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Assessment of risk for asthma initiation and cancer and heart disease deaths among patrons and servers due to secondhand smoke exposure in restaurants and bars

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ABSTRACT

Background Despite efforts to reduce exposure to secondhand smoke (SHS), only 5% of the world's population enjoy smoke-free restaurants and bars.

Methods Lifetime excess risk (LER) of cancer death, ischaemic heart disease (IHD) death and asthma initiation among non-smoking restaurant and bar servers and patrons in Minnesota and the US were estimated using weighted field measurements of SHS constituents in Minnesota, existing data on tobacco use and multiple dose-response models.

Results A continuous approach estimated a LER of lung cancer death (LCD) of 18×10^{-6} (95% CI 13 to 23×10^{-6}) for patrons visiting only designated non-smoking sections, 80×10^{-6} (95% CI 66 to 95×10^{-6}) for patrons visiting only smoking venues/sections and 802×10^{-6} (95% CI 658 to 936×10^{-6}) for servers in smoking-permitted venues. An attributable-risk (exposed/non-exposed) approach estimated a similar LER of LCD, a LER of IHD death about 10^{-2} for non-smokers with average SHS exposure from all sources and a LER of asthma initiation about 5% for servers with SHS exposure at work only. These risks correspond to 214 LCDs and 3001 IHD deaths among the general non-smoking population and 1420 new asthma cases among non-smoking servers in the US each year due to SHS exposure in restaurants and bars alone.

Conclusions Health risks for patrons and servers from SHS exposure in restaurants and bars alone are well above the acceptable level. Restaurants and bars should be a priority for governments' effort to create smoke-free environments and should not be exempt from smoking bans.

BACKGROUND

Secondhand smoke (SHS) exposure has many adverse health effects and causes over 600 000 deaths annually.¹ Despite efforts to reduce SHS exposure in public places, national smoke-free environment (SFE) regulations for restaurants and bars protect only 5% of the world's population.² A quarter of the US population remains unprotected by SFE policies in restaurants and 35% in bars.³ Tobacco companies use multiple strategies to oppose SFE policies and sometimes succeed in overturning existing smoking bans; for example, by July 2012 a total of 16 US municipalities had repealed, weakened, or postponed their SFE regulations due to such efforts.⁴ Patronising restaurants and bars may be a predominant source of SHS

exposure for those living in smoke-free homes, and occupational SHS exposure of servers may be very high.

Jamrozik estimated that 54 deaths from lung cancer, ischaemic heart disease (IHD) or stroke among hospitality workers were attributed to their workplace SHS exposure.⁵ The published risk assessments^{6–9} are limited to servers only, and the lung cancer or heart disease risks were based on non-representative exposure data; none have estimated health risks for patrons, nor have any examined asthma initiation. A comprehensive risk assessment based on more comprehensive accurate exposure assessment is imperative for policymakers who do not have SFE policies or are considering overturning SFE policies in restaurants and bars.

Steenland described two general approaches to assess risk due to SHS exposure,¹⁰ a unit risk (UR) or continuous approach based on field measurements and a categorical approach based on relative risks (RR) comparing exposed to non-exposed populations. Both approaches are used in this paper. This paper focuses on the lifetime excess risks (LER) of non-smoking servers and adult patrons for cancer death, IHD death and asthma induction. LER is the difference in lifetime risks/probability of having a disease between exposed and unexposed populations. LER of cancers due to exposure to various risk factors has been frequently assessed by researchers.^{11–14} Assessment of LER of heart diseases due to SHS exposure has also been applied by the US Occupational Safety and Health Administration (OSHA)¹⁵ and by Steenland.¹⁰

METHODS

Before the October 2007 implementation of SFE policies covering indoor public places and workplaces in Minnesota, a study was conducted to collect detailed SHS exposure data from a statistically representative sample of 65 restaurants and bars that permitted smoking within a 20-mile radius of downtown Minneapolis. Substrata were sampled in proportion to the number of employees working in smoking-permitted venues and the expected SD of SHS concentrations. From February through to September 2007, 2423 short-term visits (median: 12 min) were made to conduct systematically real-time area monitoring of fine particulate matter (PM_{2.5}), and to observe the number of lit cigarettes, customers and workers at 3 different times of day (lunch, dinner and evening) on 4

different day types (Fridays, Saturdays, Sundays and other weekdays) in each venue. These peak-patronage-time visits are used as the basis for estimating exposures. Another 210 2 h visits were conducted at dinner and in the evening to make the same observations, monitor PM_{2.5} and sample multiple gas phase SHS tracers, including nicotine, 3-ethenylpyridine, pyridine, pyrrole, picroine, quinoline and myosmine; most (n=186) of these were conducted on Fridays and Saturdays. For venues restricting smoking to designated sections (n=40), most sampling was conducted in smoking sections, with observations recorded from both sections. Simultaneous measurements were made in both sections during 16 visits.

Estimate of cancer risk using a continuous approach based on field measurements

Repace and Lowrey¹⁶ developed a model to predict the risk of lung cancer death (LCD) due to SHS exposure using PM_{2.5} as a tracer (SHS-PM_{2.5}). The model was validated by predicting epidemiologically derived observational data to within 5%. They estimated that, for the general US population, the risk of LCD is 5×10^{-5} for exposure to 1 mg/day of SHS-PM_{2.5} for 1 year and the LER of LCD can be estimated by equation 1:

$$\text{LER of LCD} = 5 \times 10^{-5} (\text{mg/day-year})^{-1} \times \text{Daily dose} (\text{mg/day}) \times \text{Years} \quad (1)$$

$$\text{Daily dose} (\text{mg/day}) = C_{\text{SHS-PM}} (\text{mg/m}^3) \times \text{BR} (\text{m}^3/\text{h}) \times \text{Time} (\text{h/day}) \times f$$

Where daily dose is the exposure dose of SHS-PM_{2.5} in mg/day; years is the number of years exposed to SHS; $C_{\text{SHS-PM}}$ is the average concentration of SHS-PM_{2.5} in mg/m³ during the period of exposure; BR is the breathing rate in m³/h; time is the average hours per day exposed to SHS; f is the days per week exposed divided by 7 days.

