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A Pilot Study of Concurrent Lead and Cotinine Screening for Childhood Tobacco Smoke Exposure: Effect on Parental Smoking

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Abstract

Purpose—To investigate whether a biomarker screening approach for tobacco smoke exposure (TSE) conducted concurrently with lead screening at well-child visits would increase parental smoking cessation and implementation of home smoking restrictions.

Design—Observational, quasi-experimental.

Setting—Pediatric clinic in Minneapolis, Minnesota.

Subjects—Eighty parents who smoked and their children presenting for well-child visits.

Intervention—Children in the intervention group had serum cotinine measured with lead screening. Laboratory results were sent to providers and parents and a counselor proactively contacted parents to offer an eight-session telephone intervention to help parents stop smoking. The comparison group, a historical control, received usual care.

Measures—Parental smoking, engagement in tobacco treatment, and home and car smoking policies 8 weeks later.

Analysis—Mean/standard deviation for continuous data or frequency/percentage for categorical data.

Results—Eighty-four percent of eligible parents agreed to have their child tested for TSE along with lead testing. Measurable cotinine was identified in 93% of children. More parents in the intervention group received tobacco treatment than in the comparison group (74% vs. 0%) and more parents reported 7-day point-prevalent abstinence from smoking at 8 weeks (29% vs. 3%).

Conclusion—These data demonstrate the feasibility of adding cotinine measurement to routine well-child lead screening to document TSE in small children. Data suggest providing this information to parents increases engagement in tobacco treatment and prompts smoking cessation.

Keywords

Tobacco Smoke Exposure; Secondhand Smoke; Smoking Cessation; Lead Screening; Prevention Research; Manuscript format; research; Research purpose; intervention testing; Study design; quasi-experimental; Outcome measure; behavioral; Setting; clinical/health care; Health focus; smoking control; Strategy; education; Target population age; adults and children; Target population circumstances; all education levels; all income levels; all U.S. locations; all races/ethnicities

PURPOSE

Among children, tobacco smoke exposure (TSE) is associated with health problems such as low birth weight, asthma induction, asthma exacerbation, increased ear and lower respiratory infections, and Sudden Infant Death Syndrome.¹ Parental smoking cessation benefits both parent and child, but pediatric providers rarely intervene on parental smoking.² We hypothesized that laboratory documentation of childhood TSE would have an effect on both provider and parental behavior by providing medical evidence of the unseen risk of TSE to children.

National guidelines also recommend that all U.S. children at high risk have blood lead concentrations measured at 1 and 2 years of age.³ Experience with lead testing suggests providers can incorporate the routine screening of children for exposure to environmental toxins into practice, including collecting biological specimens, which suggests the same might be true for TSE testing.

The objective of this pilot study was to examine the feasibility of concurrent lead and cotinine screening during well-child visits with tobacco biomarker feedback to providers and parents. We sought to estimate whether a biomarker screening approach similar to lead

screening would increase detection of children with TSE, enrollment of parents in smoking cessation treatment, participation in treatment, parental cessation, and implementation of home smoking restrictions.

METHODS

Design

We describe provider clinical response and parent smoking behavior following receipt of TSE biomarker feedback, and compare it to a group of parents and providers who did not receive biomarker feedback (a historical comparison group) using parent survey data. Participants were enrolled between September 2010 and June 2011. The study protocol was approved and monitored by the University of Minnesota Institutional Review Board.

Sample

Children coming to clinic for a 12- or 24-month well-child visit were eligible for participation if the parent accompanying the child to the visit was a current cigarette smoker (defined as smoking 10 or more cigarettes daily for the past year), spoke English, and had a telephone. For the intervention group, study staff reviewed clinic schedules to identify children with upcoming 12- or 24-month well-child visits. They telephoned the child's home during the week prior to the visit to screen the parent planning to accompany the child for eligibility. If the parent was interested, research staff met them at the time of the child's visit to answer further questions and obtain informed consent for the parent and the child. For the control group, staff called parents of children noted to have exposure to secondhand smoke in the electronic medical record during the visit prior to the 12- or 24-month visit (9- and 18-month visits, respectively). If the parent smoked at the time of the index visit they were invited to enroll.

Measures

For the intervention group, the data coordinator screened parents by telephone prior to the child's visit and conducted baseline assessments in person at the clinic, including demographics, smoking history, and home and car smoking policy descriptions. Outcome data were collected 8 weeks later by telephone, including receipt of tobacco treatment (defined as consultation with a health care provider specifically for tobacco treatment, enrollment in a tobacco treatment program, consultation with a quitline, or receipt of tobacco dependence treatment medications), quit attempts, 7-day point prevalent abstinence, and current home and car smoking policies. For the historic comparison group, the data coordinator contacted parents 2 to 6 months after their index visit and collected identical data elements. During the single call the parent described his or her smoking status at the time of the index visit, provider activities related to tobacco use, participation in tobacco treatment, and smoking and home policy status 8 weeks later.

