HIGH-TEMPERATURE FRYING

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1. Exposure Data

1.1 Definition

'Cooking fumes' or 'cooking oil fumes' is the term commonly used to describe the visible emissions generated during cooking by frying with oil. However, these emissions are not technically 'fumes'. In occupational and environmental hygiene, 'fumes' are defined as submicron-sized solid particles (particulate matter) created by the cooling of hot vapour. During cooking, such vapour is formed when the cooking oil is heated above its boiling point. In addition to this ultrafine particulate matter, cooking, especially frying and grilling, generates aerosol oil droplets, combustion products, organic gaseous pollutants, and steam from the water contents of the food being cooked.

1.2 Constituents of cooking fumes

Cooking, in particular frying, generates substantial amounts of airborne particulate matter (PM), which includes ultrafine particles (UFP) and fine PM ($PM_{2.5}$), and is a major contributor to their indoor levels. In addition, particles created during cooking have organic substances adsorbed on their surface. These include polycyclic aromatic hydrocarbons (PAHs) and heterocyclic amines. Certain gaseous pollutants such as formaldehyde (IARC, 2006), acetaldehyde (IARC, 1999), acrylamide (IARC, 1994) and acrolein (IARC, 1995) are also produced during cooking.

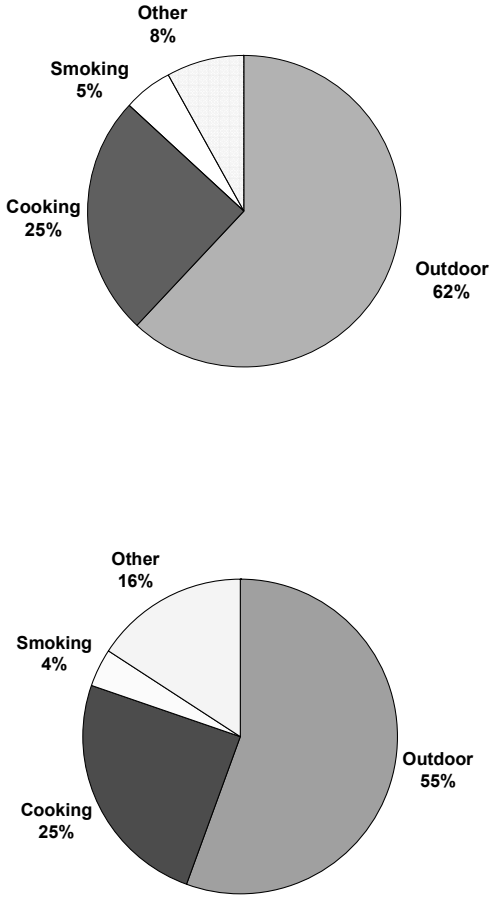
The concentrations of these constituents measured in cooking fumes in field and controlled studies are presented below.

1.2.1 *Ultrafine and fine particulate matter*

The Particle Total Exposure Assessment Methodology (PTEAM) study was carried out by the Research Triangle Institute and the Harvard University School of Public Health in the USA in 1989–90 (Clayton *et al.*, 1993). Particle concentrations were measured for a probability-based sample of 178 nonsmokers who represented the non-institutionalized population of Riverside, CA (~139 000 persons). Personal samples of PM_{10} were taken;

the indoor and outdoor samples included both PM_{10} and $PM_{2.5}$. Cooking produced both fine and coarse particles. Homes where cooking took place during monitoring (about 55%) had average PM_{10} concentrations ~20 μg/m^3 higher than those where no cooking took place (Özkaynak *et al.*, 1996a,b). The proportion of $PM_{2.5}$ and PM_{10} due to cooking was 25% for both particle sizes (Figure 1.1). However, when considered as a fraction of particles due to indoor sources alone, the proportion was 65% and 55%, respectively (Özkaynak *et al.*, 1996b).

Figure 1.1 Fraction of $PM_{2.5}$ due to cooking (top); fraction of PM_{10} due to cooking (bottom).



A large-scale study of personal, indoor and outdoor exposures was undertaken for more than 100 persons living in Seattle, WA, USA (Seattle Study; Liu *et al.*, 2003). Based on 195 cooking events, the average $PM_{2.5}$ concentration due to cooking was estimated to be 5.5 (standard error [SE], 2.3) μg/m^3 (Allen *et al.*, 2004).

A study of personal, indoor and outdoor exposure to $PM_{2.5}$ and associated elements was carried out on 37 residents of the Research Triangle Park area in North Carolina, USA (Research Triangle Park Study; Wallace *et al.*, 2006a,b). Burned food added an average of 11–12 μg/m^3 to the indoor concentration (Wallace *et al.*, 2006b). In continuous measurements, the mean estimated $PM_{2.5}$ personal exposures during more than 1000 h of cooking were found to be 56 μg/m^3 higher than background (Wallace *et al.* 2006b). The 24-h average increase due to cooking was about 2.5 μg/m^3. A different analysis of the results from this study concluded that cooking contributed 52% of personal exposure to $PM_{2.5}$ and more than 40% of the indoor concentration of $PM_{2.5}$ (Zhao *et al.*, 2006).

A long-term study of indoor and outdoor particle concentrations was carried out between 1997 and 2001 in an occupied townhouse in Reston, VA, USA (Reston, VA Townhouse Study). Cooking produced about an order of magnitude higher number of the smallest UFP (10–50 nm) and from 1.2- to 9.4-fold higher levels of the larger particles compared with identical times when no cooking occurred (Table 1.1; Wallace *et al.*, 2004). The mean mass concentration increased at dinner (4-h averages) from 3.7 μg/m^3 to 11.8 μg/m^3 assuming a density of combustion particles of 1 g/cm^3. About 70% of the particles emitted during dinnertime were <0.05 μm.

Table 1.1. Number and concentration of $PM_{2.5}$ during dinnertime cooking compared with no cooking

Size (μm)	Dinnertime cooking		No cooking	
	Mean	SE	Mean	SE
Number[a]				
0.010–0.018	6472	165	465	10
0.018–0.05	13363	342	1507	22
0.05–0.1	7085	221	1701	29
0.1–0.2	2226	77	807	11
0.2–0.3	277	10	128	1.4
0.3–0.5	76	3	16	0.15
0.5–1	5	0.086	1.3	0.015
1–2.5	1	0.016	0.15	0.0016
Concentration (μg/m^3)				
0.010–0.018	0.0085	0.0002	0.0006	0.00001
0.018–0.05	0.3	0.01	0.039	0.0006
0.05–0.1	1.4	0.04	0.4	0.006
0.1–0.2	2.9	0.10	1.1	0.014
0.2–0.3	2.6	0.12	1.2	0.013
0.3–0.5	2.4	0.05	0.5	0.005

Table 1.1. (contd)

Size (μm)	Dinnertime cooking		No cooking	
	Mean	SE	Mean	SE
Concentration (μg/m^3) (contd)				
0.5–1	0.8	0.01	0.2	0.003
1–2.5	1.4	0.04	0.3	0.003
Sum ($PM_{2.5}$)	11.8		3.7	

From Wallace *et al*. (2004)
PM, particulate matter; SE, standard error
[a] No. of samples between 2400 and 12 800

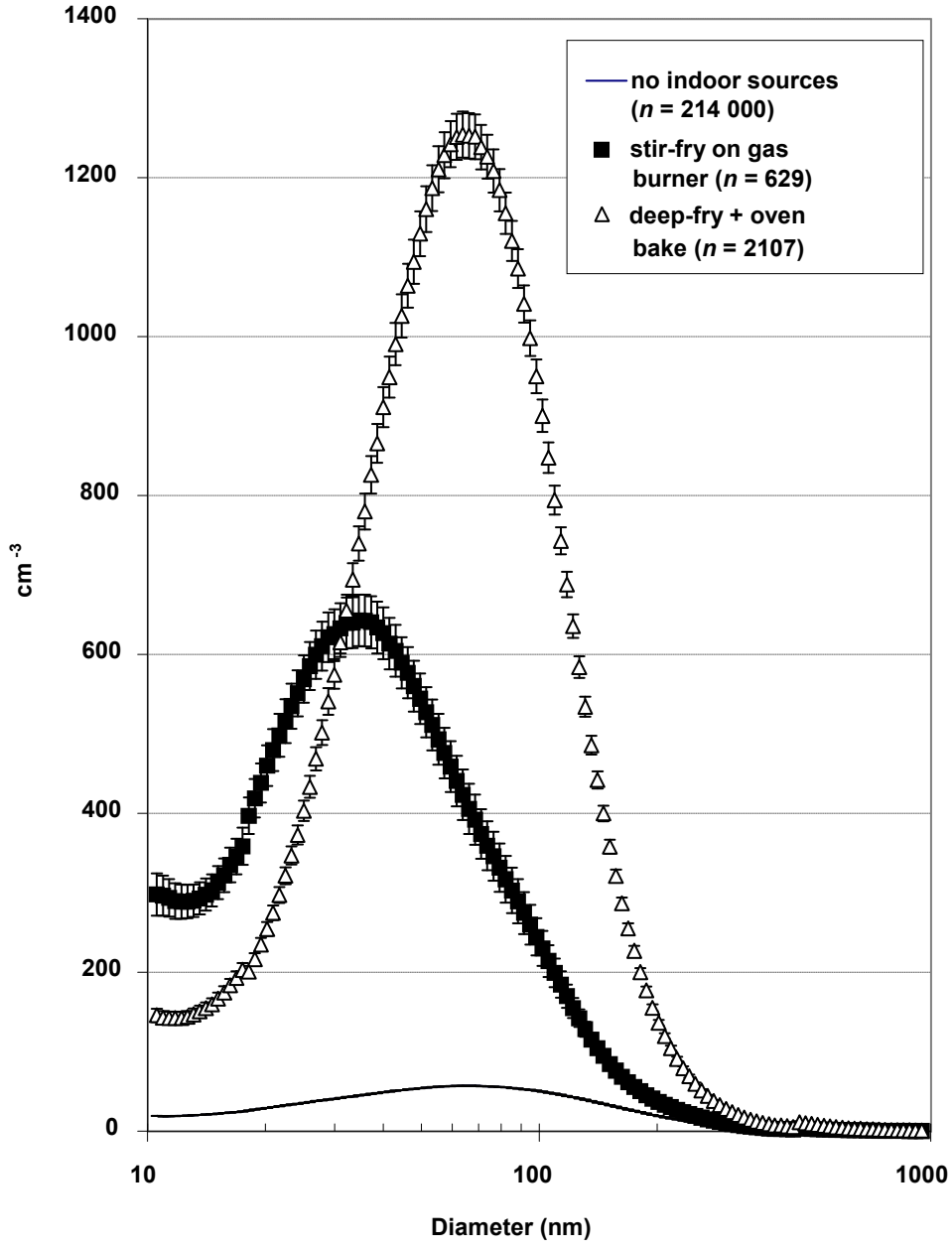
In a more detailed analysis, 44 high-particle-production (frying, baking, deep-frying) cooking episodes on a gas stove were assessed (Wallace *et al.*, 2004). Most of the particles were in the ultrafine range, but the largest volume was contributed by particles between 0.1 μm and 0.3 μm in diameter. The total particle volume concentration created by the 44 high-particle-production cooking events averaged a little more than 50 (μm/cm)^3, corresponding to an average concentration of about 50 μg/m^3, about an order of magnitude higher than average values for all types of cooking combined.

The size distribution of ultrafine particles during cooking was studied by Wallace (2006) and Ogulei *et al.* (2006). Stir-frying using one gas burner produced a peak of PM ~35 nm, whereas deep-frying using one gas burner followed by baking in the oven produced a peak about twice as high and at a diameter of 64 nm (Figure 1.2).

Brauer *et al.* (2000) reported $PM_{2.5}$ concentrations in the range of 24–201 g/m^3 in residential kitchens during frying, with peak $PM_{2.5}$ concentrations above 400 μg/m^3. Kamens *et al.* (1991) estimated that 5–18% of an 8-h personal particle exposure could be attributed to cooking one meal in one of three homes that they studied.

Abt *et al*. (2000) studied 17 selected cooking events in three homes that provided mean peak volume concentrations of particles between 20 and 500 nm ranging between 29 and 57 (μm/cm)^3. Long *et al.* (2001) studied nine homes for 6–12 days each and found mean peak volume concentrations for UFP (20–100 nm) of 2.2–18.2 (μm/cm)^3. He *et al*. (2004a) studied 15 homes for 48 h during cooking under good and poor ventilation conditions and found a range of peak submicrometer number concentrations for cooking events between 16 000 and 180 000 particles/cm^3. Estimates of the emission rate ranged between 0.2–4 × 10^{12} particles/min. Finally, 24 cooking events with high concentrations and well-shaped decay curves, including concurrent air exchange rate measurements, were analysed more accurately, taking into account losses due to deposition during the lag time required to reach the peak, for their source strengths (Wallace *et al.*, 2004). A value of 3×10^{12} UFP/min was obtained.

Figure 1.2. Size distribution of ultrafine particles from cooking. *n* = number of 5-min measurements. Error bars are standard errors. Stir-frying on one gas burner produced a peak at ~35 nm; deep-frying on one gas burner followed by baking in the oven produced a peak at 64 nm that was twice as high.



A study in Amsterdam and Helsinki found that cooking increased $PM_{2.5}$ concentrations by 1.9–3.4 μg/m^3 (14–24%) among two groups of 47 and 37 elderly residents in the two cities, respectively (Brunekreef *et al.*, 2005; the ULTRA Study).

Kleeman *et al.* (1999) used an industrial charbroiling facility to cook >100 hamburgers. The particle mass consisted mainly of organic compounds, with a very small amount of elemental carbon, and a large unknown component. Most of the particle mass came from particles between 0.1 and 0.4 μm in diameter.

Emission rates during cooking with commercial institutional-scale deep-fryers have been reported (Schauer *et al.*, 1998). Professional chefs prepared vegetables by stir-frying in soya bean or canola oil and deep-frying potatoes in oil. Fine particle emission rates were 21.5±1.2, 29.5±1.3 and 13.1±1.2 mg/kg for stir-frying vegetables in the two oils and deep-frying potatoes, respectively. [Emissions during food preparation by a professional chef using large commercial cookers may differ substantially from emissions in a residence.]

In a recent study in a residential setting in Canada (Evans *et al.* 2008), real-time measurements were taken during frying to estimate the time-integrated exposure to PM associated with frying food. The production rates and concentrations of UFP and $PM_{2.5}$ during and at the end of frying a variety of breakfast foods typical of the Canadian diet at medium temperatures were assessed (Table 1.2).

Table 1.2. The production rates and concentrations of UFP and $PM_{2.5}$ during and at the end of frying of various types of foods

Food	Food temperature (°C)[a]	Production rate during frying		Concentration at the end of frying	
		UFP (particles/cm^3 s)	$PM_{2.5}$ (μg/m^3 s)	UFP (particles/cm^3)	$PM_{2.5}$ (μg/m^3)
Bacon	314	45	0.092	$2.2*10^4$	38
Pancakes	297	25	0.17	$2.5*10^4$	55
Peppers and onions	336	78	0.12	$2.0*10^4$	60
Vegetable stir-fry	280	31	ND	$2.0*10^4$	ND
Vegetable mix	249	59	ND	$4.5*10^4$	ND
Fried egg	271	60	ND	$2.5*10^4$	ND
Fried rice	274	6	ND	$1.0*10^4$	ND
Breaded eggplant	280	88	1.1	$8.0*10^4$	1000
Overall		44	0.13		

From Evans *et al*. (2008)
ND, not determined because no elevated $PM_{2.5}$ concentration was observed; PM, particulate matter; UFP, ultrafine particles
[a] Refers to maximum temperature

1.2.2 *Volatile organic compounds*

A large proportion of the vapours generated during cooking is steam from the water contents of the food or from the water used to cook the food. However, during frying (with oil), fatty acid esters that are constituents of edible oils and fat can decompose and produce volatile organic compounds, as well as semi-volatile compounds that can condense to form particles. A wide variety of organic compounds have been identified in cooking emissions, including alkanes, alkenes, alkanoic acids, carbonyls, PAHs and aromatic amines. Felton (1995) reported that the main volatile compounds generated during frying were aldehydes, alcohols, ketones, alkanes, phenols and acids. Of particular concern in relation to carcinogenicity are PAHs, heterocyclic amines and aldehydes.

(a) PAHs

Dubowsky *et al.* (1999) reported peak total particle-bound PAH concentrations in a range from undetectable to 670 ng/m^3 during cooking when measured with a Gossen PAS monitor.

A study in Taiwan found several PAHs in the fumes of three cooking oils (safflower, vegetable and corn oil) (Chiang *et al.*, 1999a).

By contrast, Wallace (2000) did not measure increased concentrations of total PAHs during cooking.

(b) Aldehydes

Schauer *et al.* (1998) reported emissions of 20 100 µg formaldehyde/g of food during stir-frying of vegetables on an institutional-size cooker. They reported emissions of 12 400 µg/g formaldehyde and 20 900 µg/g acetaldehyde during deep-frying of potatoes.

(c) Aromatic amines

One study found the aromatic amines 2-naphthylamine and 4-aminobiphenyl in the fumes of three different cooking oils (sunflower oil, vegetable oil and refined lard) (Chiang *et al.*, 1999b).

(d) Other volatile compounds

Rogge *et al*. (1991) measured the fine aerosol emission rates for single organic compounds from charbroiling and frying hamburger meat. The compounds detected were *n*-alkanes, *n*-alkanoic acids, *n*-alkenoic acids, dicarboxylic acids, *n*-alkanals and *n*-alkenals, *n*-alkanones, alkanols and furans.

Ho *et al.* (2006) studied emissions of 13 carbonyl compounds in cooking exhaust fumes from 15 restaurants in Hong Kong Special Administrative Region, China, and developed a new method of analysis using Tenax coated with a hydrazine compound followed by thermal desorption and mass spectrometry. This allowed them to separate three similar compounds: acetone, acrolein and propanal. The most prevalent compounds were formaldehyde (in all but four of the restaurants), acrolein, acetaldehyde and nonanal,

which accounted for 72% of all carbonyl emissions. Based on a small sample of restaurants, the authors estimated total annual emissions for acrolein, formaldehyde and acetaldehyde of 7.7, 6.6 and 3.0 tonnes per year from cooking compared with 1.8, 10 and 33 tonnes per year, respectively, from vehicles.

1.3 Effect of different parameters of cooking on emissions

The chemical composition of cooking emissions varies widely depending on the cooking oils used, the temperature, the kind of food cooked, as well as the method and style of cooking adopted.

1.3.1 *Effect of the type of oil and temperature*

(a) Mixture of volatile components

Studies were undertaken to identify qualitatively the volatile components emitted during the heating of cooking oils to 265–275°C (Li, *et al.* 1994; Pellizzari *et al.* 1995; Shields *et al.* 1995; Chiang *et al.*, 1999a; Wu *et al.* 1999). The oils tested were rapeseed, canola, soya bean and peanut. The major constituents identified in the oil vapours were saturated, unsaturated and oxygenated hydrocarbons. These studies detected a variety of agents in emissions from heated cooking oils including 1,3-butadiene, benzene, benzo[*a*]pyrene, dibenz[*a,h*]anthracene, acrolein, formaldehyde and acetaldehyde. Emissions were highest for rapeseed oil and lowest for peanut oil. In one study, the emission levels of 1,3-butadiene and benzene were approximately 22-fold and 12-fold higher, respectively, for rapeseed oil than for peanut oil (Shields *et al.*, 1995). Compared with rapeseed oil heated to 275°C, fourfold and 14-fold lower levels of 1,3-butadiene were detected when the oils were heated to 240°C and 185°C, respectively.

(b) PAHs and nitro-PAHs

In a study performed in a controlled environment (Air Resources Board of the State of California Study; Fortmann *et al.*, 2001), five untreated cooking oils were extracted and analysed for PAHs (Table 1.3). All were found to contain some PAHs; olive oil and peanut oil contained generally higher concentrations than rapeseed, corn or vegetable oils.

