

C O S H

HONG KONG COUNCIL ON SMOKING AND HEALTH

Passive smoking and risks for heart disease and cancer in Hong Kong catering workers 2001

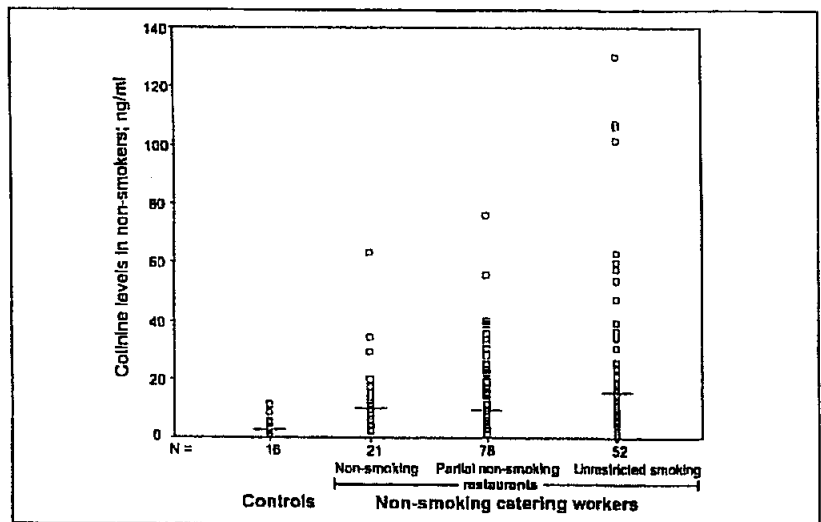
May 2001

Report No. 8

Second-hand smoke exposures and passive smoking in non-smoking catering workers in Hong Kong: the combined risks for heart disease and cancer

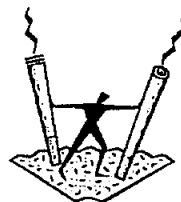
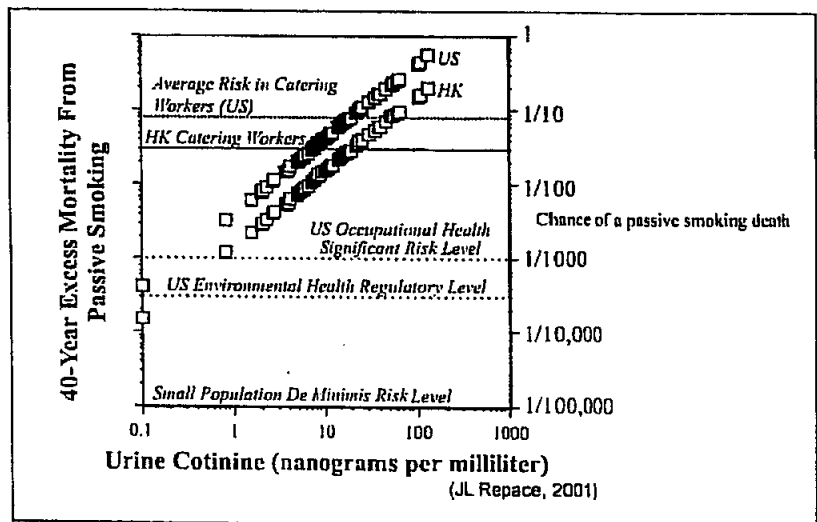
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Urine cotinine levels in non-smoking catering workers and controls



104 catering workers, exposed to second-hand smoke only 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



1. Background

Second-hand smoke and passive smoking: Passive smoking results from non-smokers breathing air which is contaminated with second-hand smoke made up of *mainstream smoke* exhaled by smokers and *side-stream smoke* emitted from the tips of burning cigarettes and cigars. Second-hand smoke is extremely poisonous; it contains over 4000 chemicals in the form of particles and gases.

Health hazards: Exposures to second-hand smoke are the cause of many health problems in non-smokers. These include extreme irritation to mucous membranes in the eyes, nose and throat; chronic respiratory symptoms such as cough, phlegm and wheeze and exacerbations of asthma. Asthmatics experience a decline in lung function when exposed to second-hand smoke. Passive smoking also causes damage to blood vessels so that non-smokers are at increased risk of heart attacks and stroke. Passive smoking is a hazard to the health of pregnant women and the foetus. Children are extremely sensitive to second-hand smoke and those with passive smoking exposures have more health problems including middle ear disease, bronchitic symptoms, acute chest infections and emergency admissions to hospital.

Second-hand smoke contains a high concentration of carbon monoxide which is implicated as one cause of heart disease in smokers. Tobacco smoke also increases platelet aggregation and causes changes in blood clotting mechanisms. Cancer causing compounds in second-hand smoke are inhaled and pass into the circulation. Exposure of non-smokers to tobacco smoke leads to increased blood and urinary concentrations of tobacco-specific cancer causing substances.

The US Environmental Protection Agency (EPA) and the UK Government Department of Health Scientific Committee on Tobacco and Health (SCOTH) and many other national and international agencies accept the evidence that exposures to passive smoking cause lung cancer and conclude that second-hand smoke is a *proven human carcinogen*.

