-specific carcinogen, 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone [**NNK**]).[7] Urine was extracted via syringe from 2 cotton pads placed in the infants' diapers.[35] Published methods were used for quantifying cotinine, 3HC,[36] and NNAL.[37] The limit-of-quantification (**LOQ**) of NNAL is 0.25 picograms (**pg**)/ml; cotinine's LOQ is 0.05 ng/ml; and 3HC's LOQ is 0.1 ng/ml.

3. RESULTS

All 5 infants were admitted to the NICU on their date of birth. All values have been adjusted by subtracting out nicotine found in blanks (*M*=2.6 ng/wipe). All participants reported living with other smokers, that other smokers visited the infant, and allowing smoking inside their homes or cars or both.

There was greater variability across other factors likely to be associated with surface nicotine and urine outcomes (see Table). Participants tended to report light smoking (<10 cigarettes/day)[38,39] and most participants visited daily. Participant 1's, 2's, and 3's infants were hospitalized for ≥3 weeks and all reported discontinuing or never initiating breastfeeding. Participant 4's and 5's infants were hospitalized <2 weeks and both reported current breastfeeding. Participants 2 and 4 did not smoke on the day of measurement and had low finger nicotine levels; whereas, participants 3 and 5 smoked on the collection day and had greater finger nicotine levels.

Surface nicotine levels of all incubators/cribs were similar and within the lower range of surface nicotine found inside smoking households that ban indoor smoking.[17] THS levels on furniture tended to be much higher and were similar to average levels generally observed in smoking households that ban indoor smoking. However, the one repeated furniture measurement taken (participant 3) was substantially higher at the 2nd measurement and suggested a value closer to a home that allows indoor smoking[17] (**see** Supplementary Figure 1).

Data for infant urine cotinine, 3HC, and NNAL were all >LOQs for each respective metric, except participant 4's 3HC. Participants 2, 3, and 4 had highly similar cotinine, 3HC, and NNAL values. Participant 5's infant was still breastfeeding and had greater cotinine, 3HC, and NNAL values (see Table).

4. DISCUSSION

For NICU infants visited by smokers, THS may be transported to and adhere to surfaces in the NICU at levels which are similar to those found in households where smokers reside. Further, this pilot study of NICU infants from smoking households were exposed to measurable levels of nicotine and a known carcinogen (NNK), raising the possibility of exposure due to THS reemission (off-gassing). These findings demonstrate that exposure is taking place in at least one NICU and raises the possibility that such exposure contributes to morbidity and premature mortality in vulnerable babies. Results warrant confirmation and more complete assessment of NICU micro-environments, sources of contamination, and their relationships to home environments to which children are discharged.

A majority of samples had surface nicotine levels above the LOD and one had a level commonly found in households that permit *indoor* smoking. Surface nicotine levels on infants' incubators/cribs tended to be lower than furniture levels. Infants receive a new, thoroughly cleaned incubator every 30 days, which is not true of furniture. Other possibilities for lower levels include increased cleaning attention for cribs/incubators or relatively little time spent at the crib's side in favor of sitting on the furniture. The greater

levels on NICU furniture could suggest that clothing worn by visitors is transferring much of the THS residue.

These data have implications for further research and policies. For example, whether NICU exposures will cause acute or long-term harm is unknown. However, there is no safe level of SHS[40] and whether there is a safe level of THS exposure for immuno-compromised infants is yet to be verified by large, long-term epidemiological studies.

NICUs often require visitors to wash/sanitize their hands, wear protective gowns or gloves, and take other precautions before entering the NICU. We only recorded data on these practices on the day of the assessment, and incomplete use of protective gowns/gloves and handwashing by study participants was observed. Two (of five) participants smoked on the assessment day and only one reported washing their hands since their most recent cigarette. Research staff did not observe any glove use or handwashing. Studies show handwashing policies are not universally enforced (e.g., a review of hand washing in 65 ICUs reported 40% median compliance)[41] and it is unknown whether hand washing or sanitization significantly reduces the amount of nicotine transported. Further research on the effectiveness of these procedures for reducing THS is clearly needed.

