Effects on Chinese Restaurant Workers of Exposure to Cooking Oil Fumes: A Cautionary Note on Urinary 8-Hydroxy-2′-Deoxyguanosine

Chih-Hong Pan,[1,2] Chang-Chuan Chan,[1] and Kuen-Yuh Wu[3]

[1]Institute of Occupational Medicine and Industrial Hygiene, College of Public Health, National Taiwan University; [2]Institute of Occupational Safety and Health, Council of Labor Affairs, Executive Yuan, Taipei, Taiwan, People's Republic of China and [3]Division of Environmental Health and Occupational Medicine, National Health Research Institute, Miaoli, Taiwan, People's Republic of China

Abstract

This study evaluates oxidative DNA damage in workers who are exposed to cooking oil fumes (COFs) in Chinese restaurants. The study participants were 387 nonsmoking Chinese restaurant workers, 202 kitchen staff, and 185 service staff at 23 Chinese restaurants in Taiwan. Airborne particulate matter and particulate polycyclic aromatic hydrocarbon levels were monitored in kitchens and dining areas. Urinary 1-hydroxypyrene (1-OHP) was used as an internal dose of exposure to COFs, and urinary 8-hydroxy-2′-deoxyguanosine (8-OHdG) was used as an oxidative DNA damage marker. The relationship between workers' 8-OHdG and 1-OHP levels was estimated using linear mixed-effects models. Airborne particulate matter and polycyclic aromatic hydrocarbons levels in kitchens significantly exceeded those in dining areas. The kitchen staff's geometric mean levels of urinary 8-OHdG (7.9 μg/g creatinine) and 1-OHP (4.5 μg/g creatinine) were significantly higher than those of the service staff, which were 5.4 and 2.7 μg/g creatinine, respectively. Urinary 1-OHP level, work in kitchens, gender, and work hours per day were four significant predictors of urinary 8-OHdG levels after adjustments are made for covariates. Oxidative DNA damage was associated with exposure of Chinese restaurant workers to COFs. Female restaurant workers had a greater oxidative stress response to COFs than male restaurant workers, providing additional evidence of the link between lung cancer in Chinese women and exposure to COFs. (Cancer Epidemiol Biomarkers Prev 2008;17(12):3351–7)

Introduction

Epidemiologic studies have established that Chinese women who are exposed to cooking oil fumes (COFs) at home suffer from high risks of respiratory diseases (1), lung cancer (2-8), bladder cancer (9), and cervical intraepithelial neoplasm (10). However, studies have not investigated the health effects of exposure to COFs on cooks in Chinese restaurants, who may have potentially high occupational exposure to COFs. Two studies have yielded inconsistent results regarding the mortality of cooks in the United Kingdom. One prospective cohort study showed that cooks who had retired from the British Army suffered excess mortality from lung and large intestinal cancers, ischemic heart disease, cerebrovascular disease, and digestive disease over that of the general population (11). The other retrospective study found that the mortality of cooks by all causes was markedly lower than that of the general population of England and Wales (12).

COFs are produced and released into the environment when food is fried, stir-fried, or grilled using cooking oil at high temperatures. The degradation of sugar, the pyrolysis of proteins and amino acids, and the degradation of fat during the high-temperature treatment of food can generate harmful degradation products (13), such as particulate matter (PM), polycyclic aromatic hydrocarbons (PAH; ref. 2), aromatic amines (9), nitro-PAHs (2, 5), and aldehydes (13, 14). Traditional Chinese cooking includes stir-frying and deep-frying, which generate significant amounts of COFs (15).

Reactive oxygen species in living cells may cause oxidative damage to nucleic acids, proteins, and lipids. Such damage is regarded as being associated with aging, cancer, and other degenerative diseases. 8-Hydroxy-2′-deoxyguanosine (8-OHdG) is the most common DNA lesion that is induced by the reaction of hydroxyl radicals with guanosine at the C-8 position in DNA (16). DNA damage may be repaired by the base excision repair pathway, and the resulting repair product, urinary 8-OHdG, is affected by neither diet nor cell turnover (17). Exposure to COFs promotes the lipid peroxidation of lung epithelial cells according to an *in vitro* study (15). Oxidative stress caused by COFs may be implicated in the development of lung cancer in Chinese women (18). The measurement of urinary 8-OHdG is useful in evaluating risks of lung cancer (19) and other oxidative stress-related diseases (20).

Because the detrimental health effects of COFs are related to long-term exposure, a biological monitoring approach that can reveal an individual's accumulated internal dose is the preferred method for evaluating

Received 1/24/08; revised 8/21/08; accepted 9/19/08.