The SHS-PM_{2.5} concentration was defined as the difference between indoor and outdoor measurements during each visit. The sample was analysed as a 2-stage stratified cluster sample with the first stage consisting of the 9 venue-type/size strata from which 65 venues (clusters) were drawn, and the second stage consisting of 12 day-time strata from which a sample of all possible visits in a year was drawn by quasisystematic sampling. The Complex Samples module of Predictive Analytics SoftWare (PASW) 18.0.0 (SPSS, Inc., Chicago, Illinois, USA) was used to compute statewide averages in smoking-permitted venues/sections for each day-time period, with each measurement or observation weighted by $1/(\text{Venue selection probability} \times \text{visit selection probability})$ (weight 1). The SE was computed by the Complex Samples module using a Taylor linearisation formula¹⁷ that takes the sample design into account. These 'venue-weighted' concentrations were further weighted by the number of patrons in smoking venues/sections to estimate the average concentration to which all patrons were exposed in smoking venues/sections (weight 2). The average SHS-PM_{2.5} level to which patrons in designated non-smoking section were exposed was estimated by multiplying the SHS-PM_{2.5} level in smoking sections by 0.525 (the ratio of the average non-smoking-section SHS-PM_{2.5} level to the average smoking-section level according to simultaneous side-by-side measurements during 16 visits),¹⁸ then weighted by weight 1 and the number of patrons in non-smoking sections (weight 3). To estimate the average SHS-PM_{2.5} level to which servers were exposed, the smoking and non-smoking section concentrations were combined in proportion to the number of patrons in each section (weight 4), assuming that

the time spent by servers in each section was proportional to the number of patrons. See online table S1 of the supplementary materials for these weighted concentrations.

To estimate the daily dose, breathing rates (in equation 1) recommended by the US Environmental Protection Agency (EPA) were used: 1.6 m³/h for servers at a moderate activity level and 1.0 m³/h for patrons at a light activity level.¹⁹ To avoid overestimating servers' exposure during work, their exposure time was assumed to be 4 h a day during peak patronage times. OSHA strives to make the workplace safe for everyone, and the agency uses a 45 year working life as standard. Thus, a typical assumption of working 5 days per week for a working life of 45 years was applied. Patrons were assumed to visit a restaurant or bar once every week, with an average time of 86 min each time²⁰ for 60 years.

The risk of cancer due to exposure to volatile organic compounds from SHS (SHS-VOCs) was quantified by using the cancer unit risk estimate (URE) reported by the US EPA²¹ or the equivalent cancer UR reported by the California EPA.²² URE and UR both describe the excess cancer risk associated with a daily inhalation exposure to 1 µg/m³ of a given chemical for a lifetime of 70 years, assuming 20 m³/day of inhalation. The LER of cancers can be estimated by equation 2:

$$\text{LER of cancers} = C_{\text{SHS-VOC}} (\mu\text{g/m}^3) \times \text{URE or UR} (\mu\text{g/m}^3)^{-1} \\ C_{\text{SHS-VOC}} (\mu\text{g/m}^3) = C_{\text{SHS-PM}} (\mu\text{g/m}^3) \times (\text{EF}_{\text{SHS-VOC}}/\text{EF}_{\text{SHS-PM}}) \times F \quad (2)$$

Where $C_{\text{SHS-VOC}}$ is the daily average concentration of a SHS-VOC during a 70 year lifetime, µg/m³; URE is the cancer URE reported by the US EPA, (µg/m³)⁻¹; UR is the cancer UR reported by the California EPA, (µg/m³)⁻¹; years is the number of years exposed to SHS; $C_{\text{SHS-PM}}$ is the average concentration of SHS-PM_{2.5} during peak patronage time, µg/m³; $\text{EF}_{\text{SHS-VOC}}$ and $\text{EF}_{\text{SHS-PM}}$ are the average emission factors of SHS-VOC and SHS-PM_{2.5} from the literature, respectively, µg/cigarette; F is the adjustment factor, which is $(4 \text{ h/day} \times 1.6 \text{ m}^3/\text{h} / (20 \text{ m}^3/\text{day}) \times 5 \text{ day}/7 \text{ day} \times 45 \text{ years}/70 \text{ years})$ for servers and $(1.4 \text{ h/day} \times 1.0 \text{ m}^3/\text{h} / (20 \text{ m}^3/\text{day}) \times 1 \text{ day}/7 \text{ day} \times 60 \text{ years}/70 \text{ years})$ for patrons.

The overall cancer risk from exposure to SHS-VOCs was estimated by summing the risk of cancers of all sites from exposure to individual SHS-VOCs. The URE was used when it was available from the US EPA website; otherwise, the UR reported by the California EPA was used. Since SHS-PM_{2.5} was most intensively monitored, and the ratios of simultaneous measurements of SHS-PM_{2.5} and SHS-VOC tracers were quite similar to the ratios of their emission factors (EF) reported in the literature (see online table S2 in the supplementary material), exposure to SHS-VOCs was estimated from the ratios of their EFs to SHS-PM_{2.5} EF. Nine SHS-VOCs with URE/UR and EF were available, thus the LER of cancers due to SHS-VOCs exposure was estimated as the sum of cancer risk from these nine SHS-VOCs.

