

# **Preliminary Analysis of Dose, Exposure, and Risk for 104 Hong Kong Catering Workers Exposed to Second Hand Smoke (SHS) at Work Only**

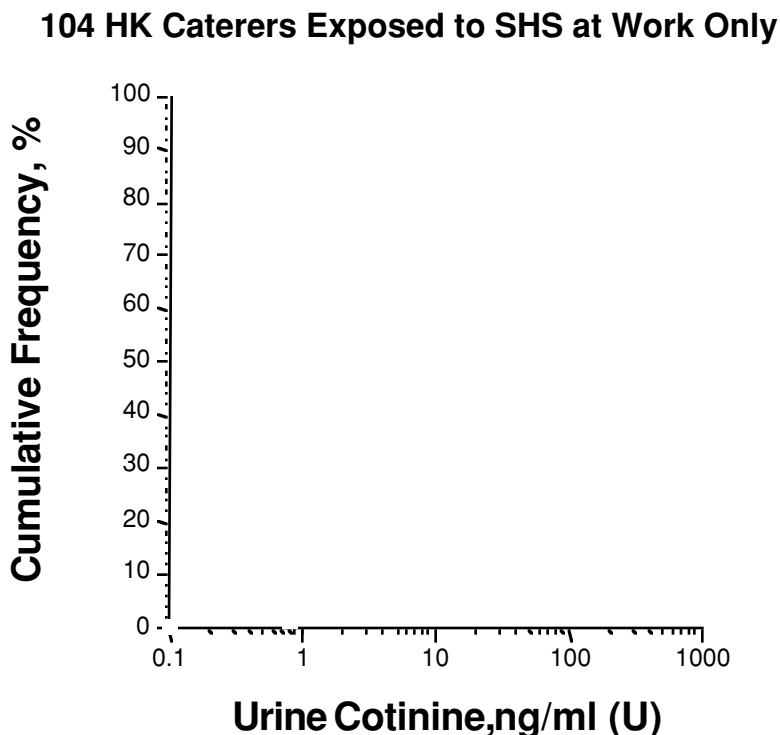
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## **Abstract**

**An estimated 150 Hong Kong Catering workers (3% of workers studied) die annually from passive-smoking induced coronary heart disease or lung cancer due to workplace passive smoking. This amounts to 6,000 deaths from a working lifetime exposure to second-hand smoke in 200,000 Hong Kong catering workers. These results were based on levels of urinary cotinine, a nicotine metabolite, found in 104 workers measured at MetLife Laboratories, USA and sampled and analyzed by the Hong Kong Council on Smoking and Health, University of Hong Kong, Chinese University of Hong Kong, and Repace Associates Inc, USA. Two thirds of the workers are estimated to be exposed to levels of respirable particulate air pollution on the job which exceed the Annual Hong Kong Air Quality Objective level of 55 micrograms per cubic meter, and one third exceeded the 24-hour AQO because of second-hand smoke exposure alone. Asthmatic workers are estimated to have their risk of an asthmatic attack doubled at the mean exposure level of the workers, and increased as much as ten-fold at the extreme.**

## Content

The Cumulative Frequency Distribution for Urine Cotinine for 104 Hong Kong catering workers shows the following results: 10th percentile, 3.7 ng/mL; median 11.1 ng/mL; mean 18.6 ng/L; sd 22.6; 90th percentile 39.1 ng/mL. Figure 1 gives the complete distribution.

