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

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Objectives. We studied the impact of clean indoor air law exemptions and preemption policies on the prevalence of a tobacco-specific lung carcinogen—4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK)—among nonsmoking bar and restaurant workers.

Methods. We collected urine specimens from 32 nonsmoking bar and restaurant workers from communities in Oregon where smoking is prohibited in bars and restaurants, and from 52 participants from communities in Oregon where smoking is allowed. Urine specimens collected before and after a workshift were analyzed for 3 NNK metabolites and reported as total 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol (NNAL). Urinalysis results from participants protected from workplace secondhand smoke were compared with results from participants who were exposed to it.

Results. Participants exposed to workplace secondhand smoke were more likely to have any detectable level of NNAL ($P = .005$) and higher mean levels of NNAL ($P < .001$) compared with nonexposed participants. Increased levels of NNAL were also associated with hours of a single workplace exposure ($P = .005$).

Conclusions. Nonsmoking employees left unprotected from workplace secondhand smoke exposure had elevated levels of a tobacco-specific carcinogen in their bodies. All workers—including bar and restaurant workers—should be protected from indoor workplace exposure to cancer-causing secondhand smoke. (*Am J Public Health.* 2007;97:1457–1463. doi:10.2105/AJPH.2006.094086)

Epidemiological studies have shown that exposure to secondhand smoke among nonsmokers increases their risk of lung cancer, heart disease, and asthma, perinatal complications such as sudden infant death syndrome and low birthweight, and other chronic and acute diseases.[1–7] Research has also shown that nonsmoking workers exposed to workplace secondhand smoke are at elevated risk for these diseases.[2,5,7–11] This evidence of increase in disease risk among nonsmokers exposed in the workplace has led to the passage of clean indoor air acts that ban smoking in indoor work environments. Such laws now protect a large majority of workers from indoor secondhand smoke[12,13] and have the added benefit of facilitating smoking cessation among smokers in workplaces where smoking has been prohibited.[14–17]

In spite of the progress made in protecting workers from secondhand smoke exposure, at the time of this study, only 11 states had comprehensive clean indoor air acts that banned smoking in all indoor workplaces.[18] In the other 39 states, clean indoor air acts exempt certain workplaces, especially bars and restaurants.[19,20] As a result of the exemptions, millions of food service workers are at elevated risk of secondhand smoke exposure.[12,21,22] Smoky bars and restaurants also create the impression that smoking is an acceptable behavior,[23] especially among young people who frequent these types of establishments.

In the absence of statewide clean indoor air acts that include bars and restaurants, the tobacco control community adopted a strategy to protect nonsmoking workers by passing local comprehensive clean indoor air ordinances.[24–26] Not only do these local ordinances protect workers in their jurisdictions, but enactment of a substantial number of local ordinances in a state also can facilitate the passage of statewide comprehensive clean indoor air laws. Indeed, in California, the first state to totally ban smoking in restaurants and bars, the statewide law followed the enactment of hundreds of local ordinances.

The tobacco industry responded to the tobacco control community's strategy of passing of local ordinances that restrict smoking in public places by using its influence to promote passage of state-level preemption laws that eliminate local jurisdictions' authority to regulate tobacco.[24,25,27,28] As of 2004, 19 states had at least 1 preemptive provision in their clean indoor air legislation, and the Centers for Disease Control and Prevention's (CDC) assessment is that since 1999, almost no progress had been made toward the 2010 goal[29] of eliminating all preemptive state smoke-free indoor air laws.[30]

Oregon is one state that currently has both a preemptive provision and exemptions in its clean indoor air legislation. Oregon's statewide comprehensive Tobacco Prevention and Education Program began in 1997 with dedicated funds from a voter-mandated tobacco tax increase. In accordance with guidance from the CDC,[22,31] Oregon's Tobacco Prevention and Education Program funded local (county-level) coalitions to create smoke-free environments, including support for local clean indoor air ordinances that had no exemptions. Indeed, beginning in 1997, several Oregon cities passed local clean indoor air ordinances that had no exemptions. The passage of these local ordinances and the indication that more local ordinances without exemptions were forthcoming

led to the enactment of a statewide clean indoor air law in 2001. This law included exemptions for bars and restaurants with areas posted "off limits" to minors and preemptive provision that prohibited passage of more stringent local clean indoor air ordinances. When preemption was legislated, however, previously enacted local ordinances that prohibited smoking in all indoor workplaces, including all bars and restaurants, were permitted to remain in place. The fact that some nonsmoking food service workers in Oregon are protected from secondhand smoke by local ordinance, while others cannot be protected because of clean indoor air exemptions and preemption, provides an opportunity to assess the extent to which these policies create a health disparity among the unprotected nonsmoking workers.

