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# Reduction of cooking oil fume exposure following an engineering intervention in Chinese restaurants

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#### ABSTRACT

**Background** A new engineering intervention measure, an embracing air curtain device (EACD), was used to increase the capture efficiency of cooker hoods and reduce cooking oil fume (COF) exposure in Chinese restaurants. **Methods** An EACD was installed in six Chinese

restaurants where 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 changes in levels of urinary 8-hydroxy-2'deoxyguanosine (8-OHdG) and malondialdehyde (MDA). The association between PM and PAHs in air and 8-OHdG and MDA in urine was evaluated by linear mixed-effects regression analysis.

**Results** Results showed that geometric mean kitchen air levels of  $PM_{10}$ ,  $PM_{2.5}$ ,  $PM_{1.0}$  and total particulate PAHs were significantly reduced after the EACDs were introduced. Urinary levels of 8-OHdG and MDA in cooks were also significantly lower after EACD instalment.  $PM_{2.5}$ ,  $PM_{1.0}$  and benzo(a)pyrene (BaP) levels were positively associated with urinary 8-OHdG levels after adjusting for key personal covariates. Urinary MDA levels in cooks were also positively associated with BaP levels after adjusting for key personal covariates.

**Conclusion** This study demonstrates that the EACD is effective for reducing COF and oxidative stress levels in cooks working in Chinese kitchens.

#### INTRODUCTION

A previous study demonstrated that cooking had a major influence on the total mutagenic activity of indoor air.<sup>1</sup> 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 therefore occupationally exposed to COF. Particulate matter (PM) and polycyclic aromatic hydrocarbons (PAHs)<sup>2</sup> are important components of COF. Airborne PM are associated with cardiopulmonary mortality and morbidity,<sup>2</sup> oxidative stress<sup>4</sup>  $^{5}$  and inflammation in humans.<sup>6</sup> PAHs are associated with lung and bladder cancers.<sup>7</sup> Therefore, effective measures are necessary to reduce human exposure to COF. Exterior cooker hoods have poor capture efficiency, so airborne contaminants easily reach the breathing zone of workers. Since exterior cooker hoods are used in Chinese restaurants, increasing the capture efficiency of the hoods and reducing levels of PM and PAHs in workers' breathing zones is very important.

#### What this paper adds

- Oxidative DNA damage in Chinese restaurant workers was associated with exposure to cooking oil fumes (COF).
- This is the first study to evaluate oxidative stress in cooks after COF exposure was reduced using an embracing air curtain device (EACD).
- The EACD is effective in reducing COF and oxidative stress in cooks working in Chinese kitchens.
- Increased emphasis should be placed on installing the EACD to lower the risk of exposure to particles and carcinogens among kitchen staff.

Previous studies indicated that urinary 8-hydroxy-2'-deoxyguanosine (8-OHdG)<sup>8</sup> and malondialdehyde (MDA)<sup>9</sup> were two useful biomarkers for assessing oxidative stress induced by COF exposure. Two other studies revealed that the mutagenicity of COF was significantly reduced if the antioxidant catechin was added to cooking oils before heating.<sup>10</sup> <sup>11</sup> Air curtains (jets of air) are used in air conditioners, fire safety systems and the food industry, and for indoor air quality.<sup>12</sup> This study used a new engineering intervention measure, an embracing air curtain device (EACD; Smoke Guider, Acxing Industrial, Taipei, Taiwan) to increase the capture efficiency of kitchen cooker hoods and reduce COF exposure for Chinese restaurant workers. The effects of the EACD in reducing COF were evaluated by air monitoring of PM and particulate PAHs, and biological monitoring of urinary 8-OHdG and MDA in cooks. We are unaware of any previous study evaluating oxidative stress in cooks after COF exposure was reduced using an air curtain.

