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Secondhand tobacco smoke exposure and pulmonary function among non-smoking employees of bar and restaurants in Santiago, Chile

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Secondhand tobacco smokeexposure and pulmonary function among non-smoking employees of bar and restaurantsin 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 restaurantsin 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 (FVC), 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 FEF25 / 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.

Keywords: Secondhand smoke exposure, chronic exposure, pulmonary function, urine cotinine, workers.

Strengths and limitations of this study

- The effects of occupational 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 studied chronic SHS exposure.
- Our sample included mainly young workers being reasonable to infer that the sample not accumulated sufficient 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 various times of day, according to the availability and shifts of the workers and establishments.

Introduction

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 and the smoke exhaled by the smoker [1, 2, 3, 4]. 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. There is no known safe exposure level [1, 4].

Because SHS contains the same toxic substances that a smoker inhales, SHS exposure can lead to the same health problems associated with active smoking [5], with risk levels increasing as a function of hours of exposure [6, 7, 8, 9, 10]. 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 [2, 4,]. 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 (Table 1). This lack of evidence may be attributable to the methods use to measure SHS exposure, which range from self-report [11, 12, 13, 14] to measurement of exposure biomarkers [11, 12, 13, 14, 15].

Table 1. Effects of passive smoking on pulmonary function parameters

Author (year)	Sample size	Exposure assessment	Source of exposure	Main results
Kunzli et al. (2000)	3534	Questionnaire	Occupational	FEV_1 (β= -0.1%; CI95% -1.3 to 1.1%) FVC (β= -0.7%; CI95% -0.4 to 1.8%) FEF 25/75 (β=-1.9%; CI95% -4.2 to 0.5%)
Janson et al. (2001)	7882	Questionnaire	Total exposure*	FEV_1 (β= -63 ml; CI95% -111 to -15 ml)
Chen et al. (2001)	301	Questionnaire. Are you regularly exposed to tobacco smoke from other people? Three sources of exposure of SHS were given; workplace, home, and other places. On average, for how many hours a day are you exposed to other people's tobacco smoke?. Blood cotinine	Occupational	FEV_1 (β= -254 ml; CI95% -84 to -240 ml) FVC (β= -273 ml; CI95% -60 to -480 ml)
Eisner (2002)	10581	Questionnaire. Does anyone who lives here smoke cigarettes in the home? At work, how many hours per day are you close enough of people who smoke so that you can smell the smoke? Exposure dif was ≥ 1 hr a day. Blood cotinine.	Total exposure*	FEV_1(β= -100 ml; CI95% -143 to -56 ml) FVC (β= -119 ml; CI95% -168 to -69 ml) FEV_1/CVF (β= -1,77%; CI 95% -2.18 to -1.36%)
Fidan et al. (2004)	207	Questionnaire	Occupational	FEV_1 (β= -5.1%; p value=0.011) FVC (β= -3.4%; p value=0.080)
Alipour et al. (2005)	302	Questionnaire	Occupational Total exposure*	FEV_1 (β= 2.45%; CI95% -5.17 to -0.28%) FEV_1 (β= 2.90%; CI95% -5.59 to -0.23%) FVC (β= -3.16%; CI95% -5.67 to -0.64%) FEF 25/75 (β= -9.87%; p value=0.009)
Fahim et al. (2012)	55	Questionnaire	Occupational	FVC (β= -6%; p value=0.041) VEF_1/FVC (β= -4.2%; p value=0.001) FEF_{75} (β= -7.5%; p value=0.017)

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 [16, 17, 18]. Chronic exposure to SHS has been measured through questionnaire and by hair nicotine concentration [19, 20].

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 than100 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 nonsmokers. Therefore, "hospitality" workers were unprotected from SHS exposure, becoming the workplace, in many cases, the main source of SHS exposure [21, 22].

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 [23]. In brief, the sampling framework included the 5 municipalities with the largest numbers of facilities, according to data provided by the National Institute of Statistics (Spanish acronym INE, for *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[23]. Non-smoking workers in these facilities were then invited to participate in the study. Workers were excluded if they did not provide a urine sample (n=5) or had a contraindication for spirometry (n=1) [24, 25]. A total of 92 non-smoking workers participated in the study after providing written informed consent.

Outcome variables

Pulmonary function parameters: Certified personnel used an *Easy One Diagnostic*® to measure forced vital capacity (FVC) and forced expiratory volume in 1s (FEV_1), and then calculated the FEV_1 to FVC ratio (FEV_1/FVC) and forced expiratory flow 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 [24, 25]. A maximum of 8 spirometry trials were performed. The criteria for including a participant's spirometry data in the analysis was achieving at least 3 acceptable and 2 reproducible trials, as described in the norms published by Spanish Society of Pneumology and Thoracic Surgery (Spanish acronym SEPAR, for *Sociedad Española de Neumología y Cirugía Torácica*) [24, 25]. 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 [26]. As a quality control, duplicate samples were obtained and analyzed. 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 3 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 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 dichotomized using the median as cutoff. 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 analyzed using multiple linear regression models adjusted by covariates

potentially associated with both, the outcome and the exposure considering a p-value of<0.10 [27], as well as variables commonly controlled for in the literature.

Results

A total of 17 participants (18.5%) were excluded due to spirometry results that failed to meet the criteria for acceptability and reproducibility. The final sample was 75 workers. Median age was 35 years (P_{25}–P_{75}: 19–68 years), and 61% of participants were male. 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 (58.7%), followed by owners or managers (28%), and finally cooks (13.3%). The number of hours worked per week was similar for workers in smoking, mixed, and non-smoking facilities. Workers in smoking facilities reported higher number of weekly hours and number of years exposed to SHS compared to workers in mixed and non-smoking facilities (Table 2).