No safe threshold: In terms of its cancer inducing potential there is no known safe level of second-hand smoke. Neither simple measures designed to separate smokers from non-smokers nor ventilation engineering will prevent passive smoking when a common air space is contaminated with tobacco smoke.

In 1999 the American Society of Heating, Refrigerating and Air-Conditioning Engineers (ASHRAE) eliminated all reference to any level of smoking being permissible from the ANSI/ASHRAE indoor air quality standards. This standard now makes it clear that the governing standard (ANSI/ASHRAE 62-1989) is based on a totally non-smoking environment.

The prevention of passive smoking: There is increasing awareness and acceptance in Hong Kong, on the part of the Government and the general public, that effective controls are needed to prevent involuntary passive smoking in all public places and in the workplace. At the present time very few indoor places meet the necessary criteria to ensure that the public and the workforce are protected against second-hand smoke exposures.

One major deficiency in the present legislation concerns the catering industry where there is no protection for most customers and none at all for workers. The requirement for restaurants with 200 or more seats to offer one third of

seating in "smoke-free" sections is a token arrangement which cannot meet even the minimum criteria and public health requirements for a smoke-free indoor environment.

Two previous reports on Hong Kong public opinion by COSH, in 1995 and 2000, showed that the overwhelming majority of the public wanted smoke-free dining facilities and that patrons would eat out more often given assurances of smoke-free facilities; in other words it would be good for business. A large proportion of the customers in these surveys frequently experienced adverse exposures to second-hand smoke including foul odour, contamination of clothes and hair, irritation of eyes, nose and throat, and asthma/ wheezing or other respiratory problems. Over one third formed an unfavourable impression of the restaurants concerned and considered taking their patronage elsewhere.

Two previous studies in Hong Kong have shown that passive smoking in the workplace is a major cause of chronic respiratory problems in Hong Kong. This report examines the preliminary results of a new survey of non-smoking workers in the catering industry, which aimed to assess their passive smoking exposures in different work settings and their risks for heart disease and cancer.

2. Objectives

The objectives of this pilot study were to

- document workplace and other exposures to second-hand tobacco smoke in non-smoking catering workers
- collect and analyse urine samples for cotinine which is a breakdown product of nicotine and an indicator of passive smoking in non-smokers
- estimate the combined working-lifetime risks for heart disease and lung cancer in Hong Kong catering workers.

3. Subjects and Methods

Subjects: A total of one hundred and eighty four catering workers were recruited to the study and 165 provided complete data on exposures to second-hand smoke. All were volunteers, invited on the basis that they were non-smokers but any smokers who wished to participate were accepted. They received \$100-\$150 (including travel expenses) for their participation. They were asked to complete an interview schedule and give a 50 ml sample of urine. All subjects were tested using a monitor to detect carbon monoxide in their breath (expired air). Carbon monoxide levels in human breath are usually less than 10 parts per million (ppm) in non-smoking subjects. Fourteen subjects were found to be (or declared that they were) occasional or regular smokers and 170 (83 male and 87 female) were non-smokers. Seven subjects were found to be *regular smokers* either because of self-declaration or raised breath carbon monoxide (>9 ppm) and seven more admitted to being *occasional smokers*, defined as using less than 7 cigarettes per week. Their results are included in the findings for comparison with the other groups (Table 1). The majority (86%) of workers were employed in restaurants which permitted smoking. The remainder were from catering facilities which did not permit any smoking by customers (Table 2).

An additional sample group of 16 control subjects were recruited, being physicians, nurses or university researchers. All were non-smokers who worked in a smoke-free workplace and who generally avoided smoky environments.

Table 1: Catering workers and urinary cotinine levels by exposure to second-hand smoke at work, home and leisure activities

Subjects	Exposure outside work	Non-customer exposure	Mean	N	SD	Range
Controls	no exposure outside work	nil	3.3	13	3.5	0-11.2
	home or leisure exposure	nil	5.5	3	4.9	1.1-10.8
	Total	nil	3.7	16	3.7	
Worker in non-smoking restaurant	no exposure outside work	nil	6.4	3	6.6	2.6-14.0
		other staff or break	14.0	10	17.7	2.2-62.9
		Total	12.3	13	15.9	
	home or leisure exposure	nil	20.3	5	11.9	3.9-34.1
		other staff or break	9.9	3	3.9	5.8-13.6
		Total	16.4	8	10.7	
	Total	nil	15.1	8	12.0	
	other staff or break	13.1	13	15.5		
	Total	13.8	21	14.0		
Worker in partial smoking restaurant	no exposure outside work	nil	6.1	6	6.4	1.5-18.6
		other staff or break	14.3	50	10.8	2.0-55.3
		Total	13.4	56	10.7	
	home or leisure exposure	nil	7.1	1		7.1
		other staff or break	16.6	21	17.2	1.0-76.4
		Total	16.2	22	17.0	
	Total	nil	6.3	7	5.8	
	other staff or break	14.9	71	13.0		
	Total	14.2	78	12.7		
Workers in unrestricted smoking restaurant	no exposure outside work	nil	15.9	4	6.5	7.6-23.1
		other staff or break	28.7	34	33.9	0-129.4
		Total	27.4	38	32.3	
	home or leisure exposure	nil	26.5	3	10.5	14.7-34.6
		other staff or break	20.0	11	21.9	0.03-62.3
		Total	21.4	14	19.8	
	Total	nil	20.4	7	9.5	
	other staff or break	26.6	45	31.4		
	Total	25.7	52	29.4		
Occasional smoker	no exposure outside work	other staff or break	145.0	6	118.4	2.2-286.8
	home or leisure exposure	other staff or break	881.4	1		
	Total	other staff or break	250.2	7	298.6	
Regular smoker	no exposure outside work	other staff or break	2996.3	3	1695.0	1281-4671
	home or leisure exposure	other staff or break	4034.0	4	1274.1	
	Total	other staff or break	3589.2	7	1441.2	

