

Bartenders' Respiratory Health After Establishment of Smoke-Free Bars and Taverns

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Context.—The association between environmental tobacco smoke (ETS) exposure and respiratory symptoms has not been well established in adults.

Objective.—To study the respiratory health of bartenders before and after legislative prohibition of smoking in all bars and taverns by the state of California.

Design.—Cohort of bartenders interviewed before and after smoking prohibition.

Setting and Participants.—Bartenders at a random sample of bars and taverns in San Francisco.

Main Outcome Measures.—Interviews assessed respiratory symptoms, sensory irritation symptoms, ETS exposure, personal smoking, and recent upper respiratory tract infections. Spirometric assessment included forced expiratory volume in 1 second (FEV₁) and forced vital capacity (FVC) measurements.

Results.—Fifty-three of 67 eligible bartenders were interviewed. At baseline, all 53 bartenders reported workplace ETS exposure. After the smoking ban, self-reported ETS exposure at work declined from a median of 28 to 2 hours per week ($P < .001$). Thirty-nine bartenders (74%) initially reported respiratory symptoms. Of those symptomatic at baseline, 23 (59%) no longer had symptoms at follow-up ($P < .001$). Forty-one bartenders (77%) initially reported sensory irritation symptoms. At follow-up, 32 (78%) of these subjects had resolution of symptoms ($P < .001$). After prohibition of workplace smoking, we observed improvement in mean FVC (0.189 L; 95% confidence interval [CI], 0.082-0.296 L; 4.2% change) and, to a lesser extent, mean FEV₁ (0.039 L; 95% CI, -0.030 to 0.107 L; 1.2% change). Complete cessation of workplace ETS exposure (compared with continued exposure) was associated with improved mean FVC (0.287 L; 95% CI, 0.088-0.486; 6.8% change) and mean FEV₁ (0.142 L; 95% CI, 0.020-0.264 L; 4.5% change), after controlling for personal smoking and recent upper respiratory tract infections.

Conclusion.—Establishment of smoke-free bars and taverns was associated with a rapid improvement of respiratory health.

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THE LONG-TERM health effects of exposure to environmental tobacco smoke (ETS) have been established during the past 2 decades. Strong epidemiologic evidence links ETS exposure with lung cancer^{1,2} and atherosclerotic cardiovascular disease.³⁻⁶ As a result, ETS has been estimated as the third leading preventable cause of death.⁷ By contrast, the more immediate impact of ETS exposure on adult respiratory health has received less attention.⁸ Although household ETS exposure is a known cause of respiratory symp-

ptoms in children,^{2,9} studies examining the effect of ETS on adult respiratory symptoms have yielded conflicting results.^{2,10-18}

While most epidemiologic studies have focused on household ETS,^{12,11,19} the workplace is now recognized as a major site of exposure.^{10,20-25} Bar and tavern workers,

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in particular, are exposed to high ambient levels of ETS, reaching levels 4 to 6 times higher than in other workplaces.²⁶⁻²⁹ This high-level exposure is paralleled by an increased risk of lung cancer in bartenders, after controlling for personal smoking.^{30,31} The effect of ETS on respiratory symptoms and lung function, however, has not been examined in these heavily exposed workers.

Recent California statewide legislation,³² which now mandates smoke-free bars and taverns, provided an unusual opportunity to prospectively evaluate the effect of reduced ETS exposure on respiratory symptoms, sensory irritation symptoms (eye, nose, and throat), and pulmonary function in bartenders.

METHODS

Overview

California State Assembly Bill 13 amended the California Labor Code (section 6404.5) to prohibit tobacco smoking in bars and taverns starting January 1, 1998.^{32,33} From December 1 to 31, 1997, we interviewed and performed spirometry on participating bartenders in their workplaces (bar or tavern). Follow-up interviews and spirometry were performed from February 1 to 28, 1998, to evaluate changes in symptoms or lung function following the institution of smoke-free bars.

Recruitment of Freestanding Bars and Taverns

The present study was approved by the University of California, San Francisco, Committee on Human Research. We obtained a list of all bars and taverns ($N = 366$) in the city and county of San Francisco from a commercial yellow pages directory (under subject headings "bars," "cocktail lounges," or "taverns"). After review of the listings, we excluded businesses known to be restaurants ($n = 66$) or associated with hotels ($n = 4$). Of the 296 listings, we randomly sampled 105 freestanding establishments. Each bar or tavern proprietor was contacted by a letter describing the study and given the opportunity to decline participation by prepaid, self-addressed postcard. We telephoned each owner who did not return the decline postcard and requested permission to visit their bar or tavern to recruit bartenders for the study.

Of the 105 freestanding bars and taverns sampled, 13 establishments were no longer in business and 9 were located in restaurants, leaving 83 eligible businesses. In 22 cases, the owner could not be reached by telephone despite 6 or more

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attempts. The owners of 36 bars declined study participation; 7 returned the decline postcard and 29 declined by telephone. During telephone contact, the reasons provided for declining were disagreement with the change in the Labor Code ($n = 8$, 28%), inconvenience ($n = 3$, 11%), or not stated ($n = 18$, 61%). Ultimately, 25 bars and taverns (30%) still in business participated. As presented later in the "Methods" section, we found no evidence of systematic bias introduced by bar or tavern nonparticipation.

