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Secondhand tobacco smoke exposure and pulmonary function: a cross-sectional study among non-smoking employees of bar and restaurants in Santiago, Chile

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ABSTRACT

Introduction The workplace remains a significant source of secondhand smoke (SHS) exposure. This pollutant is known to be associated with respiratory and cardiovascular problems, but its effects on specific pulmonary function parameters remain largely unexplored. The objectives of this study were to measure SHS exposure among non-smoking employees of bar and restaurants in Santiago, Chile and to evaluate the effects of such exposure on pulmonary function.

Methods Cross-sectional design. The study sample included non-smoking workers from 57 restaurants and bars in Santiago, Chile. The outcome variable was pulmonary function and the exposure variables were urine cotinine concentration, a biomarker for current SHS exposure, and years of SHS exposure in the workplace as proxy of chronic exposure. Personal and occupational variables were also recorded. Data analysis was performed using linear regression models adjusted by confounders.

Results The median age of the workers was 35 years and the median employment duration at the analysed venues was 1 year. Workers in smoking facilities reported greater SHS exposure (36 hours per week) than workers in smoke-free locations (4 hours per week). Urine cotinine levels were inversely correlated with forced vital capacity, but the finding was not statistically significant (β=−0.0002; 95% CI −0.007 to 0.006). Years of exposure to SHS showed to be significantly associated with forced expiratory flow$_{25/75}$ (β=−0.006; 95% CI −0.010 to −0.0004).

Conclusion These findings suggest that cumulative exposure to SHS at work may contribute to deterioration of pulmonary function in non-smoking employees.

Strengths and limitations of this study

- The effects of occupational secondhand smoke (SHS) exposure on specific pulmonary function parameters has been scarcely explored.
- This study is the first in Chile to evaluate occupational SHS exposure and its association with specific pulmonary function parameters.
- The use of the variable 'number of years exposed to SHS at workplace' was appropriate to study chronic SHS exposure.
- Our sample included mainly young workers being reasonable to infer that the employees did not accumulate enough years of SHS exposure to register greater changes in pulmonary function.
- Daily fluctuations of the timing of the spirometry measurements may have affected the results, since these were performed at different times of day, according to the availability and shifts of the workers and establishments.

INTRODUCTION

The secondhand smoke (SHS) is the smoke that remains in the air after someone has consumed tobacco, including the smoke coming from the burning end of the cigarette (side-stream smoke) and the smoke exhaled by the smoker (mainstream smoke).[1–5] Side-stream smoke contains higher concentration of harmful substances than main stream as it contains a greater amount of toxic gases and smaller particles that reach greater depth in the lungs when inhaled.[6] SHS is a common indoor pollutant in restaurants and bars that poses a serious health risk for non-smokers as it contains over 50 substances known to be carcinogenic in humans.[7 8] There is no known safe exposure level.[1 4] Some of the highest and most sustained occupational exposure to SHS occur in bar staff, with non-smoking areas providing only limited protection.[9]

SHS exposure can lead to the same health problems associated with active smoking,[1 7 8] with risk levels increasing as a function of hours of exposure.[10–14] Common scenarios associated with chronic SHS exposure include living with a spouse or parent who smokes and working in a location where smoking is allowed.[3 5] Previous studies have not been

consistent in showing a decline in specific pulmonary function parameters in people affected by SHS exposure at work or at home.[9 15–20] This lack of evidence may be attributable to the methods use to measure SHS exposure, which range from self-report to measurement of exposure biomarkers.[15–19]

One of the most common ways of measuring SHS exposure is measuring concentration of cotinine, the principle metabolite of nicotine. Cotinine can be measured in the blood or urine and shows high sensitivity and specificity for acute SHS exposure (over the past 3–4 days), although some authors have also used it to evaluate longer term exposure.[21–23] Chronic exposure to SHS has been measured through questionnaire and by hair nicotine concentration.[24 25]