In a similar study, PAHs levels in samples of five raw cooking oils (canola, olive, corn, soya bean and vegetable oil) were not increased compared with the blank (Kelly, 2001).

Fume samples from three different commercial cooking oils commonly used in Taiwan, China (lard oil, soya bean oil and peanut oil), were collected and tested for PAHs. All samples contained dibenz[*a,h*]anthracene and benz[*a*]anthracene; extracts of fume samples from the latter two also contained benzo[*a*]pyrene (Chiang *et al.*, 1997). In a later study, fume samples from safflower, olive, coconut, mustard, vegetable and corn oil were similarly tested (Chiang *et al.*, 1999a). Extracts of fumes from safflower oil,

vegetable oil and corn oil contained benzo[*a*]pyrene, dibenz[*a,h*]anthracene, benzo-[*b*]fluoranthene, and benz[*a*]anthracene. Concentrations are shown in Table 1.4.

Table 1.3. Concentrations (ng/g) of polycyclic aromatic hydrocarbons in untreated cooking oils

Compound	Olive	Peanut	Rapeseed	Corn	Vegetable
Acenaphthylene	ND	ND	ND	ND	ND
Acenaphthene	19.9	ND	ND	ND	ND
Phenanthrene	10.7	ND	ND	ND	ND
Anthracene	1.12	2.60	1.12	1.54	0.56
Fluoranthene	4.07	1.28	0.71	0.65	1.64
Pyrene	7.10	10.2	1.79	ND	ND
Benz[*a*]anthracene	4.49	13.6	6.51	ND	2.22
Chrysene	3.29	14.7	ND	ND	2.22
Benzo[*b+j+k*]fluoranthene	77.3	72.8	ND	4.68	5.28
Benzo[*e*]pyrene	0.26	19.4	ND	2.70	3.66
Benzo[*a*]pyrene	8.32	24.5	ND	11.0	4.22
Indeno[1,2,3-*cd*]pyrene	16.2	30.3	2.67	2.03	9.84
Benzo[*ghi*]perylene	5.31	26.6	18.7	3.20	8.40
Fluorene	1.73	ND	0.21	0.28	0.30
1-Methylphenanthrene	4.25	0.74	3.56	3.59	4.38
Perylene	1.50	15.5	ND	1.90	3.06
Dibenzo[*a,h+a,c*]anthracene	9.26	27.1	ND	0.59	9.20
Naphthalene	31.7	13.9	15.5	13.3	17.6
1-Methylnaphthalene	10.1	ND	ND	ND	0.66
Biphenyl	2.99	0.12	0.72	0.26	ND
2,6+2,7-Dimethyl naphthalene	8.63	ND	ND	ND	ND
2,3,5+i-Trimethyl naphthalene	4.63	0.16	0.63	ND	0.32

From Fortmann *et al*. (2001)
ND, not detected

Table 1.4. The polycyclic aromatic hydrocarbon contents (μg/m^3) of fumes from various oils heated to 250±10°C for 30 min

Carcinogens	Cooking oil		
	Safflower	Vegetable	Corn
Benzo[*a*]pyrene	22.7±1.5	21.6±1.3	18.7±0.9
Dibenz[*a,h*]anthracene	2.8±0.2	3.2±0.1	2.4±0.2
Benzo[*b*]fluoranthene	1.8±0.3	2.6±0.2	2.0±0.1
Benz[*a*]anthracene	2.5±0.1	2.1±0.4	1.9±0.1

From Chiang *et al.* (1999a)

Wei See *et al.* (2006) studied three ethnic food stalls in a food court for levels of $PM_{2.5}$ and PAHs. PAHs varied from 38 to 141 to 609 ng/m^3 at the Indian, Chinese and Malay stalls, respectively. The trend was considered to be related to the cooking temperature and amount of oil used (simmering, stir-frying and deep-frying). Frying provided relatively more high-molecular-weight PAHs compared with simmering, which produced relatively more low-molecular-weight PAHs.

In addition to PAHs, fumes from three different commercial cooking oils frequently used in Chinese cooking (lard oil, soya bean oil and peanut oil) also contained nitro-PAHs such as 1-nitropyrene and 1,3-dinitropyrene (Table 1.5) (Wu *et al.*, 1998).

Table 1.5. Concentrations of PAHs and nitro-PAHs (μg/m^3) in fumes from various oils heated to 250±10°C for 30 min

Carcinogens	Type of cooking oil		
	Lard	Soya bean	Peanut
PAHs			
Benzo[*a*]pyrene	ND	21.1±0.8	19.6±0.5
Benz[*a*]anthracene	2.3±0.2	2.1±0.5	1.5±0.2
Dibenz[*a,h*]anthracene	2.0±0.3	2.4±0.4	1.9±0.1
Nitro-PAHs			
1-Nitropyrene	1.1±0.1	2.9±0.3	1.5±0.1
1,3-Dinitropyrene	0.9±0.1	3.4±0.2	0.4±0.1

From Wu *et al.* (1998)
ND, not detected

Zhu and Wang (2003) studied 12 PAHs in the air of six domestic and four commercial kitchens. Mean concentrations of benzo[*a*]pyrene were 6–24 ng/m^3 in the domestic kitchens and 150–440 ng/m^3 in the commercial kitchens. Cooking oils were ranked lard>soya bean oil>rapeseed oil. Increases in cooking temperature produced increased PAH concentrations.

Various samples of cooking oil fumes were analysed in an effort to study the relationship between the high incidence of pulmonary adenocarcinoma in Chinese women and cooking oil fumes in the kitchen (Li *et al.*, 1994). The samples included oil fumes from three commercial cooking oils. All samples contained benzo[*a*]pyrene and dibenz[*a,h*]anthracene. The concentration of dibenz[*a,h*]anthracene in the fume samples was 5.7–22.8 times higher than that of benzo[*a*]pyrene. Concentrations of benzo[*a*]pyrene and dibenz[*a,h*]anthracene were, respectively, 0.463 and 5.736 μg/g in refined vegetable oil, 0.341 and 3.725 μg/g in soya bean oil and 0.305 and 4.565 μg/g in vegetable oil.

(c) Heterocyclic amines

Hsu *et al.* (2006) studied the formation of heterocyclic amines in the fumes from frying French fries in soya bean oil or lard. Lard was more susceptible to form these compounds than soya bean oil heated alone (Hsu *et al.*, 2006). Fumes from soya bean oil heated alone were found to contain three heterocyclic amines, namely, 2-amino-3-methylimidazo[4,5-*f*]quinoxaline (IQx), 2-amino-3-methylimidazo[4,5-*f*]quinoline (IQ) and 1-methyl-9*H*-pyrido[4,3-*b*]indole (Harman), whereas two additional amines, 2-amino-3,4-dimethylimidazo[4,5-*f*]quinoline (MeIQ) and 3-amino-1,4-dimethyl-5*H*-pyrido[4,3-*b*]indole (Trp-P-1), were generated with lard.

(d) Aldehydes and other volatile organic compounds

Higher aldehydes [C>7] have been detected in emissions from pan-frying beefsteak using four different types of oil (Table 1.6) (Sjaastad & Svendsen 2008). The aldehyde *trans*,*trans*-2,4-decadienal (*t,t*-2,4-DDE) has been found and quantified in both frying oils and fumes generated during frying. The quantity of *t,t*-2,4-DDE in fried potatoes was considered to be dependent on the oil used, on the frying process and, to a lesser extent, on oil deterioration. The degree of unsaturation of the frying oil was also considered to promote the formation of *t,t*-2,4-DDE.

Table 1.6. Levels[a] of total particles (mg/m^3) and higher aldehydes (µg/m^3) measured in the breathing zone of the cook during pan-frying of beefsteak using different oils or margarine

	Margarine	Rapeseed oil	Soya bean oil	Olive oil
Total particles	11.6 (0.7)	1.0 (0.3)	1.4 (0.7)	1.0 (1.1)
t,*t*-2,4-Decadienal	10.33 (2.52)	0.63 (1.32)	0.52 (0.80)	ND
2,4-Decadienal	25.33 (4.51)	ND	ND	ND
t-2-Decenal	25.33 (9.70)	3.60 (6.40)	0.50 (1.20)	0.50 (1.20)
s-2-Decenal	ND	0.82 (1.08)	2.20 (5.29)	3.67 (2.94)
2-Undecenal	20.67 (7.64)	3.81 (5.21)	2.02 (3.62)	3.33 (2.34)
Alkanals	426.00 (70.00)	107.00 (75.00)	128.00 (53.00)	121.00 (85.00)
Alkenals	55.70 (11.00)	1.80 (4.00)	4.00 (2.70)	0.90 (1.30)

From Sjaastad & Svendsen (2008)
ND, not detected; ; *s*, *cis*; *t*, *trans*
The results are given as arithmetic mean (standard deviation)

Emissions of low-molecular-weight aldehydes from deep-frying with extra virgin olive oil, olive oil and canola oil (control) were investigated at two temperatures, 180 and 240°C, for 15 and 7 h, respectively. Seven alkanals (C-2 to C-7 and C-9), eight 2-alkenals (C-3 to C-10) and 2,4-heptadienal were found in the fumes of all three cooking oils. The

generation rates of these aldehydes were found to be dependent on heating temperature, and showed significant increases with increases in temperature. The emissions of low-molecular-weight aldehydes from both kinds of olive oil were very similar and were lower than those observed from canola oil under similar conditions (Fullana *et al.*, 2004a,b).

The composition of the fumes was studied at different temperatures (190–200, 230–240 and 270–280°C). A strong peak was observed within the wavelength range of 260–270 nm in each condensate sample. From gas chromatography–mass spectrometry results, it was tentatively deduced that there were some 2,4-dialkylenaldehydes and other conjugated compounds in the condensates. Large amounts of hexanal and 2-heptenal were present in the cooking oil fumes. The total aldehyde peak areas of the condensates from four kinds of oil were around 30–50% of the total peak area at 270–280°C (Zhu *et al.*, 2001).

Concentrations of ethylene oxide and acetaldehyde were assessed during the simulated frying of soya bean oil without or with flavouring herbs and spices (garlic, onion, ginger, basil) under nitrogen or air at 1atm (Lin *et al*., 2007). The tests were performed at 130, 150, 180 and 200°C.

The concentration of both ethylene oxide and acetaldehyde in the oil and vapour phases increased with frying temperature within the range of 130 to 200°C. Under air, the amounts of ethylene oxide and acetaldehyde generated in either phase were several times higher when compared with amounts generated under nitrogen. In the oil phase, concentrations of ethylene oxide and acetaldehyde increased linearly from 7.6 ppm at 130°C to 26.2 ppm at 200°C, and from 6.0 ppm to 16.6 ppm, respectively. Similarly, ethylene oxide concentrations in the vapour phase increased from 7 ppm to 85 ppm.

The impact of the combination of flavouring sources and soya bean oil was assessed. Both ethylene oxide and acetaldehyde were distributed between the gas phase and the oil phase after cooking each herb or spice at 150°C for 5 minutes under either atmosphere. In each scenario, the amounts of ethylene oxide and acetaldehyde produced were different when compared with heating soya bean oil alone.

1.3.2 *Effect of the type of food, type of cooking or mode of frying*

(a) Studies in a controlled environment

In an experimental study, airborne cooking by-products from frying beef (hamburgers), pork (bacon strips) and soya bean-based food (tempeh burgers) were collected, extracted and chemically analysed. 2-Amino-1-methyl-6-phenylimidazo[4,5-*b*]pyridine (PhIP) was the most abundant heterocyclic amine, followed by 2-amino-3,8-dimethylimidazo[4,5-*f*]quinoxaline (MeIQx) and 2-amino-3,4,8-trimethylimidazo[4,5-*f*]quinoxaline (DiMeIQx). No 2-amino-9*H*-pyrido[2,3-*b*]indole (AαC) was detected in the food samples fried at about 200°C, although it was present in the collected airborne products. The total amounts of heterocyclic amines in the smoke condensates were 3 ng/g

from fried bacon, 0.37 ng/g from fried beef and 0.177 ng/g from fried soya-based food (Table 1.7) (Thiébaud *et al.*, 1995).

Table 1.7. Concentration of heterocyclic amines from frying meat and soya-based patties (ng/g of cooked samples)

Food sample (average temperature)	In the fried food sample				In the bead-trap smoke condensate			
	MeIQx	DiMeIQx	PhIP	AαC	MeIQx	DiMeIQx	PhIP	AαC
Beef patties (198°C)	4.3	1.3	4.9	ND	0.14	0.006	0.14	0.084
Beef patties (277°C)	16	4.5	68	21	1.1	0.25	1.8	4.0
Bacon strip (208°C)	45	12	106	ND	ND	ND	1.0	2.0
Soya-based patties (226°C)	ND	ND	ND	ND	ND	ND	0.007	0.17

From Thiébaud *et al.* (1995)
AαC, 2-amino-9*H*-pyrido[2,3-*b*]indole; DiMeIQx, 2-amino-3,4,8-trimethylimidazo[4,5-*f*]quinoxaline; ND, not detected (<0.1ng/g); PhIP, 2-amino-1-methyl-6-phenylimidazo[4,5-*b*]pyridine; MeIQx, 2-amino-3,8-dimethylimidazo[4,5-*f*]quinoxaline

One study compared emissions of particles, nitrogen oxides, carbon monoxide, PAHs and formaldehyde in an experimental chamber during seven different types of cooking activity including pan-frying (Table 1.8; Kelly, 2001). Samples were integrated over periods of 1–4 h. [Temperatures were measured but not reported.]. Except for the hamburger cooked on gas, all tests showed an increase in total PAHs, with indoor levels averaging about twice or more the outdoor concentrations. Since the outdoor concentrations would be expected to be roughly half of those indoors in the absence of indoor sources, the increase over normal indoor levels is by a factor of about 3. For seven particle-bound PAHs that are considered to be probably carcinogenic, indoor:outdoor ratios averaged from 1–1.5. Emissions of nitrogen dioxide were found only when the gas stove was used, and were 10 mg/kg for pan-frying of hamburgers. Emissions of formaldehyde remained below 10 ppb (see footnote in Table 1.8).

Another major controlled study of cooking emissions was sponsored by the Air Resources Board of the State of California (Fortmann *et al.*, 2001). $PM_{2.5}$ and PM_{10} particles, carbon monoxide, nitrogen oxide, nitrogen dioxide, PAHs and aldehydes were measured. Cooking activities included wok stir-frying of chicken and vegetables, deep-frying of French fries and pan-frying of bacon, tortillas or hamburgers. The cooking activities were studied under standard conditions or worst-case scenarios. Wok stir-frying was performed with 65 g peanut oil for 1 or 3 min at high temperatures, using chicken and vegetables as food. The concentrations of $PM_{2.5}$ particles emitted during the cooking activities under different conditions are given in Table 1.9. Of the 13 PAHs targeted for analysis, pyrene, benzo[*e*]pyrene, benzo[*a*]pyrene and benzo(*b*+*j*+*k*)phenanthrenes were detected in more than 60% of the samples. Duplicate samples collected during the worst-case stir-fry test showed that the precision of the PAH sampling method was poor.

[Because of the short test, the mass of PAHs in the samples was low, and there was large analytical uncertainty associated with the measurement.]

Table 1.8. Concentrations[a] of $PM_{2.5}$, PAHs and formaldehyde in a research house during pan-frying

Type of stove Food cooked	$PM_{2.5}$ (μg/m^3)		Total PAHs (ng/m^3)		Seven PAHs[b] (ng/m^3)		Formaldehyde[c] (ppb)
	Stove	Kitchen	Indoor	Outdoor	Indoor	Outdoor	Indoor
Gas							
Hamburger	115	60	294	288	0.93	1.76	3
Steak	2270	2670	833	189	3.70	1.93	48
Electric							
Hamburger	252	160	425	251	3.59	1.68	<2
Steak	542	457	610	431	2.56	3.24	9

From Kelly (2001)
PAH, polycyclic aromatic hydrocarbon; PM, particulate matter
[a] Average of three replicate runs
[b] Benz[*a*]anthracene, chrysene, benzo[*b*+*k*]fluoranthene, benzo[*a*]pyrene, indeno[1,2,3-*cd*]pyrene, dibenzo[*a*,*h*]anthracene, benzo[*ghi*]perylene
[c] Values were confounded by background emissions from building materials and by variations due to purging air between tests.

(b) Field studies

Samples of cooking oil fumes from three catering shops were analysed (Li *et al.*, 1994). All samples contained benzo[*a*]pyrene and dibenz[*a*,*h*]anthracene. PAH concentrations at the three catering shops showed levels of benzo[*a*]pyrene of 41.8 ng/m^3 at a Youtiao (deep-fried twisted dough sticks) shop, 22.8 ng/m^3 at a Seqenma (candied fritters) workshop and 4.9 ng/m^3 at a kitchen of a restaurant; concentrations of dibenz[*a*,*h*]anthracene were 338, 144 and 30.3 ng/m^3, respectively.

Another study in China showed that the cooking method affected the concentration of benzo[*a*]pyrene in kitchen air (Du *et al.*, 1996). In the same kitchens, the level of benzo[*a*]pyrene was elevated in indoor air from the baseline value of 0.41 μg/100m^3 to 0.65 μg/100m^3 when meat was boiled, and was further increased to 2.64 μg/100m^3 when meat was stir-fried.

Li *et al.* (2003) measured PAHs emitted from the rooftop exhausts of four types of restaurant in Taiwan, China. Although gaseous PAHs outweighed particle-bound PAHs by about 4:1, when expressed in benzo[*a*]pyrene-equivalents, the ratio was reversed. Chinese food contributed the majority of the level of benzo[*a*]pyrene-equivalents, while western food contributed about seven times less and fast food and Japanese food contributed negligible amounts. Compared with traffic in the city, restaurants contributed somewhat less total PAHs but about 10 times the benzo[*a*]pyrene-equivalent amount.

Zhu and Wang (2003) studied 12 PAHs in the air of six domestic and four commercial kitchens. Mean concentrations of benzo[*a*]pyrene were 6–24 ng/m^3 in the

Table 1.9. $PM_{2.5}$ concentrations under different cooking conditions in a research house

Type of cooking	Food	Type of stove	Conditions	Temperature (°C)[a]		$PM_{2.5}$ concentration ($\mu g/m^3$)			
				Food	Burner	Kitchen	Living room	Bedroom	Outdoors
Stir-frying	Chicken and vegetables	Gas	Standard[d]	79.6	85[b]	241	191	185	7
			Replicate[d]	88.3–100	418–439	185	323	301	8.8
			Worst case[d]	119–124	284–398	1289	850	798	8.1
			Vegetable oil	95.3–104	295–513	392	294	303	8.1
		Electric	Standard	105	289	214	1124	364	5.6
Deep-frying	French fries	Gas	Standard	182[c]	729	195	71.9	83.3	4.2
			Replicate	186.9[c]	277	162	91.9	70.5	4.1
		Electric	Standard	171.4[c]	446	374	94.7	90.2	5.7
Pan-frying	Bacon	Gas	Standard	148–156	105–108[b]	482	142	286	7
			Worst case	143.6–184.1	268–337	484	711	771	8.8
		Electric	Standard	72.8–73.7	272–298	207	276	235	5.7
	Tortillas	Gas	Standard	172[c]	97[b]	566	260	77.4	4.2
		Electric	Standard	232.9[c]	ND	1269	1175	1173	5.7
	Hamburger	Gas	Cast iron pan	93.0–93.7	270–304	153	7.73	8.64	1.5
		Gas	Cast iron pan	95.3	ND	51.9	8.6	8.8	3.6
		Gas	Pan lid	NR	253–300	355	5.8	6.4	4

From Fortmann *et al.* (2001)
ND, not detected; NR, not reported; PM, particulate matter
[a] Peak temperature of the food during the test; average temperature for burner or oven during the test
[b] Thermocouple proble location for this test was inconsistent with later tests that yielded variable flame temperatures, but other parameters indicate similar cooking temperatures.
[c] Temperature of cooking oil
[d] Peanut oil

domestic kitchens and 150–440 ng/m^3 in the commercial kitchens. Cooking practices produced PAHs in the rank order broiling>frying>>boiling.

