Thirdhand smoke: here to stay

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Tob Control 2011 20: 1-3
doi: 10.1136/tc.2010.040279

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The first time I had any inkling that nicotine might linger in a room was when I listened to a group of nicotine chemists complaining about how hard it is to keep nicotine out of their laboratories. Their gas chromatography and mass spectroscopy machines are expensive, complex and very sensitive. In order to detect nicotine and cotinine in samples from non-smokers exposed to secondhand smoke, they must scrupulously exclude nicotine and tobacco smoke from their laboratories. One chemist told a story of how experiments in his laboratory were ruined for weeks after new data cables were installed in the ceiling of the laboratory. Probable culprit: nicotine in the ceiling tiles and the dust above them, dating back 30 years to when people still smoked in laboratories at the university.

Thirdhand smoke is a new concept in the field of tobacco control. While everyone who has ever noticed the lingering smell of stale smoke knows that something stays around after the smoke clears, exactly what that something is, how long it stays and what it means for human health has been little studied to date.

The paper by Matt *et al*[1] in this issue of *Tobacco Control* advances the study of thirdhand smoke by exploring one of the situations most likely to isolate thirdhand smoke exposure from concurrent exposure to secondhand smoke: rental housing. Their findings demonstrate that nicotine persists in homes previously occupied by smokers, and that non-smokers who move into these units have elevated levels of nicotine on their skin and in their bodies. The design of this experiment was very challenging; one can imagine approaching complete strangers who were in the middle of moving house and asking them to let researchers examine their homes and bodies, and the group is to be commended for persuading as many people to participate as they did.

We do not know what the potential health effects of this low-level exposure to thirdhand smoke may be. Nicotine is a toxin that effects development of the nervous system and the lungs, but we don't know if it has effects at concentrations this low. However, nicotine is not the only chemical to consider. Nicotine on indoor surfaces can react with the low levels of oxidant gases that are normally present in homes to form nitrosamines, including 1-(*N*-methyl-*N*-nitrosamino)-1-(3-pyridinyl)-4-butanal (NNA) and 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK).[2] Both of these tobacco-specific nitrosamines are normally found in the particulate phase, which means that once they form on a surface they will tend to stay in place. We do not know whether these nitrosamines, in turn, react and form other compounds, or whether they accumulate over time. If they do accumulate, this could have important implications for the epidemiology of lung cancer. NNK is a lung carcinogen that will cause tumours in the lung whether it is inhaled, injected, or ingested. If concentrations of NNK in rooms where smoking takes place build steadily over time, then this exposure may be partly responsible for the lung cancer seen in smokers and in non-smokers exposed to secondhand smoke. Nicotine can also react to form volatile compounds including formaldehyde.[3] Both formaldehyde and NNK are known human carcinogens for which there is no safe level of exposure.[4 5]

We also do not know what the levels of nicotine and cotinine seen in the study indicate about the level of exposure to the other components of thirdhand smoke. Most studies relating biomarkers of nicotine and nitrosamine exposure have been conducted with smokers. A recent paper by Benowitz *et al* demonstrated that measurement of urinary cotinine can underestimate exposure to tobacco-specific nitrosamines in non-smokers.[6] The ratios observed between 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol (NNAL; a metabolite of the nitrosamine NNK) and cotinine in smokers were between 0.09 and 0.23. The ratios observed between NNAL and cotinine in non-smokers were between 1.10 and 5.50. This means that if urinary cotinine data from smokers is used to estimate exposure to the carcinogen NNK in non-smokers, one could underestimate their exposure by 5–60-fold. I hope that Matt *et al* were able to reserve portions of their samples to test for nitrosamines and nitrosamine metabolites, so we can begin to learn what the relationships are for thirdhand smoke exposure.

