Research Article

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Tobacco Smoke Exposure in Nonsmoking Hospitality Workers before and after a State Smoking Ban

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Abstract

Secondhand smoke exposure is estimated to account for 3,000 cancer deaths per year. Although several countries and states in the United States have passed comprehensive smoke-free laws to protect all employees, a significant number of workers are still not protected. The purpose of this study was to determine the effects of passing a comprehensive smoking ban that included bars and restaurants on biomarkers of nicotine and carcinogen exposure. The urines of nonsmoking employees ($n = 24$) of bars and restaurants that allowed smoking before the smoke-free law were analyzed before and after the law was passed in Minnesota. The results showed significant reductions in both total cotinine and 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol (free plus glucuronidated) after the ban was instituted. These results provide further support for the importance of protecting employees working in all venues. *Cancer Epidemiol Biomarkers Prev; 19(4); 1016–21. ©2010 AACR.*

Introduction

Exposure to secondhand smoke (SHS) has been consistently identified as a public health hazard and cause of disease (1). The U.S. Environmental Protection Agency (EPA) classified SHS as a group A carcinogen (2) and the International Agency for Research on Cancer (IARC) also classified SHS as carcinogenic to humans (3). SHS exposure has been associated with the increased risks for lung cancer (1, 3), coronary heart disease (1), and possibly respiratory disease (1). The health risks of SHS are no longer in dispute, leading to a number of local ordinances and state legislative initiatives that have restricted smoking in public places. Due to these smoking bans, cotinine levels have decreased approximately 50% to 70% in nonsmokers as observed in state and national surveys (4, 5). Although causality cannot be inferred, a cross-sectional study has shown a dose-response relationship between exposure to SHS, as measured by cotinine, and extensiveness of clean indoor air acts in subject's county of residence (6). As of August 2009, 27 states had implemented smoke-free workplace policies that include restaurants and bars (7).

Even with an overall increase in restrictions in smoking in public places, a significant number of smoke-free workplace policies still do not include hospitality venues such as bars, restaurants, and casinos. Establishment owners have proposed exemptions to these laws claiming economic hardship if these venues were to become smoke free. Unfortunately, these exemptions have resulted in greater SHS exposure among some hospitality workers. For example, bartenders have a 2- to 4-fold increase in SHS exposure compared with table waiting staff (8). Siegel and Skeer (9) found that workers in these venues had three to four times greater excess risk of lung cancer mortality than those exposed to the "typical de manifestis" levels used to determine obligatory regulation of a hazardous worksite. Similarly, in the Boston pubs tested by Repace et al. (10), before a smoking ban, indoor air quality did not meet the Occupational Health and Safety Significant Standards for environmental exposure. According to the EPA Air Quality Index, levels of outdoor fine particulate matter at several of these pubs averaged in the "very unhealthy" category. These findings on SHS exposure indicate that hospitality workers may be at an increased health risk in the absence of comprehensive indoor air policies.

The enactment of comprehensive smoke-free workplace laws in some U.S. states as well as in other countries has provided an opportunity to examine the effect of these laws on tobacco toxicant exposure among workers in these venues. The results from these studies should allay the questions about positive health benefits from comprehensive bans. These studies show significant reductions in air levels of small particulate matter (11-18), particulate polycyclic aromatic hydrocarbons (10, 19), nicotine (20), and benzene (11) after enactment of smoking bans. Similarly, countries that have instituted nationwide laws prohibiting indoor smoking in public places showed significantly lower levels of small particulate matter in these venues than countries without such bans (21). Additionally, nonsmoking workers had a significant reduction in levels

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of cotinine, a metabolite of nicotine (4, 11-15, 20, 22-24), or nicotine levels in hair (25). To date, no study has examined employee exposure to both nicotine and 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone, a potent tobacco-specific lung carcinogen found in tobacco smoke, before and after the implementation of a comprehensive ban. Such an opportunity occurred when Minnesota enacted a comprehensive statewide law prohibiting smoking in virtually all indoor workplaces, including bars and restaurants, beginning October 1, 2007.