Recruitment of Bartenders

At prearranged times, a single study investigator (M.D.E.) visited each participating bar or tavern and attempted to recruit all bartenders who worked there at least 1 daytime shift per week. Because study participation required about 15 minutes per subject, we were unable to conduct the study during peak business hours. The 25 participating bars and taverns employed 124 bartenders, with 67 bartenders working at least 1 weekly daytime shift. Fifty-four of the daytime bartenders (81%) completed baseline interviews and spirometry; 53 of these subjects (98%) completed follow-up. A small number of subjects ($n = 3$, 6%) were no longer working in bars or taverns at the time of the follow-up interview and lung function assessment (these subjects were retained for analysis). The mean interval (SD) between baseline and follow-up interviews was 56 (9) days (median, 56 days).

The estimated annual average number of bartenders employed in San Francisco was 1930 (1994 data based on the California Employment Development Department Labor Market Information Database). Our study sample of bartenders, then, represents approximately 2.8% of all bartenders employed in San Francisco.

Interviews

All subjects underwent a standard baseline interview conducted by a single study investigator (M.D.E.) in their workplaces. Respiratory symptoms were assessed with 5 questions from the International Union Against Tuberculosis and Lung Disease (IUATLD) Bronchial Symptoms Questionnaire.³⁴ The questions related to wheezing, dyspnea, morning cough, cough during the rest of the day or night, and phlegm production. The IUATLD instrument has been validated against the criterion of bronchial hyper-responsiveness.^{34,35} To evaluate change in respiratory symptoms during a short period, we modified the IUATLD questions to assess symptoms during the past 4 weeks rather than the previous 12 months. In addition to the IUATLD battery, we also assessed sensory irritation symptoms, which can result from ETS-related

noxious stimulation of upper respiratory tract and corneal mucous membranes.² Three questions ascertained the presence of red, teary, or irritated eyes; runny nose, sneezing, or nose irritation; and sore or scratchy throat during the past 4 weeks.

Personal, active cigarette smoking was measured using questions developed for the National Health Interview Survey.³⁶ In 3 additional questions, we evaluated ETS exposure duration in work, home, and other settings during the previous 7 days (in hours per week).

Several questions focused on baseline health and demographic characteristics. Using a question from the National Health and Nutrition Examination Survey (NHANES), we assessed whether subjects had physician-diagnosed asthma.³⁷ In addition, medication use for asthma was ascertained. We evaluated whether subjects had an upper respiratory tract infection (URI) during the past 4 weeks with the following question: "In the last 4 weeks, have you had a cold?" Finally, demographic information was collected, including age, sex, race, and education.

Conducted about 8 weeks later, follow-up interviews contained the same questions about respiratory symptoms, sensory irritation symptoms, personal smoking, ETS exposure, and URIs. At the end of the second interview, we ascertained personal beliefs about the health effects of ETS exposure and attitudes about the prohibition of smoking in bars and taverns.

Spirometry

All participating bartenders underwent spirometry at both baseline and follow-up in their workplaces. We measured lung function with a portable spirometer (Creative Biomedics, San Clemente, Calif). Using a standard protocol conforming to American Thoracic Society Guidelines, we had each subject perform at least 3 forced expiratory maneuvers.³⁸ Forced expiratory volume in 1 second (FEV_1), forced vital capacity (FVC), and forced expiratory flow, midexpiratory phase ($FEF_{25\%-75\%}$) were determined.

Participating and Nonparticipating Bars and Taverns

To assess the comparability of participating and nonparticipating establishments, we obtained information about San Francisco bars and taverns from several sources. The State of California Department of Alcoholic Beverage Control provided liquor license issuance dates and license status. We extracted data about establishment size (square meters), county health district (based on census tract), length of time in business (either same owner or same establishment name), and health code violations from Department of

Public Health Environmental Health Section inspection records. Table 1 shows that there were no statistically significant differences between participating and nonparticipating bars ($P > .40$ in all cases).

To estimate how closely our sample of bartenders matched the target population of San Francisco bartenders, we reviewed demographic data for an available comparison group—unionized San Francisco bartenders ($n = 462$)—obtained from the Hotel and Restaurant Employees and Bartenders Union Local 2 (affiliated with the American Federation of Labor—Congress of Industrial Organizations). Compared with union members, the bartenders in our sample were younger (mean [SD], 42.2[14] vs 51.0[11.4] years; $P < .001$) and more likely to be female (28% vs 17%; $P = .05$). There was no statistically significant difference in the proportion of nonwhite bartenders in our sample compared with union members (37% vs 29%; $P = .25$).

Statistical Analysis

Our general analytic framework compared the respiratory health of bartenders before and after prohibition of smoking in bars and taverns. The study had 2 central hypotheses. First, respiratory and sensory irritation symptoms would improve among bartenders after reduced ETS exposure following the legislative ban. Second, bartenders' pulmonary function would improve after reduction in workplace ETS exposure.

Interview and spirometry data were analyzed using SAS software version 6.12 (SAS Institute Inc, Cary, NC), unless otherwise noted. We compared the change in work duration (hours per week), personal smoking, and ETS exposure using the paired t test for normally distributed variables, paired Wilcoxon signed rank test for nonnormally distributed continuous variables, and McNemar χ^2 test for dichotomous variables.

To reduce the number of statistical comparisons, we defined 2 a priori primary symptom end points: any respiratory symptom (wheeze, shortness of breath, morning cough, cough during the rest of the day or night, or phlegm production) and any sensory irritation symptom (eye, nose, or throat). The McNemar χ^2 test was used to compare the observed change in each symptom end point with that expected by chance. We then performed secondary analyses to evaluate the change in each symptom type during follow-up.

To address the potential confounding effect of recent URIs, we repeated the primary analyses excluding these subjects. To control for personal smoking, we also repeated the analyses stratified by smoking status. Using Stata software version 5.0 (Stata Corp, College Station,

Tex), we then performed conditional logistic regression analysis³⁹ on the entire sample to estimate the impact of reduced workplace ETS exposure on the 2 primary symptom outcomes, controlling for the effects of URIs and personal cigarette consumption at both interviews.

