

論文

以工程介入降低中式餐廳勞工之烹飪油煙暴露

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摘要

本研究嘗試應用新的工程介入方法—包圍型氣簾式導煙機來增進抽油煙機捕集效率，以降低中式餐廳勞工之烹飪油煙暴露。研究針對廚師抱怨過度暴露烹飪油煙的六家中式餐廳進行工程介入，比較裝設包圍型氣簾式導煙機前後之廚房作業環境空氣中懸浮微粒 (Particulate Matter, PM) 與多環芳香烴化合物 (Polycyclic Aromatic Hydrocarbons, PAHs) 濃度、廚師尿液中8-羥基-2-去氧鳥嘌呤核甘 (8-hydroxy-2'-deoxyguanosine, 8-OHdG) 與丙二醛 (Malondialdehyde, MDA) 濃度，並以線性混合效應迴歸模式評估廚師尿液中8-OHdG、MDA與作業環境空氣中PM、PAHs的相關性。

在裝設包圍型氣簾式導煙機進行烹飪油煙工程介入後，6家中式餐廳廚房在作業環境空氣中PM₁₀，PM_{2.5}，PM_{1.0}，與粒狀PAHs的幾何平均濃度皆顯著降低；廚師尿液中8-OHdG、MDA的濃度亦皆顯著降低。在校正干擾因子後，作業環境空氣中PM_{2.5}，PM_{1.0}，benzo(a)pyrene(BaP)與廚師尿液中8-OHdG濃度分別呈顯著正相關，且作業環境空氣中BaP與廚師尿液中MDA濃度亦呈顯著正相關。裝設包圍型氣簾式導煙機可有效地降低中式餐廳廚房工作人員之烹飪油煙暴露與氧化傷害的程度。

關鍵字：烹飪油煙、氣簾、尿液中8-羥基-2-去氧鳥嘌呤核甘、尿液中丙二醛、氧化傷害、餐館業勞工

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Research Articles

Reduction of Cooking Oil Fume Exposure by Engineering Intervention in Chinese Restaurants

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Abstract

This study used a new engineering intervention measure, embracing air curtain device, to increase the capture efficiency of kitchen range hoods and reduce COF exposure for Chinese restaurant workers. The embracing air curtain devices (EACD) were installed in six Chinese restaurants in which the cooks complained of COF exposure. Before- and after- installation measurements were taken to compare changes in particulate matter (PM) and polycyclic aromatic hydrocarbons (PAHs) in kitchen air, and in levels of urinary 8-hydroxy-2'-deoxyguanosine (8-OHdG) and malondialdehyde (MDA). The association of PM and PAHs in air and 8-OHdG and MDA in urine was evaluated by linear mixed-effects regression analysis. The results for the six restaurants studied showed that geometric mean kitchen air levels of PM₁₀, PM_{2.5}, PM_{1.0}, and total particulate PAHs were significantly reduced after the engineering intervention was implemented with the EACD. Urinary levels of 8-OHdG and MDA in cooks were significantly lower after the EACD installment. PM_{2.5}, PM_{1.0}, and benzo(a)pyrene (BaP) levels were positively associated with urinary 8-OHdG levels after adjusting for key personal covariates. Furthermore, urinary MDA levels in cooks were also positively associated with BaP levels. This study demonstrates that the EACD is effective for reducing COF and oxidative stress levels in cooks working in Chinese kitchens.

Keywords: Cooking oil fumes, Health risks, Air curtain, Urinary 8-hydroxy-2'-deoxyguanosine, Urinary malondialdehyde, Oxidative stress, Restaurant workers

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Introduction

A previous study demonstrated that the cooking process was the main influence on the total mutagenic activity of indoor air [1]. Cooking oil fumes (COF) are created and released into the environment when food is fried, stir-fried or grilled using cooking oil at high temperatures. Cooks are occupationally exposed to COF. Particulate matter (PM) and polycyclic aromatic hydrocarbons (PAHs) [2] are important components of COF. Airborne PM are associated with cardiopulmonary mortality and morbidity [3], oxidative stress [4-5], and inflammation response in humans [6]. PAHs are related to lung and bladder cancers [7]. Therefore, effective measures are necessary to reduce human exposure to COF. Exterior hoods are well known for having poor capture efficiency, which means airborne contaminants can easily reach the breathing zone of workers. Since kitchen range hoods in Chinese restaurants are exterior hoods, increasing capture efficiency of kitchen range hoods and reducing levels of PM and PAHs in worker breathing zones becomes critically important.

Previous studies indicated that urinary 8-hydroxy-2'-deoxyguanosine (8-OHdG) [8] and malondialdehyde (MDA) [9] were two useful biomarkers for assessing oxidative stress induced by COF exposure. Another two previous studies revealed that the mutagenicities of COF were significantly reduced by adding the antioxidant catechin to cooking oils before heating [10-11].