This initial, post-hoc study has limitations. For example, the half-life of NNAL in adults is approximately 10–16 days[42] and the half-life for infants (and how long in utero exposure takes to wash out) is unknown. Thus, some (or all) of the infants' NNAL may have come from in utero exposure via the mother. Cotinine has a much shorter half-life of 16–22 hours, which is similar for adults and infants,[43,44] but less understood in premature infants. Further, this small sample is unable to tease out the influence of other variables including previous-room-occupant smoking, staff smoking, visitation frequency/length (including visitation by other household members), and breastfeeding (particularly for 2 participants). Also, we assessed infant rooms where the mother was a smoker (and other smokers visited), which likely have greater levels of nicotine deposits than infants visited by non-smokers. Residual nicotine adhesion and dynamics differ across surface type [e.g., 12,45]. Surface-nicotine variability has been found across settings, including dashboards sampled in rental cars (interquartile range [IQR]: 0.1–3.1 $\mu g/m^2$ [designated-smoking cars]; 0.0–1.2 $\mu g/m^2$ [designated non-smoking cars])[46]; homes (IQR: 0.7–13.7 $\mu g/m^2$)[19]; and, hotels show significant variability based on indoor-smoking-ban policies. For example, non-smoking hotels (IQR: 0.0–3.4 $\mu g/m^2$) have the least surface nicotine, and non-smoking (IQR: 0.0–10.3 $\mu g/m^2$) and smoking rooms (IQR: 7.3–353.2 $\mu g/m^2$) in hotels without complete bans tend to have the greatest surface nicotine. Finally, research should quantify the *cumulative* amount of THS that is absorbed by ongoing contact, as much of the health effect may be due to a relatively large "dose" achieved by cumulative exposure. These data raise questions that require replication with rigorous methodology in larger samples.

5. CONCLUSION

This research highlights THS's pervasiveness, even in closely guarded healthcare settings. Future work is needed to understand exposures and health consequences in such a vulnerable population. Indeed, the death rate among NICU infants is high[18] and the role of

environmental carcinogens is unknown. It may be important to implement hospital policies and interventions to reduce THS exposure, even ahead of causal data given the potential risks for NICU patients. Extending smoke-free policy definitions to include THS could have the added benefit to hasten the elimination of SHS in other environments.[31]

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

The authors wish to thank Maleeha Arshad, Mary MacGregor, Maria Ortiz, Mackenzie Spellman, Jennifer Meeks, Lora Bunge, and Rose Young for their help initiating and conducting the study. Kayo Watanabe and Dana Datuin assisted with the surface nicotine analysis. Additionally, the authors would like to thank the staff of the Children's Memorial Hermann Hospital.

FUNDING

This work was supported by the National Heart, Lung, and Blood Institute (R01 HL107404, PI=A.L. Stotts; R01 HL103684-02, PI=M.F. Hovell) and the National Institute on Drug Abuse (P30 DA012393; PI=Reese T. Jones) at the U.S. National Institutes of Health, and Department of Health and Human Services. This work was also supported by the California Consortium on Thirdhand Smoke, California Tobacco-Related Disease Research Program (20PT-0184; PIs=N.L. Benowitz & P. Jacob) and the California Tobacco-Related Disease Research Program (TRDRP) for "Certifying Smoke-Free Used Cars: Effects on Value and Consumer Behavior" (21RT-0142; PI=G.E. Matt).

References

1. Jung JW, Ju YS, Kang HR. Association between parental smoking behavior and children's respiratory morbidity: 5-year study in an urban city of South Korea. Pediatr Pulmonol. 2012; 47(4): 338–45. [PubMed: 22006579]
2. Singer BC, Hodgson AT, Nazaroff WW. Gas-phase organics in environmental tobacco smoke: 2. Exposure-relevant emission factors and indirect exposures from habitual smoking. Atmos Environ. 2003; 37(39–40):5551–61.
3. Vaughan WM, Hammond SK. Impact of "designated smoking area" policy on nicotine vapor and particle concentrations in a modern office building. J Air Waste Manag Assoc. 1990; 40(7):1012–7.
4. Sleiman M, Logue J, Luo W, et al. Inhalable constituents of thirdhand tobacco smoke: Chemical characterization and health impact considerations. Environ Sci Technol. 2014
5. Martins-Green M, Adhami N, Frankos M, et al. Cigarette smoke toxins deposited on surfaces: Implications for human health. PLoS ONE. 2014; 9(1):e86391. [PubMed: 24489722]
6. Rehan VK, Sakurai R, Torday JS. Thirdhand smoke: A new dimension to the effects of cigarette smoke on the developing lung. Am J Physiol Lung Cell Mol Physiol. 2011; 301(1):L1–8. [PubMed: 21478255]
7. Hang B, Sarker AH, Havel C, et al. Thirdhand smoke causes DNA damage in human cells. Mutagenesis. 2013; 28(4):381–91. [PubMed: 23462851]
8. Prins JM, Wang Y. Quantitative proteomic analysis revealed N′-nitrosonornicotine-induced down-regulation of nonmuscle myosin II and reduced cell migration in cultured human skin fibroblast cells. J Proteome Res. 2013; 12(3):1282–8. [PubMed: 23305604]
9. Dreyfuss JH. Thirdhand smoke identified as potent, enduring carcinogen. CA Cancer J Clin. 2010; 60(4):203–4. [PubMed: 20530799]
10. 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(1):e1–e1. [PubMed: 21037269]

