ORIGINAL ARTICLE

Cross shift changes in lung function among bar and restaurant workers before and after implementation of a smoking ban

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Objective: To study possible cross shift effects of environmental tobacco smoke (ETS) on pulmonary function among bar and restaurant employees before and after the implementation of a smoking ban in Norway.
Methods: The study included 93 subjects employed in 13 different establishments in Oslo. They were examined at the beginning and end of a workshift both while ETS exposure was present and when smoking was banned. The mean exposure level of nicotine and total dust before the ban was 28 μg/m^3 (range 3–65) and 275 μg/m^3 (range 81–506), respectively. Following the smoking ban, the mean level of nicotine and total dust was 0.6 μg/m^3 and 77 μg/m^3, respectively. Assessment of lung function included dynamic lung volumes and flows.
Results: The cross shift reduction in forced vital capacity (FVC) among 69 subjects participating in both examinations changed from 81 ml (SD 136) during exposure to ETS to 52 ml (SD 156) ($p=0.24$) following the smoking ban. The reduction in forced expired volume in one second (FEV1) during a workshift, was borderline significantly reduced when comparing the situation before and after the intervention, by 89 ml (SD = 132) compared to 46 ml (SD = 152) ($p=0.09$), respectively. The reduction in forced mid-expiratory flow rate ($FEF_{25-75\%}$) changed significantly from 199 ml/s (SD = 372) to 64 ml/s (SD = 307) ($p=0.01$). Among 26 non-smokers and 11 asthmatics, the reduction in FEV1 and $FEF_{25-75\%}$ was significantly larger during ETS exposure compared to after the smoking ban. There was an association between the dust concentration and decrease in $FEF_{25-75\%}$ before the ban among non-smokers ($p=0.048$).
Conclusions: This first study of cross shift changes before and after the implementation of a smoking ban in restaurants and bars shows a larger cross shift decrease in lung function before compared with after the implementation of the ban.

In past years there has been increasing concern regarding the health consequences of exposure to environmental tobacco smoke (ETS) for employees working in restaurants and bars. These workers have reached ETS levels several times greater than that of other occupational groups.[1] Whereas employees in most workplaces since 1988 have performed their tasks without passively being exposed to tobacco smoke in Norway, those employed in bars and restaurants have not been able to do so until recently.

Sidestream smoke contains the same irritative and toxic compounds as mainstream smoke, and it is plausible that the biological effects of ETS are similar to those of active smoking.[2] Irritation of the eyes, airways, and throat has been described in places with high levels of tobacco smoke.[3] Passive smokers in workplaces have reported more symptoms from the airways and more days lost from work due to chest colds than control subjects, and exposure to tobacco smoke may be even higher among such employees than people living with smokers.[1]

Acute smoke exposure activates cells such as neutrophils and macrophages in the airways, has a suppressive effect on the numbers of eosinophils, and may result in tissue damage.[4] Repeated smoke exposure may promote a chronic inflammatory process resulting in thicker, inflamed, deformed, and narrow airways with emphysematous changes around them.[5]

In Norway a revision of the Environmental Tobacco Smoke Act was put forward on 1 June 2004, prohibiting smoking in restaurants and bars. Few countries have so far implemented similar regulations and little is known about the potential benefits to health of such interventions in the workplaces. Only one study from the US has been published describing the lung function of bartenders before and after smoking cessation in the workplace. Both forced vital capacity (FVC) and forced expired volume in one second (FEV1) increased during follow up, according to the authors, due to the establishment of a smoke-free workplace.[6]

The aim of the present investigation was to compare cross shift changes in pulmonary function among employees in restaurants and bars before and after enforcement of smoke-free bars and restaurants and to examine if there were any associations between nicotine/dust exposure levels and changes in lung function before the implementation of the ban.