To test for this possible disparity, we examined the prevalence of metabolites of 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK) among nonsmoking food service workers in Oregon communities where such workers were either protected or not protected from secondhand smoke. A potent carcinogen, NNK has an important role in the induction of lung cancer in smokers.[32–35] In rodents, NNK has been shown to induce adenocarcinoma of the lung,[34,36,37] the same type of tumor most prevalent among nonsmokers exposed to secondhand smoke.[38,39] The presence of NNK and its biomarkers in the human body is specific to tobacco use or tobacco smoke exposure.[35,38,40,41] Therefore, its presence cannot be attributed to other factors.[34,35,38]

A number of studies have documented the urinary biomarkers for NNK: 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol (NNAL) and its glucuronides (NNAL-*O*-Gluc and NNAL-*N*-Gluc), which demonstrate NNK uptake and metabolism in nonsmokers exposed to secondhand smoke. Specifically, increases in levels of NNAL have been detected among (1) nonsmoking subjects experimentally exposed to secondhand smoke[42]; (2) nonsmoking female spouses of smokers[38]; (3) nonsmoking children exposed in homes and cars[41]; (4) nonsmoking patrons exposed during a 4-hour casino visit[43]; (5) nonsmoking hospital workers who performed some of their duties in areas where patients smoke[44]; and (6) nonsmoking food service workers exposed to workplace secondhand smoke.[45] In the latter study, there were significant increases in total NNAL on working days compared with nonworking days, which strongly suggests that workplace exposure to secondhand smoke increases NNK levels among nonsmoking workers.

The 2 studies of NNK among nonsmokers exposed in the workplace[44,45] employed relatively small sample sizes ($n<21$) and have not shown that workplace exposure to secondhand smoke increases the proportion of workers with NNK metabolite levels above the limit of detection. In addition, no studies have assessed increases in NNK levels within a single workshift exposure. Our study's sample size and analytic approach allowed us to address both issues. We hypothesized that (1) those participants working in establishments where smoking is allowed would be more likely to have any detectable level and higher levels of NNAL in their urine, compared with those workers protected from workplace secondhand smoke by local ordinances and (2) among those exposed to secondhand smoke at work, levels of total urinary NNAL would rise between the beginning and end of a workshift. In addition to analyzing participants' pre- and postworkshift urine samples for total NNAL, we supplemented the NNAL analyses with tests for cotinine and nicotine, as previous research has shown that levels of these tobacco metabolites increase with workplace exposure.[22,46–50]

METHODS

Recruitment, Enrollment, and Data Collection

Data were collected from November 2004 through August 2005. Participants were recruited through advertisements in local newspapers, through flyers, and by word of mouth. The recruitment materials indicated that participants (1) must be nonsmokers employed in either bars or restaurants anywhere in Oregon where smoking is allowed and practiced or in a community in Oregon where smoking is prohibited by local ordinance (i.e., Eugene, Corvallis, or Philomath), (2) must provide a pre- and postworkshift urine sample, and (3) would receive a $50 incentive.

Once potential participants called study staff, they received a description of the study and were screened for eligibility. Participants were considered eligible if they reported (1) being either never smokers or former smokers who had not smoked, even a puff, within 6 months prior to enrollment, (2) having no history of using any other form of tobacco or any nicotine product in the past 6 months, and (3) being in good health.

Eligible participants provided informed consent and received a brief (10- to 15-minute) telephone survey that covered demographic information, smoking history, and worksite smoking practices. After the telephone survey was completed, we mailed a urine sample collection kit to the participant that included instructions to provide a urine sample within an hour before and after the targeted workshift. At this time participants were told that they would need to provide the urine samples for analysis of tobacco by-products only, as well as proof of employment at their specified workplace, and a breath sample that would be tested for smoking before final study enrollment.

Prior to meeting participants to gather the urine samples, project staff visited each workplace to determine whether there was evidence of indoor smoking. All staff observations confirmed participants' reports of their worksites' smoking practices.

