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Risks for Heart Disease and Lung Cancer from Passive Smoking by Workers in the Catering Industry

Anthony J. Hedley,* Sarah M. McGhee,*,[1] James L. Repace,† Lai-Chin Wong,*
Marcus Y. S. Yu,‡,[2] Tze-Wai Wong,§ and Tai-Hing Lam*

*Department of Community Medicine, University of Hong Kong, Pokfulam, Hong Kong, China; †Repace Associates Inc., Bowie, Maryland 20720; ‡Hong Kong Council on Smoking and Health, Hong Kong, China; and §Department of Family and Community Medicine, Chinese University of Hong Kong, Prince of Wales Hospital, Shatin, Hong Kong, China

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Workers in the catering industry are at greater risk of exposure to secondhand smoke (SHS) when smoke-free workplace policies are not in force. We determined the exposure of catering workers to SHS in Hong Kong and their risk of death from heart disease and lung cancer. Nonsmoking catering workers were provided with screening at their workplaces and at a central clinic. Participants reported workplace, home, and leisure time exposure to SHS. Urinary cotinine was estimated by enzyme immunoassay. Catering facilities were classified into three types: nonsmoking, partially restricted smoking (with nonsmoking areas), and unrestricted smoking. Mean urinary cotinine levels ranged from 3.3 ng/ml in a control group of 16 university staff through 6.4 ng/ml (nonsmoking), 6.1 ng/ml (partially restricted), and 15.9 ng/ml (unrestricted smoking) in 104 workers who had no exposures outside of work. Workers in nonsmoking facilities had exposures to other smoking staff. We modeled workers' mortality risks using average cotinine levels, estimates of workplace respirable particulates, risk data for cancer and heart disease from cohort studies, and national (US) and regional (Hong Kong) mortality for heart disease and lung cancer. We estimated that deaths in the Hong Kong catering workforce of 200,000 occur at the rate of 150 per year for a 40-year working-lifetime exposure to SHS. When compared with the current outdoor air quality standards for particulates in Hong Kong, 30% of workers exceeded the 24-h and 98% exceeded the annual air quality objectives due to workplace SHS exposures.

Key Words: **secondhand smoke; passive smoking; urinary cotinine; heart disease; lung cancer; catering workers; Hong Kong.**

INTRODUCTION

The United States (U.S.) Centers for Disease Control and Prevention (CDC) estimated during 1988–1991 that 88% of nonsmokers were exposed to secondhand smoke (SHS) based on the detection in serum of the nicotine metabolite cotinine (Pirkle *et al.*, 1996). Emmons *et al.* (1992) identified the workplace as responsible for ~50% of SHS exposures. This evidence and the necessity to protect workers have led to legislation designed to strengthen smoke-free policies in the United States. These policies appear to have been successful to some extent. Wortley *et al.* (2002) reported that workplace exposures of nonsmokers declined between the late 1980s and the early 1990s, but the nonsmoking workers with the highest exposures were waiters. To protect catering workers from SHS exposures, smoke-free policies in restaurants and bars have now been widely introduced in the US (American Nonsmokers' Rights Foundation, 2004) and Europe has begun to follow suit (Howell, 2004).

In Asia, on the other hand, progress in the implementation of smoke-free workplaces and public places has been slow (Lam and Hedley, 1999; McGhee *et al.*, 2002) and as in other countries, smoke-free policies have been opposed by both the catering and tobacco industries (Dearlove *et al.*, 2002). At present most caterers are not required by law to provide smoke-free areas and the Hong Kong SAR government has recently published new legislative proposals to ban smoking in all workplaces (Health, Welfare and Food Bureau, Hong Kong Government, 2005). Although surveys show a high prevalence of perceived exposure among all workers in Hong Kong (Census and Statistics Department, 2003; McGhee *et al.*, 2002), arguments that actual exposures are low and are eliminated by ventilation (Drope *et al.*, 2004) are difficult to refute without air quality or dosimetry measurements. Repace (2004) found that after a workplace smoking ban, area measurements of SHS fine particles and polyaromatic hydrocarbon carcinogens decreased by 90%. Dosimetry measurements complement such studies by directly measuring SHS

[1] To whom correspondence should be addressed at Department of Community Medicine, University of Hong Kong, 5/F, 21 Sassoon Road, Pokfulam, Hong Kong SAR, China. Fax: 852-2855–9528. Email: commed@hkucc.hku.hk.

[2] Present address: Hong Kong Applied Science and Technology Research Institute Company Ltd., 18/F, Tower 6, Gateway, 9 Canton Road, Kowloon, Hong Kong SAR, China.

biomarkers, which incorporate proximity effects and respiration rates, which cannot be assessed by area monitors. Measurements of urinary cotinine are noninvasive and objective and have been found to be a valid quantitative predictor of SHS exposure in epidemiological studies (Benowitz, 1996; Jarvis *et al.*, 1984), with significant association between the levels of urinary cotinine and increasing self-reported exposure to secondhand tobacco smoke (Cummings *et al.*, 1990; Vineis *et al.*, 2005). The detection of raised levels of cotinine in hospitality workers has been reported from several countries including Canada (Dimich-Ward *et al.*, 1997), United States (Trout *et al.*, 1998; Maskarinec *et al.*, 2000), Finland (Johnsson *et al.*, 2003), and New Zealand (Bates *et al.*, 2002). These have often been based on small samples and the levels of cotinine have not been linked to estimates of disease risk. Tulunay *et al.* (2005) identified increased carcinogen levels in restaurant and bar workers and there is now substantive evidence of the effect of SHS on the cardiovascular system, including platelet and endothelial cell function (Barnoya and Glantz, 2005). The objectives of this study were to identify exposures to second-hand tobacco smoke in nonsmoking catering workers in Hong Kong, using personal histories and measurement of urinary cotinine, and to use pharmacokinetic models of relationships between cotinine, nicotine, and SHS respirable suspended particulates, coupled with exposure- and dose-response models, to estimate the working lifetime risks of fatal heart disease and lung cancer.