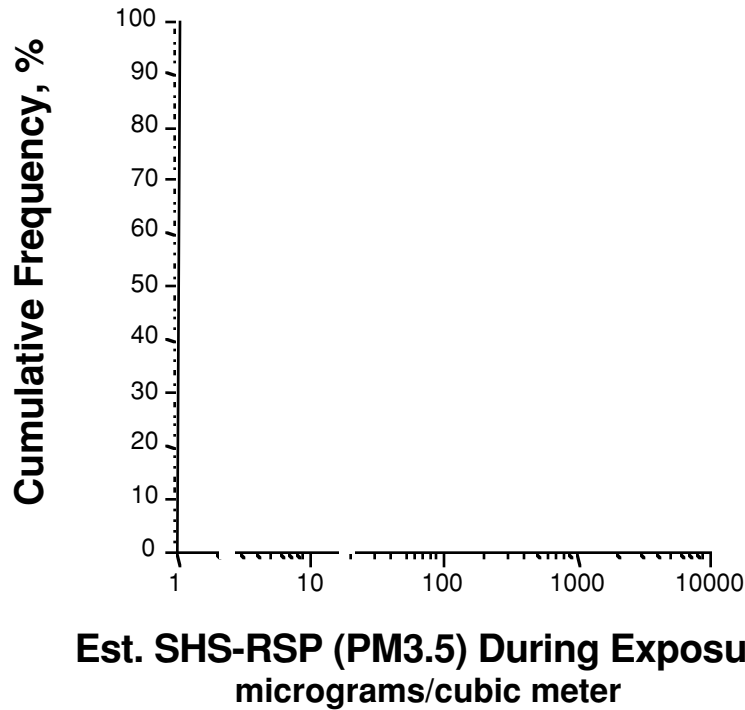


**Figure 1. Cumulative frequency distribution for urinary cotinine of 104 Hong Kong restaurant workers exposed to second-hand tobacco smoke only at work, April 2001.**

13 Hong Kong subjects with no work or other SHS exposures had a mean urinary cotinine of 3.3 ng/mL (SD 3.5). The average restaurant worker had an SHS exposure of (18.6 / 3.3) 5.6 times the controls. By comparison, a recent national sample taken in the U.S. showed that half of the population had undetectable levels of cotinine (CDC, 2001).

Mean SHS-RSP 429  $\mu\text{g}/\text{m}^3$ ; SD 522; median 257  $\mu\text{g}/\text{m}^3$ ;  
90th percentile 914  $\mu\text{g}/\text{m}^3$ ;

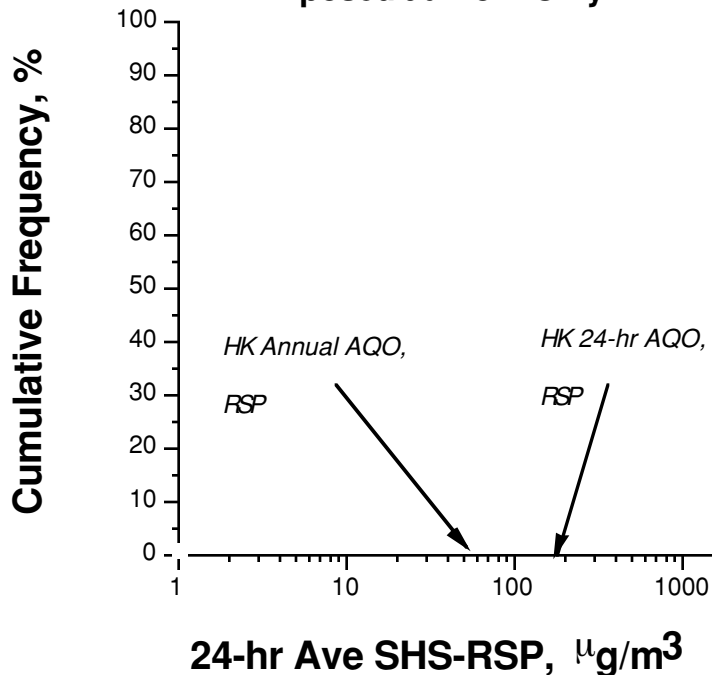
**104 HK Caterers, SHS-Exposed @ Work Only**



**Figure 2. Cumulative Frequency Distribution for 104 HK Workers Exposed to second-hand tobacco smoke only at work: Estimated SHS-RSP During Smoking: SHS-RSP = 23 U (see Figure 1).**

Mean ETS-RSP 197  $\mu\text{g}/\text{m}^3$ ; SD 239; median 118  $\mu\text{g}/\text{m}^3$ ;  
90th percentile 419  $\mu\text{g}/\text{m}^3$ ;

**104 Hong Kong Caterers, Second-hand Smoke (SHS)-Exposed at Work Only**



**Figure 3. Cumulative Frequency Distribution for 104 HK Workers exposed to second-hand smoke only at work: Estimated 24-hr Average SHS-RSP levels assume an average 11-hour workshift. Hong Kong Air Quality Objectives for RSP are shown for comparison.**

**Hong Kong Air Quality Objective (AQO) for respirable suspended particles (RSP) is 55 micrograms per cubic meter ( $\mu\text{g}/\text{m}^3$ ) annual average, and 180  $\mu\text{g}/\text{m}^3$  24-hr average. An estimated 1/3 of Hong Kong Catering Workers studied exceeded the 24-hr AQO. Assuming a 250 day work-year, 2/3 of Hong Kong Catering Workers studied exceeded the Hong Kong Annual AQO from secondhand smoke alone, assuming a zero background RSP level from non-SHS sources. However the actual annual average Hong Kong RSP background for 2000 was 50.4  $\mu\text{g}/\text{m}^3$  (Hong Kong Environmental Protection Department 2000) so the number of workers exceeding the AQO is actually greater than shown in Figure 3.**