MATERIALS AND METHODS

A detailed description of the material and methods is presented elsewhere.[7] Initially, 15 bars and restaurants were

Abbreviations: COPD, chronic obstructive pulmonary disease; ETS, environmental tobacco smoke; $FEF_{25-75\%}$, forced mid-expiratory flow rate; FEV1, forced expired volume in one second; FVC, forced vital capacity

Table 1 Characteristics of the women studied at baseline, before shift (n = 47)

	Tested before ban			Post-ban dropouts
	Total group (n = 47)	Smokers (n = 30)	Non-smokers (n = 17)	Total group (n = 8)*
	Mean (SD) (range)	Mean (SD)	Mean (SD)	Mean (SD)
Age (years)	30.1 (8.4) (19–55)	30.6 (8.2)	29.2 (9.0)	29.6 (8.5)
Height (cm)	166 (5.5) (155–181)	166.4 (5.3)	165.2 (5.8)	169 (8.1)
Weight (kg)	63.1 (8.5) (50–85)	62.5 (7.3)	64.3 (10.6)	66.9 (8.3)
FVC (l)	3.93 (0.57) (2.21–4.73)	3.88 (0.55)	4.03 (0.60)	3.91 (0.77)
FEV1 (l)	3.24 (0.53) (2.02–4.14)	3.14 (0.50)	3.41 (0.55)	3.25(0.57)
$FEF_{25-75\%}$(l/s)	3.40 (0.93) (1.42–5.41)	3.27 (1.0)	3.63 (0.76)	3.55 (0.56)
PEF (l/min)	470 (98) (234–629)	451 (99)	504 (88)	442 (79)

*Seven smokers, one ex-smoker.

selected in cooperation with the owners' and workers' organisations to be a diverse sample of establishments in the capital of Oslo. Two establishments did not want to participate and 13 agreed and were included in the study. At these 13 different restaurants and bars 112 subjects at were asked to participate, of which 93 gave their consent (participation rate 83%) (tables 1 and 2). At follow up eight individuals had quit work, eight were on leave/vacation, three subjects had changed their smoking habits, five left work before second examination due to lack of work tasks/sudden change of schedule, leaving 69 individuals (35 women) for the study. The characteristic of these individuals are shown in table 3. At baseline the mean weight of this group was 70.7 kg (SD 12.9) changing to 71.3 kg (SD 13.3) at follow up.

As for the registration of asthma, the subjects were asked: "Do you use any medication against asthma or allergy?", "Have you ever had asthma?" and, if so, "was the diagnosis made by a doctor?" In this study the participants were defined as asthmatics if they reported asthma in the history.

The results of exposure assessment including urinary cotinine, total dust, and nicotine in air have been presented elsewhere.[7] Each individual's exposure dose was expressed as the mean values of total dust and nicotine obtained at her/his establishment.

Lung function measurements were performed both before and at the end of the workshift before the smoking ban was introduced on 1 June 2004. These measurements were repeated 3–8 months after the smoking ban was implemented during the months September 2004 until February 2005. In May 2004 the mean outdoor temperature in Oslo was 12℃ (range 7.4 to 19.8) whereas it was 3℃ in the period September 2004 through February 2005 (range −7.8 to 17.9). However, most retests were performed in September/October giving a mean second temperature of 6℃. Most workers worked evening shifts meaning they started working in the evening and left work at midnight or afterwards. The same shift plan was present at follow up, thus all workers worked at the same time at follow up as compared to at baseline and thus the tests were performed during the same shift at the same day of the week for each individual on both occasions. The mean time of day for starting measurements during shift before ban was 16 minutes past six o'clock in the afternoon (18:16) (range 10:00–21:45). After the ban the mean starting time was six minutes to six o'clock (17:54) in the afternoon (range 10:30–23:40). No statistical difference between these starting points was revealed ($p = 0.23$). During the first cross shift examination the mean time between the measurements was 363 minutes (SD 77 minutes). After the implementation of the ban the mean time between the measurements was 360 minutes (SD 91). Testing was performed using the Vitalograph 2170 spirometer (Spirotrac, UK). The tests were performed by the authors (MS, KK) who were visiting five and eight establishments, respectively. They examined the same subjects both before and after shift on both occasions. The subjects were given standardised instructions on the forced maximal expiratory manoeuvres, with demonstration of the procedures. The tests were performed with the subjects sitting in a chair and breathing through a mouthpiece with a nose clip. The spirometer was calibrated daily. The best results, according to ATS criteria, of at least three flow volume manoeuvres were used in the analysis.[8] Peak expiratory flow rate (PEF), forced vital capacity (FVC), forced expired volume in one second (FEV1), and forced mid-expiratory flow rate ($FEF_{25-75\%}$) were measured.