In 2010, the time at which this study was performed, Chilean law prohibited tobacco smoking in public areas and workplaces. However, there were exceptions for 'hospitality' venues, such as casinos, bars, pubs, restaurants and cafés. Bars, pubs and restaurants with areas smaller than 100 m^2 could choose to allow smoking indoors or not, while facilities with an area larger than 100 m^2 were required to offer separate sections for smokers and non-smokers. Therefore, 'hospitality' workers were unprotected from SHS exposure, becoming the workplace, in many cases, the main source of SHS exposure.[26 27]

The objectives of this study were to measure SHS exposure among non-smoking workers in restaurants and bars in Santiago, Chile and to evaluate the effects of such exposure on pulmonary function.

METHODS

This cross-sectional study was performed as part of a larger project, 'Impact of involuntary exposure to tobacco smoke on respiratory health: study of pub and restaurant workers', carried out in Santiago, Chile between September 2010 and January 2011. This study was approved by the University of Chile School of Medicine's Ethics Committee.

Population and sample

The selection process for participating facilities has been previously described in detail.[28] In brief, the sampling framework included the five municipalities with the largest numbers of facilities, according to data provided by the National Institute of Statistics (in Spanish, Instituto Nacional de Estadísticas). Study staff visited 690 locations and used a brief survey to record the venue's name, address, type of facility (bar/pub, restaurant or other), smoking status (smoking allowed in all areas; designated smoking/non-smoking areas or smoke free) and number of non-smoking workers. Of the 690 facilities, 207 met inclusion criteria (be a bar–pub or restaurant and have non-smoking workers). Of them, 108 were visited or contacted by telephone to invite the owner or manager to participate in the study. In 63 establishments they agreed to participate (58%). For logistical reasons, only 59 of the facilities were included.[28] Smoking and non-smoking workers in these facilities were invited to participate in the main study. Only those who had not smoked in the last year were included in the current study. Workers were excluded if they did not provide a urine sample (n=5) or had a contraindication for spirometry (n=1).[29 30]

Outcome variables

Pulmonary function parameters: Certified personnel used an Easy One Diagnostic to measure forced vital capacity (FVC) and forced expiratory volume in 1 s (FEV_1), and then calculated the FEV_1 to FVC ratio (FEV_1/FVC) and forced expiratory flow (FEF) as 25%–75% of FVC (FEF_{25-75}). Spirometry measurements were performed during working hours. In compliance with international norms on collecting and interpreting spirometry data, age, sex, weight, height and race of each participant were also recorded.[29 30] A maximum of eight spirometry trials were performed. The criteria for including a participant's spirometry data in the analysis was achieving at least three acceptable and two reproducible trials, as described in the norms published by Spanish Society of Pneumology and Thoracic Surgery (Sociedad Española de Neumología y Cirugía Torácica (SEPAR)).[29 30] The equipment was calibrated weekly.

Exposure variables

Urine cotinine concentration. Each worker was asked to provide urine sample the morning after the spirometry measurements. The sample was provided, retrieved and frozen on the same day. Urine cotinine concentration was measured using ELISA at a sensitivity of 1 ng/mL. The cut-off value typically used in the literature to distinguish smokers from non-smokers is 10 ng/mL[31]. As a quality control, duplicate samples were obtained and analysed. There was a strong correlation between the original and duplicate samples (Spearman's correlation=0.96; p value=0.0005). Chronic exposure to SHS was measured as the number of years exposed to SHS at workplace (number of years worked at their three most recent job positions and whether it involved SHS exposure).

Covariables

The questionnaire included items about the participant's health history (asthma diagnosis, smoking habits); occupational history (job function at the facility, secondary employment at another facility, number of hours per day and days per week worked); occupational exposure (number of hours per day and days per week exposed to SHS) and the type of facility (smoking, mixed or non-smoking).

