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EXPOSURE TO COOKING FUMES DURING THE PAN FRYING OF BEEFSTEAK UNDER DOMESTIC AND OCCUPATIONAL CONDITIONS

Thesis for the degree of philosophiae doctor

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Norwegian University of Science and Technology Faculty of Social Sciences and Technology Management Department of Industrial Economics and Technology Management



NTNU

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SUMMARY

Possible connections have been shown between exposure to cooking fumes and adverse health effects, such as lung cancer, rhinitis and reduced lung function. The aim of this study was to monitor the personal exposure to some of the harmful components of cooking fumes during the frying of beefsteak under experimental conditions resembling both domestic and occupational cooking situations, and during real-life occupational cooking. In addition, the aim was to investigate the occurrence of the same components during a variety of frying procedures, i.e. with different frying fats, cooking appliances and kitchen exhaust hoods. The spreading of cooking fumes in the kitchen and neighbouring rooms in private homes was also explored.

In a custom-built laboratory kitchen, standardized experiments were performed under conditions similar to the frying of beefsteak in real-life domestic and occupational conditions. Personal sampling was conducted of total particles, high molecular weight aldehydes, polycyclic aromatic hydrocarbons (PAHs) and ultrafine particles. In addition, stationary measurements of ultrafine and submicrometer particles were performed in the kitchen and in the neighbouring room. Tests were conducted under the use of different frying fats (margarine, soya bean oil, rapeseed oil, olive oil), different types (4 types) and settings (2 levels) of kitchen exhaust hoods and different cooking appliances (gas, electric). In addition, personal sampling of total particles, high molecular weight aldehydes and polycyclic aromatic hydrocarbons was conducted in three restaurant kitchens.

The studies confirmed the presence of carcinogenic components (higher aldehydes and PAHs) in fumes from Norwegian cooking styles which were collected in the breathing zone of the cook. It was also shown that the fumes spread to neighbouring rooms, and that leaving the kitchen extraction hood on for 30 minutes after the end of frying reduced the spreading significantly. The use of different types and settings of kitchen extraction hoods resulted in different exposure conditions. The choice of frying fat also seemed to affect the concentrations of particulate matter and chemical components produced during the frying of beefsteak, but the studies were not comprehensive enough to recommend preferable fats. In addition, the results indicated that frying on a gas stove caused a higher exposure to some of

the hazardous components in cooking fumes than frying on an electric stove.

Furthermore, the results suggested that cooking under experimental conditions may give a correct reflection of occupational exposure conditions if numerous repetitions of a very specific and standardized cooking method are performed. The level of total particles and the level of PAHs and higher aldehydes did not covariate, suggesting that the measured levels of particulate matter alone are unsuitable as indicators of the actual health risk inflicted upon persons exposed to cooking fumes.

It seems vital to reduce the exposure to cooking fumes as far as possible. In the area of extraction hoods, more research is needed in order to develop optimal systems. Additional studies are required in order to be able to identify relations between exposure to and adverse effects of cooking fumes.

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LIST OF ABBREVIATIONS

AA	Aromatic amine
BaA	Benzo[a]anthracene,
BaP	Benzo[a]pyrene
BbFA	Benzo[b]fluoranthene
BghiP	Benzo[ghi]perylene
BkFA	Benzo[k]fluoranthene
DBahA	Dibenzo[a,h]anthracene
EC	Elemental carbon
ELCR	Excess lifetime cancer risk
FT-IR	Fourier Transform Infrared Spectroscopy
GC-MS	Gas Chromatography-Mass Spectrometry
HCA	Heterocyclic amine
HPLC	High-performance liquid chromatography
IARC	International Agency for Research on Cancer
IP	Indeno[1,2,3-cd]pyrene
nitroPAH	nitro-polycyclic aromatic hydrocarbons
OC	Organic carbon
OEL	Occupational exposure limit
PAH	Polycyclic aromatic hydrocarbon
PMx	Particulate matter, aerodynamic diameter < x
PMx-y	Particulate matter, aerodynamic diameter x-y
SD	Standard deviation
SMPS	Scanning Mobility Particle Sizer
TLV	Threshold limit value
tt-DDE	trans,trans-2,4-decadienal
UFP	Ultrafine particles
US EPA	United States Environmental Protection Agency

LIST OF PAPERS

PAPER I: Sub-micrometer particles: Their level and how they spread after pan frying of beefsteak Sjaastad AK, Svendsen K, Jørgensen RB.

Indoor and Built Environment 2008;17:230-236.

PAPER II: Exposure to mutagenic aldehydes and particulate matter during panfrying of beefsteak with margarine, rapeseed oil, olive oil or soybean oil. Sjaastad AK, Svendsen K. Annals of Occupational Hygiene 2008;52(8):739-745

PAPER III: Different types and settings of kitchen canopy hoods and particulate exposure conditions during pan-frying of beefsteak. Sjaastad AK, Svendsen K. Indoor and Built Environment 2010;19:267–274

PAPER IV: Exposure to polycyclic aromatic hydrocarbons (PAHs), mutagenic aldehydes and particulate matter during pan frying of beefsteak.

Sjaastad AK, Jørgensen RB, Svendsen K.

Occupational and Environmental Medicine 2010;67(4):228-232

PAPER V: Exposure to polycyclic aromatic hydrocarbons (PAHs), mutagenic aldehydes, and particulate matter in Norwegian a la carte restaurants.

Sjaastad AK, Svendsen K.

Annals of Occupational Hygiene 2009;53(7):723-729

INTRODUCTION

Aims of the study

Several studies have shown possible connections between exposure to cooking fumes and adverse health effects, such as lung cancer, rhinitis, reduced lung function and other airway symptoms. Based on this, the aims of this study were:

- to investigate the personal exposure to cooking fumes from the pan frying of beefsteak under experimental conditions, aiming to confirm the presence of some of the components known to give adverse health effects during frying under domestic and occupational conditions in Norway
- to monitor the personal exposure to the same harmful components during real-life occupational cooking
- to compare the effect of using different kitchen exhaust hoods, frying fats and cooking appliances on the exposure to cooking fumes under domestic conditions
- to investigate the spreading of cooking fumes in the kitchen and neighbouring rooms in private homes

Characteristics of cooking fumes

"Cooking fumes" is the term commonly used to describe the visible emissions generated during cooking. During cooking, submicron-sized solid particles (particulate matter) are created by the cooling of hot vapour which is formed when cooking oil is heated above its boiling point. In addition, cooking, especially frying and grilling/charbroiling, generates aerosol oil droplets, combustion products, organic gaseous pollutants and steam from the water content of the food being cooked (IARC 2010).

A large proportion of the vapours generated during cooking is steam from the water content of the food or from the water used to cook the food. However, several chemical processes take place during the high temperature treatment of food. The most important of these processes are the degradation of sugars, pyrolysis of proteins and amino acids and the degradation of fats (Svendsen et al. 2002). Several compounds may be generated in these processes, e.g. fatty acids (n-alkanoic and n-alkenoic acids), aldehydes, such as formaldehyde, acrolein,

acetaldehyde or higher aldehydes (alkanes, alkenes, dienaldehydes), ketones, alkanols, esters, furans, lactones, nitriles, polycyclic aromatic hydrocarbons (PAHs), nitro-polycyclic aromatic hydrocarbons (nitroPAHs), heterocyclic amines (HCAs) and aromatic amines (AAs) (Vainiotalo and Matveinen, 1993, Felton 1995, Thiébaud et al. 1995, Chiang et al. 1999a,b, Wu et al. 2001, Schauer et al. 1999, Zhu et al. 2001, Schauer et al. 2002, He et al. 2004a, To et al. 2007, Zhao et al. 2007a,b). It has been reported that the main volatile compounds generated during the frying of beef are aldehydes, alcohols, ketones, alkanes, phenols and acids (Felton 1995).

Cooking is also one of the most important sources of indoor particulate matter, including coarse (PM10, aerodynamic diameter $<10 \ \mu$ m), fine (PM2.5, aerodynamic diameter $<2.5 \ \mu$ m) and ultrafine particles (UFP, aerodynamic diameter $<0.1 \ \mu$ m) (Kamens et al. 1991, Abt et al. 2000a,b, Long et al. 2000, Dennekamp et al. 2001, Evans et al. 2008, Wallace and Ott 2010). UFP may be formed during the combustion process of frying and emitted directly to the atmosphere as UFP, but hot vapours in the cooking fumes may also nucleate to form UFP when being cooled (Sioutas et al. 2005, Lai and Ho 2008). UFP represent a major portion of particle emissions from typical cooking activities. In addition, larger aerosols will arise from the physical stirring of foods which induces spattering of frying fats and liquids (Long et al. 2000). Most of the particles emitted from the grilling or frying of meat contain organic compounds (Kleeman et al. 1999, McDonald et al. 2003, Zhu and Wang 2003), and the mass concentrations and chemical characteristics of the emissions depend strongly on the cooking method and food ingredients (Rogge et al. 1991, Lee et al. 2001, See et al. 2006, Zhao et al. 2007a,b, See and Balasubramanian 2008).

The particulate matter in cooking fumes

Several studies have measured particle concentrations in the PM10 fraction during various cooking procedures (Abt et al. 2000a, Kelly 2001, Lee et al. 2001, Fortmann 2002, Lee et al. 2007, Pan et al. 2008a), but investigations on size distributions of the particulate matter which are produced have shown that a larger part of this size fraction is comprised of particles smaller than 2.5 μ m (Kelly 2001, Zhao et al. 2007a,b). In fact, studies on the particle size distribution of cooking aerosols have shown that a large percentage (~80-90%) of the measured particles is UFP (Li et al. 1993, He et al. 2004c, Hussein et al. 2006, See and

Balasubramanian 2006a).

Particle number size distributions resulting from cooking have been reported to be unimodal with the number median diameter ranging from 0.022 to 0.94 μ m (He et al 2004b). Other studies have reported a number mode of 0.064 μ m during the cooking of a dinner involving deep-fried and baked tortillas (Wallace 2006), and a number mode at about 0.09 μ m during the miscellaneous cooking of dinners (Ogulei et al. 2006). An investigation measuring the submicrometer size distribution of aerosol emissions from meat cooking operations, showed a broad, single modal mass distribution, with a peak around 0.2 μ m in particle diameter (Hildemann et al. 1991). Some studies have reported a bimodal distribution (Abt et al. 2000b, Long et al. 2000, Lai and Chen 2007), based on the production of both ultrafine and coarse particles from cooking processes. Overall, it seems that different cooking methods applied to miscellaneous foods during the use of different cooking appliances generate peak number concentrations at various particle diameters (Wallace 2006). It has been concluded that indoor fine particle events tend to be brief, intermittent and highly variable (Long et al. 2000), and thus, difficult to characterize.

Studies on particle emissions in domestic kitchens have shown that PM2.5 increases by 30 times during frying. However, these high concentrations are transient in nature (Fortmann et al. 2002, He et al. 2004c, Evans et al. 2008). It has been reported that cooking contributed to approximately 50 % of the PM2.5 in 38 homes in North Carolina, USA, based on receptor modelling of daily filter samples collected over four seasons (Zhao et al. 2006). In four homes in Boston, USA, it was found that cooking contributed to about 25 % of indoor PM2.5 (Abt et al 2000b).

The major species of the fine particle fraction in cooking fumes is carbonaceous particles (See and Balasubramanian 2008). Carbonaceous particles are comprised of a complex mixture of substances, which are broadly classified into two main fractions: elemental carbon (EC) and organic carbon (OC) (Seinfeld and Pankow 2003). In general, the major chemical constituents of fine particles from various sources are sulfate, nitrate, ammonium, OC and EC, as well as a variety of trace metals formed in the combustion processes (Sioutas et al. 2005). In cooking fumes, organic carbon is the major constituent (Kleeman et al. 1999, Schauer et al. 1999,

Long et al. 2000, See and Balasubramanian 2008, Kleeman et al. 2008), probably constituting more than 80 % of the carbonaceous particles in cooking fumes (See and Balasubramanian 2008).

Inorganic and organic ions, metals and PAHs are also found to be considerable components of the fine particulate matter in cooking fumes. The amounts of the different components, however, seem to be dependent on the food cooked and the applied cooking methods. Generally, it seems that oil-based cooking methods release more organic pollutants (OC, PAHs, organic ions) and metals (mainly copper, iron and zinc), while water-based cooking methods account for more water-soluble ions (inorganic ions such as fluoride, chlorine and sulfate) (See and Balasubramanian 2008). In addition, the combination of compounds in the fine particulate matter of cooking fumes are different in Western and Chinese style fast food cooking (Zhao et al. 2007b) Cholesterol, myristic acid, palmitic acid, stearic acid, oleic acid, nonanal and lactones are compounds reported as possible tracers of emissions from Westernstyle meat cooking (Rogge et al. 1991), since they usually are not abundant in emissions from other particle sources. Tetradecanoic acid, hexadecanoic acid, octadecanoic acid, oleic acid, levoglucosan, mannosan, galactosan, nonanal and lactones are candidates of organic tracers used to describe and distinguish emissions from Chinese cooking (Zhao et al. 2007a).

Oil-based cooking methods also seem to generate more UFP than water-based cooking methods, according to a study where ultrafine particles < 50 nm accounted for 69-90 % of all particles during oil-based cooking as compared to 55 % during steaming and 62 % during boiling (See and Balasubramanian 2006a). This can be attributed to the high-temperature heating of cooking oil (fatty acids) which presumably generates more particles than the boiling of water. In addition, water with a boiling point of 100°C is much more volatile than, for example, corn oil which has a boiling point of 245°C. Therefore under the high-temperature cooking, water droplets are more likely to exist in the gaseous phase than in the particulate phase, while less volatile oil droplets tend to remain as particles. In addition, during the steaming and boiling of water, the humidity in the kitchen is probably higher than during oil-based cooking. This may cause water vapour to condense on UFP to form larger particles (See and Balasubramanian 2006a), or the hydroscopic growth of particles (Wallace and Howard-Reed 2002).

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The chemical components in cooking fumes

Over 90 organic compounds have been identified in emissions from Chinese cooking (He et al. 2004b), and also in American meat grilling (Schauer et al. 1999), but the mixtures of compounds in fumes from the different cooking styles are very dissimilar (He et al. 2004b). The emissions of different chemical compounds seem to be significantly impacted by the cooking ingredients (including frying fats) and the applied cooking methods (Thiébaud et al. 1995, Gertz 2000, He et al. 2004b, Zhao et al. 2007a,b, See and Balasubramanian 2008). A study in China showed that the cooking method affected the concentration of benzo[a]pyrene (BaP) in kitchen air (Du et al. 1996). In the same kitchens, the level of BaP was elevated in indoor air from the baseline value of 0.41 μ g/100m³ to 0.65 μ g/100m³ when meat was boiled, and to 2.64 μ g/100m³ when meat was stir-fried. In addition, the ingredients and methods affect not only the quantity, but also the presence of different compounds. For example, monosaccharide anhydrides and β -sitosterol are detected in the emissions from Chinese cooking due to the high use of vegetables, but not in the emissions from meat cooking. In addition, nonanedioic acid is an abundant dicarboxylic acid found in Chinese cooking; however, hexanedioic acid is a dominant one in meat cooking (Rogge et al. 1991, He et al. 2004b, Zhao et al. 2007a).

The duration of cooking may affect the quantity and composition of the cooking fumes, e.g. it has been shown that the fine particle mass emission rate per unit of grilled meat was approximately proportional to the cooking time on the grill (Schauer et al. 1999). However, in a study where four different cooking oils were heated, it was shown that the generation rate of volatile compounds was almost constant regarding the time for all oils (Katragadda et al. 2010). The same study indicated that the temperature had an impact on the total quantity of volatile emissions. During the process of heating the oils, the same compounds were present in the emissions, but the quantities increased significantly when the temperature was risen. This was also shown in a study on emissions of low molecular weight aldehydes from the heating of frying oils (Fullana et al. 2004a,b), and in a study on airborne mutagens produced by the frying of beef, bacon and a soya-based food (Thiébaud et al. 1995). Here the generation rates of emissions were found to be dependent on temperature, showing significant increases with rising temperatures. However, it has also been shown that some mutagenic cooking fume components are produced at all frying temperatures, whereas others may require higher

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temperatures (Felton 1995).

The International Agency for Research on Cancer (IARC) have stated that PAHs, HCAs and aldehydes are of particular concern in relation to carcinogenicity caused by cooking fumes (IARC 2010). In several previous studies, PAHs, HCAs and AAs have been identified as the main mutagenic compounds in cooking fumes (Kiel 1986, Vainiotalo and Matveinen 1993, Li et al. 1994, Thiébaud et al. 1995, Felton 1995, Chiang et al. 1997, Yang et al. 1998, Chiang et al. 1999a,b). In recent years, attention has been shifted towards other compounds as the most mutagenic constituents in cooking fumes. For instance, it has been indicated that trans,trans-2,4-decadienal (tt-DDE), a high molecular weight aldehyde found in cooking oil fumes may make a more important contribution than PAHs (BaP) to the cell survival and proliferation of lung cancer cells (Hung et al. 2007).

Based on the results of recent studies, we chose to focus the present study on investigating the presence of and exposure to some of the components in cooking fumes, i.e. PAHs and high molecular weight aldehydes, in addition to the particulate matter. Low molecular weight aldehydes were not registered because we had identified them in previous studies, and found that the exposure levels of acrolein, formaldehyde and acetaldehyde for cooks in Norwegian restaurants were well below Norwegian TLVs (Svendsen et al. 2002).