Air curtain is a jet air that is applied to air conditioner, fire safety system, food industries, and indoor air quality [12]. However, we are unaware

of any previous study evaluating the influence of oxidative stress for cooks by reducing COF exposure via air curtain. This study attempted to use a new engineering intervention measure, embracing air curtain device (EACD, commercial name: smoke guider, Acxing Industrial Co., Ltd., Taipei, Taiwan), to increase the capture efficiency of kitchen range hoods and reduce COF exposure for Chinese restaurant workers. The effects of the EACD on reduction of COF were evaluated by air monitoring of particulate matter and particulate PAHs, and biological monitoring of urinary 8-OHdG, and MDA in cooks.

METHODS

Study site

This study selected six Chinese restaurants that each average inlet velocity on opening of kitchen range hood was less than 0.4 m/sec in the Taipei area of Taiwan. The cooks in each restaurant complained about the hazards of COF and their discomfort on exposure to COF.

Engineering intervention

The criteria of engineering intervention by the EACD were fitted good work practices. Meanwhile, the good work practices included: 1. the control space of EACD should cover the COF emission source; 2. the dip angle of the EACD should exceed zero to induce COF into the opening of the kitchen hood; 3. the diffusion area of EACD should be fully included in the opening of the kitchen hood. From Fig. 1, the three-piece U-type EACD[13] was introduced to these kitchens to improve kitchen

hood capture efficiency. The EACD comprised air curtain generators (blowing devices), including a control panels, and a cast-iron protection cover that encloses motors and air curtain generators. The EACD can be applied to any type and any size of kitchen range hood. The original kitchen range hoods were used as the air induction hoods. This study set the maximum flow velocity (2.6 m/sec) of the EACD to induce the COF into the kitchen range hoods. To evaluate the efficiency of the EACD, this study compared differences for PM and PAHs in air, urinary 8-OHdG, and MDA in cooks between the situations before and after these devices were applied.

Exposure assessment

Personal air monitoring and biological monitoring were used to evaluate the EACD efficiency before and after engineering intervention. Smoking was not allowed in the kitchens and dining areas because anti-smoking regulations designated all restaurants as non-smoking areas. The Institute Review Board of the National Health Research Institutes in Taiwan approved this study. All subjects provided informed consent.

Air monitoring

Air monitoring for particulate matter and particulate PAHs was monitored during the weekend (Saturday) before and four weeks after engineering intervention devices were set up.

Particulate matter

PM₁₀, PM_{2.5} and PM_{1.0} were monitored continuously for each study subject using a Dust-

Check Portable Dust Monitor (Grimm Model 1.108, Grimm Labortechnik Ltd., Ainring, Germany), and 1-minute mass concentration, temperature, and humidity were continuously reported. A dust monitor was placed near the breathing zone of each subject and worn throughout the workday. Collocated Rupprecht and Patashnick 1400a tapered element oscillating microbalance (TEOM) samplers (Thermo Electron Corporation, East Greenbush, NY, USA) were used to calibrate the mass concentrations of PM₁₀, PM_{2.5} and PM_{1.0} measured using the DUST-check monitor in a previous study. Concurrent PM measurements indicated a strong association between the data collected by these two monitors for all three size fractions-PM₁₀ ($r^2=0.91$), PM_{2.5} ($r^2=0.90$) and PM_{1.0} ($r^2=0.80$).

Particulate PAHs

This study collected particulate PAHs samples using personal monitoring for 45 cooks in kitchens of six Chinese restaurants. Particulate PAHs in the workplace were collected using IOM (Institute of Occupational Medicine, England) samplers with glass fiber filters (diameter: 25mm, pore size: 0.7 μ m) at a flow rate of 2.0 L/min. The samplers were placed near the breathing zone of the workers. Personal particulate PAHs measurements were summarized for further statistical analysis. Five PAH species, including pyrene, benzo(k)fluoranthene (BkF), benzo(a)pyrene (BaP), benzo(ghi)perylene (Bghip), and dibenzo(a,e)pyrene (DBaP), were quantified using high performance liquid chromatograph (HPLC). The coefficient variation for these repeated analyses was below 2% for all 5 PAHs. Meanwhile, 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 DBaP, respectively.

Biological monitoring

Urinary 8-OHdG and MDA

Before engineering intervention, urine samples were collected for all participants after they finished their shift on a Saturday night. Then they have worked for six consecutive days from Monday through Saturday. Four weeks after engineering intervention of EACD was established, again, urine samples were once again collected for all participants when they completed their shift on a Saturday night after having worked for six consecutive days from Monday through Saturday. All participants were asked to wash their hands prior to urine collection to avoid environmental contamination. Urinary 8-OHdG and MDA levels for each individual were corrected according to urine creatinine values, which were determined by using an automated method based on the Jaffe reaction [14]. Urinary 8-OHdG was analyzed via HPLC/MS/MS with the use of an isotope-labeled coeluting internal standard which has been described previously [15]. A detection limit of 5.7 ng/L was obtained from seven repeated analyses of deionized water. The coefficients of variation were below 5% for the interday and intraday tests.