Estimate of health risk by attributable risk assessment method (exposed/non-exposed)

This method has been used to assess disease burden due to SHS exposure worldwide¹ and in the US.²³ Briefly, the burden of a specific disease due to SHS exposure was estimated from the population attributable fraction (PAF), defined as the proportional reduction in disease that would occur if the exposure was

reduced to zero. The attributable burden (AB) of a disease due to SHS exposure can be estimated by equation 3^{1 23} and the attributable risk of death/case can be estimated by equation 4:¹⁰

$$AB_{SHS} = B_{ns} \times PAF_{SHS}$$

$$B_{ns} = (B - AB_{sm}) - (B - AB_{sm}) \times p_{sm} = (B - AB_{sm}) \times (1 - p_{sm})$$

$$PAF_{SHS} = p_{SHS} (RR_{SHS} - 1) / [p_{SHS} (RR_{SHS} - 1) + 1] \tag{3}$$

$$AER_{SHS} = AB_{SHS} / P_{risk} \tag{4}$$

$$LER_{SHS} = AER_{SHS} \times \text{Years}$$

Where B is the total number of deaths/cases per year among the whole population; B_{ns} is the number of deaths/cases per year among non-smokers; AB_{sm} is the attributable burden of a disease in number of deaths/cases per year among smokers due to smoking; AB_{SHS} is the attributable burden of a disease in number of deaths/cases per year among non-smokers due to SHS exposure; PAF_{SHS} is the PAF of the disease burden due to SHS exposure among non-smokers; p_{sm} is the prevalence of current smoking; p_{SHS} is the prevalence of SHS exposure; RR_{SHS} is the relative risk of a disease due to SHS exposure among non-smokers; AER_{SHS} is the annual excess risk of death/case due to SHS exposure among non-smokers; P_{risk} is the population at risk, that is, current non-smokers aged 35 years or older; LER_{SHS} is the life time excess risk of death/case due to SHS exposure; years is the number of years of exposure during lifetime.

To estimate the number of LCD and IHD deaths due to SHS exposure among non-smoking adults in Minnesota and the US, the latest (2004) disease burden of these two diseases among all adults aged 35 and older (B in equation 3) and that attributed to smoking (AB_{sm} in equation 3) were obtained from the US Centers for Disease Control (CDC) website;²⁴ the prevalence of current smoking (p_{sm}) in the US and Minnesota and the prevalence of SHS exposure (p_{SHS}) in the US were obtained from CDC reports.²⁵⁻²⁷ The prevalence of SHS exposure (p_{SHS}) for Minnesotans relative to the US was assumed to be proportional to the ratios of prevalence of current smoking among the Minnesota population and the US population. RR of LCD or IHD due to SHS exposure (RR_{SHS}) was acquired from the meta-analysis in the US Surgeon General's Report 2006.²⁸ Thus, the LER_{SHS} is the LER of non-smoking adults due to exposure to the average SHS concentration of all sources, not just of restaurants and bars.

The 1992-1994 *National Human Activity Pattern Survey* for the US determined the percentage of time exposed to SHS that

occurred in restaurants and bars was 8.5% for males and 9.1% for females.²⁹ Although exposure intensity varies in different microenvironments, with SHS levels in bars and restaurants often higher than other indoor locations,³⁰ these fractions were applied conservatively to estimate the LCD and IHD deaths due to SHS exposure in restaurants and bars for the general population without adjustment for relative exposure intensities.

Similar approaches were used to estimate the risk of asthma initiation due to SHS exposure at work for never-smoking restaurant and bar servers in Minnesota and the US According to the CDC's *Work-Related Lung Disease Surveillance System*,³¹ the average prevalence of current asthma (asthma attack in the past 12 months) among never-smoking servers aged 18 and over was 3.8% (95% CI 3.0% to 4.6%) from 1997 to 2005. Asthma incidence rate was estimated to be 10% of the prevalence of current asthma.³² Data on restaurant and bar server employment in 2004 was obtained from the US Bureau of Labor Statistics³³ and the never-smoking rate for the server population was assumed the same as the general population.^{25 26}

To estimate the population attributable fraction (PAF_{SHS}) of asthma initiation, the RR was taken from the most relevant study available,³⁴ and the prevalence of SHS exposure at work in restaurants and bars was estimated from the percentage of the population not covered by smoking bans for restaurants and/or bars in May 2007 in Minnesota and in January 2012 in the US.^{3 35}

RESULTS

Continuous approach based on field measurements

The average SHS-PM_{2.5} concentration to which patrons were exposed during their visits to smoking venues/sections was 134 µg/m³ (SE 12 µg/m³), the corresponding average for patrons visiting non-smoking sections was 30 µg/m³ (SE 4 µg/m³) and the weighted average for servers working in smoking-permitted venues was 78 µg/m³ (SE 7 µg/m³).

Based on the risk model developed by Repace and Lowrey,¹⁶ this exposure corresponds to a LER of LCD 18 × 10⁻⁶ (95% CI 13 to 23 × 10⁻⁶) for patrons visiting only designated non-smoking sections and 80 × 10⁻⁶ (95% CI 66 to 95 × 10⁻⁶) for patrons visiting only smoking venues/sections for 1.4 h per week in 70 years and 802 × 10⁻⁶ (95% CI 658 to 936 × 10⁻⁶) for servers who are exposed for 20 h per week for 45 years (table 1). The LER of overall cancer death due to exposure to nine SHS-VOCs is 48 × 10⁻⁶ for servers, 4.8 × 10⁻⁶ for patrons visiting only smoking venues/sections and 1.1 × 10⁻⁶ for patrons visiting only designated non-smoking sections; most of these cancers risks were non-lung cancer risks (table 2).

