Dissertation

Characterization of Polycyclic Aromatic Hydrocarbons and their Derivatives on Indoor Biomass Burning in Rural Thailand

タイ農村の屋内バイオマス燃焼に由来する多環芳香族炭化水素とその誘導体の特性解析

Graduate School of Medical Sciences

Kanazawa University

Division: Pharmaceutical Science

Laboratory: Hygienic Chemistry

School registration No.: 1429012002

Name: Walaiporn Orakij

Primary Supervisor name: Akira Toriba

Dissertation Summary

My dissertation focuses on exposure of Thai rural residents to polycyclic aromatic hydrocarbons (PAHs) and their nitro-derivatives (NPAHs) from indoor air emissions of biomass burning. Indoor air pollution plays a significant role on human health to most people who spend their major time in indoor environment, especially in developing countries. The use of biomass fuels for domestic energy is the largest source of indoor air pollution on a global scale. Airborne particulate matters emitted from the incomplete combustion of biomass burning contain hazardous organic pollutants such as PAHs and NPAHs which are carcinogenic to humans and can impact indoor air quality and human health. Lung cancer incidence in northern Thailand is higher than that in other region areas of the country. The facts motivated me to identify important factors resulting in the high incidence rate of lung cancer in northern Thailand. This study focused on combustion sources such as biomass fueled cooking in daily life. My dissertation consists of two studies: (1) characterization of atmospheric PAHs and NPAHs from indoor biomass fueled cooking in rural Thailand; (2) evaluation of personal exposure of rural residents to fine particulate matters ($PM_{2.5}$), PAHs, and NPAHs in northern Thailand.

The first study of rural households in Chiang Mai investigated indoor air pollution from open-fire cooking with wood as the fuel. Severe $PM_{2.5}$, PAHs, and NPAHs contamination of the indoor air was observed during cooking periods. Time-dependent changes in $PM_{2.5}$ counts inside two study houses demonstrated that $PM_{2.5}$ level increased during cooking periods. The indoor PAH levels recorded in this study were higher than those found in similar studies of homes using biomass. The indoor to outdoor concentration ratios and diagnostic ratio using PAHs and NPAHs or carbonaceous fractions also demonstrated the large contribution of biomass burning to indoor air pollution. The composition profiles of PAHs and NPAHs showed that benz[*a*]anthracene, benzo[*k*]fluoranthrene, and benzo[*a*]pyrene made the greatest contribution to total PAHs, while 9-nitroanthracene made the greatest contribution to total NPAHs. The correlations of PAHs and NPAHs with levoglucosan (LG) as a tracer for biomass burning ($p < 0.01$) confirmed that the main source of PAHs and NPAHs was biomass combustion. The carcinogenic risk from the indoor air exceeded the guidelines for human health, suggesting that inhalation exposure to

emissions of biomass burning through open-fire for cooking may increase the risk of lung cancer in this area.

The second study observed characterization of personal inhalation exposure to $PM_{2.5}$, PAHs and NPAHs, and the cancer risk assessment of rural residents in Lampang for the first time. The levels of the monitored components for the subjects were higher than those from stationary samplings, suggesting the unreliability of estimating personal exposure from microenvironments in subjects' lives using only the results of stationary sampling. The atmospheric environment in the residential area contributed less to PAH concentrations because these were strongly affected by individual exposure from microenvironments such as indoor air. The smoking behavior of the residents was not reflected in their exposure to PAHs and NPAHs compared to other sources. Cooking activity was the most important factor concerning exposure to PAHs. The diagnostic ratios for PAHs and NPAHs, 1-nitropyrene/pyrene and benzo[*a*]pyrene/benzo[*ghi*]perylene, were used to identify the combustion sources. Urban ambient air was dominated by vehicle exhaust, whereas exposure to residents was affected by sources related to their personal lifestyle in addition to the atmospheric environment during haze periods. Personal inhalation cancer risks for all rural subjects during the study period exceeded the guideline value set by the USEPA, suggesting that the residents have a potentially increased cancer risk. In particular, the subjects who cooked using charcoal open fires showed the highest cancer risk.

All of these populations in the both study sites need to reduce exposure to severe indoor air pollution. Ways of improving indoor air quality in households that use biomass as fuel should be found to reduce exposure and prevent health problems arising in the future. Residential environments may be improved by the adoption of high-efficiency wood stoves, the installation of ventilation system, and the transition to cleaner fuels such as LPG or electricity.

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List of Abbreviations

Ant	Anthracene
BaA	Benz[*a*]anthracene
BaP	Benzo[*a*]pyrene
BbF	Benzo[*b*]fluoranthene
BghiPe	Benzo[*ghi*]perylene
BkF	Benzo[*k*]fluoranthene
BSTFA	*N*,*O*-Bis(trimethylsilyl) trifluoroacetamide
CAA	Clean Air Act
Chr	Chrysene
CO_2	Carbon dioxide
COPD	chronic obstructive pulmonary disease
DBA	Dibenz[*a*,*h*]anthracene
DCM	Dichloromethane
DHHS	The Department of Health and Human Services
DMSO	Dimethyl sulfoxide
EC	Elemental carbon
EPA	Environmental Protection Agency
FL	Fluorescence
Flu	Fluoranthene
GC	Gas Chromatography
HPLC	High Performance Liquid Chromatography
IARC	The International Agency for Research on Cancer
IDP	Indeno[1,2,3-*cd*]pyrene
LG	Levoglucosan
LPG	Liquid petroleum gas
MS	Mass Spectrometry
NA	Nitroanthracene
NAAQS	National Ambient Air Quality Standards
NBaA	Nitrobenz[*a*]anthracene
NBaP	Nitrobenzo[*a*]pyrene

NC	Nitrochrysene
NFR	Nitrofluoranthene
NP	Nitropyrene
NPAH	Nitropolycyclic aromatic hydrocarbon
NPh	Nitrophenanthrene
NPL	The National Priorities List
OC	Organic carbon
PAH	Polycyclic aromatic hydrocarbon
PCD	Pollution Control Department
PM	Particulate matter
Pyr	Pyrene
SD	Standard deviation
TC	Total carbon
TEFs	Toxic equivalent factors
TSP	Total suspended particulate matter
US EPA	United States Environmental Protection Agency
WHO	World Health Organization

CHAPTER 1

Introduction

1.1 Background information

Indoor air pollution exposure plays a significant role on human health due to most people spend their major time in indoor environments, particularly those women, young children, and elderly. Sources of indoor air pollution in the residential scale include emissions from cooking (both from fuel and food) and heating system, tobacco smoke, cleaning, consumer product emissions, and infiltration of outdoor air pollution. The use of solid fuels for domestic energy seems to be the most important source of indoor air pollution globally. Approximately half the world's populations, almost all in rural areas in developing countries, still rely on solid fuels (e.g., wood, charcoal, animal dung, crop residues, and coal) for their daily cooking and heating. These fuels are typically burnt indoors with low efficiency stoves, resulting in severe air pollution from incomplete combustion. Other sources of indoor air pollution in developing countries in addition to the use of solid fuels through low quality stove include smoke from nearby houses, usage of kerosene lamps, forest fires, burning of agricultural land and household waste, and emissions from industrial plant and vehicle.

Improvements of indoor air environments in developed countries have improved due to a shift from biomass fuels (such as wood) to cleaner energy sources (such as LPG and electricity). However, in developing countries, households often continue to use biomass fuels due to lack access to clean or modern energy, especially, in rural area. Concentrations of indoor air pollutants vary considerably in each microenvironment and are dependent on several factors such as house structure, ventilation condition, and combustion condition. Hence, a detailed understanding of combustion sources, emission factors and indoor levels of pollutants in different microenvironments is very important to improve the estimation accuracy for exposure and reduce human exposure and health risk.

Indoor air emissions from biomass burning are in general hazardous to human health, but the most important pollutants are respirable particles, carbon monoxide, nitrogen oxides, sulfur oxides, benzene, formaldehyde, and polycyclic aromatic compounds (De Koning et al. 1985).

Exposure to the pollutants is responsible for premature deaths annually among children and adults from stroke, chronic obstructive pulmonary disease (COPD), ischaemic heart disease, and lung cancer (WHO 2016a). Consequently, indoor air quality has been considerable interest in recent years.

1.2 Airborne particulate matter

Particulate matter (PM) is a heterogeneous mixture of solid and liquid particles suspended in the air. PM is commonly composed of sulfates, nitrates, ammonium, inorganic ions (e.g., ions of calcium, sodium, magnesium, potassium, and chloride), organic carbon, elemental carbon, soil material, particle-bound water, organic compounds (e.g., polycyclic aromatic hydrocarbons and their derivatives), and metals (e.g., cadmium, copper, nickel, zinc, and vanadium) (USEPA 2016a).

Sources of PM can be directly emitted (primary particles) into the environment or produced in the atmosphere (secondary particles). Secondary particles are formed from gaseous precursors (e.g., oxides of nitrogen, sulfur dioxide, ammonia, and non-methane volatile organic compounds) via chemical reaction. Both primary and secondary particles can be released from anthropogenic and natural sources. (WHO 2013). Anthropogenic sources include industrial and agricultural activities, solid fuel (biomass, coal, lignite, and heavy oil) combustion, erosion of the pavement by road traffic, and abrasion of brakes and tires. Natural sources include forest fires, living vegetation, volcanoes, dust storms, and sea spray (Kim et al. 2015).

PM has been classified by particle size (or aerodynamic diameter) which can describe its transport ability in the atmosphere or inhaling ability through a respiratory organism (Kim et al. 2015). Particles with a diameter between 2.5 and 10 μm ($PM_{2.5-10}$) are defined as coarse particles and a diameter less than 2.5 μm ($PM_{2.5}$) as fine particles (Anderson et al. 2012). Total suspended particles (TSP) means particles of any size below 30 μm in diameter suspended in the air. It is also intuitively true that as particles size greater than 30 μm remain suspended for a relatively short period of time before deposition when compared to smaller particles. (De Kok et al. 2006).

The 1970 Clean Air Act (CAA) was the first major American regulatory effort aimed at both studying and setting limits on emissions and air pollution (US EPA 2016b). The CAA, which was last amended in 1990, requires EPA to set National Ambient Air Quality Standards (NAAQS) for pollutants considered harmful to public health and the environment. These

standard set limits on six primary pollutants found in air include carbon monoxide, nitrogen dioxide, sulfur dioxide, ozone, lead, and PM. According to the latest guideline, the standards for $PM_{2.5}$ were set at 35 μg/m^3 for a 24 h average, and 12 μg/m^3 for an annual average meanwhile the PM_{10} standard for 24 h is 150 μg/m^3 (US EPA 2013). Moreover, WHO also recommended guideline values for $PM_{2.5}$ and PM_{10} at levels of 10 and 20 for annual mean and 25 and 50 for 24 h mean, respectively (WHO 2016b).

Several studies have reported that exposure to PM is associated to numerous health effects including respiratory symptoms, exacerbation of chronic respiratory and cardiovascular diseases, decreased lung function, and premature mortality (Samoli et al. 2008; Halonen et al. 2009; Guaita et al. 2011; Perez et al. 2012). The toxicity of PM is related to their size and the chemicals which are absorbed on them. Many toxic compounds are associated to PM such as polycyclic aromatic hydrocarbons (PAHs) and nitro polycyclic aromatic hydrocarbons (NPAHs) (Albinet et al. 2007).

1.3 Toxicity of PAHs and NPAHs

PM contains several inorganic and organic compounds including PAHs and their derivatives such as NPAHs. They are of great concern because of their carcinogenicity and mutagenicity to human (Ames et al. 1975). About 85% of PAHs and NPAHs in atmosphere were mainly associated with fine particles or $PM_{2.5}$ (Ringuet et al. 2012). Generally, they are emitted to the atmosphere through incomplete combustion or pyrolysis of organic materials such as coal, petroleum, oil, and biomass (Harvey 1997). Fig. 1.1 shows typical PAHs and NPAHs observed in the atmosphere.

PAHs are a group of several hundred individual organic chemicals consisting of carbon and hydrogen atoms with a fused ring structure including at least two benzene rings. Some PAHs have been classified as carcinogenic materials by many organizations including US EPA, IARC, and the Department of Health and Human Services (DHHS). Table 1.1 shows the carcinogen classification of 16 priority PAHs by the IARC, the US EPA, and the DHHS (Lee and Vu 2010).

NPAHs are a class of aromatic compounds with at least one nitro-(NO_2) functional group on the aromatic ring of a PAH. The potent mutagenic NPAHs were observed in the organic extracts of atmospheric particulate matter (Pitts et al. 1977) and also observed in the extracts of diesel exhaust in few years later (Rappaport et al. 1980). This observation has led to increased

interest in the environmental occurrence of these compounds. Some NPAHs are more toxic than their parent PAHs. Thus, they have been pointed out as direct-acting genotoxicity (Oanh et al. 2002; Vicente et al. 2016).

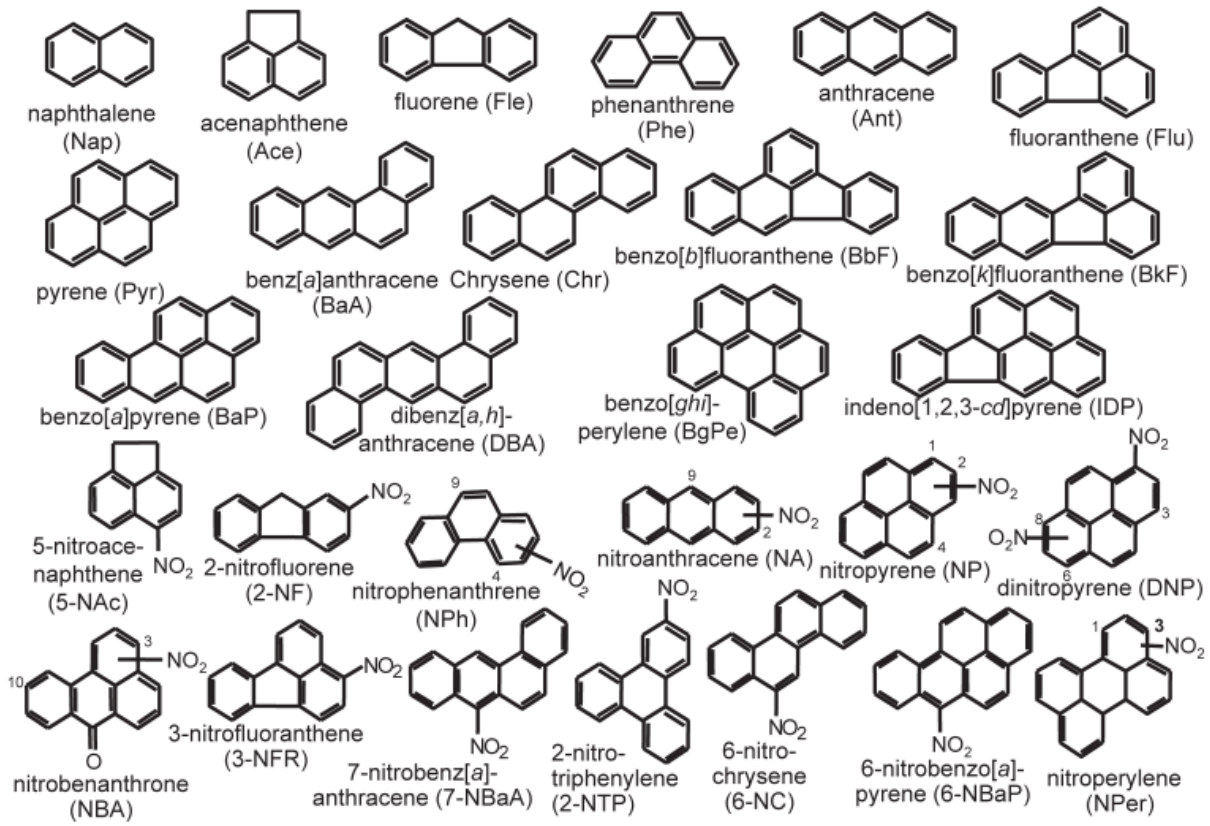


Fig. 1.1 Typical PAHs and NPAHs observed in the atmosphere (Hayakawa 2016)