Grant support: Institute of Occupational Safety and Health, Council of Labor Affairs of the Republic of China, Taiwan, contract IOSH94-M304.

Requests for reprints: Chang-Chuan Chan, Institute of Occupational Medicine and Industrial Hygiene, College of Public Health, National Taiwan University, Room 722, No. 17, Xu-Zhou Road, Taipei, 10020 Taiwan, People's Republic of China. Phone: 886-2-3322-8082; Fax: 886-2-2322-2362. E-mail: ccchan@ntu.edu.tw

doi:10.1158/1055-9965.EPI-08-0075

occupational exposure to COFs. Urinary 1-hydroxypyrene (1-OHP), a metabolite of pyrene (21) and one of the components of PAHs in COFs (22), is a possible biological marker of exposure to COFs. Studies have shown that urinary 1-OHP is a good biological marker of exposure to PAHs in fire-fighting workers (23), iron foundry workers (24), and coke oven workers (25, 26).

This study uses urinary 1-OHP level as an internal dose of COF exposure and 8-OHdG as a DNA oxidative damage marker. It evaluates the relationship between urinary 1-OHP and 8-OHdG and determines whether work in kitchens and the urinary 8-OHdG levels of workers in Chinese restaurants are related to their COF exposures.

Materials and Methods

Study Subjects. Originally, 548 restaurant workers who had been employed for at least 1 year in 23 Chinese restaurants in Taiwan in 2005 were recruited from two trade unions to respond to a questionnaire survey and to undergo a health check. All completed the survey and health check. Because cigarette smoking is a strong confounding factor of COF exposure, only the 387 nonsmokers were used as study subjects. The 23 Chinese restaurants, including both kitchen and dining areas, were designated as nonsmoking areas according to antismoking regulations. Trained interviewers met the participants between July 2005 and December 2005 to collect their demographic information, including age, work experience, main cooking methods, height, weight, number of years of working in kitchens, and health condition as well as information on their life-style, including cooking at home and exposure to secondhand smoke. Cooking at home and secondhand smoke exposure were deemed positive if either occurred on at least 4 days/wk.

Based on job titles obtained from responses to the questionnaire survey, the 387 Chinese restaurant workers (CRW) were classified into two groups. The kitchen staff included chefs, sous chefs, sauce chefs, executive chefs, and assistant cooks, who were relatively close to COFs in the restaurants. The service staff included receptionists, cashiers, waiters, and valets, who were exposed to relatively little COFs in the restaurants. Spot urine samples from the subjects were collected post-work shift during the weekend. All participants were asked to wash their hands before urine collection to prevent contamination. The Institute Review Board of the National Health Research Institutes in Taiwan approved the study. Informed consent was obtained from all subjects.

Exposure Measurement

Particulate PAHs. Daily area monitoring of particulate PAHs was done in the kitchens and dining areas of all 23 Chinese restaurants over 2 consecutive work days. Particulate PAHs in the workplace were sampled using the Institute of Occupational Medicine samplers with glass fiber filters (diameter, 25 mm; pore size, 0.7 μm) at a flow rate of 2.0 L/min. The samplers used in area monitoring were set at a height that was close to that of the breathing zone. Duplicate samples were obtained at each sampling location. Airborne particulate PAHs were monitored in the kitchens and dining areas on 2 consecutive work days, for 12 h on each work day, and then analyzed by high-performance liquid chromatography. Five PAH species, pyrene, benzo(*k*)fluoranthene (BkF), benzo(*a*)pyrene (BaP), benzo(*ghi*)perylene (BghiP), and dibenzo(*a*,*e*)pyrene (DBaeP), were quantified using high-performance liquid chromatography. Each collected sample was extracted using 2 mL *n*-hexane in an ultrasonic bath for 20 min and 4 mL 5% NaOH was added before centrifugation at 3,000 rpm for 20 min. Then, dimethyl sulfate was added to a 1.5 mL suspension solution and condensed under nitrogen gas (N_2). The PAH content of the final solution was determined using a Shimadzu system high-performance liquid chromatography (Japan) with a system controller (SCL-10A) and a fluorescent detector (RF-10AXL) that was equipped with a semi-micro column (Kaseisorb LC ODS-60-5; 4.6 × 250 mm). The mobile phase comprised CH_3CN/H_2O (9:1) solution at a flow rate of 1.0 mL/min. The detection limits were determined by conducting seven repeated analyses of the lowest standards of each PAH species. The coefficient of variation among these repeated analyses was <2% for all five PAHs. The detection limits were 0.28 pg for pyrene, 0.72 pg for BkF, 0.28 pg for BaP, 0.63 pg for BghiP, and 0.43 pg for DBaeP.