**US National Ambient Air Quality Standards for fine particulate air pollution:** In 1997, the EPA promulgated new 24-hour and annual standards for  $\text{PM}_{2.5}$ , of 65 and 15  $\mu\text{g}$  per cubic meter, respectively, based on consistency with the literature on health effects (Ware, 2000). The U.S. National Ambient Air Quality Standard (NAAQS) for  $\text{PM}_{2.5}$  is designed to protect against such fine particle health effects as premature death, increased hospital admissions, and emergency room visits (primarily the elderly and individuals with cardiopulmonary disease); increased respiratory symptoms and disease (children and individuals with cardiopulmonary disease such as asthma); decreased lung function (particularly in children and individuals with asthma); and against alterations in lung tissue and structure and in respiratory tract defense mechanisms. (Fed. Reg., 1997).

**Estimated Asthmatic Health Effects from SHS Air Pollution in Hong Kong Restaurants.** Increased risk of an adverse asthmatic health effect for asthmatic Hong Kong Catering workers may be estimated as follows. An exposure-response relationship for  $\text{PM}_{3.5}$  can be estimated (6% increase in asthmatic attack probability per 10  $\mu\text{g}/\text{m}^3$  increase in 24-hr Ave. fine particulate RSP levels, From Figure 3, Hong Kong Catering workers have a mean 24-hour average  $\text{PM}_{3.5}$  exposure due to SHS estimated at 197  $\mu\text{g}/\text{m}^3$ . If these are combined an exposure-response relationship may be estimated (Table 1), the increased probability of an adverse asthmatic health effect may be estimated, by assuming SHS-RSP has the same respiratory toxicity as outdoor RSP. The estimated increased risk of a fatal asthmatic attack for the HK workers is  $(197 \mu\text{g}/\text{m}^3)(6.8\% \text{ per } 10 \mu\text{g}/\text{m}^3) = 134\%$ , or 2.3 times. For the top 5%, the increased risk of an asthma death ranges from  $(667 \text{ to } 1373 \mu\text{g}/\text{m}^3)(6.8\% \text{ per } 10 \mu\text{g}/\text{m}^3) = 454\% \text{ to } 934\%$  or about 5 to 10 times

**Table 1. Effects of a 10  $\mu\text{g}/\text{m}^3$  increase in indicated 24-hr average  $\text{PM}_{10}$  particle air pollution levels on Asthmatics (Dockery & Pope, 1994).**

Asthmatic Effect	per 10 $\mu\text{g}/\text{m}^3$ increase in 24 hr $\text{PM}_{10}$ level	per 10 $\mu\text{g}/\text{m}^3$ increase in 24 hr $\text{PM}_{3.5}$ level*
Emergency Room Visits	+3.4%	+6.8%
Hospital Admissions	+1.9%	+3.8%
Bronchodilator use	+2.9%	+5.8%
Asthmatic Attacks	+3.0%	+6.0%
Respiratory Deaths	+3.4%	+6.8%

\*Estimated, this work, based on the conversion of 24-hr average  $\text{PM}_{3.5}$  levels into their  $\text{PM}_{10}$  24-hr equivalent, using the conversion factor:  $\text{PM}_{10} = 2\text{PM}_{3.5}$  (see text below).