Table 2. Characteristics of the study sample. Santiago, Chile 2010-2011.

	Smoking status restaurant/bar/pub		
	Smoking	**Mixed**	**Non-smoking**
Nº employees (%)	27 (36.0)	31 (41.3)	17 (22.7)
Sociodemographic characteristics			
Age, Median (P_{25}-P_{75})	40.0 (32.0-47.0)	35.0 (24.0-47.0)	31.0 (23.0-42.0)
Sex, n (%)			
Male	17 (63.0)	19 (61.3)	10 (59.0)
Scholarship, n (%)			
≤8 years	3 (11.1)	2 (6.5)	-
9-12 years	11 (40.8)	19 (61.3)	11 (64.7)

>12 years	13 (48.1)	10 (32.2)	6 (35.3)
Asthma, n (%)			
Yes	1 (3.7)	7 (22.6)	-
No	26 (96.3)	24 (77.4)	17 (100)
Occupational exposure			
Job function at the facility, n (%)			
Owners/managers	7 (25.9)	1 (3.2)	13 (76.5)
Wait staff/bartenders/cashiers	13 (48.2)	27 (87.1)	4 (23.5)
Cooks	7 (25.9)	3 (9.7)	-
Number of months of work in the local, Median (P_{25}-P_{75})	12.0 (1.0-192.0)	9.0 (1.0-468.0)	12.0 (2.0-60.0)
Number of weekly working hours, Median (P25-P75)	48.0 (40.0-54.0)	48.0 (40.0-60.0)	45.0 (40.0-48.0)
Number of hour per week exposed to SHS, Median (P_{25}-P_{75})	36.0 (21.0-56.0)	28.0 (6.0-48.0)	4.0 (2.0–7.0)
Number of years exposed to SHS workplace, Median (P_{25}-P_{75})	3.0 (0.9-7.1)	2.2 (0.8-6.9)	1.5 (0.0-5.0)

As shown in Table 3 we compared the results for pulmonary function and urine cotinine concentration based on covariables. Males had greater pulmonary function values than females, except for FEV_1/FVC ratio, where no differences were observed. 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, owners and managers had the highest levels, followed by kitchen workers

and then finally the group of wait staff, bartenders, and cashiers (44.4 ng/ml, 25.0 ng/ml, and 13.2 ng/ml, respectively). Urine cotinine concentration varied by number of hours per week of SHS exposure as self-reported by participants and by the smoking status of the facility. Workers with over 26 hours per week of SHS exposure had urine cotinine values 24.5 ng/ml higher than those who reported 26 or fewer hours of exposure per week, while workers in smoking facilities show levels of urine cotinine 17.7 ng/ml higher than workers in non-smoking facilities.

Table 3. Urine cotinine concentration and pulmonary function at non-smoking workers. Santiago, 2010-2011.

Variables		Pulmonary function parameters				Urine cotinine concentration (ng/ml)
	n	FVC ml (RIC)*	FEV_1 ml (RIC)*	FEV_1/FVC (%)	$FEF_{25\%/75\%}$ (ml)	Med (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)
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)
p value ±		0.0001	0.0001	0.116	0.014	0.944
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)
>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)
p value ±		0.0002	0.0001	0.049	0.0009	0.787
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)	44.4 (29.3-46.1)
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)
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)
p value +		0.03	0.04	0.04	0.03	0.08
Hours per week exposed to SHS						

≤26 hrs*	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)
>27 hrs	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 (11.6-48.2)
p value±		0.279	0.457	0.173	0.603	0.0003
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
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
p value ±		0.825	0.845	0.06	0.176	0.0012

*Variable dichotomized in median value; + Kruskal Wallis test; ±Wilcoxon Test

Consistent with the literature, sex, age and weight were significantly associated with pulmonary function parameters (Table 4). 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.

Table 4. Bivariate association of pulmonary function parameters in non-smokers workers according to covariables of interest.

	FVC (ml)		FEV_1 (ml)		FEV_1/FVC (ml)		$FEF_{25/75}$ (ml)	
	β (CI95%)	R^2	β (CI95%)	R^2	β (CI95%)	R^2	β (CI95%)	R^2
Sociodemographic variables								
Sex								
Male	1,260 (0.880 to 1.650)	0.371	0.91 (0.601 to 1.213)	0.321	-0.03 (-0.064 to -0.0003)	0.053	0.61 (0.110 to 1.103)	0.076
Age	-0.03 (-0.05 to -0.02)	0.161	-0.03 (-0.04 to -0.02)	0.237	-0.001 (-0.003 to -0.003)	0.083	-0.037 (-0.055 to -0.019)	0.189
Weight	0.04	0.207	0.02	0.154	-0.001	0.056	0.014	0.029