PAHs

PAHs are formed mainly during carbonisation and the incomplete combustion of organic materials (Zhu and Wang 2003). PAHs can exist in gaseous or particulate phase, depending on many environmental factors such as temperature and relative humidity (Lai and Ho 2008). Naphthalene (with two rings) is normally present almost entirely in the gaseous phase. Compounds with three or four rings (e.g anthracene, pyrene) tend to be present in both gaseous and particulate phases, and compounds with five or six rings (e.g. BaP) exist almost entirely in particulate form (Rappaport et al. 2004). PAHs are complex mixtures of hundreds of chemicals. Among the PAH's identified in fumes from the heating of different cooking oils are BaP, dibenzo[a,h]anthracene (DBahA), benzo[a]anthracene (BaA) and benzo[b]fluoranthene (BbFA). For example, these have been detected in fumes from corn oil, vegetable oil, and safflower oil (Chiang et al. 1999a). In addition, BaP and BaA, as well as naphthalene, have been registered in fumes from soya bean oil, rapeseed oil and lard (Zhu and

Wang 2003). Naphthalene is the most volatile member of the PAH class of pollutants. It is ubiquitously discharged into the human environment by incomplete combustion processes from industrial, domestic and natural sources. The most important pathway by which the general public is exposed to naphthalene is by inhalation due to the release of this substance from combustion fuels, moth repellents and cigarette smoke (Preuss et al. 2003).

High molecular weight aldehydes

Aldehydes constitute a group of relatively reactive organic compounds. They occur as natural flavouring constituents in a wide variety of foods and food components, often in relatively small, but occasionally in very large, concentrations. They are also widely used as food additives (Feron et al. 1991). They have a high electrophilicity, and can easily react with the macromolecules in organisms (Esterbauer et al. 1986). Aldehydes are commonly formed during the degradation of edible oils and will create unpleasant flavours (Dung et al. 2006). When heating edible oils, aldehyde production will also occur from the thermal and oxidative decomposition of the oils (Wang et al. 2010). At high temperatures, peroxidation of the polyunsaturated fatty acids in cooking oils will form high molecular weight aldehyde species, i.e. trans-2-alkenals, trans,trans-alka-2,4-dienals and n-alkanals (Gertz 2000). Among these aldehydes, tt-DDE, a polar dienaldehyde, has been identified as the most abundant and major mutagenic and cytotoxic component in oil fumes (Chiang et al. 1997, Wu et al. 2001, Zhu et al. 2001, Fullana et al. 2004a,b, Yang et al. 2007).

tt-DDE has been detected in cooking fumes resulting from heating oils as, for example, peanut oil (Wu et al. 2001), rapeseed oil, soya bean oil (Zhu et al., 2001), olive oils, canola oil (Fullana et al. 2004a,b), soya bean oil, sunflower oil, lard (Dung et al. 2006) safflower oil and coconut oil (Katragadda et al. 2010). A linear correlation between the contents of free fatty acids and mutagenicity as well as the contents of tt-DDE of cooking oils has been indicated (Yen and Wu 2003). tt-DDE and some 2-alkenals (2-decenal and 2-undecenal) have also been identified in cooking fumes from barbecuing and grilling of meat without the addition of cooking oils (Schauer et al. 1999, Yang et al. 2007), indicating that the oils are not the only source of high molecular weight aldehydes.

Adverse health effects caused by exposure to cooking fumes

Several epidemiological studies indicate that direct exposure to cooking oil fumes generated from the Chinese cooking style (open-wok cooking) has strong correlations to the development of rhinitis (Ng and Tan 1994), reduced lung function, airway symptoms (Ng et al. 1993) and lung cancer, the latter found in non-smoking Chinese women (Ko et al. 1997, Wu et al. 1999, Zhong et al. 1999, Metayer et al. 2002, Yu et al. 2006, Subramanian and Govindan 2007, Wang et al. 2009). In India, a significantly elevated risk (adjusted for smoking) for lung cancer has been found in cooks (Notani et al. 1993). Epidemiological studies in Europe have also reported an increased risk of respiratory tract diseases and cancer in some chefs and bakers (Coggon et al. 1986a, Lund & Borgan 1987, Foppa and Minder 1992). In addition, an association between lung cancer and occupation has been found in white, male cooks in Massachusetts, USA (Dubrow and Wegman 1984). In Italy, a significant effect on urinary mutagenicity has been shown in healthy non-smoking subjects caused by exposure to cooking fumes under domestic conditions (Pavanello et al. 2007). Norwegian studies have shown a higher frequency of respiratory symptoms in kitchen employees compared to members in a control group (Svendsen et al. 2003). An increase in the number of alveolar macrophages, which are biomarkers of pulmonary irritation, was found in kitchen workers at fast food stores and grill restaurants (Sivertsen et al. 2002), and minor short term spirometric effects, mainly affecting forced expiratory time (FET) from short term exposure to cooking fumes was found in an experimental setting (Svedahl et al. 2009). Thus, increased respiratory disease and cancer may not only be limited to people in Asian countries but is probably related to exposure to fumes generated during cooking (Young et al. 2010).

Associations between exposure to cooking fumes and cervical intraepithelial neoplasms, known to be precancerous lesions, have also been shown in both occupational chefs and women cooking in private homes (Wu et al. 2004). In addition, an increased risk of developing bladder cancer has been reported in cooks and workers processing animal and vegetable fats (Schoenberg et al. 1984, Coggon et al. 1986b, Teschke et al. 1989). In Sweden, a possible relation between air pollutants in kitchen environments and ischemic heart disease has been indicated (Sjögren et al. 2009). Cooking oil fumes are genotoxic, as shown by their ability to induce mutations in bacteria and base oxidation, single strand DNA breaks, sister chromatid exchange and DNA cross-links, as well as cell malignant transformation in mammalian cells (Chiang et al. 1997, Chiang et al. 1999a,b, Wu and Yen 2004, Wu et al. 2008). Emissions from high temperature frying have been classified as 'probably carcinogenic to humans (Group 2A)' by IARC (IARC 2010). In addition, cooking oil fumes have been found to induce oxidative stress *in vitro* (Pu et al. 2002). Cooking oil fumes contain many mutagens and carcinogens, including HCAs, PAHs, aromatic amines and nitro-polycyclic aromatic hydrocarbons (Vainiotalo and Matveinen 1993, Li et al. 1994, Thiébaud et al. 1995, Wu et al. 1998, Chiang et al. 1999a,b). According to recent studies, the two major components identified as being associated with mutagenicity and carcinogenicity in cooking oil fumes are PAHs (Zhu and Wang 2003, Wu et al. 2004) and dienaldehydes (Wu et al. 2001, Chang et al. 2005, Dung et al. 2006).

In the following section, the health effects of particulate matter, PAHs and high molecular weight aldehydes will be outlined.

Particulate matter

Particle emissions from typical cooking activities are comprised of particles in different size fractions, but ultrafine particles constitute a major portion (Long et al. 2000). UFP have been considered to be a factor contributing to a series of health problems, including premature death, aggravated asthma, chronic bronchitis and modulations in the immune system (Oberdörster et al. 2005a,b, Sioutas et al. 2005, Chang 2010). Toxic effects have been documented in pulmonary, cardiac, reproductive, renal, cutaneous and cellular levels (Ostiguy et al. 2008). UFP are characterized by a high number concentration, low mass concentration and a big surface area (Oberdörster et al. 1995). This gives them a higher deposition rate in the peripheral lung and a greater ability to cross the pulmonary epithelium than larger particles (Oberdörster 2001). The large number concentration of UFP decreases the ability of the alveolar macrophage to eliminate foreign particles (Donaldson et al. 2001, Biswas and Wu 2005). This causes an increase in exposure time between particles and lung epithelial cells and also a strong size-selective difference in particle immobilisation (Semmler-Behnke et al. 2007). Transport of UFP to the brain via the olfactory nerve has also been demonstrated (Oberdörster et al. 2004).

The small size of particles also contributes to the transcytosis across epithelial and endothelial cells into the blood and lymph circulation, reaching potentially sensitive target sites such as bone marrow, lymph nodes, spleen and heart (Oberdörster et al 2005a,b). It has also been shown that UFP localize in the mitochondria in cells where they induce major structural damage (Li et al. 2003). The greater surface area of UFPs implies that they can carry large amounts of adsorbed pollutants, oxidant gases, organic compounds and transition metals (Oberdörster 2001). The surface properties of UFPs generated at different sources and during the ageing of the particles are dynamically different in toxicity. The primary particles emitted from the sources interact through chemical reactions in the atmosphere with oxygen, nitrogen dioxide, ozone, sulfur dioxide and organics producing secondary particles of diverse reactivity and characteristics. The toxicity and adverse health effects caused by UFP are thus heterogenous, depending on the source and mixed exposures of primary and secondary UFP (Gwinn and Vallyathan 2006).

The physical properties of UFP combined with their ability to carry adsorbed compounds give them an enhanced capability of producing reactive oxygen species (Brown et al. 2001, Li et al. 2003, Dick et al. 2003, Sioutas et al. 2005) and causing oxidative stress (Li et al. 2003, Beck-Speier et al. 2005, Sioutas et al 2005), leading to allergic and inflammatory reactions to a greater degree than coarser particles (Oberdörster 2001, Brown et al. 2001, Alessandrini et al. 2006). Size, composition and solubility seem to be strongly linked with health effects, and it has been shown that even ultrafine particles composed of a low-toxicity material such as polystyrene have proinflammatory activity (Brown et al. 2001).

Calculations have estimated that as much as 71 % of the total particle counts sampled in a Chinese food stall were deposited within the body during cooking hours due to their relatively smaller sizes, compared to only 58 % of the total particle counts observed during non-cooking hours (See and Balasubramanian 2006b). Others have shown that the deposition efficiency of carbon UFPs in human subjects was >60 % and this increased with exercise and in subjects with asthma (Chalupa et al. 2004).

The fine and coarse particle fractions, which include sub-micrometer and ultrafine particles, may cause the same negative effect as the ultrafine fraction, although not necessarily to the

same extent (Oberdörster 2001, Li et al. 2003, See and Balasubramanian 2008).

PAHs

Data from animal studies indicate that several PAHs may induce a number of adverse effects, such as immunotoxicity, genotoxicity, carcinogenicity, reproductive toxicity and may possibly also influence the development of atherosclerosis. However, carcinogenicity is the most well documented effect of several PAHs (WHO 2000). The carcinogenic potency tends to be greatest in a few particle-bound four to six ring PAHs, notably BaP, DBahA, BaA, BbFA, benzo[k]fluoranthene (BkFA), indeno[1,2,3-cd]pyrene (IP) and benzo[ghi]perylene (BghiP) (Rappaport et al. 2004). The biologic properties of the majority of these PAHs are as yet unknown. BaP is the PAH most widely studied, and the abundance of information on the toxicity and occurrence of PAHs is related to this compound. Using BaP as an indicator of general PAH mixtures from the emissions of coke ovens and similar combustion processes in urban air, a lifetime risk of respiratory cancer of 8.7 x 10⁻⁵ per ng/m³ BaP was calculated (WHO 2000).

Among the PAHs identified in cooking fumes, IARC have classified BaP, DBahA and BaA as probable human carcinogens (group 2A) (IARC 1998). Naphthalene and BbFA are considered to be possible human carcinogens (group 2B) (IARC 2002, IARC 1998).

High molecular weight aldehydes

The higher aldehydes present in cooking fumes have primarily been connected to the mutagenic and carcinogenic effects of cooking fume exposure. Being highly electrophilic, aldehydes can easily diffuse into cells and react directly with the macromolecules in organisms, causing damage to, for example, DNA molecules (Esterbauer et al. 1986). In a study on mutagenic compounds in peanut oil heated to about 100°C, the following compounds were identified as the ones with the strongest mutagenicity in the Ames test (in descending order): trans,trans-2,4-decadienal, trans,trans-2,4-nonadienal, trans-2-decenal and trans-2-undecenal (Wu et al. 2001). tt-DDE is considered to be the major mutagenic and cytotoxic compound in oil fumes (Zhu et al. 2001, Wang et al. 2010), and thus it is the high molecular weight aldehyde that is most widely studied with regard to occurrence and health effects.

tt-DDE has been shown to generate reactive oxygen species (ROS) and induce oxidative injury in human leukemia cells and A-549 cells (Nappez et al. 1996, Wu & Yen 2004). tt-DDE-induced oxidative stress and genotoxicity has also been shown in human lung carcinoma cells (Wu and Yen 2004, Chang et al. 2005) and human bronchiolar epithelial cells (Young et al. 2010). In male mice who were intratracheally instilled with tt-DDE, several known risk factors associated with increased lung adenocarcinoma development were detected, suggesting that tt-DDE may pose a risk in lung carcinogenesis (Wang et al. 2010).

Of the other mutagenic aldehydes identified (Wu et al. 2001), trans-2-decenal is also shown to cause significant oxidative damage in human A-549 cells (Wu and Yen 2003). In addition, it has been shown that extracts of oil fumes from soya bean oil, sunflower oil and lard cause cytotoxicity and oxidative DNA-damage in human A-549 cells. These fume extracts contained not only trans,trans-2,4-decadienal, but also trans-2-decenal, trans,trans-2,4-nonadienal and trans-2-undecenal (Dung et al. 2006).

Studies on exposure to cooking fumes in commercial and domestic kitchens

When referring to the various studies that have been made on the production of and exposure to cooking fumes in domestic and commercial kitchens, the main emphasis will be on results that resemble the ones obtained in the papers constituting this thesis. Whenever the method used in a study is referred to as "sampling", it means that pollutants were collected on a medium (most often a filter) connected to a pump, before concentrations were determined by analysis. If the method is called "measuring", it means that the results were obtained by use of some type of monitor where real-time concentrations can be read on a display as well as from a memory log.

Particulate matter

Real-life conditions in domestic kitchens

He et al. (2004c) performed stationary measurements (2 m from the stove) of PM2.5 and submicrometer particle concentrations during various cooking activities in 15 homes in Brisbane, Australia. Two of the houses had gas stoves, the others had electric stoves. Data on kitchen exhaust hoods in the homes are not given. Median peak values of 745 μ g/m³ (PM2.5)

and 154 000 submicrometer particles/cm³ were reported during four episodes of frying. They also registered a range of peak submicrometer number concentrations between 16 000 and 180 000 particles/cm³ when performing an experimental cooking test under normal (windows and doors open) and poor (windows and doors closed) ventilation conditions.

In a study on particle sizes and emission rates during indoor activities in a house in Prague, a maximum concentration of 180 000 particles/cm³ (0.015-0.530 μ m in diameter) was measured in the kitchen and in the living room during various cooking activities on an electric stove, with more than 90% of that being UFP. The particle number concentration in the living room was also affected significantly during cooking, especially when the living room door was open (Hussein et al. 2006).

A study conducted in Boston, USA, was performed to characterize indoor particle sources in four non-smoking households, some equipped with gas stoves and some with electric stoves (Abt et al. 2000a). Continuous particle size and volume concentration data were measured during one or two periods of 6 days, with monitoring equipment placed in a single indoor location adjacent to the areas of high activity, i.e. kitchen and living room. Mean peak volume concentrations of PM0.02-0.5 during frying (4 episodes) and barbecuing (3 episodes) were 28.86 μ m³/cm³ and 57.39 μ m³/cm³ respectively. Combustion particles may have a density close to 1 g/cm³. The use of this specific gravity of 1.0 for combustion particles (Wallace et al. 2004, Wallace 2006) implies that a particle volume of 1 $\mu m^3/m^3$ corresponds to a mass of 1 μ g/m³ (Olson and Burke 2006). Thus, the mean peak mass concentrations of PM0.02-0.5 registered by Abt et al. (2000a) during frying and barbecuing can be estimated to $28.86 \ \mu g/m^3$ and 57.39 μ g/m³ respectively. Corresponding mean peak PM0.7-10 concentrations were 19.45 and 12.76 μ g/m³ respectively. In a related study, PM2.5 concentration and size distribution particulate data were obtained from nine non-smoking homes in the Boston area, some with gas stoves and some with electric stoves. The measurement equipment was placed in a central room in the main living area (living room, dining room) (Long et al. 2000). Mean peak mass concentrations of PM2.5 during frying (20 episodes) and barbecuing (2 episodes) were 40.5 and 14.8 µg/m³ respectively. Estimated mean peak mass concentrations of PM0.02-0.1 during frying and barbecuing were 3.6 and 2.3 μ g/m³ respectively.

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PM2.5 concentrations were measured using personal monitors for 7 consecutive days in each of four consecutive seasons on 37 persons living in North Carolina, USA (Olson and Burke 2006). For all cooking events (n=411), an average PM2.5 concentration of 188 μ g/m³ was registered, whereas for frying (105 events), the average PM2.5 concentration was 341 μ g/m³. The results also showed average PM2.5 concentrations of 377 μ g/m³ and 189 μ g/m³ when frying on electric (75 events) and gas stoves (30 events) respectively.

In a town house in Virginia, USA, stationary measurements of PM2.5 and UFP (0.01-0.1 μ m) was conducted semi-continuously in the kitchen throughout 24 selected cooking episodes, mainly including frying of various food types (Wallace et al. 2004). During cooking of dinner on a gas burner, a mean PM2.5 concentration of 11.8 μ g/m³ and a mean number concentration of 26 920 ultrafine particles/cm³ were measured. After three years of measuring ultrafine and accumulation mode particles in the same town house, an average number concentration of 2673 PM0.1-1.0 particles/cm³ and 30 456 UFP/cm³ during 225 events (average duration: 83 min) of cooking of miscellaneous dinners was reported (Wallace 2006). During five events of smoky cooking oil (average duration: 128 min), the average number concentration of PM0.1-1.0 and UFP were 10 308 and 33 509 particles/cm³ respectively.

As a part of a large-scale study of personal indoor and outdoor particulate exposures in residents in Seattle, USA, data collected from personal measurements during 195 cooking events gave an estimated average PM2.5 concentration due to cooking of $5.5 \ \mu g/m^3$ (Allen et al. 2004). Data on the use of gas or electric stoves were not given.