We may not yet know whether exposure to thirdhand smoke has negative effects on health, but we do know who will be most exposed to it: poor people. In many countries, the poorer you are, the more likely you are to smoke. In the US, 31.5% of adults with incomes below the federal poverty level smoked, while only 19.6% of those above the poverty level did.[7] Internationally, this trend holds among both men and women of high-income nations and among men in mid-income and most low-income nations.[8] Smoking rates in California are the second lowest in the US at only 13.8%, but in a recent survey of cotinine concentrations in patients admitted to the county hospital in San Francisco, which serves the poor and uninsured, 55% were either smokers or exposed to very high levels of secondhand smoke.[9]

Poor people are also more likely to be exposed to secondhand smoke. In the US, geometric mean urinary cotinine levels in children from families with a poverty level income were over five times higher than those from children from families with incomes four or more times the poverty level.[10] Another recent study of nicotine levels in house dust found that non-smoking households with income below the median income for the study had higher nicotine concentrations in dust than non-smoking households with income above the median.[11]

The effect of this disparity on housing stock at the low end of the price range is obvious. If the smoking rate among renters is 13.8%, then a rental home that has been occupied by five different families has a 36% chance of having been occupied by at least one smoker. If the smoking rate is 25%, then the home has a 75% chance of having been occupied by at least one smoker. The median household income of the families in this study was above the poverty level, but not far enough to allow them free choice of rental housing in San Diego County. The median income of the non-smoking households was between $33 000 and $37 200 and the median income of the smoking households was $25 500. The median household income in San Diego county is $62 820[12] and the median rent for a two-bedroom unit is $1324.[13] If thirdhand smoke is a health hazard, then this exposure may

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be yet another contributor to the existing health disparities between the rich and the poor.

I had my first child in 2007 and my first thought upon reading this research was to imagine how stressful it would be for new parents to know that their home was polluted with nicotine and all the other things that go along with it, and to be unable to afford to move. Babies and toddlers, because of their size, the way they play on the floor, their habit of putting everything in their mouths and the fact that they are growing and developing, are most affected by any pollutant in a home. House dust is believed to be the main route of exposure to indoor pollutants for young children and house dust was one of the main reservoirs for nicotine in the homes Matt *et al* studied.

When I speak publicly about secondhand or thirdhand smoke, I am always asked how to clean a home that smells of smoke. Sadly, the answer there is also: 'we don't know'. All the homes in this study were cleaned before the new families moved in. The homes that were occupied by tenants who smoked were more likely than non-smoking homes to have been painted and have new carpets installed, and they also stood vacant longer before being rented again. None of these methods got rid of all the nicotine. Studies of the dynamics of nicotine in furnished rooms suggest that nicotine sticks to surfaces rapidly and comes off very slowly. Increasing ventilation in a home will not remove the nicotine stuck to surfaces and dust.[14]

A recent study of polycyclic aromatic hydrocarbon pollution in house dust found that the strongest predictor of the concentration of this family of carcinogens in house dust was the age of the house.[15] Older homes had higher levels of polycyclics than new homes. A study of nicotine in house dust by the same group found that the smoking status of occupants for the months and years prior to the study was a stronger predictor of elevated nicotine concentrations than whether smoking was allowed in the house at the time of the study.[11] This study also detected a significant correlation between nicotine concentration and the age of the home. Evidence like this suggests that many different kinds of chemical compounds accumulate in homes and that standard cleaning and maintenance methods do not remove them effectively.

When the first evidence that SHS was hazardous to human health began to emerge in the late 1960s, it was hard for many people, including scientists, to believe that an exposure that was so much less concentrated than active smoking could have any effect. It took at least 20 years of research and public health advocacy for the tide to turn, and some scientists dismissed the significance of SHS until very recently. Yet the evidence is now considered definitive[16] and has given new insight how the human cardiopulmonary system works. The health effects of SHS exposure are, in fact, different from those of active smoking: the majority of smoking-attributable mortality is due to cancer,[17] while the majority of SHS exposure-attributable mortality is due to cardiovascular disease.[18 19] Studying how exposure to even low concentrations of inhaled smoke increases risk of cardiovascular disease has revealed new biological mechanisms and is changing how we view the significance of all kinds of particulate air pollution.[20–22]

The emerging science of thirdhand smoke may reveal equally important information about our exposure to indoor pollutants. Many of the phenomena originally observed in outdoor air pollution are now being discovered indoors. Scientists have known for years that gas phase pollutants in the atmosphere can react to form ultrafine particles.[23] Very recently, research has shown that gas phase chemicals from air fresheners, cleaning products and, (just published this month) nicotine can react with normal gases present indoors to form ultrafine particles.[24–26] Likewise one of the signal discoveries of outdoor environmental pollution—the fact that some pollutants (for example, dichlorodiphenyltrichloroethane (DDT)) persist for years—appears to be emerging in the indoor environment.