Arithmetic means and standard deviations for lung function parameters were calculated. Student's paired *t* tests were used to compare continuous outcomes before and after the workshift when the individuals were compared with themselves. Simple linear regression analysis was used to assess possible effects of dust and nicotine on change in FVC, FEV1, and $FEF_{25-75\%}$. Correlations were calculated using

Table 2 Characteristics of the men studied at baseline, before shift (n = 46)

	Tested before ban			Post-ban dropouts		
	Total group (n = 46)	Smokers (n = 30)	Non-smokers (n = 16)	Total group (n = 11)	Smokers (n = 7)	Non-smoker (n = 4)
	Mean (SD) (range)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)
Age (years)	31.3 (7.4) (19–54)	32 (7.6)	30 (7.3)	29.5 (8.7)	32.3 (9.9)	24.5 (2.4)
Height (cm)	179 (6.5) (165–194)	178.7 (6.5)	179.8 (6.7)	179 (6.1)	180.1 (6.6)	177.3 (5.6)
Weight (kg)	80.1 (9.7) (62–104)	79.3 (10.3)	81.6 (8.7)	81.5 (12.1)	84.4 (11.3)	76.5 (13.4)
FVC (l)	5.34 (0.77) (3.63–6.72)	5.24 (0.75)	5.53 (0.79)	5.37 (0.72)	5.14 (0.74)	5.76 (0.55)
FEV1 (l)	4.33 (0.67) (2.42–5.48)	4.29 (0.72)	4.41 (0.59)	4.59 (0.68)	4.40 (0.76)	4.94 (0.37)
$FEF_{25-75\%}$(l/s)	4.45 (1.57) (1.32–8.68)	4.54 (1.68)	4.27 (1.38)	5.31 (1.59)	5.23 (1.90)	5.44 (1.07)
PEF (l/min)	647 (133) (380–950)	614 (138)	709 (98)	631 (156)	575 (165)	729 (80)

Table 3 Characteristics of the woman and men who participated both before and after ban, values at baseline, before shift (n = 69)

	Tested before and after ban, women			Tested before and after ban, men		
	Total group (n = 35)	Smokers (n = 21)	Non-smokers (n = 14)	Total group (n = 34)	Smokers (n = 22)	Non-smokers (n = 12)
	Mean (SD) (range)	Mean (SD)	Mean (SD)	Mean (SD) (range)	Mean (SD)	Mean (SD)
Age (years)	30.7(8.8) (19–55)	31.4 (8.3)	29.6 (9.7)	31.9 (7.1) (21–54)	31.9 (7.1)	31.8 (7.5)
Height (cm)	165.1 (4.7) (155–175)	166.1 (4.5)	163.6 (4.8)	179.3 (6.8) (165–194)	178.5 (6.6)	180.7 (7.1)
Weight (kg)	61.9 (8.8) (50–85)	61.6 (7.8)	62.5 (10.7)	79.9 (9.0) (62–95)	78.0 (9.8)	83.2 (6.5)
FVC (l)	3.91 (0.55) (2.78–4.73)	3.85 (0.47)	4.00 (0.65)	5.36 (0.79) (3.63–6.72)	5.31 (0.76)	5.45 (0.86)
FEV1 (l)	3.20 (0.54) (2.13–4.14)	3.08 (0.49)	3.38 (0.58)	4.27 (0.66) (2.42–5.28)	4.29 (0.72)	4.24 (0.55)
$FEF_{25-75\%}$(l/s)	3.29 (0.99) (1.42–5.41)	3.10 (1.08)	3.58 (0.79)	4.20 (1.50) (1.32–8.68)	4.38 (162)	3.88 (1.27)
PEF (l/min)	475 (106) (234–629)	458 (111)	500 (95)	656 (126) (380–950)	631 (132)	702 (106)

Spearman's rank test. All tests were two sided and a significance level of 5% was chosen.[9] SPSS for Windows (SPSS version 13) was used in the data analysis.