Statistical analysis

Data analysis was performed using the program STATA V.12. The quantitative variables were assessed for normality using the Shapiro-Wilk test. Descriptive statistics were calculated, including median and interquartile ranges (P_{25}–P_{75}) for quantitative variables and relative

frequency for qualitative variables. Quantitative exposure variables and covariables, such as number of hours per week of SHS exposure or age were dichotomised using the median as cut-off. Kruskal Wallis test and Wilcoxon test were used to assess difference of pulmonary parameters and exposure variables between the categories of the covariables. Finally, the association between pulmonary function parameters and exposure to SHS was analysed using multiple linear regression models adjusted by covariates potentially associated with both the outcome and the exposure considering a p value of <0.10[32] and the variables commonly controlled for in the literature.

RESULTS

We evaluated 92 non-smoking workers. A 18.5% were excluded due to spirometry results failed to meet the criteria for acceptability and reproducibility. The final sample was 75 workers. Median age was 35 years (P_{25}–P_{75} 19 to 68 years) and 61% of participants were male. Former smokers were 29.3% and the median of time they quit smoking was 8.5 years (P_{25}–P_{75} 2 to 15 years). They were homogeneously distributed at the different facility type. On average, participants had worked at the studied venue for 12 months. Independent of the facility type, the sample was mainly composed of waiting staff, bartenders and cashiers (70.7%). Workers in smoking facilities reported higher number of weekly hours exposed to SHS compared with workers in mixed and non-smoking facilities (p value=0.0001) (table 1).

As shown in table 2, we compared the results for pulmonary function and exposure to SHS based on covariables. Men had greater pulmonary function values than women, except for FEV_1/FVC ratio, where no differences were observed. No differences in pulmonary function were observed between former smokers and never smokers groups. In terms of the occupational exposure variables, employees working in the kitchen had lower values for FVC, FEV_1 and $FEF_{25/75}$ than the group of wait staff, bartenders, cashiers and managers. Regarding the number of hours per week of SHS exposure and pulmonary function, exposure greater than 26 hours per week was associated with a 0.02% decrease in FEV_1/FVC and a 230 mL decrease in $FEF_{25/75}$, although these results were not statistically significant. Workers in smoking venues had $FEF_{25/75}$ 400 mL lower and FEV_1/FVC ratios 0.03% lower than those of workers in non-smoking venues. In terms of urine cotinine concentration, although differences were observed between categories of job function and the hours per week exposed to SHS, these differences were strongly influenced by workplace's smoking policy. For example, in the case of wait staff/bartenders/cashiers working in venues where smoking was allowed, they had

Table 1 Characteristics of the study sample (Santiago, Chile 2010–2011)

	Smoking status restaurant/bar/pub				
	Total	**Smoking**	**Mixed**	**Non-smoking**	**p Value**
N° employees n (%)	75 (100)	27 (36.0)	31 (41.3)	17 (22.7)	
Sociodemographic characteristics					
Age, median (P_{25}–P_{75})	35.0 (19.0–62)	40.0 (29.0–52.0)	35.0 (21.0–57.0)	31.0 (22.0–44.0)	0.081*
Sex, n (%)					
Male	46 (61.3)	17 (63.0)	19 (61.3)	10 (59.0)	0.963†
Asthma, n (%)					
Yes	8 (10.7)	1 (3.7)	7 (22.6)	0	0.018†
No	67 (89.3)	26 (96.3)	24 (77.4)	17 (100)	
Occupational exposure					
Job function at the facility, n (%)					
Owners/managers	8 (10.7)	7 (25.9)	1 (3.2)	0	
Wait staff/bartenders/cashiers	53 (70.7)	13 (48.2)	27 (87.1)	13 (76.5)	0.005†
Cooks	14 (18.7)	7 (25.9)	3 (9.7)	4 (23.5)	
Number of months of work in the local, median (P_{25}–P_{75})	12.0 (0.08–8.0)	12.0 (1.0–192.0)	9.0 (1.0–468.0)	12.0 (2.0–60.0)	0.606*
Number of hour per week exposed to SHS, median (P_{25}–P_{75})	25.0 (0–77.0)	36.0 (21.0–56.0)	28.0 (6.0–48.0)	4.0 (2.0–7.0)	0.0001*
Number of years exposed to SHS workplace, median (P_{25}–P_{75})	2.2 (0–26.0)	3.0 (0.9–7.1)	2.2 (0.8–6.9)	1.5 (0.0–5.0)	0.369*