Particulate Matter. Continuous-area PM_{10}, $PM_{2.5}$, and $PM_{1.0}$ monitoring was done in each restaurant using laser dust monitors for masses in the range 0 to 100 mg/m^3 (Grimm Model 1.108; Grimm Labortechnik); 1-min mass level, temperature, and humidity were continually obtained. The dust monitors were placed near the PAH samplers in the workplace. The sampling duration and schedule for monitoring PM were the same as those for PAHs.

Urinary 1-OHP and 8-OHdG. Urinary 1-OHP was analyzed using high-performance liquid chromatography with a fluorescence detector. The detection limit was about 0.1 μg/L, based on seven repeated analyses of 1-OHP at 15.0 μg/L, and the variation in the coefficients from the repeated analyses of urinary 1-OHP was <10%. Urinary 8-OHdG level was measured using a high-performance liquid chromatography/MS/MS as has been described elsewhere (27). A detection limit of 5.7 ng/L was obtained using seven repeated analyses of deionized water. The coefficients of variation in interday and intraday tests were <5%. Each individual's urinary 1-OHP and 8-OHDG levels were corrected based on the urine creatinine values, which were determined using an automated method based on the Jaffe reaction (28).

Statistical Methods. Urinary 8-OHdG and 1-OHP levels were first log-transformed to normalize their distributions before the Student's *t* test or regression analysis was done. Student's *t* and χ^2 statistics were used to compare the personal covariates, which were the urinary 8-OHdG and 1-OHP of kitchen and service staff. Nonparametric Mann-Whitney *U* tests were conducted to compare the workplace PM_{10}, $PM_{2.5}$, $PM_{1.0}$, and PAH levels of the kitchen and service staff. Spearman correlation analysis was adopted to evaluate the correlation between pyrene levels and levels of other PAHs in kitchens and dining areas.

All data from 387 nonsmoking restaurant workers were then included in linear mixed-effects models to identify significant predictors of workers' urinary 8-OHdG and 1-OHP levels. The subjects' gender, age, body mass index (BMI), work experience, whether they

Table 1. Descriptive statistics for 387 workers by job title in 23 Chinese restaurants

	Service staff (n = 185)	Kitchen staff (n = 202)	P
Personal characteristics, mean ± SD			
Age (y)	40.4 ± 12.7	42.5 ± 9.7	0.062
Height (cm)	159.7 ± 7.8	160.0 ± 7.9	0.670
Weight (kg)	62.4 ± 11.4	64.8 ± 12.0	0.044
BMI (kg/m^2)	24.4 ± 4.0	25.2 ± 4.0	0.042
Work experience, mean ± SD			
Working years	10.8 ± 10.7	13.4 ± 9.2	0.009
Work days per week	5.2 ± 0.4	5.3 ± 0.4	0.094
Work hours per day	8.3 ± 1.7	8.9 ± 2.5	0.005
Cooking hours at work per day	0.0 ± 0.0	4.2 ± 2.2	—
Gender, n (%)			
Male	61 (33.0)	93 (46.8)	0.009
Female	124 (67.0)	109 (53.2)	
Cooking at home*	24 (13.0)	103 (51.0)	<0.001
Secondhand smoke exposure†	92 (49.7)	102 (50.5)	0.880

*Cooking at home at least 4 days/wk.
† Secondhand smoke exposure at least 4 days/wk.

cooked at home, and whether they were exposed to secondhand smoking were treated as fixed effects, and each restaurant was treated as a random effect in the data analysis. The level for statistical significance was set to $\alpha = 0.05$ in all tests. All data analyses were done using the S-PLUS 2000 program (MathSoft).

Results

Table 1 provides descriptive statistics of 387 restaurant workers by job title. Their weight, BMI, years worked, work hours per day, gender distribution, and cooking-at-home status differed significantly between service staff and kitchen staff. In contrast, age, height, hours worked per day, and exposure to secondhand smoke did not differ significantly between these groups.

Table 2 presents the levels of PM and particulate PAHs in the kitchens and dining areas of 23 Chinese restaurants. PM and PAHs were monitored in 23 kitchens and 23 dining areas. Each measurement was an average over duplicate samples collected at each location on 2 consecutive work days. Because the samples were few, nonparametric Mann-Whitney U tests were used to compare the PM and PAHs between the kitchens and dining areas. The median levels of PM_{10}, $PM_{2.5}$, $PM_{1.0}$, pyrene, BkF, BaP, BghiP, and DBaeP in the kitchens significantly exceeded those in the dining areas. The median total level of PAHs in the kitchens also significantly exceeded that in the dining areas.