Materials and Methods

The study was submitted to and approved by the University of Minnesota's Institutional Review Board: Committee on the Use of Human Subjects.

Eligible subjects were nonsmokers who worked in bars, restaurants, or bowling alleys where smoking was allowed before October 1, 2007 when the statewide smoke-free law took effect. Subjects were recruited through local tobacco control leaders. They were recruited from the following Minnesota communities: Thief River Falls, Duluth, St. Cloud, Red Wing, and Moorhead. Recruitment of subjects was done by local tobacco control leaders, who contacted nonsmoking bar, restaurant, and bowling alley employees who reported work exposure to tobacco smoke. The local recruiters informed the potential participant of the opportunity to participate in a study with the University of Minnesota and ClearWay Minnesota[SM] (an independent, nonprofit organization funded by the state's 1998 tobacco settlement with the tobacco industry). They were told that this study would investigate the health effects of the upcoming statewide smoke-free law. Interested potential subjects were instructed to telephone the University of Minnesota's Tobacco Use Research Center, at which time they provided oral consent to be screened over the phone for study eligibility.

Potential participants who reported they were currently not using tobacco or nicotine products and had not used them for the last 6 mo, lived in a nonsmoking household, and were employed in a hospitality venue where they were exposed to SHS for shifts of 6 or more hours were informed of the study procedures. Interested subjects were mailed a packet that included a letter describing the study, a consent form, and a Demographics and Smoke Exposure Environment form with questions about typical work hours and extent of tobacco smoke exposure at work, in the home and when socializing. The questionnaire also included questions on their employment venues, size of smoking and nonsmoking sections, and their employer's policy on smoking. Subjects mailed the consent form and the Smoke Exposure questionnaire back to the Tobacco Use Research Center in a self-addressed, stamped envelope. Subjects received a urine cup in the packet for the sample collection that was to occur within the 2 wk before the law going into effect on October 1, 2007. A Sample Day Questionnaire was to be completed the morning the urine sample was collected. This questionnaire asked about the number of hours worked the day preceding the sample collection, occupancy at the hospitality venue, and the estimated percentage of patrons who smoked during the subjects' shift. Subjects were instructed to collect the urine sample from their first void in the morning, after they had completed at least a 6 h shift the day before. The urine samples and the Sample Day Questionnaire were returned to the local contact where the sample was frozen until shipment. The samples were sent to the University of Minnesota on ice in thermal coolers through overnight shipment.

Urine cups and the second Sample Day Questionnaire were mailed out to the subjects to obtain the post–October 1 sample. Samples were collected 4 to 8 wk after the law went into effect. The post–October 1 sample was also frozen and shipped by the local contact to the University of Minnesota. Subjects were compensated $100 for providing both urine samples.

Analysis was done by the University of Minnesota Transdisciplinary Tobacco Use Research Center Biomarkers Core laboratory. Samples were analyzed for creatinine and urinary metabolites of 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone, 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol (NNAL) and its glucuronides (NNAL-Glucs), or total NNAL, as previously described (26). In addition, samples were analyzed for total cotinine (cotinine plus cotinine-*N*-glucuronide) as previously described (27). The biomarker values are presented as both unadjusted and adjusted for creatinine to correct for urine volume.

Summary statistics included number and percentage of subjects for categorical variables and the mean, SD, median, range, and 95% confidence intervals for continuous variables. For statistical analysis, total NNAL levels below the detection limit were given a value of 0.0025 pmol per milligram of creatinine, whereas those for total cotinine were assigned a value of 1.0 nanogram per milligram of creatinine, both of which are approximately half the detection limit. Due to a skewed distribution to high values, both total cotinine and total NNAL were transformed to the natural logarithmic scale. The difference of the log-transformed biomarkers before and after the smoking ban is reported as the corrected geometric mean and confidence interval using Cox's method (28). These mean values represent the estimated average ratio of the measurements before and after the ban. The differences in total NNAL and total cotinine levels before and after the smoking ban were evaluated with the two-sided Wilcoxon signed-rank test.