Diurnal variation of indoor submicrometer (0.007-0.808 μ m) particle number and PM2.5 concentrations was investigated in 15 houses in Brisbane, Australia (Morawska et al. 2003). Measurements of the particulate mass were performed continuously for more than 48 h in the kitchen of each house, with the instruments placed stationary, 2 m from the stove. Data on gas or electric stoves were not given. All houses were naturally ventilated. Maximum concentrations of submicrometer particles and PM2.5 registered during cooking were 286 000 particles/cm³ and 535.4 μ g/m³ respectively.

Controlled experiments in domestic kitchens

In a study on particle size distribution during cooking on a gas stove in a domestic kitchen in Taiwan, it was found that concentrations of submicrometer particles increased from 15 000 particles/cm³ to 150 000 particles/cm³ during cooking. Maximum values of 260 000 submicrometer particles/cm³ during the frying of chicken and 180 000 submicrometer particles/cm³ during the scrambling of eggs were measured. Each frying/scrambling episode lasted 15 minutes, and was repeated three times. Measurements were performed stationary, 3 m from the stove. 80-85 % of the submicrometer particles measured during frying/scrambling were UFP (Li et al. 1993).

A series of cooking tests with both a gas and an electric appliance was conducted in a test chamber (17.3 m³) (Kelly 2001). The chamber was supplied with filtered outside air and had an air exhaust placed at the opposite wall, but no exhaust hood above the cooking appliances. Various cooking activities with both the gas and the electric appliances were conducted at a medium heat setting, using a single burner or heating element. Particulate emissions were sampled over the time periods in which cooking took place, including some time after cooking was finished. Both oven broiling of steak and pan frying of hamburgers were registered as high emitting processes in terms of particulate emissions. During pan frying of hamburgers on gas appliances (about 5 hours) and electric appliances (about 3 hours), average PM2.5 and PM10 concentrations in the chamber were about 500 and 1250, 655 and 1547 $\mu g/m^3$ respectively. On average, PM2.5 comprised 88 % of the PM10 mass, and nearly all particle emissions were in the size ranges below about 0.5 μm in diameter. The electric appliance produced a size distribution that was shifted toward larger particles, relative to that produced by the gas appliance. However, the main observation was that particle size distributions with both appliances were dominated by submicron particles.

Based on the experiences from the chamber tests, a series of cooking emission tests were performed under realistic conditions in an instrumented research house in Chicago, USA (Kelly 2001). The house was a one-story single family house with three bedrooms and a full basement. On the main floor, the kitchen was open towards the living room. The kitchen was not equipped with a kitchen exhaust hood. The house was ventilated between the repetitions. Particulate emissions were sampled over the time periods in which cooking took place, including only active cooking. Three repetitions of various cooking activities were conducted on both a gas and an electric stove, according to standardized procedures. During cooking, the highest indoor particle levels usually occurred at the appliance (in the breathing zone of the cook), and the lowest in the living room or bedroom. Frying hamburgers on the gas appliance (192 minutes) and the electric appliance (216 minutes) produced an average PM2.5 and PM10 concentration at the appliance of about 115 and 230, 118 and 328 μ g/m³ respectively. In the living room, the corresponding PM2.5 concentrations were about 35 and 100 μ g/m³.

In a study performed in a domestic test kitchen in California, USA, sampling of UFP and PM2.5 was performed with stationary samplers placed in the breathing zone of the cook (Fortmann et al. 2002). The samplers used were able to detect particles down to the size of 0.03 µm in diameter. UFP concentrations were sampled throughout the cooking period (about 1 h when frying minced beef, 2 h when frying bacon), while sampling of PM2.5 and PM10 was conducted throughout the cooking period and 1 hour of post-cooking sampling (about 1-2 h when frying minced beef, 3 h when frying bacon). The test kitchen was equipped with an exhaust hood over the range which ventilated to the outdoors, but this was not in use at all times. During the pan frying of minced beef on a gas stove under standard conditions with and without operating the exhaust hood (about 1 hour), the average UFP concentrations in the kitchen were 1556 and 10 520 particles/cm³ respectively. The corresponding average PM2.5 concentrations were 144 and 102 μ g/m³, and PM10 concentrations were 207 and 144 μ g/m³ respectively. Concurrent average PM2.5 concentrations in the living room, separated from the kitchen by a wall with an open doorway, were 8.2 and 7.8 μ g/m³ respectively. During frying of bacon on a gas stove and an electric stove without operating the exhaust hood, UFP concentrations in the kitchen were 38 769 and 14 197 particles/cm³ respectively. Corresponding average PM2.5 concentrations in the kitchen were 482 and 207 μ g/m³, and PM10 concentrations were 261 and 289 µg/m³ respectively. Average PM2.5 concentrations in the living room were 142 and 276 μ g/m³. The particles emitted during cooking were primarily in the size fractions of less than 1 um.

In a laboratory kitchen with no mechanical ventilation and the windows closed, various cooking experiments were performed on a gas and an electric stove while conducting stationary measurements of UFP (Dennekamp et al. 2001). The frying of bacon on the gas stove (6 repetitions à 7 min) caused the highest peak concentration of numbers of UFP, 590 000 UFP/cm³. This was significantly higher than the peak concentration of UFP

registered when frying bacon on the electric stove (5 repetitions à 7 min), which was 159 000 UFP/cm³. It was also found that the peak concentration of UFP generated by frying fatty foods was significantly higher than that generated by frying vegetables on gas.

During about 45 minutes of domestic-style pan frying of hamburgers (minced beef) on an electric stove in a laboratory chamber (32 m³), the maximum concentration of ultrafine particles was approximately 150 000 particles/cm³ (Afshari et al. 2005). Measurements were made in the middle of the room, 1.2 m above the floor. The chamber was not equipped with a kitchen exhaust hood. The maximum concentration of particles $\geq 1.0 \ \mu m$ was 25 particles/cm³. Particles $\geq 1.0 \ \mu m$ and ultrafine particles reached their maximum concentration simultaneously.

The mass concentration of PM2.5 during steaming, boiling, stir-frying, pan frying or deepfrying of one pack of tofu in corn oil was registered in the kitchen of a mid-level apartment in Singapore (See and Balasubramanian 2008). The cooking experiment was repeated five times for each cooking method. All cooking was performed on a gas burner, without use of an exhaust extractor and with doors and windows closed. Stationary sampling in the breathing zone of the cook revealed an average PM2.5 concentration at 130 μ g/m³ during pan frying. Following the same procedure in the same kitchen, the size distribution of ultrafine particles emitted from the different cooking methods was also investigated (See and Balasubramanian 2006a). The mean number concentration of particles < 0.1 μ m measured during pan frying was 100 000 particles/cm³.

Concentrations and production rates of UFP and PM2.5 from the frying of different foods (bacon, eggs, pancakes, rice and vegetables) on an electric stove in a single home in Canada were measured stationary in the breathing zone of the cook (Evans et al. 2008). All experiments were repeated three times. During frying, the kitchen exhaust hood was run on the highest setting. At the end of frying the bacon, concentrations of 22 000 particles/cm³ and $38 \mu g/m^3$ were registered for UFP and PM2.5 respectively.

Real-life conditions in commercial kitchens

Stationary measurements of PM2.5 and PM10 were performed in the main dining areas (1.5 m above the floor) of four restaurants with different cooking styles (Korean barbecue, Chinese hot pot, Chinese dim sum and Western canteen) in Hong Kong (Lee et al. 2001). The measurements were conducted throughout two of the busiest hours in the restaurants. The Western canteen did not have a cooking stove in the dining area, while all the others had a gas stove without a canopy hood situated in these areas. Mean PM2.5 and PM10 levels were 1167 and 1442 (Korean barbecue), 81.1 and 105.3 (Chinese hot pot), 28.7 and 33.9 (Chinese dim sum) and 21.8 and 38.8 (Western canteen) μ g/m³.

A study which was conducted using stationary sampling in the breathing zone of the cook during normal work hours (12 hours) in restaurants with Chinese, Malay and Indian cooking, detected mean PM2.5 levels of 201.8, 245.3 and 186.9 μ g/m³ respectively (See et al. 2006). All restaurants had gas stoves and no mechanical ventilation. The Malay and Chinese style of cooking involved a great deal of deep-frying and stir-frying respectively, mainly of vegetables. The Indian style is more based on simmering the food.

Geometric mean levels of particles (PM2.5 and PM10) of 60 and 80 μ g/m³ were reported in a Chinese study based on stationary measurements near the breathing zone of the workers in 23 different Chinese restaurant kitchens (Pan et al. 2008a). Stir-frying, deep-frying and grilling of the food were methods frequently used. Sampling was performed throughout two working days, à 12 h. Data on the stoves used and the ventilation conditions are not given.

A Korean study of indoor air pollutants in five different restaurants (two Korean barbecue, Japanese, Chinese and Italian) measured average PM1.0, PM2.5 and PM10 levels in the breathing zone of the cook in a Korean barbecue restaurant during frying of meat on a gas stove of 18.2, 32.0 and 71.1 μ g/m³ respectively (Lee et al. 2007). Before cooking started, the corresponding levels were 4.5, 7.2 and 12.6 μ g/m³. During the grilling of meat in a Korean barbecue restaurant using charcoal as the heat source, average PM1.0, PM2.5 and PM10 levels of 63.7, 124.1 and 169.4 μ g/m³ were registered. Before cooking started, the corresponding levels were 9.1, 13.7 and 20.7 μ g/m³. Measurements were performed during a busy period in the restaurants. Data on the use of a kitchen exhaust hood are not given. After

completing the cooking of the meat and eating the food, the registered levels of particulate matter were still approximately twice as high as before the cooking started. The levels registered in the Korean barbecue restaurant were higher than in the other restaurant types.

The average number and mass concentration of PM2.5 sampled stationary in the breathing zone of the cook during cooking hours (07.30-19.30) in a Chinese food stall in Singapore with four gas stoves and natural ventilation were 770 000 particles/cm³ and 312.4 μ g/m³ respectively (See and Balasubramanian 2006b). During non-cooking hours, the corresponding concentrations were 9 100 particles/cm³ and 26.7 μ g/m³. The smallest particles in the size range of 0.008-0.029 μ m constituted the largest particle number concentration, and particles in the range of 0.160-0.626 μ m dominated the mass concentrations.

In a food court in Boston, USA, PM2.5 and UFP concentrations were measured with instruments placed in the centre of the seating area (Levy et al. 2002). Measurements were performed on weekdays for approximately 2 h in the morning and 2 h in the afternoon, throughout a period of about 2 months. The food court comprised numerous grills. Data on the use of range exhaust hoods and gas or electric stoves are not given. The mean concentrations of PM2.5 and UFP were 200 μ g/m³ and 140 000 particles/cm³ respectively.

Controlled experiments in commercial kitchens

Typical Chinese style and Western style standard recipes were prepared in a model kitchen with a gas stove and an electric griddle as well as an exhaust hood (Yeung and To 2008). The kitchen was set up to resemble commercial conditions. Stationary measurements of UFP were conducted throughout 5 minutes of frying on each temperature level. During pan frying of steak in Western style cooking with the griddle temperature set to 210°C and 240°C, the average number concentrations of ultrafine aerosols were 306 000 and 288 000 particles/cm³ respectively. The investigations also showed that the typical Chinese style cooking process of frying vermicelli with beef on the gas stove generated significantly more submicrometer and ultrafine aerosols than Western style pan frying processes (frying of steak, chicken fillets and pork chops) on the electric stove.

Total particles

Personal samplings of fat aerosol and total particle concentrations have been performed in all our investigations on cooking fumes in order to be able to compare the different studies internally.

In a study on exposure to fat aerosols and low molecular weight aldehydes in Norwegian restaurant kitchens, personal sampling of total particles was performed during 1.5-2.5 hours of a work shift for three consecutive days in 19 different restaurants (hotel kitchens, hamburger chains, à la carte restaurants and small local restaurants). Electric stoves were the dominating type of appliances in the kitchens. Glass fibre filters were exposed to polluted air in the breathing zone of the cooks and analysed for fat aerosols using Fourier Transform Infrared Spectroscopy (FT-IR) (Svendsen et al. 2002). Miscellaneous foods were prepared, but the frying of meat and deep-frying of potatoes were performed in all kitchens. All restaurants were equipped with extraction hoods over the cooking ranges. Mean concentrations of fat aerosols were in the range of 0.1-1.9 mg/m³. The arithmetic mean mass concentration of fat aerosols for all restaurants was 0.62 mg/m³, and the peak concentration sampled was 6.6 mg/m³. The peak levels were registered in a small, local restaurant, mainly preparing fried meat and deep-fried potatoes.

PAHs

Real-life conditions in domestic kitchens

A study on sources and patterns of PAH pollution in kitchen air in China, showed that naphthalene was the most predominant PAH in domestic kitchens (Zhu and Wang, 2003). The levels of naphthalene sampled stationary in the breathing zone of the cook in homes of non-smoking families were in the range of $0.42-2.7 \ \mu g/m^3$. The levels of total PAHs were in the range of $3.6-7.7 \ \mu g/m^3$. Conventional Chinese cooking on gas stoves was performed in the kitchens during the sampling, which lasted for 12 hours (8.00-20.00) on two consecutive days. Three of the four kitchens had a kitchen exhaust hood which was turned on during the cooking periods. The dominance of naphthalene was partly explained as a result of oil fumes from cooking and indoor smoking, but mainly as the evaporation from mothballs containing large quantities of naphthalene which were stored in wardrobes in the bedroom. It was presumed that the bedroom air was easily transported to the kitchen by air movement.

Measurements of particulate PAH release during cooking in the living room in a residential home in the USA indicated that frying/sautéing on a gas stove yielded a maximum peak concentration of total PAH of 670 ng/m³ and an average peak release during 9 episodes (29-225 minutes) of frying/sautéing of 315 ng/m³ (Dubowsky et al. 1999). Data on the use of a kitchen exhaust hood are not given.

Controlled experiments in domestic kitchens

Naphthalene levels in the range of 0.0-646 ng/m³ and BaP levels in the range of 0.0-20.6 ng/m³ were sampled in the kitchen during stir frying (about 3 h/test) of different foods in vegetable oils on a gas stove under experimental conditions in a domestic kitchen (described previously) in California, USA (Fortmann et al. 2002). The kitchen exhaust hood was not operated during these tests.

During the frying of chicken curry on a gas stove in a model kitchen, a maximum level of total PAH of about 50 ng/m³ was measured stationary at nose height near the stove. Average levels during the whole cooking period (about 20 minutes) were about 5 ng/m³ (Flückiger et al. 2000). During cooking, the kitchen exhaust hood was run at the minimum level.

The chemical constituents of PM2.5 produced during steaming, boiling, stir-frying, pan frying or deep-frying of one pack of tofu were investigated in the kitchen of a mid-level apartment in Singapore (described previously) (See and Balasubramanian 2008). One of the constituents analysed in this study was PAHs. All cooking was performed on a gas burner without the use of an exhaust extractor and with doors and windows closed. Stationary sampling in the breathing zone of the cook revealed average PAH concentrations at 25.0 ng/m³ during pan frying. The corresponding concentration of naphthalene was 0.37 ng/m³.

A series of cooking tests with both a gas and an electric appliance was conducted in a test chamber (described previously) (Kelly 2001). The concentration of 17 PAH compounds and 7 particulate-phase PAHs designated as B2 carcinogens (probable human carcinogens) by the US Environmental Protection Agency (US EPA) were sampled during the periods in which cooking was performed. However, the PAH levels registered were not substantially elevated above those found in the inlet air into the chamber, and the contribution of the cooking to the PAH levels could not be distinguished. Based on the experiences from the chamber tests, some changes to the sampling methods were made before a series of cooking emission tests were performed under realistic conditions in the research house (described previously) in Chicago, USA (Kelly 2001). Indoor PAH concentrations were determined by sampling in the living room of the research house during the periods in which cooking was performed. Indoor total PAH levels were often elevated relative to outdoor levels in cooking tests, but not always to a large extent. On average, the various cooking activities elevated indoor total PAH by roughly a factor of two. Total PAH concentrations registered during the frying of hamburgers on gas (192 min) and electric appliances (216 min) were 294 and 425 ng/m³ respectively. Corresponding concentrations of particulate-phase PAHs designated as B2 carcinogens (probable human carcinogens) by the US EPA were 0.929 and 3.59 ng/m³.

Real-life conditions in commercial kitchens

In a Finnish study describing possible exposure from cooking fumes, several PAH's were measured by stationary sampling in the active working area, as close to the breathing zone of the kitchen workers as possible (Vainiotalo and Matveinen, 1993). Sampling of PAHs was performed at five different workplaces where meat was fried and frying temperatures were high (250-300°C). The duration of sampling was in the range of 0.5-3 hours. Naphthalene (1.6-25.6 μ g/m³) and low levels (0.02-2.3 μ g/m³) of fluorene, phenanthrene, anthracene, pyrene, benzo(a)fluorine, chrysene, BaP and BghiP were detected in some samples. Data on the use of gas or electric stoves and kitchen exhaust hoods are not given.

A study which was conducted using stationary sampling in the breathing zone of the cook during normal work hours in restaurants with Chinese, Malay and Indian cooking (described previously) detected average particulate naphthalene levels of 1.9, 2.8 and 3.9 ng/m³ respectively. Corresponding average levels of particulate BaP and acenaphthylene were 5.6, 16.0, 0.9 and 2.4, 5.6, 2.7 ng/m³ respectively (See et al. 2006).

Stationary samplers were placed in the active work area (50 cm above the oil surface) in a Chinese restaurant and two Chinese fast food shops mainly preparing deep-fried foods (vegetables and dough). Sampling lasted for 2-4 hours. Data on the use of kitchen exhaust hoods and gas or electric stoves are not given. BaP and DBahA concentrations were registered in the range of 4.9-41.8 and 30.3-338 ng/m³, respectively (Li et al. 1994).

Air samples were taken stationary throughout two working shifts in the kitchen of various types of Chinese restaurants using coal gas as cooking fuel (Cui et al. 1995). Data on the use of kitchen exhaust hoods and gas or electric stoves are not given. PAHs were present in the samples, including BaP which was found in both particulates and vapour. Higher concentrations of BaP were found in vapour and on small airborne particles than on larger particulate matter. At a frying work station in a Roast Duck Shop, the concentrations of pyrene and BaP in vapour and on airborne particles were 244 and 56.6 ng/m³, and 14.0 and 18.7 ng/m³ respectively.