Table 1.1 Sixteen priority PAHs were classified by IARC in comparing those by the DHHS and the US EPA

PAHs	No. of ring	US EPA	IARC	DHHS
Naphthalene	2	- [a)]	2B	-
Acenaphthylene	3	Not classifiable	-	-
Acenaphthene	3	-	3	-
Fluorene	3	Not classifiable	3	-
Phenanthrene	3	Not classifiable	3	-
Anthracene	3	Not classifiable	3	-
Fluoranthene	4	Not classifiable	3	-
Pyrene	4	Not classifiable	3	-
Chrysene	4	Probably carcinogen	2B	-
Benz[*a*]anthracene	4	Probably carcinogen	2B	Animal carcinogen
Benzo[*b*]fluoranthene	5	Probably carcinogen	2B	Animal carcinogen
Benzo[*k*]fluoranthene	5	Probably carcinogen	2B	-
Benzo[*a*]pyrene	5	Probably carcinogen	1	Animal carcinogen
Dibenz[*a,h*]anthracene	5	Probably carcinogen	2A	Animal carcinogen
Benzo[*ghi*]perylene	6	Not classifiable	3	-
Indeno[1,2,3-*cd*]pyrene	6	Probably carcinogen	2B	Animal carcinogen

IARC classification: Group 1 (carcinogenic); 2A (probably carcinogenic); 2B (possibly carcinogenic); 3 (not classifiable). Data from Lee and Vu (2010). [a)] no information

IARC has classified PAHs especially, Benzo[*a*]pyrene (BaP) as Group 1, as it is carcinogenic to humans (IARC 2010). This has prompted it to be a widely studied PAHs because it can be used as marker for carcinogenic risk levels in environmental studies (Ramirez et al. 2011). Several countries and organizations have set up protective health standards for BaP based on the carcinogenic potential of inhaled particulate PAHs in aerosols. Example of such is the guideline value of BaP is 1 ng/m^3 proposed by the 4th Daughter Directive (2004/107/EC); 0.25 ng/m^3 in USA; 0.25 ng/m^3 in UK; 10 ng/m^3 in China (Taylor and Nakai 2012). Meanwhile, 1-nitropyrene (1-NP) has been classified in group 2A; probably carcinogenic to humans by IARC and several other PAHs and NPAHs in group 2B; possibly carcinogenic to humans (Hayakawa

2016). Exposure to PAHs and their derivatives via various channels such as inhalation and intestinal and dermal absorption is associated with increased risk of various diseases such as lung cancer, respiratory and cardiovascular diseases (IARC 2010; Jarvis et al. 2014).

1.4 Sources, formation, and characteristics of PAHs

PAHs can be formed through pyrogenic (or pyrolytic), petrogenic, and biological processes. Pyrogenic PAHs are generated when organic substances are exposed to high temperatures under oxygen-deficient conditions. The examples of pyrolytic processes are from thermal cracking of petroleum residuals and the incomplete combustion of fuels or biomass. Meanwhile, petrogenic PAHs are formed during crude oil maturation. Major sources of petrogenic such as oceanic and fresh water oil spills, underground and aboveground storage tank leaks, and the accumulation of vast numbers of small releases of gasoline, motor oil, and related substances associated with transportation. Moreover, PAHs can be synthesized through biological activities of certain plants and bacteria. It can also be formed during degradation of vegetative matter (Abdel-Shafy and Mansour 2016).

The US EPA has classified sixteen of the PAHs as priority pollutants based on their toxicity (US EPA 1982). The listed 16 PAHs (Table 1.1) were considered to be included in the priority list because of the four reasons as followed: (1) more information is available on them than on others; (2) they are suspected to be more harmful than others and they exhibit harmful effects that are representative of PAHs in general; (3) there is a greater chance for exposure to these PAHs than to the others; and (4) all the analyzed PAHs exhibited the highest concentrations at the National Priorities List (NPL) hazardous waste site (ATSDR 1995).

The general characteristics of PAHs are high boiling and melting points (thus they are solid), low vapor pressure, and very low aqueous solubility. Aqueous solubility of PAHs decreases in each additional ring. PAHs are very soluble in organic solvent as they are highly hydrophobic. Moreover, they are mostly colorless, white, or pale yellow solids (Abdel-Shafy and Mansour 2016). Most PAHs are persistent organic pollutants in the environment. Many of them are chemically inert. PAHs can be dispersed through atmospheric transport and exist almost everywhere due to widespread sources and persistent characteristics. However, PAHs can be photochemically decomposed under strong ultraviolet light or sunlight, and thus some PAHs can be decomposed through the atmospheric reactions. Also, PAHs can react with ozone, hydroxyl

radicals, nitrogen and sulfur oxides, and nitric and sulfuric acids, which affect the environmental fate of PAHs (Lee and Vu 2010).

In general, PAHs are distributed in both gaseous and particulate phases, depending upon their volatility (vapor pressure) and ambient temperature. Low molecular weight or two-, three-rings PAHs are emitted dominantly in the gaseous phase, while high molecular weight PAHs or harmful PAHs, with five or more rings, are emitted mainly in the particulate phase (Lee and Vu 2010). The majority of compounds, especially those with three or four rings, are considered as semi-volatile and such compounds partition between the gas and particle phases. Naphthalene (NaP) having two aromatic rings exist almost entirely as gaseous phase (Keyte et al. 2013). The PAH emission profile for a given source depends on the PAH formation processes. During low temperature processes such as biomass burning, low molecular weight PAHs are usually formed, whereas high temperature processes, such as the combustion of fuels in engines, emit higher molecular weight PAH compounds (Tobiszewski and Namiesnik 2012).

Human beings are exposed to PAH mixtures in ambient air. Long-term exposure to high concentrations of PAHs is associated with adverse health problems. Since some PAHs are considered carcinogens, inhalation of PAHs in particulates is a potentially serious health risk linked to an excess risk of lung cancer. Thus, studies on PAHs in PM such as PM_{10} and $PM_{2.5}$ in ambient air have become the focus of attention in recent years (Lee and Vu 2010).

1.5 Sources, formation, and characteristics of NPAHs

NPAHs can be originated as direct (primary) or indirect (secondary) products of incomplete combustion. The primarily produced NPAHs are formed through nitration during incomplete combustion processes such as diesel engines (Schuetzle et al. 1982; Paputa-Peck et al. 1983; IARC 1989), gasoline vehicles (Cecinato and Zagari 1997), coal combustion (Tang et al. 2005; Hattori et al. 2007; Tang et al. 2011), and biomass burning (Ding et al. 2012; Shen et al. 2012; Cheusaard et al. 2014). The most abundant NPAH produced from diesel exhaust is 1-NP, which has not been detected in any gas-phase reactions (Paputa-Peck et al. 1983). 1-NP is also emitted by gasoline powered vehicles (Gibson 1983) and coal burning power plants (Harris et al. 1984). Other NPAHs such as 3-nitrofluoranthrene (3-NFR) and 9-nitrophenanthrene (9-NPh) were also observed in particle samples directly emitted from the combustion chamber of diesel engines (Paputa-Peck et al. 1983). Secondarily produced NPAHs are formed in the atmosphere

by the reactions of their parent PAHs with hydroxyl (OH) radicals during the day or with nitrate (NO_3) radicals in the presence of NOx during the night. These reactions may only occur in the gas phase, but the NPAHs produced are quickly adsorbed on the PM (Atkinson and Arey 1994). 2-Nitrofluoranthene (2-NF) and 2-nitropyrene (2-NP) are representatives of secondarily formed NPAHs (Fan et al. 1996). 2-NP is produced from the reaction of OH radicals with pyrene in the presence of NO_2, whereas 2-NF is produced from gas-phase reactions of either OH or NO_3 radicals with fluoranthene in the presence of NO_2 (Feilberg et al. 2001). The main formation process of NPAHs in the atmosphere has been suggested that it is formed from the gas-phase reactions of parent PAHs with four rings or less (Atkinson et al. 1990).

NPAHs occur in the environment as a mixture together with their parent PAHs and hundreds of other organic compounds. They are usually present in much smaller quantities than PAHs (IPCS 2003). NPAHs are semi-volatile which have the potential to equilibrate between the vapor phase and adsorption on particulate matter in the atmosphere, depending on their vapor pressure under ambient conditions and the airborne particle loading, as for PAH. NPAHs with two-rings were found only in the vapor phase, and three-rings were detected in both the gas and particulate phases. On the other hand, NPAHs with four or more rings were mainly distributed in particulate phase (Dimashki et al. 2000).

NPAHs can be 100,000 times more mutagenic and 10 times more carcinogenic compared to their parent-PAHs since they are direct-acting mutagen, while the PAHs require an initial enzymatic activation (Durant et al. 1996). Some NPAHs exhibited high direct-acting mutagenic potency in the *Salmonella* bacterial mutagenicity assay (Pitts et al. 1978) and in forward mutation assays based on human B-lymphoblastoid cells (Durant et al. 1996) and on human lung tissue (Tokiwa et al. 1998). Consequently, NPAHs is of growing interest, particularly in the environmental analytical community despite at much lower concentrations than those of their parent PAHs (Bamford et al. 2003a). There are evidences for little ($\leq$10%) direct-acting mutagenicity of some NPAHs, including nitrofluoranthenes and nitropyrenes (Arey et al. 1988; Atkinson et al. 1988; Atkinson and Arey 1994). Furthermore, the direct-acting mutagenicity of ambient air samples, which were collected at seven sites in California, did not correlate with the PAH concentrations but rather correlate with the 2-NP concentrations (Atkinson et al. 1988; Atkinson and Arey 1994). Since 2-NP is formed in the atmosphere from the OH radical-initiated reaction of gas-phase pyrene, the remainder of the ambient air direct-acting mutagenicity may be

associated with the OH radical reaction products of organic compounds and may be due to the mutagenicity of the 2- to 4-ring PAH reaction products other than the NPAH. For example, it is interesting that the NPAH account for 5% or less of the products of the gas-phase OH radical-initiated reactions of the 2- to 4-ring PAH and 10% or less of the ambient air particle phase, direct-acting mutagenicity (Atkinson and Arey 1994)

1.6. Wood smoke emission and tracer

Wood is a renewable energy source and widely used for domestic cooking and heating especially, developing countries. Wood smoke emission can cause short- and long-term health effects such as acute respiratory infections, asthma, chronic obstructive pulmonary disease, tuberculosis, blindness, headache, and reduced birth weight (Bari et al. 2011). Wood consists of primarily of two polymer such as cellulose (approximately 50-70% by weight) and lignin (approximately 30% by weight). For other biomass fuels, such as grasses and wheat stubble, also compose these polymers but their relative proportions are different (Naeher et al. 2007).

Levoglucosan (1,6-anhydro-β-D-glucopyranose; LG), is a sugar anhydride (Fig. 1.2) released during pyrolysis of cellulose and comprises atmospheric aerosol with other stereoisomeric monosaccharide anhydrides including mannosan (1,6-anhydro- β-D-mannopyranose) and galactosan (1,6-anhydro-β-D-galactopyranose). Among these monosaccharide anhydrides, LG is the most abundant in the atmosphere due to its emission amount compared to the others (Zdrahal et al. 2002). LG can be present in other sources of biomass burning such as tobacco smoke, grasses, and rice straw. However, LG has been considered as a unique tracer for biomass burning due to its source specificity and stability in atmosphere (Naeher et al. 2007; Urban et al. 2012).

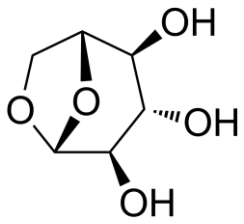


Fig. 1.2 Chemical structure of levoglucosan

1.7 Carbonaceous aerosol

Atmospheric carbonaceous particles have been received more attention in recent years because of their influence on climate and adverse health effects (Han et al. 2009). Carbon fraction or total carbon (TC) is a prominent constituent of atmospheric PM. PAHs and NPAHs are distributed in a fraction of TC (Poschl 2005). TC includes organic carbon (OC) and elemental carbon (EC, sometimes called black carbon) (Han et al. 2009). Both OC and EC are formed from emissions of coal, fossil fuels, biomass, and industrial activities. It has been estimated that EC may be the second most important anthropogenic constituent contributing to global warming, after CO_2 since EC has high absorption (Jacobson 2000). Globally, emission of EC originates from burning of biomass and biofuel about 62%, and fossil fuel combustion about 38% (Han et al. 2010). EC can be subdivided into two categories: char-EC and soot-EC. Char-EC is generally black colored material left as combustion residuals. It is formed at low combustion temperatures (<600°C) with morphological features similar to its source material. In contrast, soot-EC is formed at high temperatures (>600°C) via gas-to-particle conversion (Bond et al. 2004). Char-EC is larger particle (diameter range generally from 1 to 100 μm) than soot-EC (diameter range 0.1-1 μm). The differences of char- and soot-EC in chemical and physical properties result in different optical and radiative properties (Han et al. 2007).

1.8 Indoor air pollution from household fuel combustion and health impact

There are numerous sources of indoor air pollutions in residential scale such as cooking, heating, smoking, and cleaning. Outdoor pollutants can also be a source of indoor air pollutions through infiltration and ventilation depending on house structure and ventilation conditions (Ma and Harrad 2015) According to a WHO report, people in China, India, South East Asia (>75%), and South America and Africa (50-75%) use combustion of solid fuels (such as wood, animal dung, charcoal, crop wastes, and coal) for daily cooking (Zhu et al. 2009). These fuels are usually burnt with low combustion efficiency stoves in poor ventilation kitchens for domestic cooking resulting in high levels of indoor air pollution as shown in Fig. 1.3 (WHO 2016c).

The indoor pollution emitted when biomass is burnt contains several hazardous chemicals including PAHs and NPAHs which related to human health (Naeher et al. 2007; Taylor and Nakai 2012). Currently, there were about 4.3 million premature deaths annually among children

and adults from illness attributable to household air pollution due to cooking on inefficient biomass stoves (WHO 2014b). Among these deaths, 34% from stroke, 26% from ischaemic heart disease, 22% from chronic obstructive pulmonary disease (COPD), 12% resulted from pneumonia, and 6% from lung cancer (WHO 2016a).

Moreover, IARC has classified indoor air pollution from combustion of biomass fuel (mostly wood) are probably carcinogenic to humans (Group 2A) (IARC 2006). These suggested that indoor air pollution from solid fuel combustion in rural region is recognized as the most important source contributor to the global burden of disease today (Bhargava et al. 2004).

Recently, studies on indoor air pollution from household fuel combustion have been conducted in various developing countries including Philippines (Saksena et al. 2007), China (Ding et al. 2012; Chen et al. 2016), Bangladesh (Dasgupta et al. 2006), India (Bhargava et al. 2004), and Kenya (Boleij et al. 1989).

Fig. 1.3 High levels of indoor air pollutants in a poorly ventilated household where biomass was used as cooking fuel with low efficiency stoves (WHO 2016c)

1.9 Motivation of the dissertation

Thailand is classified as a developing country. Majority of Thai people, especially in rural area, are working in agriculture sector. The use of biomass fuels, mainly wood or corn residues with traditional open fire stoves for daily cooking in rural household is common in Thailand. It has been noted earlier that exposure to indoor air pollution from household fuel combustion associated with lung cancer mortality. The indoor air pollution may be one factor resulting in high incidence rate of lung cancer in northern Thailand (Kamnerdsupaphon et al. 2008; Wiwatanadate 2011).