Urinary 1-OHP is a metabolite of pyrene, which is just one of the many chemicals in COFs. Accordingly, the extent to which pyrene correlates with the other COF-related chemicals is important given these other chemicals are likely causes of most of the oxidative stress. Therefore, a Spearman correlation analysis was used to investigate correlations between average pyrene levels and average levels of other PAHs. Pyrene levels were significantly correlated with BkF, BaP, BghiP, and total PAHs in both kitchens and dining areas as shown in Table 3.

Table 4 compares urinary levels of 8-OHdG and 1-OHP by job title and gender using Student's t test. Overall, the geometric mean 8-OHdG and 1-OHP levels of kitchen staff significantly exceeded those of service staff. This difference was significant for male CRWs. Only 8-OHdG levels differed significantly between the two locations for female CRWs. A significant gender difference existed in the 1-OHP levels of the service staff: females had significantly higher levels than males (P = 0.009). No gender difference was found for 1-OHP and 8-OHdG in kitchen staff or for 8-OHdG in service staff. This result is consistent with a previous study of nonoccupationally exposed subjects (29). It also agrees an

Table 2. Comparisons of PM and PAHs levels in kitchens and dining areas in 23 Chinese restaurants

	Dining areas (n = 23)		Kitchens (n = 23)		P*
	Median	Geometric mean (geometric SD)	Median	Geometric mean (geometric SD)	
PM_{10} (μg/m^3)	30.3	34.4 (1.8)	74.9	81.3 (1.8)	<0.001
$PM_{2.5}$ (μg/m^3)	24.5	26.6 (1.8)	56.9	58.8 (1.6)	<0.001
$PM_{1.0}$ (μg/m^3)	21.8	23.3 (1.8)	41.4	44.2 (1.6)	<0.001
Pyrene (ng/m^3)	0.3	0.4 (3.4)	2.9	2.7 (4.7)	<0.001
BkF (ng/m^3)	0.3	0.2 (2.6)	1.5	1.5 (4.0)	<0.001
BaP (ng/m^3)	1.3	1.1 (2.7)	6.2	6.9 (4.2)	<0.001
BghiP (ng/m^3)	1.1	0.6 (3.8)	5.5	5.6 (3.9)	<0.001
DBaeP (ng/m^3)	0.4	0.6 (6.4)	1.1	2.3 (6.3)	0.001
Summed PAHs† (ng/m^3)	4.8	4.5 (2.6)	24.8	28.0 (3.4)	0.001

*Mann-Whitney U tests.
† Summed PAHs: Sum of pyrene, BkF, BaP, BghiP, and DBaeP.

Table 3. Correlation coefficients of pyrene and other PAHs in work locations of 23 Chinese restaurants

	Pyrene		
	Dining areas (n = 23)	Kitchens (n = 23)	Kitchens and dining areas (n = 46)
BkF	0.385*	0.855*	0.860*
BaP	0.533*	0.399*	0.421*
BghiP	0.295*	0.270*	0.293*
DBaeP	0.344	0.012	0.047
Summed PAHs †	0.572*	0.495*	0.517*

*$P < 0.05$.
† Summed PAHs: Sum of pyrene, BkF, BaP, BghiP, and DBaeP.

earlier study, which found that urinary 1-OHP level was significantly higher in female police officers than male police officers (30).

Exactly how urinary 8-OHdG and 1-OHP levels are related was evaluated using the Spearman correlation analysis as shown in Fig. 1. Individual urinary 8-OHdG levels were positively related to individual urinary 1-OHP levels: greater urinary excretion of 1-OHP was associated with greater urinary excretion of 8-OHdG (Spearman correlation coefficient $r = 0.329$; $P < 0.001$; $n = 387$).

Table 5 presents the results of linear mixed-effects regression models for predictors of urinary 1-OHP and 8-OHdG levels in restaurant workers. Kitchen work and gender were significant and positive predictors of urinary 1-OHP levels after adjustments were made for other covariates. Kitchen staff had higher urinary 1-OHP levels than service staff. The mean urinary 1-OHP levels of female CRWs significantly exceeded those of male CRWs ($P < 0.05$). However, cooking at home, exposure to secondhand smoke, number of hours worked per day, and other personal covariates were not significant predictors of urinary 1-OHP. However, urinary 1-OHP, work in kitchens, gender, and number of hours worked per day were significant predictors of urinary 8-OHdG levels after adjustments were made for other covariates. The increase in urinary 1-OHP was significantly related to the increase in urinary 8-OHdG ($P < 0.001$). The urinary 8-OHdG levels of the kitchen staff significantly exceeded those of the service staff ($P < 0.05$). The mean urinary 8-OHdG levels of female CRWs were also significantly higher than those of male CRWs ($P < 0.05$). Work in kitchens still had an effect after controlling for the effect of urinary 1-OHP in our models. However, cooking at home, exposure to secondhand smoke, number of years worked, number of days worked per week, age, and BMI were not significant predictors of urinary 8-OHdG levels in the restaurant workers.