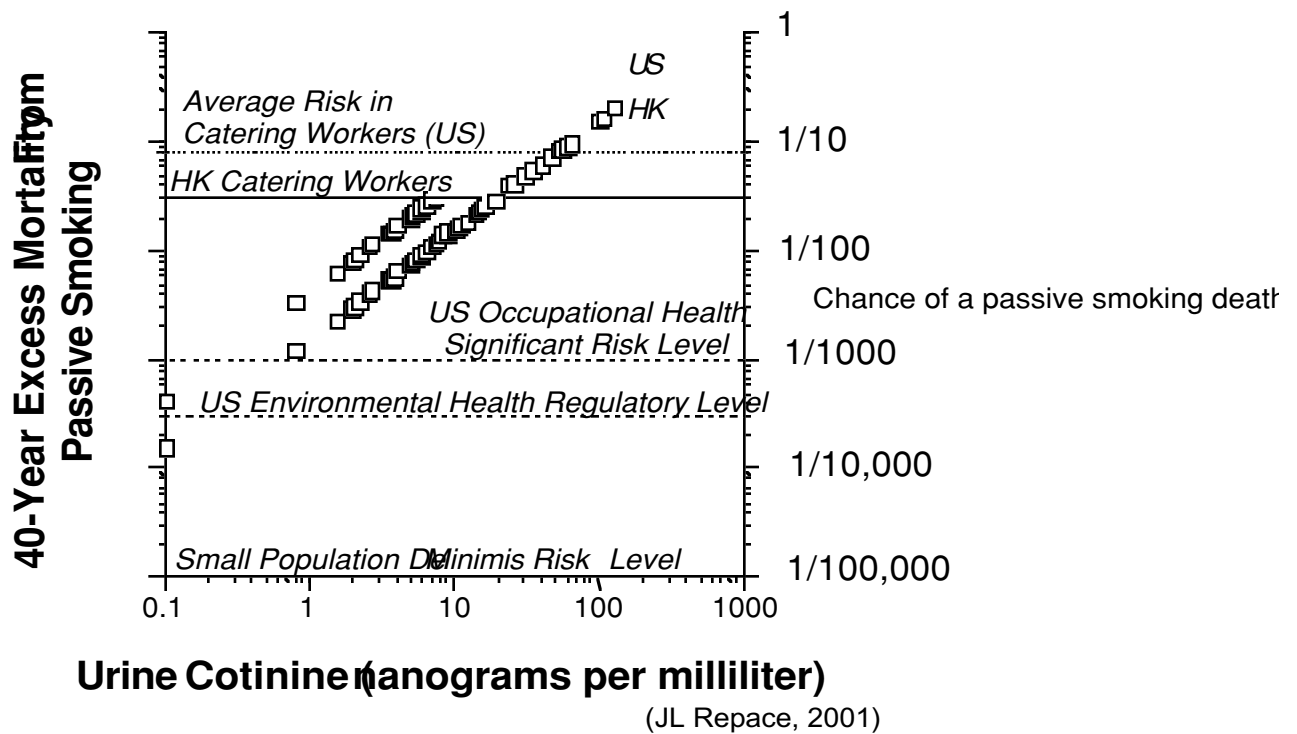
Outdoor  $\text{PM}_{10}$  contains about 50% fine fraction (Brook et al., 1997), called  $\text{PM}_{2.5}$  (particles 2.5 microns in diameter or less) which has greater effect on the lung than the coarse fraction (Fed Reg, 1997). SHS is mostly fine fraction (Repace & Lowrey, 1980, 1982).  $\text{PM}_{2.5}$  and  $\text{PM}_{3.5}$  are quite similar (Wallace, 1996).

**Figure 4. 104 Hong Kong Catering workers, exposed to second-hand smoke at work: Working lifetime combined risk from fatal heart disease & lung cancer based on both US and Hong Kong mortality rates and Hong Kong exposures to passive smoking**

**For US mortality rates:** Mean WLT Risk 7.8%; SD 9.5% ; 10th percentile: 2%; median 4.7% ;

90th percentile 17%; risk range for top 5%: 27%-55%.

**For HK mortality rates:** Mean WLT Risk = 3% with risk for the top 5%: 10% to 20%



**REGULATION OF RISK.** Several U.S. federal regulatory agencies promulgate regulations and standards to protect the public from involuntary exposure to environmental carcinogens and toxins. It is of interest to inquire as to what levels of population risk typically trigger regulation, what levels are beneath regulatory concern, and how consistently are they applied among various federal agencies. These are defined in two technical risk assessment terms: *de minimis* risk and *de manifestis* risk. A *de manifestis* risk is literally "a risk of obvious or evident concern," and has its roots in the legal definition of an "obvious risk", i.e., one recognized instantly by a person of ordinary intelligence. *De manifestis* risks are those that are so high that U.S. federal regulatory agencies almost always act to reduce them, and *de minimis* risks are so low that agencies almost never act to reduce them. For various reasons, risks falling in between these extremes are regulated in some cases but not in others; however, residual risks after control are generally *de minimis*. *De manifestis* risk corresponds to a lifetime excess probability of death of 3 in 10,000 ( $3 \times 10^{-4}$ ), and US *de minimis* risk corresponds to a probability of death of 1 in 1,000,000 ( $1 \times 10^{-6}$ ) (Travis, 1990; Repace and Lowrey, 1993). For smaller populations, a *de minimis* risk level of 1 per 100,000 is often adopted. In addition, US OSHA's *Significant Risk* level is 1 death per 1000 persons at risk, when exceeded, is regarded as resulting in "significant risk of material impairment of health," and may include serious irreversible morbidity (e.g. a heart attack) as well as mortality.