#### METHODS Study site

Six Chinese restaurants where the each average air inflow velocity of cooker hoods was less than 0.4 m/s were selected 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**

Good work practice was followed when fitting the EACDs close to the cooking surface: (i) the EACD enclosed the COF emission source; (ii) the dip angle

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Accepted 17 January 2010 Published Online First 30 September 2010 of the EACD exceeded zero to blow COF into the cooker hood; and (iii) the EACD air jets were directed into the cooker hood. The three-piece U-type EACD<sup>13</sup> (figure 1) consists of a control panel, and a cast-iron cover enclosing the motors and air curtain generators (blowing devices). The EACD can be used with all types and sizes of cooker hoods. The maximum flow of the EACDs was set at 2.6 m/s. The efficiency of the EACDs was evaluated by comparing values for PM and PAHs in air, and urinary 8-OHdG and MDA in cooks measured before and after the devices were installed.

#### **Exposure assessment**

Personal air monitoring and biological monitoring before and after intervention were used to evaluate EACD efficiency. Smoking was not allowed in the kitchens and dining areas under anti-smoking regulations designating all restaurants as nonsmoking 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 PM and particulate PAHs was carried out during the weekend (Saturday) before and 4 weeks after the EACDs were installed.

#### **Particulate matter**

 $PM_{10}$ ,  $PM_{2.5}$  and  $PM_{1.0}$  were monitored continuously for each study subject using a Dust-Check Portable Dust Monitor (Grimm model 1.108; Grimm Labortechnik, Ainring, Germany) and 1 min 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, East Greenbush, New York, USA) were used to calibrate the mass concentrations of  $PM_{10}$ ,  $PM_{2.5}$  and  $PM_{1.0}$ measured using the Dust-Check monitor in a previous study. Concurrent PM measurements indicated a strong association between collection date using these two monitors for all three size fractions:  $PM_{10}$  ( $r^2$ =0.91),  $PM_{2.5}$  ( $r^2$ =0.90) and  $PM_{1.0}$ ( $r^2$ =0.80).



**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 cover enclosing motors and air curtain generators.

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#### Particulate PAHs

Particulate PAHs in the workplace were collected for the 45 cooks using IOM (Institute of Occupational Medicine, UK) samplers with glass fibre filters (diameter: 25 mm, pore size:  $0.7 \,\mu$ m) at a flow rate of 2.0 l/min. The samplers were placed near the breathing zones of the workers. Personal particulate PAH measurements were summarised 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 (DBaeP), were quantified using high performance liquid chromatography (HPLC). The coefficient of variation for these repeated analyses was below 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.

#### **Biological monitoring**

#### Urinary 8-OHdG and MDA

Before EACD installation, urine samples were collected from all participants following their Saturday night shift after having worked for six consecutive days from Monday through Saturday. Then 4 weeks after the EACDs were introduced, urine samples were again collected from all participants following their Saturday night shift 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 analysed using HPLC/MS/MS with an isotope-labelled coeluting internal standard which has been described previously.<sup>15</sup> A detection limit of 5.7 ng/l was obtained from seven repeated analyses of deionised water. The coefficients of variation were below 5% for the interday and intraday tests.

Urinary MDA concentration was measured using an HPLC instrument (model 980-PU; JASCO, Tokyo, Japan) with a  $C_{18}$  column and an ultraviolet-visible detector at 532 nm (UV-975; JASCO). The mobile phase was methanol/potassium phosphate (9:11) buffer and flow rate was 1.2 ml/min. The within-run and run-to-run precisions of urinary MDA were evaluated. The samples were analysed for MDA based on thiobarbituric acid (TBA) reaction, with HPLC separation of the MDA (TBA)<sub>2</sub> adduct, using tetraethoxypropane as a standard. A detection limit of 0.06 µg/l was obtained from seven repeated analyses of deionised water, and the variation in the coefficients of repeated analyses was below 10%.

#### Questionnaire survey

In total, 54 of 58 eligible cooks participated in the study, giving a participation rate of 77.6%. The subjects were monitored during the weekends before and after the EACDs were installed. All 58 eligible cooks completed the questionnaire but only 45 completed the questionnaire, air monitoring and biological monitoring as 13 cooks were not working during the study weekend. Trained interviewers met participants between January 2006 and April 2006 and conducted a questionnaire survey to gather data on age, years working in restaurants, height, weight, educational level, health and lifestyle, including cigarette smoking. Cigarette smokers were defined as smoking cigarettes on at least 4 days each week for at least 6 months. Data were also gathered on main cooking methods, type of cooking oil, cooking time, rate of food oil use, kitchen size, and air inflow velocity of cooker hoods.