Stationary samples of particulate PAHs were collected throughout two whole working days near the breathing zone of the workers of 19 Chinese restaurants. The main cooking methods in the restaurants were stir-frying, deep-frying and some grilling. Data on the use of kitchen exhaust hoods and gas or electric stoves are not given. Pyrene and BaP were quantified at median levels of 3.3 and 5.9 ng/m³ respectively. The summed median level of several PAHs was 23.9 ng/m³ (Pan et al. 2008b). In a parallel study, particulate pyrene and BaP were registered at median levels of 2.9 and 6.2 ng/m³, based on stationary samples collected near the breathing zone of the workers in 23 different restaurant kitchens (described previously). The summed median level of several particulate PAHs was 24.8 ng/m³ (Pan et al. 2008a).

PAH levels have also been registered with stationary samplers in the breathing zone of the kitchen workers in other Chinese commercial kitchens with gas stoves (Zhu and Wang 2003). Kitchen exhaust hoods were operated during cooking in all restaurants. Naphthalene and BaP levels were registered in the range of $1.5-3.0 \ \mu g/m^3$ and $0.15-0.44 \ \mu g/m^3$ respectively. The mean concentration of total PAHs was $17 \ \mu g/m^3$. Three- and four-ring PAHs were predominant. When heated to the same temperatures (180, 200 and 230°C), it was found that the concentration of PAHs were highest in fumes from lard, followed by fumes from soya bean oil and rapeseed oil.

In a food court in Boston, USA, particulate PAH concentrations were measured in the centre of the seating area (Levy et al. 2002). Measurements were performed on weekdays for

approximately 2 h in the morning and 2 h in the afternoon throughout a period of about 2 months (as described previously). The mean PAH concentration registered was 9 ng/m^3 .

High molecular weight aldehydes

Some studies have investigated the occurrence of aldehydes with lower molecular weights, such as formaldehyde, acrolein and acetaldehyde (Vainiotalo and Matveinen 1993, Lin and Liou 2000, Svendsen et al. 2002, Fortmann et al. 2002) by stationary or personal sampling in the breathing zone of the cook, but these are not comparable to the investigations presented in this thesis.

In two parallel studies, emissions of high molecular weight aldehydes from deep-frying of extra virgin oil, olive oil and canola oil were investigated at two temperatures, 180 and 240°C, for 15 and 7 hours respectively. In one of the studies, the oils were heated in a closed experimental system, where the fumes were collected in Tedlar bags and analysed by gas chromatography-mass spectrometry (GC-MS) (Fullana et al. 2004a). In the other study, the fumes were heated in the same way, adsorbed onto tenax and analysed by GC-MS (Fullana et al. 2004b). Seven alkanals, eight 2-alkenals and 2,4-heptadienal were found in the fumes of all three cooking oils when collected in tedlar bags. When adsorbed onto tenax, six alkanals, seven 2-alkenals and 3 alkadienals (including 2,4-decadienal) were found in the fumes from all three oils. The generation rates of the aldehydes were found to be dependent on temperature, showing significant increase with increased temperatures. The results suggested that frying in any type of olive oil effectively decreased the generation of volatile aldehydes in the exhaust, compared to frying in canola oil.

Dung et al. (2006) studied levels of mutagenic aldehydes in cooking oil fumes collected on a filter paper connected to a vacuum pump, placed in the breathing zone of the cook during the heating of three different cooking oils under experimental conditions for 7.5 minutes. The mean contents of t-2-decenal, t,t-DDE, and t-2-undecenal were reported to be 5.8, 66.4 and 18.0 μ g/g in fumes from soya bean oil, 9.8, 35.9 and 9.98 μ g/g in fumes from sunflower oil, 13.8, 40.3 and 42.8 μ g/g in fumes from lard respectively.

The emission of volatile organic compounds, including aldehydes, formed during the heating of coconut, safflower, canola and extra virgin olive oils were studied at different temperatures: 180, 210, 240 and 270°C after 6 h (Katragadda et al. 2010). In the experimental system used, the fumes were collected in Tedlar bags and analysed by GC/MS. Emission rates (mg/h and l_{oil}) were obtained for all oils and temperatures. For example, the emission rates of 2.4-decadienal were highest when heating extra virgin olive oil to temperatures over 180°C. The lowest rates of 2.4-decadienal were registered when heating coconut oil.

In a study on the content of tt-DDE in the exhaust air from sixteen commercial restaurants in Taiwan which were categorized as barbecue (4), Chinese (8) and Western (4), the barbecue restaurants gave the highest emission factors based on the numbers of customers served (Yang et al. 2007), indicating that tt-DDE emission was affected by cooking methods.

Strategies to prevent or reduce exposure to cooking fumes

Several studies have shown that reducing the exposure to cooking fumes by the use of kitchen fume extractors may reduce the risk of developing cancer (Ko et al. 1997, Chiang et al. 1998, Chiang et al. 1999a, Wu et al. 2004, Chen et al. 2007). In a study on PAH exposure in Chinese cooks it was shown that the urinary level of a biomarker of oxidative stress (8-OHdG) was increased in exposed cooks working without an exhaust hood in the kitchen compared to exposed cooks working under similar conditions, but with an exhaust hood (Ke et al 2009). It has also been shown that the effect of the fume extractor may depend upon its location in the kitchen, i.e. the distance between the extraction hood and the cooking oil surface (Chiang et al. 1998), or the ventilation rate setting (Flückiger et al. 2000).

In Norway, most private households and commercial kitchens have some kind of exhaust hoods for the removal of cooking fumes. Most common in the private kitchen is a canopy or range hood fitted above the stove (figure 1). The hoods are frequently in use during cooking, especially during frying. However, little is known on how effective these extraction systems are in the removal of cooking fumes. In many restaurants, the exhaust hood is of a canopy style, often about 1 m above the cooking surface (figure 1). With this style of hood, the breathing zone of a cook will include the area directly between the point where volatile products of cooking are generated and where they are being exhausted. In other restaurants, the maximum height of the hood above the surface is about 0.65 m, and the air is drawn through a slot positioned toward the back of the hood, i.e. away from the breathing zone of the cook.



Figure 1: Kitchen exhaust hood (canopy/range hood) in a domestic kitchen (to the left) and in a commercial kitchen (to the right)

For comparison to capture velocity recommendations, the velocities near the cooking surface where potential contaminants are generated were examined (Teschke et al. 1989). The distance of 1 m between the cooking surface and the canopy implied that air velocity often could not be detected near these surfaces, and was in all cases less than or equal to 0.25 m/s. The other hood design showed a moderately improved performance, giving velocities of 0.15-0.38 m/s. The hood designs in all cases had to rely almost exclusively on convection for contaminants to be drawn into the hoods. The movement of the cooks could be expected to counteract the draw of the hoods and allow airborne products of the cooking process to be taken out of the direct path from the grill to the exhaust.

As illustrated by Teschke et al. (1989), canopy or range hoods in restaurant kitchens cannot achieve optimal environmental control because the distance between the updraft of the local exhaust and the stove is too great, and cooking movements break up the exhaust current. A hood's ability to collect fumes depends on the size of its opening, its exhaust volume, the relative location of the fume source and exhaust hood, and the degree of external interference with the airflow. To counteract this, the air curtain exhausting ventilation system has been designed (Lin and Lee 2010). The system is based on the existing household kitchen hood,

functioning as a suction hood, with the air curtains creating a push-pull effect, and is comprised of a local exhaust device mounted on a wall with a hood facing the stove (figure 2). On three sides of the stove (left, right and front), air curtains are installed, blowing air towards the hood and leading the cooking fumes into the exhaust. This design is supposed to obtain a maximum reduction in the concentrations of, for example, particulate matter and PAHs.

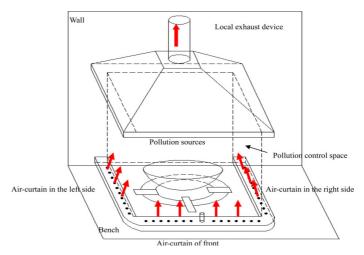


Figure 2: The air curtain exhausting ventilation system (Lin and Lee 2010)

Another design is the one where the exhaust fan is near the cooking surface, like the table edge fume extractor. This makes the extractor able to collect the cooking fumes from the cooking pot immediately before they are inhaled (Chiang et al. 1998).

To reduce the exposure to fumes causing adverse health effects, actions have also been taken to alter some of the ingredients frequently used in cooking. During the processing of peanut oil, peanut kernels are roasted at about 200°C for 45 min and pressed to obtain the oil. The oil is then kept in room temperature for 7-10 days before filtering the precipitate. For the preparation of degummed peanut oil, the oil is mixed with deionized water (5,15 or 25 g), stirred (500 rpm, 20 min at 40°C, 60°C or 80°C respectively) and cooled to room temperature before centrifuging (10 000 rpm, 1h at 20°C). It is then dried under reduced pressure (60°C, 76 mm Hg vacuum, 4h) and the precipitate and the degummed oil are separated. Studies have

indicated that a degumming treatment of peanut oil consequently gave a lower free fatty acid content, a higher smoke point and the production of less fumes when heating the oil to its smoke point. Moreover, compared to untreated peanut oil, the mutagenicity of oil fumes from degummed peanut kernels was reduced in the Ames test. The contents of trans,trans-2,4-decadienal, trans,trans-2,4-nonadienal, trans-2-decenal and trans-2-undecenal in fumes from degummed peanut oil were drastically decreased, especially the tt-DDE (Yen and Wu 2003).

When evaluating the preventive effect of three natural antioxidants (Y-tocopherol, lecithin and catechin) for the reduction of mutagenicity and amounts of PAH and nitroPAHs of fumes from cooking oils, it was found that the concentration of BaP were significantly reduced by adding catechin to cooking oils before heating (Wu et al. 1998). BaA, DBahA and two nitroPAHs were not detected when the concentration of catechin was 500 ppm in all the three cooking oil fumes that were tested. It has also been reported that lowering the cooking temperatures decreased the amount of mutagenic volatile emissions from heated rapeseed, soya bean, peanut, coconut, safflower, canola and extra virgin olive oils (Shields et al. 1995, To et al. 2007, Katragadda et al. 2010). In addition, the reheating of oil is not recommended as used oils will contain a higher free-fatty acid content, which will result in higher emissions of volatiles at lower temperatures (Katragadda et al. 2010).

MATERIAL AND METHODS

In the laboratory kitchen (papers I-IV)

The location

A large part of the present study was performed in a laboratory kitchen specially built for this purpose. The size of the kitchen was 19 m^2 (56.1 m³), and it was equipped with some basic kitchen furniture (cupboards, drawers, workbenches), an electric stove and a gas stove. The kitchen was prepared for installing exhaust hoods both centrally on one of the walls in the room and in the middle of the room (a so-called island hood). The island hood was included in the study because this is a trend in modern kitchens in Norway today. Table 1 shows a description of the different extraction hoods used in the study. All hoods had three different capacity levels. All installations in the laboratory kitchen were made flexible, making it easy to change the parameters during different experimental parts of the study. The basic ventilation in the kitchen room was 119 m³/h of air supply, and 112 m³/h outlet (except the kitchen hood ventilation). In paper I, measurements were also made in the neighbouring room which was connected to the laboratory kitchen by a small passage (4.5 m²). The neighbouring room separate ventilation, such as a kitchen exhaust hood, in the neighbouring room.

Hood	Model	Location of the hood	Given maximum air flow rate (m ³ /h)	Measured maximum air flow rate (m ³ /h)
A	Standard canopy hood, older model, exhausting outside the building	Installed under a cupboard, hanging above the stove	300	275
В	Modern canopy hood, design model, exhausting outside the building	On the wall, above the stove	630	500
С	Modern canopy hood, design model, exhausting outside the building	In the middle of the room, above the stove (cooking island)	630	589
D	Hood B with a charcoal filter, recycling the kitchen air	On the wall, above the stove	630	500

Table 1. Description of the kitchen extraction hoods used in the laboratory kitchen.

The frying of beefsteak

In papers I, II and III, all experiments were performed during the frying of beefsteak following a standardized procedure representing real-life conditions, that is, conditions similar to a common Norwegian home with regard to the location, ventilation conditions and the frying procedure. In paper IV, the measurements were performed according to standardized procedures resembling real-life conditions during the frying of beefsteak in a Western European restaurant. All procedures were standardized in order to give comparable results. The frying of beefsteak was chosen because this is a type of food that is frequently fried in both private households and restaurants in Norway. Beefsteak from the shoulder of bovine ox and the different frying fats that were used were purchased from a local grocery store.

The frying procedure

The pan was heated on the hotplate (210 mm diameter) on the top of the electric stove (papers I, II, III, IV) or on a burner on the gas stove (paper IV) using the maximum effect setting (2100 W on the electric stove) until the surface temperature in the middle of the pan reached 100°C. At 100°C, 20 g of margarine (papers I, II, III, IV) or 20 ml of cooking oil (papers II, IV) was added to the pan. After heating the margarine/oil for about 30-40 seconds, two pieces of beefsteak were added, each of them about 150 g (papers I, II, III) or 200 g (paper IV) with a thickness of 1.5-3.5 cm. At that moment, the temperature in the pan was 190-200°C. The beefsteak was left for 2 min and was then turned. At 3 min, the heat was reduced to medium effect, and at 4 min the beefsteak was turned for the second time. After this, the steak was turned once a minute until the frying ended 10 minutes after it started (papers I, II, III) or 15 minutes after it started (paper IV). Eight minutes after adding the steak to the pan, 10 g of margarine or 10 ml of oil was added. As the beefsteak was removed from the pan, the pan was also removed from the heat and the electricity or gas was turned off. The temperature in the pan was measured with a TES 1322A remote non-contact thermometer, using a laser pointer to measure surface temperatures. During frying, the temperature on the side of the beefsteak facing the pan was 280-300°C.

Different combinations of parameters applied in the laboratory kitchen

Paper I: Sub-micrometer particles: Their level and how they spread after pan frying of beefsteak

Extraction hood C was used on the maximum capacity level. Three different types of experiments were performed: In experiment type 1, the exhaust hood was turned off as the beefsteak was removed from the heat. In experiment type 2, the exhaust hood was left on for 30 minutes after the frying had ended. Experiment types 1 and 2 were repeated four times. In experiment type 3, the extraction hood was turned off when the beefsteak was removed from the heat and the air supply in the neighbouring room was turned off during the whole experiment. Experiment type 3 was repeated three times. After each repetition of all experiment types, the kitchen was left until the next day, allowing the particle levels to return to baseline. Measurements of particles >0.3 μ m and ultrafine particles were performed during all the experiments, except two of the three repetitions of experiment type 3, where the system for measuring UFP (described later) was unavailable.

Paper II: Exposure to mutagenic aldehydes and particulate matter during panfrying of beefsteak with margarine, rapeseed oil, olive oil or soybean oil

Extraction hood B was used on the medium capacity level. The standard frying procedure was repeated two or three times during each day of frying. After every repetition, the kitchen was ventilated with the aid of an extra exhausting fan until the level of particles in the air was within 7-14 part/cm³ in the size fraction 0.3-0.5 μ m, measured with the Met One particle counter (described later). Measurements of total particles and particles >0.3 μ m and sampling of high molecular weight aldehydes was performed.

Paper III: Different types and settings of kitchen canopy hoods and particulate exposure conditions during pan-frying of beefsteak

All kitchen extraction hoods were used on both the medium and the maximum capacity level. The standard frying procedure was repeated three times during each day of frying. After every repetition, the kitchen was ventilated with the aid of an extra exhausting fan, until the level of particles in the air was within 7-14 part/cm³ in the size fraction 0.3-0.5 μ m, measured with the Met One particle counter (described later). The air velocity into the kitchen extraction hood was controlled before the first and after the third repetition, using a TSI Model 8345/8346 Velocicalc Anemometer. The air velocity was measured in all four corners and in the middle of the inlet by holding the probe of the anemometer 1 cm below the inlet of the hood.

Sampling of total particles and measurements of particles $>0.3 \mu m$ were performed.

Paper IV: Exposure to polycyclic aromatic hydrocarbons (PAHs), mutagenic aldehydes and particulate matter during pan frying of beefsteak

Extraction hood B was used on the medium capacity level. Sampling and measurements were conducted continuously during 1 day of frying (214-229 min). The standard frying procedure (15 min) was repeated five times during each day of frying. The repetitions were separated by a 25 min break without activating additional ventilation other than the extraction hood in the kitchen. There was also a 25 min break after the last repetition, in which the kitchen door was kept closed and all sampling and measuring continued. Sampling of total particles, PAHs and higher aldehydes was performed, as well as measurements of ultrafine particles.

In restaurants

Paper V: Exposure to polycyclic aromatic hydrocarbons (PAHs), mutagenic aldehydes and particulate matter in Norwegian à la carte restaurants

Three restaurants in the city of Trondheim in the middle of Norway were chosen. These were à la carte restaurants with a majority of meat dishes based on beefsteak (bovine ox) on their menu. The aim of this choice was to obtain as much cooking fumes from the frying of beefsteak as possible. The three kitchens had devices for deep frying, combined cooking and frying tops, and grills. All devices were equipped with standard exhaust hoods for restaurant kitchens. Personal sampling of total particles, PAHs and higher aldehydes was performed. One person in each restaurant carried three pumps, each connected to a sampling device. The person selected to carry the sampling devices was the person who was reckoned to do the most frying of beefsteak during the hours of sampling. All samplings were repeated for 3 days during the 4 hours which were supposed to be peak hours regarding the number of customers in the restaurant.

