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Developing Wood–Based Activated Carbon Fibers Paperboard for Thermal Absorption Filtration of Oil Fume

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This study evaluated Wood–Based Activated Carbon Fibers Paperboard (WACFP) made of different proportions of wood–based activated carbon fibers (WACFs) with wood pulp as the oil fume absorbing material. The WACFPs, in basis weights of 180 g/m^2 and with 0, 10, and 40% WACFs, absorbed peanut oil fume at the smoke point, absorption saturation was reached at 80, 110, and 120 min, and oil fume absorption was 0.0203–0.0304 g/cm^2. The WACFPs, in basis weights of 360 g/m^2 and with 0, 10, and 40% WACFs, reached absorption saturation at 210, 250, and 260 min, and oil fume absorption was 0.0424–0.0604 g/cm^2. For Ames test, the oil fume absorbed by WACFPs extracted by methanol at concentrations higher than 0.10 mg/plate had cytotoxicity with *Salmonella typhimurium* TA98 (±S9) and TA100 (±S9); and the Mutagenicity ratio of methanol oil fume extracts at concentration of 0.05 mg/plate for *Salmonella typhimurium* TA98 (±S9) and TA100 (±S9) were higher than 2, representing very high mutagenicity for *Salmonella typhimurium* TA98 and TA100. According to the GC–MS analysis of the n–hexane soluble fraction of oil fume, the trans–trans–2,4–decadienal (t–t–2,4–DDE) was the main mutagenic compound of peanut oil fume probably.

Key words: Wood–Based Activated Carbon Fibers (WACFs), Wood–Based Activated Carbon Fibers Paperboard (WACFP), Oil Fume, Ames Test, t–t–2,4–DDE

INTRODUCTION

As the socioeconomic structure changes, people's living standards and health/medical treatments have improved. The leading causes of death in Taiwan have changed from acute and communicable diseases to chronic diseases, such as malignant tumors, cardiovascular disease, etc., and malignant tumors have been the leading cause of Taiwanese deaths for 33 years (since 1982); among cancers, lung cancer is the leading cause of death of males and females (Ministry of Health and Welfare, 2015a; 2015b). Lung cancer is the primary epithelial malignant tumor, and grows in the trachea, bronchus, and lungs. According to the World Health Organization statistics, from among all cancers, lung cancer is the leading cause of deaths in the world, and as it is a unique cancer, the number of patients steadily increases. Air pollution (almost half of all females with lung cancer are attributed to kitchen oil fumes) and the increased smoking population (70% of males and 30% of females with lung cancer are attributed to smoke damage) are the key factors in the steadily climbing number of lung cancer cases (Ministry of Health and Welfare, 2015b). It is indicated that, regarding nonsmoking lung cancer patients, chronic exposure to cooking oil fume environments is an important risk factor (Taiwan Clinical Oncology Research Foundation, 2011).

In addition, long–term exposure to cooking oil fume environments is significantly related to lung cancer and engaging in the cooking process without a range hood has higher correlation (Wu, 2004; Wu and Pan, 2014). Wu and Pan (2014) also indicated that the lung cancer standard incidence ratio of technicians of Chinese culinary to the control group is 1.35. Mu *et al.* (2013) reported that the ratio of the lung cancer risks of nonsmoking women who do not use a range hood during cooking to those who use a range hood during cooking in Taiyuan, China for over 10 years is 1.78. According to the findings of cooking exposure, the lung cancer risk of a kitchen without a range hood is higher than that with a range hood by 3.7–4.8 times. The high lung cancer risk; therefore, is highly correlated with cooking oil fume (Ko and Lee, 1997; Lee and Ko, 2000).

Sun *et al.* (2007) indicates that edible oil heated at high temperature produces a great deal of oil fume, which contains complex chemical substances, including volatile organic compounds (VOCs), polycyclic aromatic hydrocarbons (PAHs), aromatic amine and long–chain aldehydes, and Benzene pyrene (BaP) of PAHs, which cause an inflammatory reaction, reduce antioxidant enzyme activity, and increase oxidative stress and cell proliferation. Most of which are toxic and may be correlated with cancer incidence (Chang *et al.*, 2005; Dung *et al.*, 2006; Hung *et al.*, 2007). Lee *et al.* (2007) reported that exposure to kitchen oil fume may be correlated with the survival and proliferation of pulmonary adenocarcinoma cells, as oil fume particles are 0.15–0.56 μm, which are easily to be inhaled. There are no apparent symptoms at the initial stage of lung cancer. Once a person feels bad and the lung cancer is detected, the golden time for

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treatment has been missed; therefore, lung cancer has high global incidence, and the highest mortality rate (American Cancer Society, 2015).