* Kruskal Wallis.
† χ^2.
SHS, secondhand smoke.

Table 2 Pulmonary function and exposure to secondhand smoke at non-smoking workers (Santiago, 2010–2011).

		Pulmonary function parameters				Urine cotinine concentration (ng/mL)	Number of years exposed to SHS workplace
	n	FVC (mL)	FEV_1 (mL)	FEV_1/FVC (%)	$FEF_{25/75}$ (mL)		
Variables		**Median (P_{25}–P_{75})**	**Median (P_{25}–P_{75})**	**Median (P_{25}–P_{75})**	**Median (P_{25}–P_{75})**	**Median (P_{25}-P_{75})**	**Median (P_{25}-P_{75})**
Sex							
Male	46	4.82 (4.23–5.42)	3.94 (3.41–4.38)	0.81 (0.76–0.84)	3.95 (3.00–4.66)	18.6 (6.2–39.5)	3.5 (1.0–11.3)
Female	29	3.48 (3.16–3.90)	2.89 (2.65–3.34)	0.81 (0.79–0.89))	3.25 (2.56–3.83)	13.6 (7.3–41.1)	1.0 (0.16–4.0)
p Value‡		0.0001	0.0001	0.116	0.014	0.944	0.01
Age*							
≤35 years	38	4.79 (3.93–5.36)	3.91 (3.37–4.38)	0.83 (0.79–0.88)	4.07 (3.27–4.59)	21.4 (5.1–40.7)	1.0 (0.25–5.0)
>36 year	37	3.78 (3.21–4.42)	2.95 (2.61–3.62)	0.80 (0.78–0.83)	3.12 (2.53–3.95)	15.2 (9.7–38.1)	4.0 (1.0–11.7)
p Value‡		0.0002	0.0001	0.049	0.0009	0.787	0.02
Smoking status							
Never smokers	53	4.23 (3.45–4.89)	3.49 (2.88–4.06)	0.81 (0.79–0.86)	3.69 (2.85–4.39)	21.7 (5.7–43.8)	1.0 (0.75–5.0)
Former smokers	22	4.33 (3.58–5.32)	3.53 (2.99–4.26)	0.81 (0.76–0.85)	3.77 (3.0–4.59)	12.9 (9.4–36.8)	6.3 (0.83–11.7)
p Value‡		0.767	0.684	0.452	0.907	0.629	0.04
Job function at the facility							
Owners/managers	8	4.84 (3.47–6.09)	3.94 (2.66–4.48)	0.77 (0.72–0.80)	3.22 (2.19–3.90)	41.0 (29.3–46.1)	1.0 (0.8–4.1)
Wait staff/bartenders/cashiers	53	4.42 (3.74–5.17)	3.56 (3.14–4.20)	0.82 (0.79–0.86)	3.94 (3.11–4.59)	13.2 (5.1–39.5)	3.0 (0.4–7.1)
Cooks	14	3.38 (2.96–4.24)	2.81 (2.56–3.62)	0.82 (0.79–0.86)	3.08 (2.53–3.80)	25.0 (9.7–36.9)	1.6 (0.8–4.0)
p Value†		0.03	0.04	0.04	0.03	0.08	0.711
Hours per week exposed to SHS*							
≤26 hours	39	4.05 (3.58–4.75)	3.44 (2.85–3.91)	0.82 (0.78–0.87)	3.81 (2.89–4.59)	11.3 (3.0–26.0)	2.0 (0.25–6.9)
>27 hours	36	4.40 (3.45–5.40)	3.64 (2.89–4.32)	0.80 (0.77–0.84)	3.58 (2.78–4.38)	35.8 (11.6–48.1)	3.3 (0.9–7.04)
p Value‡		0.279	0.457	0.173	0.603	0.0003	0.474
Facility							
Smoking/mixed	58	4.24 (3.32–5.26)	3.49 (2.85–4.23)	0.81 (0.77–0.84)	3.58 (2.73–4.44)	21.8 (10.5–44.7)	2.6 (0.9–7.0)
Non-smoking	17	4.24 (3.83–4.55)	3.49 (3.28–3.83)	0.84 (0.80–0.88)	3.98 (3.25–4.48)	4.1 (1.5–26.0)	1.5 (0–5.0)
p Value‡		0.825	0.845	0.06	0.176	0.0012	0.161