Table 1 Servers' and patrons' risk of lung cancer death (LCD) due to exposure to secondhand smoke in restaurants and bars in Minnesota

	Servers	Patrons	
		Smoking venues/sections	Non-smoking sections
Weighted SHS-PM _{2.5} levels, mean (95% CI), µg/m ³	78 (64 to 91)	134 (110 to 158)	30 (22 to 38)
Dose response according to Repace and Lowrey ¹⁶	5 × 10 ⁻⁵ per year for exposure to 1 mg per day		
Breathing rates, m ³ /h	1.6	1.0	1.0
Average hours per day exposed to SHS, h/day	4	1.4	1.4
Days per week exposed to SHS, day/week	5	1	1
Number of years exposed to SHS, years	45	60	60
Lifetime attributable risk of LCD (95% CI), 10 ⁻⁶	802 (658 to 936)	80 (66 to 95)	18 (13 to 23)

The 95% CIs presented here indicate only the variance of the weighted SHS-PM_{2.5} levels during peak patronage times; uncertainties from other sources are not integrated. SHS, secondhand smoke; PM_{2.5}, fine particulate matter.

Table 2 Servers' and patrons' cancer risk due to exposure to nine SHS-VOCs in Minnesota restaurants and bars

Carcinogenicity effect	URE/UR* (10 ⁻⁶)	EF† (µg/ cigarette)	Servers		Patrons, smoking venues/sections		Patrons, non-smoking sections	
			Level‡ (µg/m ³)	LER§ (10 ⁻⁶)	Level‡ (µg/m ³)	LER§ (10 ⁻⁶)	Level‡ (µg/m ³)	LER§ (10 ⁻⁶)
PM _{2.5}		12471	11.5	–	1.1	–	0.2	–
Acetaldehyde	Nasal cancer in rats	2.20	2.11	4.6	0.20	0.5	0.04	0.1
Acrylonitrile	Lung cancer in humans	68.0	0.16	10.6	0.01	1.1	0.00	0.2
Benzene	Leukaemia in humans	7.80	0.40	3.1	0.04	0.3	0.01	0.06
1,3-Butadiene	Lymphohaematopoietic cancer in humans	30.0	0.26	7.7	0.02	0.78	0.01	0.2
Ethylbenzene	Kidney cancer in rats	2.50	0.12	0.3	0.01	0.03	0.00	0.01
Formaldehyde	Nasal cancer in humans	13.0	1.01	13.2	0.09	1.3	0.02	0.3
Naphthalene	Nasal cancer in rats	34.0	0.04	1.4	0.00	0.1	0.0009	0.03
<i>N</i> -Nitrosodimethylamine	Liver cancer in rats	14000	0.00052	7.3	0.00005	0.7	0.00001	0.2
<i>N</i> -Nitrosopyrrolidine	Liver cancer in rats	600	0.10	0.00010	0.06	0.00001	0.005	0.000002
Total risk of all cancers	–	–	–	48.3	–	4.8	–	1.1
Total risk of non-lung cancers	–	–	–	37.7	–	3.8	–	0.8

*URE/UR: unit risk estimate reported by the US EPA or unit risk reported by the California EPA.

†Average of EFs reported in the literature (see table A2 in online appendices).

‡Level: daily average exposure concentration during a lifetime of 70 years, adjusted by the factor in equation 2.

§LER, lifetime attributable risk of cancers.

Only nine SHS-VOCs with EF (in µg/cigarette, available from the literature) and URE or UR available from the US EPA or the California EPA website are included in this table.

Compounds for which either EF or URE/UR were unavailable are not included even though they are known carcinogens.

EF, emission factor; EPA, Environmental Protection Agency; LER, lifetime excess risk; PM_{2.5}, fine particulate matter; SHS-VOC, volatile organic compounds from SHS; SHS, secondhand smoke; UR, unit risk; URE, unit risk estimate.

Attributable risk assessment approach (exposed/non-exposed)

This approach estimates the LER to be 800×10^{-6} (95% CI 430 to 1180×10^{-6}) for LCD and 7670×10^{-6} (95% CI 4830 to $10\,510 \times 10^{-6}$) for IHD death for the general non-smoking population (including patrons and servers), due to exposure to the average SHS concentration of all sources in Minnesota, and 890×10^{-6} (95% CI 480 to 1290×10^{-6}) for LCD and $12\,530 \times 10^{-6}$ (95% CI 8430 to $16\,620 \times 10^{-6}$) for IHD death in the US (table 3). The LER of asthma initiation is estimated to be 7.2% (95% CI 2.4% to 11.9%) for non-smoking servers due to SHS exposure in restaurants and bars in Minnesota and 4.1% (95% CI 0.7% to 7.5%) for non-smoking servers in the US. These risks correspond to 3 LCDs and 32 IHD deaths per year among the general non-smoking population, 53 new asthma cases per year among non-smoking servers in Minnesota, 214 LCDs and 3001 IHD deaths per year among the general non-smoking population and 1420 new asthma cases per year among non-smoking servers in the US (tables 3 and 4).

DISCUSSION

This paper is the first quantitative risk assessment for restaurant and bar patrons, and of asthma and cancers other than lung cancer. The quality of the underlying exposure data is vastly superior to that in any of the previous SHS risk studies.^{6–9} Measurements from only one US restaurant⁹ were used in any of these SHS risk assessment papers; and all those studies were based on convenience sample of sites or subjects. In contrast, the 65 Minnesota bars and restaurants reported here were chosen systematically to represent venues permitting smoking, and over 2000 measurements were made methodically at different times of day and days of the week.

