Exposure to Polycyclic Aromatic Hydrocarbons (PAHs), Mutagenic Aldehydes, and Particulate Matter in Norwegian à la Carte Restaurants

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Objectives: The aim of the study was to characterize the exposure regarding polycyclic aromatic hydrocarbons (PAHs) and higher mutagenic aldehydes in the breathing zone of the cook during work in Norwegian à la carte restaurants. Levels of particle exposure were also measured to make the results comparable to other studies.

Methods: 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.

Results: Naphthalene was detected within the range of 0.05–0.27 µg m⁻³ air, and the total mean value for all three restaurants was 0.18 µg m⁻³ air. The measured levels of mutagenic aldehydes were between 1.03 and 17.67 µg m⁻³ air. The mean mass concentration of total particles measured in the three restaurants was 1.93 mg m⁻³, and the levels registered were within the range 0.32–7.51 mg m⁻³.

Conclusions: 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.

Keywords: cooking fumes; occupational exposure; trans,trans-2,4-decadienal

INTRODUCTION

Cooking fumes, especially from frying, contain fine and ultrafine particles and several specific agents which may give adverse health effects in the lung (Vainiotalo and Matveinen, 1993; Svendsen et al., 2002; Wallace et al., 2004; Afshari et al., 2005).

In addition, cooking fumes contain substances with mutagenic activity, and they may be a risk factor in lung cancer (Chen et al., 1992; Metayer et al., 2002). Emissions from high-temperature frying have recently been classified as ‘probably carcinogenic to humans (Group 2A)’ by the International Agency for Research on Cancer (IARC, 2006). An increased risk of respiratory tract cancer in cooks and bakers has been reported (Coggon et al., 1986). Among the compounds which have been identified as mutagenic in cooking fumes are polycyclic aromatic hydrocarbons (PAHs), heterocyclic amines (HCAs), and higher aldehydes (Chiang et al., 1999a,b; Wu et al., 2001).

It has been documented that the major factors contributing to the level and type of substances in cooking fumes include the type of fuel, oil, food, and cooking methods employed during the operation (See et al., 2006). Several previous studies, mostly performed under Asian conditions, have identified different substances, including higher aldehydes and PAHs, in cooking fumes from frying with various combinations of these factors (Chiang et al., 1999a, Zhu et al., 2001, Zhu and Wang, 2003, See et al., 2006).

Among the PAHs identified in fumes from the heating of different cooking oils are benzo[a]pyrene (BaP), dibenzo[a,h]anthracene (DBa,hA), benzo [a]anthracene (BaA), and benzo[b]fluoranthene (BbFA). These have been detected in fumes from corn oil, vegetable oil, and safflower oil (Chiang et al., 1999a). In addition, BaP and BaA have been registered in fumes from soybean oil, rapeseed oil,
and lard (Zhu and Wang, 2003). BaP, DBahA, and BaA are considered to be probable human carcinogens (Group 2A) and BbFA is considered to be a possible human carcinogen (Group 2B) by IARC (1998).

Naphthalene (two rings) 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, cigarette smoke (Preuss et al. 2003). Naphthalene is classified as a possible human carcinogen (Group 2B) by IARC (2002) and has been registered in fumes from rapeseed oil (9.4–13 ng m\(^{-3}\)), soybean oil (11–14 ng m\(^{-3}\)), and lard (15–16 ng m\(^{-3}\)) (Zhu and Wang, 2003).

The thermal stressing of cooking oils rich in polyunsaturated fatty acids also generates various higher aldehyde species, i.e. aldehydes with a higher number of carbon atoms, such as \textit{trans}-2-alkenals, \textit{trans}-\textit{trans}-alka-2,4-dienals, and \textit{n}-alkenals, arising from the fragmentation of conjugated hydroperoxy-diene precursors (Gertz, 2000). Alkenals are saturated aldehydes (i.e. without double bonds), and alkenals are unsaturated aldehydes with one or more double bonds.

When investigating mutagenic compounds in fumes from peanut oil heated to temperatures of \(\sim 100^\circ\)C, the following compounds were identified as the ones with the strongest mutagenicity in the Ames test (in descending order): \textit{trans},\textit{trans}-2,4-decadienal, \textit{trans},\textit{trans}-2,4-nonadienal, \textit{trans}-2-decenal, and \textit{trans}-2-undecenal (Wu et al., 2001). \textit{Trans},\textit{trans}-2,4-decadienal has also been detected in cooking fumes resulting from heating other oils, such as rapeseed oil and soybean oil, and is considered to be the major mutagenic and cytotoxic compound in oil fumes (Zhu et al., 2001).