SUBJECTS AND METHODS

Subjects

The assessment of exposure of catering workers to SHS was conducted as an outreach activity of a Smoking Cessation Health Center, based in a hospital outpatient clinic and operated by the Hong Kong Council on Smoking and Health (Abdullah *et al.*, 2004). The center provided advice, counseling, treatment for tobacco dependency, and information about protection from passive smoking free of charge. The information reported here on personal exposure histories and urinary cotinine levels was gathered between February 2000 and May 2001.

There were two sampling procedures. The first was a stratified sample of workplaces chosen to represent the three types of catering facilities in Hong Kong, nonsmoking, unrestricted smoking, and partially restricted smoking (*i.e.*, with a nonsmoking area). After agreement with the catering facility's manager, a team from the Smoking Cessation Health Center went to the venue and carried out the screening. The second method was a general invitation, which was advertised in newspapers and by leaflets in catering establishments, offering a screening service to any nonsmoking catering worker. Through this method, 184 catering workers were recruited of whom 14 were smokers; 151 were nonsmokers (77 males; 74 females), who provided complete information; 53.6% of these subjects were screened in their workplace. These workers were employed in restaurants and bars in both private and public facilities representing a spread of different types and sizes of establishments serving either Western-style food or Chinese food (Table 1). Although the service was offered to nonsmoking workers, any smoker who wished to participate was accepted for testing.

A group of 16 control subjects were recruited as a convenient sample from those associated with the center and included physicians, nurses, and university public health researchers who were nonsmokers who worked in a smoke-free workplace, lived in a smoke-free home, and usually avoided smoky environments.

TABLE 1
Number (%) of Nonsmoking Workers by Type of Catering Facility

Type of facility	*n*	(%)	Males	Females
Nonsmoking restaurants	24	(14.1)		
Fast-food	22		3	19
Western/Eastern	1		1	
Canteen	1		1	
Smoking restaurants	146			
Chinese restaurants	70	(41.2)	36	34
Cha Charn Ting[a]	31	(18.2)	14	17
Club/canteen/cafeteria	31	(18.2)	21	10
Western/Eastern	8	(4.7)	6	2
Fast-food shop	6	(3.5)	1	5
Total	170		83	97

[a]Chinese tea shop.

A full protocol for this study, including recruitment of subjects, method of obtaining consent, methods of investigations, and the publication of this report, has been approved by the Institutional Review Board of the University of Hong Kong. The purposes of the tests were explained to each of the subjects who requested the assessment, and all participants were provided with a report and interpretation of their own urinary cotinine results.

Exposure Assessment

Self-reported exposure to passive smoking. Workers and controls completed a standard interview with demographic information and data on their past exposure to secondhand smoke, including workplace, home, and leisure exposures and their past smoking history. The questionnaire was also designed to capture information about the characteristics of the respondent's workplace with respect to passive smoking. In particular we obtained details of their job, smoking restrictions, indoor ventilation and duration of shift-work.

Expired air carbon monoxide. Carbon monoxide (CO) measurements were taken to identify any smokers. Middleton and Morice (2000), using a Bedfont Smokerlyzer (Bedfont Scientific Ltd, Rochester, England), suggested a cut-off of 6 ppm CO in expired air for classification as a nonsmoker. In a previous occupational health study (McGhee *et al.*, 2002) we found that none of the workers who claimed to be nonsmokers had an expired air CO level greater than 9 ppm.

Cotinine measurement. A 50-ml sample of urine was collected from each participant in a sterile plastic container and transported to a central laboratory in an ice box and frozen at −80°C within 4 h. Nicotine undergoes metabolic breakdown in the liver into several compounds, including cotinine which is the best available biomarker of SHS exposure (Benowitz, 1996; Repace and Lowrey, 1993; Repace *et al.*, 1998) and can be measured in blood, saliva, and urine. The urinary cotinine levels of all subjects in this survey were measured by the MetLife Laboratory in New York City (Dr. N. J. Haley) using an ELISA assay (EIA) with a 93% specificity for cotinine (gas chromatography/mass spectrometry [GC/MS]) as the standard) and a 10% cross-reactivity with 3-hydroxycotinine (3-HC) (Niedbala *et al.*, 2002). Cross-reactivity with four other structurally related nicotine metabolites was negligible. The limit of detection for cotinine in this study was 0.1 ng/ml, and the limit of quantification was taken as 1 ng/ml. Benowitz (1996) reported that the ratio of 3HC to cotinine measured in 12 subjects was 2.94:1 (39.1–13.3%). Assuming that this ratio holds generally, cotinine in our study may be overestimated relative to GC/MS by a factor of (1[0.93] + 2.94[0.1]) = 22%, and underestimated relative to radioimmunoassay (RIA) values by about the same amount (Watts *et al.*, 1990).