The influence of frying conditions (deep-frying, pan-frying) was studied (Boskou *et al.*, 2006). In all cases tested, the highest concentration of *trans*,*trans*-2,4-decadienal was detected during deep-frying.

Studies have shown that the total amount of organic compounds per milligram of particulate organic matter is much higher in western-style fast food cooking than in Chinese cooking; however, Chinese cooking has a much greater contribution of PAHs to particulate organic matter (Table 1.10) (Zhao *et al.*, 2007a,b).

Table 1.10. Concentrations of organic compounds from western-style fast food and from Chinese cooking (ng/mg of particulate organic matter)

Organic compounds	Western-style fast food cooking[a]	Chinese cooking[b]
n-Alkanes	3 863	1 883
Polycyclic aromatic hydrocarbons	40	2 855
n-Alkanals	29 172	3 444
n-Alkanones	22 702	2 443
Lactones	13 323	2 142
Amides	4 692	531
Saturated fatty acids	374 699	26 804
Unsaturated fatty acids	93 299	29 028
Dicarboxylic acids	57 877	2 051
Monosaccharide anhydrides	97	314
Sterols	487	1 684
Other compounds	63	208

From Zhao *et al*. (2007a,b)

[a] Average of six samples

[b] Average of four different styles of Chinese cooking

1.4 Human exposure

Neither occupational nor non-occupational exposure to emissions from cooking has been characterized systematically. Most of the available studies examined the nature and amount of emissions produced during different types of cooking in different settings, including the release of emissions from kitchens into the ambient environment. As the substances measured varied widely among studies, it is difficult to summarize quantitatively exposures in different settings. Furthermore, co-exposures were not specifically mentioned. Results from various field studies, carried out primarily in South-East Asia, are summarized in Tables 1.11 and 1.12.

Only one recent study provided information on biological monitoring of exposure and effect in the occupational setting (Table 1.11) (Pan *et al.*, 2008).

Table 1.11. Occupational exposures to emissions from high-temperature frying

Reference, location	Setting	Study design/ samples	Exposure(s) measured	Main results
Vainiotalo & Matveinen (1993), Finland	8 workplaces (2 bakeries, a food factory, 5 restaurant kitchens)	Field measurements, sampling during frying/grilling of meat or fish or during deep-frying	Fat aerosol Acrolein Formaldehyde Acetaldehyde Heterocyclic amines PAHs	Highest concentrations (9–16 mg/m^3) in kitchens using the ordinary frying method; lower concentrations at other workplaces (<0.01–3.2 mg/m^3) Range, 0.01–0.59 mg/m^3 Highest concentrations in grill kitchens (0.24 and 0.75 mg/m^3) Highest concentrations in bakeries (0.67 and 1.5 mg/m^3) Mutagenic heterocyclic amines below detection limits Low concentrations
Svendsen *et al.* (2002), Norway	4 hotels, 2 hamburger chain restaurants, 10 à la carte restaurants and 3 small local restaurants, serving mostly fried food	Personal sampling in kitchens	Fat aerosols Aldehydes	Highest concentration (6.6 mg/m^3) in a small local restaurant; arithmetic mean for all kitchens, 0.62 mg/m^3 Highest level of the sum of the aldehydes, 186 µg/m^3; arithmetic mean, 69 µg/m^3
He *et al*. (2004b), Shen Zhen, China	2 cooking styles of Chinese cuisine: Hunan cooking and Cantonese cooking	Sampling of cooking fumes during regular operation	PM, organic compounds	More than half of the $PM_{2.5}$ mass is due to organic compounds, and over 90 species of organic compound were identified and quantified, accounting for 26.1% of bulk organic particle mass and 20.7% of $PM_{2.5}$. Fatty acids, diacids and steroids were the major organic compounds emitted from both styles of cooking. Of the quantified organic mass, over 90% was fatty acids. The mass of organic species, and the molecular distribution of *n*-alkanes and PAHs indicated the dissimilarities between the two different cooking styles, but generally the major parts of the organic particulate emissions of the two restaurants were similar.

Table 1.11. (contd)

Reference, location	Setting	Study design/ samples	Exposure(s) measured	Main results
He *et al*. (2004b), Beijing, China	2 commercial restaurants, 1 with Chinese foods cooked over gas flame, 1 Uigur style (mutton charbroiled by charcoal)	Sampling during regular operation	$PM_{2.5}$, organic compounds including series of alkanes, *n*-alkanoic acids, *n*-alkanals, alkan-2-ones and PAHs	Mass concentrations of fine particles, alkanes, *n*-alkanoic acids and PAHs in air emitted from the Uigur [Chinese Islamic] style cooking were a hundred times higher than ambient $PM_{2.5}$ in Beijing.
Lee & Jeong (2008), South Korea	3 types of restaurants: Korean barbecue house, Chinese restaurant, Japanese restaurant	Personal exposure measurements in the breathing zone during eating periods	PM [PM_{10}, $PM_{2.5}$ and $PM_{1.0}$]	Highest concentrations at Korean barbecue house, with average concentrations of PM_{10}, $PM_{2.5}$ and $PM_{1.0}$ of 169, 124, and 63 μg/m^3, respectively; average exposure ratios for $PM_{1.0}/PM_{10}$, $PM_{2.5}/PM_{10}$ and $PM_{1.0}/PM_{2.5}$ at the barbecue house were 0.38, 0.73 and 0.52, respectively, which were much higher than those at other restaurants. Second highest $PM_{2.5}$ and PM_{10} concentrations at Chinese restaurant
			Formaldehyde	Range, 89.7–345.9 μg/m^3; highest concentrations in the Japanese restaurant
Pan *et al.* (2008), Taiwan, China	23 Chinese restaurants	Cross-sectional study; measurements in kitchens and dining areas	Airborne PM and PAHs	Airborne PM and PAH levels in kitchens significantly exceeded those in dining areas.
			Urinary 1-hydroxypyrene (1-OHP)	Geometric mean: kitchen staff, 4.5 μg/g creatinine; service staff, 2.7 μg/g creatinine (significantly higher)
			Urinary 8-hydroxy-2'-deoxyguanosine (8-OHdG)	Geometric mean: kitchen staff, 7.9 μg/g creatinine; service staff , 5.4 μg/g creatinine (significantly higher)
				Urinary 1-OHP level, work in kitchens, gender and work hours per day were four significant predictors of urinary 8-OHdG levels after adjustments for covariates.

Table 1.11. (contd)

Reference, location	Setting	Study design/ samples	Exposure(s) measured	Main results
Yeung & To (2008), Hong Kong, China	Commercial cooking settings	Survey during commercial cooking processes	Size distributions of the aerosols	Log normal distribution; mode diameter of aerosols increased with increasing cooking temperature, especially in the size range between 0.1 and 1.0 μm.

PAH, polycyclic aromatic hydrocarbon; PM, particulate matter

Table 1.12. Environmental exposure to cooking emissions from commercial restaurants

Reference, location	Setting	Study design/ samples	Exposure(s) measured	Main results
To *et al.* (2007), Hong Kong, China	Commercial kitchens of Chinese restaurants, western restaurants and food servicing areas	Territorial-wide survey on the quantification of cooking fumes discharged from commercial kitchens	Organic compounds (*n*-alkanes, PAHs, fatty acids and aromatic amines)	Wide spectrum of organic compounds including *n*-alkanes, PAHs, fatty acids and aromatic amines PAHs: no statistically significant difference in the composition of fumes between restaurants; *n*-alkanes: mean concentrations in fumes from exotic food servicing areas significantly higher than those for Chinese or western restaurants ($p<0.05$)
Yang *et al.* (2007), Taiwan, China	16 restaurants with 3 types of cooking: Chinese, western and barbecue	Samples from kitchen exhausts	*trans*,*trans*-2,4-decadienal (*t*,*t*-2,4-DDE)	Emission factor (μg/customer): barbecue, 1990 > Chinese, 570 > Western, 63.8.

Table 1.12. (contd)

Reference, location	Setting	Study design/ samples	Exposure(s) measured	Main results
Zhao *et al.* (2007a), Guang Zhou, China	1 commercial western-style fast food restaurant	Sampling from exhaust	Chemical composition of particulate organic matter (POM)	The total amount of quantified compounds of per mg POM in western-style fast food cooking is much higher than that in Chinese cooking. The predominant homologue is fatty acids, accounting for 78% of total quantified POM, with the predominant one being palmitic acid. Dicarboxylic acids display the second highest concentration in the quantified homologues with hexanedioic acid being predominant, followed by nonanedioic acid. C-max of *n*-alkanes occurs at C25, but they still appear at relatively higher concentrations at C29 and C31. The relationship of concentrations of unsaturated fatty acids (C16 and C18) with a double bond at C9 position and C9 acids indicates the reduction of the unsaturated fatty acids in the emissions could form the C9 acids. Moreover, the non-linear fit indicates that other C9 species or other compounds are also produced, except for the C9 acids. The potential candidates of tracers for the emissions from western-style fast food cooking could be: tetradecanoic acid, hexadecanoic acid, octadecanoic acid, 9-octadecenoic acid, nonanal, lactones, levoglucosan, hexanedioic acid and nonanedioic acid.

Table 1.12. (contd)

Reference, location	Setting	Study design/ samples	Exposure(s) measured	Main results
Zhao *et al.* (2007b), Guang Zhou, China	4 Chinese restaurants: Cantonese style, Hunan style, Sichuan style and Dongbei style	Sampling from exhaust	Chemical composition of POM in $PM_{2.5}$	The quantified compounds account for 5–10% of total POM in $PM_{2.5}$. The dominant homologue is fatty acids, constituting 73–85% of the quantified compounds. The emissions of different compounds are impacted significantly by the cooking ingredients. The candidates of organic tracers used to describe and distinguish emissions from Chinese cooking in Guangzhou are tetradecanoic acid, hexadecanoic acid, octadecanoic acid, oleic acid, levoglucosan, mannosan, galactosan, nonanal and lactones.

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2. Studies of Cancer in Humans

2.1 Introduction

Since the 1970s, a total of 17 case–control studies have explored the relationship between exposure to cooking fumes and the risk for lung cancer. These studies were conducted in Chinese populations residing in China (including Taiwan and Hong Kong Special Administrative Region) and Singapore. While active tobacco smoking is a well-established major cause of lung cancer in Chinese men and women, a relatively high proportion of lung cancer in Chinese women, many of whom are nonsmokers, can not be explained by active smoking. Thus, one motivation for these studies was to investigate the role of other lifestyle factors, including indoor air pollution from cooking oil fumes, in the etiology of lung cancer in Chinese women.

Exposure assessment of cooking practices and cooking oil fumes varied substantially (Tables 2.1 and 2.2). Two aspects related to cooking oil fumes have been investigated: (i) the types of oil used and practices of high-temperature cooking, including frequency, stir-frying, deep-frying and pan-frying, and (ii) cooking practices, including the availability of a separate kitchen, ventilation in the kitchen based on the number and size of windows, the use of a fume extractor, personal assessment of ventilation, such as frequency of eye irritation during cooking and smokiness in the kitchen, duration of exposure (years of cooking) and susceptible time of exposure (age started to cook). In four studies (Lan *et al*., 1993; Dai *et al*., 1996; Shen *et al*., 1996; Wang *et al*., 1996), results were based on a single variable that represented some aspect of cooking practices. In contrast, exposure assessment was more comprehensive in seven studies (Gao *et al*., 1987; Ko *et al*., 1997; Zhong *et al*., 1999; Ko *et al*., 2000; Lee *et al*., 2001; Metayer *et al*., 2002; Yu *et al*., 2006). In several studies, the authors specified that past cooking practices or those experienced earlier in life (Seow *et al*., 2000) or at a particular age or time period in life (Ko *et al*., 1997, 2000; Lee *et al*., 2001) were investigated. Behaviours related to the type of cooking oil used most often and the frequency of high-temperature cooking (stir-frying, pan-frying, deep-frying) were also frequently examined. However, in most of the studies, no discussion was included regarding the timing of exposure or whether the information collected was related to current, usual or past cooking practices. Other factors included frequency of eye irritation during cooking, frequency of smokiness in the house, location of the kitchen, windows in the kitchen and the presence of fume extractors; these are viewed as indirect measures to assess the severity of exposure to cooking fumes and general household ventilation. Greater attention was paid to the measures of exposure that were considered to be more objective and whether duration, frequency and intensity of exposure to cooking oil fumes were assessed.

Of the 17 case–control studies that have investigated the relationship of exposure to cooking oil fumes and lung cancer, one was a study of lung cancer mortality (Lei *et al*.,

Table 2.1. Assessment of cooking practices/fumes included in the published case–control studies of lung cancer

Reference	Cooking in separate kitchen	Windows in kitchen/size	Fumes visible	Smokiness in kitchen	Eye irritation	No. of meals cooked/day	Age started cooking	Years of cooking
MacLennan *et al*. (1977)	–	–	–	–	–	–	No/yes (cooking)	–
Gao *et al*. (1987)	–	–	–	–	Never to frequent	–	–	–
Xu *et al*. (1989)	Cooking in bedroom (yrs)	–	–	–	–	–	–	–
Wu-Williams *et al*. (1990)	–	–	–	–	Never to frequent	–	–	–
Liu *et al*. (1991)	–	–	–	–	–	–	≤10 vs. >15 yrs	≤30, 31–44, ≥45
Ger *et al*. (1993)	–	–	–	–	–	–	–	–
Lan *et al*. (1993)	–	–	–	–	–	–	–	–
Liu *et al*. (1993)	No/yes	Size; chimneys	–	Ventilation (no/yes)	–	0–1, 2, 3	–	–
Dai *et al*. (1996)	–	–	–	–	–	–	–	–
Koo *et al*. (1996)	–	–	–	–	–	–	–	<25, 26–40, ≥41
Lei *et al*. (1996)	Size kitchen	–	–	–	–	–	–	Infrequent, ≤20, 20–40, >40
Shen *et al*. (1996)	–	–	–	No/yes	–	Times/week (no results)	–	–
Wang *et al*. (1996)	–	–	No/yes	–	–	–	–	–
Ko *et al*. (1997)	–	–	Fume extractor (no/yes)	–	–	–	7–20 vs. ≥ 21 yrs	–
Zhong *et al*. (1999)	No/yes	Area of window	No/yes	None to considerable	Never to frequent	–	–	–

Table 2.1. (contd)

Reference	Cooking in separate kitchen	Windows in kitchen/size	Fumes visible	Smokiness in kitchen	Eye irritation	No. of meals cooked/day	Age started cooking	Years of cooking
Ko *et al*. (2000)	–	<2 vs. ≥2, size of opening: small or medium, large	Fume extractor	Ventilation Poor/good	Rarely vs. frequently	Daily (no/yes) meals (1, 2, ≥3)	≤20 vs. 20 yrs	1–20, 21–40, ≥40
Seow *et al*. (2000)	–	–	<Daily/daily	–	–	–	–	–
Zhou *et al*. (2000)	(location) Separate Living room, Bedroom	–	Medium/heavy vs slight	None, slight, medium, heavy	Never to frequent	–	–	–
Lee *et al*. (2001)	–	–	Fume extractor	–	–	–	≤20 vs. 20 yrs	–
Metayer *et al*. (2002)	–	–	–	No to considerable by oil type	Ever to frequent by oil type	≤2 vs. ≥3	≤13, 14–16, ≥17 yrs	≤29, 30–39, 40–49, ≥50
Chan-Yeung *et al.* (2003)	–	–	–	–	–	–	–	
Shi *et al.* (2005)	–	–	Fuel smoke, cooking oil smoke	–	–	–	–	–
Yu *et al*. (2006)	–	–	Fume extractor/ exhaust fan	–	–	–	–	–

Table 2.2. Assessment of cooking practices/fumes by type of oil, type of frying and type of fuel included in the published case–control studies of lung cancer

Reference	Rapeseed oil	Other type of oil	Amount of oil	No. of times stir-frying	No. of times deep-frying	No. of times pan-frying	No. of times boiling	Fuel for cooking	Fuel for heating
MacLennan *et al.* (1977)	–	–	–	–	–	–	–	Gas	Kerosene
Gao *et al*. (1987)	Never to frequent	Never to frequent	–	≤20–≥30/wk	0–≥3/wk	–	≤3–≥12/wk	Coal/gas/ wood	
Xu *et al*. (1989)	–	–	–	–	–	–	–	Gas	Coal
Wu-Williams *et al.* (1990)	–	–	–	–	0–≥3/mo	–	–	Coal	Coal
Liu *et al*. (1991)	–	–	–	–	–	–	–	Coal	Wood
Ger *et al*. (1993)	–	–	–	No/yes	No/yes	No/yes	No/yes	Coal	–
Lan *et al*. (1993)	Never vs. often	–	–	–	–	–	–	Coal	–
Liu *et al*. (1993)	–	–	–	–	–	–	–	Coal/gas/ wood	–
Dai *et al*. (1996)	–	–	–	–	≤5 vs. ≥5/mo*	≤5 vs. ≥5/mo*	–	Coal	Kerosene
Koo & Ho (1996)	–	–	–	–	–	–	–	Gas	Kerosene
Lei *et al*. (1996)	–	–	–	–	–	Preferred/ average/not preferred	–	–	–
Shen *et al*. (1996)	–	–	Use per mo (no results)	–	–	–	–	Solid/non-solid fuel	Coal
Wang *et al*. (1996)	–	–	–	–	–	–	–	–	–

Table 2.2. (contd)

Reference	Rapeseed oil	Other type of oil	Amount of oil	No. of times stir-frying	No. of times deep-frying	No. of times pan-frying	No. of times boiling	Fuel for cooking	Fuel for heating
Ko *et al*. (1997)	–	No/lard/ vegetable oil	–	0–4 vs. ≥5/wk	0–4 vs. ≥5/wk	0–4 vs. ≥5/mo	–	Gas/coal/ wood	–
Zhong *et al*. (1999)	Used frequently	Soya bean used frequently	–	<7, 7, >7/wk	≤1 vs. >1/wk	≤1 vs. >1/wk	–	Coal/coal gas/gas	–
Ko *et al*. (2000)	–	–	–	No/yes after fumes, fume extractor	No/yes after fumes, fume extractor	No/yes after fumes, fume extractor	–	Coal	Gas
Seow *et al*. (2000)	–	Unsaturated vs. saturated oil	–	Not daily vs. daily	–	–	–	–	–
Zhou *et al*. (2000)	–	–	–	–	–	0–1 vs. ≥2/wk	–	–	–
Lee *et al*. (2001)	–	Lard/vegetable oil	–	No/yes after fumes, fume extractor	No/yes after fumes, fume extractor	No/yes after fumes, fume extractor	–	Gas/coal/ wood	–
Metayer *et al*. (2002)	No/yes	Linseed/ perilla/ hempseed oil	Catty/mo ≤3–≥6	≤15–≥3/mo	≤1–≥3/mo	–	–	Coal/wood	–
Chan-Yeung *et al*. (2003)	–	–	–	–	–	No exposure <3.5/wk 3.5–7/wk >7/wk	–	–	–
Yu *et al*. (2006)	Never/seldom, sometimes, always	Peanut/corn oil		≤50 dish-years 51–100 101–150 151–200 >200	≤50 51–100 101–150 151–200 >200	≤50 51–100 101–150 151–200 >200		–	–

* deep-frying and pan-frying combined

1996); the other studies included six population-based (Gao *et al*., 1987; Xu *et al*., 1989; Wu-Williams *et al*., 1990; Lan *et al*.., 1993; Zhong *et al*., 1999; Metayer *et al*., 2002) and 10 hospital-/clinic-based studies of incident lung cancers (Ger *et al*., 1993; Dai *et al*., 1996; Shen *et al*., 1996; Wang *et al*., 1996; Ko *et al*., 1997, 2000; Seow *et al*., 2000; Zhou *et al*., 2000; Lee *et al*., 2001; Yu *et al*., 2006). Twelve studies included only women (Gao *et al*., 1987, Wu-Williams *et al*., 1990; Lan *et al*., 1993; Dai *et al*., 1996; Wang *et al*., 1996; Ko *et al*., 1997; Zhong *et al*., 1999; Ko *et al*., 2000; Seow *et al*., 2000; Zhou *et al*., 2000; Metayer *et al*., 2002; Yu *et al*., 2006), seven of which studied only nonsmokers (Lan *et al*., 1993; Dai *et al*., 1996; Wang *et al*., 1996; Ko *et al*., 1997; Zhong *et al*., 1999; Ko *et al*., 2000; Yu *et al*., 2006). Men and women, smokers and nonsmokers were included in the other five studies (Xu *et al*., 1989; Ger *et al*., 1993; Lei *et al*., 1996; Shen *et al*., 1996, Lee *et al*., 2001).