When project staff met participants to collect the urine samples, they tested the participants for alveolar carbon monoxide with a Vitalograph–BreathCO Monitor, model 29.700 (Vitalograph Inc, Lenexa, Kan). Those whose carbon monoxide levels were greater than 8 ppm were to have been excluded; however, none had a reading more than 4 ppm. Staff then ascertained participants' nonworksite secondhand smoke exposure in the 7 days prior to the targeted workshift and the number of hours they had worked during the workshift. Those with more than 2 hours of self-reported nonworkplace secondhand smoke exposure or less than 4 hours worked during the targeted workshift were excluded.

Collected samples were brought to the Oregon State Public Health Laboratory, and frozen aliquots (30 mL) of urine samples were batched and mailed to the University of Minnesota Cancer Center laboratory for

chemical analysis. The mailed samples were blind to participant name, worksite, and pre–post workshift status.

Urine Analysis

Urine specimens were tested for the presence of NNAL, a metabolite of NNK, and for cotinine and nicotine. We report results as total NNAL (the sum of the concentrations of NNAL, NNAL-*O*-Gluc, and NNAL-*N*-Gluc), total cotinine (the sum of the concentrations of cotinine and cotinine-*N*-Gluc), and total nicotine (the sum of the concentrations of nicotine and nicotine glucuronides). Chemical analysis of total cotinine and total nicotine at the University of Minnesota's Cancer Center Laboratories was performed using gas chromatography and mass spectrometry as described previously[51]; analysis of total NNAL, also performed at the Cancer Center Laboratories, was carried out using gas chromatography of the trimethylsilyl ether derivative of NNAL, as described previously.[52]

Exposure to Secondhand Smoke Measures

We measured workplace secondhand smoke exposure by asking participants for the number of hours they worked during the targeted workshift. For those participants exposed to workplace secondhand smoke, duration of exposure was set equal to zero for the preworkshift urine sample and was set equal to the hours of the workshift for the postworkshift sample; for those participants protected from workplace secondhand smoke, duration of exposure to secondhand smoke was set to zero for both the pre- and postworkshift urine samples. For nonworkplace secondhand smoke exposure, we asked participants about the places and amount of time they were exposed to secondhand smoke outside work (i.e., in the home, in vehicles, at other worksites, and during leisure time away from home and work) in each of the 7 days prior to the targeted workshift. These data were summed and coded as total minutes of nonworkplace secondhand smoke exposure.

Statistical Methods

Statistical analyses were conducted with SPSS version 11.5 (SPSS Inc, Chicago, Ill) with a .05 level of significance for statistical tests. We first compared demographic characteristics of participants exposed to workplace secondhand smoke and those protected from workplace secondhand smoke with the Fisher exact test for dichotomous variables and the Welch *t* tests for continuous variables.

Next, we fitted 3 different regression models with all the participants. First, we fitted a logistic regression model to determine whether being exposed to workplace secondhand smoke was associated with having any detectable level of total urinary NNAL at postworkshift (the dependent variable). Second, we fitted a linear regression model to determine whether workplace secondhand smoke exposure status was associated with postworkshift level of total NNAL. For this model, we used the natural log of total NNAL as the dependent variable because the distributions were highly skewed. The model coefficients were then back-transformed to estimate the multiplicative increase in NNAL levels between exposed and unexposed workers. Third, we fitted a linear mixed model to determine whether length of exposure to secondhand smoke in a single workshift was associated with changes in level of total NNAL. For this determination, we used 2 measures of total NNAL for each participant: one from the preworkshift urine sample and the other from the postworkshift sample. Again in this model, we used the natural log of total NNAL as the dependent variable. Model coefficients were back-transformed to estimate the multiplicative increase in NNAL levels for each hour of workplace secondhand smoke exposure. We also included a random participant-by-intercept term in this model. In both the linear regression and mixed models, a value of one half the limit of detection was assigned to samples with nondetectable NNAL.

In addition, we conducted the same 3 analyses with measures of nicotine or cotinine as the dependent variables, which were also highly skewed. In all of the models (NNAL, nicotine, and cotinine), we adjusted for participant's age, gender, and number of minutes exposed to secondhand smoke outside the workplace in the week prior to the targeted workshift.

RESULTS

Among the 163 people who volunteered to participate, 60 did not meet the initial eligibility requirements, 5 subsequently either chose not to participate or did not come to the face-to-face meeting, 2 had preworkshift cotinine levels higher than 100 ng/mL and were deemed to be smokers, 3 worked less than 4 hours during the targeted workshift, and 9 reported more than 2 hours of nonworkplace exposure in the week preceding the targeted workshift. Thus, 84 individuals comprised the final sample: 32 participants from 22 worksites located in Eugene, Corvallis, and Philomath where smoking in bars and restaurants is prohibited by local ordinance, and 52 participants from 39 worksites located in the remainder of Oregon where smoking is allowed in bars and restaurants.