Wood–based activated carbon fibers (WACFs) prepared from wood pulp or recycled paper waste as a precursor have the natural fiber form and surface pore structure of wood, and there are monolayer and multilayer adsorption phenomena on the pore wall. Furthermore, WACFs prepared by steam activation have larger specific surface area and adsorbability than traditional granular, powdered, or commercial carbons, as well as can be applicable to gas/liquid adsorption. WACFs also have excellent adsorbability, and the Ames test result shows it is free of cytotoxicity and mutagenicity (Lin *et al*., 2015a). Lin *et al*. (2015b) also indicated that toxicity test results showed no adverse effect on animals fed with WACFs for 28 days, meaning there was no the safety problem in the food. In addition, according to Brunauer–Deming–Deming–Teller (BDDT) classification (Gregg and Sing, 1982), WACFs are presented in Type Ⅳ, and the monolayer and multilayer adsorption phenomena are generated in the pore wall, which are applicable to gas/liquid adsorption (Lorenc–Grabowska and Gryglewicz, 2007).

Therefore, this study prepared wood pulp into WACFs that made with the fiber material of wood pulp into WACFP to investigate the absorbability for the oil fume derived from the adsorbed oil by high temperature heating. Methanol was used as a solvent to extract the oil fume absorbed by WACFP, and this oil fume methanol extracts were obtained after decompression concentration for Ames test, including cytotoxicity and mutagenicity, in order to evaluate the carcinogenicity for the human body. The extracts were analyzed by GC–MS to realize the absorbed toxic substances. Hopefully, this study intends to use WACFP as an oil fume absorbing material to provide an option to prevent lung cancer in oil fume kitchen environments.

MATERIALS AND METHODS

Test materials

Wood pulp

Nadelholz unbleached kraft pulps (NUKP) and Laubholz unbleached kraft pulps (LUKP) were supplied from Cheng Loong Pulp, Taiwan.

Peanut oil

It was bought from the Chiayi City Hsinchang Cooking Oil Co.

Test strains

Salmonella typhimurium (*S. typhimurium*), including TA98 and TA100, was bought from the Bioresource Collection and Research Center, Food Industry Research and Development Institute.

Rat liver mixture

The rat liver mixture (S9) (Organ Teknika Co., Switzerland) was prepared from Sprague–Dawley male rats treated with Aroclor 1254.

Experimental methods

Preparation of Wood–based activated carbon fibers (WACFs)

60 g LUKP by absolute dry weight was carbonized for the first stage at nitrogen flow 200 mL/min, carbonization temperature of 850°C, and heating rate 10°C/min. Stage Ⅱ activation was implemented at steam flow 90 mL/h and activation temperature of 850°C for 60 min. Finally, it was cooled at nitrogen flow 200 mL/min for 4 h, and removed at normal temperature to obtain WACFs. The yield of WACFs–L850 (specimen code) is 14.32%; the iodine value is 1007.43 mg/g; the BET specific surface area is about 775 m^2/g. According to Brunauer–Deming–Deming–Teller (BDDT) classification (Gregg and Sing, 1982), WACFs–L850 is presented in Type Ⅳ (Lin *et al*., 2015a).

Wood–based activated carbon fibers paperboard (WACFP) making method

The basis weight of WACFP was 180 and 360 g/m^2, NUKP 20% and LUKP 80% were mixed using the beater method, mixed with 0, 10, and 40% WACFs–L850 by weight with a cylinder paper machine, referring to the CNS11212 method of making handmade paper for physical testing. The WACFP codes are WACFP–180 or –360–L10 or –L40, and the control group is PBO–180 or –360 (without WACFs). The above making methods are referred by Lin *et al*. (2015a).

Determination of moisture content of WACFP

The moisture content in WACFPs was determined by referring to CNS 3086 method of test for determination of moisture content in pulp, paper and board (Oven–drying method).

Smoke point determination

Referring to the measuring method of the American Oil Chemists' Society (AOCS) (1980), in an unventilated and light–proof environment, the oil (peanut oil) was heated and observed visually, and the temperature at which the peanut oil emits thin fume was the smoke point.