Validation of exposure measurements. The information obtained was validated by direct or indirect measures, as appropriate. For the self-reported information on types of venue, the investigators were able to directly observe the sites included in the stratified sample and record the smoking arrangements. For self-reported smoking status, all subjects were tested for expired breath carbon monoxide. None of those who declared themselves to be nonsmokers had raised levels of CO.

The objectively measured cotinine levels provided evidence to support the workers' declarations of exposures as follows: (1) for workplace restrictions (or lack of them) on smoking and smoking by non-customers, we observed a gradient in median cotinine levels between the different types of venue with respect to whether smoking was allowed; (2) for declared time elapsed from last shift in the different types of facilities, we observed lower concentrations of cotinine in those who reported a longer time elapsed since their shift.

Analyses. Subjects from both samples were pooled for analysis. Urinary cotinine levels were analyzed by main and sub-groups as defined by their worker or control status, workplace type, and reported exposures to tobacco smoke from any source. The classification of subjects was done *a priori* using the terms "control" or "catering worker" and the place of work as "non-smoking" or "smoking" catering facilities, further categorized as "unrestricted smoking" or "partially restricted smoking" with designated smoke-free areas. The findings were then further analyzed by subgroups, including "non-waiter" (*e.g.*, accounts clerks, housekeepers, chefs, others), and "waiter" (anyone serving at tables or a bar), by time elapsed from last shift; by declarations of "other exposures" including exposure during rest times, home, and leisure activities; and by the presence of air-conditioning at place of work.

All analysis was done using STATA version 6. Cotinine levels for the groups are presented as means and standard deviations and also as box-plots with medians, interquartile ranges (IQ), values up to 1.5 times the IQ, and outliers. The Kruskal-Wallis rank test for equality of populations was used to compare cotinine levels between pre-defined groups. The significance of trends was estimated using Cuzick's nonparametric test for trend across ordered groups, ranked as controls or workers and type of restaurant.

Risk assessment. The risks of heart disease and lung cancer in this sample were estimated from pharmacokinetic risk models (Repace and Lowrey, 1993; Repace *et al.*, 1998) that allow cotinine levels to be related to ambient nicotine (N) and respirable suspended particulate (R) concentrations and to the risk of lung and heart disease in passive smokers. The relationship of steady-state SHS nicotine concentration, *N*, in micrograms per cubic meter ($\mu g/m^3$) to daily urinary cotinine concentration, *U,* in nanograms of cotinine per milliliter of urine (ng/ml) (Repace and Lowrey, 1993) was calculated as follows:

$$N = (U\delta_T V_u)/(1000\phi\alpha\delta_R \rho H), \quad (1)$$

where δ_T is the total cotinine clearance in ml/min, V_u is the daily urine flow in ml/day, 1000 is the conversion from nanograms to micrograms, ϕ is the nicotine-to-cotinine conversion efficiency by the liver, α is the nicotine absorption efficiency by the lung, δ_R is the renal cotinine clearance in ml/min, ρ is the worker respiration rate during exposure in m^3/h, and H is the number of hours of exposure per day. Typical values for the parameters in equation 1 are:$\delta_T = 64$ ml/min, $V_u = 1300$ ml/day, $\phi = 0.78$, $\alpha = 0.71$, $\delta_R = 5.9$ ml/min, $\rho = 1$ m^3/h (Repace and Lowrey, 1993; Repace *et al.*, 1998), and the average work shift for the Hong Kong catering workers reported in this study was about H = 11 h per day. With these values, equation 1 yields the following equations for the estimated steady-state SHS-nicotine and SHS-respirable suspended particulates (RSP) concentrations during an 11-h work shift in a Hong Kong restaurant:

$$N = U(64)\,(1300)/[(1000)\,(0.78)\,(0.71)\,(5.9)\,(1)\,(11)] = 2.33\,\mathrm{U} \quad (2)$$

and

$$R = 10\,N = 23.3\,U, \quad (3)$$

where *U* is in ng/ml, and *N* and *R* are in $\mu g/m^3$. Thus, urinary cotinine can be used to estimate SHS-RSP exposure concentration ($\mu g/m^3$) over an 11-h work day using equation (3), where the nicotine and particulate concentrations are dependent on the conditions of smoking prevalence, occupancy, and air exchange discussed below.

Risks of heart disease and lung cancer mortality. In the risk assessment we assumed that the lung cancer exposure–response relationship is 5 lung cancer deaths per 100,000 person-years per milligram of exposure to SHS-RSP per day (Repace and Lowrey, 1985*a*). This assumption was derived from the lung cancer mortality rates found between cohorts of lifelong nonsmoking Seventh Day Adventists with very low exposures to SHS and a cohort with typical community exposures (Phillips *et al.*, 1980). The heart disease exposure–response relationship was estimated from the 10:1 ratio of passive-smoking–induced heart disease deaths (HDD) to passive-smoking lung cancer deaths (LCD) (Repace *et al.*, 1998), by averaging U.S. population estimates published by Repace and Lowry (1985b), Repace and Lowrey (1990), and the U.S. Environmental Protection Agency (1991) and HDD estimates by Wells (1994), Glantz and Parmley (1991), and Steenland (1992). This averaging was done on the conservative assumption that this ratio is constant with age, although nonsmokers' HDDs actually increase faster with age than LCDs (National Cancer Institute, 1997).