Table 2: Number (%) of non-smoking workers by type of catering facility

Non-smoking restaurants		24
Fast-food	22	
Western/Eastern	1	
Canteen	1	
Smoking restaurants		146
Chinese restaurants	70	(41.2)
Cha Cham Ting	31	(18.2)
Fast food shop	6	(3.5)
Western/Eastern	8	(4.7)
Club/canteen/caf	31	(18.2)
Total		170

Cotinine: When nicotine in tobacco smoke is absorbed into the circulation it undergoes metabolic breakdown in the liver into other compounds, including *cotinine* which can be measured in blood, saliva and urine. In this way it can be used as a marker of exposure to the toxic components of second-hand smoke in non-smokers who become passive smokers. The urinary cotinine levels of all workers and the controls in this survey were measured by the MetLife Laboratory in New York (Dr N Haley). The cotinine values are expressed as nanograms (ng) per milliliter of urine.

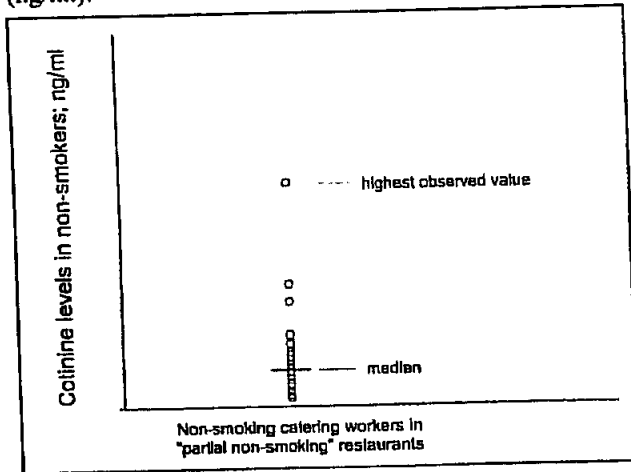
Interview: In addition to basic demographic information, workers were asked about workplace, home and leisure exposures to tobacco smoke. The numbers and proximity to them of smokers in their workplace were documented whenever possible. The workers' past active smoking history was recorded when relevant and the time since quitting was recorded. Finally questions about respiratory and cardiovascular health, including diagnoses and current symptoms were included.

Analyses: *Urinary cotinine levels* were analysed by main groups and sub-groups, defined by their worker or control status, workplace type and reported exposures to tobacco smoke from any source.

The classification of subjects has initially been carried out on an *a priori* basis using their criteria for selection (ie "control", or "catering worker") or their place of work (ie "non-smoking" or "smoking" catering facilities).

These findings have been further explored by subgroups, including "non-waiter" (eg accounts clerks, housekeepers, chefs, others), and "waiter" (anyone serving tables as waiter or senior restaurant supervisors). Exposures have been examined by the workers' declarations of "other exposures" including staff smoking, exposure during rest times, home and leisure activities.