Several studies have been investigated on ambient air pollution in northern Thailand (Pengchai et al. 2009; Wiriya et al. 2013; Phoothiwut and Junyapoon 2013; Chuesaard et al. 2014) meanwhile few study on indoor air pollution. A study related to indoor air pollution has been reported but they mainly focused on public building in Bangkok, capital city of Thailand (Klinmalee et al. 2009). Although indoor air quality on residential scale caused by the use of biomass fuel for cooking has been of particular interest in recent years, there is a lack of information on indoor air pollution in this country.

Therefore, studies on contribution of indoor air pollution from biomass burning to its health effects in rural residents would help provide information for air quality management and evaluating the health risk from exposure to PAHs and NPAHs. The obtained results should lead to improve air quality and population health in developing countries. In this study, we focus on emission of biomass burning as a source of carcinogenic compounds in rural household, Thailand. We investigated the levels of pollutants emitted from biomass burning both inside and outside of the houses during cooking and noncooking periods. Characterization of contribution pattern of indoor PAHs and NPAHs was conducted. Furthermore, carcinogenic potential of exposure to PAHs and NPAHs was also estimated. As strong carcinogenic PAHs and NPAHs mostly existed in particulate phase, only atmospheric particulate phases were selected for investigations in this study. The structures of targeted ten PAHs and eleven NPAHs are shown in Fig. 1.4 and 1.5, respectively.

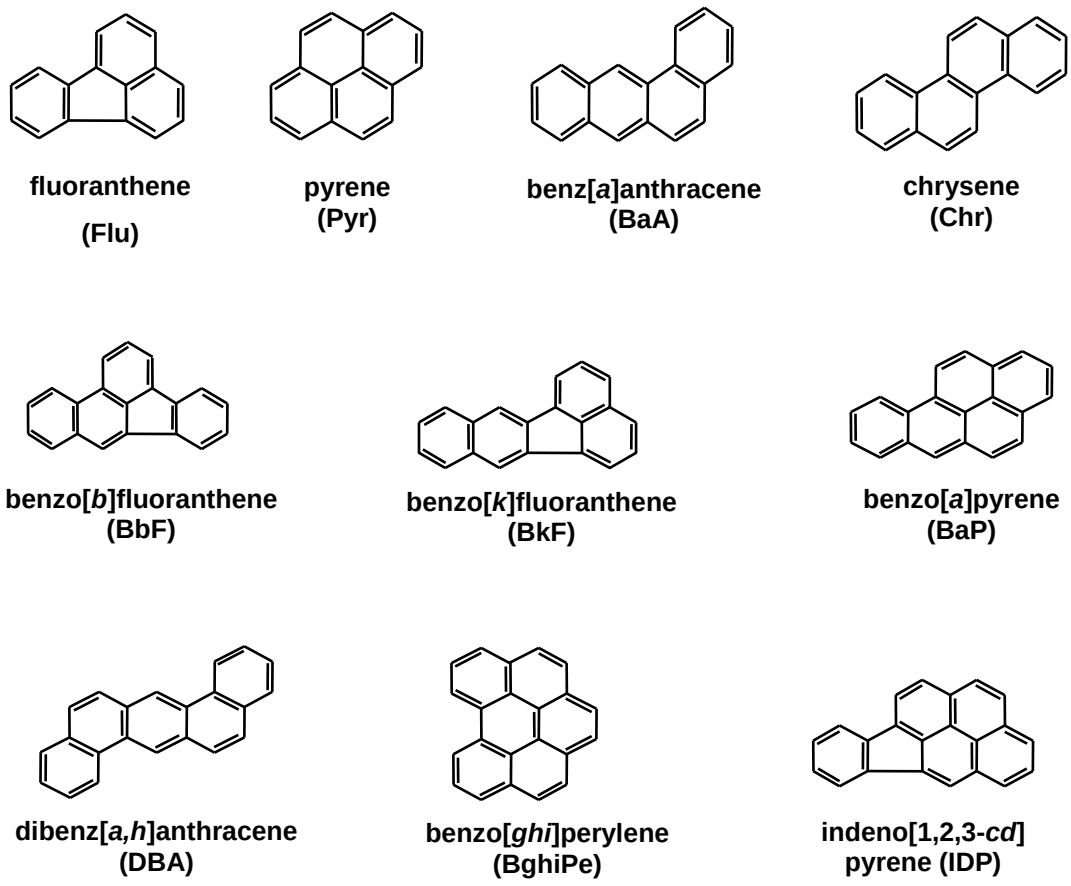
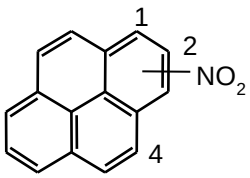
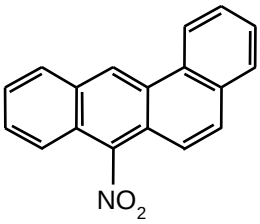


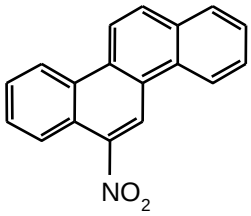
Fig. 1.4 Structure of targeted ten PAHs



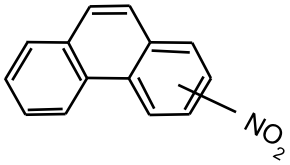
nitropyrene
(NP)



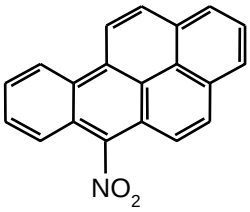
7-nitrobenz[*a*]anthracene
(7-NBaA)



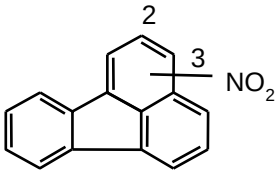
6-nitrochrysene
(6-NC)



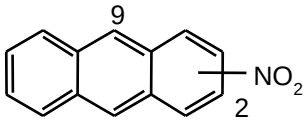
9-nitrophenanthrene
(9-NPh)



6-nitrobenzo[*a*]pyrene
(6-NBaP)



nitrofluoranthene
(NFR)



nitroanthracene
(NA)

Fig. 1.5 Structure of targeted eleven NPAHs

CHAPTER 2

Polycyclic aromatic hydrocarbons and their nitro-derivatives from indoor biomass fueled cooking in rural Thailand

2.1 Introduction

The IARC has been classified PM as carcinogenic to humans (Group 1). The IARC evaluation showed an increasing risk of developing lung cancer as the level of exposure to air pollution and particulate matter increase (IARC 2013). Air pollution is now the single biggest environmental health risk, according to the WHO. In a 2012 report, the WHO attributed around 7 million deaths to air pollution, or approximately one in eight of all deaths. Approximately 4.3 million deaths were attributed to indoor air pollution mainly due to the use of wood, coal, or biomass fuel for cooking inside the dwelling (WHO 2014a, 2014b).

Biomass fuels, mainly comprising wood or crop residues, are important primary energy sources. They contribute approximately 13% of the total final fuel consumption worldwide and are used for cooking and heating by 39% of the global population. They are, especially, significant in the rural household sector of developing countries (Shen et al. 2012). The IARC has classified the indoor emissions arising from household combustion of biomass fuel (mainly wood) as falling within Group 2A, probably carcinogenic to humans (IARC 2006). Residential biomass combustion is one of the most important sources of air pollution that, release hazardous chemicals including polycyclic aromatic hydrocarbons (PAHs) and their derivatives (NPAHs), which are known carcinogens and mutagens (Oanh et al. 2002; Claxton et al. 2004; Shen et al. 2011; Vicente et al. 2016). Among PAHs, BaP has been classified as carcinogenic to humans (Group 1) by the IARC (IARC 2010). BaP is among the most widely studied PAHs, as it can be used as a marker for carcinogenic risk levels in environmental studies (Ramirez et al. 2011). Some NPAHs are assumed to be more toxic than their parent PAHs and have been identified as direct-acting genotoxins (Oanh et al. 2002; Vicente et al. 2016).

Exposure to PAHs and their derivatives via inhalation and intestinal and dermal absorption is associated with increased risk of a range of diseases including lung cancer, respiratory diseases, and cardiovascular diseases (IARC 2010; Jarvis et al. 2014). Lung cancer is

one of the most significant health problems in Thailand and has been the most common cause of death since 1999 (Kamnerdsupaphon et al. 2008). A higher lung cancer rate has been reported in northern Thailand than in other areas (Kamnerdsupaphon et al. 2008; Wiwatanadate 2011). However, there has been little research on indoor air pollution in rural areas in northern Thailand, where biomass is widely used as the cooking fuel. Many villagers still use traditional open fire for cooking and use firewood or corn residues as fuel. Little information is currently available on the impact of exposure to PAHs and NPAHs from indoor biomass burning, especially, for the case of rural households in Thailand.

This study investigated the level, composition, and carcinogenic risk of exposure to PAHs and NPAHs from residential biomass combustion based on a monitoring of two houses for 2 days. A better understanding of indoor air pollution from biomass combustion and its health effects in rural areas of Thailand will provide information for air quality management, help evaluate the health risk of PAHs and NPAHs, and suggest improvements in the health of rural populations in developing countries.

2.2 Materials and methods

2.2.1 Chemicals

The EPA 610 PAH mixture (including fluoranthene (Flu), pyrene (Pyr), benz[*a*]anthracene (BaA), chrysene (Chr), benzo[*b*]fluoranthene (BbF), benzo[*k*]fluoranthene (BkF), benzo[*a*]pyrene (BaP), dibenz[*a,h*]anthracene (DBA), benzo[*ghi*]perylene (BghiPe) and indeno[1,2,3-*cd*]pyrene (IDP)), 2- and 9-nitroanthracene (2- and 9-NA), 1-, 2-, and 4-nitropyrene (1-, 2-, and 4-NP), 2- and 3-nitrofluoranthene (2- and 3-NFR), levoglucosan (1,6-anhydro-β-D-glucopyranose) (LG), and pyridine were purchased from Sigma-Aldrich (St. Louis, MO, USA). 6-nitrochrysene (6-NC), 7-nitrobenz[*a*]anthracene (7-NBaA) and 6-nitrobenzo[*a*]pyrene (6-NBaP) were obtained from Chiron AS (Trondheim, Norway). 9-Nitrophenanthrene (9-NPh) was purchased from AccuStandard, Inc. (New Haven, CT, USA). Three deuterated PAHs (Pyr-d_{10}, BaA-d_{12} and BaP-d_{12}), deuterated 6-NC (6-NC-d_{11}), and a stable isotope labeled LG (LG-$^{13}C_6$) were obtained from the Cambridge Isotope Lab. Inc. (Andover, MA, USA). 1,4-dithioerythritol was from Wako Pure Chemicals (Osaka, Japan), and a silylating agent, *N,O*-Bis (trimethylsilyl) trifluoroacetamide (BSTFA) with 1% trimethylchlorosilane (TMCS) was purchased from Sigma-

Aldrich (Supulco). All solvents and other chemicals were high performance liquid chromatography (HPLC) or analytical-reagent grade.

2.2.2 Study site and PM Sampling

The study houses were located in the rural area of Pong Yeang, Mae Rim district (geographic coordinates: latitude 18°53′28.3″ North, longitude 98°49′39.0″ East, elevation 1,255.32 meters above sea level), Chiang Mai province, in the northern part of Thailand (Fig. 2.1). The site is approximately 30 km from the center of Chiang Mai, the provincial capital. The rural village Buakchan is a hill tribe village, which is surrounded by mountains. There are no major roads, industries, or other emission sources nearby. The village comprised 145 houses, with a population of 1,009. Biomass fuels such as wood are widely used for cooking.

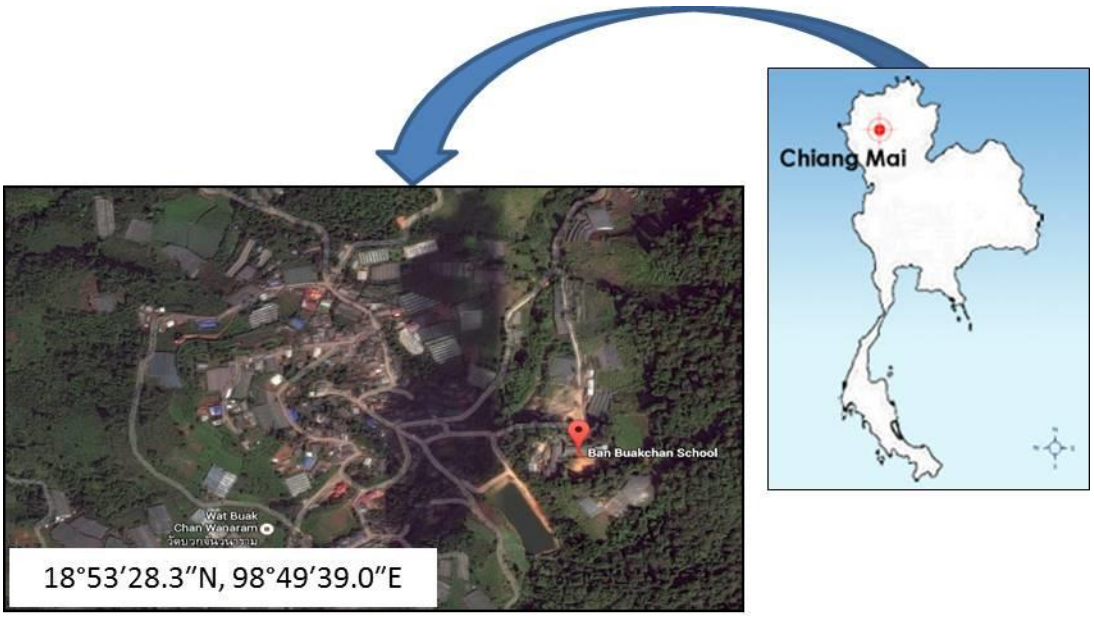


Fig. 2.1 Two study households located in a rural village Buakchan, Chiang Mai province, Thailand

Sampling was conducted in two houses. One house a large family of 17 people (House 1), and the other was a family of two (House 2), as shown in Fig. 2.2. House 1 was built of concrete blocks with a galvanized iron roof and had no windows. There were ventilation blocks at the upper part of the wall but no ventilation fan, thus ventilation of the kitchen was very poor. Two traditional open stoves fueled by wood were normally used for cooking. House 2 was a wooden structure and had better ventilation than House 1 despite of lacking a ventilation fan because of air exchange through the rough lattice-shaped material of the walls. Cooking in House 2 was done on an open fire. In both houses, meals were prepared twice a day. No member of either household smoked cigarettes.