Using these models, Repace *et al.* (1998) associated an average serum cotinine of 0.4 ng/ml with a 40-year working lifetime (WLT_{40}) increase in mortality in the United States from lung cancer of 1 in 1000 person-years (PY), and 1 in 100 PY for heart disease, giving a combined total risk of 11 deaths per 1000 PY of exposure. Using a urine-to-serum cotinine ratio of 6.5, this dose–response relationship estimates that a urinary cotinine level of 2.6 ng/ml corresponds to a combined risk of 11 deaths in 1000 PY. Alternatively, a urinary cotinine level of 1 ng/ml corresponds to a lifetime risk of approximately four deaths per 1000 PY (Repace and Lowrey, 1993).

The previous model was developed based on U.S. mortality rates. In Hong Kong, the unadjusted mortality rate in 1998 from heart disease (ICD9 390–429) was 78 per 100,000 (Department of Health, 2000), compared with 268/100,000 in the United States (Centers for Disease Control/National Center for Health Statistics, 2000), whereas the mortality rate for lung cancer (ICD9 162) was slightly lower at 48 per 100,000 compared with 57 per 100,000 in the United States. Therefore, for Hong Kong, the final estimates of risk were scaled to reflect the Hong Kong rates.

Using this model, a health- based standard for passive smoking, based on SHS-R levels, was developed for the United States (Repace and Lowrey, 1985*b*). The *de minimis* or acceptable WLT_{40} risk level of 1 death per million nonsmokers at risk in the U.S. occurs at 2.6 picograms of cotinine per milliliter of urine (Repace *et al.*, 1998).

Validation of the model. The average exposure of the U.S. population was modeled using time-activity pattern studies, and the results were consistent with a national probability sample of serum cotinine data measured by the U.S. Centers for Disease Control and Prevention (Repace *et al.*, 2005). The model also predicted both the lung cancer mortality rate and risk ratio for the American Cancer Society cohort of passive smokers to within 5% (Repace and Lowrey, 1985*a*).

When the risk of a 40-year exposure is linearly scaled to a 20-year exposure, the heart disease risk corresponding to an average serum cotinine dose of 0.4 ng/ml becomes 5 per 1000. In comparison, in the British Regional Heart Study, Whincup *et al.* (2004), found that a serum cotinine of 0.4 ng/ml at enrollment in a cohort of 2105 nonsmoking men was associated with a 20-year risk (1980–2000) of 5.4 coronary heart disease events per 1000 person-years.

RESULTS

Controls

Thirteen of the 16 control subjects declared no known work or other exposures to passive smoking. This group had a mean

cotinine level of 3.3 ng/ml. The other three subjects with declared possible exposure outside work had a mean of 5.5 ng/ml (Table 2).

Smoking Workers

The mean urine cotinine in occasional smokers was 250.2 ng/ml (SD 298.6); the declared use of tobacco in this group was variable and very low in some subjects. For regular smokers, the mean urine cotinine was over 3589 ng/ml (SD 1441). All smokers were excluded from further analysis.

Nonsmoking Facilities

In facilities that did not permit customer smoking, a majority of workers (13/21; 62%) were exposed to other workers' SHS because of smoking at break times. Their mean cotinine levels ranged from 9.9 ng/ml to 14.0 ng/ml. Three workers with no exposures outside of work and who declared no exposures from other workers had a mean cotinine level of 6.4 ng/ml (Table 2).

Partially Restricted Smoking Facilities

These findings relate to any worker employed in a facility that permitted smoking but that had various forms of ''smoke-free'' areas or seating. Those workers with no exposure outside work and no exposures from other workers ($n = 6$) had a mean cotinine level of 6.1 ng/ml (Table 2). Workers with additional exposures to tobacco smoke from home, leisure venues, and other workers had higher mean levels ranging from 7.1 ng/ml in one subject associated with home and leisure exposure, to 14.3 ng/ml in workers with exposures from co-workers ($n = 50$) and 16.6 ng/ml in 21 workers with home, leisure, and other worker exposures ($n = 21$). For the upper quartile of this group, cotinine levels ranged from 18.6 ng/ml with no exposures from co-workers to 55 ng/ml in those with exposures from other staff.

TABLE 2
Urinary Cotinine Levels (ng/ml) in Nonsmoking Staff by Exposure to Secondhand Smoke at Work, Home, and Leisure Activities

		Exposure					
Subjects	*N*	Home/ leisure	Other staff	Mean	SD	Median	Range
Controls	13	No	No	3.3	3.5	2.6	0–11.2
	3	Yes	No	5.5	4.9	4.5	1.1–10.8
Total	16			3.7	3.7	2.7	0–11.2
Nonsmoking restaurants	3	No	No	6.4	6.6	2.7	2.6–14.0
	10	No	Yes	14.0	17.7	8.9	2.2–62.9
	5	Yes	No	20.3	11.9	19.6	3.9–34.1
	3	Yes	Yes	9.9	3.9	10.3	5.8–13.6
Total	21			13.8	14.0	10.3	2.2–62.9
Partially restricted smoking restaurants	6	No	No	6.1	6.4	4.2	1.5–18.6
	50	No	Yes	14.3	10.8	9.6	2.0–55.3
	1	Yes	No	7.1		7.1	
	21	Yes	Yes	16.6	17.2	12.0	1.0–75.4
Total	78			14.2	12.7	9.5	1.0–75.4
Unrestricted smoking restaurants	4	No	No	15.9	6.5	16.5	7.6–23.1
	34	No	Yes	28.7	33.9	17.3	0–129.4
	3	Yes	No	26.5	10.5	30.2	14.7–34.6
	11	Yes	Yes	20.0	21.9	10.4	0–62.3
Total	52			25.7	29.4	15.9	0–129.4

Unrestricted Smoking Facilities

In workers with no exposures outside of work, and no exposures from other workers ($n = 4$), the mean cotinine level was 15.9 ng/ml compared with 28.7 ng/ml in workers with non-customer workplace exposure ($n = 34$) (Table 2). For workers with home, leisure, and other worker exposures ($n = 14$), the mean cotinine ranged from 20.0 to 26.5 ng/ml. In the upper quartile of this group, the cotinine levels ranged from 23.1 ng/ml to 129.4 ng/ml.