*Variable dichotomised in median value.
†Kruskal Wallis test.
‡Wilcoxon test.
FEF, forced expiratory flow; FEV, forced expiratory volume; FVC, forced vital capacity; SHS, secondhand smoke.

Table 3 Bivariate association of pulmonary function parameters in non-smokers workers according to covariables of interest

	FVC (mL)	FEV_1 (mL)	FEV_1/FVC (%)	$FEF_{25/75}$ (mL)
	β	β	β	β
	(95% CI)	(95% CI)	(95% CI)	(95% CI)
Sociodemographic variables				
Sex				
Male	1.26	0.91	-0.03	0.61
	(0.880 to 1.650)	(0.601 to 1.213)	(–0.064 to –0.0003)	(0.110 to 1.103)
Age				
	–0.03	–0.03	-0.001	–0.04
	(–0.05 to –0.02)	(–0.04 to –0.02)	(–0.003 to –0.003)	(–0.055 to –0.019)
Weight				
	0.04	0.02	-0.001	0.01
	(0.02 to 0.05)	(0.01 to 0.04)	(–0.002 to –0.0001)	(–0.004 to 0.034)
Size				
	0.08	0.06	-0.001	0.05
	(0.07 to 0.10)	(0.050 to 0.074)	(–0.002 to 0.001)	(0.029 to 0.076)
Asthma				
Yes	0.04	–0.17	-0.05	–0.67
	(–0.731 to 0.802)	(–0.750 to 0.422)	(–0.100 to –0.010)	(–1.470 to 0.122)
Occupational exposure variables				
Job function at the facility				
Wait staff/bartenders/cashiers Reference		Reference	Reference	Reference
Owners/managers	0.37	0.003	-0.06	–0.83
	(–0.370 to 1.110)	(–0.570 to 0.570)	(–0.113 to –0.021)	(–1.613 to –0.047)
Cooks	–0.70	–0.64	-0.02	–0.77
	(–1.290 to –0.120)	(–1.090 to –0.190)	(–0.061 to 0.022)	(–1.391 to –0.151)
Hours per week exposed to SHS				
	0.01	0.01	-0.0004	0.002
	(0.002 to 0.020)	(–0.0005 to 0.014)	(–0.001 to 0.0002)	(–0.008 to 0.011)
Facility				
Non-smoking	Reference	Reference	Reference	Reference
Smoking/mixed	0.10	–0.05	-0.03	–0.41
	(–0.460 to 0.672)	(–0.486 to 0.381)	(–0.071 to 0.003)	(–1.003 to 0.177)

FEF, forced expiratory flow; FEV, forced expiratory volume; FVC, forced vital capacity.

a median urinary cotinine concentration of 40.7 ng/mL. Employees working in mixed venues (with smoking and non-smoking areas) had a median of 13.5 ng/mL and those who working in smoke-free venues had a median of 2.5 ng/mL. In the same way, the information regarding urinary cotinine concentration in people working over 27 hours per week exposed to SHS in venues where smoking was allowed was 45.2 ng/mL, in those working in mixed venues the median was 13.6 ng/mL and in those working in smoke-free venues the median was 2.0 ng/mL. The number of years exposed to SHS workplace varied according to sex, age and smoking status of employees.