Discussion

The above results show that ambient levels of total PAHs were closely correlated with pyrene levels inside restaurants. Both Student's t tests and linear mixed-effects models indicated that urinary 1-OHP levels differed significantly between CRWs with different exposures to COFs. These findings reveal that 1-OHP is a suitable internal dose of exposure to COFs among CRWs. Notably, cooking at home, exposure to secondhand smoke, number of years worked, number of work days per week, number of work hours per day, age, and BMI were not significant predictors of urinary 1-OHP in this study ($P > 0.1$). This finding is consistent with a previous study of urinary 1-OHP in male restaurant workers (31).

Cooking at home is not a significant predictor of urinary 8-OHdG levels of CRWs. Cooking methods that are commonly adopted in restaurant kitchens include deep-frying, stir-frying, and grilling, all of which generate significant quantities of COFs (15, 32). In contrast, the main cooking methods used by restaurant workers at home were steaming and stewing, neither of which generate significant amounts of COFs. These restaurant workers do not deep-fry, stir-fry, or grill foods at home because they are aware of the hazards of COFs as a result of having attended government training programs and thus are exposed to few or no COFs. However, the fact that cooking at home takes a few minutes while cooking at work continues for hours may also explain why home cooking is not a significant predictor of urinary 8-OHdG levels. The likelihood of exposure to secondhand smoke was small, because both the kitchen and the dining areas of all of the 23 surveyed

Table 4. Comparisons by job title and gender of urinary 8-OHdG and 1-OHP levels (μg/g creatinine) in 387 kitchen and service staff at Chinese restaurants

Marker	Gender	Service staff		Kitchen staff		P*
		Geometric mean (geometric SD)	n	Geometric mean (geometric SD)	n	
8-OHdG	Male (n = 154)	4.7 (2.0)	61	7.0 (2.0)	93	0.001
	Female (n = 233)	6.0 (2.2)	124	8.6 (2.2)	109	<0.001
	All (n = 387)	5.5 (2.2)	185	7.9 (2.1)	202	<0.001
1-OHP	Male (n = 154)	1.8 (4.2)	61	4.1 (4.6)	93	0.002
	Female (n = 233)	3.4 (4.9)	124	4.9 (5.1)	109	0.150
	All (n = 387)	2.7 (4.9)	185	4.5 (4.7)	202	0.005

*Student's t test.

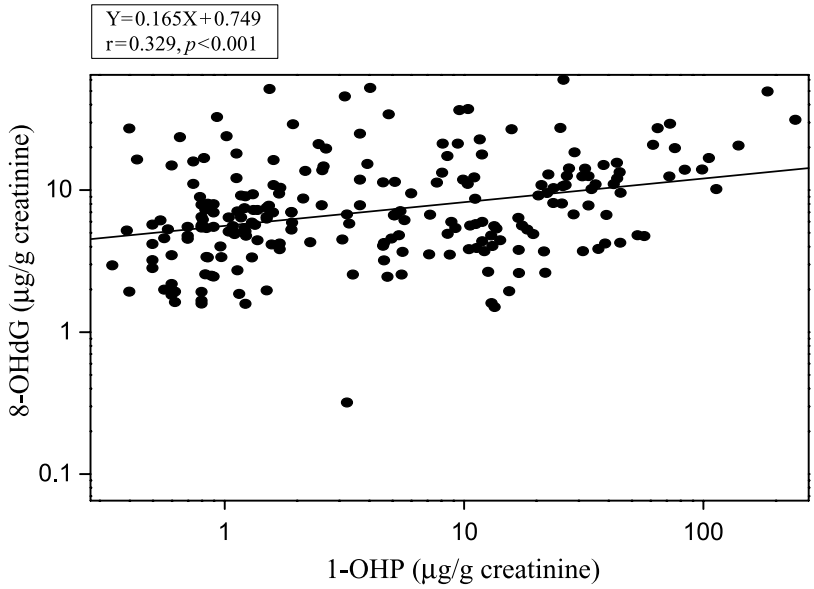

Figure 1. Relationship between urinary excretion of 8-OHdG and 1-OHP among 387 CRWs.

restaurants were designated nonsmoking areas. Restaurant workers who smoked thus typically did so outdoors or in designated smoking areas. Therefore, secondhand smoke exposure did not significantly affect urinary 8-OHdG levels. This finding is consistent with a previous longitudinal study of urinary 8-OHdG levels in 68 healthy adults (33).