The study participants tended to be women, aged 18 to 29 years, with household incomes less than $25 000 per year (Table 1). More than one third did not have health insurance coverage. The most frequently mentioned work roles were servers (47.6%) or bartenders (40.5%). Protected participants had significantly lower incomes and were somewhat more likely to work as servers or in other roles (e.g., cooks, bouncers) compared with the exposed workers. On average, participants' targeted workshift lasted slightly more than 7 hours, and they reported slightly more than 14 minutes of nonworkplace secondhand smoke exposure in the 7 days prior to the workshift.

Being exposed to workplace secondhand smoke was significantly associated with having a detectable level of total urinary NNAL (Table 2). In fact, those exposed to workplace secondhand smoke had almost 6 times the odds of having a detectable urine level of total NNAL, compared with protected workers (adjusted odds ratio [OR]=5.66; P=.005). In addition, being exposed to workplace secondhand smoke was strongly associated with having any detectable level of nicotine (adjusted OR=109.01; P<.001) and cotinine (adjusted OR=95.21; P<.001) in the urine.

With multiple linear regression, we found that being exposed to workplace secondhand smoke was significantly associated with almost a 3-times greater increase (adjusted

TABLE 1—Characteristics of Nonsmoking Food Service Workers, by Workplace Secondhand Smoke Exposure (Exposed vs Protected): Oregon, November 2004–August 2005

	Full Sample (n = 84)	Exposed (n = 52)	Protected (n = 32)	P[a]
Age, y, %				.129
18–29	58.3	51.9	68.8	
30–39	20.2	26.9	9.4	
40–49	8.3	5.8	12.5	
50–59	13.1	15.4	9.4	
Gender, %				.191
Women	66.7	71.2	59.4	
Men	33.3	28.8	40.6	
Household income, %				.008
< $15 000	27.4	13.5	50.0	
$15 000–$24 999	35.7	44.2	21.9	
$25 000–$34 999	19.0	23.1	12.5	
$35 000–$49 999	7.1	7.7	6.3	
≥ $50 000	10.7	11.5	9.4	
Health insurance, %				.183
No coverage	35.7	40.4	28.1	
Coverage	64.3	59.6	71.9	
Occupation,[b] %				
Server	47.6	40.4	59.4	.071
Bartender	40.5	48.1	28.1	.056
Other[c]	26.2	19.2	37.5	.057
Mean length of shift, h (SD)	7.3 (2.1)	7.3 (1.8)	7.3 (2.7)	.976
Mean nonwork secondhand smoke exposure, min (SD)	14.3 (33.3)	17.2 (37.4)	9.7 (25.3)	.275

[a]*P* values derived from the Fisher exact test (for dichotomous variables) and the Welch *t* tests (for continuous variables) that compared those exposed and those protected from workplace secondhand smoke exposure.
[b]Occupation categories are not mutually exclusive; participants were asked to list all workplace functions.
[c]Other category includes busperson, cook, seating host, karaoke host, manager, disc jockey, dancer, and bouncer.

increase=2.85; $P<.001$) in the level of total urinary NNAL (Table 3). Exposure was also significantly associated with large increases in levels of total urinary nicotine (adjusted increase=15.12; $P<.001$) and cotinine (adjusted increase=10.52; $P<.001$).

Further, we found that duration of exposure to secondhand smoke in a single workshift was significantly associated with the level of total urinary NNAL (Table 4). Each hour of exposure was associated with about a 6% increase in total NNAL (adjusted increase=1.06; $P=.005$). In addition, each hour of exposure was associated with about a 33% increase in level of total nicotine (adjusted increase=1.33; $P<.001$) and a 12% increase in total cotinine (adjusted increase=1.12; $P<.001$).

DISCUSSION

We found that workplace exposure to secondhand smoke was highly associated with elevated levels of urinary NNAL, a biomarker for the potent tobacco-specific lung carcinogen NNK. Whereas more than 3 out of 4 exposed workers had a detectable level of NNAL, fewer than half of the unexposed workers had a detectable level. Exposed workers also had higher levels of NNAL, and their levels increased by about 6% for every hour they worked in an establishment where smoking was allowed. These findings are consistent with earlier studies that showed uptake of NNAL among nonsmokers exposed to secondhand smoke in various settings,[38,41,44] as well as those showing a pre–post increase in NNAL after exposure in casinos[43] and in bars and restaurants.[45] Our results extend these findings by documenting significant differences in any detectable level of NNAL between exposed and nonexposed nonsmoking workers and by estimating the hourly impact of workplace secondhand smoke exposure on levels of NNAL.