In addition, three papers^{6–8} used a single dose-response model published by Repace and Lowrey¹⁶ to estimate the LCD risk and/or IHD risk for workers; one⁹ based its dose-response

model on potency factors for diesel exhaust. By contrast, our study used multiple approaches to estimate the health risks and our results converged.

Restaurants and bars are major employers³⁶ and are important public places for the general population; 66% of adults eat out at least weekly.³⁷ However, about 30% of the US population and 95% of the world population remain unprotected by SFE policies in restaurants and bars.^{2,3}

A LER of 1×10^{-6} has been considered a de minimis risk, below regulatory concern, while a LER of 3×10^{-4} has been considered a de manifestis risk, one of obvious concern that public agencies will usually regulate to mitigate when recognised.³⁸ Among workers, a LER of 1×10^{-3} has been considered a significant risk, an unsafe level often used as a benchmark by the US Occupational Safety and Health Administration. Results of this study indicate that SHS-induced LER of LCD for restaurant and bar servers is much higher than the de manifestis risk level, regardless of which dose-response model is used for the estimates. The LER of non-lung cancers due to exposure to SHS-VOCs adds to the LER of LCD and is underestimated because we estimated effects for only nine SHS-VOCs for which EF and cancer UREs have been reported, while the impact of other carcinogens such as tobacco specific nitrosamines was omitted. The LER of IHD death for restaurant and bar non-smoking servers is probably much higher than the significant risk. The LER of asthma initiation could be higher than 10% for non-smoking servers. Other studies around the world have reported higher SHS levels in restaurants and bars than this study.^{39–42} The health risks from SHS exposure in those cities/countries are expected to be higher.

However, by July 2012 SFE laws already enacted in 16 US municipalities had been repealed, weakened, or postponed⁴ due to anti-smoking ban efforts of the tobacco industry; and a bill proposed to allow smoking in Minnesota bars in 2011.⁴³ The significant disease burden imposed on the population from SHS

Table 3 Attributed death and lifetime attributable risk of LCD and IHD death due to SHS exposure in Minnesota and US, 2004*

	Minnesota†		USA†	
	Lung cancer	Ischaemic heart disease	Lung cancer	Ischaemic heart disease
Total burden (B), 2004, deaths/year‡	2352	4861	157908	450043
Attributable burden due to smoking (AB _{sm}), 2004, deaths/year‡	1838	776	125542	72715
Population at risk, current non-smokers aged 35+ (P _{risk}), 2004‡	Male: 1 026 443 Female: 1 140 400		Male: 57 058 144 Female: 65 584 599	
Prevalence of current smoking (p _{sm}), 2004, %§	Male: 22.0 (17.7, 26.3) Female: 19.5 (15.8, 23.2)		Male: 23.4 (21.6, 25.2) Female: 18.5 (17.1, 19.9)	
Prevalence of SHS exposure (p _{SHS}), 2003–2004, %¶	Male: 48.8 (41.6, 55.9) Female: 46.6 (38.8, 54.4)		Male: 51.9 (44.3, 59.5) Female: 44.2 (36.8, 51.6)	
Relative risk due to SHS exposure (RR _{SHS})**	1.22 (1.12, 1.32)	1.27 (1.17, 1.37)	1.22 (1.12, 1.32)	1.27 (1.19, 1.36)
Disease burden among non-smokers (B _{ns}), 2004, deaths/year	410 (380, 439)	3235 (3019, 3450)	25 794 (24 031, 27 557)	298 365 (278 356, 318 373)
Overall death rate among non-smokers aged 35+, per 10 ⁴	1.9 (1.7, 2.1)	14.9 (13.5, 16.3)	2.1 (1.9, 2.3)	24.3 (22.0, 26.6)
PAF of disease burden among non-smokers due to SHS exposure (PAF _{SHS}), %	9.5 (5.4, 13.6)	11.4 (7.3, 15.5)	9.5 (5.4, 13.6)	11.4 (7.9, 15.0)
Attributable burden among non-smokers due to SHS exposure (AB _{SHS}), deaths/year	39 (26, 52)	369 (273, 466)	2412 (1630, 3195)	34 143 (26 281, 42 004)
Annual attributable risk due to SHS exposure (AAR _{SHS}), 10 ⁻⁶	17.8 (9.5, 26.1)	170.4 (107.3, 233.5)	19.7 (10.8, 28.6)	278.4 (187.4, 369.3)
Lifetime attributable risk due to SHS exposure for 45 years (LER _{SHS}), 10 ⁻⁶	800 (430, 1180)	7670 (4830, 10 510)	890 (480, 1290)	12 530 (8430, 16 620)
Percentage of SHS exposure in restaurants and bars of total SHS exposure in terms of time, %††	Male: 8.5 Female: 9.1		Male: 8.5 Female: 9.1	
Deaths attributed to SHS exposure in restaurants and bars, no./year	3 (2.6, 4.3)	32 (26, 38)	214 (164, 263)	3001 (2512, 3490)

*Assessment of disease burden was conducted separately for male and female populations, but aggregated data are presented in this table to save space; Overall death rate among non-smokers aged 35+, PAF_{SHS}, AAR_{SHS} and LER_{SHS} were weighted estimates by population at risk of males and females.

†Except ‡ § ¶ ** ††, all the other numbers were estimated according to equation 3 or 4; the CIs were estimated by propagation of uncertainties of reported parameters.