Consistent with the literature, sex, age and weight were significantly associated with pulmonary function parameters (table 3). In terms of job function, the owners and managers had FEV_1/FVC values 60% lower and $FEF_{25/75}$ values 830 mL lower than the group of wait staff, bartenders and cashiers. The kitchen workers had 700 mL lower FVC values, 640 mL lower FEV_1 values and 772 mL lower $FEF_{25/75}$ than the group of wait staff, bartenders and cashiers. Workers in smoking facilities had 413 mL lower $FEF_{25/75}$ and 3% lower FEV_1/FVC than workers in non-smoking venues.

Association between pulmonary function and SHS exposure

The crude model revealed that the association between pulmonary function and urine cotinine concentration was not statistically significant (table 4). The multivariate

Table 4 Crude and adjusted association between pulmonary function parameters and SHS exposure of non-smoking workers of bars and restaurants

	FVC (mL)		FEV_1 (mL)		FEV_1/FVC (%)		FEF25/75 (mL)	
	β (95% CI)	R^2	β (95% CI)	R^2	β (95% CI)	R^2	β (95% CI)	R^2
Urine cotinine								
Crude model	0.002	0.002	0.002	0.003	0.0002	0.002	0.002	0.002
	(−0.010 to 0.010)		(−0.010 to 0.010)		(−0.001 to 0.001)		(−0.010 to 0.010)	
Adjusted model	−0.0002	0.781	0.001	0.795	0.0004	0.33	0.005	0.672
	(−0.007 to 0.006)*		(−0.003 to 0.006)*		(−0.0003 to 0.001)†		(−0.006 to 0.015)†	
Number of years exposed to SHS at work								
Crude model	−0.025	0.046	−0.022	0.061	−0.0008	0.013	−0.022	0.032
	(−0.051 to 0.002)		(−0.042 to −0.001)		(−0.002 to 0.0008)		(−0.050 to 0.006)	
Adjusted model	−0.013	0.79	−0.01	0.802	0.0006	0.324	−0.006	0.964
	(−0.030 to 0.0025)*		(−0.022 to 0.002)*		(−0.001 to 0.002)†		(−0.010 to −0.0004)†	

*Adjusted by sex, age, weight, size and job function at the facility.
†Adjusted by sex, age, size, asthma status and job function at the facility.
FEF, forced expiratory flow; FEV, forced expiratory volume; FVC, forced vital capacity.

analysis was based on a parsimonious model that included the covariate 'job function', as this variable was related to pulmonary function and urine cotinine concentration with a p value <0.10, as well as the variables sex, age, weight, height and asthma status, all of which are recognised as variables that affect pulmonary function according to SEPAR.[29 32] The adjusted model did not demonstrate a significant association between urine cotinine concentration and decreased pulmonary function. Conversely, the number of years of SHS exposure in workplace showed an inverse and significant association with FEV_1. Each year of SHS exposure was associated with a 200 mL decrease in FEV_1 (95% CI −0.042 to −0.001). The other pulmonary function variables were also inversely associated with years of SHS exposure in workplace, although the association in these cases did not reach significance. The adjusted model showed an inverse and in some cases statistically significant association between the number of years of SHS exposure and pulmonary function parameters, specifically in $FEF_{25/75}$ (β=−0.006; 95% CI −0.010 to −0.0004).