### **Original article**

#### **Statistical methods**

Paired Student t tests were used to compare personal  $PM_{10}$ ,  $PM_{2.5}$ ,  $PM_{1.0}$ , PAH, 8-OHdG and MDA levels before and after the EACDs were installed. The PM, PAH, 8-OHdG and MDA measurements, and other personal covariates with abnormal distributions, were first log-transformed to normalise their distributions before conducting paired Student t test analysis.

The differences between the pre-intervention and post-intervention levels in urinary 8-OHdG and MDA were calculated by subtracting the concentration before EACD installation from that after installation. The differences in PM and particulate PAH levels were similarly calculated. Linear mixed-effects regressions models were then used to investigate the effects of PM and particulate PAHs on urinary 8-OHdG and MDA concentrations, after adjusting for age and body mass index (BMI). 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 at  $\alpha$ =0.05 in all tests. All data analyses were performed using the S-PLUS 2000 program (MathSoft, Cambridge, Massachusetts, USA).

#### RESULTS

Table 1 lists the background information and characteristics of the 45 male cooks. Each cook used all types of cooking methods. Main cooking methods in order of frequency were: stir frying, deep frying, stewing, steaming and roasting, and soybean oil was the main oil used. Mean cooking time per day was  $6.3\pm0.6$  h and average use of food oil was  $14.1\pm7.2$  l/day. Mean air inflow velocity of the cooker hoods was  $0.28\pm0.05$  m/s. The size of the kitchens varied from 47.3 to 437.0 m<sup>3</sup>. The cooks' ages ranged from 15 to 56 years, with a mean $\pm$ SD of  $34.0\pm10.6$  years. The subjects had an average of  $13.6\pm9.2$  years of cooking experience. Twenty of the 45 subjects (44.4%) were current cigarette smokers. Demographic characteristics did not differ significantly between the 45 participants and the 13 non-participants (p>0.1).

Figure 1 illustrates a U-type three-piece EACD. The air curtain can consist of one, two or three line type devices, or be a fourpiece enclosed type. Figure 2 shows an EACD with three pieces set up in a kitchen. The air curtain generator sucks in air from outside the cooking area and generates a vertical upwards air curtain to blow COF towards the cooker hood where they are captured.

Table 2 lists personal and occupational PM and PAH exposure data before and after EACD installation. Levels of  $PM_{10}$ ,  $PM_{2.5}$  and  $PM_{1.0}$  in kitchen air were significantly reduced after

 Table 1
 Background information and characteristics of the study population

Restaurant information (n=6), mean $\pm$ SD (range)	
Type of cooking oil	Soybean oil
Cooking time (hours per day)	6.3±0.6 (5.4-7.0)
Rate of food oil use (l/day)	14.1±7.2 (7.6-27.0)
Air inflow velocity of hood (m/s)	0.28±0.05 (0.24-0.36)
Size of kitchen (m <sup>3</sup> )	138.5±154.9 (42.2—437.0)
Personal characteristics of cooks (n=45)	
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)
Body mass index (kg/m <sup>2</sup> )	23.4±2.8 (17.6-32.9)
Current smokers, n (%)	20 (44.4%)

Values are mean ± SD (range).

installation, with 55.7–65.8% reductions in PM levels. The geometric means of particulate pyrene, BkF, BaP, Bghip, DBaeP and summed PAHs levels in kitchen air were also significantly reduced, with a 71.8–77.7% reduction in particulate PAH levels.

Geometric mean urinary 8-OHdG and MDA concentrations in cooks before and after installation of the EACDs are presented in table 3, and show significant reductions in geometric mean urinary 8-OHdG and MDA levels in smokers, non-smokers and all subjects combined, with a 56.5–56.7% reduction in urinary 8-OHdG levels and a 51.4–59.1% reduction in urinary MDA levels. Geometric mean 8-OHdG and MDA concentrations did not differ significantly between smokers and non-smokers before EACD installation.