The Spearman correlation measured the association between total NNAL and total cotinine levels and exposure to SHS at work. P values of <0.05 were considered statistically significant. All analyses were done using SAS version 9.1 (SAS Institute, Inc.).

Results

Thirty-one subjects provided oral consent by telephone and 24 returned the written consent form, completed the

Table 1. SHS exposure before and after the smoking ban

Amount of SHS exposure	Before smoking ban	After smoking ban
Duration of shift, h (mean ± SD)	8.0 ± 2.1 (range, 6-15)	7.7 ± 2.4 (range, 4-15)
Time in smoking areas, h (mean ± SD)	7.2 ± 2.9 (range, 1-15)	—
Smoke exposure outside of work (no. of subjects)	4	1
Smoke exposure outside of work, h (mean ± SD)	1.9 ± 2.8 (range, 0-6)*	0.25

*The extents of exposure were 15, 35 min, 1, and 6 h for the four individuals.

additional questionnaires, and returned both urine samples. All subjects were Caucasian and 15 were female. They worked in a bar and grill ($n = 7$), bar ($n = 5$), restaurant ($n = 6$), bowling alley ($n = 5$), and one subject did not provide information on their hospitality workplace. They averaged 29.6 years of age (range, 18-58) and worked an average of 3.9 (SD, 1.4) shifts per week and 6.9 (SD, 1.3) hours per shift. The mean percent of the hospitality venues considered as a smoking section was 52.2% (SD, 35.5%; range, 10-100%). The mean number of hours spent in the smoking section before the ban was estimated to be about 7.2 (SD, 2.9; range, 1-15 hours). Nineteen subjects indicated that they were around smokers most or all of the time at work and four indicated exposure to SHS outside of work some of the time ($n = 3$) or very often ($n = 1$). None or very little exposure to SHS outside of the work environment was reported by 20 of the participants.

Table 1 shows the extent of SHS exposure at the workplace and outside the workplace just before the time of urine collection before and after the statewide smoke-free law went into effect.

Table 2 shows the data for each subject. Although one subject (#14) had a seemingly high total cotinine level, the total NNAL level was consistent for an SHS-exposed nonsmoker; therefore, his data were retained in the analysis. One subject (#24) had total NNAL and total cotinine levels that were higher than would be expected for a nonsmoker. The data were analyzed with and without this data point, and because no differences were found in the analyses, the subject's data were retained.

Nineteen of 24 workers (79%) showed at least a 50% reduction in total cotinine and 13 (54%) showed at least a 50% reduction in total NNAL. One subject had levels at the limit of detection for total cotinine before and after the ban; two subjects had levels that were at the limit of detection before the ban and slightly higher after the ban; and one subject had slightly higher levels after the ban when the value was adjusted for creatinine. For total NNAL, the respective numbers of subjects in these categories were 6, 2 and 1, respectively.

Table 3 shows the median percent decreases in total cotinine and total NNAL after the ban, geometric means for the before the ban/after the ban ratios of total NNAL and total cotinine, and the median differences in these biomarkers before and after the ban. Significant reductions were seen for all measures ($P \leq 0.001$). Levels of total NNAL (pmol/mg creatine) were significantly correlated with number of hours worked in the smoking section ($r = 0.43$; $P < 0.05$), but total cotinine (ng/mg creatinine) was not ($r = 0.20$; $P > 0.30$).

Discussion

The results of this study are consistent with others that we have conducted, examining SHS exposure levels in patrons and workers in restaurants and bars (29-31). In one of the prior studies, total NNAL and total cotinine were measured in workers at bars and restaurants that allowed smoking (30). Twenty-four hour urine collections were obtained during and after their work day and during a nonwork day. The results showed that for total cotinine the median difference was 7.5 ng/mL and the mean difference was 11.6 ng/mL on work days compared with nonwork days. The values were 0.025 and 0.033 pmol/mL, respectively, for total NNAL. In another study, total NNAL and cotinine levels of 32 nonsmoking workers in bars and restaurants that prohibited smoking were compared with 52 nonsmoking employees of bars and restaurants where smoking was allowed (31). The employees of restaurants and bars where smoking was permitted were significantly more likely to have detectable levels of urinary total NNAL as well as urinary total cotinine and to experience thrice greater increase in levels of total NNAL and 10 times greater increase in total cotinine levels compared with workers in similar venues that did not allow smoking.