RESULTS

The levels of airborne nicotine and dust are presented elsewhere.[7] The mean concentration of nicotine and dust before the smoking ban varied between the different restaurants and bars (table 4). All baseline results obtained by spirometry are shown in tables 1–3.

Three individuals changed their smoking habits during follow up, two subjects had incomplete data, and 19 were lost to follow up and were thus not included in the second examination leaving 69 for the last examination. The cross shift changes of lung function, both in absolute and relative numbers, before and after the implementation of the smoking ban are shown for the remaining 69 individuals in table 5. There was a statistically significant decrease in $FEF_{25-75\%}$, when the pre-ban measurements were compared to those of the post-ban, from 199 ml/s (SD 372) to 64 ml/s (SD 307) ($p = 0.01$) on average, respectively.

Cross shift lung function changes for the 26 non-smokers, the 11 asthmatics (non-smokers and smokers), and the 43 smokers are shown in table 6. For the 26 non-smokers, the mean pre-ban cross shift fall in FEV1 was 120 ml compared to 37 ml ($p = 0.03$) after the intervention, and the corresponding decrease in $FEF_{25-75\%}$ was 218 ml/s compared to 65 ml/s ($p = 0.01$). Eleven asthmatics had a statistically significant larger decrease in FVC, FEV1, and $FEF_{25-75\%}$ across the workshift before than after the ban. As for the smokers the only change was a significantly larger increase in PEF and an almost significantly larger decrease in $FEF_{25-75\%}$ across the workshift when the pre-ban results were compared with those after the ban.

A near significant association was observed between the change in lung function during follow up and weight gain, for FVC ($\beta = -0.02$, $p = 0.09$).

A statistically significant association between the total dust concentration during the first cross shift examination and decrease in $FEF_{25-75\%}$ among the 33 non-smoking subjects who were present both pre and post-ban was observed ($p = 0.05$) (fig 1). The association between FEV1 and total dust did not reach statistical significance ($p = 0.15$), (table 7).

Table 4 Nicotine and total dust levels in 13 different restaurants and bars before and after implementation of smoking ban

	Pre-ban mean (SD) (range)	Post-ban mean (SD) (range)
Nicotine (μg/m^3)	28 (15) (3–65)	0.6 (0.4) (ND–1)
Total dust (μg/m^3)	275 (130) (81–506)	77 (39) (17–170)

ND, not detected.

DISCUSSION

Among non-smokers and people with asthma in the history a larger decrease in FEV1 and $FEF_{25-75\%}$ was demonstrated during a pre-ban workshift compared to the same work situation after a smoking ban was implemented in the bars and restaurants studied. Among smoking subjects a significant increase in PEF and a near significant decrease in $FEF_{25-75\%}$ was found when comparing the pre-ban situation with that of the post-ban. Before the ban there was an association between fall in $FEF_{25-75\%}$ and levels of total dust.

Our main result indicates an effect of ETS on small airways that may be related to total dust levels during ETS exposure in restaurants and bars. The effect was not observed after the intervention. The results agree with the results found after experimental exposure in chambers where ETS exposure gives a reduction of FEV1 among asthmatics[10] and a reversible reduction in pulmonary function among non-smokers.[11] Studies of workers and in the household have indicated a decrease in small airways after ETS exposure.[12 13] The small particulate components of ETS can be drawn deeply into the lungs, thus causing pathological changes in the peripheral airways. Further exposure to cigarette smoke

Table 5 Change in lung function during a workshift before and after implementation of smoking ban (n = 69)