The change in pulmonary function after reduction in workplace ETS exposure was examined using paired *t* tests. The primary end points were FEV₁, FVC, and FEF_{25%-75%}. As before, we repeated the analyses excluding subjects with URIs. We also controlled for the potential confounding effect of personal smoking by further stratifying the analysis by smoking status. Because the legislative ban was only partially successful in prohibiting smoking in some bars, we were able to examine the effect of complete (vs partial) cessation of workplace ETS exposure on each pulmonary function parameter using multiple linear regression analysis. The multivariate model controlled for personal smoking, reduced daily cigarette consumption at follow-up, and URIs during the 4 weeks prior to baseline. To evaluate a dose-response relationship, we repeated the multivariate analysis using complete workplace exposure cessation as the referent case and 2 dichotomous indicator variables for moderate exposure (1-6 h/wk) and high exposure (≥ 7 h/wk). These cut points provided similar subject numbers in moderate-level (*n* = 14) and high-level (*n* = 15) categories.

RESULTS

Bartender Characteristics

For the 53 participating bartenders completing follow-up interviews and spirometry, the average (SD) age was 42.5 (14.0) years. A substantial proportion of subjects were female (28%) and non-white (38%) (Table 2). The mean duration of employment at the current bar or tavern was 6.1 (SD, 7.1) years (median, 3.0 years). Other subject characteristics are summarized in Table 2.

Cigarette Smoking and ETS Exposure

Forty (76%) of the 53 bartenders reported a history of ever smoking, with 24 (45%) currently smoking at baseline (Table 3). There was no change in the proportion of current smokers from baseline to follow-up interviews, after prohibition of workplace smoking. Among the current smokers, there was no overall change in daily cigarette consumption during the follow-up period.

At baseline, all 53 subjects reported ETS exposure while working in bars or taverns during the 7 days prior to interview. After prohibition of smoking, there was no significant change in weekly work dura-

Table 1.—Comparison of Participating and Nonparticipating Bars and Taverns in San Francisco*

Characteristic	Participating Bars (n = 25)	Nonparticipating Bars (n = 58)
Health center location, No. (%)		
2	5 (20)	13 (22)
3	2 (9)	8 (14)
4	7 (28)	14 (24)
5	11 (44)	23 (40)
Establishment size, mean (SD), m ²	143.91 (95.76)	126.36 (84.06)
Years in business, mean (SD), y	13.6 (9.5)	15.3 (8.3)
Current liquor license duration, mean (SD), y	9.9 (6.5)	9.0 (7.0)
Liquor license with active status, No. (%)	25 (100)	58 (100)
Any health code violation in past 1 year, No. (%)	11 (46)	25 (43)

**P* > .40 in all comparisons of participating vs nonparticipating bars. The sources of data are the Department of Public Health Environmental Health Section inspection records (health center location, establishment size, years in business, and health code violations) and State of California Department of Alcoholic Beverage Control (liquor license information).

tion from baseline (mean [SD], 33.4 [14.9] hours) to follow-up interviews (32.2 [17.5] hours; *P* = .48). However, self-reported workplace ETS exposure sharply declined from a median of 28 to 2 h/wk (*P* < .001) after the smoke-free workplace law went into effect (Table 3). We observed a parallel decrease in other (non-work) and total ETS exposure. Despite the prohibition of smoking, 29 subjects (55%) continued to report some ETS exposure (≥ 1 h/wk) while working as bartenders.

Respiratory and Sensory Irritation Symptoms

Thirty-nine (74%) of the 53 bartenders reported respiratory symptoms at baseline, while only 17 (32%) were still symptomatic at follow-up (Table 4). Of the 39 bartenders reporting baseline symptoms, 23 subjects (59%) no longer indicated any respiratory symptoms after prohibition of smoking (*P* < .001). The majority of bartenders also had at least 1 sensory irritation symptom at baseline (77%), with fewer reporting symptoms at follow-up (19%). With introduction of smoke-free workplaces, sensory symptoms were no longer present in 32 (78%) of the 41 previously symptomatic subjects (*P* < .001).

Since URIs can be associated with both respiratory and sensory irritation symptoms, we repeated the analyses excluding the 8 subjects who reported a recent URI at baseline interview. A majority of the remaining 45 bartenders (69%) still reported respiratory symptoms at baseline, with most of these subjects (65%) indicating resolution of symptoms at follow-up (*P* < .001). Similarly, most bartenders without recent URIs noted sensory irritation symptoms at baseline (76%). At follow-up interview, the majority of these subjects (79%) no longer reported any sensory symptoms (*P* < .001).

We recognized that smoke-free bars and taverns might lead bartenders to curtail their personal smoking, which could diminish respiratory symptoms. After stratifying the analysis by smoking status, we observed similar results. Of the previ-

Table 2.—Baseline Bartender Characteristics (*n* = 53)

Age, mean (SD), y	42.5 (14.0)
Sex, No. (%) female	15 (28)
Race, No. (%)	
White, non-Hispanic	33 (62)
Hispanic	10 (19)
African American	2 (4)
Asian/Pacific Islander	6 (11)
Other	2 (4)
Education, highest level attained, No. (%)	
High school	13 (25)
College or greater	40 (76)
Duration of current bar employment, y	
Mean (SD)	6.1 (7.1)
Median (25th-75th interquartile range)	3.0 (1.5-8.0)
History of physician-diagnosed asthma, No. (%)	9 (17)
Currently receiving asthma medications, No. (%)	4 (8)

ously symptomatic smoking bartenders, the majority no longer reported respiratory (63%) or sensory irritation symptoms (80%) at follow-up (*P* < .001 in both cases). Similarly, most nonsmoking bartenders with baseline symptoms reported resolution of respiratory (53%) or sensory irritation symptoms (76%) (*P* = .02 and *P* < .001, respectively).