11. Daisey JM, Mahanama KR, Hodgson AT. Toxic volatile organic compounds in simulated environmental tobacco smoke: Emission factors for exposure assessment. J Expo Anal Environ Epidemiol. 1998; 8(3):313–34. [PubMed: 9679214]
12. Bahl V, Jacob P, Havel C, et al. Thirdhand cigarette smoke: Factors affecting exposure and remediation. PLoS ONE. 2014; 9(10):e108258. [PubMed: 25286392]
13. Hoh E, Hunt RN, Quintana PJ, et al. Environmental tobacco smoke as a source of polycyclic aromatic hydrocarbons in settled household dust. Environ Sci Technol. 2012; 46(7):4174–83. [PubMed: 22397504]
14. Matt GE, Quintana PJ, Destaillats H, et al. Thirdhand tobacco smoke: Emerging evidence and arguments for a multidisciplinary research agenda. Environ Health Perspect. 2011; 119(9):1218–26. [PubMed: 21628107]
15. Schick SF, Farraro KF, Perrino C, et al. Thirdhand cigarette smoke in an experimental chamber: Evidence of surface deposition of nicotine, nitrosamines and polycyclic aromatic hydrocarbons and de novo formation of NNK. Tob Control. 2013; 23(2):152–59. [PubMed: 23716171]
16. Sleiman M, Gundel LA, Pankow JF, et al. Formation of carcinogens indoors by surface-mediated reactions of nicotine with nitrous acid, leading to potential thirdhand smoke hazards. Proc Natl Acad Sci U S A. 2010; 107(15):6576–81. [PubMed: 20142504]
17. Matt GE, Quintana PJ, Hovell MF, et al. Households contaminated by environmental tobacco smoke: sources of infant exposures. Tob Control. 2004; 13(1):29–37. [PubMed: 14985592]
18. Stotts AL, Green C, Northrup TF, et al. Feasibility and efficacy of an intervention to reduce secondhand smoke exposure among infants discharged from a neonatal intensive care unit. J Perinatol. 2013; 33(10):811–16. [PubMed: 23619375]
19. Northrup TF, Matt GE, Hovell MF, et al. Thirdhand smoke in the homes of medically fragile children: Assessing the impact of indoor smoking levels and smoking bans. Nicotine Tob Res. 2015:1–9. epub.
20. Becquemin MH, Bertholon JF, Bentayeb M, et al. Third-hand smoking: Indoor measurements of concentration and sizes of cigarette smoke particles after resuspension. Tob Control. 2010; 19(4): 347–8. [PubMed: 20530137]
21. Ferrante G, Simoni M, Cibella F, et al. Third-hand smoke exposure and health hazards in children. Monaldi Arch Chest Dis. 2013; 79(1):38–43. [PubMed: 23741945]
22. 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. 2013; 23(3):264–72. [PubMed: 23669058]
23. Martin JA, Hamilton BE, Sutton PD, et al. Births: Final data from 2003. Natl Vital Stat Rep. 2005; 54:1–116.
24. Koumbourlis AC, Motoyama EK, Mutich RL, et al. Longitudinal follow-up of lung function from childhood to adolescence in prematurely born patients with neonatal chronic lung disease. Pediatr Pulmonol. 1996; 21(1):28–34. [PubMed: 8776263]
25. Fernandez E, Fu M, Martinez C, et al. Secondhand smoke in hospitals of Catalonia (Spain) before and after a comprehensive ban on smoking at the national level. Prev Med. 2008; 47(6):624–8. [PubMed: 18845179]
26. Duffy SA, Scholten RL, Karvonen-Gutierrez CA. The relation of tobacco use during hospitalization to post-discharge smoking cessation among US veterans. Prev Med. 2010; 50(5–6): 285–87. [PubMed: 20122956]
27. Rigotti NA, Arnsten JH, McKool KM, et al. Smoking by patients in a smoke-free hospital: Prevalence, predictors, and implications. Prev Med. 2000; 31(2 Pt 1):159–66. [PubMed: 10938217]
28. Sarna L, Bialous SA, Sinha K, et al. Are health care providers still smoking? Data from the 2003 and 2006/2007 Tobacco Use Supplement-Current Population Surveys. Nicotine Tob Res. 2010; 12(11):1167–71. [PubMed: 20937670]
29. Brooks B, Firek BA, Miller CS, et al. Microbes in the neonatal intensive care unit resemble those found in the gut of premature infants. Microbiome. 2014; 2(1):1. [PubMed: 24468033]