Comparison Between Types of Catering Facility

The average cotinine levels found in the nonsmoking catering workers varied by type of catering facility (Fig. 1). There was a statistically significant difference between groups ($\chi^2 = 27.8$; $p = 0.0001$) (test for equality of populations) and a significant trend ($z = 4.98$; $p < 0.01$). The highest cotinine levels of 100 ng/ml or more were observed in unrestricted smoking facilities. However, the median levels were similar for all groups, and there was marked overlap of the interquartile ranges.

Waiters and Non-waiters

There was no significant difference in cotinine levels between waiters and non-waiters. The mean cotinine level for non-waiters in partially restricted smoking facilities was 13.9 ng/ml (SD 0.9), compared with 13.0 ng/ml (SD 12.6) for waiters. In the facilities with unrestricted smoking, the mean cotinine for non-waiter staff was 23.2 ng/ml (SD 16.8), compared with 26.9 ng/ml (SD 33.2) for waiters.

Variation by Place of Exposure, Time since Exposure and Gender

In the entire group of 151 workers, 104 workers with work exposures only had a mean cotinine level of 18.6 ng/ml (SD 22.6), compared with a slightly lower mean 17.0 ng/ml (SD 17.0) in the group as a whole. A total of 81 workers were screened during their working shift, and they had a higher mean cotinine level (22.1, SD 2.5) than those screened up to 12 h later (12.5, SD 15.2) or more than 12 h later (14.2, SD 20.7; p for trend 0.026). There was no significant difference in cotinine levels between male (mean 15.3, SD 16.1) and female (mean 16.4, SD 22.3) workers.

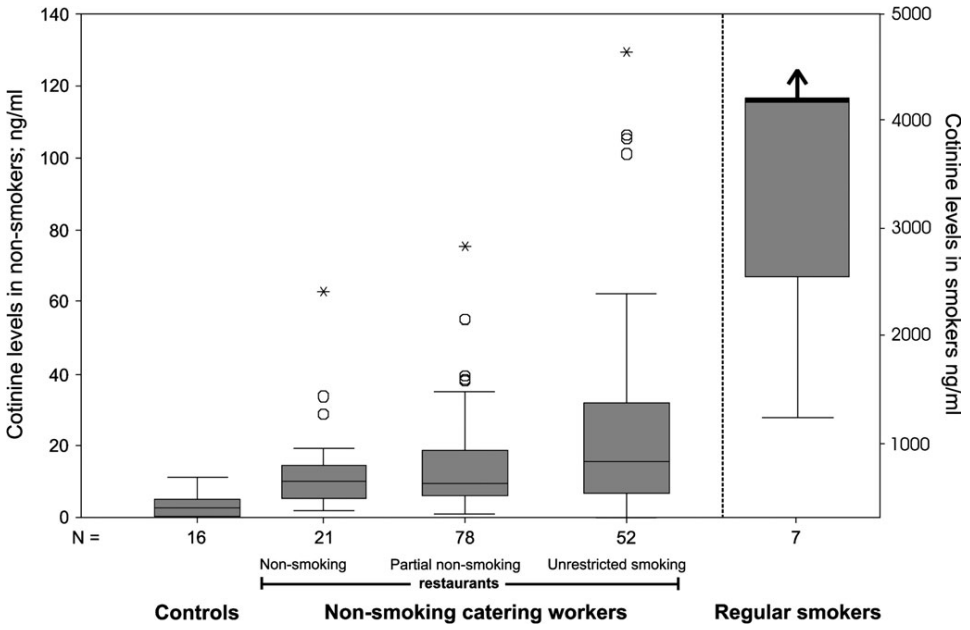

FIG. 1. Urine cotinine levels (ng/ml) in controls and nonsmoking catering workers by type of restaurant.

Ventilation and Cotinine Levels

Ninety three percent of workers stated that air-conditioning units operated in their workplace; their mean cotinine levels were higher (27.6 ng/ml) than those of workers in non-air-conditioned premises (14.3 ng/ml). However, there were only six small establishments without air-conditioning, and there was a wide range of values in both types of venue.

Workplace Respirable Particulates from Secondhand Smoke

In a log-probability plot of the estimated cumulative frequency distribution for workplace SHS-RSP exposure ($\mu g/m^3$) (Fig. 2), based upon the reported average work day of 11 h for the 104 nonsmoking workers who were exposed only at work, the 10th percentile of cumulative exposure was 88 $\mu g/m^3$, mean 429 $\mu g/m^3$ (SD 522, median 257 $\mu g/m^3$) and the 90th percentile 914 $\mu g/m^3$. These estimated SHS-RSP exposure concentrations are comparable to previously reported area measurements of SHS-RSP in hospitality industry workplaces, which ranged from about 100 to 1000 $\mu g/m^3$ (Repace, 2004; Repace and Lowrey, 1980; Travers *et al.*, 2004; U.S. EPA, 1992).