Parameter	Before smoking ban: Pre-shift, mean (SD)	Post-shift, mean (SD)	Change, mean (SD)	Change (%)	After smoking ban, mean (SD): Pre-shift, mean (SD)	Post-shift, mean (SD)	Change, mean (SD)	Change (%)	p Value comparing changes
FVC	4.63 l (0.99)	4.55 l (1)	−81 ml (136)	−1.8	4.49 l (0.93)	4.44 l (0.95)	−52 ml (156)	−1.2	0.24
FEV1	3.73 l (0.80)	3.64 l (0.8)	−89 ml (132)	−2.4	3.52 l (0.71)	3.48 l (0.73)	−46 ml (152)	−1.4	0.09
$FEF_{25-75\%}$	3.74 l/s (1.35)	3.54 l/s (1.33)	−199 ml/s (372)	−5	3.33 l/s (1)	3.26 l/s (1)	−64 ml/s (307)	−1.7	0.01
PEF	564 l/min (147)	577 l/m (154)	14 l/min (87)	3.9	576 l/min (152)	584 l/min (150)	9 l/min (63)	2.5	0.11

*Cross shift changes significantly changed from before application of smoking ban $p<0.05$ (Student's paired *t* test).

Table 6 Change in lung function during a workshift before and after implementation of smoking ban among non-smokers (n = 26), individuals with asthma in the history (n = 11), and smokers (n = 43)

	Before ban, mean (SD)	Change (%)	After ban, mean (SD)	Change (%)	p Value
Non-smokers					
FVC (ml)	−97 (168)	−2.2	−56 (115)	−1.4	0.24
FEV1 (ml)	−120 (155)	−3.1	−37 (127)	−1.1	0.03
$FEF_{25-75\%}$(ml/s)	−218 (286)	−5.4	−65 (274)	−1.3	0.01
PEF (l/min)	22 (67)	0.4	2 (74)	1.9	0.22
Asthmatics					
FVC (ml)	−89 (121)	−2.3	6 (63)	0.02	0.04
FEV1 (ml)	−122 (130)	−3.6	12 (90)	0.2	0.02
$FEF_{25-75\%}$(ml/s)	−316 (469)	−9.2	21 (274)	0.8	0.01
PEF (l/min)	28 (65)	4.6	2 (65)	1.4	0.49
Smokers					
FVC (ml)	−71 (113)	−1.6	−49 (177)	−0.9	0.51
FEV1 (ml)	−71 (114)	−2.1	−52 (167)	−1.4	0.57
$FEF_{25-75\%}$(ml/s)	−187 (420)	−5.0	−64 (329)	−2.1	0.12
PEF (l/min)	26 (89)	5.8	13 (56)	2.6	0.02

*Cross shift changes significantly changed from before application of ban $p<0.05$ (Student's paired *t* test).

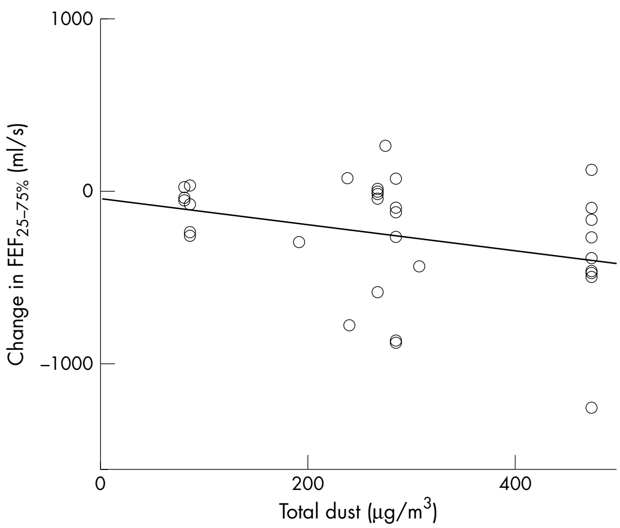

Figure 1 Cross shift lung function decline ($FEF_{25-75\%}$) associated with the concentration of total dust among 33 non-smokers before application of the smoking ban. The regression line of this group is shown.

can cause progression from involvement of small airways to disease of the bronchial and alveolar part of the lung.[3 14]

We found a smaller cross shift increase in PEF after ban compared to before ban. The changes in PEF are mostly due to effort and may explain, to some extent, the opposite effect on small airways. Effort depending increased PEF may lead to a compression of air in the upper airways. We believe that the effect is small but it could nevertheless partly explain the larger fall in dynamic lung volumes before implementation of the ban compared with after the ban.