Oil fume absorption test for WACFP

The measuring methods of Wu and Yen (2000) and Lee *et al*. (2007) are referred to and modified partially. 200 g of peanut oil was poured in a flat pan with a diameter of 25 cm, heated by an electromagnetic oven to the smoke point and monitored by a thermometer. The oil temperature was controlled at smoke point ± 3°C, the peanut oil was extracted by an oil vacuum pump (Oil pump, ROCKER Tanker 150) and heated to the smoke point. The absorption equipments for the oil fume are shown in Fig. 1. WACFP with different proportions of WACFs and PBO were used as the absorbing material during extraction. The absorption area of WACFs and PBO was 19.63 cm^2, 50 cm above the oil surface, and the oil fume was adsorbed at 142 L/min. The WACFP and

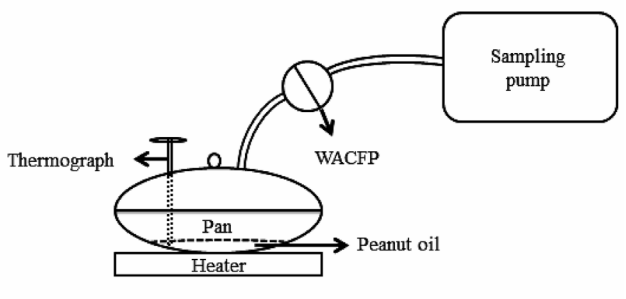

Fig. 1. Adsorption equipments of peanut oil fume.

PBO were measured at 0.5, 1, 1.5, 2, 3, 4, 5, 10, 15, 20, 25, 30, 35, 40, 45, 50, 55, 60, and 70 min detection points (time of heating procedure), respectively. Afterwards, it was measured every 10 min, where each absorbance point was calculated and tested by Duncan's multiple range test analysis, in order to ensure each WACFP reaches absorption saturation. The computing equation is oil fume absorption (g/cm^2) = (WACFP or PBO absorption weight – air–dried weight of WACFP or PBO) / (WACFP or PBO absorption area)

Collection and compounds extraction of oil fume

As oil products deteriorate if heated too long (Ichikawa, 2012), the subsequent Ames test and compounds analysis is influenced. In this experimental, the peanut oil, WACFP and PBO; therefore, were altered after 10 min absorption. The measuring methods of compounds extraction of oil fume are referred to (Wu and Yen, 2000; Lee *et al*., 2007). Besides, according to the results of Wu (1997), using methanol as the solvent to extract peanut oil fume results in the maximum yield. Hence, the PBO and various WACFPs collected from oil fume were extracted in the dark by 200 mL methanol at room temperature, and shaken in a digital rotary shaker (Orbital shaker, YIH DER TS–585D) at 100 rpm/min during extraction. The methanol extraction liquid was obtained after 24 h, and then, the methanol in the extraction liquid was concentrated to dry by a rotary decompression concentrator (Rotatory vacuum evaporator, Heidolph LABOROTA 4000) at 40°C of temperature. The end products were methanol extracts.

Mutagenicity of oil fume methanol extracts

The Ames test, as proposed by Ames *et al*. (1975) and Maron and Ames (1983), which is also known as the *Salmonella* Reversion Assay, uses a microbial system to test the gene mutation of the specimens, where the mutagenic capability can be rapidly and simply detected, including cytotoxicity and mutagenicity tests. The *S. typhimurium* TA98 and TA100 are used as test strains. TA98 results in a strain sensitive to frame–shifted mutation for specific mutagen, while TA100 is used to test the base substitution mutated strain (Mortelmans and Zeiger, 2000). In addition, some mutagenic substances have no mutagenicity until in vivo metabolism is activated; thus, liver enzyme S9 is added (+S9) to simulate the in vivo metabolism (Ames *et al*., 1973). If the tested specimens for mutagenicity testing is cytotoxicity to the strain, the test results is influenced, thus, cytotoxicity testing have to be implemented first, in order to observe the effect of the specimen on strain growing ability.

Cytotoxicity

0.01, 0.05 and 0.10 mg/plate of peanut oil fume were put in the test tube and mixed with 0.1 mL phosphate buffer saline (pH 7.4) and 0.1 mL *S. typhimurium* TA98 and TA100, and cultured overnight oxoid nutrient broth No. 2. If there was additional S9 mix, the aforesaid addition level was changed to 0.2 mL phosphate buffer saline, 0.1 mL *S. typhimurium* TA98 and TA100 and cultured overnight in nutrient broth and 0.5 mL S9 (with S9 mix) or zero S9 (without S9 mix). Afterwards, the test tube was pre–cultured at 37°C for 20 min. The mixed diluent was taken out, and then, 1 mL diluent was put in the plate, mixed with nutrient agar and shaken up. When the mixture solidified, the plate was placed in an incubator at 37°C for 48 h. The colony (bacterial) count was calculated; if the bacterial count of the test group (including with or without S9 mix) was greater than the bacterial count of the control group by 80% (the bacterial count rate, Survival; %), there was no cytotoxicity Ames *et al*. (1975). The survival of cytotoxicity is calculated as follows:

Survival (%) = (the bacterial count of test group / the bacterial count of control group) × 100