\textit{Trans},\textit{trans}-2,4-decadienal has been reported to inhibit human erythroleukemia cell growth, to affect cell viability, to reduce the cellular glutathione level, and to be involved in the initiation of DNA fragmentation \textit{in vitro} (Nappez et al., 1996). Other studies indicate an association between this aldehyde and genotoxic effects due to the reaction with nucleic acid bases (Lourenço et al., 2000).

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

The aim of the present study was to characterize the exposure regarding PAHs, higher mutagenic aldehydes, and particulate matter 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 measured in cooking fumes produced in a model kitchen under conditions intended to be similar to the conditions in a Western European restaurant kitchen during the frying of beefsteak.

Personal measurements were performed at three restaurants in the city of Trondheim in the middle of Norway. The levels of PAHs (16 EPA standard) and higher aldehydes (\textit{trans},\textit{trans}-2,4-decadienal, 2,4-decadial, \textit{trans},\textit{trans}-2,4-nonadienal, \textit{trans}-2-decenal, \textit{cis}-2-decenal, \textit{trans}-2-undecenal, 2-undecenal, as well as various alkanals and alkenals) were measured. The mass concentration of total particulate matter was also registered.

To our knowledge, data on personal exposure to PAHs and higher mutagenic aldehydes from cooking fumes produced in occupational settings have not previously been reported.

**METHODS**

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 since this is what we have been investigating in previous, experimental studies. The three kitchens had devices for deep frying, combined cooking and frying tops, and grills. All devices were equipped with standard ventilation hoods for restaurant kitchens, considered to be able to take care of the main part of the cooking fumes produced. The ventilation hoods seemed to work properly except for one evening in Restaurant 3, when the kitchen was filled with cooking fumes. The staff had to work under these conditions because they were unable to repair the system before the next day.

Table 1 contains a description of the three different restaurants where measurements were made.

All measurements were repeated for 3 days, during the 4 h which were supposed to be peak hours regarding the number of customers in the restaurant. In all three restaurants, this period of time occurred in the late afternoon and evening (\(\sim 6\) to 10 p.m.).

The measurements were performed as personal measurements and each person carried three pumps, each connected to a sampling device. In two of the restaurants, measurements were performed on two different persons, one person carried the sampling...
devices for 2 days and the other person carried them for 1 day. In the third restaurant, the same person carried the sampling devices through all the 3 days. The person selected to carry the sampling devices was the person supposed to do most frying of beefsteak during the measurements. The measuring devices were mounted on a small rucksack with the three pumps in the sack and the sampling devices mounted on the straps as near the breathing zone as possible.

**PAH measurements**

The levels of PAHs were measured by use of a pump connected to two XAD(II)-tubes (backup and sampling tube) and a glass fiber filter (37 mm) in a closed-face filter cassette. The pump used was a SKC Sidekick Air Sampling Pump, model 224-52TX. The sampling flow rate was 1 l min\(^{-1}\) according to standard procedures. 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 analyzed by gas chromatography–mass spectrometry (GC/MS) for a selection of PAH components (16 EPA standard), following a method of analysis which is a modified version of AMI L5, NIOSH 5515, ISO/CD 12884, and VDI 3873. The analyzes were performed by a certified commercial laboratory, with Danish accreditation no. 168 (DANAK 168).

The 16 PAHs determined were naphthalene, acenaphthylene, acenaphthene, fluorene, phenanthrene, anthracene, fluoranthene, pyrene, BaA, chrysene, benzo[\(b+k\)]fluoranthene, benzo[e]pyrene, BaP, indeno[1,2,3-cd]pyrene, DBa,hA, and benzo[g,h,i]-perylene.

**Aldehyde measurements**

The levels of higher aldehydes were measured by use of stainless steel tubes [automatic thermal desorber (ATD) tubes] with 220 mg Tenax TA. The sampling flow rate was 100 ml min\(^{-1}\), given by the laboratory for best performance regarding equipment capacity. The pump used was a SKC Pocket Pump model 210-1002. After sampling, the tubes were closed with end caps and stored in room temperature until analysis.

The ATD tubes were analyzed by thermic desorption in an ATD 400 (Perkin Elmer, Waltham, MA, USA) and GC/MS in a Focus GC–DSQ (ThermoElectron Corporation, Waltham, MA, USA) following standard procedures for qualitative/semi-quantitative MS-Full-Scan analyzes (Health and Safety Executive, 1993). The identified aldehydes were quantified as equivalents based on the response of hexanal. The analyzes were performed by a certified commercial laboratory.