	(0.02 to 0.05)		(0.01 to 0.04)		(-0.002 to -0.0001)		(-0.004 to 0.034)	
Size								
	0.08	0.596	0.06	0.595	-0.001	0.009	0.052	0.222
	(0.07 to 0.10)		(0.050 to 0.074)		(-0.002 to 0.001)		(0.029 to 0.076)	
Asthma								
Yes	0.04	0.001	-0.17	0.004	-0.054	0.071	-0.673	0.038
	(-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	*Ref.*	0.097	*Ref.*	0.104	*Ref.*	0.1	*Ref.*	0.113
Owners/managers	0.37		0.003		-0.06		-0.828	
	(-0.370 to 1.110)		(-0.570 to 0.570)		(-0.113 to -0.021)		(-1.613 to -0.047)	
Cooks	-0.7		-0.64		-0.02		-0.772	
	(-1.290 to -0.120)		(-1.090 to -0.190)		(-0.061 to 0.022)		(-1.391 to -0.151)	
Hours per weekworked								
	0.001	0.0003	-0.003	0.003	-0.001	0.034	-0.014	0.035
	(-0.02 to 0.02)		(-0.02 to 0.01)		(-0.002 to 0.0002)		(-0.02 to 0.003)	
Hours per weekexposedto SHS								
	0.01	0.077	0.01	0.046	-0.0004	0.022	0.002	0.002
	(0.002 to 0.020)		(-0.0005 to 0.014)		(-0.001 to 0.0002)		(-0.008 to 0.011)	
Years of work								
	-0.01	0.005	-0.001	0.0001	0.003	0.061	0.028	0.017
	(-0.06 to 0.03)		(-0.04 to 0.04)		(0.0003 to 0.006)		(-0.021 to 0.078)	
Facility								
Non-smoking	*Ref.*	0.002	*Ref.*	0.001	*Ref.*	0.044	*Ref.*	0.026
Smoking/mixed	0.1		-0.05		-0.03		-0.413	
	(-0.460 to 0.672)		(-0.486 to 0.381)		(-0.071 to 0.003)		(-1.003 to 0.177)	

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 5). The multivariate 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 recognized as variables that affect pulmonary function according to SEPAR [24, 27]. The

adjusted model did not demonstrate a significant relation 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).

Table 5. 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 (ml)		FEF25/75 (ml)	
	β (CI95%)	R^2	β (CI95%)	R^2	β (CI95%)	R^2	β (CI95%)	R^2
Urine cotinine								
Crude model	0.002 (-0.010 to 0.010)	0.002	0.002 (-0.010 to 0.010)	0.003	0.0002 (-0.001 to 0.001)	0.002	0.002 (-0.010 to 0.010)	0.002
Adjusted model	-0.0002 (-0.007 to 0.006)*	0.781	0.001 (-0.003 to 0.006)*	0.795	0.0004 (-0.0003 to 0.001)+	0.33	0.005 (-0.006 to 0.015)+	0.672
Number of years exposed to SHS at work								
Crude model	-0.025 (-0.051 to 0.002)	0.0462	-0.022 (-0.042 to -0.001)	0.061	-0.0008 (-0.002 to 0.0008)	0.013	-0.022 (-0,050 to 0,006)	0.032
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

Discussion

This study is the first in Chile to evaluate occupational SHS exposure and its association with specific pulmonary function parameters. The results indicate that there was an inverse association between the number of years of SHS exposure in workplace and pulmonary function parameters as FEV_1 and $FEF_{25/75}$. In terms of job function, kitchen workers showed lower pulmonary function values than the group of wait staff, bartenders, and cashiers as compared to 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 [28]. In our study, it was not possible to analyze differences according this variable because 100% of the establishments used gas stoves.

Although the present study was not able to find a significant association between FVC and urine cotinine concentration a trend can be observed. A possible explanation for these results is the use of urine cotinine concentration as biomarker of exposure. As noted above, urine cotinine levels reflect recent exposure to tobacco smoke[16, 17, 26] while chronic exposure to SHS is likely implicated in a decline in pulmonary function parameters. In fact, analysis of the exposure variable *number of years of SHS exposure in workplace* (including the 3 most recent job positions) did reveal significant associations between SHS exposure

and FEV_1 (β= -0.022; 95% CI -0.042 to -0.001) and $FEF_{25/75}$ (β= -0.006; 95% CI -0.010 to -0.0004), suggesting that this variable is useful in studies of cumulative SHS exposure. It should also 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. Other studies that have addressed this topic have produced varying results[11, 12, 13, 16, 17, 26, 29, 30] reported a significant inverse association between SHS exposure (evaluated through self-report) and FVC and FEV_1. In our study, self-reported SHS exposure measured in *hours per week* was inversely correlated with FEV_1/FVC and FEF_{25-75}, but the association did not reach significance. 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 [12].

A possible limitation of this study was that 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.

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 various times of day, according to the availability and shifts of the workers and establishments.

While this study did not find a significant association between years of SHS exposure in workplace and urine cotinine concentration (β=0.060, p-value=0.264; R^2=0.017) there was a significant association between weekly hours of exposure and urine cotinine (β= 0.365, p-value<0.001; R^2=0.24). This finding suggests that a self-reported weekly hour of exposure is an acceptable qualitative biomarker of recent exposure if quantitative measurements are not available.

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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Competing interest

The authors have no conflict of interest to declare**.**

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Contributorship statement

Parro Javiera. Substantial contributions to the conception and design of the work on pulmonary function parameters; acquisition, analysis and interpretation of data, drafting the work, and final approval of the version to be published;

Aceituno Paulina. Substantial contributions to the conception and design of the work, revising it critically for important intellectual content, final approval of the version to be published.

Droppelman Andrea. Substantial contributions to the conception, design of the work and interpretation of exposure data, final approval of the version to be published.

Mesías Sthepanie. Substantial contributions to the acquisition and analysis of exposure data, final approval of the version to be published.

Muñoz Claudio. Substantial contributions to the acquisition, analysis and interpretation of data, final approval of the version to be published.

Marchetti Nella. Substantial contributions to the conception of the work and interpretation of data, final approval of the version to be published.