Air sampling was conducted on two days, March 9-10 and 11-12, 2012. The atmospheric conditions during sampling period were characterized as dry season in Thailand. In general, northern Thailand has a quite high temperature with a mean of 28.1°C (range, 21.8°C–36.1°C, data from 1981-2010) and has a low precipitation from mid-February to mid-May. The meteorological conditions during sampling period are described in Table 2.1. Indoor (kitchen) and outdoor $PM_{2.5}$ samples were collected over 24 hours on both days. All samples were collected using a personal air sampler with an ATPS-20H impactor (Shibata Sci. Tech., Tokyo, Japan) connected to a portable MP-∑300 pump (Shibata) that provides an air flow of 1.5 L/min. Particles >10 μm in diameter were collected on a metal impaction plate coated with grease immediately downstream of the inlet. Particles 2.5-10 μm passed through the impaction plate and were collected on a 10 mm Fiberfilm filter (heat resistant borosilicate glass fiber coated with fluorocarbon, T60A20, Pall Life Sciences, Ann Arbor, MI, USA) with a 50% cutoff point of 10 μm ($PM_{2.5–10}$) that was placed on the second impaction stage. Particles 2.5 μm or less were collected on a 20 mm Fiberfilm filter with a 50% cutoff point of 2.5 μm ($PM_{2.5}$) located in the final stage of the sampler. In House 1, the total suspended particulate matter (TSP) was also collected on a 32 mm quartz filter for carbon analysis during cooking periods of the first day. A modified personal dust sensor (PDS-2, Shibata) equipped with an ATPS-20H impactor was used for real time $PM_{2.5}$ monitoring at 10 sec intervals. The indoor sampling equipments were placed in a basket on top of a cabinet, 5-6 m away from the cooking area (Fig. 2.3). For outdoor sampling, the other basket with equipments was hung from a crossbeam facing the yard at a height of approximately 2 m (Fig. 2.3).

House 1

House 2

Fig. 2.2 Two study houses in Buakchan village in Chiang Mai, Thailand. House 1 is a concrete house with two traditional open wood stoves. House 2 is a wooden house with an open fire place.

Table 2.1 Metrological conditions during the sampling period in the study region [a)]

Date	Temperature (°C)	Humidity (%)	Precipitation (mm)	Sea level pressure (hPa)	Wind speed (km/hr)
9-Mar, 2012	26 (17-35) [b)]	45 (18-72) [b)]	0	1007.81	2
10-Mar, 2012	26 (20-33)	52 (29-68)	0	1009.59	4
11-Mar, 2012	28 (24-31)	67 (51-83)	0	1012.18	7
12-Mar, 2012	28 (23-34)	68 (43-88)	0	1013.73	5

[a)] Data are available on http://thai.wunderground.com
[b)] Mean (minimum – maximum)

Fig 2.3 Indoor and outdoor sampling site of house 1 and house 2

The air samples from noncooking periods and the two meal preparation periods (dinner and breakfast) were collected separately. The noncooking period sampling was performed between 10:00 and 16:30. The $PM_{2.5}$ samples from the dinner and breakfast preparation periods were collected from approximately 16:30 to 20:30 and 20:30 to 10:00, respectively, as the cooking periods were not precisely the same each time. The samples collected from 20:30 to 10:00 were defined as breakfast period because the cooking accounted for almost PM generation during the sampling time. In total, 75 samples were obtained. These were stored at -20°C until analysis.

2.2.3 Sample preparation and analysis

The $PM_{2.5}$ filter samples were extracted using 5 mL of dichloromethane (DCM) under ultrasonication for 15 min. The extraction procedure was repeated three times to obtain a first fraction volume of 15 mL. The filters were then ultrasonically extracted a second time using 5 mL of a mixture of methanol and DCM (1/1, v/v) for 15 min to obtain the second fraction. The first and second fractions were evaporated to dryness and then redissolved in 1 mL and 0.5 mL of methanol, respectively.

The crude DCM extract (200 µL) was used for the determination of PAH and NPAH. The extract was evaporated until dry and then dissolved in hexane (5 mL). The hexane solution was treated with tandem cartridges of silica (Sep-Pak Plus cartridge, 690 mg) and aminopropyl silica (Sep-Pak Plus Light cartridge, 130 mg) (both from Waters Co., Milford, Massachusetts, USA). Prior to fractionation, each SPE cartridge was conditioned sequentially with DCM (10 mL), followed by hexane (10 mL), and the extract was then applied to the cartridges. SPE elution was performed using 20 mL of hexane to collect the PAH fraction, and 10% DCM in hexane (10 mL) was followed by 50% DCM in hexane (10 mL) to collect the NPAH fraction. The final volumes of the PAH and NPAH fractions were 25 and 20 mL, respectively. Finally, after the evaporation, the residues were dissolved in ethanol at 200, 500, or 1,000 µL, depending on the sample type (indoor or outdoor, cooking or noncooking period) for the PAH fractions, and 200 µL for all NPAH fractions. These were passed through a membrane filter (HLC-DISK 3, 0.45 µm pore size, Kanto Chemical Co., Inc., Tokyo, Japan) prior to HPLC injection with volumes of 20 µL for analysis of PAH and 100 µL for analysis of NPAH. The deuterated compounds Pyr-d_{10}, BaA-d_{12}, BaP-d_{12}, and 6-NC-d_{11} were used as internal standards.

Ten PAHs (Flu, Pyr, BaA, Chr, BbF, BkF, BaP, DBA, BghiPe, and IDP) were determined using HPLC with fluorescence detection (HPLC-FL), following the procedure of Toriba et al. (2003). The system consisted of two HPLC pumps (LC-30A), a fluorescence detector (RF-20Axs), a system controller (CMB-20A), a degasser (DGU-20A5R), an auto sample injector (SIL-30AC) and a column oven (CTO-20AC) (all from Shimadzu, Kyoto, Japan). PAHs were separated on a guard column (Inertsil ODS-P, 10 × 3.0 mm i.d., 5 µm, GL Sciences, Tokyo, Japan) and an analytical column (Inertsil ODS-P, 250 × 3.0 mm i.d., 5 µm, GL Sciences) with acetonitrile/water gradient and fluorescence detection.

Eleven NPAHs (9-NPh, 2-NA, 9-NA, 2-NFR, 3-NFR, 1-NP, 2-NP, 4-NP, 6-NC, 7-NBaA, and 6-NBaP) were measured using an HPLC-FL method based on the chemiluminescence method reported in previous studies (Tang et al. 2005; Chuesaard et al. 2014). The HPLC system of NPAH determination is shown in Fig. 2.4. The system comprised four HPLC pumps (LC-20AD), a system controller (CBM-20A), a degasser (DGU-20A5), an auto sample injector (SIL-20AC), a column oven (CTO-20AC), a six port switching valve, and a fluorescence detector (RF-20Axs); all the components were from Shimadzu (Kyoto, Japan). The NPAHs were purified using a clean-up column (Cosmosil 5NPE, 150 × 4.6 mm i.d. 5 µm, Nacalai Tesque, Kyoto, Japan) with a guard column and were then reduced to their amino derivatives by the use of a reduction column (NPpak-RS, 10 × 4.0 mm i.d. JASCO, Tokyo, Japan) under heating at 80°C. The mobile phase in the clean-up column and reduction column was acetate buffer (pH 5.5)-ethanol (5/95, v/v) with a flow rate of 0.2 mL/min. The mobile phase eluted from the reduction column was mixed with 30 mM ascorbic acid at a flow rate of 1.6 mL/min before entering a concentration column (Spheri-5 RP-18, 30 × 4.6 mm i.d. 5 µm, Perkin Elmer, MA, USA) at the switching valve. A fraction of the amino derivative was trapped in the concentration column using the switching valve with a switching time of 13.5–22.5 min. The concentrated fraction was passed through two separation columns (Inertsil ODS-P, 250 × 4.6 mm i.d. 5 µm, GL, Sciences, Tokyo, Japan) in tandem. All columns were maintained at 20°C. A gradient elution of the separation columns was performed using 10 mM imidazole buffer (pH 7.6) as eluent A and acetonitrile as eluent B. The gradient conditions (B concentrations and flow rate) for the separation of the amino derivatives are presented in Table 2.2. The eluted fraction from the separation columns was detected by the dual-channel fluorescence detector and

wavelengths used for the reduced NPAHs are provided in Tables 2.3. Representative chromatograms of NPAH standards are shown in Fig. 2.5.

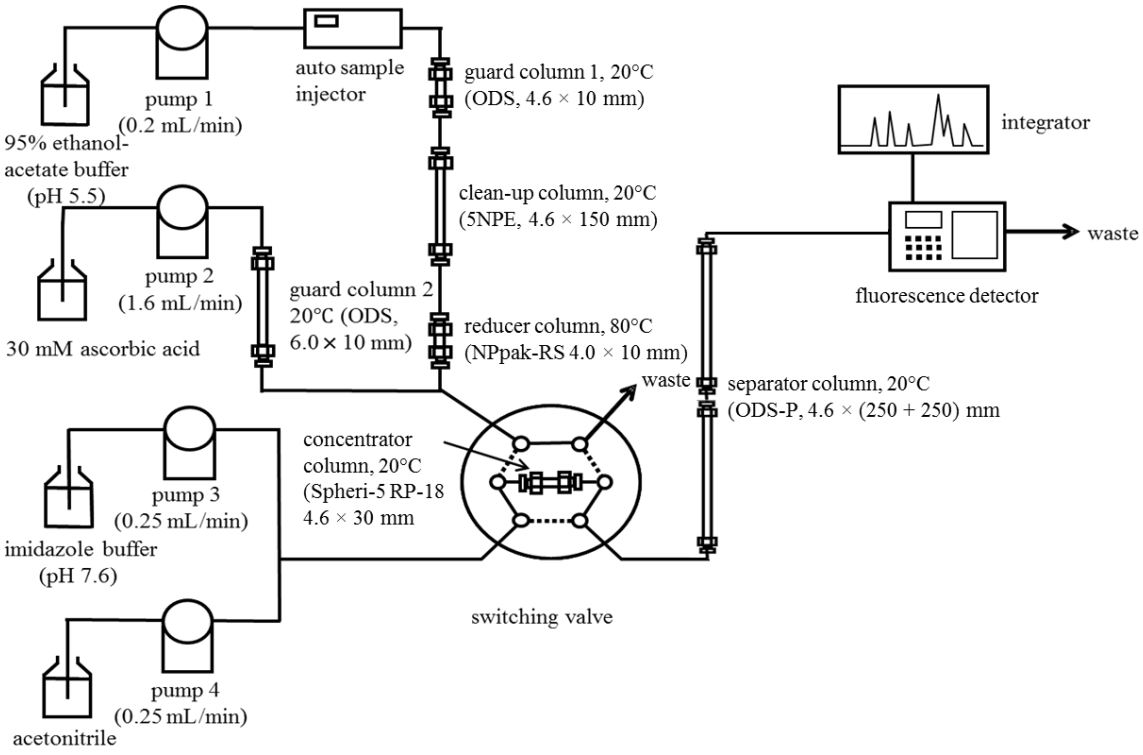


Fig. 2.4 HPLC system for NPAHs analysis

Table 2.2 Gradient conditions for the separation of amino derivatives of NPAHs

Acetonitrile concentration		Flow rate	
Time (min)	**B (%) [a)]**	**Time**	**Total flow (mL/min)**
0–13.50	20	0–13.50	0.5
13.50–32.50	70	13.50–22.50	0.5–0.8 [c)]
32.50–52.50	80	22.50–32.50	0.8
52.50–65.00	100	32.50–52.50	0.9
65.00–80.00	20 [b)]	52.50–65.00	0.9–1.8 [c)]
		65.00–70.00	1.0–0.5 [b, c)]
		70.00–80.00	0.5 [b)]

[a)] The concentration varied using the stepwise gradient mode

[b)] Initializing process after analysis

[c)] The flow rate increased linearly with time

Table 2.3 Excitation and emission wavelengths for the detection of amino derivatives of NPAHs

Chanel		Time	Excitation wavelength (nm)	Emission wavelength (nm)
1	$W_{1\text{-}1}$ [a)]	0.00–41.75 min	247	430
	$W_{1\text{-}2}$	41.75–44.30 min	283	513
	$W_{1\text{-}3}$	44.30–46.40 min	300	530
	$W_{1\text{-}4}$	46.40–65.00 min	273	437
2	$W_{2\text{-}1}$	0.00–44.40 min	260	490
	$W_{2\text{-}2}$	44.40–48.00 min	360	430
	$W_{2\text{-}3}$	48.00–55.00 min	300	475
	$W_{2\text{-}4}$	55.00–65.00 min	283	513

[a)] The marks for wavelength changes are shown in Fig. 2.5.

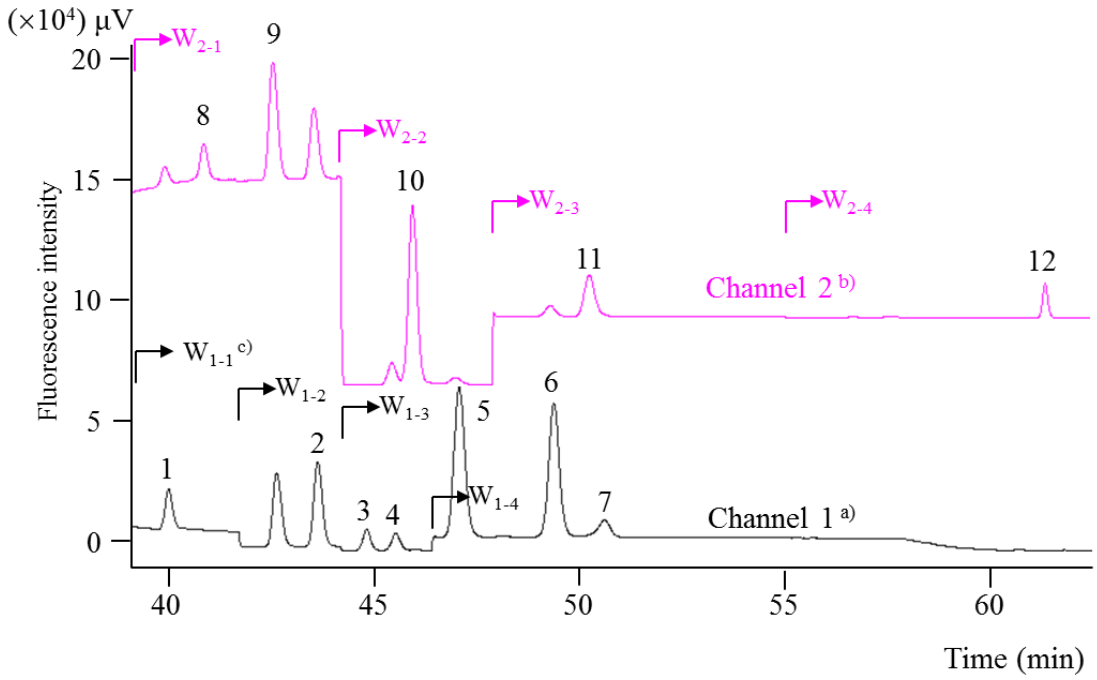


Fig. 2.5 Representative chromatograms of NPAH standards by the HPLC method using the dual-channel fluorescence detector.

[a)] Chanel 1 (black line); 1: 9-NPh (1.1 ng/mL), 2: 2-NFR (2.5 ng/mL), 3: 3-NFR (1.2 ng/mL), 4: 4-NP (1.2 ng/mL), 5: 2-NP (1.2 ng/mL), 6: 6-NC-d_{11} (internal standard), 7: 6-NC (0.3 ng/mL).

[b)] Chanel 2 (pink line); 8: 9-NA (1.1 ng/mL), 9: 2-NA (1.1 ng/mL), 10: 1-NP (1.2 ng/mL), 11: 7-NBaA (1.4 ng/mL), 12: 6-NBaP (1.5 ng/mL).

[c)] The times for wavelength changes. The detailed information is described in Table 2.2.

LG analysis was performed using gas chromatography with mass spectrometry (GC-MS) by following the method reported in Chuesaard et al. (2014). Briefly after combining the two fractions (first/second fraction; 2:1 v/v), the mixture was evaporated until dryness and the residue then derivatized by adding toluene, pyridine, and a silylating agent. The mixture was heated to 80°C for 1 h before being injected into GC-MS equipment with a DB-5MS column (30 m × 250 μm i.d., 0.25 μm film thickness). The isotope-labeled ($^{13}C_6$) LG was used as an internal standard.