Applied methods

Personal exposure sampling

Total particles (papers II, III, IV and V)

The sampling of total particles was performed using preweighed, double Gelman AE glass fibre filters (37 mm). The filters were placed in a closed face, clear styrene, acrylonitrile (SAN) cassette connected to a pump with an air flow of 2 l/min. The filter cassette was placed on one of the shoulders of the cook. The sampling was run continuously through one day of frying. Before and after sampling, the filters were conditioned in an exicator for 24 hours. The filters were analysed gravimetrically, using a Mettler balance (0.01 mg resolution). An inner calibration was performed on the Mettler balance before every weighing. Blank filters were included in the analysis in order to control for deviations caused by temperature or humidity.

High molecular weight aldehydes (papers II, IV and V)

Sampling of higher aldehydes was conducted by use of stainless steel tubes with 220 mg Tenax TA connected to a pump with a sampling flow rate of 100 ml/min. The tubes were placed on one of the shoulders of the cook. In paper II the sampling started when the beefsteak was added to the pan and ended when the pan was removed from the heat, a total of ten minutes. In papers IV and V the sampling was conducted continuously throughout each day of frying. After sampling, the tubes were closed with end caps and stored in room temperature until analysis.

The Tenax tubes were analysed by thermic desorption in an ATD 400 (Automatic Thermal Desorber, Perkin Elmer, Waltham, USA) and gas chromatography-mass spectrometry in a Focus GC–DSQ (Thermo-Electron Corporation, Waltham, USA) following standard procedures for qualitative/semi-quantitative MS-Full-Scan analyses (Health and Safety Executive 1993). The identified aldehydes were quantified as equivalents based on the response of hexanal. The analyses were performed by a certified commercial laboratory.

PAHs (papers IV and V)

Sampling of polycyclic aromatic hydrocarbons was conducted by use of a pump connected to two XAD(II) tubes (backup and sampling tube) and a glass fibre filter (37 mm) in a closed face filter cassette. The sampling flow rate was 1 l/min according to standard procedures. It

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was run continuously throughout each day of frying. After sampling, the tubes and the filter cassette were closed with end caps and stored in a refrigerator until analysis.

The XAD(II) tubes and the filter from the filter cassette were desorbed in dichloromethane and analysed by gas chromatography-mass spectrometry (GC-MS) for a selection of PAH components (16 US EPA standard), following a method of analysis which is a modified version of AMI L5, NIOSH 5515, ISO/CD 12884 and VDI 3873. The analyses were performed by a certified commercial laboratory, with Danish accreditation no 168 (DANAK 168). The 16 PAH's determined were: naphthalene, acenaphtylene, acenaphthene, fluorene, phenanthrene, anthracene, fluoranthene, pyrene, benzo(a)anthracene, chrysene, benzo(b+k)fluoranthene, benzo(e)pyrene, benzo(a)pyrene, indeno(1,2,3-cd)pyrene, dibenzo(a,h)anthracene and benzo(ghi)perylene.

Ultrafine particles (paper IV)

In paper IV, a TSI 3936 Scanning Mobility Particle Sizer (SMPS) system was used to perform personal exposure measurements of the total number concentration of ultrafine particles. The following settings were used for these measurements: The electrostatic classifier was fitted with a 0.0457 cm impactor nozzle with a flow rate of 0.3 l/min and a sheath flow rate of 3.0 l/min. This corresponds to a measurement range of 14.3-673.2 nm. Scanning time was 2 min 15 s. Measurements started every 3 min and lasted throughout the entire frying. Air was drawn through a flexible silicone tube. This tube fits the impactor of the electrostatic classifier optimally (1/4 inch). The length of the tube was 2.65 m. In paper IV, the inlet of the silicone tube was placed on the right shoulder of the cook.

Stationary measurements

Particles >0.3 μm (papers I, II and III)

The level of particles in the kitchen was determined by the use of a Met One model 237B laser particle counter in the kitchen. This particle counter registers the number of particles in 0.1ft³ (2832 cm³) of air in the particle sizes $0.3 - 0.5 \mu$ m, $0.5 - 0.7 \mu$ m, $0.7 - 1.0 \mu$ m, $1.0 - 2.0 \mu$ m, 2.0 - 5.0 μ m and above 5.0 μ m. In papers I-III, the number of particles in the size fractions >1.0 μ m was not reported since they contained only a small percentage (<5 %) of the total number of particles registered. All measurements were started manually, and lasted for one

minute. Measurements were performed before frying and 1 min, 4 min, 7 min, 10 min, 12 min, 15 min and 18 min after the start of frying. The inlet of the particle counter was located 1 m above the floor and 1.3 m to the side of the stove. In paper I, a second Met One particle counter was placed in the neighbouring room, located in the middle of the room, 1 m above the floor.

Submicrometer particles (paper I)

The TSI 3936 SMPS system was used to measure the total number concentration of submicrometer (including ultrafine) particles and the associated size distribution in the neighbouring room. The following settings were used for these measurements. The electrostatic classifier was fitted with a 0.0457 cm impactor nozzle with a flow rate of 0.3 l/min and a sheath flow rate of 3.0 l/min. This corresponds to a measurement range of 14.3-673.2 nm. Scanning time was 3 min 15 s. Measurements started every 4th minute and lasted through the whole of experiments type 1, 2 and 3. The apparatus was placed in the middle of the room, 1 m above the floor.

SUMMARY OF PAPERS I-V

PAPER I: SUB-MICROMETER PARTICLES: THEIR LEVEL AND HOW THEY SPREAD AFTER PAN FRYING OF BEEFSTEAK

The number concentration of sub-micrometer particles and their spread during and after the frying of beefsteak were measured in a laboratory kitchen and in the neighbouring room. The kitchen was equipped with a modern extraction hood which exhausted outside the building and an electric stove, which was placed in a cooking island in the middle of the room. The frying was performed according to an experimental, standardized procedure, mimicking actual cooking conditions in a private household in Norway. The aim was to study the spread of particles to the neighbouring room, and to register how the particle concentration decreased after frying in relation to the use of the kitchen extraction hood. The level of particles increased rapidly in the kitchen when frying was started, regardless of the use of the hood. The sub-micrometer particles spread rapidly to the neighbouring room and reached a maximum 10-40 minutes after the top level in the kitchen, depending on the type of ventilation. The highest level of particles in the range 0.3-0.5 in the neighbouring room was on average 5.8 % of the level in the kitchen (SD 2 %). In both rooms, the main size fraction of the particles was below 0.5 μ m. Continuing the extraction for 30 min after the end of frying gave a significantly reduced number of particles in all size fractions in the neighbouring room.

PAPER II: EXPOSURE TO MUTAGENIC ALDEHYDES AND PARTICULATE MATTER DURING PANFRYING OF BEEFSTEAK WITH MARGARINE, RAPESEED OIL, OLIVE OIL OR SOYBEAN OIL.

The aim of the study was to see if higher mutagenic aldehydes could be detected in fumes from the frying of beefsteak collected in the breathing zone of the cook, and to compare exposure conditions during the use of different frying fats. The frying was performed in a laboratory kitchen, according to an experimental, standardized procedure, mimicking actual cooking conditions in a private household in Norway. The stove was placed centrally on one of the walls in the kitchen. A modern extraction hood, exhausting outside the building, was mounted 50 cm above the stove. The levels of higher aldehydes (*trans,trans-*2,4-decadienal, 2,4-decadienal, *trans,trans-*2,4-nonadienal, *trans-*2-decenal, *cis-*2-decenal, *trans-*2-undecenal, 2-undecenal, as well as various alkanals and alkenals) and total particles were sampled in the breathing zone of the cook during the panfrying of beefsteak with four different frying fats: margarine, soybean oil, virgin olive oil and rapeseed oil. In addition, the number of particles in the size intervals $0.3-0.5 \ \mu\text{m}$, $0.5-0.7 \ \mu\text{m}$ and $0.7-1.0 \ \mu\text{m}$ were measured in the kitchen. Registered mean concentrations of mutagenic aldehydes were between non-detectable (s-2-decenal) and 25.33 $\ \mu\text{g/m}^3$ (2,4-decadienal, t-2-decenal). The exposure level of total particles was between 1.0 and 11.6 mg/m³. Higher aldehydes were detected in all samples, and mutagenic aldehydes were detected in most of the samples. Frying with margarine gave statistically significantly higher levels of mutagenic aldehydes and particles in all size fractions than frying with the three different kinds of oil. When comparing the three different types of oil, the levels of mutagenic aldehydes and particles were not statistically significantly different.

PAPER III: DIFFERENT TYPES AND SETTINGS OF KITCHEN CANOPY HOODS AND PARTICULATE EXPOSURE CONDITIONS DURING PAN-FRYING OF BEEFSTEAK

The aim of the study was to compare four different kinds of canopy hoods in common use in private Norwegian households in regard to their ability to protect the cook from exposure to particles from cooking fumes and to reduce the spreading of fume particles in the kitchen. The hoods were tested during the pan-frying of beefsteak under different combinations of the following parameters: medium or maximum flow rate in the hood, two different heights above the cooking surface (50 or 60 cm) and three different locations in the kitchen (in a corner, at the wall or in the middle of the floor). The mass concentration of total particles was sampled in the breathing zone of the cook and the number concentration of particles in the size interval 0.3-0.5 μ m in the kitchen was measured. To achieve the best possible effect of a kitchen canopy hood under the conditions described, it is best to mount it in a corner with a 60 cm distance to the cooking surface and run it on the maximum flow rate. Also, it is best to install hoods that extract the polluted air out of the house rather than recycling it through a charcoal filter.

PAPER IV: EXPOSURE TO POLYCYCLIC AROMATIC HYDROCARBONS (PAHs), MUTAGENIC ALDEHYDES AND PARTICULATE MATTER DURING PAN FRYING OF BEEFSTEAK

The aim of the study was to see if PAHs and higher mutagenic aldehydes could be detected in fumes from the pan frying of beefsteak which were collected in the breathing zone of the cook. Also, the aim was to see if there were differences between levels of PAHs, higher aldehydes and particulate matter when frying on an electric or a gas stove with two different frying fats (margarine and soya bean oil). Frying was performed in a laboratory kitchen, under conditions similar to those in a Western European restaurant kitchen. The concentrations of PAHs (16 US EPA standard), higher aldehydes (trans,trans-2,4-decadienal, 2,4-decadienal, trans,trans-2,4-nonadienal, trans-2-decenal, cis-2-decenal, trans-2-undecenal, 2-undecenal) and total particles were sampled during frying on an electric or gas stove with margarine or soya bean oil as the frying fat. The number concentration of particles <100 nm in size (ultrafine) was measured. Levels of naphthalene were in the range of 0.15-0.27 μ g/m³. BaP were detected in two samples during frying with margarine on the gas stove (0.14 μ g/m³). Phenathrene (0.06 μ g/m³) and DBahA (1.3 μ g/m³) were detected in single samples. Registered concentrations of mutagenic aldehydes were between non-detectable and 61.80 $\mu g/m^3$. The exposure level of total aerosol was between 1.6 and 7.2 mg/m³. Peak number concentrations of ultrafine particles were in the range of 60 000-896 000 particles/cm³. Naphthalene and mutagenic aldehydes were detected in most of the samples. The levels were variable, and seemed to be dependent on many factors involved in the frying process. According to the results, frying on a gas stove instead of an electric stove causes increased occupational exposure to some of the components in cooking fumes which may cause adverse health effects.

PAPER V: EXPOSURE TO POLYCYCLIC AROMATIC HYDROCARBONS (PAHs), MUTAGENIC ALDEHYDES AND PARTICULATE MATTER IN NORWEGIAN À LA CARTE RESTAURANTS

The aim of the study was to characterize the exposure regarding PAHs and higher mutagenic aldehydes in the breathing zone of the cook during work in Norwegian à la carte restaurants. In addition, we wanted to verify the results obtained in experimental studies, where PAHs and higher mutagenic aldehydes were sampled in cooking fumes produced in a laboratory kitchen under conditions intended to be similar to the conditions in a Western European restaurant kitchen during the frying of beefsteak. Personal measurements of the levels of PAHs, higher aldehydes, and total particles were performed in three restaurants in the city of Trondheim in the middle of Norway. Levels of particle exposure were registered to make the results comparable to other studies. Naphthalene was detected within the range of $0.05-0.27 \,\mu g/m^3$. Acenaphthylene (0.04 μ g/m³) was found in one single sample. The total mean value for all three restaurants was 0.18 µg/m^3 air. The registered concentrations of mutagenic aldehydes were between 1.03 and 17.67 μ g/m³. The mean mass concentration of total particles in the three restaurants was 1.93 mg/m^3 , and the levels registered were within the range 0.32-7.51mg/m³. The reported results were comparable to the ones obtained in the laboratory kitchen, and were assumed to be representative for the exposure that professional cooks are subject to during the frying of beefsteak in a Western European restaurant. Working as a cook in a Norwegian à la carte restaurant with some manual pan frying involves exposure to components in cooking fumes which may cause adverse health effects. Additional studies are necessary in order to identify relations between exposure levels and the adverse health effects of cooking fumes.

DISCUSSION

Methods

The main intention with the experiments conducted in the present study was to study what cooks are exposed to during different cooking activities. To do this, we had to perform cooking activities under standardized conditions, so that it was possible to compare the results obtained from various situations. However, this proved to be complicated. Kelly (2001) describes the difficulty of conducting cooking tests in a repeatable manner in words that are very transferrable to our studies: "The intent of the testing was to perform the cooking activities in a realistic manner, and that was achieved. However, differences in the food cooked, the specific actions of different cooks and the normal variability in chemical measurements and control of the chamber resulted in about a factor of two variability in the purchase of food in large enough quantities to supply multiple test runs. This degree of variability may be typical of cooking emissions in a home or a commercial kitchen as well. Some conditions, such as air exchange rate, uniformity of food cooked, background air composition and the cook's activities may even be less controlled in normal, real-life conditions than in the experimental tests performed in this study".

Total particles

Glass fibre filters were chosen as the sampling medium for total particles because these filters are non-hydrophilic. The filters were analysed gravimetrically. This was chosen on the basis of a previous study (Svendsen et al. 2002), where the glass fibre filters were analysed by FT-IR, to find the concentration of fat aerosols. As it is not known whether the fat aerosols or other aerosols cause harmful effects, we found it more appropriate and less complicated to perform gravimetric analyses. The results from the former FT-IR analyses of fat on the filters may be lower than the results from the samples that are analysed gravimetrically, but as most of the particles present in the kitchen atmosphere are supposed to be fat aerosols, the differences are not likely to be substantial.

Particles >0.3 µm

Measurements of particles with aerodynamic diameter larger than $0.3 \mu m$ were conducted with a diluter connected to the Met One particle counter. The factor of dilution used was 10. Some settling of particles throughout the diluter and tube has probably occurred. However, the results are considered to be good indicators of particle levels, and they allow for internal comparison as the same device and settings were used for all the experiments.

Ultrafine particles

A SMPS system was used to measure the number concentration of submicrometer particles in the neighbouring room (paper I) and UFP in the breathing zone of the cook (paper IV). This is an established, widely used and highly accepted method for particle measurements. In paper IV, however, a tube was used to make it possible to measure in the breathing zone of the cook. It is expected that some loss of particles will occur due to settling as the air travels through the tube. This was not accounted for when presenting the results. However, the results reported in paper IV are reckoned to be internally comparable. In addition, this is only one of many factors affecting the results of particle number concentrations measurements during various types of cooking, and the impact of it alone is not reckoned to be significant when comparing different studies.

PAHs

In papers IV and V, PAHs were sampled on glass fibre filters (particulate phase) and XAD(II) tubes (vapour phase). The filters and tubes were analysed by GC-MS for a selection of PAH components (16 US EPA standard) by a commercial laboratory. In an Asian study, samples of air in various types of Chinese restaurants were taken on glass fibre filters and polyurethane foam plastic piece absorbents (Cui et al. 1995) and analysed by both GC-MS and high-performance liquid chromatography (HPLC). They stated that the fluorometric detector of the HPLC had effectively greater sensitivity for PAHs than the GC/MS. However, in samples from a frying work station in a Hepingmen Roast Duck Shop, Cui et al. (1995) found concentrations of BaP in the vapour and airborne particulate phase of 0.014 and 0.019 μ g/m³ respectively, when analyzing the sampled material with HPLC. In our studies, we found concentrations are in the same order of magnitude and even higher than the ones registered by Cui et al. (1995). This may indicate that we were able to register concentrations of BaP or

other PAHs in the samples even though our laboratory did not use HPLC to analyse the material.

High molecular weight aldehydes

In papers II, IV and V, high molecular weight aldehydes were sampled on Tenax tubes connected to a pump. According to the commercial laboratory performing the analyses, this active sampling method will mainly sample aldehydes in the gaseous phase, even though some condensed aldehydes of varying volatility also will be trapped. Yang et al. (2007) reported that 83 % of tt-DDE was found in the particulate phase when sampling at the exhaust stacks from 16 different restaurants. However, the distribution of tt-DDE between the gaseous and particulate phases seemed to depend on a number of factors, such as equilibrium temperature and the available adsorptive area of the particulate matter. It must be noted that we sampled higher aldehydes much closer to the source than Yang et al. (2007), possibly allowing less time for the aldehydes present in the gaseous phase to condense onto particles. It must also be mentioned that there is no standard, validated method for the sampling of high molecular weight aldehydes.

The main results

Several studies have investigated the production of particulate matter and various chemical components during the cooking of miscellaneous types of food under different conditions. However, the variations are great, and make it almost impossible to make direct comparisons of the results found. Even within the different studies great diversities may occur, illustrating that not only cooking styles, cooking temperatures, the duration of cooking and types of food and frying fats, but also the intensity of cooking activities, characteristics of sampling sites, sampling heights, the nature of instrumentation employed for collecting fumes, ventilation rate, the use and quality of kitchen exhaust hoods, indoor temperature, relative humidity, appliances and quality of fuels used may have an effect on the composition of cooking fumes.