Intervention

A finger-stick blood sample for lead and hemoglobin was part of routine well-child visit procedures at 12- and 24-month visits in the pediatric clinic. An additional 0.5 mL of blood was collected and analyzed for cotinine, a metabolite of nicotine. Total cotinine

concentration in plasma was quantified by liquid chromatography tandem mass spectrometry analysis as previously described.⁴

Clinic providers asked that a masters-prepared certified tobacco treatment specialist execute a protocol to reach all parents whose children had measurable cotinine levels. Providers wanted to avoid taking on the task of making individual referrals themselves, as that might increase provider workload and could potentially lead to failures to refer.

Results were mailed to parents in a form letter modeled on typical laboratory result correspondence 1 to 3 weeks after the visit. The letter included an explanation that cotinine came from tobacco exposure and that the normal value was zero (absent). One week later the tobacco counselor telephoned the parent to explain the laboratory result; to describe potential sources of TSE, including thirdhand smoke⁵; and to convey what is known about the potential health effects of TSE for his or her child. The counselor used a combination of motivational interviewing and cognitive behavioral therapy to engage the parent in a quit-smoking attempt. All parents were encouraged to institute a strict home and car no-smoking policy, regardless of whether they wanted to stop smoking. The counselor attempted at least weekly contact for up to 8 weeks to continue engaging the parent and embarked on an evidence-based telephone smoking cessation protocol⁶ if the parent wanted to stop smoking. The tobacco treatment specialist described prescription and over-the-counter medication options for smoking cessation to parents, but the study did not provide medications or funding to obtain them. If parents requested help finding sources of medications, the specialist worked to facilitate access.

Analysis

Descriptive statistics are presented as mean and standard deviation for continuous data or frequency and percentage for categorical data. Statistical analysis was conducted using SAS 9.1.3 (SAS Institute Inc., Cary, North Carolina) statistical software.

RESULTS

Of 661 children with upcoming 12- and 24-month visits who were assessed for eligibility, 49 (7%) had parents who were smokers. Among these parents, 41 of 49 (84%) agreed to have their child tested for TSE along with lead testing. One child failed to have blood drawn, yielding 40 child-parent pairs in the intervention group.

Parent demographics are shown in Table 1. There were no statistically significant differences between the intervention group and comparison group on baseline characteristics. Measurable cotinine was identified in 37 of 40 children (93%). Assessments were completed in 38 of 40 participants in the intervention group. Of the 38, 36 received at least one counselor call, 25 received two calls, and 14 received three or more calls. The majority of parents, 28 of 38 (74%), engaged in smoking counseling, and 9 of 38 (24%) accessed smoking cessation medications. Eight weeks after the index visit, 11 of 38 parents in the intervention group (29%) reported 7-day point-prevalent abstinence. In contrast, only one parent in the comparison group reported abstinence from smoking ($p = .001$). There were fewer quit attempts (mean .9 vs. .2, $p = .001$) and less readiness to quit (mean

Contemplation Ladder score 6.6 vs. 4.7, $p < .001$) in the comparison group (Table 2). All parents described low rates of receipt of tobacco treatment from the child's doctor. There was little change in household or car rules about smoking 8 weeks after the index visit, but parents reported a high rate of total restriction at baseline (Table 2).

DISCUSSION

Summary

Results from our pilot study show that it is feasible to conduct concurrent screening for lead and TSE at well-child visits. The majority of parents who smoked agreed to have their child provide a blood sample at the time of lead screening to test for tobacco exposure. Pediatric providers quickly instituted a clinic system that virtually guaranteed parents of all children who had a cotinine value indicating TSE would receive smoking intervention. Nearly all parents thought they protected their children from TSE, but cotinine values indicated this perceived protection was incomplete. The rate of engagement in telephone treatment, which was optional, was high. Nearly one-third of parents who received the biomarker intervention reported short-term quitting.

Other studies that used children's biomarkers to address parental smoking show varying efficacy,⁷⁻¹¹ but knowledge of TSE effects and the social environment for smoking has changed radically in the 15-year period over which these studies took place; it is possible that biomarker feedback may be more potent in the current social climate than in the past.

Limitations

Limitations to this observational study include the lack of random assignment to the intervention or comparison groups and the inclusion of multiple components in the intervention. It is possible that parents who chose to participate in the intervention group were more inclined to stop smoking than smokers in the general population. Because the comparison group was recruited using a different method than the intervention group, there is potential for selection bias. Data were ascertained historically in the comparison group, and there may have been errors in recall. Finally, smoking status was not biochemically confirmed, so it is possible that parents overreported abstinence.

