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 $18 \times 10^{-6}$ (95% CI 13 to 23×$10^{-6}$) for patrons visiting only designated non-smoking sections, $80 \times 10^{-6}$ (95% CI 66 to 95×$10^{-6}$) for patrons visiting only smoking venues/sections and $802 \times 10^{-6}$ (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×$10^{-6}$ 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sometimes succeed in overturning existing smoking bans; for example, by July 2012 a total of 16 US municipalities had repealed, weakened, or postponed their SFE regulations due to such efforts.4 Patronising restaurants and bars may be a predominant source of SHS exposure for those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 assessments6–9 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 field measurements of SHS constituents, customers and workers at 3 different days of the week (lunch, dinner and evening) on 4

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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 PM2.5 and sample multiple gas phase SHS tracers, including nicotine, 3-ethenylpyridine, pyridine, pyrrole, picoline, 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 Lowrey developed a model to predict the risk of lung cancer death (LCD) due to SHS exposure using PM2.5 as a tracer (SHS-PM2.5). 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-PM2.5 for 1 year and the LER of LCD can be estimated by equation 1:

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

(1)

Where daily dose is the exposure dose of SHS-PM2.5 in mg/day; years is the number of years exposed to SHS; \(C_{\text{SHS-PM}}\) is the average concentration of SHS-PM2.5 in mg/m^3 during the period of exposure; BR is the breathing rate in m^3/h; time is the average hours per day exposed to SHS; \(f\) is the days per week exposed divided by 7 days.

The SHS-PM2.5 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 quasystematic 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/(Venue selection probability \times visit selection probability) (weight 1). The SE was computed by the Complex Samples module using a Taylor linearisation formula 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-PM2.5 level to which patrons in designated non-smoking section were exposed was estimated by multiplying the SHS-PM2.5 level in smoking sections by 0.525 (the ratio of the average non-smoking-section SHS-PM2.5 level to the average smoking-section level according to simultaneous side-by-side measurements during 16 visits), then weighted by weight 1 and the number of patrons in non-smoking sections (weight 3). To estimate the average SHS-PM2.5 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 m^3/h for servers at a moderate activity level and 1.0 m^3/h for patrons at a light activity level. 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 time 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 EPA or the equivalent cancer UR reported by the California EPA. URE and UR both describe the excess cancer risk associated with a daily inhalation exposure to 1 μg/m^3 of a given chemical for a lifetime of 70 years, assuming 20 m^3/day of inhalation. The LER of cancers can be estimated by equation 2:

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

Where \(C_{\text{SHS-VOC}}\) is the daily average concentration of a SHS-VOC during a 70 year lifetime, \(\mu g/m^3\); URE is the cancer UR reported by the US EPA, (μg/m^3)^{-1}; UR is the cancer UR reported by the California EPA, (μg/m^3)^{-1}, years is the number of years exposed to SHS; \(C_{\text{SHS-PM}}\) is the average concentration of SHS-PM2.5 during peak patronage time, \(\mu g/m^3\); \(\text{EF}_{\text{SHS-VOC}}\) and \(\text{EF}_{\text{SHS-PM}}\) are the average emission factors of SHS-VOC and SHS-PM2.5 from the literature, respectively, μg/cigarette; F is the adjustment factor, which is (4 h/day×1.6 m^3/h/(20 m^3/day)×5 day/7 day×45 years/70 years) for servers and (1.4 h/day×1.0 m^3/h/(20 m^3/day)×1 day/7 day×60 years/70 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-PM2.5 was most intensively monitored, and the ratios of simultaneous measurements of PM2.5 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-PM2.5 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 worldwide and in the US. 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\textsuperscript{1,23} and the attributable risk of death/case can be estimated by equation 4:10

\[ AB_{\text{SHS}} = B_{\text{ns}} \times PAF_{\text{SHS}} \]

\[ B_{\text{ns}} = (B - AB_{\text{sm}}) - (B - AB_{\text{sm}}) \times p_{\text{sm}} = (B - AB_{\text{sm}}) \times (1 - p_{\text{sm}}) \]

\[ PAF_{\text{SHS}} = p_{\text{SHS}}(RR_{\text{SHS}} - 1) \left/ \frac{p_{\text{SHS}}(RR_{\text{SHS}} - 1) + 1} \right] \]

\[ AERSHS = ABSHS / P_{\text{risk}} \]

\[ LERSHS = AARSHS \times \text{Years} \]