Mutagenicity

The mutagenicity is analyzed by using the method proposed by Maron and Ames (1983). The test oil fumes for this mutagenicity test, with or without S9 mix, are the same as for the cytotoxicity test. 0.1 mL phosphate buffer, 0.1 mL *S. typhimurium* TA98 and TA100 cultured overnight in nutrient broth were put into the test tube, mixed with 0.5 mL phosphate buffer saline and cultured at 37°C for 20 min. The test mixture was mixed with 2 mL Molten Top Agar (including 0.05 mM L–histidine, 0.05 Mm Biotin and 0.09 M NaCl) uniformly; the nutrient agar then was poured into the plate. When the mixture solidified, the plate was put in the incubator at 37°C for 48 h, and then, the colony count was calculated. In addition, the phosphate buffer saline is only a pair of control groups (Blank). If the colony count of the TA98 and TA100 test groups is greater than that for the control group by more than two times, the specimen has mutagenicity. In other words, the Mutagenicity ratio = induced revertants per plate/spontaneous revertants per plate (Blank).

Oil fume extracts compounds analysis

Referring to the method of Ron and Louisa (1972), the oil fume methanol extracts obtained was dissolved by 2 mL n–hexane, separated by 10 mL 0.1 N HCl and 0.1 N NaOH, respectively, and concentrated to obtain the n–hexane soluble and insoluble fractions. Afterwards, the n–hexane soluble fraction was dissolved and diluted with n–hexane 1000 times, filtered through 0.22 μm filter membrane, and put in the gas chromatography–mass

spectrometry (GC–MS, GC 6890; MS 5973) for compounds analysis. The initial temperature was set as 40°C, which was kept for 1 min, then increased at 4°C/min to 180°C, increased at 15°C/min to 260°C continuously, and kept for 10 min finally. The shot volume was 1 μL, the injection port temperature was 270°C, the ionization voltage was 70 eV, and the helium gas carrying velocity was 1 mL/min. The oil fume extracts compounds were compared with the standard mass spectrum, and the database is searched via Wiley/NBS, Nation Institute of Standard and Technology (NIST), and combined with the Kovat's index (KI) value for comparative analysis and confirmation. The compositions quantification calculated the content of various compounds according to the gas phase mass spectrogram peak area.

Statistical analysis

The test results are represented by a mean (standard deviation), and the control group and test group are compared by Duncan's Analysis. If the ρ value is smaller than 0.05, meaning a significant difference between the test group and the control group, it is represented by different superscript upper case letters.

RESULTS AND DISCUSSION

Air–dried moisture content of WACFP

The air–dried moisture content of PBO–180 and 360 was 8.56–8.59%, while that of WACFPs was 8.67–8.91% (not tabulated). According to Duncan's multiple range test analysis, there was no significant difference between PBO and the various WACFPs.

Oil fume absorbability

Smoke point

The smoke point of oil and fat is the temperature at which the constituents are decomposed under heat, between the melting point and boiling point. The more free of fatty acid the oil and fat contain, or the shorter the fatty acid carbon chain, the lower the smoke point, which is an important indicator of the refinement and quality of oil and fat. When the oil temperature exceeds this temperature, the quality deteriorates, producing free radicals and polymers that are harmful to the human body (Kao, 2013). The smoke point of the peanut oil in this range of the study was 105±1°C (not tabulated). The oil products with a smoke point lower than 170°C is classified as low smoke point oil and fat, which is only suitable for cold use, dressing with sauce, and boiling. The oil for deep frying must have high smoke point oil and fat at above 200°C (Chen, 2014). The smoke point; therefore, is an important index of choosing oils and fats for cooking food.

Oil fume absorption by WACFP

The absorption results of peanut oil fumes at the smoke points of WACFPs and PBO, with different proportions of WACFs and basis weights (180 and 360 g/m^2), are shown in Fig. 2. According to Duncan's multiple range test analysis, the PBO–180, WACFP–180–L10 and WACFP–180–L40 reached their adsorption saturations at 80, 110, and 120 min, where oil fume absorption was 0.0203, 0.0243, and 0.0304 g/cm^2, respectively. The PBO–360, WACFP–360–L10, and WACFP–360–L40 reached their absorption saturations at 210, 250, and 260 min, respectively, where oil fume absorption was 0.0424, 0.0508 and 0.0604 g/cm^2, respectively. The oil fume absorption of various WACFPs increased with the proportions of WACFs. Take the basis weight of 360 g/m^2 as an example, the oil fume absorption of WACFP–360–L40 were 1.42 and 1.19 times of that of PBO–360 and WACFP–360–L10, meaning the additional percent of WACFs influenced the absorbability. WACFPs reaching their absorption saturation were 30 to 50 min later than PBO–180 and PBO–360. The WACFPs have a relatively long time for absorbing oil fume, and the oil fume absorption of PBO–360, WACFP–360–L10, and WACFP–360–L40, with a basis weight of 360 g/m^2, is higher than that of 180 g/m^2 by 2.0–2.04 times.