These studies used heterogeneous methodologies and included different sources of cases, types of controls, methods of data collection and use of surrogate respondents; the degree of pathological confirmation of lung cancer diagnoses also differed. Relevant information regarding each of the case–control studies (i.e. study population, study period, sources of cases and controls, number of cases and controls, response rate, number of proxy interviews, percentage of pathologically/cytologically confirmed cases) and selected results are shown in Table 2.3.

2.2 Case–control studies

2.2.1 Northern China

Two large population-based case–control studies carried out in industrial areas in northern China during the late 1980s provided information on cooking practices and the risk for lung cancer. The main objectives of these two studies were to examine the role of active and passive smoking, and pollution from industrial and domestic sources. Xu *et al*. (1989) studied men and women who had lung cancer in Shenyang while Wu-Williams *et al*. (1990) examined the pattern of risk for lung cancer among women in Harbin and Shenyang.

The study in Shenyang included 1249 lung cancer cases (729 men, 520 women) and 1345 population-based controls (788 men, 557 women); 86% of male cases and 70% of male controls were smokers; the corresponding figures in women were 55% of cases and 35% of controls (Xu *et al*., 1989). Nearly 80% (85.1% in men, 75.0% in women) of the lung cancers were pathologically/cytologically confirmed; 31% of these were adenocarcinoma of the lung. After adjusting for age, education and active smoking, the risk for lung cancer was higher when cooking took place in the bedroom or entry corridor to the bedroom than in a separate kitchen or elsewhere in the house. In men, the adjusted odds ratios were 1.0, 1.2 and 2.1 in relation to cooking in the bedroom for 0, 1–29 and ≥30 years, respectively (p trend <0.05); the corresponding adjusted odds ratios in women were 1.0, 1.5 and 1.8 (p trend <0.05).

Table 2.3. Case–control studies of cooking practices/fumes and lung cancer in China

Reference, study location, period	Characteristics of cases, histological confirmation (HC), cell type (%)	Characteristics of controls	Data collection, response rate	Exposure categories	No. of cases	Relative risk (95% CI)	Adjustment factors	Comments (covariates considered)
Gao *et al.* (1987), Shanghai, 1984–86	672 women; 81% HC; 61% ADC; 22% SqCC; 6% SCLC; 11% other; 236 smokers; aged 35–69 years permanent residents of the area, Shanghai Cancer Registry ICD-9 (162)	735 frequency-matched by age and selected from the general population of Shanghai; 130 smokers	Response rate: cases, 672/765 (88.0%); controls, 735/802 (91.3%)	*Oil used*			Age, education, smoking	Study population and exposure indices defined clearly; use of coal/gas was unrelated to risk
				Soya bean	269	1.0		
				Rapeseed	322	1.4 (1.1–1.8)		
				Stir-frying (dishes/week)				
				≤20	336	1.0		
				20–24	198	1.2 (0.9–1.5)		
				25–29	48	1.2 (0.8–1.9)		
				≥30	34	2.6 (1.3–5.0)		
				Deep-frying (dishes/week)				
				0	502	1.0		
				1	85	1.5 (1.0–2.1)		
				2	21	1.6 (0.8–3.2)		
				≥3	8	1.9 (0.5–6.8)		
				Boiling (dishes/week)				
				≤3	96	1.0		
				4–7	390	1.0 (0.7–1.3)		
				8–11	63	1.8 (1.1–3.0)		
				≥12	67	2.2 (1.3–3.7)		
				Eye irritation/ smokiness				
				Never/none	244	1.0		
				Never/considerable	55	1.6 (1.0–2.5)		
				Frequent/none	212	1.6 (1.2–2.1)		
				Frequent/considerable	109	2.6 (1.8–3.7)		

Table 2.3. (contd)

Reference, study location, period	Characteristics of cases, histological confirmation (HC), cell type (%)	Characteristics of controls	Data collection, response rate	Exposure categories	No. of cases	Relative risk (95% CI)	Adjustment factors	Comments (covariates considered)
Xu *et al.* (1989), Shenyang, 1985–87	1249 (729 men, 520 women); 79% HC; 31% ADC; 43% SqCC; 16% SCLC; 10% other; 86% men and 55% women smoked; aged 30–69 yrs; newly diagnosed with primary lung cancer; Shenyang Cancer Registry ICD-9 (162)	1345 (788 men, 557 women) population controls, frequency-matched on age and sex; 70% men and 35% women smoked	Response rate: cases, 1249/1318 (94.8%); controls, 100%	*Cooking in bedroom* 0 year 1–29 yrs ≥30 yrs *p* for trend 0 year 1–29 yrs ≥30 yrs *p* for trend	*Men* 570 75 84 *Women* 503 25 29	 1.0 1.2 2.1 <0.05 1.0 1.5 1.8 <0.05	Age, education, smoking	CIs not reported; coal use was not adjusted for in the analysis
Wu-Williams *et al*. (1990), Shenyang, 1985–87	965 women (520 from Shenyang, 445 from Harbin); 74% HC; 44% ADC; 28% SqCC; 16% SCLC; 12% other; 545 smokers (56.7%); aged 30–69 years; Shenyang Cancer Registry; 729 men from Shenyang	959 female (555 from Shenyang, 404 from Harbin) population controls, frequency-matched by age; 357 smokers (37.2%); 788 male controls from Shenyang	Response rate: cases, 962/1049 (92.7%); controls, 100%	*Deep-frying (times/month)* 0 1 2 ≥3 *Eye irritation* Never/rarely Occasionally Frequently *Burning kangs* 0 1–20 21+	 324 326 170 121 647 218 89 677 106 173	 1.0 1.2 (1.0–1.5) 2.1 (1.5–2.8) 1.9 (1.4–2.7) 1.0 1.6 (1.2–1.8) 1.8 (1.3–2.6) 1.0 1.2 (0.9–1.7) 1.5 (1.1–2.0)	Age, education, smoking, study area	

Table 2.3. (contd)

Reference, study location, period	Characteristics of cases, histological confirmation (HC), cell type (%)	Characteristics of controls	Data collection, response rate	Exposure categories	No. of cases	Relative risk (95% CI)	Adjustment factors	Comments (covariates considered)
Ger *et al*. (1993), Taipei, Taiwan, 1990–91	131 hospital patients (92 men, 39 women); 100% HC; 50% ADC; 27% SqCC; 14% SCLC; 48 nonsmokers	524 (262 hospital, 262 neighbourhood) matched to cases on age, sex, insurance status/ residence; 229 nonsmokers (111 hospital controls, 118 neighbourhood controls)	In-person interview; response rate: cases, 131/143 (92%); hospital controls, 88%; neighbourhood controls, 83%	**ADC**				Results shown were based on neighbour-hood controls. Matched analysis: variables included were not specified. Definition of cooking practices was not presented.
				Frying				
				No	46	1.0		
				Yes	26	0.71 (0.36–1.39)		
				Stir-frying				
				No	28	1.0		
				Yes	44	1.19 (0.58–2.44)		
				Deep-frying				
				No	63	1.0		
				Yes	9	0.63 (0.26–1.55)		
				Boiling				
				No	38	1.0		
				Yes	34	1.75 (0.99–3.12)		
				SqCC-/SCLC				
				Frying				
				No	44	1.0		
				Yes	15	0.93 (0.37–2.32)		
				Stir-frying				
				No	33	1.0		
				Yes	26	1.00 (0.47–2.14)		
				Deep-frying				
				No	51	1.0		
				Yes	8	1.22 (0.42–3.52)		
				Boiling				
				No	36	1.00		
				Yes	23	1.26 (0.61–2.60)		

Table 2.3. (contd)

Reference, study location, period	Characteristics of cases, histological confirmation (HC), cell type (%)	Characteristics of controls	Data collection, response rate	Exposure categories	No. of cases	Relative risk (95% CI)	Adjustment factors	Comments (covariates considered)
Lan *et al.* (1993), Xuan Wei County, 1988–90	139 female farmers; 39.6% HC; 49.1% ADC; 36.4% SqCC; 14.6% NOS with primary lung cancer; all nonsmokers	139 female farmers from the general population matched ± 2 years; all nonsmokers	In-person interview; response rate not reported	*Rapeseed oil* Never Occasional Often	 24 106 9	 1.00 1.26 (0.68–2.63) 4.58 (0.56–37.08)	Age, length of menstrual cycle, menopause age, family history of lung cancer	Coal use was not adjusted for in the analysis. Definition of occasional use was not provided.
Dai *et al.* (1996), Harbin, 1992–93	120 women with primary lung cancer; 100% HC; 100% ADC; aged 30–69 years; Harbin resident at least 10 years; all nonsmokers	120 population 1:1 matched by age (±5 yrs); all nonsmokers	In-person interview in the hospital or at home; response rate not reported	*Pan-fried and deep-fried* ≤5 times/month >5 times/month	 	 1.0 9.20 (1.53–55.3) p=0.152	Income, area of resident, years of coal use in bedroom, years of coal heating, exposure to coal, intake of carrot, family history of cancer	

Table 2.3. (contd)

Reference, study location, period	Characteristics of cases, histological confirmation (HC), cell type (%)	Characteristics of controls	Data collection, response rate	Exposure categories	No. of cases	Relative risk (95% CI)	Adjustment factors	Comments (covariates considered)
Lei *et al*. (1996), Guangzhou, 1986	792 (563 men, 229 women) who died from lung cancer; 0% HC; no information on cell type; 566 smokers (443 men, 123 women)	792 (563 men, 229 women); 1:1 matched on sex, age (± 5 years), year of death, block of residence; no history of respiratory disease; 422 smokers (361 men, 61 women)	In-person interview with next of kin; response rate, 792/831 (95.3%); home interviews with spouses or relatives·	**Men**	Deaths			Crude analysis was presented; odds ratios were calculated based on the data presented. Definition of frying was not provided.
				Kitchen space				
				<1	18	1.0		
				1–2	66	[0.70]		
				≥2	431	[0.78]		
				Cooking activity				
				Infrequent	339	1.0		
				≤20 yrs	83	[0.92]		
				20–40 yrs	79	[1.10]		
				>40 yrs	30	[1.00]		
				Cooking frying				
				Preferred	192	1.0		
				Average	177	[0.72]		
				Not preferred	177	[0.89]		
				Women				
				Kitchen space				
				<1	6	1.0		
				1–2	28	[1.20]		
				≥2	179	[1.82]		
				Cooking activity				
				Infrequent	29	1.0		
				≤20 yrs	28	[0.72]		
				20–40 yrs	83	[0.88]		
				>40 yrs	62	[0.75]		
				Cooking frying				
				Preferred	55	1.0		
				Average	93	[0.88]		
				Not preferred	77	[1.06]		

Table 2.3. (contd)

Reference, study location, period	Characteristics of cases, histological confirmation (HC), cell type (%)	Characteristics of controls	Data collection, response rate	Exposure categories	No. of cases	Relative risk (95% CI)	Adjustment factors	Comments (covariates considered)
Lin *et al*. (1996), Harbin City	122 cases of adenocarcinoma; nonsmokers aged 30–69 years		122 matched controls by gender and age; non-smokers	>3 times per month frying	NR	3.00 (1.35–6.69)		Age adjusted
Shen *et al*. (1996), Nanjing, 1986–93	263 (men and women); 100% HC; ≥20 years old	263 general population; healthy residents of Nanjing, matched on age (±5 years), sex, neighbourhood	In-person interview; response rate not reported	**Cooking fumes** *SqCC* No Yes *ADC* No Yes		 1.0 3.81 (1.06–13.73) 1.0 2.99 (1.68–5.34)	Active smoking, chronic bronchitis, family history of cancer, coal stove for heating, fuel index.	Many limitations in the study methods—no information on gender, smoking or other factors
Wang *et al*. (1996), Shenyang, 1992–94	135 newly diagnosed cases of lung cancer; 57% HC; 100% HC; 54.5% ADC; 20% SCLC; 16.4% SqCC; 9.1% other; aged 35–69 years; all nonsmokers; ICD-9 (162)	135 general population matched on age (±5 years), sex, lifetime nonsmoking status	In person interview; response rate not reported	*Exposed to cooking fumes* No Yes Yes (adjusted)*	 77	 1.0 3.79 (2.29–6.27) 4.02 (2.38–6.78)*	* All study variables were considered in multivariate analysis, but results on cooking fumes were adjusted for coal smoke during cooking	

Table 2.3. (contd)

Reference, study location, period	Characteristics of cases, histological confirmation (HC), cell type (%)	Characteristics of controls	Data collection, response rate	Exposure categories	No. of cases	Relative risk (95% CI)	Adjustment factors	Comments (covariates considered)
Ko *et al*. (1997), Kaohsiung, Taiwan, 1992–93	117 female cases with primary lung cancer; 64.8% ADC; 17.1% SqCC; 15.2% SCLC; 2.9% LCC; 106 nonsmokers included in analysis ICD-9 (162)	117 hospital controls matched on age (±2 years), date of interview, nonsmoking-related disease	Personal interviews; response rate: cases, 117/128 (91.4%); controls, 117/125 (93.6%)	**Use of fume extractor** *Stir-frying* 0–4/week ≥5/week *Pan-frying* 0–4/week ≥5/week *Deep-frying* 0–4/month ≥5/month *Age when first cooking* ≥21 yrs 7–20 yrs *Before 20 yrs of age* Yes No *At 20–40 yrs of age* Yes No No (adjusted)* *After 40 yrs of age* Yes No	 14 91 29 76 82 23 36 67 7 60 25 78 76 22	 1.0 2.4 (1.1–5.2) 1.0 2.3 (1.2–4.6) 1.0 0.9 (0.5–1.9) 1.0 1.6 (0.8–3.0) 1.0 5.3 (1.1–25.6) 1.0 6.4 (2.9–14.1) 8.3 (3.1–22.7)* 1.0 2.3 (1.1–5.1)	Social class, residential area, and education were adjusted in all analysis. *Additional adjustment for tuberculosis, cooking fuels, living near industrial district	Coal use not significant Wood/charcoal use was significant ≤40 yrs Cooking fuel use was only adjusted for in selected analysis.

Table 2.3. (contd)

Reference, study location, period	Characteristics of cases, histological confirmation (HC), cell type (%)	Characteristics of controls	Data collection, response rate	Exposure categories	No. of cases	Relative risk (95% CI)	Adjustment factors	Comments (covariates considered)
Ko *et al.* (1997) (contd)				**Cooking oils**				
				Before 20 yrs of age				
				No cooking	38	1.0		
				Lard	51	1.6 (0.8–3.1)		
				Vegetable oil	16	2.0 (0.8–4.8)		
				At 20–40 yrs of age				
				No cooking	2	–		
				Lard	38	1.0		
				Vegetable oil	65	1.4 (0.8–2.6)		
				After 40 yrs of age				
				No cooking	2	–		
				Lard	7	1.0		
				Vegetable oil	91	0.5 (0.1–2.2)		

Table 2.3. (contd)

Reference, study location, period	Characteristics of cases, histological confirmation (HC), cell type (%)	Characteristics of controls	Data collection, response rate	Exposure categories	No. of cases	Relative risk (95% CI)	Adjustment factors	Comments (covariates considered)
Zhong *et al*. (1999), Shanghai, 1992–94	504 nonsmoking women ~77% HC; 76.5% ADC; 12.4% SqCC; 1.8% SCLC; 0.3% LCC; 9.0% mixed cells; aged 35–69 years; permanent residents of the area; Shanghai, China Cancer Registry; 145 smokers excluded from analysis	601 nonsmoking general population frequency-matched to age distribution by 5-year age intervals; 74 smokers excluded from analyses	In-person interview at hospital, home or work; response rate: cases, 649/706 (91.9%) (for smokers and nonsmokers); controls, 84%	*High-temperature cooking*			Age, education, income, intake of vitamin C, respondent status, exposure to passive smoking, family history of lung cancer, employment in high-risk occupation	
				No	339	1.0		
				Yes	165	1.64 (1.24–2.17)		
				Most frequently used oil				
				Soya bean oil	444	1.0		
				Rapeseed oil	49	1.84 (1.12–3.02)		
				Both oils	11	0.92 (0.37–2.28)		
				Stir-frying (no./week)				
				<7	40	1.0		
				7	434	0.38 (0.19–0.75)		
				>7	30	2.33 (0.68–7.95)		
				Pan-frying (no./week)				
				≤1	464	1.0		
				>1	40	2.09 (1.14–3.84)		
				Deep-frying (no./week)				
				≤1	469	1.0		
				>1	35	1.88 (1.06–3.32)		
				Smokiness in kitchen				
				None	177	1.0		
				Somewhat	241	1.67 (1.25–2.21)		
				Considerable	86	2.38 (1.58–3.57)		
				Eye irritation				
				Never	338	1.0		
				Rarely	49	1.49 (0.91–2.43)		
				Occasionally	74	1.75 (1.16–2.62)		
				Frequently	43	1.68 (1.02–2.78)		

Table 2.3. (contd)

Reference, study location, period	Characteristics of cases, histological confirmation (HC), cell type (%)	Characteristics of controls	Data collection, response rate	Exposure categories	No. of cases	Relative risk (95% CI)	Adjustment factors	Comments (covariates considered)
Ko *et al.* (2000), Kaohsiung, Taiwan, 1993–96	131 women with primary carcinoma of the lung; 100% HC; 19.8% SqCC; 62.6% ADC; 13.7% SCLC; 2.3% LCC; 1.5% NOS; >40 years of age; nonsmokers ICD-9 (162)	252 hospital eye or orthopaedic patients, or in for check-ups (diseases unrelated to smoking); 262 community, age-matched randomly selected from a computerized population database.; matched for age and date of interview; nonsmokers	Personal interviews; response rate: cases, 131/148 (88.5%); hospital controls, 252/281 (89.7%); community controls, 262/294 (89.1%)	*Daily cooking* No Yes *Age cooking started* >20 yrs ≤20 yrs *Yrs cooking at home* 1–20 21–40 ≥40 *Meals cooked/day* 1 2 ≥3 *Windows in kitchen* <2 ≥2 *Ventilation of kitchen* Poor Good	 1 130 47 83 36 74 20 13 71 46 62 69 71 60	 1.0 5.9 (0.7–53.6) 1.0 1.5 (0.9–2.4) 1.0 1.3 (0.6–2.6) 1.0 (0.4–2.9) 1.0 3.1 (1.6–6.2) 3.4 (1.6–7.0) 1.0 1.3 (0.8–2.1) 1.0 0.9 (0.6–1.4)	Socio-economic status, occupation, previous lung disease, passive smoking	Results shown are based on comparison with community controls. Role patterns were similar for hospital controls