The results were presented as the quantity of the aldehydes \textit{trans,trans}-2,4-decadienal, 2,4-decadienal, \textit{trans}-2-decenal, \textit{trans}-2,4-heptadienal. The group ‘alkanals’ consisted of eight different alkanals: butanal, pentanal, hexanal, heptanal, octanal, nonanal, decanal, and undecanal. The group ‘alkenals’ consisted of 2-heptenal, 2-octenal, 2-nonenal, and \textit{trans,trans}-2,4-heptadienal.

**Sampling of total particles**

The sampling of total particles was performed using preweighed, double Gelman AE glass fiber filters (37 mm). The filters were placed in a closed-face, clear styrene, acrylonitrile cassette connected to a pump (Casella Vortex standard 2 Personal Air Sampling Pump) with an airflow of 2 l min\(^{-1}\). Before and after sampling, the filters were conditioned in an exicator for 24 h. The filters were analyzed gravimetrically, using a Mettler balance (0.01 mg resolution). An inner calibration was performed on the Mettler

<table>
<thead>
<tr>
<th>Restaurant no.</th>
<th>No. of cooks employed</th>
<th>No. of seats in the restaurant</th>
<th>Type of frying fat used</th>
<th>Activity</th>
<th>Gas stove</th>
<th>Estimated time spent on frying during measurements (mean no. of minutes per 4 h)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>10</td>
<td>330</td>
<td>Rapeseed oil</td>
<td>Four cooks in activity at same time. Pizza is baked in oven, both dark and white meat is braised. Some fish is also braised and there is a deep frying device in the kitchen.</td>
<td>No</td>
<td>175</td>
</tr>
<tr>
<td>2</td>
<td>4</td>
<td>150</td>
<td>Soybean oil and some butter</td>
<td>A typical gourmet restaurant with the kitchen open to the restaurant. During the measurements duck, lamb, beef, deer, mushroom, and salmon were braised.</td>
<td>Yes (combined with an electric stove)</td>
<td>140</td>
</tr>
<tr>
<td>3</td>
<td>7</td>
<td>128</td>
<td>Soybean oil</td>
<td>A steak house with beef as a specialty. On Day 2, it seemed the ventilation system was broken as the cooking fumes was strongly perceptible.</td>
<td>No</td>
<td>198</td>
</tr>
</tbody>
</table>
balance before every weighing. Blank filters were included in the analysis in order to control for deviations caused by temperature or humidity.

Statistical analyzes

Data were handled by means of SPSS version 16.0.

RESULTS

Polycyclic aromatic hydrocarbons

In this study, naphthalene was the only PAH registered in samples from all the different restaurants. The mean levels of PAHs (micrograms per cubic meter) measured in the breathing zone of the cook in the different restaurants are presented in Table 2.

When measuring naphthalene, levels above the detection limit were registered in eight of nine samples. The non-detectable value was substituted by \( L/2^{1/2} \), where \( L \) is the detection limit (0.04 \( \mu g \) m\(^{-3} \)) (Hornung and Reed, 1990).

Acenaphthylene was registered above the detection limit in one single sample, which was collected in Restaurant no. 2 (Table 2).

No other PAHs were detected in the samples.

Aldehydes

Table 3 describes the quantity of \( \text{trans},\text{trans}-2,4\text{-decadienal}, \ 2,4\text{-decadienal}, \ \text{trans}-2\text{-decenal, 2-undecenal, alkanals, and alkenals measured in the breathing zone of the cook.} \)

In a series of measurements where some values were non-detectable, these values were substituted by \( L/2^{1/2} \), where \( L \) is the detection limit (Hornung and Reed, 1990). For example, when measuring \( \text{trans},\text{trans}-2,4\text{-decadienal, levels above the detection limit were registered in only one of the three samples from Restaurant no. 1. When measuring 2,4\text{-decadienal, levels above the detection limit were registered in one of the three samples from Restaurant no. 1 and in two samples from Restaurant no. 2. Levels of \( \text{trans}-2\text{-decenal and 2-undecenal were registered above the detection limit in two samples from Restaurant no. 3. In the group alkenals there were no registrations above the detection limit in one sample from Restaurant no. 1 and Restaurant no. 3. } \)

Total particles

The mean mass concentrations of total particles measured in the breathing zone of the cook during 4 h of work in a Norwegian à la carte restaurant are given in Table 4.