Table 4 lists the effects of PM and PAHs on urinary 8-OHdG levels. Differences between pre-intervention and post-intervention levels of PM and COF particulate PAH variables predicted the end-of-week measurement differences between pre-intervention and post-intervention levels for both urinary 8-OHdG and MDA in linear mixed-effects regression models. PM<sub>2.5</sub>, PM<sub>1.0</sub> and BaP levels were positively associated with urinary 8-OHdG levels after adjusting for age, BMI and cigarette smoking. However, PM<sub>10</sub>, pyrene, BkF, Bghip and DBaeP levels were not significantly associated with urinary 8-OHdG levels after adjusting for other covariates. PM<sub>10</sub>, PM<sub>2.5</sub>, PM<sub>1.0</sub>, BkF, Bghip and DBaeP levels were not significantly associated with urinary MDA levels after adjusting for other covariates. PM<sub>10</sub>, PM<sub>2.5</sub>, PM<sub>1.0</sub>, BkF, Bghip and DBaeP levels were not significantly associated with urinary MDA levels after adjusting for other covariates. PM<sub>10</sub>, PM<sub>2.5</sub>, PM<sub>1.0</sub>, BkF, Bghip and DBaeP levels were not significantly associated with urinary MDA levels.

Analyses using self-reported smoking status (yes/no) found no association between urinary 8-OHdG levels and cigarette smoking, nor were urinary MDA levels 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, nor were age or BMI significantly associated with urinary MDA levels.

#### DISCUSSION

COF emissions in restaurants are both an environmental and occupational hazard. The carcinogenic potency of particulate PAH emissions was greater than that of gaseous PAH emissions in COF.<sup>16</sup> Air pollution contains large amounts of oxidants that induce the generation of reactive oxygen species (ROS). ROS have been posited to be associated with ageing, cancer and certain degenerative diseases because they cause oxidative damage to nucleic acids, proteins and lipids.<sup>17</sup> Levels of urinary 8-OHdG, a biomarker of oxidative DNA damage and repair, increased after occupational exposure to PM<sub>2.5</sub> in boilermakers.<sup>18</sup>



**Figure 2** The three-piece embracing air curtain device installed in a kitchen to reduce levels of cooking oil fumes (COF).

Table 2	Comparisons	of PM	and PAHs	s before an	d after	EACD	installation	(n=45)	
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	Before EACD installation		After EACD installation			
Variable	GM	GSD	GM	GSD	Rate of reduction (%)	p Value*
PM <sub>10</sub> (μg/m <sup>3</sup> )	131.4	2.6	44.9	1.5	65.8	< 0.001
PM <sub>2.5</sub> (μg/m <sup>3</sup> )	90.6	2.2	37.0	1.6	59.2	< 0.001
PM <sub>1.0</sub> (μg/m <sup>3</sup> )	65.7	2.0	29.1	1.5	55.7	< 0.001
Pyrene (ng /m <sup>3</sup> )	3.7	4.4	1.0	3.9	73.0	< 0.001
Benzo(k)fluoranthene (ng /m <sup>3</sup> )	1.8	2.1	0.5	1.9	72.2	< 0.001
Benzo(a)pyrene (ng/m <sup>3</sup> )	8.5	2.3	2.4	2.2	71.8	0.033
Benzo(ghi)perylene (ng/m <sup>3</sup> )	4.8	2.7	1.2	2.4	75.0	0.003
Dibenzo(a,e)pyrene (ng/m <sup>3</sup> )	0.9	4.3	0.2	6.3	77.7	< 0.001
Summed PAHs (ng/m <sup>3</sup> )†	24.2	2.7	5.9	2.7	75.6	0.007

\*Paired Student t test after log-transformation of measurements from before and after EACD installation.

+Summed PAHs: sum of pyrene, benzo(k)fluoranthene, benzo(a)pyrene, benzo(ghi)perylene and dibenzo(a,e)pyrene.