It is notable that eight of the subjects in the current study did not have detectable levels of total NNAL before the ban. Five of these eight subjects were employees of two restaurants. The low levels of total NNAL may have been a function of the level of exposure on the day that they collected urine samples; perhaps they had higher exposure on other days. The 57% to 77% reductions in total NNAL levels is consistent with studies that have shown reductions in particulate matter or respirable suspended particles that range from 68% to 99%, with the majority of studies showing >80% reductions (10-17, 19, 32). The percent reduction in total NNAL exceeds the 30% coefficient of variation observed across repeated total NNAL measurements in smokers, according to our

Table 2. Preban and postban urinary total cotinine and total NNAL levels in nonsmoking hospitality workers

ID	When	Total cotinine		Total NNAL	
		ng/mL*	ng/mg creatinine†	pmol/mL*	pmol/mg creatinine†
1	Preban	26	11.4	LOD	LOD
	Postban	5	3.2	LOD	LOD
2	Preban	39	23.9	0.168	0.103
	Postban	5	8.6	0.026	0.0448
3	Preban	18	9.1	0.035	0.0179
	Postban	6	4.7	LOD	LOD
4	Preban	12	21.4	LOD	LOD
	Postban	2	2.9	0.021	0.0304
5	Preban	25	18.7	0.097	0.0726
	Postban	3	2.2	LOD	LOD
6	Preban	26	10.1	0.049	0.0192
	Postban	LOD	LOD	LOD	LOD
7	Preban	26	12.0	0.084	0.0387
	Postban	10	12.5	0.041	0.0516
8	Preban	7	6.1	0.018	0.0157
	Postban	LOD	LOD	LOD	LOD
9	Preban	42	25.9	0.127	0.0782
	Postban	7	3.9	0.019	0.0104
10	Preban	LOD	LOD	0.008	0.0034
	Postban	3	Not available	LOD	LOD
11	Preban	66	58.4	0.219	0.194
	Postban	LOD	LOD	LOD	LOD
12	Preban	8	8.1	0.055	0.0553
	Postban	2	1.2	LOD	LOD
13	Preban	LOD	LOD	LOD	LOD
	Postban	5	2.2	LOD	LOD
14	Preban	390	500	0.069	0.0888
	Postban	9	20.0	0.022	0.0489
15	Preban	18	14.3	0.104	0.0828
	Postban	LOD	LOD	LOD	LOD
16	Preban	4	3.3	LOD	LOD
	Postban	LOD	LOD	0.017	0.0120
17	Preban	LOD	LOD	LOD	LOD
	Postban	LOD	LOD	LOD	LOD
18	Preban	6	5.1	LOD	LOD
	Postban	LOD	LOD	LOD	LOD
19	Preban	3	5.5	0.050	0.0909
	Postban	LOD	LOD	LOD	LOD
20	Preban	9	6.8	0.038	0.0288
	Postban	LOD	LOD	LOD	LOD
21	Preban	5	6.9	LOD	LOD
	Postban	2	2.3	LOD	LOD
22	Preban	7	5.3	0.092	0.0692
	Postban	LOD	LOD	LOD	LOD
23	Preban	15	20.5	LOD	LOD
	Postban	LOD	LOD	LOD	LOD
24	Preban	1,820	1,456	0.763	0.611
	Postban	651	455	0.509	0.356

*Unadjusted for creatinine.

†Adjusted for creatinine; total cotinine limit of detection (LOD), 1.0 ng per mg creatinine; total NNAL LOD, 0.0025 pmol per mg creatinine.