Conditional logistic regression analysis was performed to estimate the independent impact of reduced bar ETS exposure on the primary symptom end points. A 5-hour reduction in workplace ETS exposure was associated with a lower risk of respiratory symptoms at follow-up (odds ratio [OR], 0.7; 95% confidence interval [CI], 0.5-0.9), after controlling for URIs and daily cigarette consumption at both interviews. In a similar analysis, a 5-hour decrement in bar ETS exposure was associated with reduced risk of sensory irritation symptoms at follow-up (OR, 0.5; 95% CI, 0.3-0.8). Excluding the 3 subjects no longer working as bartenders at follow-up did not appreciably affect these risk estimates.

Pulmonary Function

After prohibition of smoking, the mean FVC and FEV₁ both increased at follow-up (Table 5). Flow rate at midlung volumes (FEF_{25%-75%}), which was

Table 3.—Personal Smoking and Environmental Tobacco Smoke Exposure at Baseline and Follow-up Interviews (n = 53)*

	Baseline	Follow-up†
Personal (direct) smoking		
Ever smoked cigarettes, No. (%)	40 (76)	...
Currently smokes cigarettes, No. (%)	24 (45)	24 (45)
Daily smoking, mean No. of packs per day (SD)	1.0 (0.6)	1.0 (0.6)
Environmental tobacco smoke exposure, median (25th-75th interquartile range)		
Bar or tavern exposure, h in past 7 d	28 (20-40)	2 (0-10)‡
Other exposure, h in past 7 d	7 (0-20)	2 (0-15)§
Total exposure, h in past 7 d	40 (30-55)	10 (2-30)‡

*Environmental tobacco smoke exposure at follow-up excludes 3 subjects who no longer worked in bars. Including these subjects, exposure at follow-up was bar or tavern median, 2 (interquartile range, 0-9) hours; other, 2 (0-10) hours, and total, 10 (2-30) hours. Ellipses indicate data not applicable.

†All comparisons are $P > .99$ unless otherwise indicated (McNemar test for current smoking, paired t test for daily smoking).

‡ $P < .001$ by Wilcoxon signed rank test.

§ $P = .07$ by Wilcoxon signed rank test.

Table 4.—Respiratory and Sensory Irritation Symptoms in Bartenders Before and After Prohibition of Smoking in Bars (n = 53)

	Prevalence of Symptoms		Change in Symptoms at Follow-up*			P Value‡
	Baseline, No. (%)	Follow-up, No. (%)	Reduction, No.	Increase, No.	No Change, No.	
Any respiratory symptom†	39 (74)	17 (32)	23	1	29	<.001
Wheezing	17 (32)	8 (15)	12	3	38	.02
Dyspnea	10 (19)	4 (8)	8	2	43	.06
Cough, morning	28 (53)	12 (23)	17	1	35	<.001
Cough, rest of day or night	26 (49)	6 (11)	21	1	31	<.001
Phlegm production	28 (53)	6 (11)	22	0	31	<.001
Any sensory irritation symptom†	41 (77)	10 (19)	32	1	20	<.001
Eye	22 (42)	3 (6)	20	1	32	<.001
Nose	32 (60)	8 (15)	25	1	27	<.001
Throat	13 (25)	7 (13)	9	3	41	.08

*Reduction, symptoms at baseline but none at follow-up; increase, no symptoms at baseline and new symptoms at follow-up; and no change, either symptomatic at both interviews or asymptomatic at both interviews.

†Any respiratory symptom and any sensory irritation symptom are primary end points. Individual symptom analyses are secondary.

‡McNemar χ^2 test, comparing observed change in symptoms over time with that expected by chance. For example, of the 39 subjects with any respiratory symptom at baseline, 23 (59%) no longer had symptoms at follow-up ($P < .001$).

Table 5.—Pulmonary Function in Bartenders Before and After Prohibition of Smoking in Bars (n = 53)*

Measurement	Baseline		Follow-up		Change, Mean (95% CI)†	% Change‡
	Mean (SE) [% Predicted]	Mean (SE) [% Predicted]	Mean (SE) [% Predicted]	Mean (SE) [% Predicted]		
FEV ₁ , L/s	3.38 (0.13) [89.2 (2.4)]	3.42 (0.14) [89.9 (2.4)]	0.039 (-0.030 to 0.107)	1.2		
FVC, L	4.43 (0.15) [95.5 (2.2)]	4.62 (0.17) [99.8 (2.4)]	0.189 (0.082 to 0.296)	4.2		
FEF _{25%-75%} , L/s	3.37 (0.19) [81.6 (3.5)]	3.18 (0.17) [80.3 (3.8)]	-0.190 (-0.405 to 0.025)	-5.7		

*FEV₁ indicates forced expiratory volume in 1 second; FVC, forced vital capacity; FEF_{25%-75%}, forced expiratory flow, midexpiratory phase; and CI, confidence interval.

†95% CIs are based on paired t test.

‡Compared with baseline value of pulmonary function measurement.

highly variable, declined during the study period.