EACD, embracing air curtain device; GM, geometric mean; GSD, geometric SD; PAHs, polycyclic aromatic hydrocarbons;

PM, particulate matter.

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 lung cells and activated inflammatory cells, may contribute to hyperoxidant damage.<sup>19</sup> Measurement of MDA, an indicator of the degree of tissue damage, has been extensively used to evaluate the extent of lipid peroxidation under various pathological conditions.<sup>20</sup>

The levels of  $PM_{10}, PM_{2.5}$  and  $PM_{1.0}$  exposure in the sample cohort of cooks were significantly reduced after installation of EACDs; before the EACDs were introduced  $PM_{10}$  and  $PM_{2.5}$  levels in kitchen air exceeded National Ambient Air Quality Standards (NAAQS). However, following installation, the average  $PM_{10}$  and  $PM_{2.5}$  levels in kitchen air were below 150  $\mu g/m^3$  and 100  $\mu g/m^3$ , respectively, based on averaging over 24 h of indoor air quality according to Taiwan Environmental Protection Administration standards. <sup>21</sup> Epidemiological studies demonstrate that increased cardiovascular mortality is associated with elevated daily levels of  $PM_{10}$  and particularly  $PM_{2.5}^{-22}$  <sup>23</sup>

Previous studies indicated that submicrometre particles (PM<sub>1.0</sub>) are an environmental stressor, with the potential to induce a series of events by increasing sympathetic activation, leading to ischaemia or fatal arrhythmia in high-risk patients with underlying cardiac abnormalities.<sup>24</sup> Therefore, reducing PM exposure may decrease the risk of cardiovascular mortality. In this study, linear mixed-effect models indicated a significant association between PM<sub>2.5</sub>, PM<sub>1.0</sub> and urinary 8-OHdG levels. This finding agrees with two previous studies demonstrating a significant exposure–response relationship between PM<sub>2.5</sub> exposure and urinary 8-OHdG levels.<sup>25 26</sup> PM<sub>10</sub>, PM<sub>2.5</sub> and PM<sub>1.0</sub> levels were not significantly associated with urinary MDA levels.

MDA concentration is considered a valuable systemic oxidative stress indicator, however MDA reacts readily with other substances in the body, and its level, unlike that of urinary 8-OHdG, may not accurately reflect oxidative stress throughout the whole body.<sup>27</sup>

The levels of pyrene, BkF, BaP, Bghip and DBaeP exposure in our cohort of cooks were also significantly reduced after the introduction of the EACDs, although the average levels of BaP in kitchen air both before and after the EACD intervention were below the permissible exposure limits set by the US Occupational Safety and Health Administration (OSHA) (0.2 mg/m<sup>3</sup>).<sup>28</sup> Epidemiological studies indicate an increase in the incidence of cancer among workers exposed to PAHs, even when levels of BaP are below  $0.2 \text{ mg/m}^{3.26}$  <sup>29</sup> For example, there was a significant positive correlation between DNA adducts in lymphocytes and BaP (from 2 to 62 107 ng.m<sup>3</sup>) in inhaled air in coke oven workers.<sup>29</sup> Furthermore. Nilsson noted increased urinary 8-OHdG in engine room personnel exposed to PAHs.<sup>30</sup> Moreover, a significant increase in 8-OHdG was evident in end-ofweek urine samples compared with start-of-week urine samples in roofers exposed to PAHs.<sup>31</sup> In addition, urinary 8-OHdG levels were positively and significantly associated with PAH exposure in restaurant workers<sup>8</sup> and coke oven workers.<sup>32</sup> Urinary 8-OHdG is considered to arise from three sources: repair products of oxidised DNA, removal of oxidised dG in the nucleotide pool, and cell turnover, and thus represents the average oxidative DNA damage level throughout the body.<sup>33</sup> A study found a positive association between BaP levels and urinary 8-OHdG and MDA levels after adjusting for personal covariates. This finding is in agreement with a previous animal study.34

Table 3 Summary of urinary 8-OHdG and MDA levels before and after EACD installation (geometric mean ( $\mu$ g/g creatinine))

	Smoking status	Before EACD installation		After EACD installation			
Marker		GM	GSD	GM	GSD	Rate of reduction (%)	p Value*
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

\*Paired Student t test after log-transformation of measurements from before and after EACD installation.