Iglesias Verónica. Substantial contributions to the conception and design of the work, analysis and interpretation of data for the work; drafting the work, final approval of the version to be published, agreement to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

STROBE Statement—checklist of items that should be included in reports of observational studies

	Item No	Recommendation
Title and abstract	1	(*a*) Indicate the study's design with a commonly used term in the title or the abstract YES
		(*b*) Provide in the abstract an informative and balanced summary of what was done and what was found YES
Introduction		
Background/rationale	2	Explain the scientific background and rationale for the investigation being reported YES
Objectives	3	State specific objectives, including any prespecified hypotheses YES
Methods		
Study design	4	Present key elements of study design early in the paper YES
Setting	5	Describe the setting, locations, and relevant dates, including periods of recruitment, exposure, follow-up, and data collection YES
Participants	6	(*a*) *Cohort study*—Give the eligibility criteria, and the sources and methods of selection of participants. Describe methods of follow-up *Case-control study*—Give the eligibility criteria, and the sources and methods of case ascertainment and control selection. Give the rationale for the choice of cases and controls *Cross-sectional study*—Give the eligibility criteria, and the sources and methods of selection of participants YES
		(*b*) *Cohort study*—For matched studies, give matching criteria and number of exposed and unexposed *Case-control study*—For matched studies, give matching criteria and the number of controls per case
Variables	7	Clearly define all outcomes, exposures, predictors, potential confounders, and effect modifiers. Give diagnostic criteria, if applicable YES
Data sources/ measurement	8*	For each variable of interest, give sources of data and details of methods of assessment (measurement). Describe comparability of assessment methods if there is more than one group YES
Bias	9	Describe any efforts to address potential sources of bias YES
Study size	10	Explain how the study size was arrived at YES
Quantitative variables	11	Explain how quantitative variables were handled in the analyses. If applicable, describe which groupings were chosen and why YES
Statistical methods	12	(*a*) Describe all statistical methods, including those used to control for confounding
		(*b*) Describe any methods used to examine subgroups and interactions
		(*c*) Explain how missing data were addressed
		(*d*) *Cohort study*—If applicable, explain how loss to follow-up was addressed *Case-control study*—If applicable, explain how matching of cases and controls was addressed *Cross-sectional study*—If applicable, describe analytical methods taking account of sampling strategy YES
		(*e*) Describe any sensitivity analyses NO

Continued on next page

Results		
Participants	13*	(a) Report numbers of individuals at each stage of study—eg numbers potentially eligible, examined for eligibility, confirmed eligible, included in the study, completing follow-up, and analysed YES
		(b) Give reasons for non-participation at each stage YES
		(c) Consider use of a flow diagram NO
Descriptive data	14*	(a) Give characteristics of study participants (eg demographic, clinical, social) and information on exposures and potential confounders YES
		(b) Indicate number of participants with missing data for each variable of interest YES
		(c) *Cohort study*—Summarise follow-up time (eg, average and total amount)
Outcome data	15*	*Cohort study*—Report numbers of outcome events or summary measures over time
		Case-control study—Report numbers in each exposure category, or summary measures of exposure YES
		Cross-sectional study—Report numbers of outcome events or summary measures YES
Main results	16	(*a*) Give unadjusted estimates and, if applicable, confounder-adjusted estimates and their precision (eg, 95% confidence interval). Make clear which confounders were adjusted for and why they were included YES
		(*b*) Report category boundaries when continuous variables were categorized YES
		(*c*) If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period NO
Other analyses	17	Report other analyses done—eg analyses of subgroups and interactions, and sensitivity analyses NO
Discussion		
Key results	18	Summarise key results with reference to study objectives YES
Limitations	19	Discuss limitations of the study, taking into account sources of potential bias or imprecision. Discuss both direction and magnitude of any potential bias YES
Interpretation	20	Give a cautious overall interpretation of results considering objectives, limitations, multiplicity of analyses, results from similar studies, and other relevant evidence YES
Generalisability	21	Discuss the generalisability (external validity) of the study results YES
Other information		
Funding	22	Give the source of funding and the role of the funders for the present study and, if applicable, for the original study on which the present article is based YES

*Give information separately for cases and controls in case-control studies and, if applicable, for exposed and unexposed groups in cohort and cross-sectional studies.

Note: An Explanation and Elaboration article discusses each checklist item and gives methodological background and published examples of transparent reporting. The STROBE checklist is best used in conjunction with this article (freely available on the Web sites of PLoS Medicine at http://www.plosmedicine.org/, Annals of Internal Medicine at http://www.annals.org/, and Epidemiology at http://www.epidem.com/). Information on the STROBE Initiative is available at www.strobe-statement.org.

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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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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 (FVC), 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 FEF25 / 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.

Keywords: Secondhand smoke exposure, chronic exposure, pulmonary function, urine cotinine, workers.

Strengths and limitations of this study

- The effects of occupational 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 studied chronic SHS exposure.
- Our sample included mainly young workers being reasonable to infer that the sample not accumulated sufficient 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 various 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, 2, 3, 4, 5]. Exposure to side-stream smoke is more harmful than exposure to mainstream smoke 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, 11, 12, 13, 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, 16, 17, 18, 19, 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, 16, 17, 18, 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, 22, 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 than100 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 nonsmokers. 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 5 municipalities with the largest numbers of facilities, according to data provided by the National Institute of Statistics (Spanish acronym INE, for *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 1s (FEV_1), and then calculated the FEV_1 to FVC ratio (FEV_1/FVC) and forced expiratory flow 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 8 spirometry trials were performed. The criteria for including a participant's spirometry data in the analysis was achieving at least 3 acceptable and 2 reproducible trials, as described in the norms published by Spanish Society of Pneumology and Thoracic Surgery (Spanish acronym SEPAR, for *Sociedad Española de Neumología y Cirugía Torácica*) [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 analyzed. 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 3 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 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 dichotomized using the median as cutoff. 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 analyzed 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], as well as variables commonly controlled for in the literature.