Table 7 Correlation between dust exposure and change in lung function among 33 non-smokers before implementation of the ban

	Correlation coefficient	Significance
Total dust pre-ban		
Change in FVC	0.149	0.41
Change in FEV1	0.255	0.15
Change in FEF $_{25-75\%}$	0.347	0.048
Nicotine pre-ban		
Change in FVC	−0.126	0.485
Change in FEV1	0.081	0.655
Change in FEF $_{25-75\%}$	0.232	0.193

In the present study, the subjects with asthma in the history had their airways affected during their workshift before smoking was banned in the workplace. ETS in the workplace has been associated with asthma both among women and men.[15] However, due to methodological problems and a limited number of studies, no definite conclusion has been made concerning the role of ETS in adult asthma.[16] Recent studies, however, suggest a causal relation between ETS exposure in the workplace and both new onset asthma and asthma exacerbations among adults,[17 18] a possible increased risk of allergic sensitisation and allergic rhinitis,[19] and reduction in lung function particularly among women with asthma.[20]

A plausible mechanism of ETS mediated asthma might be attributed to the inflammatory response of ETS which may facilitate the permeability of allergens into the bronchial epithelium[21] or that asthma occurs via irritative mechanisms.

Among the non-smokers in this study a response in the airways was noted during ETS at work. This is in accordance with previous studies showing increased bronchial responsiveness,[22] and even a reduction in pulmonary function[23] after ETS exposure.

Even among the smokers there was a cross shift reduction in lung function before the implementation of the smoking ban. In support of this finding it has been shown that inhalation of cigarette smoke among smokers, who had not smoked 24 hours before examination, results in an immediate fall in FEV1 with the highest response among smokers with asthma.[24]

Several mechanisms could explain the effects observed in our study. Cigarette smoke has been shown to induce bronchoconstriction in animals through an early phase cholinergic reflex and a sensory mediated stress induced norepinephrine release could also have contributed.[11]

It is known that ETS is a potent airways irritant that can induce an inflammatory response in the airways among sensitive individuals[25] by stimulating bronchial epithelial cells to release mediators and thus recruit neutrophilis into the airways.[26] Supporting this mechanism, it has been shown that smoking cessation among young people can produce an increase in FEV1, possibly reflecting reduced inflammation and reduced bronchoconstriction.[27]

Evidence provides a plausible link between passive smoking, bronchial hyperresponsiveness, and chronic obstructive pulmonary disease (COPD). However, limited information is available on these relationships[2 28] and due to the fact that levels of ETS doses have been based on questionnaire reports,[29] effects on lung function may not have been observed at low exposures.[2] As for a possible association between COPD among employees in restaurants and bars and ETS, the levels of ETS exposure in the current study have been higher compared to other studies.[1] The mean levels of exposure in some bars in our study were very high, exceeding the air nicotine levels previously reported in 17 restaurants and in 25 different bars of 6.5 μg/m^3 and 19.7 μg/m^3, respectively,[1] and equalling that of 28–50 μg/m^3 reported among nightclub musicians.[30] As longitudinal decrease in lung function seems to coincide with cross shift fall in lung function among people exposed to dust,[31 32] an increased risk for COPD among non-smoking employees in restaurants and bars with the high levels of exposure found in our study cannot be ruled out.

In the present study the pre-shift lung function was lower after, rather than before, the ban was introduced. This finding is not supported by the results reported in a study of bartenders 10 years older than the group in the present study.[6] Cessation of workplace high exposure ETS seemed to improve lung function in the study of Eisner *et al*[6] but may not, according to Bell and Urbach,[33] represent any clinically important change. The reduction in lung function in our study during the 3–8 month follow up could to some extent be explained with weight gain. Recent studies report that even mild obesity may influence dynamic lung volumes.[35] In addition, our results could be explained by seasonal variations[36] where low outside temperature may give a decrease in lung function.[36] The second examination was performed when the temperature was on average 6℃ lower in Oslo compared to May 2004, reaching a mean temperature below zero in February 2005, and when inhalation of cold air could cause bronchoconstriction in subjects with reactive airways. However, to eliminate the effects of seasonal variation, a repeated cross shift design was chosen. Such a repeated crossover study has some attractive features, namely that the comparison of change in lung function is ''within-subjects'' rather than ''between-subjects'' thus reducing the potential for confounding substantially.