As with symptom end points, we performed additional analyses to control for the effects of recent URIs and personal smoking. Excluding the 8 subjects who reported URIs in the 4 weeks prior to baseline, we found a statistically significant improvement in both FVC (0.233 L; 95% CI, 0.124-0.343 L) and FEV₁ (0.083 L; 95% CI, 0.015-0.151 L). After further stratification by smoking status, we observed a similar estimated improvement in FVC among smokers (0.238 L; 95% CI, 0.081-0.395 L) and nonsmokers (0.229 L; 95% CI, 0.063-0.394 L). The FEV₁ in-

crease was also comparable among smokers (0.096 L; 95% CI, -0.013 to 0.204 L) and nonsmokers (0.070 L; 95% CI, -0.020 to 0.159 L), although the CIs did not exclude no significant change.

Compared with some continued ETS exposure in bars or taverns at follow-up, complete workplace exposure cessation was associated with improved FVC (0.287 L; 95% CI, 0.088-0.486 L) and FEV₁ (0.142 L; 95% CI, 0.020-0.264 L), after controlling for current smoking, decreased daily cigarette consumption, and recent URIs. Expressed as an adjusted mean percentage change, FVC and FEV₁ increased by 6.8% and 4.5%, respectively. After con-

trolling for these covariates, FEF_{25%-75%} also increased during follow-up, but the effect was not statistically significant (0.081 L; 95% CI, -0.349 to 0.511 L). A history of asthma, when added to the model, was not associated with change in pulmonary function ($P > .60$). Excluding the 3 subjects no longer working as bartenders at follow-up did not appreciably change the effect estimates or CIs.

To evaluate a dose-response relationship, we repeated the multivariate analysis using 3 categories of workplace ETS exposure at follow-up: none, moderate (1-6 hours), and high (≥ 7 hours). Bartenders with complete workplace ETS exposure cessation had improved FVC (0.326 L; 95% CI, 0.009-0.565 L) and FEV₁ (0.157 L; 95% CI, 0.001-0.303 L), relative to those indicating persistent high-level workplace exposure. Compared with bartenders reporting only moderate levels of ETS exposure in bars or taverns, complete workplace exposure cessation was associated with a smaller improvement in FVC (0.244 L; 95% CI, 0-0.489 L) and FEV₁ (0.127 L; 95% CI, -0.024 to 0.277 L).

Relationship Between Symptoms and Pulmonary Function

We repeated the multiple linear regression analyses to evaluate whether pulmonary function improved in 2 separate strata: subjects whose respiratory symptoms resolved (n = 23) and subjects with persistent or new symptoms (n = 30) at follow-up. In bartenders who reported resolution of respiratory symptoms, complete cessation of workplace ETS exposure was associated with improved FVC (0.464 L; 95% CI, 0.172-0.757 L) and FEV₁ (0.202 L; 95% CI, 0.002-0.403 L). The subjects with continued symptoms also experienced improvement in FEV₁ (0.146 L; 95% CI, -0.010 to 0.302 L) and FVC (0.139 L; 95% CI, -0.164 to 0.441 L), although the CIs overlap no change.

Bartenders' Attitudes About the Health Effects of ETS and the Prohibition of Smoking

Eleven (21%) of the 53 bartenders expressed the belief that ETS exposure has no adverse effect on their personal health. The remaining 42 bartenders believed that ETS has a slight effect (40%) or moderate-to-severe effect (40%) on their health.

When asked about their personal attitude toward the prohibition of smoking in bars, 24 (45%) of the 53 bartenders strongly or somewhat (19%) disagreed with the legislative ban. The remaining bartenders were neutral (9%), somewhat agreed (8%), or strongly agreed (19%). Most bartenders who believed that ETS has a moderate-to-severe adverse health effect agreed with the prohibition of smoking in bars

(67%), compared with those who thought ETS had a slight or no effect on their health (16%; $P < .001$).

Bartender attitudes were related to smoking status. A greater proportion of nonsmoking bartenders (52%) believed that ETS had a moderate-to-severe health effect than smoking bartenders (27%; $P = .06$). Similarly, more nonsmokers agreed with the legislation (59%) than smokers (12%; $P < .001$).

COMMENT

Our study indicates that self-reported workplace ETS exposure was ubiquitous among bartenders, with most reporting heavy exposure. After statewide legislation mandating smoke-free bars and taverns was enacted, San Francisco bartenders reported a substantial reduction in workplace ETS exposure. The prevalence of respiratory and sensory irritation symptoms, which initially affected the majority of bartenders, declined markedly after the smoking ban. Similarly, pulmonary function improved following reduction of workplace ETS exposure, after controlling for personal smoking and URIs.

In previous studies, prohibition of workplace smoking has effectively reduced employee ETS exposure. Smoke-free workplaces have been associated with decreased personal cigarette consumption,⁴⁰⁻⁴² public smoking,⁴³ and self-reported ETS exposure.⁴⁴ In addition, workplace smoking bans result in dramatic reduction of indoor airborne nicotine concentrations, reflecting decreased ambient ETS levels.^{22,43,45} The efficacy of smoking prohibition in bars and taverns, however, has been less well established. After smoking was prohibited in a sports tavern, investigators found decreased respiratory suspended particulate concentrations, suggesting lower ETS levels.⁴⁶ The present study indicates that legislative prohibition of smoking substantially reduced, but did not eliminate, self-reported workplace ETS exposure among bartenders.

In adults, the evidence linking ETS exposure with respiratory symptoms has been inconclusive. Several studies have demonstrated an association between self-reported obstructive lung disease and ETS exposure.^{15,16,47} A recent cross-sectional study of 4187 nonsmoking Swiss adults found an increased risk of wheeze, dyspnea, and bronchitis symptoms in subjects reporting ETS exposure during the past year.¹⁰ Similarly, workplace ETS exposure was related to increased cough, phlegm production, and dyspnea in 80 adults enrolling in a fitness program.⁴⁸ Other studies, however, have not demonstrated a consistent, significant increase in respiratory symptoms in adults exposed to household^{11-14,17,18} or workplace ETS.¹⁷ Furthermore, a smoking ban in sev-

eral Canadian office buildings was not associated with any significant reduction in respiratory symptoms 1 year later.⁴² Our study, which demonstrated reduced respiratory symptoms after prohibition of workplace smoking, helps confirm the adverse impact of ETS exposure on immediate respiratory health.