The TSP samples were used for the determination of the carbon fractions, including organic carbon (OC) and elemental carbon (EC). Carbonaceous fractions were analyzed using an OC/EC analyzer (Sunset Laboratory, Tigard, OR), following the IMPROVE method. Four OC fractions (OC1, OC2, OC3, and OC4) were produced under heating in a pure helium (He)

atmosphere and three EC fractions (EC1, EC2, and EC3) in 2% O_2/98% He. In this study, OC and EC were defined as ∑OC (OC1 + OC2 + OC3 + OC4) + POC (pyrolyzed carbon fraction) and ∑EC (EC1 + EC2 + EC3) − POC, respectively. The EC fraction was divided into char-EC and soot-EC (char-EC = EC1 – POC and soot-EC = EC2 + EC3). Moreover, the total carbon (TC) was OC1 + OC2 + OC3 + OC4 + EC1 + EC2 + EC3 (Han et al. 2007; Han et al. 2009; Wei et al. 2015).

2.2.4 Quality control and data analysis

Quantitative analysis of PAH and NPAH was based on the peak area ratios between the analytes and the deuterated internal standards. Validation of the analytical methods was conducted using spiked $PM_{2.5}$ samples at two different concentrations. The low concentration was three times higher than the concentration observed in the sample, and the high concentration was ten times higher. For analytes that were undetectable in the nonspiked sample, the spiked concentration was based on the limit of quantification (LOQ). The limit of detection (LOD) and the LOQ for each compound are given in Table 2.4. The LOD and LOQ values were calculated as a signal-to-noise ratio of 3:1 and 10:1, respectively. The results of accuracy and precision are shown in Table 2.5. The accuracy was 100 ± 20% for all analytes. The precision was favorable at a RSD of 10% or less for all analytes. The recoveries of the deuterated internal standards (Pyr-*d*10, BaA-*d*12, BaP-*d*12, and 6-NC-*d*11) were between 50% and 120%.

Table 2.4 LOD and LOQ values (ng/ml) of each PAHs and NPAHs

PAHs	**LOD**	**LOQ**	**NPAHs**	**LOD**	**LOQ**
Flu	0.159	0.529	9-NPh	0.015	0.048
Pyr	0.016	0.053	2-NA	0.013	0.042
BaA	0.005	0.016	9-NA	0.016	0.054
Chr	0.014	0.048	2-NFR	0.003	0.011
BbF	0.036	0.121	3-NFR	0.004	0.014
BkF	0.012	0.039	1-NP	0.001	0.004
BaP	0.049	0.163	2-NP	0.003	0.012
DBA	0.088	0.292	4-NP	0.006	0.019
BghiPe	0.028	0.094	6-NC	0.003	0.012
IDP	0.135	0.448	7-NBaA	0.005	0.016
			6-NBaP	0.025	0.085

Table 2.5 Result of method validation of PAHs and NPAHs (n = 5)

PAHs (pg/m^3)	Spiked concentration	PAHs concentration mean ± SD	Accuracy (%)	Precision (RSD%)	NPAHs (pg/m^3)	Spiked concentration	NPAHs concentration mean ± SD	Accuracy (%)	Precision (RSD%)
	0.00	46 ± 2.3	-	5.1		0.00	<LOQ	-	-
Flu	222	268 ± 7.1	100	2.6	9-NPh	1.29	1.75 ± 0.07	105	4.0
	709	806 ± 11	107	1.3		4.29	5.14 ± 0.12	110	2.3
	0.00	30 ± 1.4	-	4.6		0.00	N.D.	-	-
Pyr	111	150 ± 2.9	106	2.0	2-NA	1.11	1.28 ± 0.06	116	4.7
	354	443 ± 3.4	115	0.8		3.69	4.21 ± 0.13	114	3.0
	0.00	11± 0.4	-	3.3		0.00	<LOQ	-	-
BaA	111	129 ± 3.8	106	3.0	9-NA	2.81	3.30 ± 0.13	102	4.0
	355	403 ± 5.2	110	1.3		9.37	10.4 ± 0.60	106	5.8
	0.00	32 ± 0.7	-	2.2		0.00	3.05 ± 0.04	-	1.4
Chr	111	148 ± 5.7	104	3.8	2-NFR	8.24	11.4 ± 0.21	101	1.9
	355	434 ± 5.2	112	1.2		27.5	31.5 ± 0.96	103	3.1
	0.00	33 ± 0.6	-	1.8		0.00	N.D.	-	-
BbF	222	244 ± 6.7	96	2.8	3-NFR	0.36	0.42 ± 0.04	92	8.6
	709	747 ± 33	101	4.4		1.21	1.41 ± 0.05	116	3.5
	0.00	17 ± 0.2	-	1.0		0.00	0.47 ± 0.004	-	0.9
BkF	111	139 ± 3.8	108	2.7	1-NP	1.71	2.35 ± 0.05	108	2.1
	355	417 ± 17	112	4.2		5.69	6.90 ± 0.13	112	1.9
	0.00	16 ± 0.6	-	3.7		0.00	0.05 ± 0.01	-	9.9
BaP	111	136 ± 5.6	107	4.1	2-NP	0.31	0.38 ± 0.01	105	3.0
	355	432 ± 17	116	4.0		1.03	1.19 ± 0.02	110	1.6
	0.00	N.D.	-	N.D.		0.00	N.D.	-	-
DBA	222	244 ± 9.5	110	3.9	4-NP	0.51	0.57 ± 0.03	111	5.8
	709	788 ± 49	111	6.2		1.71	1.90 ± 0.06	111	3.0
	0.00	46 ± 3.2	-	6.9		0.00	0.13 ± 0.01	-	9.3
BghiPe	222	295 ± 11	110	3.6	6-NC	0.31	0.41 ± 0.03	93	6.5
	709	865 ± 34	115	3.9		1.04	1.18 ± 0.04	101	3.3
	0.00	29 ± 2.6	-	8.8		0.00	0.14 ± 0.01	-	8.5
IDP	111	156 ± 6.4	112	4.1	7-NBaA	1.82	1.94 ± 0.05	99	2.8
	355	419 ± 9.8	109	2.3		6.06	6.42 ± 0.20	104	3.1
						0.00	N.D.	-	-
<LOQ = less than limit of quantification					6-NBaP	2.25	2.45 ± 0.10	109	4.2
N.D. = not detected						7.49	8.04 ± 0.13	107	1.6

SPSS 17.0 software (SPSS Inc., North Castle, NY, USA) was used to calculate Spearman's rank correlation coefficients for the relationships among the concentrations of PAHs, NPAHs, and LG during the cooking period in House 1, House 2, and in the indoor and outdoor environments at a significance level of 0.05. In the case of concentrations that were below the LOQ, a value of half the LOQ was used in the data analysis. Undetected compounds were excluded from the calculations.

2.3 Results and discussion

2.3.1 Real time $PM_{2.5}$ monitoring and characteristics of carbonaceous fractions

Real time monitoring of indoor $PM_{2.5}$ was conducted in parallel with the filter collection of $PM_{2.5}$ by using the personal sampler. Time-dependent changes in $PM_{2.5}$ counts inside both houses during the sampling period are shown in Fig. 2.6. There was a substantial variation in the three sampling periods with higher levels recorded during the evening (18:00–20:00 pm) and morning (5:00–7:30 am) cooking periods due to the increase $PM_{2.5}$ generation from biomass burning. The time periods with higher concentrations than the mean counts in the noncooking periods were defined as the burning periods for cooking (Fig. 2.6). In House 1, a large mass of wood was supplied to the two stoves at the beginning of cooking, producing incomplete combustion that raised the $PM_{2.5}$ level. The highest levels were therefore observed in the first 3–23 min of these burning periods. In contrast, the mean $PM_{2.5}$ counts in House 2 were lower, as the wood was supplied in stages, producing a series of peaks in the $PM_{2.5}$ concentration. These peaks have also been observed in households in rural China (Jiang and Bell 2008). During noncooking periods, the variation disappeared, and the levels were probably consistent with those of the outside atmosphere. The contamination levels in House 1, with the larger number of inhabitants, were higher than those in House 2.

Table 2.6 shows the concentrations of carbon fractions in the indoor samples from House 1 during cooking periods. The OC and EC concentrations were higher in the breakfast period than in the dinner period. The ratios of fractions have been used to identify sources of air carbonaceous aerosols. The char-EC/soot-EC ratios are less than 1 for automobile exhaust, 1.5–3.0 for residential coal combustion, and much higher for biomass burning, rising as high as 11.6 (Cao et al. 2005) or 22.6 (Chow et al. 2004). The char-EC/soot-EC ratios in this study were similar to those observed in other studies of biomass burning. Our EC/OC ratios were also close to the reported ratios of 0.284 (Christian et al. 2010) and 0.267 (Roden and Bond 2006) for wood fire cooking. These results strongly suggest that biomass burning was the primary source of the carbonaceous aerosols.

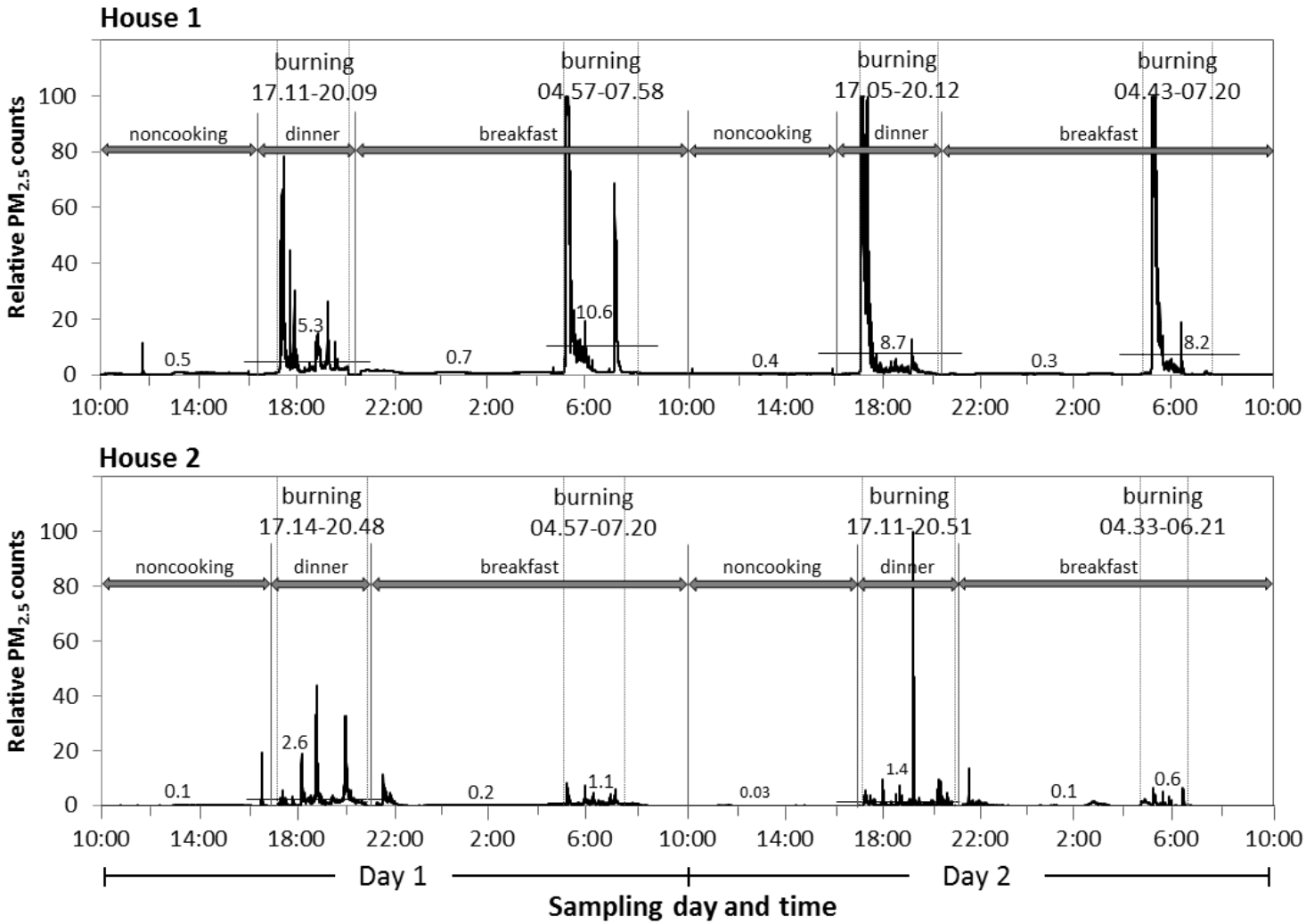


Fig. 2.6 Time-dependent changes in indoor $PM_{2.5}$ counts.

The highest level of $PM_{2.5}$ was taken as 100. The details of the three sampling periods (noncooking, dinner, and breakfast) were described in the section on the study site and PM sampling. Horizontal lines and the values in the figure show mean values of $PM_{2.5}$ counts during burning and nonburning periods.

Table 2.6 Concentrations of carbonaceous fractions of indoor TSP samples during cooking periods

Cooking periods	Concentration (μg/m^3)					Ratio	
	OC	EC	TC	Char-EC	Soot-EC	Char-EC/Soot-EC	EC/OC
Dinner	240	94.1	334	75.6	18.5	4.1	0.39
Breakfast	454	180	633	158	22.2	7.1	0.40

2.3.2 Presence and composition of PAHs and NPAHs

2.3.2.1 PAHs

Figure 2.7 shows the indoor and outdoor mean concentrations of total PAH during cooking and noncooking periods. The concentrations per volume of the samples were calculated by assuming that all emissions were generated during the burning periods. The detailed concentrations are shown in Tables 2.7 and 2.8. The indoor PAH concentrations during cooking periods in House 1 and 2 were 631–2,080 times and 17–82 times higher than in noncooking periods, respectively, suggesting that biomass burning was a major source of PAHs. The concentration during each cooking period in both houses varied, probably due to differences in fuel consumption and cooking time. The PAH concentrations during cooking in House 1 were significantly higher than those in House 2, probably due to the a greater consumption of fuels based on the greater number of inhabitants. In House 1, two traditional open stoves were used for 17 people, in comparison to an open fire for 2 people in House 2. A second factor was the difference in house structure. House 2 had a wooden construction, providing better ventilation than the concrete structure of House 1, as air exchange occurred through the rough lattice walls.