The selection of restaurants and bars was chosen in order to obtain a representative sample of the Norwegian workforce in bars and restaurants. Ninety three out of 112 subjects were initially invited to participate in the study at baseline. In our study 23% of the subjects did not participate in the second examination. In addition to many who had quit during follow up, restaurant and bar subjects work irregular hours and schedules are often changed at short notice. Thus, there were difficulties in gathering exactly the same group of employees for follow up. None of those approached a second time, however, refused further participation. The proportion of smokers and the ages were similar among those who were lost to follow up compared to those who participated. Thus we have no indication of outcome selective dropout from the study.

This first study of cross shift lung function changes before and after implementation of a smoking ban in restaurants and bars shows a larger reduction in FEV1 and $FEF_{25-75\%}$ during ETS exposure compared to after the smoking ban. These effects were most pronounced among non-smokers and asthmatics. Among the non-smokers the reduction in lung function during ETS exposure was associated with the total dust levels.

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REFERENCES

1 **Siegel M**. Involuntary smoking in the restaurant workplace. A review of employee exposure and health effects. *JAMA* 1993;**270**:490–3.
2 **Jaakkola MS**, Jaakkola JJK. Effects of environmental tobacco smoke on the respiratory health of adults. *Scand J Work Environ Health* 2002;**28**:52–70.
3 **Lefcoe NM**, Ashley MJ, Pederson LL, *et al.* The health risk of passive smoking. The growing case for control measures in enclosed environments. *Chest* 1983;**84**:90–5.
4 **Van der Vaart H**, Postma DS, Timens W, *et al.* Acute effects of cigarette smoke on inflammation and oxidative stress: a review. *Thorax* 2004;**59**:713–21.
5 **Saetta M**, Finkelstein R, Cosio MG. Morphological and cellular basis for airflow limitation in smokers. *Eur Respir J* 1994;**7**:1505–15.
6 **Eisner MD**, Smith AK, Blanc PD. Bartenders' respiratory health after establishment of smoke-free bars and taverns. *JAMA* 1998;**280**:1909–14.
7 **Ellingsen DG**, Fladseth G, Daae HL, *et al.* Airborne exposure and biological monitoring of bar and restaurant workers before and after the introduction of a smoking ban. *J Environ Monit* 2006;**8**:362–8.
8 **American Thoracic Society**. Standardization of spirometry-1987 update. *Am Rev Respir Dis* 1987;**136**:1285–98.
9 **Altman DG**. *Practical statistics for medical research.* London, UK: Chapman & Hall, 1995.
10 **Eisner MD**. Environmental tobacco smoke exposure and pulmonary function among adults in NHANES III: Impact on the general population and adults with current asthma. *Environ Health Perspect* 2002;**110**:765–70.
11 **Smith CJ**, Bombick DW, Ryan BA, *et al.* Pulmonary function in nonsmokers following exposure to sidestream cigarette smoke. *Toxicol Pathol* 2001;**29**:260–4.
12 **White JR**, Froeb HF. Small-airways dysfunction in nonsmokers chronically exposed to tobacco smoke. *N Engl J Med* 1980;**302**:720–3.
13 **Masi MA**, Hanley JA, Ernst P, *et al.* Environmental exposure to tobacco smoke and lung function in young adults. *Am Rev Respir Dis* 1988;**138**:296–9.
14 **Niewoehner DE**, Kleinerman J, Rice DB. Pathologic changes in the peripheral airways of young cigarette smokers. *N Engl J Med* 1974;**292**:755–8.
15 **Greer JR**, Abbey DE, Burchette RJ. Asthma related to occupational and ambient air pollutants in nonsmokers. *JOM* 1993;**35**:909–15.