The evidence Matt *et al* present suggests that nicotine may persist in indoor environments like some pesticides persist outdoors. Like DDT, the nicotine, polycyclic aromatic hydrocarbons and nitrosamines in cigarette smoke are members of a group of chemicals called semivolatile organic compounds. Semivolatile organic compounds are oily or waxy compounds. There are many other chemicals in this group that are used indoors, including phthalates, bisphenol A and flame retardants. Once released indoors, they are more likely to stick to surfaces than to be removed by ventilation. Once on surfaces, they can desorb slowly back into the air or react to form other chemical compounds.[27] Like nicotine, many other semivolatile organic compounds are also found in our bodies.[28] The best solution we found to the persistence of DDT in the environment was to ban its use except to control insects that cause human disease.

Is the current body of evidence on the composition and persistence of the residue from smoking enough to justify laws banning smoking in multiunit and rental housing? Perhaps not yet, but I think the evidence will come and the laws will come even faster. We don't yet know whether exposure to thirdhand smoke is harmful to human health, but we now know that most of the nicotine from every cigarette smoked indoors stays indoors, where it lingers for months, is taken up by the occupants and also reacts to form nitrosamines, formaldehyde and other harmful chemicals. No one wants that in their home.

Funding Funding was received from the Flight Attendants Medical Research Institute.

Competing interests None.

Contributors This article was written solely by SS.

Provenance and peer review Commissioned; not externally peer reviewed.

Tobacco Control 2011;**20**:1–3.
doi:10.1136/tc.2010.040279

REFERENCES

1. **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*. Published Online First: 30 October 2010 doi:10.1136/tc.2010.037382.
2. **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**:6576–81.
3. **Destaillats H,** Singer BC, Lee SK, *et al*. Effect of ozone on nicotine desorption from model surfaces: evidence for heterogeneous chemistry. *Environ Sci Technol* 2006;**40**:1799–805.
4. **IARC Working Group on the Evaluation of Carcinogenic Risks to Humans,** World Health Organization. Smokeless tobacco and some tobacco-specific N-nitrosamines. In: *IARC Working Group on the Evaluation of Carcinogenic Risks to Humans*. Geneva: World Health Organization, 2004:641.
5. **IARC Working Group on the Evaluation of Carcinogenic Risks to Humans,** World Health Organization. Formaldehyde, 2-butoxyethanol and 1-tert-butoxypropan-2-ol. In: *IARC Working Group on the Evaluation of Carcinogenic Risks to Humans*. Geneva: World Health Organization, 2006:478.
6. **Benowitz NL,** Goniewicz M, Eisner M, *et al*. Urine cotinine underestimates exposure to the tobacco-derived lung carcinogen 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone in passive compared with active smokers. *Cancer Epidemiol Biomarkers Prev* 2010;**19**:2795–800.
7. **US Center for Disease Control.** *Adult Cigarette Smoking in the United States: Current Estimate*. Atlanta, Georgia, US: US Center for Disease Control, 2009. http://www.cdc.gov/tobacco/data_statistics/fact_sheets/adult_data/cig_smoking/index.htm (accessed 20 Oct 2010).