Table 2.3. (contd)

Reference, study location, period	Characteristics of cases, histological confirmation (HC), cell type (%)	Characteristics of controls	Data collection, response rate	Exposure categories	No. of cases	Relative risk (95% CI)	Adjustment factors	Comments (covariates considered)
Ko *et al.* (2000) (contd)				*Eye irritation*				
				Rarely	84	1.0		
				Frequently	46	2.1 (1.3–3.5)		
				Stir-fry after fumes emitted				
				No	22	1.0		
				Yes	108	2.4 (1.4– 4.2)		
				Use of fume extractor				
				Before 20 yrs of age				
				Yes	40	1.0		
				No	43	0.9 (0.4–2.0)		
				Aged 20–40 yrs				
				Yes	85	1.0		
				No	45	2.2 (1.3–3.8)		
				Aged >40 yrs				
				Yes	114	1.0		
				No	12	1.3 (0.6–2.8)		

Table 2.3. (contd)

Reference, study location, period	Characteristics of cases, histological confirmation (HC), cell type (%)	Characteristics of controls	Data collection, response rate	Exposure categories	No. of cases	Relative risk (95% CI)	Adjustment factors	Comments (covariates considered)
Seow *et al*. (2000), Singapore, 1996–98	303 women; 100% HC; 54.8% ADC; 18.5% SqCC; 6.9% SCLC; 15.2% LCC; 4.6% NOS; aged <90 years; 127 smokers, 176 nonsmokers	765 hospital controls, frequency-matched for age, hospital, date of admission; no history of cancer, heart chronic disease or renal failure; 100 smokers, 663 nonsmokers	In-person interview within 3 months of diagnosis; response rate: cases, 361/380 (95.0%); controls, 765/789 (96.9%)	**Smokers** *Stir-frying* Less than daily Daily Less than daily with meat **Lifetime nonsmokers** *Stir-frying* Less than daily Daily Less than daily with meat **Smokers** *Stir-frying meat less than daily* Daily with meat Less than daily with meat with fume-filled kitchen Daily with meat with fume-filled kitchen **Lifetime nonsmokers** *Stir-frying meat less than daily* Daily with meat Less than daily with meat with fume-filled kitchen Daily with meat with fume-filled kitchen	 25 97 21 52 122 41 46 75 23 52 93 76 34 42	 1.0 ref 2.0 (1.0–3.8) 1.0 (0.5–2.4) 1.0 ref 1.0 (0.7–1.5) 0.9 (0.6–1.5) 1.0 ref 2.7 (1.3–5.5) 1.7 (0.7–3.9) 3.7 (1.8–7.5) 1.0 ref 0.9 (0.6–1.4) 1.1 (0.7–1.7) 1.0 (0.6–1.4)	Age , birthplace, family history of cancer, intake of fruits and vegetables. For smokers, odds ratios were additionally adjusted for duration of smoking (in years) and number of cigarettes smoked/day.	Current and ex-smokers grouped together

Table 2.3. (contd)

Reference, study location, period	Characteristics of cases, histological confirmation (HC), cell type (%)	Characteristics of controls	Data collection, response rate	Exposure categories	No. of cases	Relative risk (95% CI)	Adjustment factors	Comments (covariates considered)
Zhou *et al*. (2000), Shenyang, 1991–95	72 women with primary lung cancer; 100% HC; 100% ADC; aged 35–69 years; 20 smokers	72 general population, age 1:1 matched (±5 years) to cases; 23 smokers	In person interview; response rate not reported	*Eye irritation from smoke*		**Multivariate odds ratio**	Income, family history of lung cancer, number of live births	Fuel use for cooking/ heating was not considered in the analysis.
				Never		1.0		
				Slight		1.58 (0.62–4.03)		
				Medium		11.45 (3.10–42.4)		
				Heavy		3.41 (0.52–22.5)		
				p for trend		0.002		
				Location of kitchen		**Crude odds ratio**		
				Separate	6	1.00		
				In living room	63	1.40 (0.41–4.88)		
				In bedroom	3	1.00 (0.11–8.93)		
				p for trend		0.83		
				Cooking oil fumes				
				Slight	30	1.0		
				Medium/heavy	42	4.53 (2.09–9.94)		
				Deep-fried (no./week)				
				0–1		1.0		
				≥ 2	5	1.68 (0.45–6.84)		
				Extent of smoke when cooking	67			
				None	19	1.0		
				Slight	15	0.73 (0.28–1.90)		
				Medium	35	2.71 (1.09–6.80)		
				Heavy	3	1.32 (0.18–9.50)		
				p for trend		0.027		

Table 2.3. (contd)

Reference, study location, period	Characteristics of cases, histological confirmation (HC), cell type (%)	Characteristics of controls	Data collection, response rate	Exposure categories	No. of cases	Relative risk (95% CI)	Adjustment factors	Comments (covariates considered)
Lee *et al*. (2001), Kaohsiung, Taiwan, 1993–99	236 male, 291 female; only women with ADC, SqCC and SCLC retained for this analysis; 100% HC; 55.7% ADC; 20.3% SqCC; 8.6% SCLC; 2.1% LCC; 5.5% BA; 7.9% NOS; aged 18–83 years	407 hospital patients; matched to cases on sex, age (±2 years); ~2 controls per case; smoking in female controls not reported	In-person interview; response rate (presented for men and women combined): cases, 527/574 (91.8%); controls, 805/883 (91.2%)	*Kitchen with fume extractor*		***SqCC/SCLC***	Residence area (urban, suburban, rural), educational levels, socio-economic status (high, medium low), smoking (cumulative pack–years)	Wood/charcoal use was a significant risk factor; this was not adjusted for in the analysis.
				Yes	51	1.0		
				No	31	3.0 (1.3–7.1)		
				Cooking oils				
				Lard	28	1.0		
				Vegetable oil	54	0.7 (0.3–1.4)		
				Age first cooked (yrs)				
				>20	27	1.0		
				≤20	55	1.5 (0.7–3.1)		
				Stir-frying after fumes				
				No	23	1.0		
				Yes	59	0.9 (0.4–1.9)		
				Pan-frying after fumes				
				No	24	1.0		
				Yes	58	0.8 (0.4–1.5)		
				Deep-frying after fumes				
				No	44	1.0		
				Yes	38	1.0 (0.5–2.0)		

Table 2.3. (contd)

Reference, study location, period	Characteristics of cases, histological confirmation (HC), cell type (%)	Characteristics of controls	Data collection, response rate	Exposure categories	No. of cases	Relative risk (95% CI)	Adjustment factors	Comments (covariates considered)
Lee *et al*. (2001) (contd)				*Kitchen with fume extractor*		***ADC***		
				Yes	84	1.0		
				No	74	3.9 (2.3–6.6)		
				Cooking oils				
				Lard	50	1.0		
				Vegetable oil	108	1.2 (0.7–1.9)		
				Age first cooked (yrs)				
				>20	65	1.0		
				≤20	93	1.1 (0.7–1.7)		
				Stir-frying after fumes				
				No	29	1.0		
				Yes	29	2.0 (1.2–3.3)		
				Pan-frying after fumes				
				No	20	1.0		
				Yes	138	2.6 (1.5–4.5)		
				Deep-frying after fumes				
				No	68	1.0		
				Yes	90	1.6 (1.0–2.6)		
Metayer *et al*. (2002), Gansu Province, 1994–98	233 women; 37% HC; cell type distribution not presented; aged 30–75 years; 27 smokers	459 randomly selected from 1990 population census list of study areas; frequency-matched by age (± 5 years), Prefecture; 47 smokers	In-person interview; response rate: cases, 233/238 (98%); controls, 459/509 (90%)	*Type of oil (ever use)*			Age, Prefecture, socio-economic factors, respondent type.	
				Linseed	80	1.0		
				Rapeseed	53	1.65 (0.8–3.2)		
				Rapeseed + linseed	90	1.70 (1.0–2.8)		
				Perilla + hempseed	5	3.25 (0.8–14.0)		
				Stir-fying (times/month)				
				<15	71	1.0		
				15–29	60	1.96 (1.1–3.5)		
				30	52	1.73 (1.0–3.1)		
				≥31	45	2.24 (1.1–4.5)		
				p for trend		<0.05		

Table 2.3. (contd)

Reference, study location, period	Characteristics of cases, histological confirmation (HC), cell type (%)	Characteristics of controls	Data collection, response rate	Exposure categories	No. of cases	Relative risk (95% CI)	Adjustment factors	Comments (covariates considered)
Metayer *et al*. (2002) (contd)				*Deep-frying (times/month)*				
				Never/<1	70	1.0		
				1–2	86	0.82 (0.5–1.3)		
				≥3	38	0.83 (0.5–1.5)		
				Years of cooking				
				≤29	52	1.00		
				30–39	76	1.26 (0.6–2.8)		
				40–49	65	2.51 (0.9–6.8)		
				≥50	29	2.46 (0.8–7.9)		
				Age started cooking (yrs)				
				≤13	63	1.0		
				14–16	85	0.69 (0.4–1.1)		
				≥17	80	0.69 (0.4–1.2)		
				No. of meals cooked/day				
				≤2	193	1.0		
				≥3	36	1.36 (0.8–2.4)		
				Eye–throat irritation				
				Never	72	1.0		
				Occasionally/seldom	100	1.37 (0.8–2.2)		
				Frequently	54	2.82 (1.6–5.0)		
				p for trend		<0.01		
				Home smokiness				
				No	49	1.0		
				Some/little	155	0.90 (0.6–1.5)		
				Considerable	23	0.76 (0.4–1.6)		

Table 2.3. (contd)

Reference, study location, period	Characteristics of cases, histological confirmation (HC), cell type (%)	Characteristics of controls	Data collection, response rate	Exposure categories	No. of cases	Relative risk (95% CI)	Adjustment factors	Comments (covariates considered)
Chan-Yeung *et al.* (2003), Hong Kong, 1999–2001	331 histologically or cytologically proven cases of lung cancer	331 in- and out-patients without cancer; matched for age, sex	Personal interviews for cases and controls; response rates not given	**Frying foods** *Men* No or <2 yrs <3.5 yrs 3.5–7 yrs >7 yrs *Women* No or <2 yrs <3.5 yrs 3.5–7 yrs >7 yrs	 146 27 22 13 34 37 27 21	 1.0 0.69 (0.32–1.49) 0.83 (0.38–1.80) 1.22 (0.38–3.99) 1.0 1.08 (0.50–2.32) 1.05 (0.46–2.42) 1.54 (0.57–4.13)	Place of birth, educational status, family history of lung cancer, smoking (in men); educational status, smoking status (in women)	
Shi *et al* (2005), Shenyang, 2000–2002	618 newly diagnosed female patients with primary lung cancer	Randomly selected from the general population in urban districts	Face-to-face interviews	Cooking oil smoke		4.11 (2.14–7.89)		In multivariate analysis cooking oil smoke remained statistically significant but fuel smoke did not remain significant

Table 2.3. (contd)

Reference, study location, period	Characteristics of cases, histological confirmation (HC), cell type (%)	Characteristics of controls	Data collection, response rate	Exposure categories	No. of cases	Relative risk (95% CI)	Adjustment factors	Comments (covariates considered)
Yu *et al.* (2006) Hong Kong	291 women newly diagnosed with primary carcinomas; 96% participation rate; 68.5% ADC; aged 30–79 yrs; 67 smokers	661 randomly sampled residents from same districts as cases; frequency matched ±10 years; 322 (48.7% participation rate)	In-person interviews	*Total dish–years*			Age, education, employment status, previous lung diseases and history of lung cancer in first degree relatives (for model 1 regarding total dish-years); age, history of lung cancer in first degree relatives, intake of dark green vegetables, yellow orange vegetables, meat, coffee drinks, multivitamins, total dish–years	
				≤50		1.0		
				51–100		1.31 (0.81–2.11)		
				101–150		2.80 (1.52–5.18)		
				151–200		3.09 (1.41–6.79)		
				>200		8.09 (2.57–25.45)		
				Heating a wok to high temperatures				
				Never/seldom	25	1		
				Sometimes	37	1.02 (0.51–2.06)		
				Always	131	1.97 (1.06–3.65)		
				Use of fume extractor				
				Never	12	1		
				Ever	183	0.73 (0.29–1.87)		
				Use of peanut oil				
				Seldom/sometimes	70	1		
				Always	125	1.36 (0.87–2.15)		
				Use of corn oil				
				Seldom/sometimes	146	1		
				Always	49	1.27 (0.76–2.10)		
				Use of canola oil				
				Seldom/sometimes	181	1		
				Always	14	1.40 (0.59–3.30)		

ADC, adenocarcinoma; BA, basal-cell cancer; CI, confidence interval; ICD, International Classification of Diseases; LCC, large-cell carcinoma; NOS, not otherwise specified; SqCC, squamous-cell carcinoma; SCLC, small-cell cancer

The report by Wu-Williams *et al*. (1990) was based on 965 female lung cancer cases in northern China (445 in Harbin, 520 in Shenyang) and 959 female controls (404 in Harbin, 555 in Shenyang); 417 cases and 602 controls were nonsmokers. Seventy-four per cent (714/965) of the lung cancers were histologically/cytologically confirmed of which 44% were adenocarcinoma of the lung. Cases and controls were compared in terms of deep-frying practices. Compared with no deep-frying, the adjusted odds ratios were 1.2, 2.1 and 1.9 for deep frying once, twice and more than three times per month, respectively. Cases reported that their homes became smoky during cooking more often than controls and that they had irritated eyes more frequently during cooking. Compared with women who never or rarely experienced eye irritation during cooking, the risk was increased among those who occasionally (odds ratio, 1.6; 95% CI, 1.2–1.8) or frequently (odds ratio, 1.8; 95% CI, 1.3–2.6) reported such irritation. The authors noted that results were similar for squamous-/oat-cell cancers and adenocarcinomas and for smokers and nonsmokers. Pollution from coal burning for heating was a major risk factor in this area in northern China; in a multivariate analysis, deep-frying and eye irritation remained significant risk factors after adjusting for active smoking, previous lung diseases and coal burning (i.e. use of *kangs*). [The Working Group noted that, although coal heating was adjusted for in the multivariate analysis, the risk associated with frequent eye irritation may be due to fuel smoke and cooking smoke. The assessment of cooking practices was relatively limited in these two studies.]

Two small studies were conducted in Harbin (Dai *et al*., 1996) and Shenyang during the early 1990s (Wang *et al*., 1996; Zhou *et al*., 2000). The study by Dai *et al*. (1996) included 120 nonsmoking women who had adenocarcinoma of the lung and an equal number of nonsmoking controls; all were long-term (at least 10 years) residents of Harbin. The risk for adenocarcinoma of the lung was significantly influenced by frequency of frying food; women who pan-fried and deep-fried more than five times per month experienced a more than ninefold increased risk (adjusted odds ratio, 9.20; 95% CI, 1.53–55.28) after adjustment for various covariates including exposure to coal burning. [The Working Group noted that the prevalence of frying was not presented; the wide confidence interval is a concern. It is unclear whether these questions related to current or usual frying practices and whether other questions on cooking practices were asked. One Chinese study by Lin *et al.* (1996) evaluated the exposure to cooking oil fumes and the risk of lung adenocarcinoma among female nonsmokers. An age-adjusted increased risk of lung cancer (odds ratio, 3.0; 95% CI, 1.35–6.69) was observed for those who reported to fry food more than 3 times per month.

In a hospital-based study conducted in Shenyang, Wang *et al*. (1996) compared the experiences of 135 female lifetime nonsmokers who had been diagnosed with primary lung cancer and an equal number of nonsmoking female population controls. Of the lung cancers included, 57.2% were diagnosed pathologically or cytologically, 54.5% of which were adenocarcinoma. The risk for lung cancer increased significantly in association with some or frequent exposure to cooking fumes (odds ratio, 3.79; 95% CI, 2.29–6.27). In a multivariate analysis, exposure to cooking fumes remained a significant risk factor

(adjusted odds ratio, 4.02; 95% CI, 2.38–6.78) after adjusting for exposure to coal smoke and other factors. [The Working Group noted that this study was small and the exposure was limited to dichotomized (no/yes) assessment. The specific variables that were included in the multivariate analysis were not described. Coal use and exposure to coal smoke were reported in this study and may confound the findings related to cooking fumes. The validity of a diagnosis of adenocarcinoma is questionable because the authors stated that determination of the histological cell type was based on relevant medical record, chest X-rays, CT films and cytological and histological slides.]

Zhou *et al*. (2000) published another report on a subset of women from the hospital-based study in Shenyang (Wang *et al*., 1996). Specifically, 72 women (52 nonsmokers) who had been diagnosed with adenocarcinoma of the lung between 1991 and 1995 were compared with an equal number of control women (49 nonsmokers). A nonsignificant increased risk was observed in relation to deep-frying; the crude odds ratio was 1.68 (95% CI, 0.45–6.84) for deep-frying two or more times per week compared with none or once a week. The risk for adenocarcinoma increased significantly among women who reported that they experienced medium/heavy exposure to cooking fumes (crude odds ratio, 4.53; 95% CI, 2.09–9.94) or had frequent eye irritation and exposure to smoke during cooking. The risk for lung cancer was not significantly associated with whether cooking was carried out in a separate kitchen or in the living-room or bedroom. In a multivariate regression analysis, frequent eye irritation from smoke had an independent impact on risk. Compared with women who reported no eye irritation from smoke, those who reported slight, medium and heavy eye irritation showed elevated risks; the respective adjusted odds ratios were 1.58, 11.45 and 3.41 for (p for trend=0.002). [The Working Group noted that most of the lung cancer cases and controls included in the analysis by Zhou *et al*. (2000) represented a select subgroup of subjects reported by Wang *et al*. (1996) and the selection criteria were not described. This study was small and the confidence intervals were very wide.]

2.2.2 *Other parts of China and Singapore*

One of the first studies of exposure to cooking oil fumes and the risk for lung cancer was a large population-based case–control study conducted in the mid-1980s in Shanghai that was designed to examine lifestyle factors and lung cancer (Gao *et al*., 1987). The study included 672 women who had lung cancer and 735 population controls, of whom 436 cases and 605 controls were nonsmokers. Eighty-one per cent (542/672) of the lung cancers were diagnosed histologically or cytologically. Questions on cooking practices included type of oil used most often, frequency of frying, smokiness in the kitchen during cooking and frequency of eye irritation during cooking. Several measures of cooking practices were associated with an increased risk for lung cancer after adjusting for age, education and tobacco smoking. Compared with women who most frequently used soya bean oil, those who used rapeseed oil had an increased risk for lung cancer (adjusted odds ratio, 1.4; 95% confidence interval [CI], 1.1–1.8). The increased risk associated with the

use of rapeseed oil existed at each level of reported frequency of eye irritation when cooking. However, the increased risk associated with frequent eye irritation when cooking was found among both women who used soya bean oil and those who used rapeseed oil, although the highest risk was found in women who used rapeseed oil and frequently experienced eye irritation (adjusted odds ratio, 2.8; 95% CI, 1.8–4.3). There was a stepwise increase in risk associated with smokiness in the house. Specifically, women who reported occasional/frequent eye irritation and a considerable amount of smokiness in the house showed a more than twofold increased risk (adjusted odds ratio, 2.6; 95% CI, 1.8–3.7). Risk increased with increasing number of dishes prepared by stir-frying (adjusted odds ratios, 1.0, 1.2, 1.2 and 2.6 for ≤20, 20–24, 25–29 and ≥30 times per week, respectively) and deep-frying (adjusted odds ratios, 1.0, 1.5, 1.6 and 1.9 for 0, 1, 2 and ≥3 times per week, respectively). The risk patterns were similar for adenocarcinoma and squamous-cell/oat-cell carcinoma of the lung. [The Working Group noted that this was one of the first well-conducted population-based studies on this topic and had many strengths. The Working Group also noted that the increased risk was found with increasing number of dishes prepared by boiling food. Since it should produce less oil vapour than stir-frying and deep-frying, the comparably high odds ratios associated with boiling food were unexpected, although the authors suggested that oil was also added during boiling.]