Urinary 1-OHP, work in kitchens, number of hours worked per day, and gender were four major predictors of urinary 8-OHdG levels in CRWs. Urinary 1-OHP and work in kitchens were good indicators of exposure to COFs for predicting oxidative stress in workers at Chinese restaurants. The number of hours worked per day was a predictor of urinary 8-OHdG, revealing that urinary 8-OHdG is a short-term biomarker. One study showed that the half-life of the induced 8-OHdG was about 6 h (34). The use of work in kitchens as an independent predictor of 8-OHdG in linear mixed-effects regression models reveals that kitchen staff may be exposed to other unmeasured hazards, such as aldehydes (35). This finding is consistent with an earlier study, which found that exposure to COFs, PAH carcinogens, and heterocyclic amines may contribute to oxidative stress (18). Our findings suggest that kitchen staff are more likely to have oxidative stress than service area workers, which can be attributed to factors other than their urinary 1-OHP levels.

Some studies have shown an inverse relationship between urinary 8-OHdG levels and both age and BMI possibly because older or leaner individuals have higher metabolic rates than younger or obese individuals (36, 37). However, this study did not determine that age or BMI influenced urinary 8-OHdG levels. The results herein are consistent with an earlier study of firefighters (38). Another interesting finding is that female CRWs have higher excretion of urinary 1-OHP and 8-OHdG than male CRWs, which may reflect gender-related differences in the metabolism of PAHs mediated by endogenous mechanisms (30, 39, 40).

This study has some limitations. First, other unmeasured data concerning COFs, such as levels of aromatic amines (9), nitro-PAHs (2, 5), and aldehydes (35), were

Table 5. Linear mixed-effects regression analysis: predictors of urinary 1-OHP and 8-OHdG in 387 nonsmoking restaurant workers

Predictors	Log_{10} 1-OHP (μg/g creatinine), regression coefficient (95% confidence interval)	Log_{10} 8-OHdG (μg/g creatinine), regression coefficient (95% confidence interval)
Work in kitchens (kitchen vs service staff)	0.397 (0.109-0.685)*	0.137 (0.063-0.212)*
Gender (female vs male)	0.215 (0.038-0.392)*	0.082 (0.007-0.157)*
Cooking at home (yes vs no)	0.165 (-0.016 to 0.346)	0.039 (-0.006 to 0.085)
Secondhand smoke exposure (yes vs no)	0.091 (-0.071 to 0.254)	0.008 (-0.059 to 0.075)
Work years (y)	0.007 (-0.002 to 0.017)	<0.001 (-0.004 to 0.004)
Work days per week (d)	0.129 (-0.138 to 0.397)	0.038 (-0.067 to 0.143)
Work hours per day (h)	0.001 (-0.035 to 0.036)	0.021 (0.006-0.035)*
Age (y)	-0.003 (-0.011 to 0.005)	0.004 (-0.001 to 0.008)
BMI (kg/m^2)	0.019 (-0.001 to 0.039)	0.006 (-0.003 to 0.014)
Log_{10} 1-OHP (μg/g creatinine)	—	0.110 (0.068-0.152)*

*$P < 0.05$.

lacking, possibly confounding the results concerning oxidative stress. The other limitation was the lack of data on the exposure to PAHs outside occupational settings, such as from vehicle traffic emissions and through cooking at home. However, the restaurant workers herein spent >10 h/d at the restaurants, including work and rest periods, but <1 h/d in traffic. The contribution of traffic sources to the PAH exposure of restaurant workers is thus assumed to be limited. Regardless of such limitations, this study concludes that urinary 8-OHdG is a good biomarker of oxidative DNA damage for CRWs because it reflects the internal dose of COFs (urinary 1-OHP), a proxy indicator of COF exposure (work location), and cumulative COF exposure (period of work experience as CRWs).

An earlier pathologic study (41) determined that Chinese women are inclined to contract adenocarcinoma, rather than squamous cell carcinoma, indicating that lung cancer in such women may not be associated with smoking (3). Some epidemiologic studies have shown that the contamination of indoor air from cooking sources may be a risk factor for lung cancer in Chinese women. One study established that nonsmoking women had an 8.3 times higher risk of contracting lung cancer due to exposure to COFs (42). The finding in this study that female restaurant workers had a greater oxidative stress response to COFs than male restaurant workers is additional evidence of the link between lung cancer in Chinese women and exposure to COFs. The results of this study show that CRWs are a group at high risk of exposure to COFs, and further study is required to clarify the health effect.

Disclosure of Potential Conflicts of Interest

No potential conflicts of interest were disclosed.