Total particles

The results registered by personal sampling in the papers II-V showed total particle concentrations in the range of $310-11600 \ \mu g/m^3$. Most of the total particle concentrations that were reported were in the area of $1000-2000 \ \mu g/m^3$. These concentrations are similar to the

personal exposure levels registered on Norwegian professional cooks (arithmetic mean concentration of 620 μ g/m³ and peak concentration of 6600 μ g/m³, Svendsen et al. 2002). The exposure levels in Svendsen et al.'s study (2002) were found by FT-IR analysis, and reported as concentrations of fat aerosols. As most of the aerosols present in the kitchen atmosphere are reckoned to be fat aerosols, the results reported by Svendsen et al. (2002) are considered to be comparable to the levels of total particles obtained by gravimetric analyses in papers II-V.

The PM10 concentrations in the study conducted by Fortmann et al. (2002) were in the range of 144-289 μ g/m³ during the frying of minced beef and bacon. The PM10 concentrations registered by Kelly (2001) were in the range of 118-1547 μ g/m³ during the frying of hamburgers. The total particle fraction sampled with 37 mm three piece filter cassettes is estimated to have a 50 % cut size at about 20 μ m in aerodynamic diameter (PM20) (Vincent 1989). It has been indicated that almost 90 % of the PM10 fraction are PM2.5 particles (Kelly 2001). Based on this, it may be suggested that a large percent of the PM20 (total particle) fraction is comprised of particles in the PM10 fraction or smaller. If this is so, the results from our papers are in the same order of magnitude as the results from other studies conducted under conditions that are remotely similar (Kelly 2001, Fortmann et al. 2002).

When performing experimental studies mimicking the conditions during the frying of beef steak under occupational conditions in Norway (paper IV), the main results showed sampled levels of total particles in the range of 1600-7200 μ g/m³. These, and the results in paper V, obtained from sampling in real-life in three Norwegian restaurants, are not easily compared to the results from other studies performed in restaurant kitchens, mainly because of different sampling strategies. However, the total particle concentrations reported in paper IV (1600-7200 μ g/m³) are comparable to the ones in paper V (1080-3170 μ g/m³) and also to the concentrations of fat aerosols reported in Svendsen et al (2002) (peak concentration of 6600 μ g/m³). This indicates that the results from the laboratory kitchen may be representative for real-life exposure conditions in professional restaurants.

The PM10 levels reported in studies from Chinese and Korean restaurants (Lee et al. 2001, Lee et al. 2007, Pan et al. 2008a) seem to be low compared to the PM10 levels that may be

estimated, based on the total particle concentrations reported in paper V. Based on this it may be assumed that the exposure levels of particulate matter in Chinese restaurant kitchens are not higher than in Western restaurants.

Submicrometer particles

The mean submicrometer particle concentrations measured during domestic cooking conditions in our papers I-III are in the range of 147-1468 particles/cm³, and the peak levels registered in paper I are in the range of 1000-1200 particles/cm³. These are much lower levels than the ones registered in other studies performed under experimental conditions in domestic kitchens (Li et al. 1993) and also in studies performed under real-life conditions in domestic kitchens (Abt et al. 2000a, He et al. 2004c, Hussein et al. 2006). The particle counter available during most of our studies did not measure particles smaller than 0.3 μ m in diameter, while the other studies used instruments which registered particles down to the size of 0.007-0.017 nm. Considering the fact that these smaller size fractions normally include the largest number of particles, this probably is the main reason that our results are lower. Thus, a large part of the diversity may be a result of different instrumentation.

Ultrafine particles

When performing experimental studies mimicking the conditions during the frying of beef steak under occupational conditions in Norway (paper IV), peak number concentrations of UFP in the range of 60 000 -896 000 particles/cm³ were registered. The average UFP number concentrations registered by Yeung and To (2008) under conditions quite similar to the ones described in paper IV were rather much higher than the above-mentioned UFP number concentrations, even though their measurements were stationary. This may be caused by the frying period being shorter and more intense than in our study, but generally there is no obvious reason for the differences observed.

Most of the available studies on UFP number concentrations measured under real-life conditions (Long et al. 2000, Wallace et al. 2004, Wallace 2006, Morawska et al. 2003) and under experimental conditions (Dennekamp et al. 2001, Fortmann et al. 2002, Afshari et al. 2005, See and Balasubramanian 2006a, Evans et al. 2008) in domestic kitchens showed results in the same order of magnitude as the UFP concentrations measured during the frying

of beef steak under occupational conditions in our laboratory kitchen (paper IV).

Spreading of particulate matter to neighbouring rooms

In paper I, measurements of UFP and particles in size fraction 0.3-0.5 µm were conducted in the neighbouring room during the frying of beef steak in the kitchen. The rooms were separated by a passage with open doors in both ends. The arithmetic mean peak PM0.3-0.5 and UFP concentrations in the neighbouring room were in the range of 42-78 and 1500-6500 particles/cm³ respectively. These maximum levels of particles were reached 13-23 and 20-28 minutes respectively after frying started in the kitchen. In comparison, the maximum levels of PM0.3-0.5 particles in the kitchen (1000-1200 particles/cm³) were reached 10-13 minutes after the start of frying. The ventilation conditions in the two rooms had an effect on the decay rates of particles, as leaving the kitchen extraction hood on for 30 minutes after the end of frying resulted in a quicker decay in the neighbouring room, while turning off the ventilation in the neighbouring room while turning off the ventilation in the neighbouring room, while turning off the ventilation in the neighbouring room.

The actual particle concentrations measured in our studies are difficult to compare with the concentrations reported in other studies, owing to a number of reasons discussed earlier. However, it may be possible to see some trends in the spreading of particulate matter during cooking. At the start of the frying, there was an increase in particle numbers both in the kitchen and the living room, but peak levels were higher in the kitchen than in the living room. Similar results have also been reported by others (Wallace 2000, Kelly 2001, Fortmann et al. 2002, Lai and Chen 2007, Lai and Ho 2008). The longer the cooking fume particles travel before being sampled, the more particles will be lost on the way (Lai and Ho 2008), probably due to both gravitational settling and the coagulation of aerosols. The coagulation of particles may also explain why the concentration of 10 μ m particles in the living room has been observed, both by us and by others (Kelly 2001, Hussein et al. 2006, Lai and Ho 2008). The lengths of the time-lags vary, possibly related to fluctuating air exchange rates between the rooms (Hussein et al. 2006).

PAHs

Most of the PAH concentrations registered during cooking under domestic conditions (reallife and controlled experiments) are relatively low, except for the ones reported by Zhu and Wang (2003). The relatively high naphthalene levels detected in their study were mainly explained by the presence of mothballs in the bedrooms of the houses where sampling took place. It was presumed that evaporation from the mothballs was easily transported to the kitchen by air movement. However, the concentrations of naphthalene and total PAHs reported in the same study, sampled during cooking in commercial kitchens where no mothballs were present, are also relatively high, indicating that other factors may also have had an impact on the results.

As mentioned above, the PAH concentrations reported after sampling in commercial kitchens by Zhu and Wang (2003) are relatively high, standing out from the rest for no obvious reason. In addition, Vainiotalo and Matveinen (1993) registered high concentrations of naphthalene and total PAHs, compared to the other studies. In their study, PAHs were deliberately sampled at workplaces where meat was fried at relatively high temperatures (250-300°C). In the restaurant where the peak levels of PAHs and also the highest number of different PAHs were detected, the grilling of beef at 300°C was the main cooking style. The sampling time was short (30 min), thus including little other than active grilling. The other studies reporting PAH concentrations sampled in commercial kitchens were performed in Asian style restaurants, mainly cooking dishes based on vegetables. Based on this it may seem that the cooking of meat at high temperatures may generate higher PAH exposure levels. This tendency may also be seen in the results from controlled experiments performed in domestic kitchens where the frying of hamburgers (Kelly 2001) gives higher PAH levels than the frying of mixed types of food (Flückiger et al 2000, Fortmann et al. 2002, See and Balasubramanian 2008). However, it seems obvious that other factors, such as sampling methods, the location of samplers or the use of kitchen exhaust hoods, also affect the results. For example, in some of the studies, only particulate PAHs were registered. According to Cui et al. (1995), who reported that more than 42 % of the BaP found in fumes from the kitchen of four Chinese restaurants were in the vapour phase, this may imply some loss of PAHs.

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When comparing the PAH concentrations reported in paper IV (frying under experimental conditions mimicking commercial kitchens) and V (real-life sampling in restaurant kitchens) with results from other studies, they are higher than most of the results reported by others, but lower than the levels reported by Zhu and Wang (2003) and Vainiotalo and Matveinen (1993). These deviations may be caused by several factors, as mentioned above, but the fact that our results are placed within the range reported in other studies may indicate that our methods of sampling and analysis are suitable. Overall, the results indicate no systematic differences regarding PAH concentrations between Asian style and Western style cooking.

In general, the concentrations of both particulate matter and PAHs registered in our papers I-V are higher than the ones found in the other studies. Even though there may be various reasons for this, it may have been caused by the fact that we have conducted personal sampling, as opposed to most of the other studies. Fortmann et al. (2002) performed two tests with stove-top stir frying, where concentrations of particulate matter measured in the kitchen air were compared to concentrations measured with personal samplers worn by the cook. The PM2.5 concentrations for the personal samples were four to six times higher than the kitchen air concentrations, indicating that the stationary room air concentrations may underestimate the cook's exposure to PM2.5. During the sampling of larger particles on an optical scattering device, it was shown that the personal exposures of the cook may be several times the wholehouse concentrations (Wallace 2000). Others have also shown that the relation between monitoring points and room lay-out is very important and that the results from personal samplers are not necessarily comparable to the results from stationary samplers (Koyano et al. 2001, Evans et al. 2008).

High molecular weight aldehydes

High molecular weight aldehydes were found in all samples reported in paper II, IV and V, and mutagenic aldehydes were detected in most of the samples. The concentrations varied substantially, but the results in paper IV and V indicate that the pan frying of beefsteak on a gas stove may imply exposure to higher aldehyde concentrations than on an electric stove.

Some studies have investigated the emissions of high molecular weight aldehydes during different cooking processes (Schauer et al. 1999, Schauer et al. 2002, Dung et al. 2006,

Fullana et al. 2004a,b, Yang et al. 2007, Katragadda 2010), but unfortunately, it is not possible to compare these results to the findings in the present thesis, owing to different study designs and ways of presenting the results. However, these studies constitute a basis for investigations of the personal exposure to higher aldehydes during cooking.

tt-DDE has been identified as one of the most abundant of the higher aldehydes found in cooking fumes (Chiang et al. 1997, Wu et al. 2001, Zhu et al. 2001, Fullana et al. 2004a,b, Yang et al. 2007). This seems to be reflected in our studies, where tt-DDE frequently is reported in higher concentrations than other higher aldehydes, regardless of the environment in which sampling was conducted.

In paper IV, the aldehyde concentrations were considerably higher than the ones reported in paper II. This may be caused by the degradation of some of the frying oil used, which at the time of the experiments in paper IV had been stored at room temperature for about 10-12 months, while the oil used during the experiments in paper II was purchased a short time before use.

The levels of higher aldehydes registered during cooking on an electric stove in paper IV and V are in the same order of magnitude. This may indicate that the results obtained in the laboratory kitchen are representative for real-life exposure conditions in professional restaurants. The results obtained during frying on a gas stove in paper IV are not comparable to the results reported in paper V, as only one of the three restaurants had a gas grill, and this was used in combination with an electric stove.

When comparing the levels of total particles, UFP, PAHs and higher aldehydes reported in paper IV and V, they seem to fluctuate independently. The situations where the highest concentrations of total particles were registered are not necessarily the situations that produced the highest levels of PAHs and high molecular weight aldehydes, neither in the experimental studies (paper IV) nor in the commercial kitchens (paper V).

Kitchen exhaust hoods

When comparing different kitchen canopy hoods (paper III), our main conclusion was that the hood should ideally be mounted in a corner, 60 cm above the stove, and run on the maximum flow rate in order to achieve the best possible exposure conditions for the cook and other occupants of the kitchen under the conditions investigated. These results are supported by the findings of Fortmann et al (2002), where side shields from the range top to the exhaust hood proved to give lower PM2.5 concentrations in the kitchen than when using the hood without side shields. Similar findings were also reported by Flückiger et al. (2000), where integrating the exhaust ventilator into a row of cupboards on the wall increased the efficiency of pollutant removal. In addition, our results also indicated that it is best to install hoods that extract the pollutants out of the building, rather than recycling the air through charcoal filters.

In paper I, the measurements of particle concentrations in the neigbouring room showed that letting the kitchen exhaust hood run for about 30 minutes after frying had finished led to a significant reduction in the level of particles in a much shorter time than when the hood was turned off immediately after frying had finished. This is consistent with the findings of He et al. (2004c), who stated that the main effect of kitchen exhaust ventilation was on the decay behaviour of the aerosols generated by cooking, and that the decay rate under poor ventilation conditions was lower than under normal ventilation conditions.

In summary, several studies have shown that the use of a kitchen exhaust hood reduces the exposure to cooking fumes (Chiang et al. 1998, Flückiger et al. 2000, Koyano et al. 2001, Fortmann et al. 2002, Ke et al. 2009), which in many cases also implies a reduced risk of exposure to carcinogenic compounds and developing cancer (Ko et al. 1997, Chiang et al. 1998, Chiang et al. 1999a, Wu et al. 2004, Chen et al. 2007, Ke et al. 2009). However, it is important that the hood used is a powerful and well suited type, adequately installed, regularly cleaned and attended. It also needs to be used in a sensible way to make it as efficient as possible.

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Different frying fats

In paper II, indications were found that the use of margarine as frying fat implied higher exposure to particulate matter and mutagenic aldehydes than the use of rapeseed, soya bean or olive oil. The use of the three different oils resulted in equal exposure levels. These findings were somewhat contradicted by the findings in paper IV, where the concentrations of mutagenic aldehydes registered during frying with soya bean oil were considerably higher than the ones reported in paper II, indicating that the degradation of the oil over time may increase the exposure to mutagenic compounds when heating stored oils (as discussed earlier).

The smoke point of frying oil is the temperature at which the oil begins to smoke continuously and can be seen as a bluish smoke, which is an indication of the chemical breakdown of the fat to glycerol and free fatty-acids. In a study on volatile emissions from the heating of different types of cooking oils, Katragadda et al. (2010) stated that it is a general rule that the higher the smoke point, the better suited a fat is for frying; e.g. fats with smoke point below 200°C are not suitable for deep-fat frying. The temperature of any oil used for deep-frying operations should be established below its smoke point, otherwise the emission of potentially toxic compounds will increase significantly. However, even emissions below the smoke point of oils may be harmful. The tendency for volatiles to form depends on the fatty acid composition; the more free fatty acids (linoleic acid, oleic acid) the more volatiles. This, along with other studies which found various emission rates for different compounds from the heating of different frying fats (Li et al. 1994, Chiang et al 1997, Wu et al. 1998, Chiang et al. 1999a,b, Lin and Liou 2000, Chen and Chen 2001, Zhu et al. 2001, Katragadda et al. 2010), indicates that the choice of frying fats should not be random.

Gas and electric appliances

When comparing pan frying of beefsteak on a gas and an electric stove in paper IV, we found that frying on a gas stove may cause increased exposure to some of the hazardous components (total particles, UFP, some PAHs and some higher aldehydes) in cooking fumes. In addition, the only two samples where BaP was found above the detection limit were sampled during frying on the gas stove. This may be due to the higher immediate temperature the food is exposed to when frying on a gas stove, as it has been suggested that at least the production of particulate matter should depend on stove temperature (Evans et al. 2008). The tendency that gas appliances produce more air pollutants than electric appliances may also be seen in other

studies (Dennekamp 2001, Yeung and To 2008), but the variations are substantial.

In our studies, it seemed that the UFP fraction was comprised of smaller particles (40-60 nm) when frying on a gas stove than when frying on an electric stove (80-100 nm). Wallace (2000) found that cooking activities were a source of both fine and coarse particles, whereas the gas appliances produced more of the UFP. Overall, there seems to be a trend that electric appliances produce a size distribution that shifts towards larger particles in all the size fractions (UFP, PM2.5, PM10) relative to that produced by gas appliances (Flückiger et al. 2000, Dennekamp 2001, Kelly 2001, Fortmann 2002, Hussein 2006). However, it has been concluded that the type of cooking activity had a greater influence on cooking emissions than the type of appliance used (Flückiger et al. 2000, Kelly 2001), a conclusion mainly based on blank tests where both types of appliances produced minimal particle emissions (PM2.5 and PM10) in the absence of food (Kelly 2001). Concurrent measurements of UFP (<0.1 μ m) with a particle counter could possibly have altered this conclusion, since the smaller particles contribute little to the total particular mass, whereas they occur in large number concentrations.

Health risk assessment and exposure to cooking fumes

One way of assessing the health risk due to occupational exposure is to compare measured concentrations of different pollutants to their occupational exposure limit (OEL) or threshold limit value (TLV). However, there is no OEL or TLV for cooking fumes. It may be relevant to compare the registered total particle concentrations to the Norwegian TLVs for organic dust (5 mg/m³) or nuisance dust (5 (respirable fraction) or 10 (total dust) mg/m³) (Arbeidstilsynet 2009). The mean levels registered in paper I-IV are below these limit values. However, the concentrations are high in certain situations, for example, the mean total particle concentration sampled during frying on a gas stove in paper IV is 6.35 mg/m³. Also, the mean levels reported during frying with margarine in paper II are high.

Regarding the UFP, it has been stated that indoor sources such as cooking on a gas stove often provide a substantial fraction of total exposure to UFP (Wallace 2006, Long et al. 2000). The question has arisen as to whether UFP from the combustion of natural gas may be of different toxicity than UFP from common outdoor sources such as vehicles (Wallace 2006). One study found that most of the mutagenicity of ambient air in Los Angeles could be traced to gas

appliance emissions rather than automobiles and diesel vehicles (Hannigan et al. 2005). Therefore there is some evidence suggesting that UFP from indoor sources may have important health effects (Wallace 2006).