30. Elliot J, Vullermin P, Robinson P. Maternal cigarette smoking is associated with increased inner airway wall thickness in children who die from sudden infant death syndrome. Am J Respir Crit Care Med. 1998; 158(3):802–6. [PubMed: 9731008]

31. Hovell MF, Hughes SC. The behavioral ecology of secondhand smoke exposure: A pathway to complete tobacco control. Nicotine Tob Res. 2009; 11(11):1254–64. [PubMed: 19776346]

32. Ostfeld BM, Esposito L, Perl H, et al. Concurrent Risks in Sudden Infant Death Syndrome. Pediatrics. 2010; 125(3):447–53. [PubMed: 20156907]

33. 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(9):1555–63. [PubMed: 23460657]

34. Anastassiades M, Lehotay SJ, Štajnbaher D, et al. Fast and easy multiresidue method employing acetonitrile extraction/partitioning and “dispersive solid-phase extraction” for the determination of pesticide residues in produce. J AOAC Int. 2003; 86(2):412–31. [PubMed: 12723926]

35. Matt GE, Hovell MF, Quintana PJ, et al. The variability of urinary cotinine levels in young children: Implications for measuring ETS exposure. Nicotine Tob Res. 2007; 9(1):83–92. [PubMed: 17365739]

36. Jacob P, Yu L, Duan M, et al. Determination of the nicotine metabolites cotinine and trans-3′-hydroxycotinine in biologic fluids of smokers and non-smokers using liquid chromatography–tandem mass spectrometry: Biomarkers for tobacco smoke exposure and for phenotyping cytochrome P450 2A6 activity. J Chromatogr B. 2011; 879(3):267–76.

37. Jacob P III, Havel C, Lee D-H, 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(21):8115–21. [PubMed: 18841944]

38. Costello D, Dierker L, Sledjeski E, et al. Confirmatory factor analysis of the Nicotine Dependence Syndrome Scale in an American college sample of light smokers. Nicotine Tob Res. 2007; 9(8): 811–9. [PubMed: 17654294]

39. Husten CG. How should we define light or intermittent smoking? Does it matter? Nicotine Tob Res. 2009; 11(2):111–21. [PubMed: 19246425]

40. United States Department of Health and Human Services. The Health Consequences of Involuntary Exposure to Tobacco Smoke: A Report of the Surgeon General. Washington, DC: 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.

41. Erasmus V, Daha TJ, Brug H, et al. Systematic review of studies on compliance with hand hygiene guidelines in hospital care. Infect Control Hosp Epidemiol. 2010; 31(3):283–94. [PubMed: 20088678]

42. 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 Biomark Prev. 2009; 18(12):3421–25.

43. Benowitz, NL., Hukkanen, J., Jacob, P, III. Nicotine Psychopharmacology. Berlin Heidelberg: Springer; 2009. Nicotine chemistry, metabolism, kinetics and biomarkers; p. 29-60.

44. Dempsey D, Jacob P, Benowitz NL. Nicotine metabolism and elimination kinetics in newborns*. Clin Pharmacol Ther. 2000; 67(5):458–65. [PubMed: 10824624]

45. Daisey JM. Tracers for assessing exposure to environmental tobacco smoke: What are they tracing? Environ Health Perspect. 1999; 107(Suppl 2):319. [PubMed: 10350517]

46. Matt GE, Fortmann AL, Quintana PJ, et al. Towards smoke-free rental cars: An evaluation of voluntary smoking restrictions in California. Tob Control. 2013; 22(3):201–7. [PubMed: 22337558]

WHAT THIS PAPER ADDS

- Thirdhand smoke (THS) contamination is unexplored in non-smoking, protected medical environments visited by smokers.
- THS is estimated to take weeks to months to degrade in controlled environments.
- Infants cared for during extended stays in neonatal intensive care units (NICUs) are protected from secondhand smoke but exposure to THS is unknown.
- This study demonstrated that THS is deposited in rooms of NICU infants visited by smokers.
- Data showed that NICU infants are exposed to nicotine and nicotine-derived, tobacco-specific carcinogens, raising the possibility of exposure due to THS reemission in the NICU.
- These data justify more formal research documenting acute and cumulative THS exposure, sources of exposure, means of prevention, and associations with morbidity/mortality for NICU infants.