Environmental tobacco smoke contains potent respiratory irritants, such as ammonia, sulfur dioxide, acrolein, and formaldehyde,² that could potentially impair lung function. In several cross-sectional epidemiologic studies, ETS exposure was associated with small reductions in FEV₁ (2.8%-6.7%)⁴⁹⁻⁵³ and FVC (2.6%-5.4%)^{49,53} compared with unexposed subjects. Another study found no impact of ETS exposure on FEV₁ or FVC, but FEF_{25%-75%} was reduced.⁵⁴ A recent prospective investigation of 26 bar workers demonstrated a significant reduction in FEV₁ (0.042 L) immediately following ETS exposure during a work shift.⁵⁵ Not all studies, however, have found consistent pulmonary function decrements.^{11,56} Although the effect of ETS exposure cessation on adult lung function has not been characterized, the salutary effect of personal cigarette smoking cessation is well established. Several studies demonstrated modest increases in FEV₁ (1.2%-4.8%) shortly after personal smoking cessation.⁵⁷⁻⁶¹ Our study suggests that lung function may also improve, to a small degree, after cessation of heavy ETS exposure.

Although we adhered to a standard spirometry protocol,³⁸ we cannot exclude the contribution of training to the observed pulmonary function improvement. The proportionally larger increase in FVC than FEV₁, in particular, could be consistent with a learning effect. The CIs, however, are broad, with overlap between the estimated relative improvement in FEV₁ and FVC. Also, the FEV₁ improvement is similar to the acute decrement previously described in bar workers after a work shift.⁵⁶ Similarly, the unadjusted relative increase in FEV₁ (1.2%) is comparable with the FEV₁ change described in both smoking cessation studies⁵⁷⁻⁶¹ and cross-sectional studies of ETS.⁴⁹⁻⁵³ The unadjusted FVC improvement (4.2%) also seems compatible with the ETS-related decrement described in epidemiologic studies.^{49,53} Finally, the unexpected trend toward decreasing FEF_{25%-75%}, a highly variable measure, disappeared after adjustment for smoking and URIs. Overall, the improvement in lung function is consistent with a causal effect of reduced workplace ETS exposure.

The present study has several additional limitations, including the use of interviews to assess ETS exposure and symptom status. Interview administration by an unblinded investigator could

have biased subject responses, although we attempted to maintain standard conditions. Although many studies demonstrate modest correlations between self-reported ETS exposure and biomarker levels, such as serum cotinine,^{20,62,63} we cannot exclude some systematic misclassification of exposure. For example, bartenders with respiratory symptoms might be more likely to report ETS exposure, whereas asymptomatic subjects might underreport exposure. Similarly, controversy generated by the smoke-free bar and tavern legislation could have biased symptom reporting. If subjects who agreed with the smoking ban were more likely to report symptom reduction, the observed improvement in symptom status could be inflated. However, the majority of subjects disagreed with the new legislation, which would not be expected to favorably bias symptom reporting. Moreover, the objective measure of pulmonary function helps validate the reduction in respiratory symptoms.

The low participation rate by bars and taverns (30%) raises the concern of generalizability to all bars. Owner attitude toward the smoking ban could have influenced their decision to allow study participation, making systematic differences between participating and nonparticipating bars possible. However, we found no differences in any characteristic examined between participating and nonparticipating establishments. Importantly, business size, which is 1 determinant of ambient ETS concentration,⁶⁴ did not differ.

Similarly, differences between study participants and the entire population of San Francisco bartenders could limit the generalizability of our results. To assess how representative our sample was, we compared our subjects with an available bartender group from the same sampling area. Participating bartenders were younger and more likely to be female than unionized bartenders; there were no differences in race. Unionized workers, however, are generally more likely to be older and male than the general working population,⁶⁵ potentially explaining these observed differences. Although we found little evidence of systematic differences between our sample and the target population, residual differences may still limit the generalizability of our findings to all bartenders in San Francisco, and more broadly, in California overall.

The small sample size, while not likely to affect study generalizability, limited our power to detect differences in some aspects of respiratory health. The unadjusted improvement of FEV₁, in particular, had a CI including no change. Similarly, there was limited power to detect a dose-response relationship. Although we

attempted to perform parsimonious regression analyses, the sample size was small for multivariate procedures.

Finally, confounding by personal smoking and URIs could potentially explain the observed improvement in respiratory health. After statistically controlling for these issues, we still observed improvement in respiratory symptoms and lung function after prohibition of smoking. Reliance on self-report, however, raises the possibility that unmeasured baseline URIs or reduced active smoking could still partially explain the observed improvement in respiratory health indexes.

Our study demonstrates that reduced ETS exposure, occurring after implementation of smoke-free workplace legislation, was associated with improved adult respiratory health during a short observation period. In addition to potentially reducing the long-term risk of lung cancer and cardiovascular disease, workplace smoking prohibition appears to have immediate beneficial effects on adult respiratory health.