Results

The non-smoking workers evaluated in the study were 92. 17(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}: 19–68 years), and 61% of participants were male. 29.3% were former smokers and the median of time they quit smoking was 8.5 years (RIC 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 (58.7%) followed by owners or managers (28%), and finally cooks (13.3%). Workers in smoking facilities reported higher number of weekly hours exposed to SHS compared to workers in mixed and non-smoking facilities (p-value=0.0001) (Table 1).

Table 1. Characteristics of the study sample. Santiago, Chile 2010-2011.

	Smoking status restaurant/bar/pub				
	Total	**Smoking**	**Mixed**	**Non-smoking**	**p value**
Nº 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)	-	0.018**
No	67 (89.3)	26 (96.3)	24 (77.4)	17 (100)	
Occupational exposure					
Job function at the facility, n (%)					
Owners/managers	21 (28.0)	7 (25.9)	1 (3.2)	13 (76.5)	
Wait staff/bartenders/cashiers	44 (58.7)	13 (48.2)	27 (87.1)	4 (23.5)	0.005**
Cooks	10 (13.3)	7 (25.9)	3 (9.7)	-	
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; ** Chi2.

As shown in Table 2 we compared the results for pulmonary function and exposure to SHS based on covariables. Males had greater pulmonary function values than females, 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, owners and managers had the highest levels, followed by kitchen workers and then finally the group of wait staff, bartenders, and cashiers (44.4 ng/ml, 25.0 ng/ml, and 13.2 ng/ml, respectively). Urine cotinine concentration varied by number of hours per week of SHS exposure as self-reported by participants and by the smoking status of the facility. Workers with over 26 hours per week of SHS exposure had urine cotinine values 24.5 ng/ml higher than those who reported 26 or fewer hours of exposure per week, while workers in smoking facilities show levels of urine cotinine 17.7 ng/ml higher than workers in non-smoking facilities. The number of years exposed to SHS workplace varied according to sex, age and smoking status of employees.

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

Variables	n	Pulmonary function parameters				Urine cotinine concentration (ng/ml)	Number of years exposed to SHS workplace
		FVC ml (RIC)	FEV_1 ml (RIC)	FEV_1/FVC % (RIC)	$FEF_{25\%/75\%}$ ml (RIC)	Med (P_{25}-P_{75})	Med (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)	44.4 (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 hrs	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 hrs	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)	1.5 (0-5.0)
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)	2.6 (0.9-7.0)

p value ±	0.825	0.845	0.06	0.176	0.0012	0.161

*Variable dichotomized in median value; + Kruskal Wallis test; ±Wilcoxon Test

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.

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 (ml)	$FEF_{25/75}$ (ml)
	β (CI95%)	β (CI95%)	β (CI95%)	β (CI95%)
Sociodemographic variables				
Sex				
Male	1,260 (0.880 to 1.650)	0.91 (0.601 to 1.213)	-0.03 (-0.064 to -0.0003)	0.61 (0.110 to 1.103)
Age	-0.03 (-0.05 to -0.02)	-0.03 (-0.04 to -0.02)	-0.001 (-0.003 to -0.003)	-0.037 (-0.055 to -0.019)
Weight	0.04 (0.02 to 0.05)	0.02 (0.01 to 0.04)	-0.001 (-0.002 to -0.0001)	0.014 (-0.004 to 0.034)
Size	0.08 (0.07 to 0.10)	0.06 (0.050 to 0.074)	-0.001 (-0.002 to 0.001)	0.052 (0.029 to 0.076)
Asthma				
Yes	0.04 (-0.731 to 0.802)	-0.17 (-0.750 to 0.422)	-0.054 (-0.100 to -0.010)	-0.673 (-1.470 to 0.122)
Occupational exposure variables				
Job function at the facility				

Wait staff/bartenders/cashiers	*Ref.*	*Ref.*	*Ref.*	*Ref.*
Owners/managers	0.37	0.003	-0.06	-0.828
	(-0.370 to 1.110)	(-0.570 to 0.570)	(-0.113 to -0.021)	(-1.613 to -0.047)
Cooks	-0.7	-0.64	-0.02	-0.772
	(-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	*Ref.*	*Ref.*	*Ref.*	*Ref.*
Smoking/mixed	0.1	-0.05	-0.03	-0.413
	(-0.460 to 0.672)	(-0.486 to 0.381)	(-0.071 to 0.003)	(-1.003 to 0.177)

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 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 recognized 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).

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 (ml)		FEF25/75 (ml)	
	β (CI95%)	R^2	β (CI95%)	R^2	β (CI95%)	R^2	β (CI95%)	R^2
Urine cotinine								
Crude model	0.002 (-0.010 to 0.010)	0.002	0.002 (-0.010 to 0.010)	0.003	0.0002 (-0.001 to 0.001)	0.002	0.002 (-0.010 to 0.010)	0.002
Adjusted model	-0.0002 (-0.007 to 0.006)*	0.781	0.001 (-0.003 to 0.006)*	0.795	0.0004 (-0.0003 to 0.001)+	0.33	0.005 (-0.006 to 0.015)+	0.672
Number of years exposed to SHS at work								
Crude model	-0.025 (-0.051 to 0.002)	0.0462	-0.022 (-0.042 to -0.001)	0.061	-0.0008 (-0.002 to 0.0008)	0.013	-0.022 (-0,050 to 0,006)	0.032
Adjusted model	-0.013 (-0.030 to 0.0025)*	0.79	-0.01 (-0.022 to 0.002)*	0.802	0.0006 (-0.001 to 0.002)+	0.324	-0.006 (-0,010 to -0,0004)+	0.964

*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

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 (ml), FEV_1 (ml), FEV_1/FVC (ml) 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 to 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 analyze 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 apropriate when we are studying chronic effects. Other studies that have addressed this topic have produced varying results [15, 16, 17, 20, 21, 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, 13, 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 various 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 40.0 ng/ml, in a mixed venue was 13.5 ng/ml and where smoking was not allow 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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Competing interest

The authors have no conflict of interest to declare.