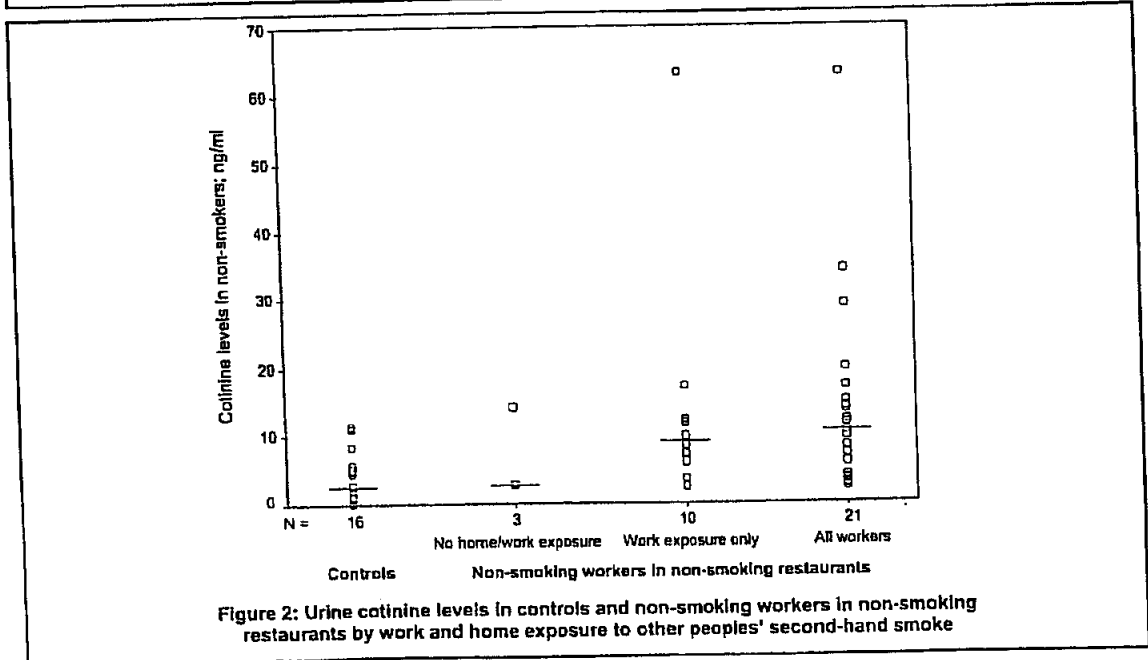
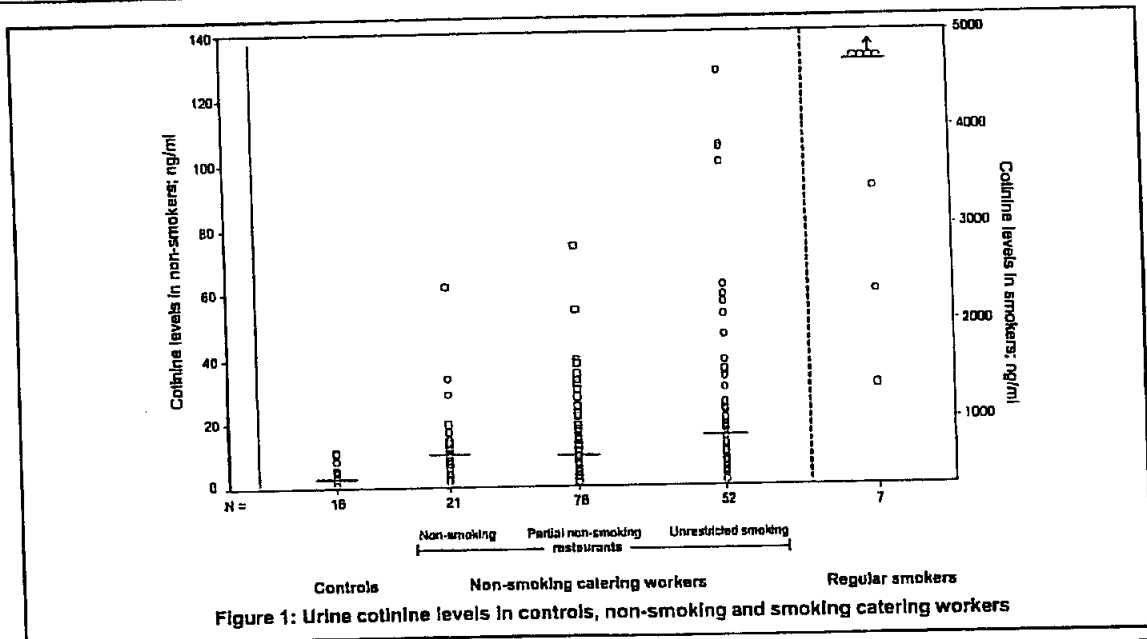
The graphics for the urinary cotinine values are presented as dot charts as shown in the example below. Each dot represents an individual cotinine value within the group tested; the lowest and highest dot indicate the range and the horizontal bar is the median or middle value. The cotinine values are measured as nanograms per milliliter (ng/ml).



The risk of heart disease and lung cancer in this sample of Hong Kong catering workers who are exposed to second-hand smoke has been estimated using a pharmacokinetic risk model developed by Repace and his co-workers. This enables cotinine levels in urine, saliva and plasma to be related to lung and heart disease in passive smokers. The risk is calculated for a 40 year working life time (WLT,). Using this model Repace and Lowrey associated an average plasma cotinine of 0.4 ng/ml with a WLT, increased mortality for lung cancer of 1 in 1000. The model of estimated mortality associated with salivary cotinine level indicates that the risk for heart disease rises from 1 in 3000 to about 1 in 100 with a gradient of salivary cotinine of 0.1 up to 1 nanogram/milliliter. This risk model successfully predicted the risk observed in the American Cancer Society Cohort Study of passive smoking and lung cancer in non-smokers.

4. Findings

Overall, our control subjects with declarations of low exposures had the lowest cotinine levels. The lowest risk group in this survey, were doctors, nurses and members of a university department of public health who were non-smokers, working in a totally smoke free environment and



who generally took action to avoid second-hand smoke exposures. At the high end of the non-smoking subjects were waiters and other staff in catering facilities with either partial smoke-free areas or no restrictions on smoking. The small group of regular smokers in the sample showed the expected very high levels of urinary cotinine which were several thousand percent higher than the controls and non-smoking workers (Figure 1).

The data are heterogeneous and show important variations in cotinine levels in catering workers by exposures to tobacco smoke from both customers and other staff, as well as home and leisure exposures. The following brief description is based on the data in the summary Table.