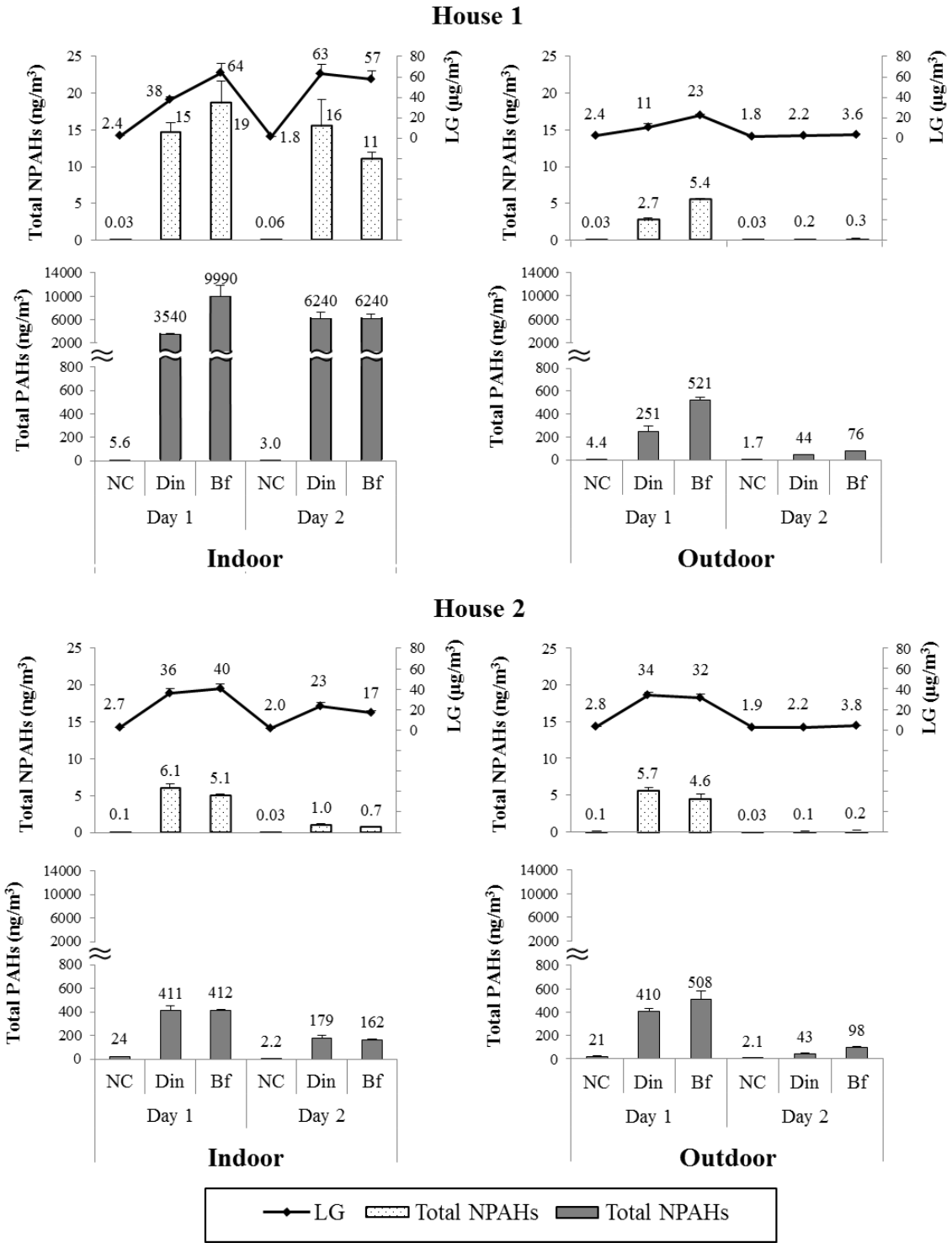


Fig. 2.7 Indoor and outdoor concentrations (mean ± SD) of total PAHs (ng/m^3), NPAHs (ng/m^3), and LG (µg/m^3) during cooking (dinner and breakfast) and noncooking periods. *NC*: noncooking; *Din*: dinner; *Bf*: breakfast

Table 2.7 Concentration (mean ± SD) of PAHs, NPAHs and LG in $PM_{2.5}$ collected inside and outside the House 1

House 1	Day 1						Day 2					
	Indoor			Outdoor			Indoor			Outdoor		
Compounds	Noncooking (n = 3)	Dinner (n = 3)	Breakfast (n = 3)	Noncooking (n = 3)	Dinner (n = 3)	Breakfast (n = 3)	Noncooking (n = 4)	Dinner (n = 4)	Breakfast (n = 4)	Noncooking (n = 3)	Dinner (n = 3)	Breakfast (n = 3)
PAHs (ng/m^3)												
Flu	<LOQ	155 ± 18	1210 ± 422	<LOQ	11.9 ± 2.7	11.9 ± 1.2	<LOQ	788 ± 163	344 ± 78	<LOQ	2.08 ± 0.2	3.3 ± 0.3
Pyr	0.63 ± 0.1	227 ± 28	1850 ± 590	0.57 ± 0.2	17.1 ± 3.1	18.2 ± 2.0	0.51 ± 0.1	1150 ± 224	555 ± 119	0.34 ± 0.1	2.22 ± 0.4	4.0 ± 0.2
BaA	0.36 ± 0.05	592 ± 11	1940 ± 336	0.15 ± 0.03	33.0 ± 6.5	74.5 ± 2.8	0.23 ± 0.1	1190 ± 229	1410 ± 127	0.08 ± 0.01	3.81 ± 0.8	5.8 ± 0.1
Chr	0.74 ± 0.1	422 ± 15	956 ± 117	0.63 ± 0.2	23.4 ± 4.9	64.5 ± 3.1	0.46 ± 0.1	618 ± 135	830 ± 38	0.24 ± 0.05	3.23 ± 0.7	6.1 ± 0.1
BbF	0.66 ± 0.03	232 ± 12	488 ± 61	0.57 ± 0.1	19.8 ± 3.6	47.0 ± 1.5	0.43 ± 0.1	300 ± 50	405 ± 30	0.30 ± 0.02	4.54 ± 0.7	8.1 ± 0.2
BkF	0.23 ± 0.03	225 ± 14	533 ± 72	0.17 ± 0.03	19.1 ± 3.8	43.4 ± 0.3	0.24 ± 0.1	326 ± 53	440 ± 42	0.11 ± 0.01	3.81 ± 0.6	7.2 ± 0.3
BaP	0.38 ± 0.1	631 ± 28	1460 ± 334	0.39 ± 0.1	47.7 ± 8.8	97.9 ± 7.5	0.40 ± 0.1	770 ± 139	1100 ± 91	<LOQ	8.97 ± 1.3	14.0 ± 0.5
DBA	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.
BghiPe	1.43 ± 0.1	576 ± 38	848 ± 39	1.17 ± 0.1	41.6 ± 7.8	94.1 ± 7.7	0.82 ± 0.1	635 ± 80	672 ± 117	0.59 ± 0.04	9.30 ± 1.1	15.6 ± 1.4
IDP	1.18 ± 0.1	479 ± 25	708 ± 25	0.92 ± 0.1	37.1 ± 8.9	69.4 ± 7.5	<LOQ	462 ± 38	487 ± 85	<LOQ	6.73 ± 1.1	11.6 ± 1.8
Total PAHs	5.61 ± 0.4	3540 ± 74	9990 ± 1800	4.44 ± 0.8	251 ± 49	521 ± 26	3.00 ± 0.7	6240 ± 1070	6240 ± 656	1.66 ± 0.2	44.0 ± 6.4	75.5 ± 2.9
NPAHs (pg/m^3)												
9-NPh	N.D.	N.Q.	N.Q.	N.D.	N.D.	N.D.	N.D.	1670 ± 179	1100 ± 146	N.D.	N.D.	N.D.
2-NA	N.D.	<LOQ	259 ± 35	N.D.	N.D.	N.D.	N.D.	195 ± 36	336 ± 18	N.D.	N.D.	N.D.
9-NA	N.D.	5590 ± 171	6570 ± 891	N.D.	1100 ± 125	1620 ± 174	N.D.	4330 ± 1150	3220 ± 237	N.D.	N.D.	N.D.
2-NFR	34.6 ± 3.0	4610 ± 524	6000 ± 1050	26.9 ± 3.5	897 ± 141	1770 ± 41	36.9 ± 2.6	4600 ± 1040	2820 ± 280	30.6 ± 2.1	100 ± 21	159 ± 8.7
3-NFR	N.D.	<LOQ	287 ± 108	N.D.	<LOQ	<LOQ	N.D.	430 ± 112	455 ± 84	N.D.	N.D.	N.D.
1-NP	<LOQ	71 ± 18	143 ± 35	<LOQ	21.2 ± 3.7	30.6 ± 1.4	<LOQ	154 ± 23	141 ± 16	<LOQ	<LOQ	<LOQ
2-NP	<LOQ	4430 ± 620	5290 ± 866	<LOQ	464 ± 86	1400 ± 56	23.0 ± 2.0	4930 ± 1330	2890 ± 353	<LOQ	68.7 ± 10	111 ± 16
4-NP	N.D.	<LOQ	179 ± 98	N.D.	<LOQ	<LOQ	N.D.	109 ± 33	89.2 ± 10	N.D.	N.D.	N.D.
6-NC	N.D.	N.Q.	N.Q.	N.D.	106 ± 6.8	338 ± 23	N.D.	N.Q.	N.Q.	N.D.	N.D.	<LOQ
7-NBaA	N.D.	N.Q.	N.Q.	N.D.	87.2 ± 9.2	283 ± 20	N.D.	N.Q.	N.Q.	N.D.	<LOQ	<LOQ
6-NBaP	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.
Total NPAHs	34.6 ± 3.0	14700 ± 1320	18700 ± 2930	26.9 ± 3.5	2680 ± 326	5440 ± 299	61.6 ± 2.1	15500 ± 3640	11000 ± 1030	30.6 ± 2.1	169 ± 31	270 ± 7.3
LG (ug/m^3)												
LG	2.37 ± 0.2	38.3 ± 2.3	64.0 ± 9.5	2.36 ± 0.2	10.8 ± 3.1	22.8 ± 0.6	1.78 ± 0.1	62.9 ± 9.2	57.4 ± 8.3	1.77 ± 0.1	2.24 ± 0.2	3.62 ± 0.3

<LOQ = less than limit of quantification

N.D. = not detected

N.Q. = not quantified because co-eluted with interfering peaks.

Table 2.8 Concentration (mean ± SD) of PAHs, NPAHs and LG in $PM_{2.5}$ collected inside and outside the House 2

House 2	Day 1						Day 2					
	Indoor			Outdoor			Indoor			Outdoor		
Compounds	**Noncooking** (n = 3)	**Dinner** (n = 3)	**Breakfast** (n = 3)	**Noncooking** (n = 3)	**Dinner** (n = 3)	**Breakfast** (n = 3)	**Noncooking** (n = 3)	**Dinner** (n = 3)	**Breakfast** (n = 3)	**Noncooking** (n = 3)	**Dinner** (n = 3)	**Breakfast** (n = 3)
PAHs (ng/m^3)												
Flu	1.4 ± 0.2	22.0 ± 4.0	<LOQ	1.5 ± 0.5	26.8 ± 1.9	14.4 ± 16	<LOQ	4.30 ± 0.5	<LOQ	<LOQ	<LOQ	3.81 ± 0.7
Pyr	1.9 ± 0.2	30.9 ± 6.0	7.5 ± 0.8	1.8 ± 0.3	37.8 ± 2.8	23.5 ± 27	0.30 ± 0.04	5.65 ± 0.7	2.56 ± 0.02	0.21 ± 0.03	1.94 ± 0.4	4.95 ± 0.7
BaA	2.7 ± 0.2	60.7 ± 9.0	59.5 ± 1.3	2.8 ± 0.2	63.7 ± 2.5	82.6 ± 12	0.58 ± 0.1	20.5 ± 1.7	15.7 ± 0.8	0.63 ± 0.1	3.56 ± 0.6	9.81 ± 2.1
Chr	2.3 ± 0.3	49.5 ± 4.9	59.3 ± 2.5	2.3 ± 0.3	45.6 ± 1.4	61.2 ± 1.2	0.16 ± 0.03	19.3 ± 2.0	17.0 ± 0.5	0.17 ± 0.02	2.23 ± 0.6	7.30 ± 1.6
BbF	1.8 ± 0.2	32.0 ± 3.0	46.1 ± 2.1	1.8 ± 0.1	29.3 ± 1.1	41.0 ± 2.8	0.29 ± 0.01	16.5 ± 0.8	20.3 ± 1.0	0.30 ± 0.02	4.45 ± 0.4	9.14 ± 0.6
BkF	1.5 ± 0.2	29.1 ± 2.8	39.0 ± 1.0	1.5 ± 0.1	26.5 ± 1.1	38.6 ± 1.5	0.12 ± 0.003	12.9 ± 0.8	15.2 ± 0.9	0.10 ± 0.01	3.75 ± 0.3	8.13 ± 0.5
BaP	4.3 ± 0.4	78.0 ± 7.8	67.9 ± 6.9	3.7 ± 0.3	74.1 ± 3.0	87.8 ± 10	<LOQ	35.2 ± 3.1	30.7 ± 2.0	<LOQ	8.60 ± 0.4	18.2 ± 0.9
DBA	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.
BghiPe	4.4 ± 0.3	64.0 ± 4.5	86.9 ± 2.8	3.2 ± 0.2	62.5 ± 4.6	89.4 ± 8.5	0.72 ± 0.03	36.8 ± 6.6	35.3 ± 2.9	0.67 ± 0.03	10.4 ± 0.5	21.2 ± 4.4
IDP	3.6 ± 0.3	45.1 ± 4.2	45.7 ± 3.3	2.4 ± 0.2	44.1 ± 3.8	69.8 ± 7.0	<LOQ	27.8 ± 5.3	25.2 ± 0.3	<LOQ	7.76 ± 0.6	15.6 ± 3.6
Total PAHs	23.9 ± 1.5	411 ± 45	412 ± 8.5	21.1 ± 2.0	410 ± 16	508 ± 72	2.18 ± 0.2	179 ± 20	162 ± 6.6	2.08 ± 0.1	43.3 ± 4.5	98.1 ± 4.1
NPAHs (pg/m^3)												
9-NPh	N.D.	477 ± 32	<LOQ	N.D.	424 ± 30	192 ± 27	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.
2-NA	N.D.	<LOQ	N.D.	N.D.	<LOQ	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.
9-NA	N.D.	1910 ± 305	1240 ± 120	N.D.	1950 ± 183	1170 ± 82	N.D.	193 ± 35	N.D.	N.D.	N.D.	N.D.
2-NFR	63.3 ± 5.1	1760 ± 118	1740 ± 84	60.7 ± 5.9	1690 ± 100	1490 ± 206	29.7 ± 1.7	352 ± 39	293 ± 21	29.0 ± 2.5	79.0 ± 8.9	120 ± 10
3-NFR	N.D.	<LOQ	<LOQ	N.D.	<LOQ	<LOQ	N.D.	<LOQ	<LOQ	N.D.	N.D.	N.D.
1-NP	<LOQ	24.7 ± 1.6	29.7 ± 1.6	<LOQ	24.5 ± 1.7	27.7 ± 1.6	<LOQ	11.6 ± 1.4	<LOQ	<LOQ	<LOQ	<LOQ
2-NP	35.5 ± 4.8	1350 ± 85	1130 ± 87	34.2 ± 8.9	1190 ± 63	924 ± 151	<LOQ	261 ± 35	264 ± 16	<LOQ	59.0 ± 5.6	76.0 ± 3.0
4-NP	N.D.	<LOQ	<LOQ	N.D.	<LOQ	<LOQ	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.
6-NC	<LOQ	535 ± 43	725 ± 48	<LOQ	396 ± 57	630 ± 58	N.D.	212 ± 17	156 ± 10	N.D.	N.D.	52.8 ± 4.6
7-NBaA	N.D.	N.Q.	191 ± 29	<LOQ	N.Q.	174 ± 25	N.D.	N.D.	<LOQ	N.D.	<LOQ	<LOQ
6-NBaP	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.
Total NPAHs	98.8 ± 9.8	6050 ± 577	5050 ± 186	94.9 ± 12	5670 ± 323	4600 ± 525	29.7 ± 1.7	1030 ± 111	712 ± 29	29.0 ± 2.5	138 ± 14	231 ± 22
LG (ug/m^3)												
LG	2.65 ± 0.02	35.8 ± 4.4	40.4 ± 5.0	2.84 ± 0.1	34.4 ± 2.2	31.8 ± 3.4	1.98 ± 0.04	23.0 ± 3.8	17.2 ± 1.8	1.91 ± 0.1	2.16 ± 0.2	3.84 ± 0.7

<LOQ = less than limit of quantification

N.D. = not detected

N.Q. = not quantified because co-eluted with interfering peaks.