Table 3. Change in total cotinine and total NNAL from before to after the smoking ban (n = 24)

Biomarker	Median percent decrease after the ban	Geometric mean of before/after (95% CI)	Median difference (before minus after)	P (before minus after)
Total cotinine ng/mL*	83.3%	12.3 (5.7-26.6)	11.0	<0.001
Total cotinine ng/mg creatinine†	78.6%	9.3 (5.1-16.9)	6.9	<0.001
Total NNAL pmol/mL*	76.6%	21.4 (6.2-73.7)	0.039	<0.001
Total NNAL pmol/mg creatinine†	56.5%	19.8 (5.4-72.8)	0.018	0.001

Abbreviation: CI, confidence interval.
*Unadjusted for creatinine.
†Adjusted for creatinine; total cotinine LOD, 1.0 ng per mg creatinine; total NNAL LOD, 0.0025 pmol per mg creatinine.

unpublished data, but no similar data are available for nonsmokers exposed to SHS.

Unlike total NNAL, the majority of subjects did have detectable levels of total cotinine in their urine before the ban. Prior studies using cotinine as an outcome measure showed a reduction in levels preban and postban that ranged from 43% to 95% with a mean reduction of ~76% (4, 11-15, 20, 22-24, 33). These observations are in line with the ~ 80% reduction observed in total cotinine in this study and these findings are consistent with the 83% and 98% reduction observed in air nicotine concentrations observed before and after bans (15, 20). Similar to total NNAL, the coefficient of variation observed across repeated total cotinine measurements in smokers is ~30%, according to our unpublished data, but no data are available for nonsmokers exposed to SHS.

The levels of 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone and nicotine exposure reported here are significantly less than found among smokers. However, in spite of these low exposure levels, SHS is believed to contribute to 3,000 lung cancer deaths and >35,000 coronary heart disease deaths per year in the United States (1) and can negatively affect the health outcomes of individuals who already have a disease (34).

With accumulating findings such as those observed in this study, there is increasingly less challenge to the concept that reducing tobacco smoke in hospitality venues will reduce employee exposure to tobacco toxicants and ultimately reduce health risk. Consistent with significant reductions in exposures, smoke-free bans have resulted in a rapid effect on reduction in hospital admissions for acute myocardial infarctions in the community (35-37) as well as decreased respiratory symptoms and increased pulmonary function in bartenders (13, 24, 38) or other hospitality workers (23) and improved systemic inflammatory markers (24). Not only do these laws provide health benefits to employees and patrons, smoke-free workplace bans provide benefits to other groups. Rates of smoking initiation after bans are reduced in adolescents (39). A review of 26 studies before and after smoking bans estimated that bans resulted in a reduction in the prevalence of smoking by 3.8%, reduction in the number of cigarettes per day by 3.1, and an estimated drop in U.S. consumption of 4.5% (40). In addition, smokers are more likely to make a quit attempt and be successful (41, 42). Studies also show minimal economic problems associated with bans (43).

A limitation of this study is that the subjects were self selected. They were recruited by local tobacco control leaders who support and in some cases work to advance comprehensive smoke-free workplace policies. It is possible that a participant may have altered his or her behavior to influence the study outcome. However, our results are consistent with other published studies on restaurant and bar workers showing decreased levels of cotinine and other tobacco related exposure biomarkers after smoking bans went into effect (11, 12, 20). Additional limitations included the lack of sensitivity in our analytic methods to adequately determine the concentrations of NNAL in all workers before the ban and only a single measurement point before and after the ban.

In summary, it is critical for states and communities to continue to support strict restrictions on smoking in workplaces to ensure that all employees, including those in the hospitality industry, are guaranteed a safe work environment, free of exposures to carcinogens and toxicants that enhance the risk of cancer, cardiovascular, and pulmonary disease.

Disclosure of Potential Conflicts of Interest

Dorothy Hatsukami is conducting a clinical trial supported by Nabi Biopharmaceuticals. The other authors disclosed no potential conflicts of interest.