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References

1. Hackshaw AK, Law MR, Wald NJ. The accumulated evidence on lung cancer and environmental tobacco smoke. *BMJ*. 1997;315:980-988.
2. California Environmental Protection Agency. *Health Effects of Exposure to Environmental Tobacco Smoke*. Sacramento: California Environmental Protection Agency, Office of Environmental Health Hazard Assessment; 1997.
3. Howard G, Wagenknecht LE, Burke GL, et al. Cigarette smoking and progression of atherosclerosis. *JAMA*. 1988;279:119-124.
4. Kawachi I, Colditz GA, Speizer FE, et al. A prospective study of passive smoking and coronary heart disease. *Circulation*. 1997;95:2374-2379.
5. Wells JA. Heart disease from passive smoking in the workplace. *J Am Coll Cardiol*. 1998;31:1-9.
6. Law MR, Morris JK, Wald NJ. Environmental tobacco smoke, exposure, and ischaemic heart disease: an evaluation of the evidence. *BMJ*. 1977;315:973-980.
7. Glantz SA, Parmley WW. Passive smoking and heart disease. *Circulation*. 1991;83:1-11.
8. Leaderer BP, Samet JM. Passive smoking and adults: new evidence for adverse effects. *Am J Respir Crit Care Med*. 1994;150:1216-1218.
9. US Environmental Protection Agency. *Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders*. Washington, DC: US Environmental Protection Agency, Office of Air and Radiation; 1992. Publication EPA/600/6-90/006F.
10. Leuenberger P, Schwartz J, Ackermann-Lieblich U, et al. Passive smoking exposure in adults and chronic respiratory symptoms (SAPALDIA study). *Am J Respir Crit Care Med*. 1994;150:1222-1228.
11. Kauffmann F, Dockery DW, Speizer FE, et al. Respiratory symptoms and lung function in relation to passive smoking. *Int J Epidemiol*. 1989;18:334-344.
12. Gillis CR, Hole DJ, Hawthorne VM, Boyle P. The effect of environmental tobacco smoke in two urban communities in the west of Scotland. *Eur J Respir Dis*. 1984;65:121-126.
13. Hole DJ, Gillis CR, Chopra C, et al. Passive smoking and cardiorespiratory health in a general population in the west of Scotland. *BMJ*. 1989;299:423-427.
14. Comstock GW, Meyer MB, Helsing KJ, Tockman MS. Respiratory effects of household exposures to tobacco smoke and gas cooking. *Am Rev Respir Dis*. 1981;124:143-148.
15. Dayal HH, Khuder S, Sharrar R, et al. Passive smoking in obstructive respiratory diseases in an industrialized urban population. *Environ Res*. 1994;65:161-171.
16. Robbins AS, Abbey DE, Lebowitz MD. Passive smoking and chronic respiratory disease symptoms in non-smoking adults. *Int J Epidemiol*. 1993;22:809-817.
17. Jaakkola MS, Jaakkola JJK, Becklake MR, Ernst P. Effect of passive smoking on the development of respiratory symptoms in young adults: an 8-year longitudinal study. *J Clin Epidemiol*. 1996;49:581-586.
18. Schwartz J, Zeger S. Passive smoking, air pollution, and acute respiratory symptoms in a diary study of student nurses. *Am Rev Respir Dis*. 1990;141:62-67.
19. Hirayama T. Nonsmoking wives of heavy smokers have a higher risk of lung cancer: a study from Japan. *BMJ*. 1981;282:183-185.
20. Pirkle JL, Flegal KM, Bernert JT, et al. Exposure of the US population to environmental tobacco smoke. *JAMA*. 1996;275:1233-1240.
21. Morris PD. Lifetime excess risk of death from lung cancer for a US female never-smoker exposed to environmental tobacco smoke. *Environ Res*. 1995;68:3-9.
22. Hammond SK, Sorensen G, Youngstrom R, Ockene JK. Occupational exposure to environmental tobacco smoke. *JAMA*. 1995;274:956-960.
23. Thompson B, Emmons K, Abrams D, et al. ETS exposure in the workplace. *J Occup Environ Med*. 1995;37:1086-1092.
24. Gerlach KK, Shopland DR, Hartman AM, et al. Workplace smoking policies in the United States. *Tob Control*. 1997;6:199-206.
25. Akbar-Khanzadeh F, Greco TM. Health and social concerns of restaurant/bar workers exposed to environmental tobacco smoke. *Med Law*. 1996;87:122-132.
26. Siegel M. Involuntary smoking in the restaurant workplace. *JAMA*. 1998;270:490-493.
27. Jarvis MJ, Russell M, Feyerabend C. Absorption of nicotine and carbon monoxide from passive smoking under natural conditions of exposure. *Thorax*. 1983;38:829-833.
28. Bergman TA, Johnson DL, Boatright DT, et al. Occupational exposure of nonsmoking nightclub musicians to environmental tobacco smoke. *Am Ind Hyg Assoc J*. 1996;57:746-752.
29. Jarvis MJ, Foulds J, Feyerabend C. Exposure to passive smoking among bar staff. *Br J Addict*. 1992;87:111-113.
30. Singleton JA, Beaumont JJ. *COMS II, California Occupational Mortality 1979-1981: Adjusted for Smoking, Alcohol, and Socioeconomic Status*. Sacramento: California Dept of Health Services; 1989.
31. Schoenberg JB, Sternhagen A, Mason TJ, et al. Occupation and lung cancer risk among New Jersey white males. *J Natl Cancer Inst*. 1987;79:13-21.
32. California General Assembly. *An Act to Add Section 6404.5 to the Labor Code, Relating to Occupational Safety and Health*, Sess (1994). Assembly Bill 13.
33. MacDonald HR, Glantz SA. Political realities of statewide smoking legislation: the passage of California's Assembly Bill 13. *Tob Control*. 1997;6:41-54.