Urinary MDA concentration was measured using an HPLC (JASCO Model 980-PU, Toyoko, Japan) with a C18 column and an ultraviolet-visible detector at 532 nm (JASCO UV-975, Toyoko, Japan). The mobile phase was methanol/potassium phosphate (9:11) buffer and the flow rate was 1.2

ml/min. The within-run and run-to-run precisions of MDA in urine were evaluated. The samples were analyzed for MDA based on thiobarbituric acid (TBA) reaction, with HPLC separation of the MDA (TBA)₂ adduct, using tetraethoxypropane as a standard. A detection limit of 0.06 µg/L was obtained from seven repeated analyses of deionized water, and the variation in the coefficients of repeated analyses was below 10%.

Questionnaire survey

The study population consisted of cooks working at six Chinese restaurants. The subjects were monitored during the weekends before and after engineering intervention devices were set up. All 58 eligible cooks completed the questionnaire survey but only 45 completed the questionnaire survey, air monitoring and biological monitoring. The reason for the non-participation of 13 workers was their not working during the study weekend. Trained interviewers met with the participants between January 2006 and April 2006 and conducted a questionnaire survey to gather participant demographic data, including age, years spent doing restaurant work, height, weight, educational level, health condition and life style - including cigarette smoking. Cigarette smokers were defined as smoking cigarettes at least four days weekly for at least 6 months. This study also gathered data on main cooking methods, cooking oil type, cooking time, food oil consumption rate, kitchen volume, and inflow velocity of kitchen hood openings in these six Chinese restaurants.

Statistical methods

Student pair t statistics were used to compare personal PM₁₀, PM_{2.5} and PM_{1.0}, PAHs levels,

urinary 8-OHdG, and MDA levels between the before and after engineering intervention situations. The concentrations of PM, PAHs, urinary 8-OHdG, and MDA levels and other personal covariates belonging to abnormal distributions were first log-transformed to normalize their distributions before conducting Student paired t analysis for the 45 cooks in the six Chinese restaurants.

The end of week measurement differences of pre-intervention and post-intervention levels in urinary 8-OHdG or MDA were calculated by subtracting the concentration before engineering intervention from that after engineering intervention. Furthermore, the end of week measurement differences of pre-intervention and post-intervention levels in PM or particulate PAHs were calculated by subtracting the concentration before engineering intervention from that after engineering intervention. This study then used linear mixed-effects regressions models to investigate the effect of PM and particulate PAHs on urinary 8-OHdG and MDA concentrations, after adjusting for age and BMI, respectively. Subject age and cigarette smoking were treated as fixed effects, and each restaurant was treated as a random effect in the data analysis. The level of statistical significance was set to $\alpha=0.05$ in all tests. All data analyses were performed using the S-PLUS 2000 program (MathSoft Inc., Cambridge, MA, USA).

RESULTS

Table 1 lists the background information and characteristics of 45 male cooks in the six Chinese restaurants. The main cooking methods included stir frying, deep frying, steaming, stewing, and

roasting. All cooks did all types of cooking. The most frequently used type of cooking was stir fry, the second was deep fry, the third was stew, the fourth was steaming, and the last was roast in these Chinese restaurants. Meanwhile, the main type of cooking oil was soybean oil. Mean cooking time per day was 6.3 ± 0.6 hours. Furthermore, the average consumption rate of food oil was 14.1 ± 7.2 L/day. The mean inflow velocity on the opening of hoods in kitchens was 0.28 ± 0.05 m/sec. Furthermore, kitchen volume ranged from 47.3 to 437.0 m³. The study population consisted of 45 male cook. Their ages ranged from 15 to 56 years, with a mean \pm SD of 34.0 ± 10.6 years. On average, the subjects had 13.6 ± 9.2 years of cooking experience. Twenty of the 45 subjects (44.4%) were current cigarette smokers. No demographic characteristics significantly differed between the 45 participants and the 13 non-participants ($p > 0.1$).

Figure 1 illustrates the U-type EACD with three-piece air curtain device. The air curtain devices can be modified to one-piece, two-piece or three-piece line type, or four-piece enclosed type, and were applied in the kitchens of Chinese restaurants for engineering intervention of COF. Figure 2 shows an example of EACD with three-piece applied in kitchens for engineering intervention of COF. Air curtain resembles an air jet. Both air curtain generator and COF source are located on the workbench. The air curtain generator sucks the outer air and generates a vertical upwards air curtain to blow COF towards the opening of the kitchen range hood where they are captured.

Table 2 lists the comparison of personal and occupational PM, and PAH exposure data between

before and after engineering. The geometric levels of PM₁₀, PM_{2.5}, and PM_{1.0} in air of kitchens were significantly reduced after engineering intervention by the EACD. These data indicate the 55.7% - 65.8 reduction in levels of PM following engineering intervention by the EACD. The geometric mean of particulate pyrene, BkF, BaP, Bghip, DBaEP, and summed PAHs levels in air of kitchens were significantly reduced after engineering intervention by the EACD. These data presents a 71.8 % - 77.7% reduction in particulate PAHs levels following engineering intervention by the EACD.