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References

1. U.S. Department of Health & Human Services. The health consequences of involuntary exposure to tobacco smoke: A report of the Surgeon General. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, Coordination Center for Health Promotion, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health; 2006.
2. Office of Health and Environmental Assessment EPAE. Respiratory health effects of passive smoking: lung cancer and other disorders. Washington, DC: EPA; 1992.
3. International Agency for Research on Cancer. Tobacco smoke and involuntary smoking. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans 83. Lyon, France: IARC; 2004.
4. Centers for Disease Control and Prevention. Reduced secondhand smoke exposure after implementation of a comprehensive statewide smoking ban—New York, June 26, 2003-June 30, 2004. MMWR Morb Mortal Wkly Rep 2007;56:705–8.
5. Pirkle JL, Bernert JT, Caudill SP, Sosnoff CS, Pechacek TF. Trends in the exposure of nonsmokers in the U.S. population to secondhand smoke: 1988-2002. Environ Health Perspect 2006;114:853–8.
6. Pickett MS, Schober SE, Brody DJ, Curtin LR, Giovino GA. Smoke-free laws and secondhand smoke exposure in US non-smoking adults, 1999-2002. Tob Control 2006;15:302–7.
7. Centers for Disease Control and Prevention. State smoking restrictions for private-sector worksites, restaurants, and bars-United States, 2004 and 2007. MMWR Morb Mortal Wkly Rep 2008;57:549–52.
8. Maskarinec MP, Jenkins RA, Counts RW, Dindal AB. Determination of exposure to environmental tobacco smoke in restaurant and tavern workers in one US city. J Expo Anal Environ Epidemiol 2000;10:36–49.
9. Siegel M, Skeer M. Exposure to secondhand smoke and excess lung cancer mortality risk among workers in the "5 B's": bars, bowling alleys, billiard halls, betting establishments and bingo parlours. Tob Control 2003;12:333–8.
10. Repace JL, Hyde JN, Brugge D. Air pollution in Boston bars before and after a smoking ban. BMC Public Health 2006;6:266.
11. Goodman P, Agnew M, McCaffrey M, Paul G, Clancy L. Effects of the Irish smoking ban on respiratory health of bar workers and air quality in Dublin pubs. Am J Respir Crit Care Med 2007;175:840–5.
12. Semple S, Maccalman L, Naji AA, et al. Bar workers' exposure to second-hand smoke: the effect of Scottish smoke-free legislation on occupational exposure. Ann Occup Hyg 2007;51:571–80.
13. Allwright S, Paul G, Greiner B, et al. Legislation for smoke-free workplaces and health of bar workers in Ireland: before and after study. BMJ 2005;331:1117.
14. Valente P, Forastiere F, Bacosi A, et al. Exposure to fine and ultrafine particles from secondhand smoke in public places before and after the smoking ban, Italy 2005. Tob Control 2007;16:312–7.
15. Ellingsen DG, Fladseth G, Daae HL, et al. Airborne exposure and biological monitoring of bar and restaurant workers before and after the introduction of a smoking ban. J Environ Monit 2006;8:362–8.
16. Alpert HR, Carpenter CM, Travers MJ, Connolly GN. Environmental and economic evaluation of the Massachusetts Smoke-Free Workplace Law. J Community Health 2007;32:269–81.
17. Waring MS, Siegel JA. An evaluation of the indoor air quality in bars before and after a smoking ban in Austin, Texas. J Expo Sci Environ Epidemiol 2007;17:260–8.
18. Travers MJ, Cummings KM, Hyland A, et al. Indoor air quality in hospitality venues before and after implementation of a clean indoor air law. MMWR Morb Mortal Wkly Rep 2003;53:1038–41.
19. Repace J. Respirable particles and carcinogens in the air of Delaware hospitality venues before and after a smoking ban. J Occup Environ Med 2004;46:887–905.
20. Mulcahy M, Evans DS, Hammond SK, Repace JL, Byrne M. Secondhand smoke exposure and risk following the Irish smoking ban: an assessment of salivary cotinine concentrations in hotel workers and air nicotine levels in bars. Tob Control 2005;14:384–8.