Food service workers have more exposure to indoor secondhand smoke than workers in any other occupation[12,22,29,53] and suffer serious health consequences because of this disparity.[8,20,54] Further, this disparity is greatest among young women who are generally overrepresented among food service workers.[55] In addition to the broader risks associated with secondhand smoke exposure, these women have increased risk of breast cancer and perinatal complications such as low birthweight, sudden infant death syndrome, and preterm delivery.[6] Our study's participants had relatively low incomes, as is the case with food service workers nationally,[21] and more than one third lacked health insurance. This vulnerable population suffers a health disparity that could be reduced by elimination of clean indoor air exemptions and preemption.

Our study is limited because participants from communities with clean indoor air exemptions may be exposed to more nonworkplace secondhand smoke if they spend leisure time in local bars and restaurants where smoking takes place. In addition, the measure of all participants' exposure to secondhand smoke outside the workplace was based on self-report. To limit the impact of this potential bias, we confined the sample to persons who reported 2 hours or less of nonworkplace secondhand smoke exposure in the past week and controlled for minutes of nonworkplace exposure in all analyses. Further, our pre–post workshift results, which showed increases in urinary levels of NNAL, cotinine, and nicotine that were directly proportional to reported hours of workplace exposure, give us confidence that the levels of NNAL reported in this study do, indeed, reflect workplace exposure.

Another potential limitation is that, despite little recent reported exposure to secondhand smoke, a fairly large proportion (45%) of protected workers had any detectable level of NNAL. However, this finding is consistent

TABLE 2—Associations Between Exposure to Workplace Secondhand Smoke and Any Detectable Level of Total NNAL, Nicotine, and Cotinine in the Postworkshift Urine of Nonsmoking Food Service Workers; Oregon, November 2004–August 2005

	No.[a]	% Having Any Detectable Level[b]	Adjusted OR (95% CI)[c]
Total NNAL			
Protected workers (reference)	31	45.2	1.00
Exposed Workers	50	76.0	5.66 (1.67, 19.14)*
Total nicotine			
Protected workers (reference)	32	9.4	1.00
Exposed workers	52	90.4	109.01 (20.42, 581.77)**
Total cotinine			
Protected workers (reference)	32	18.8	1.00
Exposed workers	52	92.3	95.21 (15.97, 567.61)**

Notes. NNAL = 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol; OR = odds ratio; CI = confidence interval.
[a]Total NNAL (pmol/mL) could not be determined in 3 participants' urine samples.
[b]Detection limits: cotinine, 2 ng/mL; nicotine, 2 ng/mL; NNAL, 0.007–0.01 pmol/mL. In nonsmokers, the half-life for nicotine is 2 hours,[56] for cotinine is 16.9 hours,[56] and for NNAL is unknown. In smokers, the half-life for nicotine is 2.6 hours,[56] for cotinine is 17.5 hours,[56] and for NNAL is 3–4 days for the distribution phase and 40–45 days for the elimination phase.[51]
[c]Odds ratios were based on logistic regression and adjusted for participant age, gender, and minutes exposed to secondhand smoke outside the workplace in the past week.
*$P < .01$; **$P < .001$.

TABLE 3—Associations Between Exposure to Workplace Secondhand Smoke and Level of Total NNAL, Nicotine, and Cotinine in the Postworkshift Urine of Nonsmoking Food Service Workers: Oregon, November 2004–August 2005

	No.[a]	Range of Levels (Untransformed)[b]	Mean Level (SD) (Untransformed)	Multiplicative Increase (95% CI)[c]
Total NNAL (pmol/mL)				
Protected workers (reference)	31	0.01–0.18	0.02 (0.03)	1.00
Exposed workers	50	0.01–0.31	0.04 (0.05)	2.85[d] (1.77, 4.60)**
Total nicotine (ng/mL)				
Protected workers (reference)	32	1.00–7.22	1.39 (1.33)	1.00
Exposed workers	52	1.00–319.00	44.36 (61.25)	15.12 (8.37, 27.33)**
Total cotinine (ng/mL)				
Protected workers (reference)	32	1.00–5.35	1.4 (0.99)	1.00
Exposed workers	52	1.00–72.80	20.20 (18.27)	10.52 (6.90, 16.04)**