The comparisons of geometric mean urinary 8-OHdG and MDA concentrations in cooks between the before and after engineering intervention situations are presented in Table 3. Current smokers, non-smokers, and all subjects geometric mean urinary 8-OHdG and MDA levels were all significantly reduced after engineering intervention compared to before engineering intervention. The geometric mean 8-OHdG and MDA concentrations before engineering intervention did not differ significantly between smokers and nonsmokers, respectively. The geometric mean 8-OHdG and MDA concentrations following engineering intervention were also not significantly different between smokers and nonsmokers, respectively. These data reveal the 56.5% - 56.7% reductions in levels of urinary 8-OHdG after engineering intervention. These data indicate the 51.4% - 59.1%

reduction in levels of urinary MDA following engineering intervention.

Table 4 lists the effects of PM, and PAHs on urinary 8-OHdG levels. Predictors of the end of week measurement differences of pre-intervention and post-intervention levels for both urinary 8-OHdG, MDA levels of cooks in linear mixed-effects regression models were used by differences of pre-intervention and post-intervention levels for PM, and particulate PAHs variables in COF. PM_{2.5}, PM_{1.0}, and BaP levels were positively associated with urinary 8-OHdG levels after adjusting for age, BMI, and cigarette smoking. However, PM₁₀, pyrene, BkF, Bghip, and DBaEP levels were not significantly associated with urinary 8-OHdG levels, respectively. Only BaP levels were positively associated with urinary MDA levels after adjusting for other covariates. However, PM₁₀, PM_{2.5}, PM_{1.0}, BkF, Bghip, and DBaEP levels were not significantly associated with urinary MDA levels.

Analyses using self-reported smoking status (yes/no) found that no association between urinary 8-OHdG levels and cigarette smoking, and urinary MDA levels were also not associated with cigarette smoking. Analyses of other personal covariates in the linear mixed-effects regression models revealed that age and BMI were not significantly associated with urinary 8-OHdG levels. Additionally, neither age nor BMI were significantly associated with urinary MDA levels.

Table 1 Background information and characteristics of study population in the six Chinese restaurants

Restaurant information (n=6), mean ± SD (range)	
Type of cooking oil	Soybean oil
Cooking time (hours per day)	6.3 ± 0.6 (5.4-7.0)
Food oil consumption rate(L/day)	14.1 ± 7.2 (7.6-27.0)
Inflow speed on the opening of hoods (m/sec)	0.28 ± 0.05 (0.24-0.36)
Kitchen volume (m ³)	138.5 ± 154.9 (42.2-437.0)
Personal characteristics of cooks (n=45), mean ± SD (range)	
Age (years)	34.0 ± 10.6 (18.0-56.0)
Years as a cook	13.6 ± 9.2 (1.0-40.0)
Height (cm)	169.6 ± 5.2 (160.0-182.0)
Weight (kg)	67.4 ± 11.7 (48.0-95.0)
BMI (kg/m ²)	23.4 ± 2.8 (17.6-32.9)
Current smokers of cooks, n (%)	20 (44.4 %)

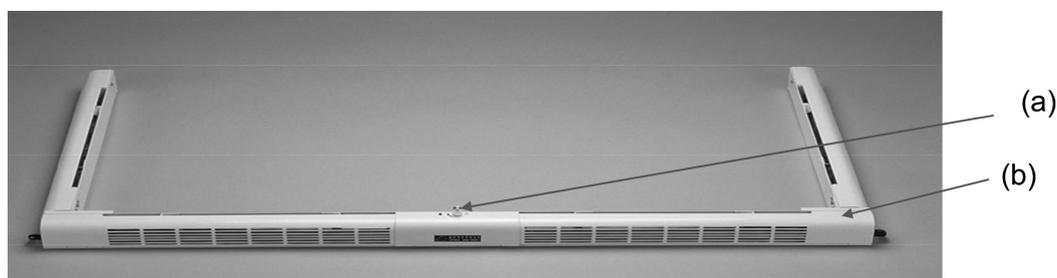


Figure 1 The three-piece U-type embracing air curtain device is a blowing device which includes (a) a control panel and (b) a cast-iron protection cover that encloses motors and air curtain generators.