‡Data obtained from the CDC Smoking-Attributable Mortality, Morbidity, and Economic Costs (SAMMEC) website.²⁴

§Data obtained from CDC Morbidity and Mortality Weekly Report (MMWR).²⁶

¶Data from CDC MMWR, which was defined as the percentage of non-smoking population with serum cotinine ≥ 0.05 ng/ml.²⁷

**Relative risks due to SHS exposure from the meta-analysis in the Surgeon General's Report.²⁸

††Data from the 1992 to 1994 National Human Activity Pattern Survey (NHAPS) for the USA.²⁹

AB, attributable burden; IHD, ischaemic heart disease; LCD, lung cancer death; LER, lifetime excess risk; PAF, population attributable fraction; RR, Relative risk; SHS, secondhand smoke.

exposure in restaurants and bars, as reported herein, supports the priority that should be accorded SFE regulation in these venues.

To oppose comprehensive smoking bans in restaurants and bars, the tobacco industry advocates designated non-smoking sections. Unfortunately, this approach offers limited protection for non-smokers, especially for non-smoking servers, who must serve customers in both sections. Our study showed that the LER of LCD is more than 10 times the de minimis risk even for non-smoking patrons who visit only designated non-smoking sections once a week during their lifetime; furthermore the LER of IHD death is substantially higher.

The estimated overall IHD death rate among general non-smokers aged 35 and over in the US in this study (24.3×10^{-4} in table 3) is quite close to the IHD death rate among general never-smokers reported in four cohort studies⁴⁴ weighted by the age and gender specific non-smoking population of 35 and over in 2004, which is 212×10^{-5} . The consistency between these indicates that the risk assessment and the underlying assumptions used in this study are reasonable and reliable.

The CIs reported in this study reflect only the variations of measured SHS levels or the reported uncertainties of related parameters used in this study and do not encompass all uncertainty of this risk assessment. The variance of servers' and patrons' exposure to SHS-PM_{2.5} in restaurants and bars in Minnesota was relatively well characterised by the selection and

weighting of the venues and the inclusion of multiple measurements taken during different times of a day and days of the week. Servers and patrons are transient populations with a high turnover, the LER estimated in this study is likely to overestimate the risk for servers and patrons who are not exposed as long as assumed. However, regulations should protect those who do make careers as servers, as well as those who visit regularly. A study reported a current smoking rate of 44.5% (95% CI 35.9% to 53.1%) among servers aged 17 years and over and of 28.3% (95% CI 26.9% to 29.8%) among the general adult population from 1988–1994.⁴⁵ Thus by using the proportion of current non-smokers of the general population, the number of new asthma cases attributed to SHS exposure for non-smoking servers may be overestimated, however, the individual lifetime risk remains the same.

Underestimation of these health risks may arise from assuming servers are occupationally exposed to SHS only 4 h per day and that patrons are exposed only 86 min per week; longer workdays or more frequent patronage in these venues would increase the risk. Only nine SHS-VOCs were included in the URE while other carcinogens were disregarded due to lack of potency or emission data. When using the attributable risk assessment approach, we assumed that SHS exposure levels in restaurants and bars were similar to the average concentration of all sources for the population. In addition, the risks for only three of the multiple diseases caused by SHS exposure were evaluated, while other health outcomes, for

Table 4 Attributed cases and lifetime attributable risk of asthma initiation among never smoking servers due to SHS exposure at work in restaurants and bars in Minnesota and in US, 2004*

	Minnesota†	USA†
Number of restaurant and bar servers employed, 2004‡	63300	2700590
Never smoking rate, 2004, %§	52.5 (49.2, 55.8)	57.6
Prevalence of current asthma among never smoking restaurant and bar servers, %¶	3.8 (3.0, 4.6)	3.8 (3.0, 4.6)
New asthma cases among never smoking restaurant and bar servers (B _{ns}), cases/year**	126 (104, 148)	5 911 (4861, 6961)
Percentage of population covered by smoke-free restaurants and/or bars (1-p _{SHS}), %††	38.1 (35.2, 41.0)	Restaurants: 74.5 Bars: 63.7
Relative risk of asthma initiation due to SHS exposure (RR _{SHS})‡‡	2.16 (1.26, 3.72)	2.16 (1.26, 3.72)
Population at risk, never smoking restaurant and bar servers (P _{risk})	33233 (31558, 34907)	1555540
PAF of new asthma cases among never smoking restaurant and bar servers due to SHS exposure (PAF _{SHS}), %	41.8 (16.0, 67.6)	24.0 (4.7, 43.3)
New asthma cases attributed to SHS exposure (AR _{SHS}), cases/year	53 (25, 80)	1420 (449, 2390)
Annual attributable risk of asthma initiation due to SHS exposure (AAR _{SHS}), 10 ⁻⁶	1588 (542, 2635)	913 (153, 1672)
Lifetime attributable risk of asthma initiation for restaurant and bar servers due to SHS exposure at work for 45 years (LER _{SHS}), %	7.2 (2.4, 11.9)	4.1 (0.7, 7.5)

*Assessment of disease burden was conducted separately for waiters/waitresses and bartenders, but aggregated data were presented in this table to save space; PAF_{SHS}, AAR_{SHS} and LER_{SHS} were weighted estimates by population at risk.

†Except ‡ § ¶ ** †† ‡‡, all the other numbers were estimated according to equation 3 or 4; the CIs were estimated by propagation of uncertainties of reported parameters.

‡Data from Occupational Employment Statistics, US Bureau of Labor Statistics.³³

§Data from CDC Morbidity and Mortality Weekly Report.^{25, 26} The variance for never smoking rate of the US population was not reported.

¶Current asthma was defined as having an asthma attack in past 12 months. Data are from CDC National Institute for Occupational Safety and Health (NIOSH) website of Work-Related Lung Disease (WoRLD) Surveillance System (see table 9–20 at <http://www2a.cdc.gov/drds/WorldReportData/>); which presents the average prevalence of current asthma among never smoking employees aged 18 and over in eating and drinking places from 1997–2005.