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Contributorship statement

Parro Javiera. Substantial contributions to the conception and design of the work on pulmonary function parameters; acquisition, analysis and interpretation of data, drafting the work, and final approval of the version to be published;

Aceituno Paulina. Substantial contributions to the conception and design of the work, revising it critically for important intellectual content, final approval of the version to be published.

Droppelman Andrea. Substantial contributions to the conception, design of the work and interpretation of exposure data, final approval of the version to be published.

Mesías Sthepanie. Substantial contributions to the acquisition and analysis of exposure data, final approval of the version to be published.

Muñoz Claudio. Substantial contributions to the acquisition, analysis and interpretation of data, final approval of the version to be published.

Marchetti Nella. Substantial contributions to the conception of the work and interpretation of data, final approval of the version to be published.

Iglesias Verónica. Substantial contributions to the conception and design of the work, analysis and interpretation of data for the work; drafting the work, final approval of the

version to be published, agreement to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

STROBE Statement—checklist of items that should be included in reports of observational studies

	Item No	Recommendation
Title and abstract	1	(*a*) Indicate the study's design with a commonly used term in the title or the abstract page 1 line 1-2
		(*b*) Provide in the abstract an informative and balanced summary of what was done and what was found page 2 line 1-22
Introduction		
Background/rationale	2	Explain the scientific background and rationale for the investigation being reported page 3-5
Objectives	3	State specific objectives, including any prespecified hypotheses page 5 line 4
Methods		
Study design	4	Present key elements of study design early in the paper page 5 line 8
Setting	5	Describe the setting, locations, and relevant dates, including periods of recruitment, exposure, follow-up, and data collection page 5 line 13-23
Participants	6	(*a*) *Cohort study*—Give the eligibility criteria, and the sources and methods of selection of participants. Describe methods of follow-up *Case-control study*—Give the eligibility criteria, and the sources and methods of case ascertainment and control selection. Give the rationale for the choice of cases and controls *Cross-sectional study*—Give the eligibility criteria, and the sources and methods of selection of participants page 5 line 13-23
		(*b*) *Cohort study*—For matched studies, give matching criteria and number of exposed and unexposed *Case-control study*—For matched studies, give matching criteria and the number of controls per case
Variables	7	Clearly define all outcomes, exposures, predictors, potential confounders, and effect modifiers. Give diagnostic criteria, if applicable page 6 line 4-23, page 7 line 1-8
Data sources/ measurement	8*	For each variable of interest, give sources of data and details of methods of assessment (measurement). Describe comparability of assessment methods if there is more than one group page 6 line 4-23, page 7 line 1-8
Bias	9	Describe any efforts to address potential sources of bias
Study size	10	Explain how the study size was arrived at NO
Quantitative variables	11	Explain how quantitative variables were handled in the analyses. If applicable, describe which groupings were chosen and why page 7 line 19-22, page 8 line 1-8.
Statistical methods	12	(*a*) Describe all statistical methods, including those used to control for confounding page 7 line 10-20
		(*b*) Describe any methods used to examine subgroups and interactions page 7 line 10-20
		(*c*) Explain how missing data were addressed not apply
		(*d*) *Cohort study*—If applicable, explain how loss to follow-up was addressed *Case-control study*—If applicable, explain how matching of cases and controls was addressed *Cross-sectional study*—If applicable, describe analytical methods taking account of

sampling strategy page 7 line 10-20

(*e*) Describe any sensitivity analyses NO

Continued on next page

Results		
Participants	13*	(a) Report numbers of individuals at each stage of study—eg numbers potentially eligible, examined for eligibility, confirmed eligible, included in the study, completing follow-up, and analysed page 8 line 1-8
		(b) Give reasons for non-participation at each stage page 8 line 1-8
		(c) Consider use of a flow diagram NO
Descriptive data	14*	(a) Give characteristics of study participants (eg demographic, clinical, social) and information on exposures and potential confounders page 8 table 1
		(b) Indicate number of participants with missing data for each variable of interest not apply
		(c) *Cohort study*—Summarise follow-up time (eg, average and total amount)
Outcome data	15*	*Cohort study*—Report numbers of outcome events or summary measures over time
		Case-control study—Report numbers in each exposure category, or summary measures of exposure
		Cross-sectional study—Report numbers of outcome events or summary measures NO
Main results	16	(*a*) Give unadjusted estimates and, if applicable, confounder-adjusted estimates and their precision (eg, 95% confidence interval). Make clear which confounders were adjusted for and why they were included page 13 Table 4
		(*b*) Report category boundaries when continuous variables were categorized page 10 Table 2
		(*c*) If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period NO
Other analyses	17	Report other analyses done—eg analyses of subgroups and interactions, and sensitivity analyses NO
Discussion		
Key results	18	Summarise key results with reference to study objectives page 14 line 2-17
Limitations	19	Discuss limitations of the study, taking into account sources of potential bias or imprecision. Discuss both direction and magnitude of any potential bias page 15 line 8-23 page 16 line 1-5
Interpretation	20	Give a cautious overall interpretation of results considering objectives, limitations, multiplicity of analyses, results from similar studies, and other relevant evidence page 16 line 15-18
Generalisability	21	Discuss the generalisability (external validity) of the study results NO
Other information		
Funding	22	Give the source of funding and the role of the funders for the present study and, if applicable, for the original study on which the present article is based page 17 line 2

*Give information separately for cases and controls in case-control studies and, if applicable, for exposed and unexposed groups in cohort and cross-sectional studies.