DISCUSSION

Polycyclic aromatic hydrocarbons

The present study shows low levels of PAH compared to the occupational exposure limit (OEL) applied in Norway (40 \( \mu g \) m\(^{-3} \) total PAH). The only two PAHs that were found to reach levels above the detection limit were naphthalene and acenaphthylene. It must, however, be mentioned that the detection limit for the more potent carcinogen BaP by our method was 0.04 \( \mu g \) m\(^{-3} \). Lung cancer risk from a lifetime exposure to PAHs in ambient air has been estimated to be \( 8.7 \times 10^{-5} \) per ng m\(^{-3} \) BaP (WHO, 2000). It has been estimated that naphthalene and acenaphthylene have a toxic equivalent factor of 0.001 compared to BaP (Nisbet and LaGoy, 1992). Thus, we assess the mean level of exposure to PAH in our study to be equal to \( \sim 0.2 \) ng m\(^{-3} \) BaP equivalents (total mean PAH \( \sim 0.2 \mu g \) m\(^{-3} \)).

In a previous study describing possible exposure from cooking fumes, several PAHs 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). Naphthalene (1.6–25.6 μg m⁻³) and low levels (0.02–2.3 μg m⁻³) of fluorene, phenanthrene, anthracene, pyrene, benzo [α]fluorine, chrysene, BaP, and benzo[ghi]perylene were detected in some samples. 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 detected mean naphthalene levels of 1.9, 2.8, and 3.9 ng m⁻³, respectively (See et al., 2006).

PAH levels have also been measured with stationary samplers in the breathing zone of the kitchen workers in other Chinese commercial kitchens (Zhu and Wang, 2003) where naphthalene levels were registered in the range of 1.5–3.0 μg m⁻³. However, these measurements were made under experimental conditions, with cooking procedures specially designed for the study. The present study describes personal exposure to cooking fumes and the sampling time includes not only frying but also boiling, garnishing, making desserts and so on.

A study on sources and patterns of PAH pollution in kitchen air in China showed that naphthalene was the most predominant two-ring kind of PAH in domestic kitchens (Zhu and Wang, 2003). This 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. However, our detection of naphthalene as the dominating PAH in cooking fumes in restaurant kitchens where no mothballs were present may indicate that cooking may be the dominating source of naphthalene also in the Chinese study.

**Aldehydes**

Higher aldehydes and mutagenic aldehydes were detected in samples from all the restaurants. The total arithmetic mean levels of mutagenic aldehydes are 9.79 μg m⁻³ (trans,trans-2,4-decadienal) and 8.23 μg m⁻³ (2-undecenal). The levels did, however, vary substantially and the highest measured level of trans,trans-2,4-decadienal was 38 μg m⁻³.

During the frying of beefsteak under experimental conditions assumed to be similar to a Western European restaurant kitchen, we were also able to detect higher aldehydes in all the samples and mutagenic aldehydes in most of the samples (A. K. Sjaastad and K. Svendsen, unpublished data). In that study, during frying on an electric stove, the total mean levels of trans,trans-2,4-decadienal, trans-2-decenal, and 2-undecenal were 14.25, 5.67, and 6.92 μg m⁻³, respectively. During frying on a gas stove, the total mean levels of trans,trans-2,4-decadienal, trans-2-decenal, and 2-undecenal were 49.07, 29.00, and 28.64 μg m⁻³, respectively. In two of the restaurants where measurements were made in the present study, no gas was used; in the third, there was a gas grill combined with an electric stove (Table 1). The levels measured when frying on the electric stove in the laboratory kitchen are in the same order of magnitude as the levels measured in the restaurants.

Several studies have focused on evaluating the mutagenicity and on finding the mutagenic components in cooking fumes (Chiang et al., 1999a,b; Wu et al., 2001). PAHs and HCAs have been identified as the main mutagenic compounds (Vainiotalo and Matveinen, 1993; Chiang et al., 1999b). A study on the mechanisms behind the association between exposure to cooking oil fumes and lung cancer indicates that these fumes induce antiapoptotic effects, contributing to the cell survival and proliferation of A-549 lung cancer cells. The results also indicate that trans,trans-2,4-decadienal from cooking oil fumes may make a more important contribution than PAHs (BaP) to the cell survival and the proliferation of A-549 lung cancer cells (Hung et al., 2007). As far as we are aware, there are no studies available that make it possible to make a risk assessment for exposure to these levels of trans,trans-2,4-decadienals or other higher aldehydes.