The estimated cumulative frequency distribution of SHS-RSP levels was averaged over 24 h and converted to an annual average, conservatively assuming 250 work days in a year. In fact, Hong Kong catering workers probably average 300 work days per year. When the 10th percentile of cumulative exposure is 77 $\mu g/m^3$, mean SHS-RSP 185 $\mu g/m^3$ (SD 164, median 131 $\mu g/m^3$) and 90th percentile 337 $\mu g/m^3$. When the Hong Kong SAR annual average PM_{10} level of 50.4 $\mu g/m^3$ is added, Figure 3, also shows the 24-h (180 $\mu g/m^3$) and annual (55 $\mu g/m^3$) air quality objectives (AQO) (Environmental Protection Department, 2000) (Fig. 3). An estimated 30% of workers exceeded the 24-h AQO and 98% exceeded the annual AQO. For the average worker, the annual levels of air pollution particulate exposures from tobacco smoke plus background levels are 185 $\mu g/m^3$, 3.7 times those from background exposures alone.

Cancer and Heart Disease Mortality

The estimated WLT_{40} combined risks from fatal heart disease and lung cancer for Hong Kong catering workers exposures to passive smoking were estimated from the dose–response relationship given earlier using both the U.S. and the Hong Kong mortality rates (Fig. 4). The mean WLT_{40} risk estimate based on U.S. mortality rates is 7.8% (SD 9.5%), 10th percentile 2%, median 4.7%, 90th percentile 17%, and the risk range for the top 5% of urinary cotinine levels 27–55%. In Hong Kong coronary heart disease death rates are lower than those in the United States by a factor of about 3. Using the US estimate for the lung cancer risk, but the lower heart disease risk, the WLT_{40} risk estimate for Hong Kong is 3% (SD 3.6%), 10th percentile 1%, median 1.7%, 90th percentile 6%, with a risk range for top 5% of 10–21%. These estimates of risk are well above the U.S. occupational health significant risk level of 1 in 1000 (Repace *et al.*, 1998).

The current population of catering workers in Hong Kong numbers around 200,000. For a 40-year working life time exposure in this population, the estimated average 3% risk translates into deaths occurring at the rate of 150 workers annually due to heart disease or lung cancer as a result of passive smoking at work.

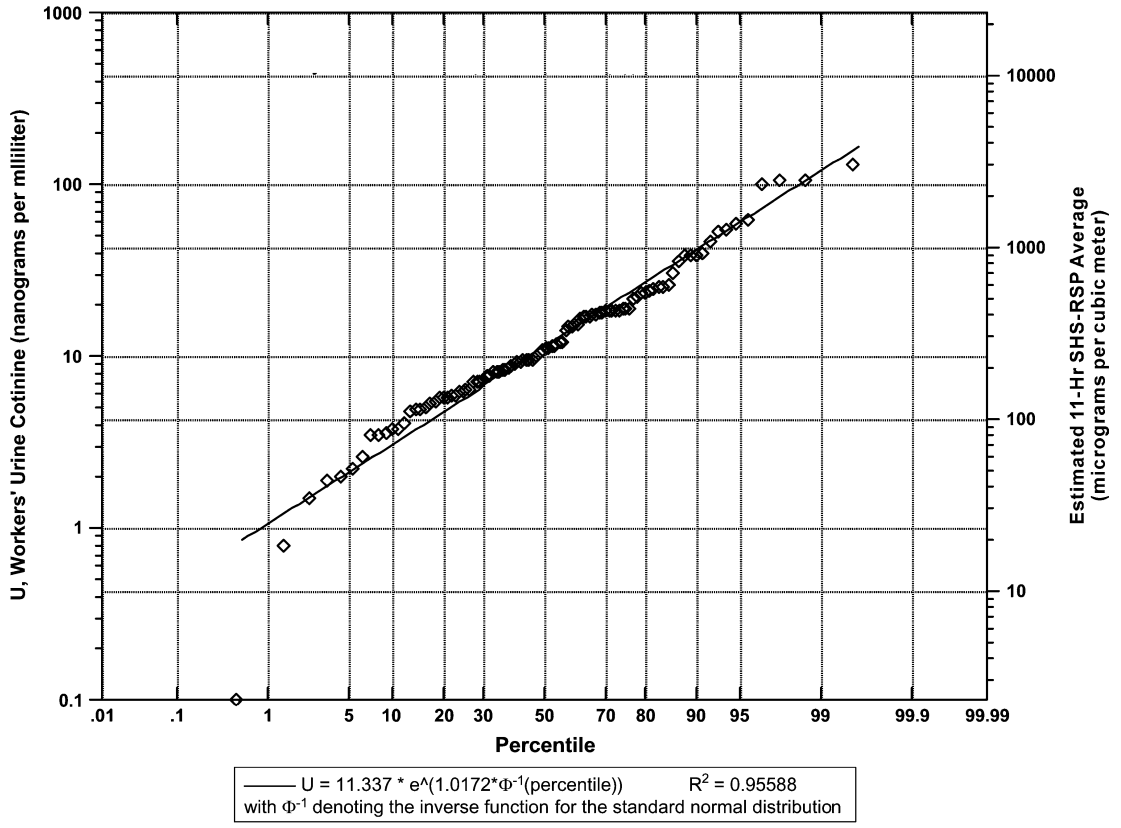

FIG. 2. Log-probability plot for urinary cotinine and estimated SHS-RSP exposure SHS-RSP = 23.15 U ($\rho = 1$ m^3/h; H = 11 h/day) for 104 Hong Kong restaurant workers exposed to secondhand tobacco smoke only at work.