Controls: A total of 16 subjects were tested. Thirteen control subjects with no work or other exposures had a mean of 3.3 (median 2.6, range 0-11.2). In an additional three subjects who declared that they had exposures outside of work the mean cotinine was 67% higher at 5.5 (median 4.5, range 1.1-10.8) (Table 1).

Workers in "non-smoking" restaurants: There was considerable variation in cotinine levels in workers in those restaurants which were designated as "non-smoking" for the purpose of their catering services to the public. Overall, the 3 workers with no exposures *outside of work* who declared that they avoided or did not receive *non-customer exposures at work* had the lowest mean cotinine level at 6.4 (median 2.7, range 2.6-14.0). However a majority of staff (13/21; 62%) were in fact exposed to *non-customer second-hand smoke* because of other staff smoking at break times. Their mean cotinine levels range from 9.9 (median 10.3, range 5.8-13.6) to 14.0 (median 9.0, range 2.2-62.9), that is 50% to 118% higher than workers not exposed to this source and 200% to 324% higher than the lowest risk controls (Figure 2).

Because of exposure to staff smoking at work the cotinine levels in many workers in "non-smoking" restaurants were as high as those in workers in "partial non-smoking" restaurants.

Workers in "partial-non-smoking" restaurants: These findings relate to any worker employed in an organisation which permitted smoking but had various forms of smoke-free areas or seating. Those workers with no exposure *outside of work* and no *non-customer exposure at work* had the lowest cotinine at 6.1 (median 4.2, range 1.5-18.6); a figure which is 85% higher than the value for the lowest risk controls in this study.

Those with any other additional exposures to tobacco smoke had higher mean levels ranging from 7.1 in one subject associated with *home and leisure exposure only*, to 14.3 (median 9.6, range 2-55.3) in those with *other staff and/or break time exposures*, and a mean of 16.6 (median 12.0, range 1-75.4) in 21 workers with both *home/leisure and staff/break time exposures*. These mean values are 333% to 403% higher than the control group (Table and Figure 1).

Workers in "unrestricted smoking" restaurants: Overall the mean cotinine levels and the ranges of values in all subgroups of workers in unrestricted smoking establishments were higher than those in workers who had lower declared exposures. In 4 workers with no exposures *outside of work*, and no *non-customer exposures*, the mean was 15.9 (median 16.5, range 7.6-23.1) compared with 28.7 (median 17.3, range 0-129.4) in 34 workers with non-

customer workplace exposure. For those with *home/leisure and/or non-customer exposures* the mean cotinines ranged from 20.0 to 26.5 (medians 10.4, 30.2, range 0.03-62.3). Overall for this group of 52 workers in unrestricted smoking establishments the mean for those who did not have exposures from other staff was 20.4 (median 18.2, range 7.6-34.6), and 26.6 (median 14.8, range 0-129.4) for those with staff/break exposures in addition to customer exposures (Table and Figure 1).

Cotinine levels in waiters and non-waiters: When workers were classified into subgroups relating to their job description, no significant differences were found in the mean cotinine values between waiters and workers in other departments in the same establishment.

However some individual waiters had the highest cotinine values observed in the survey. For example the mean cotinine for non-waiters in partial-smoking restaurants was 14.0 (median 12.1, range 1.0-35.0) compared with 14.2 (median 9.4, range 1.4-75.4) for waiters. In the restaurants with unrestricted smoking the mean cotinine for non-waiter staff was 23.0 (median 18.6, range 0.03-57.3) compared with 26.9 (median 14.7, range 0-129.4) for waiters. Lower cotinine values were observed in 3 catering workers who worked in either partial-smoking or unrestricted smoking restaurants. Two of these were non-waiters.

Variations by work exposure and gender: The average restaurant worker, who had second-hand smoke exposures at work only, had a urinary cotinine which was 464% higher than the control subjects. These 104 workers, with work exposure only, had a mean cotinine of 18.6 (median 11.1, range 0-129.4) compared with a slightly lower mean 17.0 (median 10.9, range 0-129.4) in the whole group of 170 workers (Figure 3). There is therefore no evidence that the high cotinine values observed in workers are mainly due to second-hand smoke exposures outside of their work (Figure 3). There was no significant difference in cotinine levels between male and female workers.

Ventilation and cotinine levels: The majority (98/105; 93%) of catering workers who were exposed to tobacco smoke only at work, stated that air conditioning units operated in their workplace. In general cotinine levels in these workers were as high or higher than the levels in workers without air conditioning.