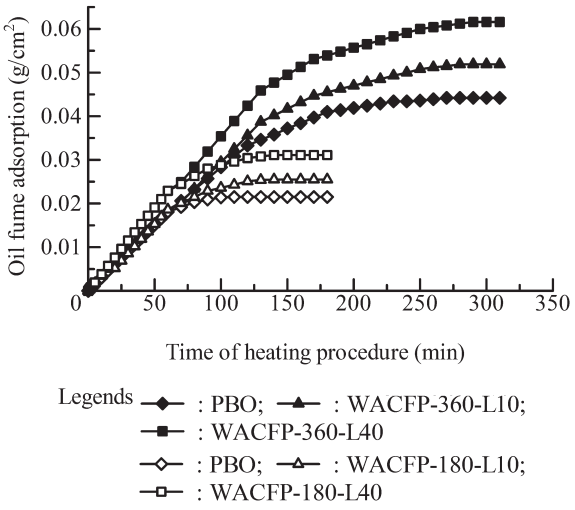

Fig. 2. Oil fume absorption from the smoke point of peanut oil for WACFP–180 and –360.

Yield of methanol extracts

After PBO–360, WACFP–360–L10, and WACFP–360–L40 absorbed peanut oil fume, the yields of the methanol solvent extraction concentrations were 0.41, 0.98, and 1.18%, respectively (not tabulated). The yield increased with the proportions of WACFs. According to Duncan's multiple range test analysis, PBO–360 was significantly different from WACFP–360–L10 and –L40.

Mutagenicity of oil fume methanol extracts

Cytotoxicity

The cytotoxicity results of methanol extracts from the peanut oil fume absorbed by different WACFPs for *S. typhimurium* TA98 and TA100 strains are shown in Table 1. The bacterial count of the blank group (Blank) of *S. typhimurium* TA98 without rat liver mixture (–S9) was 2837, and that of TA100 was 2758. The bacterial count of Blank of *S. typhimurium* TA98 with rat liver mixture (+S9) was 2897, and that of *S. typhimurium*

Table 1. Cytotoxicity of methanol extracts from different wood–based activated carbon fibers paperboard with peanut oil fume toward *Salmonella typhimurium* TA98, TA100 with or without S9 mixture

Specimen	Dose (mg/plate)	–S9				+S9			
		TA98 (cfu/plate)	Survival [3] (%)	TA100 (cfu/plate)	Survival (%)	TA98 (cfu/plate)	Survival (%)	TA100 (cfu/plate)	Survival (%)
Blank[1]	0.00	2837 ± 89 [2]	100	2758 ± 77	100	2897 ± 104	100	2866 ± 86	100
PBO–360	0.01	2838 ± 109	100	2674 ± 65	97	2812 ± 41	97	2871 ± 20	100
	0.05	2774 ± 84	98	2768 ± 75	100	2801 ± 109	97	2850 ± 79	99
	0.10	1337 ± 49	47	1513 ± 131	55	1441 ± 111	50	1634 ± 119	57
WACFP–360–L10	0.01	2924 ± 55	103	2799 ± 112	101	2737 ± 49	94	2866 ± 70	100
	0.05	2846 ± 72	100	2759 ± 81	100	2841 ± 76	98	2737 ± 85	95
	0.10	1325 ± 87	47	1245 ± 116	45	1386 ± 45	48	1381 ± 101	48
WACFP–360–L40	0.01	2770 ± 102	98	2768 ± 22	100	2693 ± 17	93	2865 ± 25	100
	0.05	2843 ± 110	100	2830 ± 112	103	2834 ± 53	98	2751 ± 47	96
	0.10	1405 ± 44	50	1525 ± 140	55	1440 ± 62	50	1567 ± 14	55

[1] Blank : Without any WACFP
[2] Mean ± standard deviation.
[3] Survival (%) = (Colony number of specimen/ Colony number of blank)*100

TA100 was 2866. Waleh *et al*. (1982) reports that the bacterial survival rate (Survival, %) of the specimen after testing is higher than the blank group by over 80%, meaning the specimen is non–toxic to cells (no cytotoxicity), and the bacterial survival rate is lower than 80%, which represents a toxic reaction with cells (with cytotoxicity).