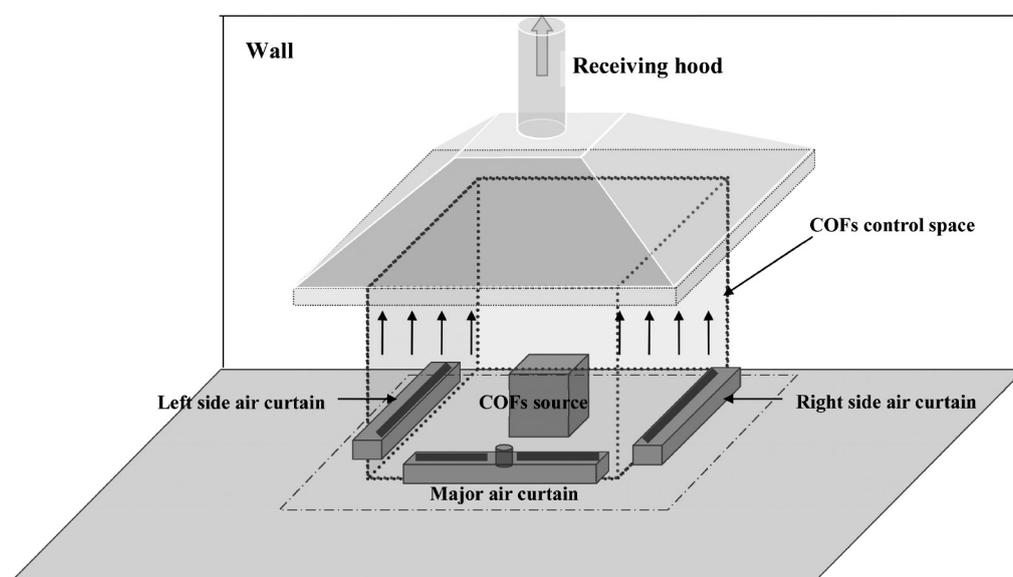


Figure 2 The three-piece embracing air curtain device is applied in the kitchens of Chinese restaurants for engineering intervention of cooking oil fumes (COF).

Table 2 Comparisons of PM and PAHs before and after engineering intervention in this Study (n=45).

Variable	Before Engineering Control		After Engineering Control		Reduction rate (%)	p value ^a
	GM	GSD	GM	GSD		
PM ₁₀ (µg/m ³)	131.4	2.6	44.9	1.5	65.8	<0.001
PM _{2.5} (µg/m ³)	90.6	2.2	37.0	1.6	59.2	<0.001
PM _{1.0} (µg/m ³)	65.7	2.0	29.1	1.5	55.7	<0.001
Pyrene (ng /m ³)	3.7	4.4	1.0	3.9	73.0	<0.001
Benzo(k)fluoranthene (ng /m ³)	1.8	2.1	0.5	1.9	72.2	<0.001
Benzo(a)pyrene (ng /m ³)	8.5	2.3	2.4	2.2	71.8	0.033
Benzo(ghi)perylene (ng /m ³)	4.8	2.7	1.2	2.4	75.0	0.003
Dibenzo(a,e)pyrene (ng /m ³)	0.9	4.3	0.2	6.3	77.7	<0.001
Summed PAHs (ng /m ³) ^b	24.2	2.7	5.9	2.7	75.6	0.007

GM: geometric mean, GSD: geometric standard deviation

^a Student pair t test after log-transformation for before and after engineering control^b Summed PAHs : Sum of pyrene, benzo(k)fluoranthene, benzo(a)pyrene, benzo(ghi)perylene, and dibenzo(a,e)pyrene.

Table 3 Summary of urinary 8-OHdG and MDA levels before and after engineering intervention in this study(Geometric mean (µg/g creatinine))

Marker	Smoking status	Before engineering intervention		After engineering intervention		Reduction rate (%)	p value [#]
		GM	GSD	GM	GSD		
8-OHdG	Smoker (n=20)	9.7	2.0	4.2	1.8	56.7	0.007
	Non-smoker (n=25)	11.5	2.4	5.0	2.0	56.5	0.005
	All(n=45)	10.6	2.2	4.6	1.9	56.6	<0.001
MDA	Smoker (n=20)	291.7	1.8	141.7	1.5	51.4	<0.001
	Non-smoker (n=25)	326.5	1.7	133.6	1.8	59.1	<0.001
	All(n=45)	309.0	1.7	137.1	1.7	55.6	<0.001

GM: geometric mean, GSD: geometric standard deviation

[#] Student pair t test after log-transformation for before and after engineering interventionTable 4 Predictors of the end of week measurement differences between pre-intervention and post-intervention levels of urinary 8-OHdG and MDA levels of cooks using the linear mixed-effects regression models (n=45)[#].

Predictors	8-OHdG (g/g creatinine)	MDA (g/g creatinine)
	Regression coefficient(95% Confidence interval)	Regression coefficient (95% Confidence interval)
PM ₁₀ (µg/m ³)	2.562 (-3.162 to 8.286)	0.668 (-2.382 to 3.719)
PM _{2.5} (µg/m ³)	7.025 (3.012 to 11.038)*	2.897 (-5.942 to 11.737)
PM _{1.0} (µg/m ³)	8.601(4.617 to 12.585)*	2.456 (-4.358 to 9.271)
Pyrene (ng /m ³)	0.345 (-11.195 to 11.886)	0.434 (-0.233 to 1.101)
Benzo(k)fluoranthene (ng /m ³)	1.421 (-3.099 to 5.842)	2.302 (-0.163 to 4.767)
Benzo(a)pyrene (ng /m ³)	0.515 (0.055 to 0.975)*	0.533 (0.241 to 0.824)*
Benzo(ghi)perylene (ng /m ³)	1.573 (-0.445 to 3.592)	0.127 (-0.275 to 0.530)
Dibenzo(a,e)pyrene (ng /m ³)	0.483 (-0.833 to 1.799)	0.047 (-0.106 to 0.199)

[#] Models adjusted for age, BMI, and cigarette Smoking.