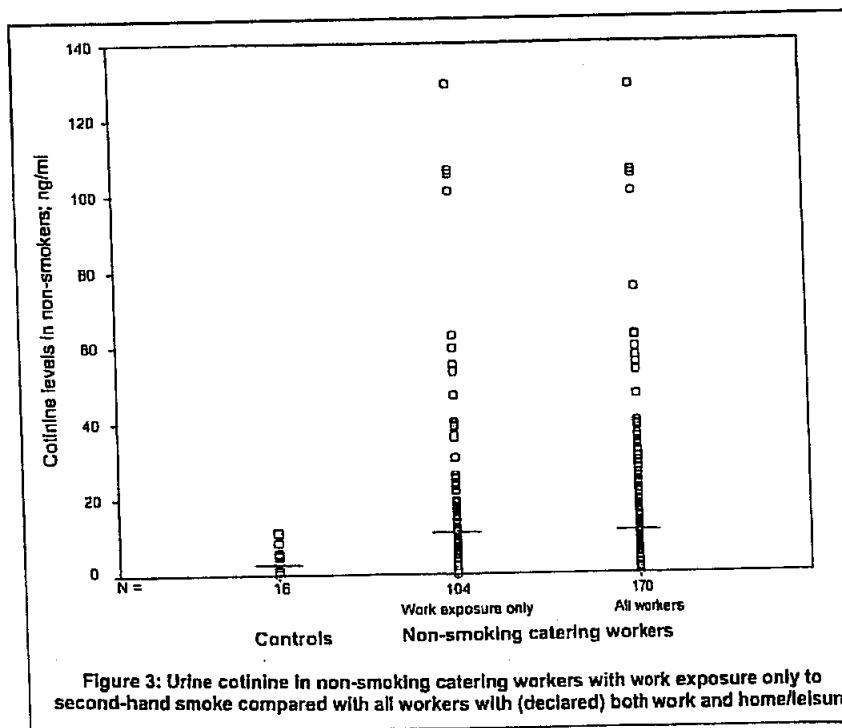
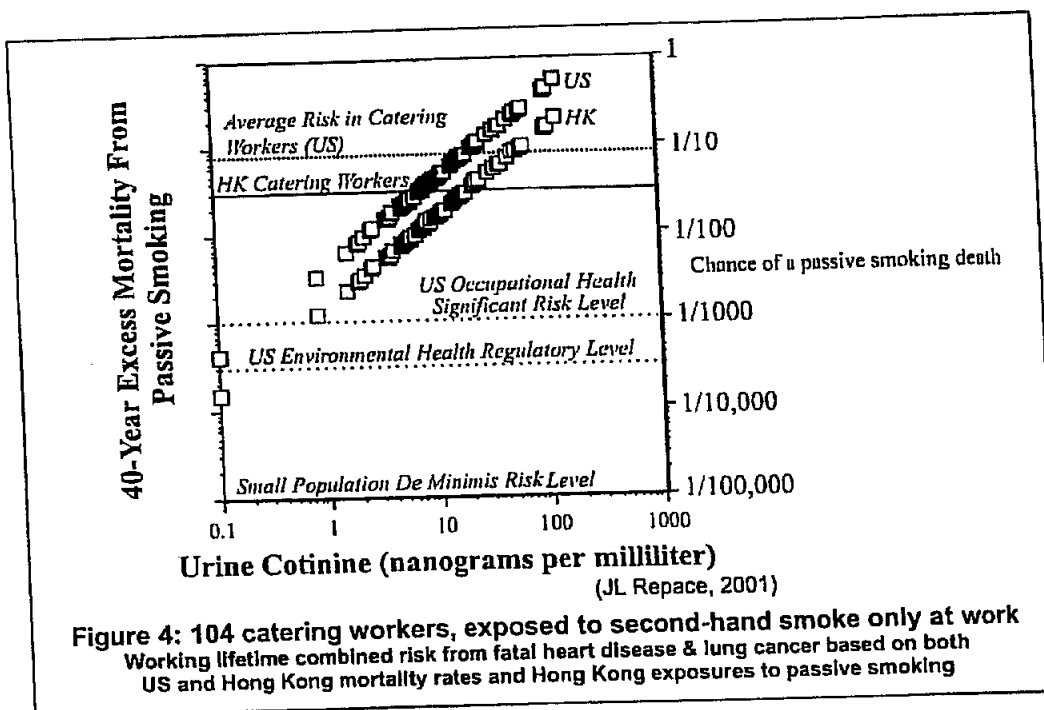


Figure 3: Urine cotinine in non-smoking catering workers with work exposure only to second-hand smoke compared with all workers with (declared) both work and home/leisure



The cotinine levels were lower in workers who had their last shift more than 12 hours previously, compared with those who had worked more recently or were at work during the survey (Table 3). This reflects the exposure levels and the biological half-life of cotinine in body fluids.

Table 3: Relationship between shift work and cotinine level

Restaurant type	Last shift		More than 12 hours ago		Less than 12 hours ago	
	Mean (SD)	N	Mean (SD)	N	Mean (SD)	N
Non-smoking	4.9 (1.3)	2	14.8 (14.4)	19	15.4 (14.1)	52
Partial smoking	11.7 (9.2)	26	21.7 (36.3)	11	26.8 (27.7)	41
Unrestricted smoking						

Declared smokers: The mean cotinine level for those who were classified as *occasional smokers* was 145.0 (median 167.9, range 2.2-286.8) for those with no exposure *outside of work*. The overall mean for this subgroup of smokers was 250.2 (median 213.9, range 2.2-881.4). The use of tobacco in this group was variable and very low in some subjects. Four out of seven had cotinine >200, the other 3 ranged from 2.2 to 121.8.

For *regular smokers* the mean cotinine was 3589 (median 4671, range 1282-4671) ng/ml. Variations within this group are likely to reflect mainly individual smoking pattern and amount rather than passive smoking exposure.