Notes. NNAL = 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol; SD = standard deviation; CI = confidence interval.
[a]Total NNAL (pmol/mL) could not be determined in 3 participants' urine samples.
[b]A value of half the limit of detection was used for nondetectable values. Limit of detection: cotinine, 2 ng/mL; nicotine, 2 ng/mL; NNAL, 0.007–0.01 pmol/mL depending on recovery.
[c]By exposure status. Multiplicative increase was based on linear regression and adjusted for participant age, gender, and exposure to secondhand smoke outside the workplace.
[d]Interpretation: Being exposed to workplace secondhand smoke was significantly associated with an almost 300 percent increase in the level of total urinary NNAL.
**$P < .001$.

TABLE 4—Associations Between Duration of Exposure to Secondhand Smoke in a Single Workshift and Changes in the Level of Total NNAL, Nicotine, and Cotinine in the Urine of Nonsmoking Food Service Workers: Oregon, November 2004–August 2005

	Multiplicative Increase per Hour (95% CI)[a]
Total NNAL[b]	1.06[c] (1.02, 1.10)*
Total nicotine[b]	1.33 (1.27, 1.39)**
Total cotinine[b]	1.12 (1.07, 1.16)**

Notes. NNAL = 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol; CI = confidence interval. The mean level of total NNAL in the exposed workers was 0.03 pmol/mL in the preworkshift urine and 0.04 pmol/mL in the postworkshift urine. Their mean levels of nicotine and cotinine, respectively, were 7.21 ng/mL and 16.63 ng/mL in the preworkshift urine and 44.36 ng/mL and 20.20 ng/mL in the postworkshift urine.
[a]Multiplicative increase was based on linear mixed model, and adjusted for participant age, gender, and minutes exposed to secondhand smoke outside the workplace in the past week.
[b]A value of half the limit of detection was used for nondetectable values. Limit of detection: cotinine, 2 ng/mL; nicotine, 2 ng/mL; NNAL, 0.007–0.01 pmol/mL depending on recovery. Total NNAL was based on 158 pre- and postworkshift urine samples from 82 participants (50 exposed). Total nicotine and cotinine were based on 166 pre- and postworkshift urine samples from 84 participants (52 exposed).
[c]Interpretation: Each hour of exposure was associated with about a 6% increase in total NNAL.
*$P < .01$; **$P < .001$.

with those from a study of nonsmoking casino patrons[43] and is likely because of the relatively long half-life for NNAL: 3 to 4 days for the distribution phase and 40 to 45 days for the elimination phase among smokers.[51] An additional limitation is that establishments and participants were not selected at random. There is, however, no reason to believe that selection bias caused by nonrandom recruitment would have any effect on the biochemical outcomes. Last, our findings with regard to the estimate of hourly increases in NNAL, cotinine, and nicotine are valid only for 4 or more hours of exposure to secondhand smoke, because we confined the sample to those who worked at least 4 hours during the targeted shift.

In conclusion, our finding of increases in metabolites of NNK among exposed nonsmoking bar and restaurant workers adds to the substantial body of research that shows health risks and adverse outcomes among nonsmokers exposed to secondhand smoke in the workplace. Policies that establish smoke-free environments effectively reduce exposure to secondhand smoke and its deleterious health effects among bar and restaurant employees.[23,57–62] Studies also show that laws that prohibit smoking in bars and

restaurants do not adversely affect either employment or sales.[63–75] There is no justification for policymakers and the public to continue to allow clean indoor air exemptions; all nonsmoking workers—including bar and restaurant workers—deserve protection from lung cancer and other cancers, heart disease, and the host of other adverse health effects that result from workplace secondhand smoke exposure. ■

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Contributors

M.J. Stark originated and designed the study, supervised all aspects of the study's implementation, and led the writing of the article. K. Rohde originated and designed the study, conducted and managed the study, led the data analysis, and contributed to the writing of the article. J.E. Maher and B.A. Pizacani originated and designed the study, assisted with the data analysis, and contributed to the writing of the article. C.W. Dent and R. Bard contributed to the writing of the article. S.G. Carmella supervised the analysis of the urine specimens. A.R. Benoit and N.M. Thomson conducted the analysis of the urine specimens. S.S. Hecht assisted with study origination.

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Human Participant Protection

This study was approved by the joint institutional review board of the Multnomah County Health Department and the Oregon Department of Human Services. Informed consent was obtained from all study participants.

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