34. Burney PGJ, Laitinen LA, Perdrizet S, et al. Validity and repeatability of the IUATLD (1984) bronchial symptoms questionnaire: an international comparison. *Eur Respir J*. 1989;2:940-945.
35. Burney PGJ, Chinn S, Britton JR, et al. What symptoms predict bronchial response to histamine? *Int J Epidemiol*. 1989;18:165-173.
36. Vital and health statistics: current estimates from the National Health Interview Survey, 1988. *Vital Health Stat 10*. 1989;173:1-250.
37. McWhorter WP, Polis MA, Kaslow RA. Occurrence, predictors, and consequences of adult asthma in NHANES I and follow-up survey. *Am Rev Respir Dis*. 1989;139:721-724.
38. American Thoracic Society. Standardization of spirometry: 1994 update. *Am J Respir Crit Care Med*. 1995;152:1107-1136.
39. Diggle PJ, Liang KY, Zeger SL. *Analysis of Longitudinal Data*. New York, NY: Oxford University Press; 1996.
40. Woodruff LJ, Rosbrook B, Pierce J, et al. Lower levels of cigarette consumption found in smoke-free workplaces in California. *Arch Intern Med*. 1993;153:1485-1493.
41. Millar WJ. Evaluation of the impact of smoking restrictions in a government work setting. *Can J Public Health*. 1988;79:379-382.
42. Broder I, Pilger C, Correy P. Environment and well-being before and following smoking ban in office buildings. *Can J Public Health*. 1993;84:254-258.
43. Stillman FA, Becker DM, Swank RT, et al. Ending smoking at the Johns Hopkins medical institutions. *JAMA*. 1990;264:1565-1569.
44. Borland R, Pierce JP, Burns DM, et al. Protection from environmental tobacco smoke in California. *JAMA*. 1992;268:749-752.
45. Becker DM, Conner HF, Waranch R, et al. The impact of a total ban on smoking in the Johns Hopkins Children's Center. *JAMA*. 1989;262:799-802.
46. Ott WR, Switzer P, Robinson J. Particle concentration inside a tavern before and after prohibition of smoking. *J Air Waste Manag Assoc*. 1996;46:1120-1134.
47. Greer JR, Abbey DE, Burchette RJ. Asthma related to occupational and ambient air pollutants in nonsmokers. *J Occup Environ Med*. 1993;35:909-915.
48. White JR, Froeb HF, Kulik JA. Respiratory illness in nonsmokers chronically exposed to tobacco smoke in the work place. *Chest*. 1991;100:39-43.
49. Xu X, Li B. Exposure-response relationship between passive smoking and adult respiratory function. *Am J Respir Crit Care Med*. 1995;151:41-46.
50. Svendsen KH, Kuller LH, Martin MJ, et al. Effects of passive smoking in the Multiple Risk Factor Intervention Trial. *Am J Epidemiol*. 1987;126:783-795.
51. Ng TP, Hui KP, Tan WC. Respiratory symptoms and lung function effects of domestic exposure to tobacco smoke and cooking by gas in non-smoking women in Singapore. *J Epidemiol Community Health*. 1993;47:454-459.
52. Brunekreef B, Fischer P, Remijn B, et al. Indoor air pollution and its effect on pulmonary function of adult non-smoking women, III. *Int J Epidemiol*. 1985;14:227-230.
53. White JR, Froeb HF. Small-airways dysfunction in nonsmokers chronically exposed to tobacco smoke. *N Engl J Med*. 1980;302:729-733.
54. Masi MA, Hanley JA, Ernst P, et al. Environmental exposure to tobacco smoke and lung function in young adults. *Am Rev Respir Dis*. 1988;138:296-299.
55. Dimich-Ward H, Lawson J, Chan-Yeung M. Work shift changes in lung function in bar workers exposed to environmental tobacco smoke. *Am J Respir Crit Care Med*. 1998;157:A505.
56. Jaakkola MS, Jaakkola JJK, Becklake MR, Ernst P. Passive smoking and evolution of lung function in young adults. *J Clin Epidemiol*. 1995;48:317-327.
57. Sherrill DL, Holberg CJ, Enright PL, et al. Longitudinal analysis of the effects of smoking onset and cessation on pulmonary function. *Am J Respir Crit Care Med*. 1994;149:591-597.
58. Anthonisen NR, Connett JE, Kiley JP, et al. Effects of smoking intervention and the use of an inhaled anticholinergic bronchodilator on the rate of decline of FEV₁. *JAMA*. 1994;272:1497-1505.
59. Burchfield CM, Marcus EB, Curb JD, et al. Effects of smoking and smoking cessation on longitudinal decline in pulmonary function. *Am J Respir Crit Care Med*. 1995;151:1778-1785.
60. Buist AS, Nagy JM, Sexton GJ. The effect of smoking cessation on pulmonary function. *Am Rev Respir Dis*. 1979;120:953-957.
61. Bake B, Oxhøj H, Sixt R, Wilhelmsen L. Ventilatory lung function following two years of tobacco abstinence. *Scand J Respir Dis*. 1977;58:311-318.
62. Emmons KM, Abrams DB, Marshall R, et al. An evaluation of the relationship between self-report and biochemical measures of environmental tobacco smoke exposure. *Prev Med*. 1994;23:35-39.
63. Defino RJ, Ernst P, Jaakkola MS, et al. Questionnaire assessments of recent exposure to environmental tobacco smoke in relation to salivary cotinine. *Eur Respir J*. 1993;6:1104-1108.
64. Repace JL, Lowry AH. Indoor air pollution, tobacco smoke, and public health. *Science*. 1980;208:464-472.
65. Western B. Institutional mechanisms for unionization in sixteen OECD countries: an analysis of social survey data. *Social Forces*. 1994;73:497-519.