Acknowledgments

The costs of publication of this article were defrayed in part by the payment of page charges. This article must therefore be hereby marked *advertisement* in accordance with 18 U.S.C. Section 1734 solely to indicate this fact.

References

1. Svendsen K, Sjaastad AK, Siverstsen I. Respiratory symptoms in kitchen workers. Am J Ind Med 2003;43:436–9.
2. Li S, Pan D, Wang D. Analysis of polycyclic aromatic hydrocarbons in cooking oil fumes. Arch Environ Health 1994;49:119–22.
3. Ko YC, Cheng LS, Lee CH, et al. Chinese food cooking and lung cancer in women nonsmokers. Am J Epidemiol 2000;151:140–7.
4. Le CH, Ko YC, Cheng LS. The heterogeneity in risk factors of lung cancer and the difference of histologic distribution between genders in Taiwan. Cancer Causes Control 2001;12:289–300.
5. Wu PF, Chiang TA, Wang LF, Chang CS, Ko YC. Nitro-polycyclic aromatic hydrocarbon contents of fumes from heated cooking oils and prevention of mutagenicity by catechin. Mutat Res 1998;403:29–34.
6. Yang SC, Jenq SN, Kang ZC, Lee H. Identification of benzo[*a*]pyrene 7,8-diol 9,10-epoxide N2-deoxyguanosine in human lung adenocarcinoma cells exposed to cooking oil fumes from frying fish under domestic conditions. Chem Res Toxicol 2000;13:1046–50.
7. Behera D, Balamugesh T. Dose-response relationship between cooking fumes exposures and lung cancer among Chinese nonsmoking women. Cancer Res 2006;66:4961–7.
8. Wu-Williams AH, Dai XD, Blot W, et al. Lung cancer among women in north-east China. Br J Cancer 1990;62:982–7.
9. Chiang TA, Pei-Fen W, Ying LS, Wang LF, Ko YC. Mutagenicity and aromatic amine content of fumes from heated cooking oils produced in Taiwan. Food Chem Toxicol 1999;37:125–34.
10. Wu MT, Lee LH, Ho CK, Wu SC, Lin LY, Cheng BH. Environmental exposure to cooking oil fumes and cervical intraepithelial neoplasm. Environ Res 2004;94:25–32.
11. Coggon D, Wield G. Mortality of army cooks. Scand J Work Environ Health 1993;19:85–8.
12. Coggon D, Wield G. Mortality of butchers and cooks identified from the 1961 census of England and Wales. Occup Environ Med 1995;52:157–9.
13. Vainiotalo S, Matveinen K. Cooking fumes as a hygienic problem in the food and catering industries. Am Ind Hyg Assoc J 1993;54:376–82.
14. Lund KH, Petersen JH. Migration of formaldehyde and melamine monomers from kitchen- and tableware made of melamine plastic. Food Addit Contam 2006;23:948–55.
15. Tung YH, Ko JL, Liang YF, Yin L, Pu Y, Lin P. Cooking oil fume-induced cytokine expression and oxidative stress in human lung epithelial cells. Environ Res 2001;87:47–54.
16. Kasai H, Crain PF, Kuchino Y, Nishimura S, Ootsuyama A, Tanooka H. Formation of 8-hydroxyguanine moiety in cellular DNA by agents producing oxygen radicals and evidence for its repair. Carcinogenesis 1986;7:1849–51.
17. Cooke MS, Evans MD, Dove R, et al. DNA repair is responsible for the presence of oxidatively damaged DNA lesions in urine. Mutat Res 2005;574:58–66.
18. Cherng SH, Huang KH, Yang SC, et al. Human 8-oxoguanine DNA glycosylase 1 mRNA expression as an oxidative stress exposure biomarker of cooking oil fumes. J Toxicol Environ Health A 2002;65:265–78.
19. Erhola M, Toyokuni S, Okada K, et al. Biomarker evidence of DNA oxidation in lung cancer patients: association of urinary 8-hydroxy-2′-deoxyguanosine excretion with radiotherapy, chemotherapy, and response to treatment. FEBS Lett 1997;409:287–91.
20. Wu LL, Chiou CC, Chang PY, Wu JT. Urinary 8-OHdG: a marker of oxidative stress to DNA and a risk factor for cancer, atherosclerosis and diabetics. Clin Chim Acta 2004;33:1–9.
21. Huang W, Smith TJ, Ngo L, et al. Characterizing and biological monitoring of polycyclic aromatic hydrocarbons in exposures to diesel exhaust. Environ Sci Technol 2007;41:2711–6.
22. Wei See S, Karthikeyan S, Balasubramanian R. Health risk assessment of occupational exposure to particulate-phase polycyclic aromatic hydrocarbons associated with Chinese, Malay and Indian cooking. J Environ Monit 2006;8:369–76.
23. Caux C, O'Brien C, Viau C. Determination of firefighter exposure to polycyclic aromatic hydrocarbons and benzene during fire fighting using measurement of biological indicators. Appl Occup Environ Hyg 2002;17:379–86.
24. Hansen AM, Omland O, Poulsen OM, et al. Correlation between work process-related exposure to polycyclic aromatic hydrocarbons and urinary levels of α-naphthol β- naphthylamine and 1-hydroxypyrene in iron foundry workers. Int Arch Occup Environ Health 1994;65:385–94.
25. Lin YC, Pan CH, Chen CJ, et al. Associations between exposure to polycyclic aromatic hydrocarbons and temporal change of urinary 1-hydroxypyrene levels in Taiwanese coke-oven workers. J Occup Environ Med 2006;48:930–6.
26. Wu MT, Pan CH, Huang YL, et al. Urinary excretion of 8-hydroxy-2-deoxyguanosine and 1-hydroxypyrene in coke-oven workers. Environ Mol Mutagen 2003;42:98–105.
27. Hu CW, Wang CJ, Chang LW, Chao MR. Clinical-scale high-throughput analysis of urinary 8-oxo-7,8-dihydro-2′-deoxyguanosine by isotope-dilution liquid chromatography-tandem mass spectrometry with on-line solid phase extraction. Clin Chem 2006;52:1381–8.
28. Huang YL, Chuang IC, Pan CH, et al. Determination of chromium in whole blood and urine by graphite furnace AAS. At Spectrosc 2000;21:10–6.
29. Chen B, Hu Y, Jin T, et al. Higher urinary 1-hydroxypyrene concentration is associated with cooking practice in a Chinese population. Toxicol Lett 2007;171:119–25.
30. Merlo F, Andreassen A, Weston A, et al. Urinary excretion of 1-hydroxypyrene as a marker for exposure to urban air levels of polycyclic aromatic hydrocarbons. Cancer Epidemiol Biomarkers Prev 1998;7:147–55.
31. Pan CH, Chan CC, Huang YL, Wu, KY. Urinary 1-hydroxypyrene and malondialdehyde in male workers in Chinese restaurants. Occup Environ Med 2008;65:732–5.
32. Reinik M, Tamme T, Roasto M, et al. Polycyclic aromatic hydrocarbons (PAHs) in meat products and estimated PAH intake by children and the general population in Estonia. Food Addit Contam 2007;24:429–37.