16 **Weiss ST**, Utell MJ, Samet JM. Environmental tobacco smoke exposure and asthma in adults. *Environ Health Perspect* 1999;**107**:891–5.
17 **Jaakkola MS**, Piipari R, Jaakkola N, *et al.* Environmental tobacco smoke and adult-onset asthma: a population-based incident case-control study. *Am J Public Health* 2003;**93**:2055–60.
18 **Eisner MD**. Environmental tobacco smoke and adult asthma. *Clin Chest Med* 2002;**23**:749–61.
19 **Topp R**, Thefeld W, Wichmann HE, *et al.* The effects of environmental tobacco smoke exposure on allergic senitization and allergic rhinitis in adults. *Indoor Air* 2005;**15**:222–7.
20 **Künzli N**, Schwartz J, Stutz EZ, *et al.* Association of environmental tobacco smoke at work and forced expiratory lung function among never smoking asthmatics and non-asthmatics. *Soz Präventivmed* 2000;**45**:208–17.
21 **Zetterström O**, Osterman K, Machado L, *et al.* Another smoking hazard: raised serum IgE concentration and increased risk of occupational allergy. *BMJ* 1981;**283**:1215–20.
22 **Janson C**, Chinn S, Jarvis D, *et al.* Effects of passive smoking on respiratory symptoms, bronchial responsivesness, lung function, and total serum IgE in the European Community Respiratory Health Survey: a cross-sectional study. *Lancet* 2001;**358**:2103–9.
23 **Chen R**, Tunstall-Pedoe H, Tavendale R. Environmental tobacco smoke and lung function in employees who never smoked: the Scottish MONICA study. *Occup Environ Med* 2001;**58**:563–8.
24 **Jensen EJ**, Dahl R, Steffensen F. Bronchial reactivity to cigarette smoke in smokers: repeatability, relationship to methacholine reactivity, smoking and atopy. *Eur Respir J* 1998;**11**:670–6.
25 **Menon P**, Rando RJ, Stankus RP, *et al.* Passive cigarette smoke-challenge studies: increase in bronchila hyperreactivity. *J Allergy Clin Immunol* 1992;**89**:560–6.
26 **Shoji S**, Ertl RF, Rennard SI. Cigarette smoke stimulates release of neutrophil chemotactic activity from cultured bronchial epithelial cells. *Clin Res* 1987;**35**:539A.
27 **Sherill DL**, Holberg CJ, Enright PL, *et al.* Longitudinal analysis of the effects of smoking onset and cessation on pulmonary function. *Am J Respir Crit Care Med* 1994;**149**:591–7.
28 **Coultas DB**. Passive smoking and risk of adult asthma and COPD: an update. *Thorax* 1998;**53**:381–7.
29 **Jaakkola MS**, Jaakkola JJK, Becklake MR, *et al.* Passive smoking and evolution of lung function in young adults. A 8-year longitudinal study. *J Clin Epidemiol* 1995;**48**:317–27.
30 **Bergman TA**, Johnson DL, Boatright DT, *et al.* Occupational exposure of nonsmoking nightclub musicians to environmental tobacco smoke. *AIHA Journal* 1996;**57**:746–52.
31 **Christiani DC**, Wang XR, Pan LD, *et al.* Longitudinal changes in pulmonary function and respiratory symptoms in cotton textile workers. A 15-yr follow-up study. *Am J Respir Crit Care Med* 2001;**163**:847–53.

32 **Erkinjuntti-Pekkanen R**, Slater T, Cheng S, *et al*. Two year follow up of pulmonary function values among welders in New Zealand. *Occup Environ Med* 1999;**56**:328–33.
33 **Bell C**, Urbach D. Bartenders' pulmonary function after establishment of a smoke-free workplace. *JAMA* 1999;**282**:629.
34 **Morgan WK**, Reger RB. Rise and fall of the FEV1. *Chest* 2000;**118**:1639–44.
35 **McKerrow CB**, Rossiter CE. An annual cycle in the ventilatory capacity of men with pneumoconiosis and of normal subjects. *Thorax* 1968;**23**:340–9.
36 **Spodnik MJ**, Cushman GD, Kerr DH, *et al*. Effects of environment on respiratory function. Weekly studies on young male adults. *Arch Environ Health* 1966;**13**:243–54.