The concentration of outdoor PAHs was also higher during cooking periods than noncooking periods (Fig. 2.7). The concentrations during noncooking were similar to or slightly higher than those reported for an urban area of Chiang Mai in the same season (3.1 ng/m^3) (Chuesaard et al. 2014). In contrast, outdoor PAH concentrations during cooking periods were markedly higher than those in Chiang Mai. These observations suggest that indoor cooking increased the PAH concentration both indoors and outdoors, particularly under the eaves, probably due to smoke leakage from the unsealed kitchen, which had no door. This is consistent with previous reports (Shen et al. 2013; Wei et al. 2014; Shen et al. 2014). The total outdoor PAH levels for House 2 during cooking were comparable to those for House 1 (Fig. 2.7), although House 1 had higher internal PAH levels. This suggested that House 2 was not airtight. The indoor to outdoor (I/O) ratios of PAH concentration were used to identify the contribution of indoor air pollution (Table 2.9). An I/O ratio >1 suggests that the major source of air pollution is indoors, whereas an I/O ratio <1 suggests that outdoor sources are dominant (Klinmalee et al. 2009). The largest total PAH I/O ratio during cooking periods was 142 for both houses, higher than those in noncooking periods (1.0–1.8) (Table 2.9). This suggested that indoor cooking played a crucial role in the exposure of residents to PAHs in this rural area. The ratios during cooking were higher than those reported for households using solid fuels in rural Shanxi (7.3–19.9) and rural Hebei (2.4–3.8) (Ding et al. 2012; Chen et al. 2016), suggesting that the households in the current study were exposed to much more severe indoor air pollution. An I/O ratio higher than 3.0 indicates that indoor sources of air pollution overwhelm outdoor sources (Chuang et al. 1991; Mitra and Ray 1995).

Table 2.9 Indoor/outdoor (I/O) ratios of PAHs and NPAHs during cooking and noncooking periods in Houses 1 and 2 (mean ± SD)

Compounds	House 1						House 2					
	Day 1			Day 2			Day 1			Day 2		
	noncooking	dinner	breakfast	noncooking	dinner	breakfast	noncooking	dinner	breakfast	noncooking	dinner	breakfast
Flu	0.99 ± 0.001	13.1 ± 1.5	102 ± 36	1.00 ± 0.001	463 ± 96	105 ± 24	0.89 ± 0.2	0.82 ± 0.2	0.06 ± 0.00002	1.00 ± 0.001	3.74 ± 0.4	0.28 ± 0.0001
Pyr	1.09 ± 0.1	13.3 ± 1.7	102 ± 32	1.50 ± 0.4	516 ± 101	138 ± 29	1.06 ± 0.1	0.82 ± 0.2	0.32 ± 0.03	1.42 ± 0.2	2.92 ± 0.4	0.52 ± 0.004
BaA	2.44 ± 0.3	17.9 ± 0.3	26.0 ± 4.5	2.92 ± 0.9	313 ± 60	244 ± 22	0.94 ± 0.1	0.95 ± 0.1	0.72 ± 0.02	0.92 ± 0.2	5.77 ± 0.5	1.60 ± 0.1
Chr	1.18 ± 0.1	18.1 ± 0.6	14.8 ± 1.8	1.88 ± 0.6	191 ± 42	137 ± 6.3	1.01 ± 0.1	1.09 ± 0.1	0.97 ± 0.04	0.90 ± 0.2	8.65 ± 0.9	2.33 ± 0.1
BbF	1.17 ± 0.1	11.7 ± 0.6	10.4 ± 1.3	1.45 ± 0.3	66.0 ± 11	50.1 ± 3.8	1.02 ± 0.1	1.09 ± 0.1	1.13 ± 0.1	1.00 ± 0.02	3.71 ± 0.2	2.22 ± 0.1
BkF	1.39 ± 0.2	11.8 ± 0.8	12.3 ± 1.7	2.24 ± 0.7	85.8 ± 14	61.0 ± 5.8	1.01 ± 0.1	1.10 ± 0.1	1.01 ± 0.03	1.16 ± 0.03	3.43 ± 0.2	1.87 ± 0.1
BaP	1.21 ± 0.2	13.2 ± 0.6	14.9 ± 3.4	2.27 ± 1.0	85.8 ± 16	78.5 ± 6.5	1.14 ± 0.1	1.05 ± 0.1	0.77 ± 0.1	0.99 ± 0.001	4.09 ± 0.4	1.69 ± 0.1
BghiPe	1.22 ± 0.1	13.9 ± 0.9	9.01 ± 0.4	1.38 ± 0.2	68.4 ± 8.6	43.1 ± 7.5	1.37 ± 0.1	1.02 ± 0.1	0.97 ± 0.03	1.09 ± 0.04	3.55 ± 0.6	1.66 ± 0.1
IDP	1.28 ± 0.1	12.9 ± 0.7	10.2 ± 0.4	1.00 ± 0.001	68.7 ± 5.6	42.1 ± 7.4	1.50 ± 0.1	1.02 ± 0.1	0.66 ± 0.1	1.18 ± 0.001	3.49 ± 0.7	1.62 ± 0.02
Total PAHs	1.26 ± 0.1	14.1 ± 0.3	19.2 ± 3.5	1.80 ± 0.4	142 ± 24	82.5 ± 8.7	1.13 ± 0.1	1.00 ± 0.1	0.81 ± 0.02	1.00 ± 0.1	4.09 ± 0.5	1.65 ± 0.1
9-NPh	- [a)]	-	-	-	-	-	-	1.12 ± 0.1	0.43 ± 0.0001	-	-	-
2-NA	-	-	-	-	-	-	-	1.00 ± 0.001	-	-	-	-
9-NA	-	5.07 ± 0.2	4.05 ± 0.6	-	-	-	-	0.98 ± 0.2	1.06 ± 0.1	-	-	-
2-NFR	1.29 ± 0.1	5.14 ± 0.6	3.40 ± 0.6	1.21 ± 0.1	45.8 ± 10.4	17.7 ± 1.8	1.04 ± 0.1	1.04 ± 0.1	1.17 ± 0.1	1.03 ± 0.1	4.45 ± 0.5	2.44 ± 0.2
3-NFR	-	1.00 ± 0.001	11.4 ± 4.3	-	-	-	-	1.00 ± 0.001	1.00 ± 0.0002	-	-	-
1-NP	0.99 ± 0.001	3.34 ± 0.9	4.67 ± 1.2	1.00 ± 0.001	24.7 ± 3.8	18.9 ± 2.1	1.03 ± 0.01	1.01 ± 0.1	1.07 ± 0.1	1.00 ± 0.001	2.18 ± 0.3	1.00 ± 0.0002
2-NP	0.99 ± 0.001	9.55 ± 1.3	3.79 ± 0.6	2.16 ± 0.2	71.7 ± 19.4	26.0 ± 3.2	1.04 ± 0.1	1.13 ± 0.1	1.22 ± 0.1	1.00 ± 0.001	4.43 ± 0.6	3.48 ± 0.2
4-NP	-	1.00 ± 0.001	3.69 ± 3.1	-	-	-	-	1.00 ± 0.001	1.00 ± 0.0002	-	-	-
6-NC	-	-	-	-	-	-	1.03 ± 0.01	1.35 ± 0.1	1.15 ± 0.1	-	-	3.62 ± 0.2
7-NBaA	-	-	-	-	-	-	-	-	1.10 ± 0.2	-	-	1.00 ± 0.0002
Total NPAHs	1.29 ± 0.1	5.49 ± 0.5	3.44 ± 0.5	1.95 ± 0.1	91.9 ± 22	40.8 ± 3.8	1.04 ± 0.1	1.07 ± 0.1	1.10 ± 0.04	1.03 ± 0.1	7.45 ± 0.8	2.98 ± 0.1

[a)] The ratios were not calculated for the existence of unquantified compounds.

Table 2.10 compares the ∑PAH from this study with those from previous studies. The mean indoor PAH level in House 1 was 6,500 ng/m^3, higher than those reported in similar studies, such as those of homes in rural India (Bhargava et al. 2004), in a Swedish residential area (Gustafson et al. 2008), in China (Ding et al. 2012; Duan et al. 2014; Shen et al. 2014; Wu et al. 2015), in rural Sierra Leone (Taylor and Nakai 2012), and in an urban area of Kaunas, Lithuania (Kliucininkas et al. 2014). The levels recorded in the present study were lower than those reported for rural Indian homes using wood (7,270 ng/m^3) or cow dung cake (12,780 ng/m^3) as fuels for cooking in winter. This was attributed to the distribution of PAHs with four rings to the particulate phase at low temperatures (Bhargava et al. 2004) A higher exposure of Chinese residents to emissions during cooking periods (7,590 ng/m^3) has been reported. This may be due to the 16 determined PAHs in both gaseous and particulate phases (Chen et al. 2016), whereas this study quantified only the 10 PAHs in the particle phase. Our results agree well with other recent reports that have identified biomass burning in homes as the main source of PAHs in the indoor air, despite being limited to short cooking periods. Such exposure may have major health impacts on household members, especially, on children.

Fig. 2.8 shows the representative concentrations of the individual PAHs found in indoors and outdoors during the cooking and noncooking periods in House 1. BghiPe was the most abundant compound during noncooking periods of both indoors and outdoors, accounting for 15.2%–23.3% of total PAHs (Fig. 2.9). Almost concentrations of individual PAHs markedly increased in House 1 during cooking periods (Fig. 2.8). As noted above, the external air was also affected. In analyzed 10 PAHs, there was no component specific to biomass burning based on the analysis of their concentrations.

BaP has been widely used as a marker for environmental levels of carcinogenic PAHs due to its strong carcinogenicity (Bostrom et al. 2002) and has been proposed for use as a wood smoke tracer (Wang et al. 2007). High levels of BaP have been recorded in emissions from wood burning by Taylor and Nakai (2012), Chen et al. (2016), and Bhargava et al. (2004), who reported kitchen BaP concentrations of 308, 636, and 730 ng/m^3, respectively. The BaP levels in our study were also high, especially, in cooking periods. The maximum recorded mean level of 1,460 ng/m^3 (indoor during cooking for breakfast of House 1 on day 1 (see Table 2.7)) was higher than those from the indoor biomass studies shown in Table 2.10.

Table 2.10 Comparison of PAHs, NPAHs, and BaP concentration in indoor air between our study and other reports

Location	Sample type	Season	Purpose of using fuel combustion	Fuel	Analyzed PAHs	∑PAHs (ng/m^3) mean (min-max)	BaP (ng/m^3) mean	Analyzed NPAHs	∑NPAHs (pg/m^3) mean (min-max)	Reference
Chiang Mai, Thailand	$PM_{2.5}$	dry season	cooking	wood	10	6500 (3540-9990)	1460	11	14980 (11000-18700)	This study
Lucknow, India	RSPM [a]	summer	cooking	wood	7	4470 (1110-11690)	500	- [b]		Bhargava et al. 2004
				cow dung cake (CDC)	7	5960 (1770-14850)	560	-		
		winter	cooking	wood	7	7270 (990-15790)	730	-		
				CDC	7	12780 (7160-20620)	1070	-		
Hagforts, Sweden	PM [c] + Gas	winter	heating	wood	27	34	0.63	-		Gustafson et al. 2008
Hebei, China	$PM_{2.5}$ + Gas	winter	cooking	straw, wood	15	6100	314	12	38000	Ding et al. 2012
		summer	cooking	straw, wood, LPG	15	2400	58.2	12	6000	
Waterloo and Tombo, Sierra Leone	$PM_{2.5}$	- [b]	cooking	wood	11	2127 (319-4282)	308	-		Taylor and Nakai 2012
				charcoal	11	158 (38-355)	25	-		
Taiyuan, China	PM [c] + Gas	heating	heating	coal (57.1%), wood (32.8%), central heating (8.0%), electricity (1.3%), others (0.8%)	15	863 (560-1208) [f]	- [b]	-		Duan et al. 2014
		non-heating	cooking	wood and crop residue (40.8%), coal (37.6%), electricity (10.5%), liquid or natural gas (8.5%), others (2.5%)	15	342 (215-456) [f]	- [b]	-		
Kaunas, Lithuania	$PM_{2.5}$	winter	heating	fire wood, wood pellet, natural gas	15	5.1-60	- [b]	-		Kliucininkas et al. 2014
East China [d]	PM [c]	September (autumn)	cooking	crop straws	15	1602	2.0	-		Shen et al. 2014
Shanxi, China	PM ($PM_{0.25}$, $PM_{0.25\text{-}1.0}$, $PM_{1.0\text{-}2.5}$, $PM_{>2.5}$), + Gas	summer	cooking	solid fuels (wood, peat, honeycomb, briquette	16	7590 [e]	131	-		Chen et al. 2016
Henan, China	$PM_{2.5}$	autumn	cooking/ heating	crop residue	15	150	18.5	-		Wu et al. 2015
		winter			15	222	19.3			

[a] Respirable suspended particulate matter (particles size ≤ 10 μm or PM_{10} (Lamare and Chaturvedi 2014))
[b] Not reported
[c] No information about particle size
[d] No information about the city
[e] The level of ∑PAHs in particle phase was determined in $PM_{0.25}$ samples
[f] Indoor PAHs concentration was estimated from measured personal and outdoor concentration, and recoded indoor and outdoor residence time

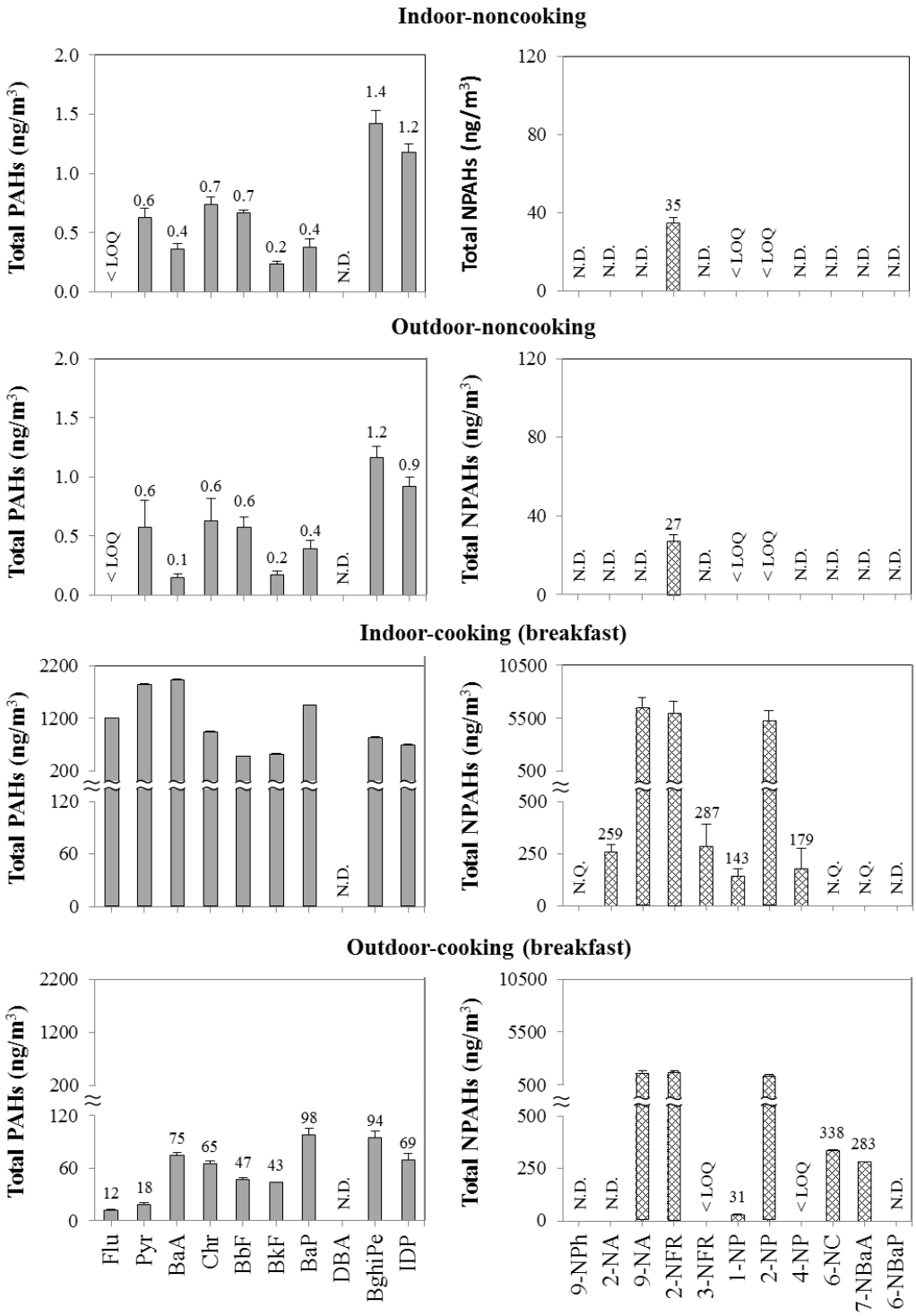


Fig. 2.8 Representative concentrations (mean ± SD) of individual PAHs and NPAHs observed in indoors and outdoors at House 1 during noncooking and cooking periods.
<LOQ = less than limit of quantification; *N.D.* = not detected; *N.Q.* = not quantified because of co-eluted with interfering peaks.