DISCUSSION

The health effects of SHS exposures are largely unobservable events, and both environmental and epidemiological analyses are needed to quantify the risks and strengthen support for policy decision making. We have shown that the estimated SHS-RSP levels are comparable to the range of SHS-RSP measured in contemporary hospitality industry studies in the United States and thus are applicable to most indoor working environments in the catering industry. Even in settings with a relatively low prevalence of smoking, average levels of SHS-RSP will indicate very high levels of risk to catering workers who typically have long working shifts. The ubiquitous nature of secondhand smoke in poorly regulated environments is clearly indicated by our highly selected low-risk group of controls. In 13 subjects with no recognized exposures to tobacco smoke the urinary cotinine was 3.3 ng/ml. This is in good agreement with a study of 30 public health workers and their spouses in Boston, Massachusetts (Hyde *et al.*, in press). This indicates that SHS exposures may often be unnoticed, especially if the levels are lower or measures to mask the odor are used.

It was necessary for us to use stratified sampling of catering facilities to obtain sufficient respondents from nonsmoking venues and to recruit workers in recent contact with SHS. Pooling of the subjects for analysis is likely to have resulted in an underestimate of cotinine levels, because the pooled sample over represents the proportion of nonsmoking establishments and probably under estimates the proportion of unrestricted smoking restaurants. However, we used this as a conservative approach. Furthermore, the workers who came to the clinic were on "days-off" from work, so the time elapsed from exposure during shift work was variable and was associated with lower average cotinine levels than in the workers recruited on-site. This also results in a conservative estimate of cotinine levels.

The model we used for the estimation of the health impact of exposure was based on one developed in the United States, which we then extrapolated to Hong Kong using the difference in mortality rates between the two populations. Possible problems with extrapolation of the model are first, the fact that Hong Kong has high ambient outdoor concentrations of air pollutants, which raises indoor levels. While this will affect absolute levels of exposure to particulates, it should not affect the estimates of excess risks from SHS exposures. It is, however, possible that at high levels of exposure the dose–response relationship is non-linear. In this case our estimates

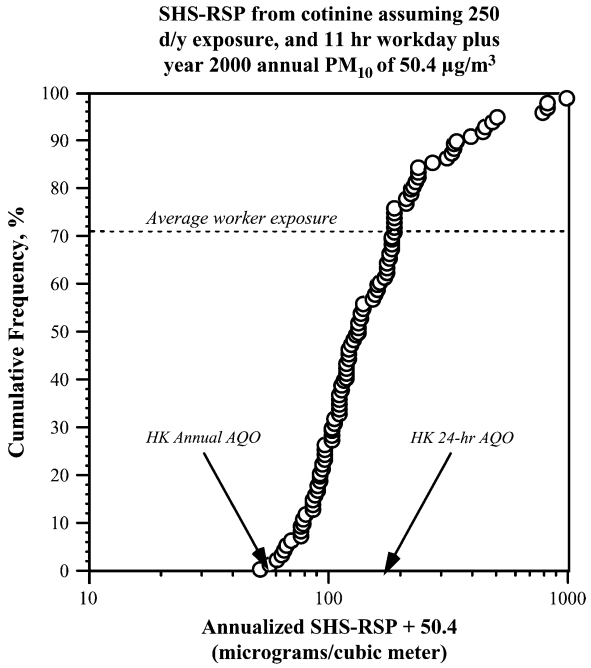

FIG. 3. Cumulative frequency distribution for 104 Hong Kong workers exposed to secondhand tobacco smoke only at work. Estimated 24-h average secondhand smoke respirable particulates levels assume an average 11-h work shift. Hong Kong air quality objectives (AQO) for RSP are shown for comparison. An estimated 30% of Hong Kong catering workers studied exceeded the 24-h AQO.

could be over estimated, but we do not think this is likely. Second, there are differences between the United States and Hong Kong in baseline risks for the commonest registered causes of death such as cancer and cardiovascular disease. However, we have taken this into account by using Hong Kong mortality rates in the model. Third, the maturity of the epidemic of smoking-related disease in Hong Kong is less than that of the United States, leading to different ratios between conditions such as lung cancer and heart disease. We have also taken this into account by using Hong Kong mortality rates for the specific diseases.

Our results show that partial smoking restrictions are of no value in significantly reducing exposures and risks to workers. The ineffectiveness of partial restrictions in shared indoor air spaces is demonstrated by the distribution of cotinine levels across groups of workers in different working environments. In our client population, workplace exposures accounted for most of their risk, as indicated by the number of staff with raised urine cotinine levels.