* p<0.05

DISCUSSION

Emissions of COF from restaurants represent both environmental and occupational hazards. The carcinogenic potency of particulate PAHs emissions was greater than that of gaseous PAHs emissions in COF [16]. Air pollution contains large amounts of oxidants that induce the generation of reactive oxygen species (ROS). ROS have been posited to be associated with aging, cancer, and certain degenerative diseases because they cause oxidative damage to nucleic acids, proteins and lipids [17]. Levels of urinary 8-OHdG, a biomarker of oxidative DNA damage and repair, increased after occupational exposure to PM_{2.5} was found in boilermakers [18]. MDA is a major product of the peroxidative degradation of the polyunsaturated fatty constituents of biological membranes. Oxygen free radicals and other toxins, formed by the lung cells themselves and by activated inflammatory cells, may contribute to hyperoxidant damage [19]. Measurement of MDA, an indicator of the extent of tissue damage has been extensively used to evaluate extent of lipid peroxidation under various pathological conditions [20].

The levels of PM₁₀, PM_{2.5}, and PM_{1.0} exposure in the sample cohort of cooks were significantly reduced after engineering intervention of EACD introduction. PM₁₀ and PM_{2.5} levels in air of kitchens exceeded National Ambient Air Quality Standards (NAAQS) before the implementation of the EACD intervention. However, following the EACD intervention implementation, the average levels of PM₁₀, and PM_{2.5} in air of kitchens were below 150 µg/m³, and 100 µg/m³ based

on averaging over 24 hours in indoor air quality recommendation standards issued by Taiwan Environmental Protection Administration [21]. Epidemiological studies demonstrate that increased cardiovascular mortality is associated with elevated daily levels of PM₁₀, and especially PM_{2.5} [22-23].

Previous studies indicated that submicrometer particles (PM_{1.0}) are an environmental stressor, with potential to induce a series of events by increasing sympathetic activation, and with potential to lead to ischemia or fatal arrhythmia in high-risk patients with underlying cardiac abnormalities [24]. This phenomenon reveals that reducing PM exposure may decrease the risk of cardiovascular mortality. In this study, linear mixed-effect models indicated that a significant association between PM_{2.5}, PM_{1.0} levels and urinary 8-OHdG levels. This finding agrees with two previous studies demonstrating a significant exposure-response association between PM_{2.5} exposure and urinary 8-OHdG levels [25-26]. However, PM₁₀, PM_{2.5}, PM_{1.0} levels were not significantly associated with urinary MDA levels. MDA level is considered a valuable systemic oxidative stress indicator, but MDA reacts readily with other substances in the body, and its level, unlike that of urinary 8-OHdG, may not accurately reflect oxidative stress in the whole body [27].

The levels of pyrene, BkF, BaP, Bghip, and DBaP exposure in our cohort of cooks were also significantly reduced after engineering intervention of the EACD introduction. Although the average levels of BaP in air of kitchens before and after EACD intervention were both below permissible exposure limit of US Occupational Safety and Health Administration (OSHA) Standards: 0.2

mg/m³ [28]. Epidemiologic studies indicate an increase in incidence of cancer among workers exposed to PAHs, even when the exposure of BaP was below 0.2 mg/m³ [26-29]. For example, a significant positive correlation between DNA adducts in lymphocytes and BaP (from 2 to 62,107 ng.m³) in the inhaled air in coke oven workers [29]. Furthermore, Nilsson noted increased urinary 8-OHdG in engine room personnel exposed to PAHs [30]. Moreover, a significant increase in 8-OHdG was evident in end-of-week urine samples compared with start-of-week urine samples in roofers exposed to PAHs [31]. In addition, urinary 8-OHdG levels were positively and significantly associated with PAH exposure in restaurant workers [8] and coke oven workers [32]. Urinary 8-OHdG is considered to arise from three sources: repair products of oxidized DNA, removal of oxidized dG in the nucleotide pool, and cell turnover. This reveals that urinary 8-OHdG represents average oxidative DNA damage level throughout the body [33]. A study found that a positive association between BaP levels urinary 8-OHdG, MDA levels, after adjusting for personal covariates. This finding is in agreement with a previous animal study [34].

Tobacco smoke contains over 50 known carcinogens and is responsible for the majority of cases of non-occupational lung cancer in industrialized countries [35]. Some previous studies have noted an increase in urinary 8-OHdG levels in smokers compared with nonsmokers [36-37]. However, this study found that mean 8-OHdG levels were not significantly different between smokers and nonsmokers.