**SHS-RSP AND CANCER AND HEART DISEASE MORTALITY RISK.** Repace and Lowrey (1985b) derived a health based standard for environmental tobacco smoke based on SHS-RSP levels. For exposure to ETS-cotinine (24-hr average), the *de minimis* or "acceptable" lifetime (40

yr. exposure) risk level of 1 lung cancer death per million nonsmokers at risk occurs at 2.6 picograms of cotinine per milliliter of urine. Repace et al. (1998) calculated that the lifetime (40 yr. exposure) risk ratio of fatal heart disease to that of fatal lung cancer was 10:1. These exposure-response relationships have been validated by physical, clinical, and epidemiological data (Repace and Lowrey, 1985, 1993, Repace et al., 1998). OSHA's *Significant Risk* level of 1 death per 1000 persons at risk, is defined as "significant risk of material impairment of health." Table 1 translates this in terms of daily average cotinine in all relevant body fluids. If lung cancer risk and heart disease risk are combined, a urine cotinine level of 2.6 ng/mL corresponds to a combined risk of  $1.1 \times 10^{-2}$ . Alternatively, a cotinine level of 1 ng/mL corresponds to a lifetime risk of  $\sim 4.23$  per 1000.

**Table 1. Estimated exposure-response and dose-response relationships used to estimate excess lifetime mortality risks from lung cancer (LCD) and coronary heart disease (CHD) based upon 24-hour average ETS cotinine biomarkers\*, assuming a worker respiration rate of  $\rho = 1 \text{ m}^3/\text{hr}$ .**

SHS Marker	Ave. Daily* Exposure/dose	LCD Risk*	CHD Risk*
Serum Cotinine	0.40 ng/mL	1 per 1000	1 per 100
Saliva Cotinine	0.46 ng/mL	1 per 1000	1 per 100
Urine Cotinine	2.6 ng/mL	1 per 1000	1 per 100

\*\*Inhaled dose. \*Exposure duration: 40 years (Repace et al., 1998; Repace & Lowrey, 1985b; 1993)

### **Estimated total passive smoking deaths among catering workers in Hong Kong.**

The number of Hong Kong catering workers is approximately 200,000. At the mean risk level of approximately 8%, estimated using US mortality rates, 16,000 catering workers would be estimated to die a passive-smoking-caused death over a working lifetime of 40 years. This results in an estimated 400 catering workers' deaths per year as a result of on-the-job passive smoking. An estimated 98% of the Catering Workers exceed the US OSHA's "Significant Risk" level resulting in "significant risk of material impairment of health."

*If the lung cancer and heart disease deaths are adjusted to current Hong Kong mortality rates the combined estimated working lifetime risk reduces to 3% and the estimated deaths to 6,000, or about 150 per year.*

### **Conclusions:**

- 1. Passive smoking dose has been quantified from urinary cotinine in 104 Hong Kong Catering Workers exposed to secondhand smoke only at work,**

2. **Based on those cotinine levels, at US mortality rates for heart disease and cancer, an estimated 400 Hong Kong catering workers would die each year from heart disease and lung cancer caused by passive smoking in the workplace. For current Hong Kong mortality rates the estimated figure is 150 per year.**
3. **An estimated 98% of Hong Kong Catering Workers sampled exceeded the US OSHA's "Significant Risk" level resulting in "significant risk of material impairment of health."**
4. **The Hong Kong Catering Workers studied had levels of cotinine in their urine that were 5-1/2 times that of a Hong Kong control group with no regular exposure to secondhand smoke at work or elsewhere.**
5. **Considering heart disease and lung cancer only, I estimate that between 3 and 8 out of 100 Hong Kong restaurant workers will die prematurely from secondhand smoke exposure at work.**
6. **At Hong Kong mortality rates the top 5% (1 in 20) of nonsmoking Hong Kong restaurant workers exposed to passive smoking at work have an estimated probability of prematurely dying from passive smoke exposure ranging from 10% to 20%.**
7. **The average catering worker in the study has an exposure to respirable particulate air pollution from ETS exposure at work which exceeds the Hong Kong Annual AQO by 2-1/2 times, assuming a zero background RSP level from non-ETS sources. Two thirds of the workers exceeded the 24-average AQO.**
8. **The risk of a fatal attack for asthmatic workers is more than doubled at the average exposure level of the workers studied. For the top 5%, the risk increase ranges from 5 to 10 times.**
9. **The estimated risks are far beyond the levels at which hazardous air pollutants are regulated in the United States by either occupational or environmental health authorities. They also violate all US annual and short-term air quality standards for fine particulate air pollution.**
10. **Secondhand smoke poses unacceptable risks to catering workers. These risks cannot be made acceptable using engineering controls. Smoke-free workplaces are the only viable control measure.**

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