Significance

A clinic system that routinely couples cotinine screening with lead screening at 12 and 24 months could take advantage of the highly developed infrastructure for lead screening and remediation. Biomarker screening with cotinine is an accurate way to detect childhood TSE and potentially could be implemented for all children when lead screening occurs, detecting TSE among parents who deny smoking and also among parents who report that they do not smoke around children. Clinics could establish protocols so that positive cotinine results or parent self-report of tobacco use would result in proactive counseling about the impact on children.

Biomarkers of TSE were detected in virtually all the children of self-reported smoking parents, even those parents who thought they were completely protecting children from

TSE. These data strongly suggest that parental reports of their strategies to limit or eliminate child exposure to TSE are inaccurate proxy measures of actual exposure. Parental smoking cessation appears to be essential to meet the goal of eliminating exposure to TSE. Finding clinical settings to engage parents in smoking cessation treatment has been a challenge^{12–15} but biomarker feedback of their own child's TSE may motivate parents to quit, promote engagement, and increase parental cessation.

Acknowledgments

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References

1. US 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.
2. Winickoff JP, Tanski SE, McMillen RC, et al. Child health care clinicians' use of medications to help parents quit smoking: a national parent survey. *Pediatrics*. 2005; 115:1013–1017. [PubMed: 15805379]
3. American Academy of Pediatrics Committee on Environmental Health. Lead exposure in children: prevention, detection, and management. *Pediatrics*. 2005; 116:1036–1046. [PubMed: 16199720]
4. Bloom J, Hinrichs AL, Wang JC, et al. The contribution of common CYP2A6 alleles to variation in nicotine metabolism among European-Americans. *Pharmacogenet Genomics*. 2011; 21:403–416. [PubMed: 21597399]
5. Matt GE, Quintana PJ, Destailats H, et al. Thirdhand tobacco smoke: emerging evidence and arguments for a multidisciplinary research agenda. *Environ Health Perspect*. 2011; 119:1218–1226. [PubMed: 21628107]
6. Stead LF, Perera R, Lancaster T. Telephone counselling for smoking cessation. *Cochrane Database Syst Rev*. 2006; (3):CD002850. [PubMed: 16855992]
7. Wilson SR, Yamada EG, Sudhakar R, et al. A controlled trial of an environmental tobacco smoke reduction intervention in low-income children with asthma. *Chest*. 2001; 120:1709–1722. [PubMed: 11713157]
8. Woodruff SI, Conway TL, Edwards CC, Hovell MF. Acceptability and validity of hair collection from Latino children to assess exposure to environmental tobacco smoke. *Nicotine Tob Res*. 2003; 5:375–385. [PubMed: 12791534]
9. McIntosh NA, Clark NM, Howatt WF. Reducing tobacco smoke in the environment of the child with asthma: a cotinine-assisted, minimal-contact intervention. *J Asthma*. 1994; 31:453–462. [PubMed: 7961322]
10. Conway TL, Woodruff SI, Edwards CC, et al. Intervention to reduce environmental tobacco smoke exposure in Latino children: null effects on hair biomarkers and parent reports. *Tob Control*. 2004; 13:90–92. [PubMed: 14985605]
11. Chilmonczyk BA, Palomaki GE, Knight GJ, et al. An unsuccessful cotinine-assisted intervention strategy to reduce environmental tobacco smoke exposure during infancy. *Am J Dis Child*. 1992; 146:357–360. [PubMed: 1543187]
12. Winickoff JP, Berkowitz AB, Brooks K, et al. State-of-the-art interventions for office-based parental tobacco control. *Pediatrics*. 2005; 115:750–760. [PubMed: 15741382]
13. Winickoff JP, Park ER, Hipple BJ, et al. Clinical effort against secondhand smoke exposure: development of framework and intervention. *Pediatrics*. 2008; 122:e363–e375. [PubMed: 18676523]

14. Rosen LJ, Noach MB, Winickoff JP, Hovell MF. Parental smoking cessation to protect young children: a systematic review and meta-analysis. *Pediatrics*. 2012; 129:141–152. [PubMed: 22201152]
15. Winickoff JP, Hillis VJ, Palfrey JS, et al. A smoking cessation intervention for parents of children who are hospitalized for respiratory illness: the stop tobacco outreach program. *Pediatrics*. 2003; 111:140–145. [PubMed: 12509567]

SO WHAT? Implications for Health Promotion Practitioners and Researchers

What is already known on this topic?

Tobacco smoke exposure is an important, but often undetected, childhood health risk. Pediatric treatment of parental smoking would benefit both parent and child. Screening for lead exposure during well-child visits provides an opportunity to screen for biomarkers of tobacco exposure.

What does this article add?