In the 1990s, Zhong *et al*. (1999) conducted another study in Shanghai that used study methods similar to those used by Gao *et al*. (1987) and included a total of 649 women who had been diagnosed with incident lung cancer during 1992–94 and 675 population controls. Subjects who had smoked at least one cigarette a day for at least 6 months (145 cases, 74 controls) were excluded from the analyses. Thus, results on cooking practices were based on 504 cases and 601 controls who were lifetime nonsmokers. Seventy-seven per cent (387/504) of the lung cancers were diagnosed histologically or cytologically; 76.5% (296/387) of these were adenocarcinoma. Women who did not cook in a separate kitchen experienced a small increased risk (adjusted odds ratio, 1.28; 95% CI, 0.98–1.68). Risk for lung cancer was higher among those who had used rapeseed oil most frequently compared with those who had used soya bean oil (adjusted odds ratio, 1.84; 95% CI, 1.12–3.02). However, the risk was not elevated when both types of oil had been used (adjusted odds ratio, 0.92; 95% CI, 0.37–2.28). Risk also increased with higher frequency of frying. Compared with women who deep-fried once a week or less often, those who deep-fried more than once a week had a nearly twofold increased risk (adjusted odds ratio, 1.88; 95% CI, 1.06–3.32). Similarly, compared with women who pan-fried food once a week or less often, those who pan-fried food more than once a week had a significantly increased risk (adjusted odds ratio, 2.09; 95% CI, 1.14–3.84). However, the risk pattern in relation to stir-frying was less consistent. Compared with stir-frying less than seven times a week, women who stir-fried seven times a week had a reduced risk (adjusted odds ratio, 0.38; 95% CI, 0.19–0.75), but those who stir-fried more than seven times a week showed an increased risk (adjusted odds ratio, 2.33; 95% CI, 0.68–7.95). Women exposed to visible fumes from high-temperature frying had an increased risk

(adjusted odds ratio, 1.64; 95% CI, 1.24–2.17). This risk more than doubled for women who reported considerable smokiness (i.e. smokiness affected vision during cooking) from 'cooking oil or fumes' (adjusted odds ratio, 2.38; 95% CI, 1.58–3.57) compared with those who reported no smokiness. There was also a trend of increasing risk with increasing frequency of self-reported eye irritation; the adjusted odds ratio was 1.68 (95% CI, 1.02–2.78) for women who reported frequent (≥5 times per week) eye irritation compared with those who reported no eye irritation. Risk patterns related to Chinese-style cooking were generally similar in analyses that were restricted to all self-respondents (400 cases, 581 controls) or to self-respondents with histologically confirmed lung cancer (308 cases, 581 controls). Results were also comparable for women who had adenocarcinomas (296 cases), non-adenocarcinomas (91 cases) or unknown cell type (i.e. diagnosed clinically/radiologically) of lung cancer (117 cases). In a multivariate regression analysis, cooking temperature, smokiness in the kitchen during cooking, type of cooking oil and the frequency of stir-frying and of pan-frying displayed independent effects on the risk for lung cancer after adjustment for variables on ventilation (e.g. area of windows, cooking in a separate kitchen). Frequency of eye irritation and frequency of deep-frying were correlated with the other variables and did not exhibit independent effects on risk. [The Working Group noted several strengths in this population-based study: it was conducted among lifetime nonsmokers, the assessment of cooking practices was comprehensive and the analyses were thorough. Results were generally consistent across various subgroup analyses by histological and respondent type. The type of fuel used for cooking (coal, gas) was not significantly associated with risk and was not adjusted for in the multivariate analysis. It should be noted that the distribution of stir-frying was skewed and the confidence intervals were wide for stir-frying. The prevalence of use of rapeseed oil was 7.2% among controls in this study compared with 47.2% in Shanghai in the mid-1980s. The reason for the large differences in the pattern of use of rapeseed oil was not discussed but may be due to differences in the questions asked in the two studies.]

Two other studies were conducted in urban areas of China to examine the relationship between exposure to cooking oil fumes and risk for lung cancer. Shen *et al*. (1996) investigated potential risk factors for lung cancer among long-term (at least 20 years) residents of Nanjing in a hospital-based, case–control study that included 263 cases of lung cancer and an equal number of population controls. Only histologically confirmed lung cancers were studied (83 squamous-cell carcinomas, 180 adenocarcinomas). Exposure to cooking fumes was associated with an increased risk for squamous-cell carcinoma (adjusted odds ratio, 3.81; 95% CI, 1.06–13.73) and adenocarcinoma (adjusted odds ratio, 2.99; 95% CI, 1.68–5.34) of the lung. [The Working Group noted that the study had serious limitations. The report lacked details regarding the study design (e.g. response rate) and characteristics of the study population (e.g. gender distribution, active smoking history). The source of information on exposures was not presented. Only significant results were presented; risk patterns in relation to the amount of oil used in cooking and frequency of cooking per week were not presented.]

Cooking practices and lung cancer mortality were investigated in a case–control study in Guangzhou (Lei *et al*., 1996). Using registered deaths that occurred in this city in 1986, the analysis was based on 792 (562 men, 229 women) lung cancer deaths reported in long-term (at least 10 years) Guangzhou residents. The comparison group included other registered decedents who were matched to cases on gender, age (±5 years) and residence and whose cause of death was unrelated to cancer or respiratory disease. A standardized interview administered to spouses or cohabiting relatives of the decedents collected information on active smoking, exposure to secondhand smoke, living conditions, cooking facilities, exposure to coal dust and dietary habits. In analyses conducted separately in men and women, cases and controls did not differ significantly in their preference of frying, years of cooking (infrequent, ≤20, 20–40, >40 years) or size the of kitchen (<1, 1–2, ≥2 m^2 per household). Similarly, living conditions (type of building, location of residence, interior dimensions of residence) and average size of the living area did not differ significantly between lung cancer cases and controls. [The Working Group noted that the study had several deficiencies. The quality of information on cooking practices obtained from next of kin is questionable; a considerable amount of information was missing; the data analysis was confined to crude analysis; and the accuracy of lung cancer diagnosis based on reviewed death records is not known for China.]

In addition to the above-mentioned studies that were conducted largely in urban areas of China, two studies were conducted in more rural parts of China: one in Xuan Wei County, Yunnan Province (Lan *et al*., 1993), an area where mortality rates for lung cancer are very high among women, and one in Gansu Province, a rural area in northwestern China (Metayer *et al*., 2002).

The study in Xuan Wei County, Yunnan Province, investigated the use of rapeseed oil in the study population and was based on 139 incident female lung cancers that were diagnosed between 1988 and 1990 and 139 age-matched controls (Lan *et al*., 1993). Of the lung cancer cases, 55 (39.6%) were diagnosed cytologically/pathologically. All cases and controls were nonsmokers. Compared with women who never used rapeseed oil, those who used it occasionally or frequently showed an increased risk; the respective adjusted odds ratios were 1.26 (95% CI, 0.68–2.63) and 4.58 (95% CI, 0.56–37.08) after adjusting for age, length of menstrual cycle, age at menopause and family history of lung cancer. [The Working Group noted that coal use was prevalent in this study population and was not considered in the analysis on cooking oil. In addition, the definition of occasional or frequent use of rapeseed oil was not provided. Few subjects (2.2% of controls) were frequent users of rapeseed oil and the confidence limits were wide. It is unclear whether other questions related to cooking practices were asked.]

Metayer *et al*. (2002) conducted a population-based case–control study that was designed to examine the association between cooking oil fumes and other sources of indoor air pollution and lung cancer in Gansu Province. The study included 233 female lung cancer cases and 459 control subjects; 206 cases and 411 controls were nonsmokers. Thirty-seven per cent of the cases were cytologically or histologically confirmed. Smokers (27 cases, 47 controls) were included in the analysis on cooking practices.

Compared with women who only used linseed oil, an elevated risk was associated with the use of rapeseed oil alone (adjusted odds ratio, 1.65; 95% CI, 0.8–3.2), rapeseed and linseed oil in combination (adjusted odds ratio, 1.70; 95% CI, 1.0–2.5) and perilla/hempseed oil (adjusted odds ratio, 3.25; 95% CI, 0.8–14.0). The risk for lung cancer was unrelated to the frequency of deep-frying (adjusted odds ratio, 1.0, 0.82 and 0.83 for never/less than once a month, 1–2 times per month and ≥3 times per month, respectively). However, there was a significant exposure–response of increased risk with increasing frequency of stir-frying (adjusted odds ratios, 1.00, 1.96, 1.73 and 2.24, for stir-frying <15, 15–29, 30 and ≥31 times per month; p for trend=0.03). Risk tended to increase with decreasing age when started to cook (adjusted odds ratio, 0.69 for started cooking at age ≥17 versus ≤13 years), with increasing number of meals cooked per day (adjusted odds ratio, 1.36 for ≥3 meals versus ≤2 meals) and with increasing years of cooking (adjusted odds ratio, 1.0, 1.26, 2.51 and 2.46 for ≤29, 30–39, 40–49 and ≥50 years) (p for trend <0.09). Although women who reported frequent eye–throat irritation showed a significantly increased risk (adjusted odds ratio, 2.82; 95% CI, 1.6–5.0) compared with those who never experienced such irritation (p trend <0.01), the general level of indoor smokiness was unrelated to risk. Risk for lung cancer was not elevated among women who reported considerable home smokiness (odds ratio, 0.76; 95% CI, 0.4–1.6) compared with those who reported no smokiness. The authors hypothesized that, as underground cave dwellings in Gansu Province reported high ventilation rates as measured by air exchanges per hour, this may explain the lack of any risk associated with general smokiness. The positive associations with stir-frying, years of cooking and eye irritation were found in women who cooked with linseed oil only (80 cases, 247 controls) and in those who cooked with rapeseed oil (148 cases, 205 controls). In addition, the authors reported that the results were generally similar when the analyses were restricted to self-respondents or to histologically confirmed lung cancer cases. [The Working Group noted that this study included a comprehensive assessment of cooking practices and conditions. Coal use for heating/cooking was not significantly associated with lung cancer risk in this population. Although coal use was not considered in the analysis on cooking practices, it is unlikely to confound the findings. The results suggest that fumes from all types of oil may have deleterious effect. This study is limited by a relatively large number of only clinically/radiologically diagnosed lung cancers and because interviews were conducted with next-of-kin respondents for 123 cases (53%) and 20 controls (4%).]

Shi *et al*. (2005) conducted a case–control study that included nonsmoking women who had been newly diagnosed with lung cancer between June 2000 and December 2002 in city hospitals of urban Shenyang. Eighty-four per cent of cases were diagnosed pathologically or cytologically. Controls were randomly selected from the general female population of urban areas and matched on age (within ±2 years). Information on demographic factors, exposure to cooking oil smoke, types of fuel used, exposure to coal smoke, use of heated *kangs*, passive smoking, history of lung disease and other factors was obtained. Risk for lung cancer increased significantly in association with exposure to

cooking oil smoke (odds ratio, 3.18; 95% CI, 2.55–3.97) and fuel smoke (odds ratio, 2.56; 95% CI, 1.83–4.55) after adjusting for education and social class. Risk was unrelated to the use of *kangs* (odds ratio, 1.12; 95% CI, 0.91–1.39). In a multivariate analysis, the increased risk associated with cooking oil smoke remained statistically significant (adjusted odds ratio, 4.11; 95% CI, 2.14–7.89) but the risk associated with fuel smoke was no longer statistically significant. [The Working Group noted that, although the finding on cooking oil smoke was adjusted for fuel smoke, it is difficult to rule out residual confounding in this study.]

Seven studies on cooking practices and the risk for lung cancer have been conducted in other parts of China, including one study in Hong Kong Special Administrative Region (Yu *et al*., 2006), four in Taiwan (Ger *et al*., 1993; Ko *et al*., 1997, 2000; Lee *et al*., 2001) and two in Singapore (MacLennan *et al*., 1977; Seow *et al*., 2000).

(a) Hong Kong Special Administrative Region

Chan-Yeung *et al*. (2003) conducted a case–control study in Hong Kong Special Administrative Region during the late 1990s which included 331 Chinese residents (212 men, 119 women) who had been diagnosed with a histologically confirmed primary lung cancer in a large teaching hospital. An equal number of age- and gender-matched residents identified from the same hospital who had non-malignant respiratory diseases were used as controls. Most of the women were nonsmokers (106 cases, 113 controls) while many of the men were smokers (160 cases, 116 controls). All cases and controls were interviewed by one interviewer and were asked about regular exposure to cooking fumes from frying in the house. Years of regular exposure to frying food was not significantly related to the risk for lung cancer in men or women. For women with no or less than 2 years of exposure, the respective odds ratios associated with <3.5 years, ≥3.5–≤7 and >7 years of exposure to frying food were 1.08 (95% CI, 0.50–2.32), 1.05 (95% CI, 0.46–2.42) and 1.54 (95% CI, 0.57–4.13) after adjustment for demographic factors and smoking habits. The corresponding risk estimates in men were 0.69 (95% CI, 0.32–1.49), 0.83 (95% CI, 0.38–1.80) and 1.22 (95% CI, 0.38–3.99). [The Working Group noted that this study included a single measure of exposure to frying in the house. Control subjects had non-malignant respiratory diseases and may have had risk factor profiles that are more similar to the lung cancer patients than control subjects selected from the general population. Thus, estimates of risk associated with exposure to frying may be underestimated.]

Yu *et al*. (2006) conducted a case–control study in Hong Kong Special Administrative Region during the early 2000s that included 200 nonsmoking Chinese women who had been diagnosed with a histologically confirmed primary lung cancer in a large oncology centre and 285 population controls. All but 12 participants (six cases, six controls) were interviewed in person using a standardized structured questionnaire that asked extensive questions about lifetime cooking habits since childhood and included number of years of cooking, the frequencies of stir-frying, pan-frying and deep-frying, the types of cooking oils used, the use of a fume extractor or exhaust fans and the habit of

heating up a wok to high temperatures. The risk for lung cancer increased significantly with increasing total cooking 'dish–years', a composite index that was constructed to account for both the frequency and the duration of cooking. The odds ratios were 1.00, 1.31, 2.80, 3.09 and 8.09, respectively, for ≤50, 51–100, 101–150, 151–200 and ≥200 'total frying dish–years' after adjusting for age, education, employment status, previous lung disease and family history of lung cancer. The results remained significant after further adjustment for factors that may contribute to indoor air pollution (e.g. radon, exposure to environmental tobacco smoke, use of kerosene, use of firewood, burning of incense and use of mosquito coils) and dietary factors. In addition, a trend of increasing risk with heating a wok to high temperature was observed; the odds ratio was 1.0, 1.02 and 1.97 in relation to never/seldom, occasionally and always engaging in such cooking habits. Risk (per 10 dish–years) was highest for deep-frying (odds ratio, 2.56; 95% CI, 1.31–5), intermediate for pan-frying (odds ratio, 1.47; 95% CI, 1.27–1.69) and lowest for stir-frying (odds ratio, 1.12; 95% CI, 1.07–1.18). However, risk was not significantly associated with the use of a particular type of oil (peanut oil, corn oil, canola oil) for cooking or with using a fume extractor. A pattern of risk associated with total cooking dish–years was observed for adenocarcinoma and for non-adenocarcinoma, although the results were stronger for adenocarcinoma of the lung, which represented 69% of the lung cancer cases included in this study. [The Working Group noted that this study included a comprehensive assessment of lifetime cooking habits. Duration and frequency of exposure was captured by a composite index, 'total cooking dish–years', which permitted a quantitative assessment of cumulative exposure. While the confidence interval for the highest exposure category (>200 dish–years) was wide, there was a monotonic increase in risk with increasing exposure. It should be noted that this index was computed based on the number of dishes cooked by the three cooking methods (stir-frying, pan-frying and deep-frying). Although the response rate among controls was modest (~50%), few differences between cases and controls were noted for demographic factors except for a higher rate of employment among controls (88%) compared with cases. Elevated risks associated with moderate to high levels of cooking (>100 dish–years) remained after further adjustment for employment status.]

(b) Taiwan (China)

Four hospital-based case–control studies of lung cancer from Taiwan investigated the role of cooking practices. The main type of oil used in Taiwan is vegetable oil (mainly peanut or soya bean oil).

Ger *et al*. (1993) conducted a hospital-based case–control study in Taipei, Taiwan, that included 131 primary lung cancers (92 men, 39 women) identified between 1990 and 1991. All were histologically confirmed. Two control groups were interviewed; 262 hospital controls were matched to cases on sex, date of birth (±5 years), date of interview (±4 weeks) and insurance status and 262 neighbourhood controls were matched to cases on age, sex and residence of case at the time of diagnosis. In total, 48 cases and 229 controls (111 hospital controls, 118 neighbourhood controls) were nonsmokers. Risk

for adenocarcinoma and squamous-/small-cell cancers in men and women combined was unrelated to cooking style; cases and controls did not differ in pan-frying, stir-frying, deep-frying or boiling practices after adjusting for active smoking and other covariates. Risk for adenocarcinoma increased significantly in persons who reported that they were professional cooks (adjusted odds ratio, 5.54; 95% CI, 1.49–20.65); no increased risk was found for squamous-cell cancer (adjusted odds ratio, 1.16; 95% CI, 0.32–422). [The Working Group noted that this study included few female lung cancer patients. Results were based on dichotomized cooking variables (e.g. no/yes frying) that were not defined.]

Three hospital-based case–control studies were conducted in Kaohsiung, a heavily industrialized city in Taiwan (Ko *et al*., 1997, 2000; Lee *et al*., 2001). The designs of these studies were similar. The first study included 117 female lung cancer cases identified between 1992 and 1993 who were compared with 117 hospital controls who were admitted for a health check-up (55 controls) or for eye diseases (62 controls) (Ko *et al*., 1997). Active smokers (11 cases, three controls) were excluded so that the analysis was based on 105 case–control pairs who were nonsmokers. In a univariate analysis, risk for lung cancer increased with increased frequency of stir-frying (odds ratio, 2.4; 95% CI, 1.1–5.2 for ≥5 versus 0–4 times per week), pan-frying (odds ratio, 2.3; 95% CI, 1.2–4.6 for ≥5 versus 0–4 times per week) but not with deep-frying (odds ratio, 0.9; 95% CI, 0.5–1.9 for ≥5 versus 0–4 times per month). Risk also increased with younger age when started to cook (odds ratio, 1.6; 95% CI, 0.8–3.0 for started at ages 7–20 versus after age 21 years). Risk for lung cancer was elevated in women who cooked in a kitchen without a fume extractor; this was found at different ages of cooking including before age 20 years (odds ratio, 5.3; 95% CI, 1.1–25.6), between the ages of 20 and 40 years (odds ratio, 6.4; 95% CI, 2.9–14.1) or after 40 years of age (odds ratio, 2.3; 95% CI, 1.1–5.1). The risk for lung cancer was not significantly related to types of cooking oil (lard versus vegetable oil). In a multivariate analysis, use of a fume extractor during cooking between the ages of 20 and 40 years remained statistically significant (adjusted odds ratio, 8.3; 95% CI, 3.1–22.7). [The Working Group noted that, while there was no increased risk associated with cooking with coal, the risk increased significantly in relation to cooking with wood or charcoal before 20 years of age and between the ages of 20 and 40 years. These investigators examined the combined effects of frying and use of fume extractors between the ages of 20 and 40 years. The increased risks associated with stir-frying and pan-frying remained regardless of use of fume extractors.]