**Estimated from ‡ † ‡ and the assumption that the incidence rate of new asthma cases is 10% of prevalence of current asthma according to Rudd and Moorman.³²

††Data from Minnesota Adult Tobacco Survey report for Minnesota³⁵ and from American Nonsmokers' Rights Foundation websites for US³ (no variance reported). The coverage percentage was for May 2007 for Minnesota and for January 2011 for USA.

‡‡Relative risk of asthma initiation related to workplace SHS exposure reported by Jaakkola *et al.*³⁴

AR, attributable risk; LER, lifetime excess risk; SHS, secondhand smoke; PAF, population attributable fraction; RR, relative risk.

example, breast cancer for young non-smoking servers, acute heart disease events, acute respiratory irritations for children, etc., were not included due to limited data to quantify these risks. Furthermore, we used a higher percentage of population covered by smoke-free restaurants and bars than would have been the case in earlier years, which can underestimate the total disease burden among the population due to SHS exposure in restaurants and bars.

The most important assumption in this risk assessment is that the association between SHS and the diseases of interests (lung cancer, IHD and asthma initiation among non-smokers) is causal. The causal relationships have been supported by several authority reports.^{28, 46, 47} This risk assessment also assumes that current SHS exposure levels have not changed during the lifetime for servers and patrons. In reality it has probably decreased. Mean air nicotine concentrations measured in restaurants and bars before 1999 ranged from 3–8 µg/m³ as reported by Hammond,⁴⁸ which is higher than 3 µg/m³ (95% CI 0 to 7 µg/m³) as measured in the Minnesota study in 2007.⁴⁹ Thus, the current risk assessment probably underestimated the LER of servers and patrons due to SHS exposure in restaurants and bars. Another important assumption is the use of a linear dose-response relationship when using the continuous risk assessment approach. Because there is no threshold effect from SHS exposure, this assumption is reasonable according to the US EPA risk assessment guidelines.⁵⁰

CONCLUSIONS

Different risk assessment approaches all revealed that the health risk for patrons visiting smoking restaurants and bars is well above the acceptable level, and that for servers exceeds the 'significant risk' level. This study provides strong evidence that smoking should be banned in hospitality venues to protect the public's health.

What this paper adds

- ▶ Secondhand smoke (SHS) exposure can cause multiple diseases, and SHS levels in restaurants and bars are often higher than in many other public places, however, few studies have comprehensively examined the health risks due to SHS exposure in restaurants and bars.
- ▶ Comprehensive smoke bans are the most effective way to protect people from SHS exposure, however, only 5% of the world's population enjoy smoke-free restaurants and bars, and there continue to be efforts by tobacco companies to remove or weaken existing smoking bans.
- ▶ Based on more comprehensive and accurate exposure assessment than the literature, this paper is the first to assess restaurant and bar patrons' health risks of cancers and heart diseases and servers' health risks of asthma initiation due to SHS exposure in restaurants and bars.
- ▶ This paper estimated the number of lung cancer and ischaemic heart disease deaths and new asthma cases among the Minnesota and the US populations due to SHS exposure in restaurants and bars alone.
- ▶ The results indicate that exposure to SHS in restaurants and bars alone can impose significant health risks to servers and patrons, and restaurants and bars should not be exempted from smoking bans.

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Supplementary Materials

Table S1 SHS PM_{2.5} concentrations in restaurants and bars by different time of day and different day of week, $\mu\text{g}/\text{m}^3$, Minnesota, 2007

			SHS PM _{2.5} , mean (SE)					
			n of visit	venue avg unweighted ^a	venue avg weighted 1 ^b	patrons_smks weighted 2 ^c	patrons_nsmks weighted 3 ^d	workers weighted 4 ^e
Restaurants	weekday	Lunch	142	35 (5)	40 (13)	75 (36)	14 (3)	36 (14)
		Dinner	154	71 (7)	66 (13)	105 (13)	26 (4)	65 (10)
		Evening	154	64 (6)	59 (10)	105 (17)	27 (6)	59 (11)
	Friday	Lunch	134	30 (4)	36 (10)	76 (29)	12 (5)	34 (12)
		Dinner	187	91 (7)	87 (16)	136 (24)	39 (5)	83 (14)
		Evening	160	108 (9)	98 (14)	158 (14)	54 (13)	107 (11)
	Saturday	Lunch	150	29 (4)	29 (7)	66 (21)	9 (2)	32 (8)
		Dinner	188	53 (4)	59 (9)	93 (15)	32 (7)	51 (7)
		Evening	165	90 (9)	76 (13)	151 (42)	31 (6)	88 (21)
	Sunday	Lunch	127	31 (4)	34 (5)	57 (11)	10 (3)	32 (6)
		Dinner	127	60 (7)	50 (11)	92 (17)	24 (4)	52 (10)
		Evening	140	61 (7)	58 (10)	98 (16)	17 (2)	56 (10)
Bars	weekday	Lunch	62	31 (6)	38 (8)	43 (13)	9 (1)	28 (6)
		Dinner	65	136 (17)	158 (51)	197 (56)	54 (10)	140 (38)
		Evening	61	155 (19)	159 (27)	181 (25)	62 (19)	156 (18)
	Friday	Lunch	63	27 (4)	29 (5)	29 (5)	24 (1)	22 (4)
		Dinner	66	180 (24)	191 (42)	217 (63)	51 (5)	164 (36)
		Evening	77	155 (19)	133 (18)	189 (32)	32 (4)	151 (21)
	Saturday	Lunch	61	45 (10)	51 (16)	88 (26)	26 (3)	38 (9)
		Dinner	70	114 (11)	124 (25)	129 (24)	49 (4)	105 (17)
		Evening	75	154 (22)	150 (27)	214 (36)	54 (3)	155 (26)
	Sunday	Lunch	56	41 (9)	56 (18)	78 (16)	4 (3)	48 (12)
		Dinner	58	91 (14)	112 (24)	145 (29)	49 (4)	95 (15)
		Evening	64	99 (11)	123 (28)	166 (47)	145 (9)	139 (33)
All	weekday	Lunch	204	34 (4)	40 (8)	63 (24)	14 (2)	33 (10)
		Dinner	219	90 (7)	98 (20)	143 (26)	31 (4)	90 (13)
		Evening	215	89 (8)	95 (12)	140 (15)	33 (7)	90 (10)
	Friday	Lunch	197	29 (3)	33 (7)	60 (21)	15 (4)	30 (9)
		Dinner	253	114 (8)	123 (18)	170 (29)	42 (4)	105 (13)
		Evening	237	123 (9)	111 (11)	172 (16)	53 (12)	120 (9)
	Saturday	Lunch	211	34 (4)	38 (7)	75 (16)	12 (3)	34 (6)
		Dinner	258	69 (5)	82 (11)	107 (13)	34 (6)	66 (7)
		Evening	240	110 (9)	103 (13)	178 (28)	35 (6)	109 (17)
	Sunday	Lunch	183	34 (4)	43 (8)	65 (9)	9 (3)	36 (5)
		Dinner	212	68 (6)	71 (11)	113 (16)	27 (5)	64 (8)
		Evening	204	73 (6)	84 (13)	130 (25)	52 (26)	84 (15)
Total			2633	72 (6)	78 (9)	134 (12)	30 (4)	78 (7)