This study shows a biomarker screening approach that is similar to lead screening during well-child visits increases detection of children exposed to tobacco smoke. Results suggest biomarker documentation of tobacco exposure increases parent tobacco treatment and prompts smoking cessation.

What are the implications for health promotion practice or research?

The addition of routine screening for tobacco smoke exposure may help reduce this hidden cause of disease in childhood. In addition to protecting children from tobacco smoke exposure, this laboratory test may help support parental tobacco dependence treatment.

Table 1

Index Visit Characteristics*

	Intervention (n = 40)	Comparison (n = 40)
Parent		
Sex, %		
Female	22 (55.0)	23 (57.5)
Male	18 (45.0)	17 (42.5)
Age, mean (SD), y	30.3 (5.7)	30.5 (6.4)
Race, %		
White	22 (55.0)	18 (46.1)
Nonwhite	18 (45.0)	21 (53.9)
Employment status, No. (%)		
Unemployed	16 (40.0)	19 (47.5)
Part-time/full-time	24 (60.0)	21 (52.5)
Individual income, No. (%), \$		
0-49,999	34 (87.2)	38 (97.4)
50,000 or more	5 (12.8)	1 (2.6)
Highest grade level completed, No. (%)		
High school	17 (42.5)	23 (57.5)
Some college	15 (37.5)	12 (30)
College +	8 (20.0)	5 (12.5)
Child		
Sex, No. (%)		
Female	21 (52.5)	21 (52.5)
Male	19 (47.5)	19 (47.5)
Visit age, No. (%), mo		
12	20 (50.0)	19 (47.5)
24	20 (50.0)	21 (52.5)
Serum cotinine corrected		
No. detectable (%)	37 (92.5)	N/A
Mean (SD), ng/mL	1.8 (2.5)	
Median, ng/mL	0.7	
Range, ng/mL	0.1-12.2	

* N/A indicates not applicable.

Table 2**Baseline and 8-Week Postvisit Smoking and Smoking Policies***

	Baseline		8-wk Post-visit	
	Intervention n = 40	Comparison n = 40	Intervention n = 38 [†]	Comparison n = 40
No smoking at all in the last 7 d, No. (%)	N/A	N/A	11 (28.9)	1 (2.5)
No. quit attempts in past 8 wk, mean (SD)	N/A	N/A	0.9 (1.8)	0.2 (0.4)
Readiness to quit, mean (SD) [†]	4.8 (1.5)	4.5 (1.1)	6.6 (2.4)	4.7 (1.4)
Which of the following best describes your household's rules about smoking?, No. (%)				
Smoking is allowed in all parts of the home	2 (5.0)	2 (5.0)	0 (0)	2 (5.0)
Smoking is allowed in some parts of the home	11 (27.5)	7 (17.5)	5 (13.2)	6 (15.0)
Smoking is not allowed in any part of the home	27 (67.5)	31 (77.5)	33 (86.8)	32 (80.0)
In your home, is smoking in the presence of children always allowed, sometimes allowed, or never allowed?, No. (%)				
Always is allowed	0 (0)	1 (2.5)	0 (0)	1 (2.5)
Sometimes is allowed	7 (17.5)	2 (5.0)	4 (10.5)	2 (5.0)
Never is allowed	33 (82.5)	37 (92.5)	34 (89.5)	37 (92.5)
Please tell me which best describes how cigarette smoking is handled in your car when children are present?, No. (%)				
No one is allowed to smoke in my car	31 (77.5)	33 (82.5)	34 (89.5)	33 (82.5)
Only special guests can smoke in my car	0 (0)	0 (0)	0 (0)	0 (0)
People can smoke in car if windows are open	7 (17.5)	6 (15.0)	2 (5.3)	6 (15.0)
People can smoke in my car at any time	2 (5.0)	1 (2.5)	2 (5.3)	1 (2.5)
Since your enrollment, has your child's doctor discussed any of the following tobacco treatments with you?, No. (%) yes				
Consultation with a health care provider specifically for tobacco treatment		N/A	3 (7.9)	5 (12.5)
Enrollment in a tobacco treatment program			2 (5.3)	0 (0)
Consultation with a telephone quitline			1 (2.6)	0 (0)
Receipt of prescription or over-the-counter tobacco dependence treatment medications			0 (0)	0 (0)
Since the time of your enrollment, have you engaged in any of the following tobacco treatments?, No. (%) yes				
Consultation with a health care provider specifically for tobacco treatment		N/A	4 (10.5)	0 (0)
Enrollment in a tobacco treatment program			2 (5.3)	0 (0)
Consultation with a telephone quitline			28 (73.7)	0 (0)
Receipt of prescription or over-the-counter tobacco dependence treatment medications			9 (23.7)	2 (5.0)

* N/A indicates not applicable.

[†]Two participants in the intervention group were lost to follow-up.