Combined heart disease and lung cancer risks: The risk calculations based on urinary cotinine levels were carried out on a selected subgroup of 104 non-smoking workers who only had work exposure to second-hand smoke. In this series the mean urinary cotinine concentration is 18.6 ng/ml and the median 11.1 ng/ml. The 90th percentile is 39.1 ng/ml and many workers have cotinine in excess of 40 ng/ml.

The 40 year working lifetime combined excess risk for heart disease and lung cancer is 7.8% (that is 1 in every 13 persons at risk) based on the US population mortality for heart disease and lung cancer (Figure 4). However, in Hong Kong, the present population mortality rates for heart disease are lower than in the US by a factor of about 2.6. The working lifetime excess risk for Hong Kong is

3% (that is 1 in 33 workers at risk) (Figure 4). This means that in the current population of catering workers (about 200,000), we predict 150 deaths per year of exposure from passive smoking, or 6,000 in a working lifetime. Of these 6,000 deaths, 3,840 (64%) will be in workers who have never smoked. Also marked on the graph in Figure 4 is the *de minimis* risk level, which corresponds to an excess lifetime mortality risk of one death in a million persons at risk and is considered acceptable from a regulatory point of view. An estimated risk level as high as 3 in 10000, marked on the graph as the *US Environmental Health Regulatory Level*, would be considered so unsafe that US Federal regulatory agencies almost always act to reduce them.

The aim of interventions and control of second-hand smoke would be to reduce the risk level to zero or at least to the *de minimis* level.

5. Comment

Based on the findings of this sample we can conclude that the majority of catering workers in Hong Kong, both waiters and other staff, have high levels of exposure to second-hand smoke in their workplace with a major risk to their current and future health.

None of the groups of workers examined had mean levels of cotinine as low as that of the control subjects and most were more than double this value. Tobacco smoke from other staff smoking (ie the *non-customer exposures*) within the workplace were apparently important sources of second-hand smoke for all catering workers. This was a major source of tobacco smoke exposure in those workers supposedly working in smoke-free restaurants. The mean levels of those exposed to non-customer smoking were more than twice the levels of those not exposed. Non-customer smoking in all restaurants is clearly a hazard to both workers and patrons, as would be expected from the well established parameters of smoke dispersion in all indoor environments.

Questions will be raised about the validity of the findings in this survey, and particularly about the possibility of misclassification of occasional smokers as non-smokers. Occasional smokers are relatively uncommon and overall

we believe that smokers have been effectively excluded from this sample by the questionnaire and breath carbon monoxide screening. Very high cotinine values (>85) have been found in other surveys, eg in non-smoking bar tenders in Buffalo, New York (Repace 2001). In our survey there were four cotinine values greater than 75 in non-smoking restaurant workers (3 female, one male; 101.1, 105.4, 106.5 and 129.4). All of these subjects worked as waiters in restaurants with unrestricted smoking; all stated that several co-workers smoked near to them and all were at work during the survey and had been at work the previous day. All stated that they had no exposure outside of work; we believe they are passive smokers. Exclusion of these four high values would only reduce the mean cotinine for all restaurant workers with work exposure from 18.6 to 15.0 ng/ml and would not affect the conclusions of the survey.

The data also show the importance of home and leisure exposures to second-hand smoke in non-smokers in Hong Kong. All of the subgroups in this pilot survey showed a marked tendency to have raised cotinine levels if they were exposed to smoke in their leisure venues or at home.

General exposures to second-hand smoke in Hong Kong are clearly widespread as only 2 (13%) out of the 16 "low-risk" control subjects had zero cotinine levels. This contrasts with a recent population survey by the US Center for Disease Control which showed that, as a result of countrywide smoking bans in public and indoor places in the United States, 50% of the sample had *undetectable* levels of cotinine.

The mean urinary cotinine in our lowest risk group in Hong Kong (those without any known home or leisure exposure) was 3.3 ng/ml, a finding which is totally unacceptable given that in the US it indicates a lifetime excess risk for coronary heart disease mortality of greater than 1 in a 100 compared with the normative *de minimis* standard of acceptable risk of 1 in 1,000,000.

The Government should increase the resources available to inform the public of the serious health hazards of second-hand smoke, including those associated with smoking in the home.

A recent study in New Zealand showed that the exposure of bar and restaurant staff to tobacco smoke can be as high as the exposure of active smokers. The hair nicotine levels of non-smoking workers in workplaces with no restrictions on smoking were as high as those in smokers.

Previous studies of non-smoking workers exposed to second-hand smoke in Hong Kong have demonstrated an increased frequency of chronic respiratory complaints (cough, phlegm and wheeze), increased health care utilization and costs and sickness absence from work.

Passive smoking is increasingly recognized as an occupational health risk world-wide. For example:

- In October 1997, 60,000 US flight attendants won a major settlement in a class action against transnational tobacco companies. The action was initiated by a non-smoking flight attendant who contracted lung cancer. The tobacco industry did not admit liability.
- In the Netherlands a court ruled in May 2000 that employers must guarantee that non-smoking staff have a working environment completely free of tobacco smoke. It upheld a postal worker's complaint that her exposure to tobacco smoke at work *infringed her right to work in a smoke-free environment*. The court ruled that her employers were bound by the constitutional rights of citizens, to protection of "physical integrity and "health", to provide such conditions. The employers failed to

satisfy this right under employment law.