Note: An Explanation and Elaboration article discusses each checklist item and gives methodological background and published examples of transparent reporting. The STROBE checklist is best used in conjunction with this article (freely available on the Web sites of PLoS Medicine at http://www.plosmedicine.org/, Annals of Internal Medicine at http://www.annals.org/, and Epidemiology at http://www.epidem.com/). Information on the STROBE Initiative is available at www.strobe-statement.org.

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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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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 (FVC), 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 FEF25 / 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.

Keywords: Secondhand smoke exposure, chronic exposure, pulmonary function, urine cotinine, workers.

Strengths and limitations of this study

- The effects of occupational 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 studied chronic SHS exposure.
- Our sample included mainly young workers being reasonable to infer that the sample not accumulated sufficient 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 various 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 than100 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 nonsmokers. 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 5 municipalities with the largest numbers of facilities, according to data provided by the National Institute of Statistics (Spanish acronym INE, for *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 1s (FEV_1), and then calculated the FEV_1 to FVC ratio (FEV_1/FVC) and forced expiratory flow 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 8 spirometry trials were performed. The criteria for including a participant's spirometry data in the analysis was achieving at least 3 acceptable and 2 reproducible trials, as described in the norms published by Spanish Society of Pneumology and Thoracic Surgery (Spanish acronym SEPAR, for *Sociedad Española de Neumología y Cirugía Torácica*)[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 analyzed. 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 3 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 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 dichotomized using the median as cutoff. 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 analyzed 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], as well as variables commonly controlled for in the literature.

Results

The non-smoking workers evaluated in the study were 92. 17(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}: 19–68 years), and 61% of participants were male. 29.3% were former smokers and the median of time they quit smoking was 8.5 years (RIC 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 to workers in mixed and non-smoking facilities (p-value=0.0001) (Table 1).

Table 1. Characteristics of the study sample. Santiago, Chile 2010-2011.

		Smoking status restaurant/bar/pub			
	Total	**Smoking**	**Mixed**	**Non-smoking**	**p value**
Nº 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; ** Chi2.

As shown in Table 2 we compared the results for pulmonary function and exposure to SHS based on covariables. Males had greater pulmonary function values than females, 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 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.

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

Variables		Pulmonary function parameters				Urine cotinine concentration (ng/ml)	Number of years exposed to SHS workplace
	n	FVC ml (RIC)	FEV_1 ml (RIC)	FEV_1/FVC % (RIC)	$FEF_{25\%/75\%}$ ml (RIC)	Med (P_{25}-P_{75})	Med (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 hrs	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 hrs	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 dichotomized in median value; + Kruskal Wallis test; ±Wilcoxon Test

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.

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 (ml)	$FEF_{25/75}$ (ml)
	β (CI95%)	β (CI95%)	β (CI95%)	β (CI95%)
Sociodemographic variables				
Sex				
Male	1.26 (0.880 to 1.650)	0.91 (0.601 to 1.213)	-0.03 (-0.064 to -0.0003)	0.61 (0.110 to 1.103)
Age	-0.03 (-0.05 to -0.02)	-0.03 (-0.04 to -0.02)	-0.001 (-0.003 to -0.003)	-0.04 (-0.055 to -0.019)
Weight	0.04 (0.02 to 0.05)	0.02 (0.01 to 0.04)	-0.001 (-0.002 to -0.0001)	0.01 (-0.004 to 0.034)
Size	0.08 (0.07 to 0.10)	0.06 (0.050 to 0.074)	-0.001 (-0.002 to 0.001)	0.05 (0.029 to 0.076)
Asthma				
Yes	0.04 (-0.731 to 0.802)	-0.17 (-0.750 to 0.422)	-0.05 (-0.100 to -0.010)	-0.67 (-1.470 to 0.122)
Occupational exposure variables				
Job function at the facility				

Wait staff/bartenders/cashiers	*Ref.*	*Ref.*	*Ref.*	*Ref.*
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	*Ref.*	*Ref.*	*Ref.*	*Ref.*
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)

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 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 recognized 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).

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 (ml)		FEF25/75 (ml)	
	β (CI95%)	R^2	β (CI95%)	R^2	β (CI95%)	R^2	β (CI95%)	R^2
Urine cotinine								
Crude model	0.002 (-0.010 to 0.010)	0.002	0.002 (-0.010 to 0.010)	0.003	0.0002 (-0.001 to 0.001)	0.002	0.002 (-0.010 to 0.010)	0.002
Adjusted model	-0.0002 (-0.007 to 0.006)*	0.781	0.001 (-0.003 to 0.006)*	0.795	0.0004 (-0.0003 to 0.001)+	0.33	0.005 (-0.006 to 0.015)+	0.672
Number of years exposed to SHS at work								
Crude model	-0.025 (-0.051 to 0.002)	0.046	-0.022 (-0.042 to -0.001)	0.061	-0.0008 (-0.002 to 0.0008)	0.013	-0.022 (-0,050 to 0,006)	0.032
Adjusted model	-0.013 (-0.030 to 0.0025)*	0.79	-0.01 (-0.022 to 0.002)*	0.802	0.0006 (-0.001 to 0.002)+	0.324	-0.006 (-0,010 to -0,0004)+	0.964

*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

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 (ml), FEV_1 (ml), FEV_1/FVC (ml) 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 to 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 analyze 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 various 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 allow 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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Data Sharing

No additional data available.

Competing interest

The authors have no conflict of interest to declare.

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Contributorship statement

Parro Javiera. Substantial contributions to the conception and design of the work on pulmonary function parameters; acquisition, analysis and interpretation of data, drafting the work, and final approval of the version to be published;

Aceituno Paulina. Substantial contributions to the conception and design of the work, revising it critically for important intellectual content, final approval of the version to be published.