In addition, the *S. typhimurium* TA98 (–S9) bacterial counts of the methanol extracts of PBO–360, WACFP–360–L10, and WACFP–360–L40, at test concentrations of 0.01 and 0.05 mg/plate, were 2770–2924 and 2774–2846 cfu/plate, respectively, while Survival were 98–103 and 98–100%, respectively. The *S. typhimurium* TA100 (–S9) bacterial counts of methanol extracts at test concentrations of 0.01 and 0.05 mg/plate were 2674–2799 and 2768–2830 cfu/plate, respectively, and Survival were 97–100 and 100–103%, respectively. The *S. typhimurium* TA98 (+S9) bacterial counts of methanol extracts at test concentrations of 0.01 and 0.05 mg/plate were 2693–2812 and 2801–2841 cfu/plate, and Survival were 93–97 and 97–98%. The *S. typhimurium* TA100 (+S9) bacterial counts of methanol extracts at concentrations of 0.01 and 0.05 mg/plate are 2865–2871 and 2737–2850 cfu/plate, and Survival were 100 and 95–99%. The above results show that the *S. typhimurium* TA98 and TA100 bacterial survival rates of the methanol extracts at concentrations of 0.01 and 0.05 mg/plate, with or without S9, are higher than 80%, representing no cytotoxicity.

However, Survival of the *S. typhimurium* TA98 (–S9) and TA100 (–S9) of the methanol extracts of PBO–360, WACFP–360–L10, and WACFP–360–L40 at a test concentration of 0.10 mg/plate were 47–50% and 45–55%, respectively; and Survival of methanol extracts at the same concentration were 48–50 and 48–57%, representing cytotoxicity with cells (Waleh *et al*., 1982). This suggests that the methanol extracts at concentrations lower than 0.05mg/plate have no cytotoxic reaction with S. *typhimurium* TA98 (±S9) or TA100 (±S9), the which can be used for mutagenicity testing.

Mutagenicity

To investigate the gene mutation caused by various methanol extracts, Table 2 shows the mutagenicity results of methanol extracts from peanut oil fume, as absorbed by different proportions of WACFPs at concentrations lower than 0.050 mg/plate for *S. typhimurium* TA98 and TA100 strains. The revertant count of the blank group (Blank) of *S. typhimurium* TA98 (–S9) was 51 revertants/plate, while that of *S. typhimurium* TA100 was 151 revertants/plate. The revertant count of Blank of *S. typhimurium* TA98 (+S9) was 48 revertants/plate, while that of *S. typhimurium* was 167 revertants/plate. In addition, the *S. typhimurium* TA98 (–S9) revertant counts of methanol extracts of PBO–360, WACFP–360–L10, and WACFP–360–L40, at test concentrations of 0.005 and 0.010 mg/plate, were 51–58 and 68–78 revertants/plate, respectively, and the Mutagenicity ratios were 0.99–1.14 and 1.34–1.53. The *S. typhimurium* TA98 (+S9) revertant counts of methanol extracts at test concentrations of 0.005 and 0.010 mg/plate were 51–53 and 78–84 revertants/plate, respectively, while the Mutagenicity ratio was 1.06–1.10. The above results show that the Mutagenicity ratio of *S. typhimurium* TA98 of methanol extracts at concentrations of 0.005 and 0.010 mg/plate, with or without S9, was lower than 2, representing no mutagenicity. As Ames *et al*. (1975) indicated, if the His+ revertants/plate induced by the specimen is higher than the spontaneous revertants by over two times, or is dose dependent, it means it has mutagenicity.

The revertant counts of *S. typhimurium* TA100 (–S9) and TA98 (+S9) of methanol extracts at test concentration of 0.005 mg/plate were 153–157 and 160–169

Table 2. Mutagenicity of methanol extracts from different wood–based activated carbon fibers paperboard with peanut oil fume toward *Salmonella typhimurium* TA98, TA100 with or without S9 mixture