Generally, the levels of naphthalene and other PAHs registered in our studies are low compared to the Norwegian OEL for total PAH ($40 \ \mu g/m^3$) and compared to both Norwegian and international OELs for naphthalene and BaP (Arbeidstilsynet 2009, BGIA 2010).

A Japanese study stated that in samples of airborne particles, higher PAH concentrations and stronger mutagenicity is associated with smaller particle diameter (Koyano et al. 2001). The study also referres to cases in which higher PAH concentrations and stronger mutagenicity was found in indoor samples compared with outdoor samples. The cause in such cases was suggested to be the influence from cooking. Thus, the exposure to particulate matter from cooking may have a stronger ability to cause adverse health effects than exposure to particles generated in other situations.

Some regulatory bodies use a quantitative risk assessment process to determine an excess cancer risk over a lifetime (ELCR). This approach mathematically calculates the probability of developing cancer over a person's lifetime at a given exposure level. It is presented as a value representing the number of extra cancers expected in a given number of people on exposure to a carcinogen at a stated dose. Estimated values of ELCR for occupational exposure to PAHs and toxic metals in cooking fumes have been reported to be on the order of 10^{-2} to 10^{-4} (See et al 2006, See and Balasubramanian 2006b). This is several orders of magnitude above the recommended acceptable limit, which according to the authors is set to 10^{-6} for ELCR by the US EPA. This suggests that cooks and other workers in the commercial kitchens are exposed to an exceedingly large amount of fine particles containing carcinogenic PAHs and other harmful pollutants. Domestic cooking is also likely to pose elevated ELCR, even though the duration of cooking is lower than that in occupational settings (Evans et al. 2008).

As far as we know, no standards or guidelines have been set for occupational exposures to tt-DDE or the other mutagenic aldehydes identified in the cooking fumes. It has, however, been suggested that prolonged exposure to non-cytotoxic, low levels of tt-DDE $(0.1 \mu M)$ can induce oxidative stress and potentially DNA damage in certain human bronchial epithelial cells (Chang et al. 2005).

Kelly (2001) concluded that residential cooking activities produce substantial increases in particles and gaseous species in indoor air, though under realistic conditions the levels do not approach health-based air quality standards. This is in consistency with our findings. However, the setting of OELs and TLVs is not based on health perspectives alone, but also as a result of political and economic evaluations. In regard to the risk of developing cancer, Felton (1995) stated that the fumes generated during the cooking of beef have about one third the mutagenic activity measured in the fried meat itself, and that occupational exposure over long periods could pose some risk, though probably less than that from consuming the meat. Overall, it seems that the health risk inflicted by exposure to cooking fumes is difficult to assess, but there is reason to reduce the exposure as far as possible.

SUMMARY AND CONCLUSIONS

Our studies confirmed the presence of PAHs and high molecular weight aldehydes in fumes from Norwegian domestic and occupational cooking styles which were collected in the breathing zone of the cook. We also showed that the fumes spread to neighbouring rooms, and that leaving the kitchen extraction hood on for 30 minutes after the end of frying reduced the spreading significantly. The use of different types and settings of kitchen extraction hoods resulted in different exposure conditions. The choice of frying fat also seemed to affect the concentrations of particulate matter and chemical components produced during the frying of beefsteak, but our studies were not comprehensive enough to recommend preferable fats. In addition, our results indicated that frying on a gas stove caused a higher exposure to some of the hazardous components in cooking fumes than frying on an electric stove.

Many studies on the emission of particulate matter and various chemical components during cooking have been conducted, but very few of them are comparable. As reported by IARC (2010), neither occupational nor non-occupational exposure to emissions from cooking has been characterized systematically. Most of the available studies have examined the nature and amount of emissions produced during different types of cooking in different settings. As the substances measured and conditions investigated vary widely between studies, it is difficult to summarize exposure conditions in different settings. Our studies indicated that cooking under experimental conditions may give a correct reflection of exposure conditions in real-life, commercial kitchens. However, numerous repetitions of a very specific and standardized cooking method under controlled conditions are necessary factors for obtaining representative results from the experimental studies.

Several studies have focused on the characterisation of cooking oil fumes obtained under experimental conditions, detecting for instance carcinogenic components such as tt-DDE and PAHs in the fumes from heated cooking oils of different kinds (Li et al. 1994, Chiang et al. 1999a,b, Lin and Liou. 2000, Chen and Chen 2001, To et al. 2000, Wu et al. 2001, Zhu et al. 2001, Fullana et al. 2004a,b, Katragadda et al. 2010). Our studies confirmed previous findings (Schauer et al. 1999, Yang et al. 2007) showing that the same components can also be detected in fumes from cooking styles that are not to the same extent based on the heating of oils. It may even seem that some of the components may be present at higher levels in

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fumes from meat-based cooking styles than in oil fumes. In addition, there is no reason to believe that Asian-style cooking generates higher concentrations of more harmful cooking fumes than Western-style cooking.

When performing our studies, we expected to find a connection between the level of total particles and the level of PAHs and higher aldehydes, but it seems that the concentrations of these components are dependent on other factors than just the levels of particles in the room. This may be considered as yet another indication of the fact that many elements have an impact on exposure conditions during cooking, and that these elements fluctuate. In addition, it implies that the measured levels of, for example, particulate matter alone are unsuitable as indicators of the actual health risk inflicted upon persons exposed to cooking fumes.

In our studies, we were able to detect PAHs and mutagenic aldehydes using personal samplers placed in the breathing zone of the cook. This implies that the frying of beefsteak under both occupational and domestic conditions causes exposure to these compounds in detectable concentrations. To our knowledge, only very few studies have investigated the presence of health effects and the actual exposure to cooking fumes simultaneously (Sivertsen et al. 2002, Pan et al. 2008a,b, Svedahl et al. 2009). To be able to identify relations between exposure to cooking fumes and their adverse effects, additional studies are required.

Considering that cooking fumes consist of a mixture of partly toxic and mutagenic compounds with no known dose-response relationship, it seems vital to reduce the exposure to these fumes as far as possible. In the area of extraction hoods, more research is needed to develop optimal systems in regard to aspects such as ventilation efficiency, noise level and design.

REFERENCES

Abt E, Suh HH, Allen G, Koutrakis P. (2000a) Characterization of indoor particle sources: A study conducted in the metropolitan Boston area. Environ Health Perspect; 108(1), 35-44.

Abt E, Suh HH, Catalano P, Koutrakis P. (2000b) Relative contribution of outdoor and indoor particle sources to indoor concentrations. Environ Sci Technol; 34, 3579-3587.

Afshari A, Matson U, Ekberg LE. (2005) Characterization of indoor sources of fine and ultrafine particles: a study conducted in a full scale chamber. Indoor Air; 15, 141-150.

Alessandrini F, Schulz H, Takenaka S, Lentner B, Karg E, Behrendt H, Jakob T. (2006) Effects of ultrafine carbon particle inhalation on allergic inflammation of the lung. J Allergy Clin Immunol; 117, 824-30.

Allen R, Wallace L, Larson T, Sheppard L, Liu LJS. (2004) Estimated hourly personal exposures to ambient and nonambient particulate matter among sensitive populations in Seattle, Washington. J Air Waste Manag Assoc; 54, 1197-1211.

Arbeidstilsynet. (2009) Veiledning om administrative normer for forurensing i arbeidsatmosfære. Available at: http://www.arbeidstilsynet.no/binfil/download2.php?tid=77907 (accessed 28.06.10).

Beck-Speier I, Dayal N, Karg E, Maier KL, Schumann G, Schulz H, Semmler M, Takenaka S, Stettmaier K, Bors W, Ghio A, Samet JM, Heyder J (2005). Oxidative stress and lipid mediators induced in alveolar macrophages by ultrafine particles. Free Radic Biol Med; 38, 1080-1092.

BGIA. GESTIS – International limit values for chemical agents 2010. Available at: http://bgiaonline.hvbg.de/LIMITVALUE/WebForm gw.aspx (accessed 21.06.10)

Biswas P, Wu C. (2005) Nanoparticles and environment. J Air Waste Manag Assoc; 29, S1015-S1016.

Brown DM, Wilson MR, MacNee W, Stone V, Donaldson K. (2001) Size-dependent proinflammatory effects of ultrafine polystyrene particles: a role for surface area and oxidative stress in the enhanced activity of ultrafines. Toxicol Appl. Pharmacol; 175, 191-199.

Chalupa DC, Morrow PE, Oberdörster G, Utell MJ, Frampton MW. (2004) Ultrafine particle deposition in subjects with asthma. Environ Health Perspect; 112(8), 879-882.

Chang LW, Lo WS, Lin P. (2005) Trans,trans-2,4-decadienal, a product found in cooking oil fumes, induces cell proliferation and cytokine production due to reactive oxygen species in human bronchial epithelial cells. Toxicol Sci; 87, 337-343.

Chang C. (2010) The immune effects of naturally occurring and synthetic nanoparticles. Journal of Autoimmunity; 34, J234-J246.

Chen BH, Chen YC. (2001) Formation of polycyclic aromatic hydrocarbons in the smoke from heated model lipids and food lipids. J Agric Food Chem; 49, 5238-5243.

Chen B, Hu Y, Jin T, Zheng L, Wang Q, Shen Y, Zhou Y. (2007) Higher urinary 1-hydroxypyrene concentration is associated with cooking practice in a Chinese population. Toxicol Lett; 171, 119-125.

Chiang TA, Wu PF, Wang LF, Lee H, Lee CH, Ko YC. (1997) Mutagenicity and polycyclic aromatic hydrocarbon content of fumes from heated cooking oils produced in Taiwan. Mut Res; 381, 157-161.

Chiang TA, Wu PF, Ko YC. (1998) Prevention of exposure to mutagenic fumes produced by hot cooking oil in Taiwanese kitchens: Environ Mol Mutat; 31, 92-96.

Chiang TA, Wu PF, Ko YC. (1999a) Identification of carcinogens in cooking oil fumes. Environ Res; 81(1), 18-22.

Chiang TA, Pei Fen W, Ying LS, Wang LF, Ko YC. (1999b) Mutagenicity and aromatic amine content of fumes from heated cooking oils produced in Taiwan. Food Chem Toxicol; 37(2-3), 125-34.

Coggon D, Pannet B, Osmond C, Acheson E. (1986a) A survey of cancer and occupation in young and middle aged men. Cancer of the respiratory tract. Br J Ind Med; 43, 332-9.

Coggon D, Pannet B, Osmond C, Acheson E. (1986b) A survey of cancer and occupation in young and middle aged men. Non-respiratory cancers. Brit J Ind Med; 43, 381-386.

Cui M, Chen Z, Zhao Z. (1995) Benzo[a]pyrene in kitchen air and urinary 1-hydroxypyrene. Indoor and Built Environment; 4, 306-310.

DANAK 168. Accreditation for testing. Available at: http://published.danak.dk/register.asp?nohead=y&lang=e&akk=168 (accessed 14.06.10)

Dennekamp M, Howarth S, Dick CA, Cherrie JW, Donaldson K, Seaton A. (2001) Ultrafine particles and nitrogen oxides generated by gas and electric cooking. Occup Environ Med; 58(8), 511-516.

Dick CA, Brown DM, Donaldson K, Stone V. (2003) The role of free radicals in the toxic and inflammatory effects of four different ultrafine particle types. Inhal Toxicol; 15, 39-52.

Donaldson K, Stone V, Clouter A, Renwick L, MacNee W. (2001) Ultrafine particles. Occup Eniron Med; 58, 211-216.

Du YX, Cha Q, Chen XW, Chen YZ, Huang LF, Feng ZZ, Wu XF, Wu JM. (1996) An epidemiological study of risk factors for lung cancer in Guangzhou, China. Lung Cancer; 14(Suppl 1), S9-S37.

Dubowsky SD, Wallace LA, Buckley TJ. (1999) The contribution of traffic to indoor concentrations of polycyclic aromatic hydrocarbons. J Expo Anal Environ Epidem; 9, 312-321.

Dubrow R, Wegman DH. (1984) Cancer and occupation in Massachusetts: A death certificate study. Am J Ind Med; 6(3), 207-230.

Dung CH, Wu SC, Yen GC. (2006) Genotoxicity and oxidative stress of the mutagenic compounds formed in fumes of heated soybean oil, sunflower oil and lard. Toxicology in Vitro; 20, 439-447.

Esterbauer H, Benedetti A, Lang J, Fulceri R, Fauler G, Comporti M. (1986) Studies on the mechanism of formation of 4-hydroxynonenal during microsomal lipid peroxidation. Bioch Biophys Acta; 876, 154-166.

Evans GJ, Peers A, Sabaliauskas K. (2008) Particle dose estimation from frying in residential settings. Indoor Air; 18, 499-510.

Felton J. (1995) Food mutagens: The cooking makes a difference. Sci Technol Rev; 3, 18-25.

Feron VJ, Til HP, de Vrijer F, Woutersen RA, Cassee FR, van Bladeren PJ. (1991) Aldehydes: occurrence, carcinogenic potential, mechanism of action and risk assessment. Mut Res; 259, 363-385.

Flückiger B, Seifert M, Koller T, Monn C. (2000) Air quality measurements in a model kitchen using gas and electric stoves: Proceedings of Healthy Buildings (Helsinki); 1, 567-572.

Foppa I, Minder CE. (1992) Oral, pharyngeal and laryngeal cancer as a cause of death among Swiss cooks. Scandinavian Journal of Work, Environment & Health; 18(5), 287-92.

Fortmann R, Kariher P, Clayton R. (2002) Indoor air quality: Residential cooking exposures. Final report, California Air Resources Board, contract 97-330, Sacramento CA, Air Resources Board.

Fullana A, Carbonell-Barrachina AA, Sidhu S. (2004a) Comparison of volatile aldehydes present in the cooking fumes of extra virgin olive, olive and canola oils. J Agric Food Chem; 52, 5207-5214.

Fullana A, Carbonell-Barrachina AA, Sidhu S. (2004b) Volatile aldehyde emissions from heated cooking oils. J Sci Food Agric; 84, 2015-2021.

Gertz C. (2000) Chemical and physical parameters as quality indicators of used frying fats. Eur J Lipid Technol; 102, 566-572.

Gwinn MR, Vallyathan V. (2006) Nanoparticles: Health effects – pros and cons. Environ Health Perspect; 114(12), 1818-1825.

Hannigan MP, Busby WF, Cass GR. (2005) Source contributions to the mutagenicity of urban particulate air pollution. J Air Waste Manag Assoc; 55, 399-410.

Health and Safety Executive. (1993) MDHS 72. Volatile organic compounds in air – Laboratory method using pumped solid sorbent tubes, thermal desorption and gas chromatography. Suffolk, UK: Health and Safety Executive Books. ISBN 0 717 61756 4

He LY, Hu M, Wang L, Huang XF, Zhang YH. (2004a) Characterization of fine organic particulate matter from Chinese cooking. J Environ Sci; 16(4), 570-575.

He LY, Hu M, Huang XF, Yu BD, Zhang YH, Liu DQ. (2004b) Measurement of emissions of fine particulate organic matter from Chinese cooking. Atmos Environ; 38, 6557-6564.

He C, Morawska L, Hitchins J, Gilbert D. (2004c) Contribution from indoor sources to particle number and mass concentrations in residential houses. Atmos Environ; 38, 3405-3415.

Hildemann LM, Markowski GR, Jones MC, Cass GR. (1991) Submicrometer aerosol mass distributions of emissions from boilers, fireplaces, automobiles, diesel trucks and meat-cooking operations. Aerosol Science and Technology; 14, 138-152.

Hung HS, Wu WJ, Cheng YW, Wu TC, Chang KL, Lee H. (2007) Association of cooking oil fumes exposure with lung cancer: Involvement of inhibitor of apoptosis proteins in cell survival and proliferation in vitro. Mutat Res; 628, 107-116.

Hussein T, Glytsos T, Ondracek J, Dohanyosova P, Zdimal V, Hämeri K, Lazaridis M, Smolik J, Kulmala M. (2006) Particle size characterization and emission rats during indoor activities in a house. Atmos Environ; 40, 4285-4307.

IARC (1998). Polynuclear Aromatic Compounds, Part 1, Chemical, Environmental and Experimental Data. IARC Monographs, Vol 32. Available at: http://monographs.iarc.fr/ENG/Monographs/vol32/volume32.pdf (accessed 18.05.10).

IARC (2002). Vol 82. Some traditional Herbal Medicines, some Mycotoxins, Naphthalene and Styrene. IARC Monographs, Vol 82. Available at: http://monographs.iarc.fr/ENG/Monographs/vol82/mono82.pdf (accessed 18.05.10).

IARC (2010). Vol 95. Household use of solid fuels and high temperature frying. IARC Monographs, Vol 95. Available at: http://monographs.iarc.fr/ENG/Monographs/vol95/mono95.pdf (accessed 18.05.10).

Katragadda HR, Fullana A, Sidhu S, Carbonell-Barrachina AA. (2010) Emissions of volatile aldehydes from heated cooking oils. Food Chemistry; 120, 59-65.

Kamens R, Lee CT, Wiener R, Leith D. (1991) A study to characterize indoor particles in three nonsmoking homes. Atmos Environ; 25A, 939-948.

Ke Y, Cheng J, Zhang Z, Zhang R, Zhang Z, Shuai Z, Wu T. (2009) Increased levels of oxidative DNA damage attributable to cooking oil fumes exposure among cooks. Inhal Toxicol; 21(8), 682-687.

Kelly T (2001) Measurement of particulate and vapor emissions from cooking activities. Final report (June 1998-June 2000). Contract no. 5083. Gas Research Institute, Des Plaines, IL.

Kiel P. Kræft og stegeos. Arbejdsmiljøfondets forskningsrapporter nr 781426. København: Arbejdsmiljøfondet; 1986.