21. Hyland A, Travers MJ, Dresler C, Higbee C, Cummings KM. A 32-country comparison of tobacco smoke derived particle levels in indoor public places. Tob Control 2008;17:159–65.
22. Abrams SM, Mahoney MC, Hyland A, et al. Early evidence on the effectiveness of clean indoor air legislation in New York State. Am J Public Health 2006;96:296–8.
23. Farrelly MC, Nonnemaker JM, Chou R, et al. Changes in hospitality workers' exposure to secondhand smoke following the implementation of New York's smoke-free law. Tob Control 2005;14:236–41.
24. Menzies D, Nair A, Williamson PA, et al. Respiratory symptoms, pulmonary function, and markers of inflammation among bar workers before and after a legislative ban on smoking in public places. JAMA 2006;296:1742–8.
25. Hahn EJ, Rayens MK, York N, et al. Effects of a smoke-free law on hair nicotine and respiratory symptoms of restaurant and bar workers. J Occup Environ Med 2006;48:906–13.
26. Carmella SG, Han S, Fristad A, Yang Y, Hecht SS. Analysis of total 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol (NNAL) in human urine. Cancer Epidemiol Biomarkers Prev 2003;12:1257–61.
27. 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.
28. Zhou XH, Gao S. Confidence intervals for the log-normal mean. Stat Med 1997;16:783–90.
29. Anderson KE, Kliris J, Murphy L, et al. Metabolites of a tobacco-specific lung carcinogen in nonsmoking casino patrons. Cancer Epidemiol Biomarkers Prev 2003;12:1544–6.
30. Tulunay OE, Hecht SS, Carmella SG, et al. Urinary metabolites of a tobacco-specific lung carcinogen in nonsmoking hospitality workers. Cancer Epidemiol Biomarkers Prev 2005;14:1283–6.
31. Stark MJ, Rohde K, Maher JE, et al. The impact of clean indoor air exemptions and preemption policies on the prevalence of a tobacco-specific lung carcinogen among nonsmoking bar and restaurant workers. Am J Public Health 2007;97:1457–63.
32. Ott W, Switzer P, Robinson J. Particle concentrations inside a tavern before and after prohibition of smoking: evaluating the performance of an indoor air quality model. J Air Waste Manage 1996;46:1120–34.
33. Fernando D, Fowles J, Woodward A, et al. Legislation reduces exposure to second-hand tobacco smoke in New Zealand bars by about 90%. Tob Control 2007;16.
34. Eisner MD, Jacob P III, Benowitz NL, Balmes J, Blanc PD. Longer term exposure to secondhand smoke and health outcomes in COPD: Impact of urine 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol. Nicotine Tob Res 2009;11:945–53.
35. Sargent RP, Shepard RM, Glantz SA. Reduced incidence of admissions for myocardial infarction associated with public smoking ban: before and after study. BMJ 2004;328:977–80.
36. Barone-Adesi F, Vizzini L, Merletti F, Richiardi L. Short-term effects of Italian smoking regulation on rates of hospital admission for acute myocardial infarction. Eur Heart J 2006;27:2468–72.
37. Pell JP, Haw S, Cobbe S, et al. Smoke-free legislation and hospitalizations for acute coronary syndrome. N Engl J Med 2008;359:482–91.
38. Eisner MD, Smith AK, Blanc PD. Bartenders' respiratory health after establishment of smoke-free bars and taverns. JAMA 1998;280:1909–14.
39. Farkas AJ, Gilpin EA, White MM, Pierce JP. Association between household and workplace smoking restrictions and adolescent smoking. JAMA 2000;284:717–22.
40. Fichtenberg CM, Glantz SA. Effect of smoke-free workplaces on smoking behaviour: systematic review. BMJ 2002;325:188.
41. Farkas AJ, Gilpin EA, Distefan JM, Pierce JP. The effects of household and workplace smoking restrictions on quitting behaviours. Tob Control 1999;8:261–5.
42. Longo DR, Johnson JC, Kruse RL, Brownson RC, Hewett JE. A prospective investigation of the impact of smoking bans on tobacco cessation and relapse. Tob Control 2001;10:267–72.
43. Eriksen MP, Cerak RL. The diffusion and impact of clean indoor air laws. Annu Rev Public Health 2008;29:171–85.