A second study conducted by the same group of investigators was based on 131 lung cancer cases identified between 1993 and 1996, 252 hospital controls and 262 community controls; all participants were nonsmokers (Ko *et al*., 2000). All lung cancers were histologically confirmed; 63% were adenocarcinoma of the lung. Of the more than 10 variables related to cooking practices that were investigated, risk for lung cancer was associated with five. There was a significant trend of increasing risk with number of meals cooked per day (adjusted odds ratios, 1.0, 3.1 and 3.4 for cooking 1, 2 and 3 meals per day, respectively). Risk was also elevated for women who cooked between the ages of 20 and 40 years without a fume extractor (adjusted odds ratio, 2.2; 95% CI, 1.3–3.8). In

addition, women who reported frequent eye irritation (odds ratio, 2.1; 95% CI, 1.3–3.5) showed significantly elevated risks. Subjects who usually waited until fumes were emitted from the oil and then stir-fried, pan-fried or deep-fried also experienced about a twofold increased risk that was statistically significant. In contrast, years of cooking at home, general ventilation in the kitchen, number of windows in kitchen (<2 versus ≥2) and size of openings (windows) to the outside did not differ between cases and controls. The risk estimates presented above were obtained when cases were compared with community controls, and risk patterns were generally similar when lung cancer cases were compared with hospital controls. [The Working Group noted that use of coal and wood/charcoal was not reported. However, since this study overlapped with the earlier study (Ko *et al*., 1997), the same comments relating to cooking fuel are applicable.]

A further expansion of the previous two studies included lung cancer patients diagnosed between 1993 and 1999 (Lee *et al*., 2001). Women who had been diagnosed with squamous-/small-cell (84 cases) cancer or adenocarcinoma of the lung (162 cases) and 407 corresponding controls were included in the analysis. Women who had other lung cancer cell types (45 cases) and men who had lung cancer were excluded from the analysis of cooking practices. Prevalence of smoking in female controls was not presented but, among female cases, 96.9% of those with adenocarcinoma of the lung and 81.6% of those with squamous-/small-cell lung cancer were nonsmokers. Risk was significantly higher for those who cooked in a kitchen without a fume extractor; the adjusted odds ratio was 3.0 (95% CI, 1.3–7.1) for squamous-/small-cell cancer and 3.9 (95% CI, 2.3–6.6) for adenocarcinoma of the lung. Women who stir-fried, pan-fried or deep-fried only when fumes were emitted from the oil showed significantly higher risk for adenocarcinoma (respective odds ratios, 2.0, 2.6 and 1.6) but not for squamous-/small-cell cancer of the lung (respective odds ratios, 0.9, 0.8 and 1.0). Risk for either cell type of lung cancer was not significantly influenced by age when first started to cook (>20 versus ≤20 years) or type of cooking oils (lard versus vegetable oils). In a multivariate regression analysis, cooking in a kitchen that was not equipped with a fume extractor remained a significant risk factor for both squamous-/small-cell lung cancer and adenocarcinoma of the lung; the respective adjusted odds ratios were 3.3 (95% CI, 1.2–9.2) and 3.8 (95% CI, 2.1–6.8). In addition, waiting to fry until the cooking oil has reached a high temperature was associated with an increased risk for adenocarcinoma of the lung (adjusted odds ratio, 2.1; 95% CI, 1.1–3.0) but not for squamous-/small-cell lung cancer. [The Working Group noted that there was an overlap of cases and controls in the three reports by Ko and colleagues. An advantage of the second report (Ko *et al*., 2000) is that a group of population controls was also included and most of the risk patterns were similar compared with both control groups. It should be noted that use of wood/charcoal, a significant risk factor for both cell types of lung cancer, was not adjusted for in the analysis on cooking practices.]

(c) Singapore

Seow *et al*. (2000) conducted a hospital-based case–control study in Singapore during the late 1990s; 303 women who had been diagnosed with a pathologically confirmed primary lung cancer (56% were adenocarcinoma of the lung) and 765 hospital controls were compared. Analyses were conducted separately for smokers (former and current smokers combined; 127 cases, 100 controls) and lifetime nonsmokers (176 cases, 663 controls). All participants were interviewed in person using a standardized questionnaire that asked extensive questions on diet, reproductive history, exposure to secondhand smoke and cooking practices. Specifically, questions included the frequency of stir-frying, types of oil used and usual cooking practice 20–30 years before diagnosis. Subjects were also asked how often the air in their kitchen became filled with oily 'smoke' during frying. For each of these cooking exposures, there were six possible responses ranging from never/less than yearly, less than monthly, to daily and more than once a day. Among smokers, the risk for lung cancer doubled in association with daily stir-frying (adjusted odds ratio, 2.0; 95% CI, 1.0–3.8) after adjusting for a large number of potential confounders. This increase in risk was confined to those who stir-fried meat on a daily basis (adjusted odds ratio, 2.7; 95% CI, 1.3–5.5). Compared with smokers who stir-fried meat less frequently than daily, risk was intermediate for those who stir-fried meat less than daily in a fume-filled kitchen (adjusted odds ratio, 1.7; 95% CI, 0.7–3.9) and was highest for those who stir-fried daily and reported a smoke-filled kitchen (adjusted odds ratio, 3.5; 95% CI, 1.8–6.9). Women who stir-fried meat daily and primarily used unsaturated oils had the highest risk (adjusted odds ratio, 4.6; 95% CI, 1.6–13.0), while risk was intermediate for those who stir-fried daily but did not use unsaturated oils exclusively (adjusted odds ratio, 2.2; 95% CI, 1.2–4.2). In contrast, the risk for lung cancer in nonsmokers was unrelated to stir-frying (adjusted odds ratio, 1.0; 95% CI, 0.7–1.5) or stir-frying meat daily (adjusted odds ratio, 0.9; 95% CI, 0.6–1.4). Risk for lung cancer in nonsmokers was not affected by smokiness of kitchen or types of oil used. [The Working Group noted that this study presented no data on pan-frying or deep-frying. Although fuel use was not considered in this analysis, it is unlikely to be an important confounder because gas/kerosene is usually used (MacLennan *et al*., 1977). However, this was one of the few studies that described the questions that were asked regarding cooking practices and that specifically addressed cooking practices during the period 20–30 years before cancer diagnosis/interview. Reasons for the differences in findings by smoking status are not apparent but the sample size of smokers was modest. The risk estimates presented in the tables were slightly different from the numbers presented in the text; the numbers presented in the tables are those given in this Monograph.]

2.3 Meta-analysis

Feng & Ling (2003) carried out a meta-analysis on case–control studies among nonsmoking women that were published between 1992 and 2002 in the English and

Chinese literature and examined the relationship between exposure to cooking oil fumes and lung cancer. Six studies (two in English and four in Chinese) were conducted in mainland China and two (in English) in Taiwan. All studies reported significantly increased odds ratios ranging from 2.10 to 9.20. The combined odds ratio using a fixed effects model was 2.94 (95% CI, 2.43–3.56). [The Working Group noted that the two studies in Taiwan had some overlap in their study subjects. Two reports by the same group of authors in China (Wang *et al*., 1996), one in English and one in Chinese, essentially overlap one another. The exposure metrics were not uniform and the rationale for selecting certain odds ratios out of a range in each paper was not entirely clear.

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3. Studies of Cancer in Experimental Animals

3.1 Cooking oil fumes

Whole-body and inhalation exposure

(a) Mouse

Four groups of 30–32 male and 30–32 female Balb/c mice (weighing 15±3 g) [age unspecified] were exposed to air heated at 22–30°C (control) or ~9, 21 and 39 mg/m^3 cooking oil fumes for 30 min per day for 2 months, then every other day for a period of 6 months (150 times overall) after which time they were killed. Oil fumes were generated by heating an unspecified volume of unrefined rapeseed oil at a temperature of 270±5°C in a steel container with an electric heating element. Fumes were directed into a cylindrical 1-m^3 exposure chamber. The incidence of lung tumours in both sexes combined was 0.0 (0/61), 15.09 (8/53; p<0.05), 20.00 (10/50; p<0.05) and 22.00% (11/50; p<0.05) for the control, low-, mid- and high-dose groups, respectively. The incidence in females was 0.00 (0/31), 12.00 (3/25; p<0.05), 25.00 (5/20; p<0.05) and 25.92% (7/27; p<0.05), respectively, and that in males was 0.00 (0.30), 17.86 (5/28; p<0.05), 16.67 (5/30; p<0.05) and 17.39% (4/23; p<0.05), respectively. The lung tumours were mainly adenocarcinomas (Zhang *et al*., 2003; Chen *et al*., 2005).

(b) Rat

Four groups of 30–35 male and 30–35 female Sprague-Dawley rats (weighing ~127 g) [age unspecified] were exposed to air or ~7, 15 and 35 mg/m^3 cooking oil fumes for 30 min every other day for 12.5 months after which they were killed. Oil fumes were generated by heating 250 mL unrefined rapeseed oil to a temperature of 260°C in steel container with an electric heating element. Fumes were directed into a cylindrical 2.2-m^3 exposure chamber. The incidence of lung carcinoma in both sexes combined was 0.0 (0/70), 6.56 (4/61), 8.96 (6/67) [p<0.05] and 12.70% (8/63) [p<0.005] for the control, low-, mid- and high-dose groups, respectively. The incidence in females was 0.0 (0/35), 6.45 (2/31), 11.76 (4/34) and 19.35% (6/31) [p<0.01], respectively, and that in males was 0.0 (0/35), 6.67 (2/30), 6.06 (2/33) and 6.25% (2/32), respectively (Long *et al*., 2005).

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4. Mechanistic and Other Relevant Data

4.1 Toxicokinetics

See the monograph on Household use of solid fuels.

4.2 Mechanisms of carcinogenesis

4.2.1 *Polycyclic aromatic hydrocarbons (PAHs)*

See the monograph on Household use of solid fuels.

Siegmann and Sattler (1996) detected a variety of genotoxic PAHs (e.g. benzo[*a*]anthracene, chrysene, benzo[*a*]pyrene) in vegetable oils (rapeseed, corn and peanut) heated to above 260ºC (1.1–22.8 μg/m^3 PAHs). Wu *et al*. (1998) detected a variety of mutagenic PAHs (e.g. benzo[*a*]pyrene) and nitro-PAHs (e.g. 1,3-dinitropyrene) in fumes of lard, soya bean oil and peanut oil heated to above 250ºC; the emission of PAHs and nitro-PAHs were reduced upon addition of the antioxidant catechin.

4.2.2 *Particles*

See the monograph on Household use of solid fuels.

4.2.3 *Genetic and related effects*

(a) Humans

Cherng *et al*. (2002) used the reverse-transcription polymerase chain reaction to investigate expression of human 8-oxoguanine DNA glycosylase 1 (HOGG1), a repair enzyme that removes 8-hydroxydeoxyguanine (8-OHdG) from damaged DNA, in the peripheral blood lymphocytes of 94 professional cooks and 43 home cooks exposed to cooking oil emissions. The results showed that HOGG1 expression in cooking oil emissions-exposed cooks was significantly higher than that in 111 control subjects. Odds ratios, adjusted for age, sex and smoking and drinking status, for home cooks versus controls and professional cooks versus controls were 3.94 (95% CI, 0.95–16.62) and 10.12 (95% CI, 2.83–36.15), respectively. Furthermore, significant induction of HOGG1 expression was confirmed *in vitro* in human lung adenocarcinoma CL-3 cells after exposure to cooking oil emissions extracts.

(b) Experimental systems

(i) *Experimental animals*

Glaser *et al*. (1989) reported that flow cytometric analyses of lung cells from Wistar rats exposed to emissions (20 mg/m^3) from fish frying in fat for 28 days showed alterations in the structure and content of nuclear DNA. In comparison with the control group, samples from exposed animals showed a significant shift and broadening of the G_1 peak, which may be caused by loss of chromosomal fragments or by chromosomal aberration during cell division.

Several studies have documented clastogenic effects, genotoxic effects and oxidative stress in experimental animals exposed to cooking oil fumes.

Intraperitoneal injection of male Kunming mice with condensates of emissions from rapeseed oil heated to 270ºC (doses of 800, 1600, 2400 or 3200 mg/kg body weight [bw]) induced a significant dose-dependent increase in the frequency of micronucleated polychromatic erythrocytes in the bone marrow. The addition of the antioxidant butylated hydroxyanisole to the oil reduced the magnitude of the effect (Chen *et al*., 1988; Qu *et al*., 1992). Two studies have shown induction of bone-marrow cell micronuclei in mice exposed to cooking oil fumes. Chen *et al*. (1992) reported a time- and dose-dependent increase in bone-marrow micronuclei in male Swiss mice exposed by inhalation to rapeseed oil fumes for 3 h per day, 6 days per week for 4 weeks. Liu *et al*. (1987) showed an increase in the frequency of bone-marrow cell micronuclei in mice exposed by inhalation for 5 days to 1/16 of the LD_{50} of cooking oil fumes from soya bean oil heated at 250–270ºC. A subsequent study by Li *et al*. (1998) showed that intratracheal instillation of refined vegetable oil (heated to 270–280ºC)-fume condensate into Sprague-Dawley rats (doses of 225, 450 or 900 mg/kg bw) elicited a significant increase in bone-marrow cell micronuclei.

Chen *et al*. (1996) revealed significant induction of chromosomal aberrations in diploid male germ cells (diakenesis/meiosis I) of ICR mice exposed to rapeseed oil emissions condensate (270–280ºC) by daily intraperitoneal injections of 100, 400 or 1600 mg/kg bw for 5 days.

Zhang *et al*. (2001) observed significant increases in DNA damage in peripheral blood lymphocytes (comet assay) of Balb/c mice following inhalation exposure for 8 months to 9.1–39 mg/m^3 fumes of heated rapeseed oil.

Kawai *et al*. (2006) showed that 4-oxo-2-hexenal (4-OHE), a mutagenic substance formed by the peroxidation of ω-3 polyunsaturated fats such as linolenic acid, was present in a condensate of smoke released during fish frying. In an earlier study, Kasai *et al*. (2005) noted that oral administration of 4-OHE to mice induced an increase in the levels of DNA adducts (4-OHE-deoxycytosine, 4-OHE-deoxyguanosine, and 4-OHE-5-methyl-deoxycytosine) in the gastrointestinal tract (i.e. oesophagus, stomach and intestine). They also showed that 4-OHE, which was detected in the volatile emissions of heated perilla oil (from *Perilla frutescens,* a member of the mint family) and broiled fish, seems to be produced by the oxidation of ω-3 fats (e.g. linolenic acid). Xi *et al*. (2003) showed that

intratracheal instillation of heated cooking oil emissions condensate into Wistar rats induced a dose- and time-dependant increase in the frequency 8-OHdG–DNA adducts in lung tissue. Li *et al*. (1998) also noted a decrease in superoxide dismutase activity and an increase in malondialdehyde (an indicator of oxidative stress) in lung tissue. Similarly, significantly decreased superoxide dismutase activity and increased malondialdehyde content in lung tissue was reported in Sprague-Dawley rats exposed by inhalation to 43 mg/m^3 fumes from cooking oil heated to 270–280ºC for 20–60 days (Rang *et al.*, 2000).

Rang *et al.* (2000) showed in the study above that lung tissue samples showed high P53 protein content. Using immunohistochemical methods, Liu *et al*. (2005) also observed overproduction of P53 and a decrease in P16 protein in lung tissues of Sprague-Dawley rats exposed by inhalation to 43.9 mg/m^3 fumes from cooking siritch oil [i.e. Chinese *Hu-Ma* oil or linseed oil] (heated to 200–220ºC) for 20–60 days. Long *et al*. (2005) showed that Sprague Dawley rats exposed by inhalation to fumes from rapeseed oil heated to 260ºC (6.9–35 mg/m^3 for 30 min every other day for 12.5 months) developed pulmonary carcinoma in addition to enhanced production of P53 and a decrease in fragile histidine triad protein in lung (bronchial epithelia) tissue sections.

A study that used the *Drosophila melanogaster* sex-linked recessive lethal assay showed that exposure to a condensate of a cooking oil fume (110, 320 and 960 mg/L in food) induced heritable mutations (Li *et al*., 1999). Wang *et al*. (1995) revealed that tracheal epithelial cells removed from Wistar rats exposed to rapeseed oil condensate by three intratracheal instillations of 0.1 or 1.5 mg/kg bw displayed a high frequency of cell transformation *in vitro*. Finally, Zhang *et al*. (1999) showed that exposure of female Kunming mice to cooking oil emissions condensates from rapeseed oil, soya bean oil and salad oil by subcutaneous injection (1.1–2.3 g/kg) caused an inhibition of the delayed hypersensitivity response and of the activity of natural killer cells in comparison with controls.

(ii) *In-vitro exposure of human cells*

Several studies investigated the effect of cooking oil emissions condensates on cultured human lymphocytes. Jin and Cu (1997) noted significant induction of unscheduled DNA synthesis in cultured human lymphocytes exposed to cooking oil emissions condensate (200ºC) from rapeseed oil and soya bean oil. Similarly, Shen *et al*. (1998) reported that fume condensates from heated rapeseed oil collected in Nanjing, China, induced unscheduled DNA synthesis in human peripheral blood lymphocytes with and without metabolic activation. Hou *et al*. (2005) reported that cooking oil emissions condensate significantly increased chromosomal aberrations but not micronucleus frequency in human peripheral blood lymphocytes.

^{32}P-Postlabelling was used to show dose-dependent induction of DNA adducts in human lung adenocarcinoma CL-3 cells exposed to extracts of cooking oil fumes from fish fried in soya bean oil. Subsequent liquid chromatography/mass spectrometry confirmed that the DNA adduct in CL-3 cells induced by exposure to cooking oil

emissions extract was benzo-[*a*]pyrene-7,8-diol-9,10-epoxide-N^2-deoxyguanosine (Yang *et al*., 2000). In addition, the comet assay showed induction of DNA damage (DNA strand breaks) in human lung adenocarcinoma CL-3 cells following exposures to 100 µg/mL cooking oil emissions condensate from fried fish (Lin *et al*., 2002). Dose-dependent induction of DNA damage, measured using the comet assay, was also observed in human lung carcinoma A549 cells treated with extracts of fumes from heated peanut oil (Wu & Yen, 2004), sunflower oil, soya bean oil and lard (Dung *et al*., 2006).

Dung *et al*. (2006) determined in the study above that *trans-trans*-2,4-decanedial (*t,t*-2,4-DDE), which is a by-product of lipid peroxidation and is one of the most abundant and potent mutagens identified in cooking oil fumes to date (Wu *et al*. 2001), was present in all three condensate samples, and induced a significant increase in the level of 8-OHdG adducts. It is also thought to induce intracellular formation of reactive oxygen species and has been shown to induce a dose-dependent increase in 8-OHdG in CL-3 cells (Cherng *et al*., 2002). Chang *et al*. (2005) also studied oxidative stress in human bronchial epithelial BEAS-2B cells, and confirmed that *t,t*-2,4-DDE induced a concentration-dependent increase in the production of reactive oxygen species and a decrease in the reduced glutathione/oxidized glutathione ratio (glutathione status). The data also suggest that *t,t*-2,4-DDE leads to cell proliferation, significant increases in unscheduled DNA synthesis (measured by bromodeoxyuridine incorporation), as well as induction of tumour necrosis factor-α and interleukin-1β gene expression and release of the corresponding cytokines in cultured BEAS-2B cells. Co-treatment of BEAS-2B cells with the antioxidant *N*-acetylcysteine prevented *t,t*-2,4-DDE-induced release of cytokines and concomitant cell proliferation.