- In May 2001 an Australian barmaid, a non-smoker, was awarded US\$235,000 for cancer caused by working for 11 years in a smoky bar. Most Australian states have already banned smoking in pubs, clubs and restaurants and a similar ban will come into force in New South Wales in September 2001.

Cotinine levels in this survey are consistently higher in establishments with partial or unrestricted smoking. Increasing smoker density in designated smoking areas increases the hazard to workers who have to service these areas. In separately ventilated smoking lounges and cigar divans the concentrations of second-hand smoke particulates and gases, including cardiovascular toxins and cancer causing substances, will predictably be very high. The contamination persists after smoking ceases and part of this comes from off-gassing from deposits on furniture and fittings. The risks to both patrons and staff are currently being ignored.

It is clear that ventilation technology cannot control and reduce the risk from second-hand smoke to minimal safety standards (1 in a million) *without massively impractical increases in ventilation and intolerable levels of air changes of "typhoon strength"* (JL Repace: Repace@erols.com).

However damage to the health of catering workers from passive smoking is wholly preventable. The establishment of smoke-free bars and taverns in California was followed by a rapid improvement in the respiratory health of the workers. The present survey confirms that workers in Hong Kong who are forced to breathe second-hand tobacco smoke in their workplace have markedly raised levels of nicotine metabolites in their circulation. We know that this is also an indicator of toxic exposures to substances which cause *heart disease and cancer* in addition to chronic *respiratory health problems*.

On the other hand the tobacco industry and many sectors of the hospitality industry continue to (i) deny that second-hand smoke is a poison, (ii) deny that both workers and customers are injured by breathing second-hand smoke, (iii) oppose the introduction of environmental and public health measures to prevent passive smoking in the workplace and public places. This is in spite of the fact that no *bone fide* economic analyses have shown any adverse impact on catering business or tourism. Tobacco industry propaganda has generated unjustified concern about loss of business and jobs. There is no reason why Hong Kong workers should not now be protected against the risks of passive smoking.

Legislation to provide and ensure totally smoke-free indoor workplaces is the only satisfactory solution to this widespread problem and it is urgently needed as a public health and occupational health measure in Hong Kong.

Voluntary agreements and codes of practice will not work and create many problems of monitoring and enforcement. Legislation on smoking bans in all public places is the only cost-effective and reliable means of protecting non-smokers. No workers, whether smokers or non-smokers should be obliged to work in a smoke contaminated workplace. *The principle on which Hong Kong's future workplace smoking controls must be based is that no worker should be required to work in an environment where tobacco products are burning.*

Adherence to this principle will not permit smoking in outdoor catering facilities. Partial smoking restrictions of all kinds leave non-smokers exposed to the risk of passive smoking.

Summary conclusions and recommendations

- 1 The world's best scientific literature on health risks from passive smoking clearly demonstrates that second-hand smoke is extremely poisonous and the cause of many health problems including chronic respiratory disease, coronary heart disease and cancers.
- 2 The majority of catering workers in Hong Kong are exposed to second-hand smoke in their workplace and most of them have markedly raised urinary cotinine concentrations which indicate markedly raised health risks for chest and heart disease and cancer in addition to many other health problems caused by passive smoking.
- 3 Most of the non-smoking subjects in this new survey have raised working lifetime excess risks for heart disease and lung cancer as a result of passive smoking. In catering workers the average excess risk was 3% or about 1 in 33. We estimate that among 200,000 catering workers, 6,000 will die from passive smoking due to heart disease and lung cancer; 3,800 (64%) of these deaths will be in never smokers.
- 4 In a group of "low risk" control subjects from smoke-free workplaces, many had detectable cotinine levels indicating that for many of them the airspaces of their home, leisure activities or other worksites visited by them are contaminated by tobacco smoke. All non-smokers in Hong Kong should have no detectable cotinine in body fluids.
- 5 There is no practical solution from ventilation engineering to the problem of second-hand smoke exposures; the only safe and most cost-effective strategy is to introduce smoke-free regulations in all catering facilities and other workplaces. The principle must be that no worker should have to work in air contaminated with tobacco smoke in order to hold a job.
- 6 There is an urgent need for effective and enforceable legislation which will ensure that all workers in all workplaces in Hong Kong do not have to breathe second-hand smoke.
- 7 There should be no exceptions to, or trade-offs in, smoke-free regulations which will lead to the health of workers being placed at risk.
- 8 There should be an urgent review by Government of designated smoking areas including smoking lounges which are separately ventilated, and particularly those which are continuously staffed such as cigar divans. The health implications for all workers who service any type of smoking lounges or other designated areas should be examined and re-assessed.
- 9 The catering and hospitality industry should take the lead now in implementing comprehensive smoke-free policies in all facilities to protect both staff and customers.
- 10 The public, the media, legislators and particularly the catering industry should be aware that the tobacco industry has for many years consistently denied and obfuscated the findings of research into second-hand smoke and passive smoking.
- 11 We fully expect that the tobacco industry will also attempt to discredit the findings of this latest investigation in Hong Kong, but there are incontrovertible reasons why Government policy to eradicate passive smoking should be fully implemented without further delay.

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