The proposals being advanced to reduce exposures through increasing ventilation and air cleaning can be evaluated using the output of this analysis, the mass-balance model for estimating SHS-RSP, and the small population *de minimis* risk level of 1 death per 100,000 workers per 40-year working lifetime. For the estimation of steady-state SHS-RSP, designated "R" in units of micrograms per cubic meter (µg/m^3), the equation is:

$$R = 220\, D_{hs}/C_v, \tag{4}$$

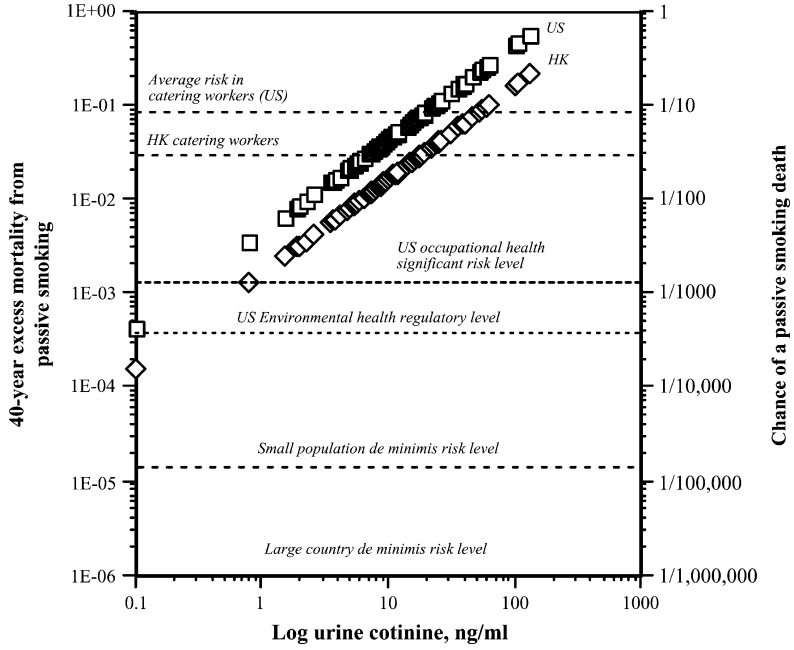

FIG. 4. Working lifetime combined risk from fatal heart disease and lung cancer based on Hong Kong exposure to secondhand tobacco smoke and both US and Hong Kong mortality rates.

where D_{hs} is the smoker density, in units of habitual smokers per hundred cubic meters (1 active smoker corresponds to three habitual smokers who smoke two cigarettes per hour each), and C_v is the restaurant air exchange rate in units of air changes per hour (ACH) (Repace, 2004). The Hong Kong Special Administrative Region (SAR) hospitality industry ventilation requirements for restaurants, pubs, bars, factory canteens, and dancing establishments are 17 m^3/h per person (m^3/h-P) or 4.5 l per second per person (L/s-P) for those who may be accommodated in the premises (Department of Justice, 1999). The corresponding seating capacity for restaurants and factory canteens is one person per 1.5m^2 or 67 persons per 100 m^2, and the estimated average air exchange rate per hour assuming a default 4-m-high ceiling is:

$$(67\mathrm{P}/100\,\mathrm{m}^2)\,(4\,\mathrm{m})\,(17\,\mathrm{m}^3/\mathrm{h}-\mathrm{P}) = (67/400)\,(16.2) = 2.8\ \mathrm{ACH}.$$

Holding D_{hs} constant, if the current average air exchange rate per hour (C_V) is 2.8 h^{-1}, corresponding to an outdoor air supply ventilation rate of 4.5 (L/s-P), and, if the risk to the typical worker is 3%, the air exchange rate per hour (C_x) to reduce the average risk to *de minimis* level is:

$$\mathrm{C_x} = [3\times10^{-2})/(1\times10^{-5})]\times[2.8\,\mathrm{h}^{-1}] = 8{,}400\,\mathrm{h}^{-1},$$

equivalent to 13,500 L/s-P. Ventilation measures cannot attain a level of *de minimis* risk without tornado-like levels of air flow (Repace, 2005). In the United Kingdom the Public Places Charter aims to reduce second-hand smoke exposures through increasing the nonsmoking area and ventilation, but Carrington *et al.* (2003) showed that the use of sophisticated ventilation systems did not have a significant effect on secondhand smoke marker concentrations in either smoking or nonsmoking areas.

In general, population samples with a history of exposure to passive smoking have strongly associated risks, with a dose–response relationship of cardiovascular and respiratory diseases and cancers. Whincup *et al.* (2004) demonstrated increased risks of coronary heart disease over 20 years using cotinine as an indicator of total exposure to secondhand smoke. A recent study (McGhee *et al.*, 2005) of mortality in nonsmokers associated with a history of living with a smoker 10 years before death found large excess risks for heart disease, stroke, chronic pulmonary disease, and cancers, and a dose–response relationship with the number of smokers at home.

The cardiovascular disease epidemics in the West and in Asia are at different stages of maturity. The age-specific mortality rates for coronary heart disease are much lower in Hong Kong than in the United States; however, ''all cardiovascular disease'' is the second most common registered cause of death. There are strong associations between particulate ambient air pollution and illness episodes and mortality from cardiovascular disease, including coronary heart disease and stroke, in Hong Kong (Wong CM *et al.*, 2002; Wong *et al.*, 2001; Wong TW *et al.*, 2002). For workers exposed to indoor pollution from second-hand smoke, there is a large additional risk. Workplaces that permit any smoking are likely to violate the 24-h AQO on a daily basis and increase the risk of fatal cardiopulmonary disease in the workforce.

Finally, there is new evidence that active smokers have additional respiratory health problems from passive smoking at work (Lam *et al.*, 2005).

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The authors certify that all research involving human subjects was done under full compliance with all government policies and the Helsinki Declaration.

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