DISCUSSION

This study is the first in Chile to evaluate occupational SHS exposure and its association with specific pulmonary function parameters. We did not find an inverse association between pulmonary function parameters and urine cotinine concentration, but when we considered number of years exposed to SHS in workplace, we found an inverse association with FVC , FEV_1, FEV_1/FVC and $FEF_{25/75}$ being significant only for the last parameter. Similar findings were described by other researchers who reported a reduction in FVC and $FEF_{25/75}$,[20 33] in FVC,[15] in FVC and FEV1[16] in subjects exposed to environmental tobacco smoke. In terms of job function, kitchen workers showed lower pulmonary function values than the group of wait staff, bartenders and cashiers as compared with the owners and managers. One possible explanation for these findings is that the SHS exposure had an additive effect with exposure to other pollutants emitted in the kitchen. In the literature has been reported that workers in kitchens with gas stoves show lower pulmonary function parameters than those in kitchens with electric stoves due to greater exposure to toxic substances in the air after cooking with gas.[34] In our study, it was not possible to analyse differences according this variable because 100% of the establishments used gas stoves.

As noted above, we did not find a significant association between pulmonary function parameters and urine cotinine concentration. A possible explanation for these results is that urine cotinine levels reflect recent exposure to tobacco smoke[21 22 31] while chronic exposure to SHS is likely implicated in a decline in pulmonary function parameters. In fact, in table 4 we can see that the proportion of the variance (R^2) explained by number of years of SHS exposure in workplace is greater than that explained by the current urine cotinine concentration, suggesting that this variable (number of years of SHS exposure) may be more appropriate when we are studying chronic effects. Other studies that have addressed this topic have produced varying results[15–17 20–22 31 35] reported a significant inverse association between SHS exposure

(evaluated through self-report) and FVC and FEV_1. As in our study, Chen *et al* did not find a significant association when serum cotinine was assess as exposure variable but did when exposure to SHS was measured through self-report.[16]

Our results are not as strong as those described in other studies.[12–14 20 33] It should be noted that our sample included mainly young workers, being reasonable to infer that the sample not accumulated sufficient years of SHS exposure to register significant changes in pulmonary function. Also the median time worked at the location was only about 1 year. About 25% of the sample had worked at the given facility for less than 3 months, and 75% of the sample had worked at the location for fewer than 2 years. This condition of high turnover rate, along with the relative youth of the workers contributes to assume that the sample not accumulated enough years of SHS exposure to register significant changes in pulmonary function. A second limitation was that although all participants were non-smokers, those who worked in non-smoking venues reported be exposed to SHS at least 4 hours a week. Also in this group the median urine cotinine concentration was 4.1 ng/mL. The lack of a true control group could have lead to underestimating the effect of SHS exposure. Another potential limitation was the timing of the spirometry measurements. The literature reports that pulmonary function varies throughout the day according to circadian rhythm, decreasing from a high point in the early morning until about noon, and then rising again to peak between about 4 and 5 in the afternoon. These daily fluctuations may have affected the results, as the lung function measurements were performed at different times of day, according to the availability and shifts of the workers and establishments. Finally, our small sample size along with the weak correlation between exposure to SHS and pulmonary function prevent us to have enough power to demonstrate a strongest association as shown in other studies.

Notwithstanding the above, our study shows that exposure to SHS among non-smoking employees working in venues where smoking is allowed appear to be substantially higher than those found in employees working in venues where smoking is not allow. The median urine cotinine in non-smoking employees working in a venue were smoking is allowed was 38.1 ng/mL, in a mixed venue was 12.5 ng/mL and where smoking was not allowed was 4.1 ng/mL. Given that SHS is a proven carcinogen in humans to which non-smoking workers of this type of venues are exposed involuntarily, a total smoking ban would provide a major protection to employees working in such venues.

CONCLUSION

The years of exposure to SHS in workplace as proxy of chronic exposure were inverse and significantly associated with the $FEF_{25/75}$ and inverse but not significant with FVC and FEV_1. These findings suggest that cumulative exposure to SHS at work may contribute to deterioration of pulmonary function in non-smoking employees.

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