(iii) *Other in-vitro systems*

Several studies have shown that exposure of Chinese hamster V79 cells to rapeseed oil cooking fumes induced a marked increase in the frequency of sister chromatid exchange (Zhu *et al*., 1990; Chen *et al*., 1992) and, moreover, the magnitude of the genotoxic effect was inversely related to the degree of hydrogenation of the cooking oil (Zhu *et al*., 1990). Qu *et al*. (1992) noted that exposure of V79 cells to an extract of cooking fumes from heated unrefined rapeseed oil and heated refined rapeseed oil induced a significant increase in sister chromatid exchange frequency; however, fume condensate from unrefined rapeseed oil supplemented with the antioxidant butylated hydroxyanisole (0.02%) failed to induce a concentration-dependent significant increase in sister chromatid exchange frequency. Additional analyses of fumes from hydrogenated rapeseed oil samples also failed to induce a significant increase in sister chromatid exchange frequency. Wu *et al*. (1999) noted a concentration-related increase in sister chromatid exchange frequency, both with and without exogenous metabolic activation, in Chinese hamster ovary (CHO-K_1) cells exposed to condensates of fumes from lard or soya bean oil. The same condensates have also been shown to induce DNA damage (SOS Chromotest) in *Escherichia coli* PQ37.

Pu *et al*. (2002) noted a time-dependent increase in DNA cross-links and single-strand breaks in rat type II lung cells exposed to cooking oil emissions condensates. A reduction in cytotoxicity, DNA cross-links and strand breaks following pretreatment with the antioxidant *N*-acetylcysteine suggested that cooking oil fumes induced oxidative stress in exposed cells. Similarly, Zhang *et al*. (2002) noted a concentration-related increase in DNA damage, as measured by the comet assay, in rat type II pneumocytes exposed to a condensate of cooking fumes (obtained from a kitchen ventilator) at concentrations up to 10 µg/mL. Yin *et al*. (1998) also noted a significant increase when these cells were exposed to cooking oil emissions condensate from vegetable oil heated to 270±5ºC.

Finally, three studies demonstrated that cooking oil fumes induced DNA damage in calf thymus DNA. Wu *et al*. (1992) demonstrated that exposure to rapeseed oil (heated to 280ºC)-fume condensate can induce adducts in naked calf thymus DNA without metabolic activation, and Yin *et al*. (1997) demonstrated that exposure to rapeseed and soya bean oil (heated to 270ºC)-fume condensates can induce cross-links in calf thymus DNA. Xi *et al*. (2003) demonstrated that exposure to cooking oil emissions condensates can induce 8-OHdG formation in calf thymus DNA.

Cooking oil emissions emissions were also investigated in several cell transformation assays. A dose-dependent increase in the frequency of morphological transformation was observed in BALB/c3T3 cells exposed to condensates of cooking fumes (Shen *et al*., 1998). Zhao *et al*. (2000) observed dose-dependent malignant transformation in KMB-17 diploid human embryo lung cells exposed to a condensate of cooking oil fumes. The transformed cells showed a variety of distinct features, including loss of density inhibition, loss of contact inhibition, growth at low serum concentration, agglutination at low concentrations of concanavalin A, aneuploidy and deviation from diploid status and loss of anchorage dependence (Zhao *et al*. 2002).

(iv) Salmonella *reverse mutation assay*

Studies have related mutagenic activity in *Salmonella* to a host of indoor activities, including cooking (e.g. Sexton *et al*., 1986; Teschke *et al*., 1989). A wide range of source-specific studies has confirmed the mutagenic activity in *Salmonella* of emissions from heated cooking oil (e.g. Qu *et al*., 1992; Nardini *et al*., 1994; Shields *et al*., 1995; Chiang *et al*., 1997, 1998; Wu *et al*., 2001) and highlighted that these sources are significant contributors to the mutagenic activity of indoor air. Moreover, several studies have noted a positive empirical relationship between the mutagenic activity of indoor air (in revertents/m^3) in *Salmonella* and the concentration of airborne PM (Mumford *et al*., 1987; Chiang *et al*., 1999). This relationship is not unexpected because combustion emissions are composed of PM, and several researchers (e.g. Maertens *et al*., 2004, 2008) have commented on the tendency for mutagens in combustion emissions, such as PAHs, to adsorb to particulate material and solid surfaces (e.g. upholstery, carpets). Chiang *et al*. (1999) noted particle concentration levels as high as 28 mg/m^3 in dwellings that were filled with cooking oil fumes.

Table 4.1 provides a summary of studies that have used the *Salmonella* assay to investigate the mutagenic activity (in revertants/m^3) of indoor air particulates from high-temperature frying. The data indicate that organic extracts of indoor air particulate material collected from areas without any obvious source of contamination have mutagenic potency values in *Salmonella* in the 1 and 10 TA98 revertants/m^3 range. Table 4.2 provides a summary of studies that investigated the mutagenic potency (in revertants/mg) in *Salmonella* of source-specific particulate emissions from high-temperature frying.

The mutagenic potency values reached several hundreds of TA98 revertants/m^3 and several thousands of TA98 revertants/mg of particle with or without exogenous metabolic activation (Sexton *et al*., 1986; Löfroth *et al*., 1991; Wu *et al*., 2001). It is interesting to note that two studies (Qu *et al*., 1992; Xu *et al*., 1995) described a relationship between the mutagenicity of cooking oil emissions condensates and heating temperature. Qu *et al*. (1992) noted that condensates of fumes from unrefined rapeseed oil did not elicit a significant response unless the oil was heated to 270ºC (TA98 with metabolic activation). Similarly, Xu *et al*. (1995) only detected a significant mutagenic response (TA98 with metabolic activation) when the rapeseed oil was heated to 230ºC or 280ºC.

Several studies have used bioassay-directed fractionation methods to identify mutagenic agents in condensates of cooking oil fumes. Wu *et al*. (2001) determined that the mutagenic activity in *Salmonella* TA98 of methanolic extracts from heated peanut oil fumes without metabolic activation is contained within a neutral fraction. Detailed chemical analyses of the neutral fraction resulted in the identification of four direct-acting alkenals: *t*,*t*-2,4-DDE, *trans*-*trans*-2,4-nonadienal, *trans*-2-decenal and *trans*-2-undecenal. The most potent agent, *t*,*t*-2,4-DDE, elicited 385 revertants/µg in TA100 and 18 revertants/µg in TA98 (without metabolic activation). Qu *et al*. (1992) hypothesized that the mutagenic agents in condensates of heated rapeseed oil are the oxidized products of unsaturated fatty acids such as linoleic and linolenic acid, and noted contrasting levels of mutagenic activity between unsaturated oil samples and highly hydrogenated samples. Unsaturated rapeseed oil samples with 10 and 12% linolenic and linoleic acid, respectively, elicited significant positive responses, whereas highly hydrogenated samples without either acid failed to elicit a positive response. Moreover, complete elimination of mutagen formation by the addition of 0.1% butylated hydroxyanisole supported this hypothesis. In addition, Shields *et al*. (1995) showed that mutagenic activity in TA98 (with metabolic activation) was induced when unsaturated fatty acids such as linoleic acid and linolenic acid were heated to 240ºC. Moreover, the mutagenic activity (TA98 with metabolic activation) of condensates from heated Chinese rapeseed oil (275–280ºC), heated peanut oil (260–265ºC) and heated soya bean oil (260–265ºC) was positively related to the content of linolenic acid. The presence of several other mutagens in the condensates of heated oils was also confirmed. These included 1,3-butadiene, benzene, acetaldehyde and acrolein.

Table 4.1. Mutagenicity in *Salmonella* of organic extracts of indoor air particulate matter from high-temperature frying (in revertants/m^3)

Source	Country	Particle concentration (μg/m^3)	Mutagenic potency in revertants/m^3		Reference
			Without metabolic activation	With metabolic activation	
TA98					
Olive oil	Italy	31600	329	108	Nardini *et al*. (1994)
Deep fry[a]	Canada	US	618	ND	Teschke *et al*. (1989)
Wok[a]	Canada	US	617	ND	Teschke *et al*. (1989)
Frying hamburger[a]	USA	US	~50	~220	Sexton *et al*. (1986)
TA100					
Frying hamburger[a]	USA	US	~960	~1180	Sexton *et al*. (1986)

ND, no data; US, unspecified
[a] Type of cooking oil not specified

Table 4.2. Mutagenicity in *Salmonella* of organic extracts of particulate emissions from high-temperature frying (in revertants/mg)

Source	Country	Particle concentration ($\mu g/m^3$)	Mutagenic potency (revertants/mg)		Reference
			Without metabolic activation	With metabolic activation	
TA98					
Rapeseed oil 230ºC	China	US	ND	[46.1]	Xu *et al.* (1995)
Rapeseed oil (unrefined) 270ºC	China	US	neg.	[62.2]	Qu *et al.* (1992)
Rapeseed oil (refined) 270ºC	China	US	neg.	[123.9]	Qu *et al.* (1992)
Rapeseed oil 275ºC	China	US	ND	[282.5]	Shields *et al.* (1995)
Rapeseed oil 280ºC	China	US	ND	[109.7]	Xu *et al.* (1995)
Sunflower oil 300ºC	China	25.1	[19.6]	[62.4]	Chiang *et al.* (1999)
Refined lard 300ºC	China	26.8	[12.5]	neg.	Chiang *et al.* (1999)
Vegetable oil 300ºC	China	28.3	[8.1]	[21]	Chiang *et al.* (1999)
Olive oil	Italy	31600	10	3	Nardini *et al.* (1994)
Lard	China	26.2	neg.	[180]	Chiang *et al.* (1997)
Lard 100°C	China	US	neg.	54	Chiang *et al.* (1998)
Lard 200°C	China	US	35	101	Chiang *et al.* (1998)
Lard 200°C	China	US	61	180	Chiang *et al.* (1998)
Lard 300°C	China	US	82	236	Chiang *et al.* (1998)
Lard 300°C	China	US	43	122	Chiang *et al.* (1998)
Soya bean oil	China	28.5	neg	[80]	Chiang *et al.* (1997)
Soya bean oil 200°C	China	US	neg.	82	Chiang *et al.* (1998)
Soya bean oil 260ºC	China	US	ND	[45.6]	Shields *et al.* (1995)
Soya bean oil 270ºC	China	US	neg.	[101]	Qu *et al.* (1992)

Table 4.2. (contd)

Source	Country	Particle concentration ($\mu g/m^3$)	Mutagenic potency (revertants/mg)		Reference
			Without metabolic activation	With metabolic activation	
TA98 (contd)					
Soya bean oil 300°C	China	US	neg.	61	Chiang *et al.* (1998)
Soya bean oil 300°C	China	US	42	112	Chiang *et al.* (1998)
Peanut oil	China	27.1	neg.	[40]	Chiang *et al.* (1997)
Peanut oil 300°C	China	US	neg.	66	Chiang *et al.* (1998)
Peanut oil	China	US	[12600]	[11600]	Wu *et al.* (2001)
Lean pork, minced[a]	Sweden	US	ND	7400	Löfroth *et al.* (1991)
Commercial pork, minced[a]	Sweden	US	ND	800	Löfroth *et al* (1991)
Pork chops	Sweden	US	ND	15	Löfroth *et al* (1991)
Baltic herring[a]	Sweden	US	ND	25	Löfroth *et al* (1991)
TA98NR					
Olive oil[b]	Italy	31600	10	ND	Nardini *et al.* (1994)
TA100					
Peanut oil	China	US	[21000]	[18030]	Wu *et al.* (2001)

ND, no data; neg., negative; US, unspecified

[a] type of cooking oil not specified

[b] spilled on a hot plate

4.3 Genetic susceptibility

See the monograph on Household use of solid fuels.

4.4 Mechanistic considerations

The mutagenicity of emissions from high-temperature frying may be due to PAHs and lipid peroxidation products, among other compounds. Unlike emissions from the combustion of wood and coal, for which extensive, positive genotoxicity data have been generated almost exclusively in humans *in vivo*, nearly all of the mutagenicity data for emissions from high-temperature frying have been generated in experimental animals and in cells *in vitro*. The large number of genotoxic end-points and largely positive results, especially in experimental animals, provide plausible evidence that a carcinogenic mechanism similar to that described for coal emissions also applies to emissions from high-temperature frying.

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5. Summary of Data Reported

5.1 Exposure data

A large proportion of the emissions generated during cooking is steam from the water contents of the food. However, during frying (with oil), fatty acid esters that make up edible oils and fat can decompose and produce volatile organic compounds, as well as semi-volatile compounds that can condense to form particles. A wide variety of organic compounds have been identified in cooking emissions, including alkanes, alkenes, alkanoic acids, carbonyls, polycyclic aromatic hydrocarbons and aromatic amines. The main volatile compounds generated during frying were aldehydes, alcohols, ketones, alkanes, phenols and acids. Of particular concern in relation to carcinogenicity are polycyclic aromatic hydrocarbons, heterocyclic amines and aldehydes. The contribution of commercial cooking operations to outdoor levels of polycyclic aromatic hydrocarbons can be substantial.

Cooking also increases the concentrations of fine and ultrafine particles.

The chemical composition of cooking emissions varies widely depending on the cooking oils used, the temperature, the kind of food cooked, and the method and style of cooking adopted.

5.2 Human carcinogenicity data

To examine the potential association between emissions from cooking oil and the risk for lung cancer, the Working Group considered studies to be more informative when cooking-related effects were separated from fuel-related effects and when the studies reported results on the exposure–response relationships between high-temperature frying (i.e. stir-frying, deep-frying and pan-frying) and lung cancer. Studies that only collected information on cooking habits (e.g. age at starting to cook, years of cooking), ventilation in the kitchen or frequency of eye irritation due to cooking or smokiness in the kitchen were considered to be less informative because they did not allow the effects of emissions from cooking oil to be distinguished from those of combustion products of cooking fuels.

On this basis, four case–control studies were considered to be the most informative. The study conducted in Hong Kong Special Administrative Region used a composite index that accounted for both the frequency and the duration of all three types of high-temperature frying; it found a significant threefold increased risk for lung cancer associated with moderate to high categories of exposure (>150 total dish–years) and an eightfold increased risk associated with the highest category (>200 total dish–years).

In the other three informative studies in Shanghai (two studies) and Gansu, China, the risk for lung cancer increased generally with increasing frequency of stir-frying, deep-frying and pan-frying and a nearly twofold increased risk was associated with the highest

frequency of high-temperature frying. In the study conducted in Gansu, however, the risk for lung cancer increased significantly with increasing frequency of stir-frying but not of deep-frying. However, potential confounding by solid cooking fuel could not be ruled out with reasonable confidence in these three studies. In the study from Hong Kong that compared risk (per 10 dish–years) for the three types of high-temperature frying, the magnitude of risk was highest for deep-frying, intermediate for pan-frying and lowest for stir-frying, but all were associated with a significantly elevated risk for lung cancer. In the studies in Shanghai and Gansu, the effects of the different types of frying were not mutually adjusted for and, because of the substantial differences in the frequency of stir-frying and deep-frying, a direct comparison of the risk estimates associated with an individual type of frying could not be made.

These four studies also provided information on the specific type of cooking oil. There was no significant difference in risk estimates for lung cancer with use of any particular type of cooking oil (peanut oil, corn oil or canola oil — a type of rapeseed oil) in the study in Hong Kong. In the three other studies, risk was higher for women who cooked with canola oil most frequently. Some increased risk was associated with cooking with linseed oil in the population-based case–control study conducted in Gansu and with cooking with soya bean oil in the study in Shanghai.

In summary, results from the four most informative studies demonstrate an exposure–response relationship between increased frequency of or cumulative exposure (frequency and duration) to high-temperature frying and increased risk for lung cancer. These four studies were conducted in different populations in Hong Kong, urban Shanghai (two studies) and rural Gansu where study characteristics differed, and where cooking practices and other co-factors may also have differed. However, confounding by cooking fuel could not be ruled out with reasonable confidence in the latter three studies. Furthermore, all epidemiological evidence was based on case–control studies and recall bias may have contributed to the positive findings in some of these studies.

5.3 Animal carcinogenicity data

Inhalation of high concentrations of emissions from high-temperature frying of unrefined rapeseed oil caused an increase in the incidence of lung carcinomas (mainly adenocarcinomas) in male and female mice in one study and female rats in another study.

5.4 Mechanistic and other relevant data

See also Section 5.4 in the monograph on household use of solid fuels.

The available information on the genotoxic and mutagenic activity of cooking oil fumes includes data from professional and home cooks that show the induction of 8-oxoguanine DNA glycosylase 1, which is a DNA repair enzyme that removes 8-hydroxydeoxyguanine. In experimental animals, cooking oil-fume condensates from rapeseed and soya bean oils induced micronuclei in the bone marrow of both mice and

rats, oxidative DNA damage, enhanced transformation of tracheal epithelia and accumulation of TP53 protein. Cooking oil-fume condensate also induced chromosomal aberrations in the diploid male germ cells of mice. In cultured human or animal cells, cooking oil fumes from a variety of oils induced DNA adducts, DNA damage (comet assay), oxidative damage, sister chromatid exchange, chromosomal aberrations, unscheduled DNA synthesis and DNA cross-links. Cooking oil fumes induced DNA damage in naked calf thymus DNA.

Extracts or condensates of emissions from cooking oil fumes are mutagenic in *Salmonella*. In strain TA98, in the presence or absence of a metabolic activation system, the mutagenic potency in terms of revertants per milligram of particle reached several thousands or in terms of revertants per cubic metre of air reached several hundreds.

Several studies showed that the mutagenicity of cooking fumes in *Salmonella* was positively correlated with heating temperature, the extent of unsaturation and the concentration of unsaturated fatty acids. Polycyclic aromatic hydrocarbons and lipid peroxidation products also contribute to the mutagenic activity of cooking oil fumes.

6. Evaluation and Rationale

There is *limited evidence* in humans for the carcinogenicity of emissions from high-temperature frying.

There is *sufficient evidence* in experimental animals for the carcinogenicity of emissions from high-temperature unrefined rapeseed oil.

Overall evaluation

Emissions from high-temperature frying are *probably carcinogenic to humans (Group 2A)*.

Rationale

Among the studies of cancer in humans, four were considered most informative because they allowed the effects of cooking-oil emissions to be distinguished from those of the fuels used for heating the stove. These studies, in four different populations, consistently showed an increased risk for lung cancer and showed an exposure–response relationship between increased frequency or duration of high-temperature frying and increased risk for lung cancer. Confounding by the fuel used to heat the stove could be ruled out with reasonable confidence in only one of these studies.

These epidemiological results are supported by the evidence from studies in experimental animals. Although positive results in experimental animals were observed only for unrefined rapeseed oil heated to high temperatures, positive results for mutagenicity were observed in virtually every category of in-vivo test. These mutagenicity data would have been enough to support an evaluation of Group 2A if the evidence of carcinogenicity in experimental animals had been less than *sufficient* or the evidence of carcinogenicity in humans had been less than *limited*. The mechanistic data also show that lipid peroxidation is an important mechanism that leads to carcinogenesis by these mixtures, although there may also be a contribution from the mechanisms by which polycyclic aromatic hydrocarbons induce cancer (see Volume 92).

The evaluation was made for 'emissions from high-temperature frying'. This wording was determined after considering several aspects of the available data.

The available studies involved frying at high temperatures. Emissions from low-temperature cooking methods can be considerably different from those studied. Data indicate that cooking oil has little mutagenic potential when heated below 100°C and high mutagenic potential when heated above 230°C.

No differences were apparent between stir-frying, deep-frying and pan-frying when these methods were investigated separately. Other high-temperature cooking methods

(e.g. baking) were not included because the Working Group reasoned that their emissions could be considerably different from those of frying.

The epidemiological data are not detailed enough to distinguish between different cooking oils and fats and experimental animal data were available for unrefined rapeseed oil only, although data are available that indicate a higher mutagenic potency for unsaturated fats.

The epidemiological data do not permit the risk to be attributed to a specific chemical compound or to the cooking oil alone. Some risk could be attributable to the food being cooked, to emissions from the heated stove or cooking vessel itself or to the fuel used to heat the stove. Nevertheless, it might be reasonable to attribute some risk to cooking oils, because in-vivo and in-vitro data indicate that emissions from some oils heated to high temperatures are mutagenic.