Specimen	Dose (mg/plate)	–S9				+S9			
		TA98 (revertants/plate)	Mutagenicity ratio [3]	TA100 (revertants/plate)	Mutagenicity ratio	TA98 (revertants/plate)	Mutagenicity ratio	TA100 (revertants/plate)	Mutagenicity ratio
Blank[1]	0.00	51 ± 2^{aA} [2]	1.00	151 ± 7^{aB}	1.00	48 ± 1^{aA}	1.00	167 ± 3^{aC}	1.00
PBO–360	0.005	58 ± 4^{bA}	1.14	153 ± 3^{aB}	1.01	51 ± 3^{aA}	1.07	169 ± 5^{aC}	1.01
	0.010	78 ± 9^{cA}	1.53	188 ± 9^{bB}	1.24	84 ± 6^{bA}	1.76	570 ± 11^{bC}	3.41
	0.050	860 ± 23^{dA}	16.87	1279 ± 14^{cB}	8.47	889 ± 34^{cA}	18.51	1381 ± 19^{cC}	8.27
WACFP–360–L10	0.005	51 ± 3^{aA}	0.99	156 ± 6^{aB}	1.04	53 ± 4^{aA}	1.10	161 ± 9^{aB}	0.96
	0.010	68 ± 6^{bcA}	1.34	181 ± 9^{bB}	1.20	78 ± 8^{bA}	1.63	538 ± 22^{bC}	3.22
	0.050	841 ± 18^{dA}	16.50	1288 ± 16^{cC}	8.53	920 ± 27^{cB}	19.17	1355 ± 50^{cC}	8.11
WACFP–360–L40	0.005	55 ± 2^{aA}	1.08	157 ± 9^{aB}	1.04	51 ± 4^{aA}	1.06	160 ± 6^{aB}	0.96
	0.010	71 ± 2^{cA}	1.40	182 ± 5^{bB}	1.20	78 ± 10^{bA}	1.63	575 ± 27^{bC}	3.44
	0.050	836 ± 25^{dA}	16.40	1273 ± 54^{cB}	8.43	864 ± 37^{cA}	18.00	1364 ± 85^{cB}	8.17

[1] Blank: Without any WACFP

[2] Mean ± standard deviation; separation within lines by Duncan's multiple range tests at 5% significant level. The horizontal axis is A, B and C; The vertical axis is a, b, c and d.

[3] Mutagenicity ratio = Induced revertants pre plate / Spontaneous revertants pre plate

revertants/plate, while the Mutagenicity ratios were 1.01–1.04 and 0.96–1.01, respectively. When the concentration was 0.01 mg/plate, the revertant count of *S. typhimurium* TA100 (–S9) was 181–188 revertants/plate, and the Mutagenicity ratio was 1.20–1.24. However, when S9 was added, the revertant count was 538–575 revertants/plate, and the Mutagenicity ratio was 3.41–3.44. Therefore, the mutagenicity of methanol extracts at a test concentration of 0.01 mg/plate appears after metabolic activation by S9. In addition, the revertant counts of *S. typhimurium* TA98 (–S9) and TA100 (–S9) of the methanol extracts of PBO–360, WACFP–360–L10, and WACFP–360–L40, at a test concentration of 0.050 mg/plate, were 836–860 and 1273–1288 revertants/plate, respectively, and the Mutagenicity ratios were 16.40–16.87 and 8.43–8.53. The *S. typhimurium* TA98 (+S9) and TA100 (+S9) revertant counts of methanol extracts at a concentration of 0.05 were 864–920 and 1355–1381 revertants/plate, while the Mutagenicity ratios were 18.00–19.17 and 8.11–8.27. Therefore, the Mutagenicity ratios of *S. typhimurium* TA98 (±S9) and TA100 (±S9) of methanol extracts at concentrations of 0.05 mg/plate were higher than two times, meaning the methanol extracts have very high mutagenicity for *S. typhimurium* TA98 and TA100, and the mutagenicity for *S. typhimurium* TA100 is higher than that of TA98, as well as there is significant difference.

Methanol extracts compounds analysis

Edible oil plays one of the important roles in daily life, as it not only provides the human body with the required calories and oil soluble vitamins, and it also enhances the flavor, color, and texture of food. However, oils and fats are oxidized, hydrolyzed, isomerized, polymerized, and cracked by high temperature heating, and the generated oil fume contains many compounds. Generally speaking, the primary products are aldehydes, ketones, hydrocarbons, fatty acid, aromatic compounds, and heterocyclic compounds (Tung, 2000; Chen, 2005). Chiang *et al*. (1997) indicates that the polycyclic aromatic compounds, meaning aldehydes and ketones in the oil fume, may have genotoxicity. The edible oil fume has mutagenicity (Wu and Yen, 2000; Tung, 2003).

According to the mutagenicity results of the oil fume methanol extracts, the concentration 0.050 mg/plate has mutagenicity for *S. typhimurium* TA98 (±S9) and TA100 (±S9) (Table 2). Therefore, the n–hexane soluble fraction was obtained by dissolving the oil fume methanol extracts with n–hexane, and diluted 1000 times with n–hexane. The GC–MS analysis results are shown in Table 3.