Table 1

Participant and Household Characteristics and Surface Nicotine and Urine Data

	Participant					
Characteristic (Measure)	**1**	**2**	**3A**	**3B**	**4**	**5**
Cigarettes Today	NC	0	8	3	0	1
Wash Hands after Last Cigarette	NC	N/A	No	No	N/A	Yes
Wash Hands during Visit	No	No	No	No	No	No
Smoked While Pregnant	No	No	Yes		Yes	No
Typical Cigarettes per Day	5	0[A]	20		5	0[A]
Days of Visitation (out of Last 7)	NC	7	0	1	7	7
Day of Life/Infant Hospitalization[B]	46	22	34	55	9	11
Visitation Minutes (on Study Visit)	60	180	140	90	45	>180[C]
Infant Held at Visit	No	Yes	Yes	Yes	Yes	Yes
Protective Gown Worn at Visit	NC	No	Yes	Yes	No	Yes
Protective Gloves Worn at Visit	NC	No	No	No	No	No
# of Other Household Smokers	2	4	2		2	2
Do Other Household Smokers Visit	NC	Yes	Yes		Yes	Yes
Indoor Home Smoking Allowed	No	Yes	Yes		Yes	No
Smoking Allowed Inside Car	Yes	Yes	No		Yes	Yes
Breastfed Infant in Last 10 days	No	No	No	No	Yes	Yes
Feeding Type at Visit[D]	Bottle	Bottle	Bottle	Bottle	Bottle[D]	Breast
Index Finger and Furniture Data						
Index Finger Nicotine (ng)	NC	44	1160	4,960	90	818
Crib/Incubator (μg/m^2)	<LOD	0.3	0.2	NC	0.2	0.2
Furniture (μg/m^2)	0.3	2.5	5.5	34.2	1.2	3.4
Infant Urine Data						
Cotinine (ng/ml) (LOQ=0.05)	NC	0.17	0.36	NC	0.37	5.01
3HC (ng/ml) (LOQ=0.1)	NC	0.63	0.46	NC	<LOQ	31.58
NNAL (pg/ml) (LOQ=0.25)	NC	0.47	1.64	NC	1.58	12.38

Note. Participant 3's visit 1 is labeled as "3A" and visit 2 is labeled as "3B". Participant 1's crib/incubator result was below the limit-of-detection (LOD; 0.1 μg/m^2). The incubator measurement was not repeated for participant 3's second measurement (i.e., PPT3B). "Cigarettes Today"=Cigarettes smoked on the day of the assessment; "Wash Hands after Last Cigarette"=On the day of the assessment, did the participant report washing their hands after their most recent cigarette; "Wash Hands during Visit"=On the day of the assessment, did research staff observe the participant washing their hands; "N/A"=Not applicable; "Days of Visitation"=Number of days visited out of the last 7 days (not including the day of the visit); "Visitation Minutes (on Study Visit)"=Number of minutes visited on the day of sample collection; "Infant Held at Visit"=Did staff observe the participant holding the infant on the day of the assessment; "Protective Gown (or Gloves) Worn at Visit"=Did the staff observe the participant wearing protective gowning/gloves on the day of the assessment; "NC"=Not collected; "# of Other Household Smokers"=Number of other smokers who live in the household; "Feeding Type at Visit"=Was the infant bottle fed or breastfed on the day of the assessment. "Furniture" = A hospital-provided couch or chair. For couches, the inner material was 100% polyurethane foam and the outer upholstery was made of 90% vinyl and 10% urethane. For chairs, the inner material was 90% polyurethane foam and 10% polyester fiber and the outer upholstery was made of 100% Paloma leather.

*A*Participants 2 and 5 reported smoking fewer than 1 cigarette a day.

*B*All 5 infants were admitted to the NICU on their date of birth.

*C*Participant 5 was rooming with her infant since the infant's admission.

*D*On the day of the assessment, participants 1–3 were bottle fed formula and participant 5 was breastfed. We did not assess whether the fluid in the bottle was expressed breastmilk or formula for participant 4.