COF can induce oxidative DNA damage and

oxidative stress in humans. Induced oxidative DNA damage may be related to lung carcinogenesis [38]. Oxidative stress involved in the exposure risk of COF may be important in the incidence of lung cancer among Chinese women [39]. Cooks face occupational exposure to COF. This study found that the introduction of the EACD is an effective engineering intervention measure for reducing levels of PM and PAHs in COF in Chinese kitchens. This finding is in agreement with a previous laboratory study [40].

This study suffers various limitations. First, this study suffered from the lack of other co-pollutant data in COF, such as aldehydes, aromatic amines, and benzene, which may have confounded its findings regarding the effects of PM and PAHs. Furthermore, this study suffered a lack of PM and PAHs exposure data from non-occupational settings, such as from vehicle traffic emissions. However, restaurant workers spent more than 10 hours daily in restaurants, including work and rest periods, compared to less than 1 hour spent in traffic daily for commuters. The contribution of traffic sources to restaurant worker PAHs exposure thus is believed to be limited. This study thus assumes only a limited contribution of traffic sources to restaurant worker PAHs exposure.

CONCLUSION

In conclusion, this study proved that engineering intervention by the EACD can effectively reduce kitchen COF levels and oxidative stress in Chinese restaurant cooks. Consequently, increased emphasis should be placed on installing the EACD to lower risk of exposure to particles and

carcinogens among kitchen staff.

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REFERENCES

- [1] Van Houdt JJ, Jongen WM, Alink GM, et al. Mutagenic activity of airborne particles inside and outside homes. *Environmental Mutagenesis* 1984;6:861-9.
- [2] Chiang TA, Pei-Fen Wu, Wang LF, et al. Mutagenicity and polycyclic aromatic hydrocarbon content of fumes from heated cooking oils produced in Taiwan. *Mutation Research* 1997;381:157-61.
- [3] Mossman BT, Borm PJ, Castranova V, et al. Mechanisms of action of inhaled fibers, particles and nanoparticles in lung and cardiovascular diseases. *Particle and Fibre Toxicology* 2007;4:4.
- [4] Lanki T, de Hartog JJ, Heinrich J, et al. Can we identify sources of fine particles responsible for exercise-induced ischemia on days with elevated air pollution? The ULTRA study. *Environmental Health Perspectives* 2006;114:655-60.
- [5] Brunekreef B, Holgate ST. Air pollution and health. *Lancet* 2002;360:1233-42.
- [6] Elbeki RH, Korashy HM, Wilis K, Gharavi N, Elkaid AOS. Benzo(a)pyrene, 3-methylcholanthrene and β -naphthoflavone induce oxidative stress in hepatoma Hepa 1c1c7 cells by an AHR-dependent pathway. *Free Radical Research* 2004; 38: 1191-1200.
- [7] Bosetti C, Boffetta P, La Vecchia C. Occupational exposures to polycyclic aromatic hydrocarbons, and respiratory and urinary tract cancers: a quantitative review to 2005. *Annals of Oncology* 2007;18:431-46.
- [8] Pan CH, Chan CC, Wu KY. Effects on Chinese Restaurant Workers of Exposure to cooking oil fumes: a Cautionary Note on Urinary 8-Hydroxy-2'-Deoxyguanosine. *Cancer Epidemiology Biomarkers & Prevention* 2008;17:3351-7.
- [9] Pan CH, Chan CC, Huang YL, et al. Urinary 1-hydroxypyrene and malondialdehyde in male workers in Chinese restaurants. *Occupational & Environmental Medicine* 2008;65:732-5.
- [10] Chiang TA, Wu Pei-Fen, Liao-Su, et al. Mutagenicity and aromatic amine content of fumes from heated cooking oils produced in Taiwan. *Food and Chemical Toxicology* 1999;37:125-34.
- [11] Wu PF, Chiang TA, Wang LF, et al. Nitro-polycyclic aromatic hydrocarbon contents of fumes from heated cooking oils and prevention of mutagenicity by catechin. *Mutation Research* 1998;403:29-34.
- [12] Pavageau M, Nieto EM, Rey C. Odour and VOC confining in large enclosures using air curtain. *Water Science & Technology* 2001;44:165-71.
- [13] Lee SY. Smoke Guiding Machine. Patent No: US 6752144 B1;2004.
- [14] Huang YL, Chuang I-Chuan, Pan CH, et al. Determination of chromium in whole blood