**Total particles**

The mass concentration of total particles in the breathing zone of the cook was measured in order to allow us to compare the results from the present study with results found in previous studies. Total particles were also measured because the method provides an easy and inexpensive indicator of exposure to cooking fumes. Although there is no OEL for this kind of particles anywhere, it may be relevant to compare measured levels to the Norwegian threshold limit values (TLVs) for organic dust (5 mg m⁻³) or nuisance dust (10 mg m⁻³). The mean levels of particles found in our study are below both these limit values.

The arithmetic mean mass concentration of total particles measured in the three restaurants in the present study was 1.93 mg m⁻³, and the levels

<table>
<thead>
<tr>
<th>Restaurant no.</th>
<th>No. of samples</th>
<th>Total particles, mean (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>3</td>
<td>1.54 (1.06)</td>
</tr>
<tr>
<td>2</td>
<td>3</td>
<td>1.08 (0.24)</td>
</tr>
<tr>
<td>3</td>
<td>3</td>
<td>3.17 (3.81)</td>
</tr>
<tr>
<td>Total</td>
<td>9</td>
<td>1.93 (2.20)</td>
</tr>
</tbody>
</table>

The levels are given as the arithmetic mean (SD) mass concentration of total particles registered during 3 days of measurements.

Table 4. The mass concentration of total particles (mg m⁻³) measured in the breathing zone of the cook during 4 h of work in a Norwegian à la carte restaurant.
registered were within the range 0.32–7.51 mg m\(^{-3}\).
In a previous study conducted on professional cooks in similar restaurants in Norway, the arithmetic mean mass concentration of total particles for all the restaurants was 0.62 mg m\(^{-3}\), and the highest measured level was 6.6 mg m\(^{-3}\) (Svendsen et al., 2002). These measurements were made during 1.5–2.5 h of a work shift in 19 different restaurants.

The mean mass concentration of total particles measured under experimental conditions in the breathing zone of the cook was 4.0 mg m\(^{-3}\), and all registered levels were within the range 0.93–8.82 mg m\(^{-3}\) (A. K. Sjaastad and K. Svendsen, unpublished data). In another previous study performed in the laboratory kitchen (Sjaastad and Svendsen, 2008), we measured a mean mass concentration of total particles in the breathing zone of the cook at 2.17 mg m\(^{-3}\), and all the registered levels were in the range of 0.12–12.07 mg m\(^{-3}\). This indicates that the results from the laboratory kitchen are representative for real-life exposure conditions in professional restaurants.

Geometric mean levels of particles (PM10) of 0.08 mg m\(^{-3}\) have recently been reported in a Chinese study from 23 different restaurant kitchens. The particles were measured stationary with a laser aerosol monitor in the breathing zone of the cooks. The measured mean level of total PAH in the same study was 0.028 μg m\(^{-3}\) (Pan et al., 2008). When comparing these results with those of our study, it can be assumed that the exposure levels in Chinese restaurant kitchens are not much higher than in Western restaurants.

In the present study, we expected to find a relationship between the level of total particles and the level of PAHs and aldehydes, but this was not the case. It seems that the levels of PAHs and higher aldehydes depend on other factors than just the level of particles in the room. In fact, the restaurant with the lowest level of total particles had the highest level of aldehydes, and nearly the same level of PAHs as the restaurant with the highest level of total particles. These inconsistencies may be explained by other factors such as the use of a gas stove or some special kind of food that were prepared. These features have to be looked at more closely in future studies.

Although the levels of exposure for each measured component lie below the limit values for those components that have a TLV, cooking fumes contain components such as mutagenic higher aldehydes and HCAs (not measured here) with unknown dose–response relationships. This should be taken into consideration when risk is assessed, with the goal to reduce exposure as far as possible.

CONCLUSIONS

To our knowledge, the occupational exposure of cooks to PAHs and mutagenic aldehydes measured with personal samples have so far not been presented. The measurements in the present study are assumed to be representative for the exposure that professional cooks are subject to during frying of beefsteak in a Western European restaurant.

Studies on both Asian (Zhong et al., 1999; Metayer et al., 2002) and Western (Svendsen et al., 2003) style cooking have indicated adverse health effects (lung cancer included) from exposure to cooking fumes. According to the present results, working as a cook in a Norwegian à la carte restaurant with some manual panfrying 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 adverse health effects of cooking fumes. A TLV for cooking fumes is desirable, until then exposure to cooking fumes has to be reduced as far as possible.

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