33. Pilger A, Germadnik D, Riedel K, Meger-Kossien I, Scherer G, Rüdiger HW. Longitudinal study of urinary 8-hydroxy-2′-deoxyguanosine excretion in healthy adults. Free Radic Res 2001;35:273–80.
34. Takeuchi T, Matsugo S, Morimoto K. Mutagenicity of oxidative DNA damage in Chinese hamster V79 cells. Carcinogenesis 1997; 18:2051–5.
35. Trush MA, Kensler TW. An overview of the relationship between oxidative stress and chemical carcinogenesis. Free Radic Biol Med 1991;10:201–9.
36. Loft S, Vistisen K, Ewertz M, Tjønneland A, Overvad K, Poulsen HE. Oxidative DNA damage estimated by 8-hydroxydeoxyguanosine excretion in humans: influence of smoking, gender and body mass index. Carcinogenesis 1992;13:2241–7.
37. Fischer-Nielsen A, Poulsen HE, Loft S. 8-Hydroxydeoxyguanosine *in vitro*: effects of glutathione, ascorbate, and 5-aminosalicylic acid. Free Radic Biol Med 1992;13:121–6.
38. Hong YC, Park HS, Ha EH. Influence of genetic susceptibility on the urinary excretion of 8-hydroxydeoxyguanosine of firefighters. Occup Environ Med 2000;57:370–5.
39. Kure EH, Ryberg D, Hewer A, et al. p53 mutations in lung tumours: relationship to gender and lung DNA adduct levels. Carcinogenesis 1996;17:2201–5.
40. Ryberg D, Hewer A, Phillips DH, Haugen A. Different susceptibility to smoking-induced DNA damage among male and female lung cancer patients. Cancer Res 1994;54:5801–3.
41. Law CH, Day NE, Shanmugaratnam K. Incidence rates of specific histological types of lung cancer in Singapore Chinese dialect groups, and their aetiological significance. Int J Cancer 1976;17: 304–9.
42. Ko YC, Lee CH, Chen MJ, et al. Risk factors for primary lung cancer among non-smoking women in Taiwan. Int J Epidemiol 1997;26:24–31.