EACD, embracing air curtain device; GM, geometric mean; GSD, geometric SD; MDA, malondialdehyde; 8-OHdG, 8-hydroxy-2'deoxyguanosine.

## Original article

Table 4	Predictors of end-of-week measurement differences between
pre-interv	ention and post-intervention levels of urinary 8-OHdG and MDA
in cooks	n=45) using linear mixed-effects regression models †

Predictors	8-OHdG (µg/g creatinine) Regression coefficient (95% Cl)	MDA (µg/g creatinine) Regression coefficient (95% Cl)
PM <sub>10</sub> (μg/m <sup>3</sup> )	2.562 (-3.162 to 8.286)	0.668 (-2.382 to 3.719)
PM <sub>2.5</sub> (µg/m <sup>3</sup> )	7.025 (3.012 to 11.038)*	2.897 (-5.942 to 11.737)
PM <sub>1.0</sub> (μg/m <sup>3</sup> )	8.601 (4.617 to 12.585)*	2.456 (-4.358 to 9.271)
Pyrene (ng /m <sup>3</sup> )	0.345 (-11.195 to 11.886)	0.434 (-0.233 to 1.101)
Benzo(k)fluoranthene (ng/m³)	1.421 (-3.099 to 5.842)	2.302 (-0.163 to 4.767)
Benzo(a)pyrene (ng/m <sup>3</sup> )	0.515 (0.055 to 0.975)*	0.533 (0.241 to 0.824)*
Benzo(ghi)perylene (ng/m <sup>3</sup> )	1.573 (-0.445 to 3.592)	0.127 (-0.275 to 0.530)
Dibenzo(a,e)pyrene (ng/m <sup>3</sup> )	0.483 (-0.833 to 1.799)	0.047 (-0.106 to 0.199)

\*p<0.05

+Models adjusted for age, body mass index and cigarette smoking.

MDA, malondialdehyde; 8-OHdG, 8-hydroxy-2'-deoxyguanosine.

Tobacco smoke contains over 50 known carcinogens and is responsible for the majority of cases of non-occupational lung cancer in industrialised countries.<sup>35</sup> Although some previous studies have noted an increase in urinary 8-OHdG levels in smokers compared with non-smokers,<sup>36 37</sup> this study found that mean 8-OHdG levels were not significantly different between smokers and non-smokers.

COF can induce oxidative DNA damage and oxidative stress in humans. Induced oxidative DNA damage may be related to lung carcinogenesis.<sup>38</sup> Oxidative stress caused by exposure to COF may be important in the incidence of lung cancer among Chinese women.<sup>39</sup> This study found that installation of a EACD is an effective intervention for reducing levels of PM and PAHs in COF in Chinese kitchens. This finding is in agreement with a previous laboratory study.<sup>40</sup>

This study has some limitations. First, it lacked data on copollutants in COF, such as aldehydes, aromatic amines and benzene, which may have confounded the findings regarding the effects of PM and PAHs. Furthermore, this study lacked PM and PAHs exposure data from non-occupational settings exposed to vehicle traffic emissions. However, restaurant workers spent more than 10 h daily in restaurants, including work and rest periods, compared to less than 1 h spent commuting in traffic each day. The contribution of traffic sources to restaurant worker PAH exposure is therefore believed to be limited. This study thus assumes that traffic sources made only a limited contribution to restaurant worker PAH exposure.

#### CONCLUSION

In conclusion, this study showed that installation of an EACD can effectively reduce kitchen COF levels and oxidative stress in Chinese restaurant cooks. Consequently, increased emphasis should be placed on installing such EACDs to lower the risk of exposure to particles and carcinogens among kitchen staff.

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#### Competing interests None.

Ethics approval This study was conducted with the approval of the Institute Review Board of the National Health Research Institutes, Taiwan.

Provenance and peer review Not commissioned; externally peer reviewed.

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