Where B is the total number of deaths/cases per year among the whole population; \( B_{\text{ns}} \) is the number of deaths/cases per year among non-smokers; \( AB_{\text{sm}} \) is the attributable burden of a disease in number of deaths/cases per year among smokers due to smoking; \( AB_{\text{SHS}} \) is the attributable burden of a disease in number of deaths/cases per year among non-smokers due to SHS exposure; \( PAF_{\text{SHS}} \) is the PAF of the disease burden due to SHS exposure among non-smokers; \( p_{\text{sm}} \) is the prevalence of current smoking; \( p_{\text{SHS}} \) is the prevalence of SHS exposure; \( RR_{\text{SHS}} \) is the relative risk of a disease due to SHS exposure among non-smokers; \( AERSHS \) is the annual excess risk of death/case due to SHS exposure among non-smokers; \( P_{\text{risk}} \) is the population at risk, that is, current non-smokers aged 35 years or older; \( LERSHS \) 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 exposed during their visits to smoking venues/sections was 134 \( \mu \)g/m\textsuperscript{3} (SE 12 \( \mu \)g/m\textsuperscript{3}), the corresponding average for patrons visiting non-smoking sections was 30 \( \mu \)g/m\textsuperscript{3} (SE 4 \( \mu \)g/m\textsuperscript{3}) and the weighted average for servers working in smoking-permitted venues was 78 \( \mu \)g/m\textsuperscript{3} (SE 7 \( \mu \)g/m\textsuperscript{3}).

Based on the risk model developed by Repace and Lowrey,\textsuperscript{16} this exposure corresponds to a LER of LCD 18 \( \times 10^{-6} \) (95% CI 13 to 23 \( \times 10^{-6} \)) for patrons visiting only designated non-smoking sections and 80 \( \times 10^{-6} \) (95% CI 66 to 95 \( \times 10^{-6} \)) for patrons visiting only smoking venues/sections for 1.4 h per week in 70 years and 802 \( \times 10^{-6} \) (95% CI 658 to 936 \( \times 10^{-6} \)) 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 SHS-PM\textsubscript{2.5} is 48 \( \times 10^{-6} \) for servers, 4.8 \( \times 10^{-6} \) for patrons visiting only smoking venues/sections and 1.1 \( \times 10^{-6} \) for patrons visiting only designated non-smoking sections; most of these cancers risks were non-lung cancer risks (table 2).

### Table 1

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

The 95% CIs presented here indicate only the variance of the weighted SHS-PM\textsubscript{2.5} levels during peak patronage times; uncertainties from other sources are not integrated. SHS, secondhand smoke; PM\textsubscript{2.5}, fine particulate matter.
Table 2: Servers’ and patrons’ cancer risk due to exposure to nine SHS-VOCs in Minnesota restaurants and bars

<table>
<thead>
<tr>
<th>SHS-VOC</th>
<th>URE/UR* (10^{-6})</th>
<th>Eff (µg/cigarette)</th>
<th>Servers Level1L (µg/m^3)</th>
<th>LER§ (10^{-6})</th>
<th>Patrons, smoking venues/sections Level1L (µg/m^3)</th>
<th>LER§ (10^{-6})</th>
<th>Patrons, non-smoking sections Level1L (µg/m^3)</th>
<th>LER§ (10^{-6})</th>
</tr>
</thead>
<tbody>
<tr>
<td>PM2.5</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acetaldehyde</td>
<td>2.20</td>
<td>2292</td>
<td>11.5</td>
<td>1.1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acrylonitrile</td>
<td>68.0</td>
<td>170</td>
<td>0.16</td>
<td>0.01</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Benzene</td>
<td>7.80</td>
<td>431</td>
<td>0.40</td>
<td>0.04</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1,3-Butadiene</td>
<td>30.0</td>
<td>279</td>
<td>0.26</td>
<td>0.02</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ethylbenzene</td>
<td>2.50</td>
<td>131</td>
<td>0.12</td>
<td>0.01</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Formaldehyde</td>
<td>13.0</td>
<td>1101</td>
<td>1.01</td>
<td>0.09</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Naphthalene</td>
<td>34.0</td>
<td>45</td>
<td>0.04</td>
<td>0.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>N-Nitrosodimethylamine</td>
<td>14000</td>
<td>0.57</td>
<td>0.00052</td>
<td>0.7</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>N-Nitrosopyrrolidine</td>
<td>600</td>
<td>0.10</td>
<td>0.00010</td>
<td>0.001</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total risk of all cancers</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total risk of non-lung cancers</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td></td>
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</tr>
</tbody>
</table>

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

†Average of Effs 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.