The main compounds of methanol extracts from peanut oil fume included Alkenals, Amine, Ester, Alkane, Alcohol, Aldehyde, Phenol, Ketones, Alkene, Polycyclic aromatic hydrocarbons, and Acid,. The relative contents were 54.21–67.89, 0.00–19.78, 11.48–17.31, 5.48–5.86, 1.34–3.20, 0.29–2.62, 0.97–1.68, 1.27–1.59, 0.36–0.50, 0.23–0.50, and 0.00–2.40%, respectively. The trans, trans–2,4–decadienal (t–t–2,4–DDE) had the maximum relative content, which was 54.21–58.41%. This result matched the findings of Wu (1997), Wu (2004), and Lo (2007) which indicate that t–t–2,4–DDE has the highest content of peanut oil fume (51.6%), and the findings of Wu (1997) shows that t–t–2,4–DDE has mutagenicity when the concentration is 0.010 mg/plate. Therefore, t–t–2,4–DDE is the main mutagenic constituent of peanut oil fume. The findings of Tung (2003) indicates that the main alkenal t–t–2,4–DDE of edible oil fume can induce

Table 3. Comparison of GC–MS results among each n–hexane extracts separated from different wood–based activated carbon fibers paperboard with peanut oil fum

	Compounds	PBO	WACP–360–L10	WACP–360–L40
Alkenals	(E,Z)–2,4–decadienal	11.37[1)]	–[2)]	–
	Trans–trans–2,4–decadienal	56.52	54.21	58.41
Amine	Hexadecanamide	–	3.70	3.11
	Oleamide	–	16.08	12.59
Ester	Bis (O,O–diethyl phosphorodithioato–S,S')–	1.18	–	–
	Methyl palmitate	5.59	3.77	3.57
	Dibutyl Phthalate	2.14	1.63	1.54
	13–Octadecenoic acid methyl ester	6.77	6.96	5.61
	Bis(2–ethylhexyl) Phthalate	1.63	0.36	0.76
Alkane	N–Nonane	–	0.29	0.28
	Decane	1.49	1.37	1.27
	2–methyl undecane	0.41	–	0.32
	Dodecane	1.27	1.03	0.99
	Tridecane	–	1.30	1.27
	Tetradecane	1.25	1.08	1.06
	Cyclododecane	0.22	–	–
	Hexadecane	0.84	0.71	0.67
Alcohol	Cedrol	0.81	0.21	0.48
	1–Heptadecanol	1.27	1.13	1.08
	Parasiticol	1.12	–	–
Aldehyde	Nonanal	–	2.62	2.53
	Decanal	0.29	–	–
Phenol	2,6–Di–tert–butyl–4–methylphenol	0.97	1.18	1.13
	Nonylphenol	–	0.24	0.55
Ketones	Hexahydrofarnesylacetone	–	0.20	0.46
	7,9–Di–tert–butyl–1–oxaspiro(4,5) deca–6,9–diene–2,8–dione	1.59	1.07	0.92
Alkene	Cyclodecene	–	–	0.25
	1–Octadecene	0.17	–	–
	Squalene	0.31	0.36	0.25
Polycyclic aromatic hydrocarbons	Naphthalene	0.39	0.50	0.23
Acid	Palmitic acid	–	–	0.67
	3–(4–N,N–Dimethylaminophenyl) propenoic acid	2.40	–	–

[1)] Relative amount of compounds (%)
[2)] – non detected

the apoptosis of human pulmonary adenocarcinoma cell strain. Wu (2006) indicates that 60% of lung cancer patients in Taiwan are pulmonary adenocarcinoma patients, and that women and cooks are more likely to get pulmonary adenocarcinoma. Therefore, t–t–2,4–DDE is the main mutagenic compounds of peanut oil fume, and may be one of the causes of human pulmonary adenocarcinoma.

CONCLUSION

The air–dried moisture content of WACFPs was 8.40–8.92%, and the smoke point of peanut oil was 105±1°C. The peanut oil fume absorption saturation of PBO–180, WACFP–180–L10, and WACFP–180–L40 at the smoke point was 0.0203–0.0304 g/cm^2; and for WACFP–360–L40, it was 0.0424–0.0604 g/cm^2. This sug-

gests the oil fume absorption of WACFPs increases with the proportions of WACFs, and the duration to absorption saturation is relatively long. The methanol extraction yield was 0.41–1.18%. According to the cytotoxicity results, methanol extracts at test concentrations higher than 0.10 mg/plate have cytotoxic reaction with *S. typhimurium* TA98 (±S9) and TA100 (±S9). According to the mutagenicity results, the Mutagenicity ratios of *S. typhimurium* TA98 (±S9) and TA100 (±S9) of methanol extracts at a test concentration of 0.050 mg/plate were higher than two times, meaning methanol extracts had very high mutagenicity. According to GC–MS analysis, trans–trans–2,4–decadienal (t–t–2,4–DDE) was the main mutagenic compounds of peanut oil fume.

AUTHOR CONTRIBUTION

Han Chien LIN designed this study, performed the experiments, analyzed the data and the statistical analysis and wrote the paper. Noboru FUJIMOTO participated in the design of the study and supervised the works. Both authors assisted in editing of the manuscript and approved the final version.

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