^a Sample average SHS PM_{2.5} concentration without any weighting;

^b Average SHS-PM_{2.5} concentration by venue type, weighted to estimate average concentrations in the smoking venue/sections before the statewide ban went into effect (weight 1);

^c Average SHS-PM_{2.5} concentration to which patrons visiting only smoking venues/sections are exposed. Weighted by weight 1 and number of patrons in each smoking venue/section (weight 2);

^d Average SHS PM_{2.5} concentration to which patrons visiting only designated non-smoking sections are exposed. Weighted by weight 1 and number of patrons in these section (weight 3);

^e Average SHS PM_{2.5} concentration to which servers working in smoking-permitted restaurants and bars are exposed. Weighted by weight 1 and the proportion of time spent working in designated smoking sections and nonsmoking sections if present (weight 4).

Table S2 Emission factors (EFs) from the literature for SHS specific compounds and volatile organic compounds and ratios of EFs and side-by-side field measurements to PM_{2.5} or 3-Ethenylpyridine (3EP)

	Mean EF ^a μg/cig	range of EF ^b μg/cig	ratio of EF _{PM} /EF _{VOC}	ratio of C _{PM} /C _{VOC} ^c	ratio of EF _{VOC} /EF _{3EP}	ratio of C _{VOC} /C _{3EP} ^d
Selected SHS tracers with field measurements						
Pyridine	348	60-530	35.8	22.4	0.83	1.50
Pyrrole	373	230-460	33.4	35.3	0.88	0.97
3,4-Picoline	312	264-350	40.0	41.3	0.74	0.60
3-Ethenylpyridine	422	84-660	29.5	36.3	1.00	1.00
Nicotine	1274	396-3070	9.8	14.4	3.02	1.80
Myosmine	122	83-160	102.6	169.3	0.29	0.20
PM _{2.5}	12471	8100-17000	1.0	1.0	29.5	36.3
Volatile organic compounds						
Acetaldehyde	2292	2042-2496	5.4		5.4	
Acetonitrile	952	858-1069	13.1		2.3	
Acrolein	363	284-404	34.4		0.9	
Acrylonitrile	170	99-250	73.4		0.4	
Benzene	431	263-590	28.9		1.0	
1,3-Butadiene	279	157-400	44.6		0.7	
2-Butanone	323	166-540	38.6		0.8	
Cresol	109	62-148	114.1		0.3	
Ethylbenzene	131	101-170	95.3		0.3	
Formaldehyde	1101	243-1333	11.3		2.6	
Isoprene	2400	1990-2810	5.2		5.7	
Methylnaphthalene	51	41-61	244.5		0.1	
Naphthalene	45	34-55	280.3		0.1	
Phenol	157	26-360	79.3		0.4	
Styrene	160	141-210	78.1		0.4	
1,2,4-Trimethylbenzene	56	25-74	221.3		0.1	
Toluene	777	364-1270	16.0		1.8	
Xylene	400	135-571	31.2		0.9	
N-Nitrosodimethylamine	0.57	0.34-0.79	22073		0.0013	
N-Nitrosopyrrolidine	0.10	0.07-0.14	119918		0.00025	

^a average of emission factors (EFs) reported by.¹⁻¹⁰

^b range of EFs indicates the range of means of EFs reported by different studies, except N-Nitrosodimethylamine and N-Nitrosopyrrolidine, for which ranges represent 95% confidence intervals reported in the single study by⁷;

^c ratios of concentration of PM_{2.5}(C_{PM}) to concentration of volatile organic compound (C_{VOC}) were derived from simple linear regression analysis of 186 side-by-side 2-hour measurements of PM_{2.5} and SHS-VOC tracers;

^d ratios of concentration of volatile organic compound (C_{VOC}) and concentration of 3-Ethenylpyridine (3EP, C_{3EP}) were from simple linear regression analysis of 186 valid side-by-side 2-hour measurements of 3EP and other SHS tracers listed on the table.

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