Kleeman MJ, Scauer JJ, Cass GR. (1999) Size and composition distribution of fine particulate matter emitted from wood burning, meat charbroiling and cigarettes. Environ Sci Technol; 33(20), 3516-3523.

Kleeman MJ, Robert MA, Riddle SG, Fine PM, Hays MD, Schauer JJ, Hannigan MP. (2008) Size distribution of trace organic species emitted from biomass combustion and meat charbroiling. Atmos Environ; 42, 3059-3075.

Ko YC, Lee CH, Chen MJ, Huang CC, Chang WY, Lin HJ, Wang HZ, Chang PY. (1997) Risk factors for primary lung cancer among non-smoking women in Taiwan. Int J Epidemiol; 26(1), 24-31.

Koyano M, Mineki S, Tsunoda Y, Endo O, Goto S, Ishii T. (2001) Effects of fish (mackerel pike) broiling on polycyclic aromatic hydrocarbon contamination of suspended particulate matter in indoor air. J Health Sci; 47(5), 452-459.

Lai ACK, Chen FZ. (2007) Modeling of cooking-emitted particle dispersion and deposition in a residential flat: A real room application. Build Environ; 42, 3253-3260.

Lai ACK, Ho YW. (2008) Spatial concentration variation of cooking-emitted particles in a residential kitchen. Build Env; 43, 871-876.

Lee SC, Li WM, Chan LY. (2001) Indoor air quality at restaurants with different styles of cooking in metropolitan Hong Kong. Sci Total Environ; 279, 181-193.

Lee, BK, Jeong UR, Dong TT. (2007) Concentration comparison of the indoor air pollutants identified from restaurants with different cooking and ventilation methods. Available at: http://www.inive.org/members_area/medias/pdf/Inive%5CIAQVEC2007%5CLee_2.pdf (accessed 23.05.10)

Levy JI, Dumyahn T, Spengler JD. (2002) Particulate matter and polycyclic aromatic hydrocarbon concentrations in indoor and outdoor microenvironments in Boston, Massachusetts. J Expo Anal Environ Epidem; 12, 104-114.

Li C, Lin W, Jenq F. (1993) Size distributions of submicrometer aerosols from cooking. Environ Int; 19, 147-154.

Li S, Pan D, Wang G. (1994) Analysis of polycyclic aromatic hydrocarbons in cooking oil fumes. Arch Environm Health; 49(2), 119-122.

Li N, Sioutas C, Cho A, Schmitz D, Misra C, Sempf J, Wang M, Oberley T, Froines J, Nel A. (2003) Ultrafine particulate pollutants induce oxidative stress and mitochondrial damage. Environ. Health Perspect; 111, 455-460.

Lin YK, Lee LH. (2010) An integrated occupational hygiene consultation model for the catering industry. Ann Occup Hyg; ePub ahead of print, doi: 10.1093/annhyg/meq017

Lin JM, Liou SJ. (2000) Aliphatic aldehydes produced by heating Chinese cooking oils. Bull Environ Contam Toxicol; 64, 817-824.

Long CM, Suh HH, Koutrakis P. (2000) Characterization of indoor particle sources using continuous mass and size monitors. J Air Waste Manag; 50, 1236-1250.

Lund E, Borgan JK. (1987) Dødelighet av kreft blant kokker [Cancer mortality among cooks]. Tidsskr Nor Laegeforen; 107, 2635-2637 (In Norwegian).

McDonald JD, Zielinska B, Fujita EM, Sagebiel JC, Chow JC, Watson JG. (2003) Emissions from Charbroiling and Grilling of Chicken and Beef. J Air Waste Manag Assoc, 53, 185-194.

Metayer C, Wang Z, Kleinerman RA, Wang L, Brenner AV, Cui H, Cao J, Lubin JH. (2002) Cooking oil fumes and risk of lung cancer in women in rural Gansu, China. Lung Cancer; 35, 111-17.

Morawska L, He C, Hitchins J, Mengersen K, Gilbert D. (2003) Characteristics of particle number and mass concentrations in residential houses in Brisbane, Australia. Atmos Environ; 37, 4195-4203.

Nappez C, Battu S, Beneytout JL. (1996) Trans,trans-2,4-decadienal: Cytotoxicity and effect on gluthatione level in human erythroleukemia (HEL) cells. Cancer Letters; 99 115-119.

Ng TP, Hui KP, Tan WC. (1993) Respiratory symptoms and lung function effects of domestic exposure to tobacco smoke and cooking by gas in non-smoking women in Singapore. J Epidemiol Community Health; 47(6), 454-8.

Ng TP, Tan WC. (1994) Epidemiology of allergic rhinitis and its associated risk factors in Singapore. Int J Epidemiol; 23(3), 553-8.

Notani PN, Shah P, Jayant K, Balakrishnan V. (1993) Occupation and cancers of the lung and bladder: A case control study in Bombay. Int J Epidemiol; 22(2), 185-191.

Oberdörster G, Gelein RM, Ferin J, Weiss B. (1995) Association of particulate air pollution and acute mortality: involvement of ultrafine particles? Inhal. Toxicol; 7, 111-124.

Oberdörster G. (2001) Pulmonary effects of inhaled ultrafine particles Int. Arch. Occup. Environ. Health; 74, 1-8.

Oberdörster G, Sharp Z, Atudorei V, Elder A, Gelein R, Kreyling W, Cox C. (2004) Translocation of inhaled ultrafine oarticles to the brain. Inhal Toxicol; 16, 437-445.

Oberdörster G, Maynard A, Donaldson K, Castranova V, Fitzpatrick J, Ausman K, Carter J, Karn B, Kreyling W, Lai D, Olin S, Monteiro-Riviere N, Warheit D, Yang H. (2005a) Principles for characterizing the potential human health effects from exposure to nanomaterials: elements of a screening strategy. Part Fibre Toxicol; 2, 1-35.

Oberdörster G, Oberdörster E, Oberdörster J. (2005b) Nanotoxicology: an emerging discipline evolving from studies of ultrafine particles. Environ Health Perspect; 113, 823-839.

Ogulei D, Hopke PK, Wallace LA. (2006) Analysis of indoor particle size distributions in an occupied townhouse using positive matrix factorization. Indoor Air; 16, 204-215.

Olson DA, Burke JM. (2006) Distributions of PM2.5 source strengths for cooking from the research triangle park particulate matter panel study. Environ Sci Technol; 40, 163-169.

Ostiguy C, Lapointe G, Ménard L et al. (2008) Health effects of nanoparticles. Report IRSST R-589, Montréal

Pan CH, Chan CC, Wu KY. (2008a) Effects on Chinese restaurant workers of exposure to cooking oil fumes: A cautionary note on urinary 8-hydroxy-2'-deoxyguanosine. Cancer Epidemiol Biomarkers Prev; 17(12), 3351-3357

Pan CH, Chan CC, Huang YL, Wu KY. (2008b) Urinary 1-hydroxypyrene and malondialdehyde in male workers in Chinese restaurants. Occup Environ Med; 65, 732-735.

Pavanello S, Lupi S, Pullioer A, Gregorio P, Saia BO, Clonfero E. (2007) Mutagenic activity of overnight urine from healthy non-smoking subjects. Environ Molec Mut; 48, 143-150.

Preuss R, Angerer J, Drexler H. (2003) Naphthalene – an environmental and occupational toxicant. Int Arch Occup Environ Health; 76, 556-576.

Pu Y, Yin L, Yuan H, Liang G. (2002) Oxidative stress of cooking oil fume on rat type II lung cells. J Hyg Res; 31, 85-87 [in Chinese]

Rappaport SM, Waidyanatha S, Serdar B. (2004) Naphthalene and its biomarkers as measures of occupational exposure to polycyclic aromatic hydrocarbons. J Environ Monit; 6, 413-416.

Rogge WF, Hildemann LM, Mazurek MA, Cass GR, Simoneit BRT. (1991) Sources of fine organic aerosol. 1. Charbroilers and meat cooking operations. Env Sci Technol; 25(6), 1112-1125.

Schauer JJ, Kleeman MJ, Cass GR, Simoneit BRT. (1999) Measurement of emissions from air pollution sources. 1. C₁ through C₂₉ organic compounds from meat charbroiling. Environ Sci Technol; 33, 1566-1577.

Schauer JJ, Kleeman MJ, Cass GR, Simoneit BRT. (2002) Measurement of emissions from air pollution sources. 4. C₁-C₂₇ organic compounds from cooking with seed oils. Environ Sci Technol; 36, 567-575.

Schoenberg JB, Stemhagen A, Mogielnicki AP, Altman R, Abe T, Mason TJ. (1984) Case-control study of bladder cancer in New Jersey. I Occupational exposures in white males. J Natl Cancer Inst; 72, 973-981.

See SW, Balasubramanian R. (2006a) Physical characteristics of ultrafine particles emitted from different gas cooking methods. Aerosol and Air Quality Research; 6(1), 89-92.

See SW, Balasubramanian R. (2006b) Risk assessment of exposure to indoor aerosols associated with Chinese cooking. Environmental Research; 102, 197-204.

See SW, Balasubramanian R. (2008) Chemical characteristics of fine particles emitted from different gas cooking methods. Atmos Environ; 42, 8852-8862.

See SW, Karthikeyan S, Balasubramanian R. (2006) Health risk assessment of occupational exposure to particulate-phase polycyclic aromatic hydrocarbons associated with Chinese, Malay and Indian cooking. J Environ Monit; 8, 369-376.

Seinfeld JH, Pankow JF. (2003) Organic atmospheric particulate material. Annu Rev Phys Chem; 54, 121-140.

Semmler-Behnke M, Takenaka S, Fertsch S, Wenk A, Seitz J, Mayer P, Oberdörster G, Kreyling WG. (2007) Efficient elimination of inhaled nanoparticles from the alveolar region: evidence for interstitial uptake and subsequent reentrainment onto airways epithelium. Environ Health Perspect; 115, 728-733.

Shields PG, Xu GX, Blot WJ, Fraumeni JF, Trivers GE, Pellizzari ED, Qu YH, Gao YT, Harris CC. (1995) Mutagens from heated Chinese and US cooking oils. J Nation Canc Inst; 87(11), 836-841.

Sioutas C, Delfino RJ, Manisha S. (2005) Exposure assessment for atmospheric ultrafine particles (UFPs) and implications in epidemiologic research. Environ Health Perspect; 113, 947-955.

Sivertsen I, Sjaastad AK, Svendsen K, Krøkje Å. (2002) Alveolar macrophages as biomarkers of pulmonary irritation in kitchen workers. Ann Occup Hyg; 46(8), 713-717.

Sjögren B, Barlow L, Weiner J. (2009) Ischemic heart disease among cooks, cold buffet managers, kitchen assistants and wait staff. Scand J Work Environ Health; Suppl 7, 24-29.

Subramanian J, Govindan R. (2007) Lung cancer in never smokers: A review. J Clin Oncol; 25(5), 561-570.

Svedahl S, Svendsen K, Quenild T, Sjaastad AK, Hilt B. (2009) Short term exposure to cooking fumes and pulmonary function. Journal of Occupational Medicine and Toxicology; 4:9. DOI: 10.1186/1745-6673-4-9. Available at: http://www.occup-med.com/content/4/1/9 (accessed 07.05.10)

Svendsen K, Jensen HN, Sivertsen I, Sjaastad AK. (2002) Exposure to cooking fumes in restaurant kitchens in Norway. Ann Occup Hyg; 46(6), 395-400.

Svendsen K, Sjaastad AK, Sivertsen I. (2003) Respiratory symptoms in kitchen workers. Am J Ind Med; 43(4), 436-439.

Teschke K, Hertzman C, Van Netten C, Lee E, Morrison B, Cornista A, Lau G, Hundal A. (1989) Potential exposure of cooks to airborne mutagens and carcinogens. Environ Res; 50, 296-308.

Thiébaud HP, Knize MG, Kuzmicky PA, Hsiah DP, Felton JS. (1995) Airborne mutagens produced by frying beef, pork and a soy-based food. Food Chem Toxic; 33(10), 821-828.

To WM, Lau YK, Yeung LL. (2007) Emission of carcinogenic components from commercial kitchens in Hong Kong. Indoor Built Environ; 16, 29-37.

Vainiotalo S, Matveinen K. (1993) Cooking fumes as a hygienic problem in food and catering industries. Am Ind Hyg Assoc J; 54, 376-82.

Vincent JH. Aerosol sampling - science and practice. John Wiley & sons, Chichester, ISBN 0471921750, 1989

Wallace L. (2000) Real-time monitoring of particles, PAH and CO in an occupied townhouse. Appl Occup Environ Hyg; 15(1), 39-47.

Wallace LA. (2006) Indoor sources of ultrafine and accumulation mode particles: Size distributions, size-resolved concentrations and source strengths. Aerosol Sci Technol; 40, 348-360.

Wallace LA, Emmerich SJ, Howard-Reed C. (2004) Source strengths of ultrafine and fine particles due to cooking with a gas stove. Environ Sci Technol; 38, 2304-2311.

Wallace L, Howard-Reed C. (2002) Continuous monitoring of ultrafine, fine and coarse particles in a residence for 18 months in 1999-2000. J Air Waste Manag Assoc; 52, 828-844.

Wallace L, Ott W. (2010) Personal exposure to ultrafine particles. J Expo Sci Environ Epidemiol; Jan 20. Epub ahead of print, doi:10.1038/jes.2009.59.

Wang XR, Chiu YL, Qiu H, Au JSK, Yu ITS. (2009) The roles of smoking and cooking emissions in lung cancer risk among Chinese women in Hong Kong. Annals of Oncology; 20, 746-751.

Wang CK, Chang LW, Chang H, Yang CH, Tsai MH, Tsai HT, Lin P. (2010) Pulmonary changes induced by trans,trans-2,4-decadienal, a component of cooking oil fumes. Eur Respir J; 35, 667-675.

WHO (2000) Air quality guidelines for Europe. 2nd edn. Copenhagen, Denmark: World Health Organization WHO regional publications European series, No. 91.

Wu PF, Chiang TA, Wang LF, Chang CS, Ko YC. (1998) Nitro-polycyclic aromatic hydrocarbon contents of fumes from heated cooking oils and prevention of mutagenicity by catechin. Mut Res; 403, 29-34.

Wu PF, Chiang TA, Ko YC, Lee H. (1999) Genotoxicity of fumes from heated cooking oils produced in Taiwan. Environ Res; 80, 122-126.

Wu SC, Yen GC, Sheu F. (2001) Mutagenicity and identification of mutagenic compounds of fumes obtained from heating peanut oil. J Food Prot; 64, 240-245.

Wu SC, Yen GC. (2003) Oxidative damage of A-549 cells induced by trans-2-decenal from oil fumes. Food Sci Agric Chem; 5, 41-46.

Wu SC, Yen GC. (2004) Effects of cooking oil fumes on the genotoxicity and oxidative stress in human lung carcinoma (A-549) cells. Toxicol in Vitro; 18-571-580.

Wu MT, Lee LH, Ho CK, Wu SC, Lin LY, Cheng BH, Liu CL, Yang CY, Tsai HAT, Wu TN. (2004) Environmental exposure to cooking oil fumes and cervical intraepithelial neoplasm: Environ Res; 94, 25-32.

Wu M, Che W, Zhang Z. (2008) Enhanced sensitivity to DNA damage induced by cooking oil fumes in human OGG1 deficient cells. Environ Molec Mut; 49, 265-275.

Yang CC, Jenq SN, Lee H. (1998) Characterization of the carcinogen 2-amino-3,8dimethylimidazo[4,5-f]quinoxaline in cooking aerosols under domestic conditions. Carcinogenesis; 19(2), 359-363.

Yang HH, Chien SM, Lee HL, Chao MR, Luo HW, Hsieh DPH, Lee WJ. (2007) Emission of trans,trans-2,4-decadienal from restaurant exhausts to the atmosphere. Atmos Environ; 41, 5327-5333.

Yen GC, Wu SC. (2003) Reduction of mutagenicity of the fumes from cooking oil by degumming treatment. Food Science Technology/Lebensm-Wiss U Technol; 36, 29-35.

Yeung LL, To WM. (2008) Size distributions of the aerosols emitted from commercial cooking processes. Indoor Built Environ; 17(3), 220-229.

Young SC, Chang LW, Lee HL, Tsai LH, Liu YC, Lin P. (2010) DNA damage induced by trans,trans-2,4-decadienal (tt-DDE), a component of cooking oil fume, in human bronchial epithelial cells. Environ Molec Mut; 51, 315-321.

Yu ITS, Chiu YI, Au JSK, Wong TW, Tang JL. (2006) Dose-response relationship between cooking fumes exposure and lung cancer among Chinese non-smoking women. Cancer Res; 66(9), 4961-4967.

Zhao W, Hopke PK, Norris G, Williams R, Paatero P. (2006) Source apportionment and analysis on ambient and personal exposure samples with a combined receptor model and an adaptive blank estimation strategy. Atmos Environ; 40, 3788-3801.

Zhao Y, Hu M, Slanina S, Zhang Y. (2007a) Chemical compositions of fine particulate organic matter emitted from Chinese cooking. Environ Sci Technol; 41, 99-105.

Zhao Y, Hu M, Slanina S, Zhang Y. (2007b) The molecular distribution of fine particulate organic matter emitted from Western-style fast food cooking. Atmos Environ; 41, 8163-8171.

Zhong L, Goldberg MS, Gao YT, Jin F. (1999) Lung cancer and indoor air pollution arising from Chinese-style cooking among nonsmoking women living in Shanghai, China. Epidemiology; 10, 488-94. Zhu X, Wang K, Zhu J, Koga M. (2001) Analysis of cooking oil fumes by ultraviolet spectrometry and gas chromatography-mass spectrometry. J Agric Food Chem; 49, 4790-4794.

Zhu L, Wang J. (2003) Sources and patterns of polycyclic aromatic hydrocarbons pollution in kitchen air, China. Chemosphere; 50, 611-618.

PAPERS I-V

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