- and urine by graphite furnace AAS. *Atomic Spectroscopy* 2000;21:10-16.
- [15] Hu CW, Wang CJ, Chang LW, et al. 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. *Clinical Chemistry* 2006;52:1381-8.
- [16] Li CT, Lin YC, Lee WJ, et al. Emission of polycyclic aromatic hydrocarbons and their carcinogenic potencies from cooking sources to the urban atmosphere. *Environmental Health Perspectives* 2003;111:483-7.
- [17] Dizdaroglu M, Jaruga P, Birincioglu M, et al. Free radical-induced damage to DNA: mechanisms and measurement. *Free Radical Biology Medicine* 2002;32:1102-15.
- [18] Kim JY, Mukherjee S, Ngo LC, et al. Urinary 8-hydroxy-2'-deoxyguanosine as a biomarker of oxidative DNA damage in workers exposed to fine particulates. *Environmental Health Perspectives* 2004;112:666-71.
- [19] Martinez-Cayuela M. Oxygen free radical and human disease. *Biochimie* 1995;77:147-61.
- [20] Li XY, Chow CK. An improved method for the measurement of malondialdehyde in biological samples. *Lipids* 1994;29:73-75.
- [21] Taiwan EPA. National Ambient Air Quality Standards, EPA; 2006.
- [22] Borja-Aburto VH, Loomis DP, Bangdiwala S, et al. Ozone, suspended particulates, and daily mortality in Mexico City. *American Journal of Epidemiology* 1997;145:258-68.
- [23] Zanobetti A, Schwartz J, Samoli E, et al. The temporal pattern of respiratory and heart disease mortality in response to air pollution. *Environmental Health Perspectives* 2003;111:1188-93.
- [24] Chan CC, Chuang KJ, Shiao GM, et al. Personal exposure to submicrometer particles and heart rate variability in human subjects. *Environmental Health Perspectives* 2004;112:1063-67.
- [25] Kim KB, Lee BM. Oxidative stress to DNA, protein, and antioxidant enzymes (superoxide dismutase and catalase) in rats treated with benzo(a)pyrene. *Cancer Letters* 1997;113:205-12.
- [26] Pilger A, Rudiger HW. 8-Hydroxy-2'-deoxyguanosine as a marker of oxidative DNA damage related to occupational and environmental exposures. *International Archives Occupational and Environmental Health* 2006;80:1-15.
- [27] Yoshida R, Ogawa Y, Mori I, et al. Associations between oxidative stress levels and total duration of engagement in jobs with exposure to fly ash among workers at municipal solid waste incinerators. *Mutagenesis* 2003;18:533-7.
- [28] US Occupational Safety and Health Administration. Standard. 1983;29 CFR 1910.1000.
- [29] Binková B, Topinka J, Mracková G, et al. Coke oven workers study: the effect of exposure and GSTM1 and NAT2 genotypes on DNA adduct levels in white blood cells and lymphocytes as determined by ³²P-postlabelling. *Mutation Research* 1998;416:67-84.
- [30] Nilsson R, Nordlinder R, Moen BE,

- et al. Increased urinary excretion of 8-hydroxydeoxyguanosine in engine room personnel exposed to polycyclic aromatic hydrocarbons. *Occupational & Environmental Medicine* 2004;61:692-6.
- [31] Toraason M, Hayden C, Marlow D, et al. DNA strand breaks, oxidative damage, and 1-OH pyrene in roofers with coal-tar pitch dust and/or asphalt fume exposure. *International Archives of Occupational and Environmental Health* 2002;75:279.
- [32] Wu MT, Pan CH, Huang YL, et al. Urinary excretion of 8-hydroxy-2-deoxyguanosine and 1-hydroxypyrene in coke-oven workers. *Environmental and Molecular Mutagenesis* 2003;42:98-105.
- [33] Loft S, Poulsen HE. Antioxidant intervention studies related to DNA damage, DNA repair and gene expression. *Free Radical Research* 2000;33:S67-S83.
- [34] Boutin AC, Shirali P, Garçon G, et al. Peripheral markers (Clara cell protein and alpha-glutathione S-transferase) and lipidoxidation (malondialdehyde) assessment in Sprague-Dawley rats instilled with haematite and benzo[a]pyrene. *Journal of Applied Toxicology* 1998;18:39-45.
- [35] Alberg AJ, Samet JM. Epidemiology of lung cancer. *Chest Journal* 2003;123:21S-49S.
- [36] Loft S, Vistisen K, Ewertz M, et al. Oxidative DNA damage estimated by 8-hydroxydeoxyguanosine excretion in humans: influence of smoking, gender and body mass index. *Carcinogenesis* 1992;13:2241-7.
- [37] Pilger A, Germadnik D, Riedel K, et al. Longitudinal study of urinary 8-hydroxy-2'-deoxyguanosine excretion in healthy adults. *Free Radical Research* 2001;35:273-80.
- [38] Ichinose T, Yajima Y, Nagashima M, et al. Lung carcinogenesis and formation of 8-hydroxydeoxyguanosine in mice by diesel exhaust particles. *Carcinogenesis* 1997;18:185-92.
- [39] 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. *Journal of Toxicology Environmental Health. Part A* 2002;65:265-78.
- [40] Wang SC, Chen CC, Chang CP. Improving Efficacy of a wall-mounted receiving hood by embracing air curtain. *AIHCE 2005 CA, USA*.