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; PM2.5, 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 10 510×10^{-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 4830 to 1290×10^{-6}) for LCD and 12 530×10^{-6} (95% CI 8430 to 16 620×10^{-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 papers6-8 used a single dose-response model published by Repace and Lowrey7 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 employers14 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.18 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...
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 estimated in this study is likely to overestimate the risks for only three of the multiple diseases caused by SHS exposure were evaluated, while other health outcomes, for example, new asthma cases attributed to SHS exposure for non-smoking servers may be overestimated, however, the individual lifetime 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 the general adult population from 1988–1994. 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 SBS-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 3: Attributed death and lifetime attributable risk of LCD and IHD death due to SHS exposure in Minnesota and US, 2004* |
|-----------------------------|-----------------------------|-----------------------------|
|                            | Minnesotat                  | 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$^6$ | 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, 235.3) | 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 estimated with the CI reported in this study re

†Data obtained from the CDC Smoking-Attributable Mortality, Morbidity, and Economic Costs (SAMMECT) website.‡‡Data obtained from CDC Morbidity and Mortality Weekly Report (MMWR).‡§Data from CDC MMWR, which was defined as the percentage of non-smoking population with serum cotinine ≥ 0.05 ng/ml.¶¶Data from the 1992 to 1994 National Human Activity Pattern Survey (NHAPS) for the USA.††Relative risks due to SHS exposure from the meta-analysis in the Surgeon General’s Report.**††Attributable burden; IHD, ischaemic heart disease; LCD, lung cancer death; LER, lifetime excess risk; PAF, population attributable fraction; RR, Relative risk; SHS, secondhand smoke.
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*

<table>
<thead>
<tr>
<th></th>
<th>Minnesota</th>
<th>USA†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of restaurant and bar servers employed, 2004‡§</td>
<td>63300</td>
<td>2700590</td>
</tr>
<tr>
<td>Never smoking rate, 2004, %§§</td>
<td>52.5 (49.2, 55.8)</td>
<td>57.6</td>
</tr>
<tr>
<td>Prevalence of current asthma among never smoking restaurant and bar servers, %¶¶</td>
<td>3.8 (3.0, 4.6)</td>
<td>3.8 (3.0, 4.6)</td>
</tr>
<tr>
<td>New asthma cases among never smoking restaurant and bar servers (Bns), cases/year**</td>
<td>126 (104, 148)</td>
<td>5911 (4861, 6961)</td>
</tr>
</tbody>
</table>
| Percentage of population covered by smoke-free restaurants and/or bars (1-Pbars), %†† | 38.1 (35.2, 41.0) | Restaurants: 74.5  
Bars: 63.7 |
| Relative risk of asthma initiation due to SHS exposure (RRbars)‡‡ | 2.16 (1.26, 3.72) | 2.16 (1.26, 3.72) |
| Population at risk, never smoking restaurant and bar servers (Pbars) | 33233 (31558, 34907) | 1555540 |
| PAF of new asthma cases among never smoking restaurant and bar servers due to SHS exposure (PAFBars), % | 41.8 (16.0, 67.6) | 24.0 (4.7, 43.3) |
| New asthma cases attributed to SHS exposure (ARbars), cases/year | 53 (25, 80) | 1420 (449, 2390) |
| Annual attributable risk of asthma initiation due to SHS exposure (AARbars)‡† | 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 (LERbars), % | 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; PAFbars, AARbars and LERbars 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.43
§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://www.who.int/globalattribution/2014;23:332–338. doi:10.1136/tobaccocontrol-2012-050831
| Contributors | RL, MGA and SKH made substantial contributions to conception and design of the study; DLB, LAG and MJH made substantial contributions to acquisition of data; RL, MJH and MGA were involved in analysing the data and drafting the manuscript; SKH made substantial contributions to interpretation of data and significant revision of the draft for important intellectual content; all authors gave final approval of the version to be published. |
REFERENCES


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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