8. **Bobak M,** Jha P, Nguyen S, *et al*. Poverty and smoking. In: Jha P, Chaloupka F, eds. *Tobacco Control in Developing Countries*. Oxford, Britain: Oxford University Press, 2000.
9. **Benowitz NL,** Schultz KE, Haller CA, *et al*. Prevalence of smoking assessed biochemically in an urban public hospital: a rationale for routine cotinine screening. *Am J Epidemiol* 2009;**170**:885—91.
10. **Max W,** Sung HY, Shi Y. Who is exposed to secondhand smoke? Self-reported and serum cotinine measured exposure in the U.S., 1999—2006. *Int J Environ Res Public Health* 2009;**6**:1633—48.
11. **Whitehead T,** Metayer C, Ward MH, *et al*. Is house-dust nicotine a good surrogate for household smoking? *Am J Epidemiol* 2009;**169**:1113—23.
12. **US Census Bureau.** *State and County Quick Facts*. Washington DC: US Census Bureau, 2010. http://quickfacts.census.gov/qfd/states/06/06073.html (accessed 10 Aug 2010).
13. **National Housing Conference.** *Most to Least Expensive Rental Markets in 2008 and 2009*. Washington, DC: National Housing Conference, 2010. http://www.nhc.org/media/files/Rental_Rankings0809.pdf (accessed 10 Aug 2010).
14. **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**:5551—61.
15. **Whitehead T,** Metayer C, Gunier RB, *et al*. Determinants of polycyclic aromatic hydrocarbon levels in house dust. *J Expo Sci Environ Epidemiol*. Published Online First: 30 Dec 2009. doi:10.1038/jes.2009.68.
16. **United States Department of Health and Human Services.** *The Health Consequences of Involuntary Exposure to Tobacco Smoke*. Washington, DC: United States Centers for Disease Control and Prevention, 2006.
17. **U.S. Department of Health and Human Services.** *The Health Consequences of Smoking: A Report of the Surgeon General*. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health, Office on Smoking and Health. Report No.: O2NLM: QV 137 H4347, 2004.
18. **U.S. Department of Health and Human Services.** *The Health Consequences of Involuntary Exposure to Tobacco Smoke: A Report of the Surgeon General—Executive Summary*. Atlanta, GA: U.S. 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. Report No.: O2NLM: WA 754 H4325, 2006.
19. **Adhikari B,** Kahende J, Malarcher A, *et al*. Smoking-attributable mortality, years of potential life lost, and productivity losses—United States, 2000—2004. *MMWR Morb Mortal Wkly Rep* 2008;**57**:1226—8.
20. **Heiss C,** Amabile N, Lee AC, *et al*. Brief secondhand smoke exposure depresses endothelial progenitor cells activity and endothelial function: sustained vascular injury and blunted nitric oxide production. *J Am Coll Cardiol* 2008;**51**:1760—71.
21. **Zanobetti A,** Schwartz J. The effect of fine and coarse particulate air pollution on mortality: a national analysis. *Environ Health Perspect* 2009;**117**:898—903.
22. **Brook RD,** Rajagopalan S, Pope CA 3rd, *et al*. Particulate matter air pollution and cardiovascular disease: An update to the scientific statement from the American Heart Association. *Circulation* 2010;**121**:2331—78.
23. **US Environmental Protection Agency.** *Researchers Study Chemical Reactions that Lead to Particle Pollution*. Washington, DC: US Environmental Protection Agency, 2009. http://www.epa.gov/airscience/pdf/ca-factsheet-atmos-chem.pdf (accessed 2 Nov 2010).
24. **Wainman T,** Zhang J, Weschler CJ, *et al*. Ozone and limonene in indoor air: a source of submicron particle exposure. *Environ Health Perspect* 2000;**108**:1139—45.
25. **Destaillats H,** Lunden MM, Singer BC, *et al*. Indoor secondary pollutants from household product emissions in the presence of ozone: a bench-scale chamber study. *Environ Sci Technol* 2006;**40**:4421—8.
26. **Sleiman M,** Destaillats H, Smith J, *et al*. Secondary organic aerosol formation from ozone-initiated reactions with nicotine and secondhand tobacco smoke. *Atmos Environ* 2010;**44**:4191—8.
27. **Weschler CJ,** Nazaroff WW. Semivolatile organic compounds in indoor environments. *Atmos Environ* 2008;**42**:9018—40.
28. **Department of Health and Human Services, Centers for Disease Control and Prevention.** *Fourth National Report on Human Exposure to Environmental Chemicals*. Atlanta, GA: Centers for Disease Control and Prevention, 2009.