Droppelman Andrea. Substantial contributions to the conception, design of the work and interpretation of exposure data, final approval of the version to be published.

Mesías Sthepanie. Substantial contributions to the acquisition and analysis of exposure data, final approval of the version to be published.

Muñoz Claudio. Substantial contributions to the acquisition, analysis and interpretation of data, final approval of the version to be published.

Marchetti Nella. Substantial contributions to the conception of the work and interpretation of data, final approval of the version to be published.

Iglesias Verónica. Substantial contributions to the conception and design of the work, analysis and interpretation of data for the work; drafting the work, final approval of the version to be published, agreement to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

STROBE Statement—checklist of items that should be included in reports of observational studies

	Item No	Recommendation
Title and abstract	1	(*a*) Indicate the study's design with a commonly used term in the title or the abstract page 1 line 1-2
		(*b*) Provide in the abstract an informative and balanced summary of what was done and what was found page 2 line 1-22
Introduction		
Background/rationale	2	Explain the scientific background and rationale for the investigation being reported page 3-5
Objectives	3	State specific objectives, including any prespecified hypotheses page 5 line 4
Methods		
Study design	4	Present key elements of study design early in the paper page 5 line 8
Setting	5	Describe the setting, locations, and relevant dates, including periods of recruitment, exposure, follow-up, and data collection page 5 line 13-23
Participants	6	(*a*) *Cohort study*—Give the eligibility criteria, and the sources and methods of selection of participants. Describe methods of follow-up *Case-control study*—Give the eligibility criteria, and the sources and methods of case ascertainment and control selection. Give the rationale for the choice of cases and controls *Cross-sectional study*—Give the eligibility criteria, and the sources and methods of selection of participants page 5 line 13-23
		(*b*) *Cohort study*—For matched studies, give matching criteria and number of exposed and unexposed *Case-control study*—For matched studies, give matching criteria and the number of controls per case
Variables	7	Clearly define all outcomes, exposures, predictors, potential confounders, and effect modifiers. Give diagnostic criteria, if applicable page 6 line 4-23, page 7 line 1-8
Data sources/ measurement	8*	For each variable of interest, give sources of data and details of methods of assessment (measurement). Describe comparability of assessment methods if there is more than one group page 6 line 4-23, page 7 line 1-8
Bias	9	Describe any efforts to address potential sources of bias
Study size	10	Explain how the study size was arrived at NO
Quantitative variables	11	Explain how quantitative variables were handled in the analyses. If applicable, describe which groupings were chosen and why page 7 line 19-22, page 8 line 1-8.
Statistical methods	12	(*a*) Describe all statistical methods, including those used to control for confounding page 7 line 10-20
		(*b*) Describe any methods used to examine subgroups and interactions page 7 line 10-20
		(*c*) Explain how missing data were addressed not apply
		(*d*) *Cohort study*—If applicable, explain how loss to follow-up was addressed *Case-control study*—If applicable, explain how matching of cases and controls was addressed *Cross-sectional study*—If applicable, describe analytical methods taking account of

sampling strategy page 7 line 10-20

(*e*) Describe any sensitivity analyses NO

Continued on next page

Results		
Participants	13*	(a) Report numbers of individuals at each stage of study—eg numbers potentially eligible, examined for eligibility, confirmed eligible, included in the study, completing follow-up, and analysed page 8 line 1-8
		(b) Give reasons for non-participation at each stage page 8 line 1-8
		(c) Consider use of a flow diagram NO
Descriptive data	14*	(a) Give characteristics of study participants (eg demographic, clinical, social) and information on exposures and potential confounders page 8 table 1
		(b) Indicate number of participants with missing data for each variable of interest not apply
		(c) *Cohort study*—Summarise follow-up time (eg, average and total amount)
Outcome data	15*	*Cohort study*—Report numbers of outcome events or summary measures over time
		Case-control study—Report numbers in each exposure category, or summary measures of exposure
		Cross-sectional study—Report numbers of outcome events or summary measures NO
Main results	16	(*a*) Give unadjusted estimates and, if applicable, confounder-adjusted estimates and their precision (eg, 95% confidence interval). Make clear which confounders were adjusted for and why they were included page 13 Table 4
		(*b*) Report category boundaries when continuous variables were categorized page 10 Table 2
		(*c*) If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period NO
Other analyses	17	Report other analyses done—eg analyses of subgroups and interactions, and sensitivity analyses NO
Discussion		
Key results	18	Summarise key results with reference to study objectives page 14 line 2-17
Limitations	19	Discuss limitations of the study, taking into account sources of potential bias or imprecision. Discuss both direction and magnitude of any potential bias page 15 line 8-23 page 16 line 1-5
Interpretation	20	Give a cautious overall interpretation of results considering objectives, limitations, multiplicity of analyses, results from similar studies, and other relevant evidence page 16 line 15-18
Generalisability	21	Discuss the generalisability (external validity) of the study results NO
Other information		
Funding	22	Give the source of funding and the role of the funders for the present study and, if applicable, for the original study on which the present article is based page 17 line 2

*Give information separately for cases and controls in case-control studies and, if applicable, for exposed and unexposed groups in cohort and cross-sectional studies.

Note: An Explanation and Elaboration article discusses each checklist item and gives methodological background and published examples of transparent reporting. The STROBE checklist is best used in conjunction with this article (freely available on the Web sites of PLoS Medicine at http://www.plosmedicine.org/, Annals of Internal Medicine at http://www.annals.org/, and Epidemiology at http://www.epidem.com/). Information on the STROBE Initiative is available at www.strobe-statement.org.