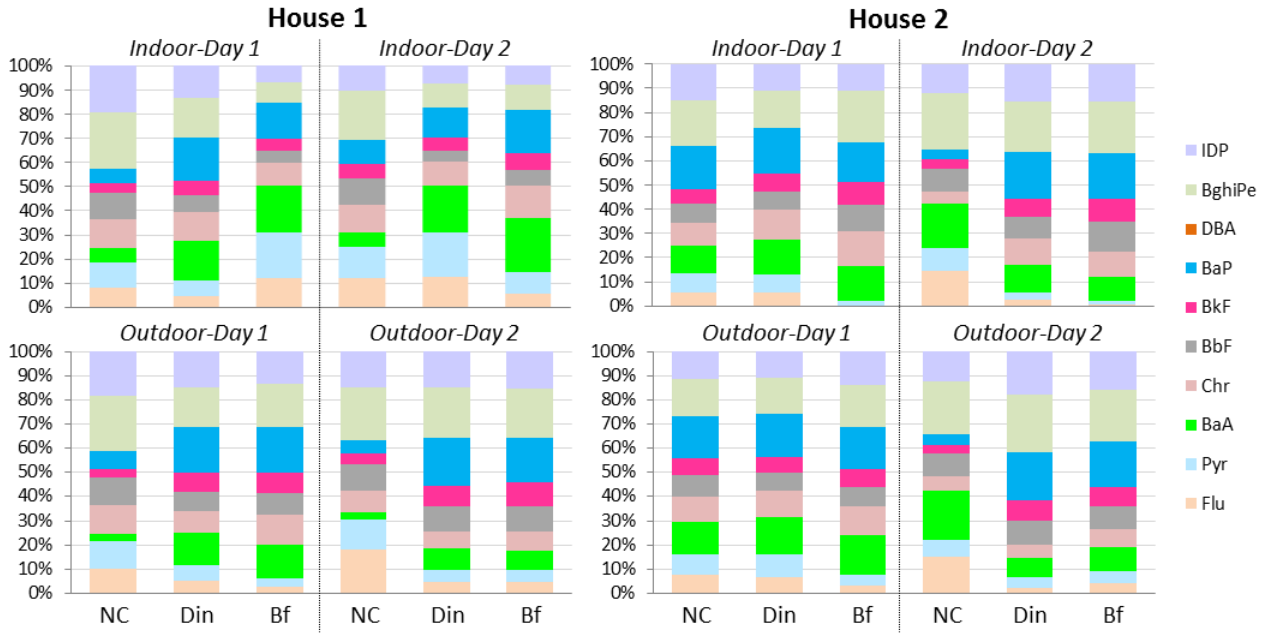


Fig. 2.9 Composition profiles of 10 PAHs indoors and outdoors at Houses 1 and 2 during noncooking and cooking periods.
The profiles of PAHs show the percentage of each compound in the total concentration.
NC: noncooking; *Din*: dinner; *Bf*: breakfast

The PAH diagnostic ratio can be used as a tool for identifying possible sources. Table 2.11 compares the diagnostic PAH ratios in this study with those from previous studies. The mean BbF/(BbF + BkF) ratio of 0.52 found in the indoor air during cooking period agreed well with the value of 0.51 for wood burning reported by Shen et al. (2013) (Table 2.11). The mean value of the IDP/(IDP + BghiPe) ratio for indoor during cooking times was 0.42, which is similar to reported values of 0.47 from wood burning stoves (Vicente et al. 2016) and 0.44 for biomass burning (Kalaitzoglou et al. 2004). However, it was not in agreement with the value of >0.5 reported for wood combustion by Yunker et al. (2002). The mean ratio of BaA/BaP observed during biomass burning inside the houses was 0.99 indicates that wood combustion is a major source of PAHs (Li and Kamens 1993). The ratio was also similar to that from crop residue combustion reported by Wu et al. (2015). We found a mean indoor BaP/BghiPe ratio of 1.19 during cooking, which is close to the reported mean ratios of 1.17 for wood combustion and crop residue combustion (winter) but is different from those of 1.91 for rice straw burning and 2.57

for rice straw burning in the field (Yang et al. 2006; Phoothiwut and Junyapoon 2013; Wu et al. 2015; Vicente et al. 2016). The residents in this study did not use rice straw as a fuel for cooking. The BaP/BghiPe ratios also revealed small extra contributions from diesel exhaust emissions (e.g., 0.46–0.81, Rogge et al. 1993). Although these PAH diagnostic ratios have been widely used for source identification, they must be treated with caution because differences in combustion conditions, sampling methods, and analytical procedures may introduce errors (Tobiszewski and Namiesnik 2012). The ratios during cooking periods observed in this study suggested a large contribution from biomass burning. Because our study focused only on particulate phase PAHs and as some of the PAHs may be distributed in the gas phase, we did not apply diagnostic ratios using PAHs with four aromatic rings.

Table 2.11 Comparison of diagnostic PAH ratios between our study (mean ± SD) and other reports

Combustion sources	BbF/(BbF+BkF)	IDP/(IDP+BghiPe)	BaA/BaP	BaP/BghiPe	Reference
Wood open-fire					
Indoor-noncooking	0.66 ± 0.08	0.40 ± 0.06	0.79 ± 0.17	0.46 ± 0.35	This study
Indoor-cooking	0.52 ± 0.04	0.42 ± 0.04	0.99 ± 0.38	1.19 ± 0.35	
Wood stove		0.47		1.17	Vicente et al. 2016
Wood stove	0.51	0.55			Shen et al. 2013
Biomass		0.44			Kalaitzoglou et al. 2004
Grass, wood, and coal		> 0.5			Yunker et al. 2002
Rice straw (in the field)		0.51		2.57	Phoothiwut and Junyapoon 2013
Rice straw				1.91	Yang et al. 2006
Crop residue ($PM_{2.5}$)					
Autumn (kitchen)		0.61	1.08	1.23	Wu et al. 2015
Winter (kitchen)		0.62	1.44	1.17	
Wood			1.0		Li and Kamens 1993

2.3.2.2 NPAHs

The mean concentrations of individual and total NPAHs are shown in Tables 2.7 and 2.8 and in Fig. 2.7. The pattern of NPAH concentrations during cooking and noncooking periods was similar to that of PAH concentrations. The highest total concentration of NPAHs was found in indoor air during cooking periods. The maximum was observed on day 1 in the indoor environment of House 1 with a mean total concentration of 18,700 pg/m^3. This was higher than the summer levels of 6,000 pg/m^3 reported for rural Chinese households using biomass as the cooking fuel (Ding et al. 2012) but was lower than those observed in winter when the kitchen window and door were completely closed (38,000 pg/m^3). The I/O ratios of the NPAH concentrations shown in Table 2.9 were calculated for the detected compounds. The ratios were above 1.0, suggesting that indoor sources were dominant but were not as large as the ratios found for PAHs. The highest I/O ratio for the total NPAH levels of 92 was observed during a cooking period and was higher than those in both noncooking periods (1.0–2.0) and ratios reported for rural Chinese households in Hebei using solid fuels for cooking (3.5–5.0) (Ding et al. 2012).

Figures 2.7 and 2.10 show that 9-NA, 2-NFR, and 2-NP were the most abundant compounds identified. The highest concentration of 9-NA found in this study was 6,570 pg/m^3. Chuesaard et al. (2014) also observed high 9-NA concentrations (249.4 pg/m^3) during the dry season in Chiang Mai. This is much higher than the concentrations reported for other Asian cities (Hayakawa et al. 2007; Pham et al. 2012). 9-NA has been suggested as a marker for biomass burning. Ding et al. (2012) also reported that 9-NA had the highest concentration (12,000 pg/m^3) among all NPAHs in the particulate phase in rural households in China that use biomass for cooking. Chuesaard et al. (2014) proposed the use of the [9-NA]/[1-NP] ratio as an indicator for biomass burning. All [9-NA]/[1-NP] ratios in this study were higher than 10 (ranging from 15 to 80), indicating a major contribution from biomass burning, with the highest ratio found during cooking on the first study day. The [9-NA]/[1-NP] ratios during cooking periods in these rural households suggested a contribution of biomass combustion much greater than that found for ambient air during severe haze period (February-March) in Chiang Mai (Chuesaard et al. 2014).

The outside and inside concentrations of 2-NFR observed during noncooking times were 26.9–63.3 pg/m^3 (see Tables 2.7 and 2.8), similar to the concentration of 164 pg/m^3 reported for the dry season in Chiang Mai (Chuesaard et al. 2014). 2-NFR and 2-NP are formed in the atmosphere as secondary products (Atkinson and Arey 2003). As extremely high internal 2-NFR

and 2-NP levels were detected during cooking periods, the possibility of their secondary formation inside the house cannot be excluded. The ratio of 2-NFR/1-NP is widely used to evaluate the relative contribution of primary emissions (combustion sources) and secondary formation (chemical reactions in the atmosphere) (Bamford and Baker 2003b; Albinet et al. 2007). A 2-NFR/1-NP ratio >5 indicates that secondary formation is dominant, whereas a ratio <5 indicates the dominance of primary emissions (Ciccioli et al. 1996). The 2-NFR/1-NP ratios during cooking and noncooking periods ranged from 14.9 to 71.2 and 8.1 to 19.8, respectively. This suggests that the secondary formation was present during cooking, adding to the primary source. The 2-NFR/2-NP ratio is often used to estimate the relative importance of OH or NO_3 radical initiated reactions in the secondary formation of NPAHs in the atmosphere. Ratios close to 10 are associated with OH radical initiated reactions, whereas ratios close to 100 are associated with NO_3 radical initiated reactions (Arey et al. 1989; Wang et al. 2011). The 2-NFR/2-NP ratios of indoor samples taken during cooking periods were less than 10, ranging from 0.9–1.5, suggesting the dominance of daytime OH radical initiated reactions in NPAH formation. Similarly, low ratios were observed at a forest site in Brazil (Ciccioli et al. 1996).

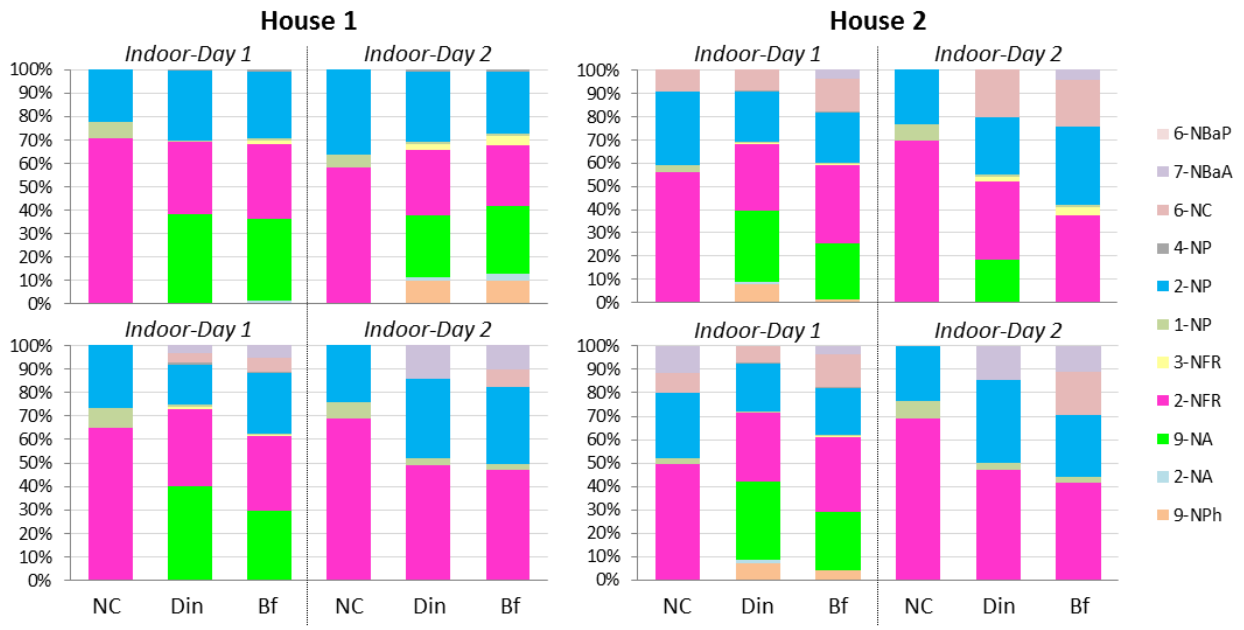


Fig. 2.10 Composition profiles of 11 NPAHs indoors and outdoors at Houses 1 and 2 during noncooking and cooking periods.
The profiles of NPAHs show the percentage of each compound in the total concentration.
NC: noncooking; *Din*: dinner; *Bf*: breakfast

2.3.3 Tracer for biomass burning

LG is the most abundant monosaccharide anhydride in the aerosol particles produced by biomass burning and is released by the combustion and pyrolysis of cellulose. Due to its source specificity and stability in the atmosphere, LG has been suggested as a marker of biomass burning (Urban et al. 2012). The LG levels in this study are shown in Fig. 2.7 and in Tables 2.7 and 2.8. High concentrations were found during cooking periods and ranged from 17.2 to 64.0 μg/m^3 indoors and 2.2 to 34.4 μg/m^3 outdoors. LG concentrations were lower when cooking was not taking place. The indoor air in House 1 on the first study day showed the highest LG concentration of 64.0 μg/m^3, as well as the highest concentrations of PAH and NPAH (Fig. 2.7). LG accounted for 10.1%–11.5% of TC (Table 2.6), higher than the values of 1%–6% at Brazilian rainforest sites heavily influenced by biomass burning (Graham et al. 2002). The LG concentrations from household biomass cooking observed in this study were significantly higher than those reported previously, for example, in a study of two rural villages where biomass fuels

(wood and crop residues) were used for cooking and space heating (Huang et al. 2015). The LG levels reported in the study in which combustion took place in stoves with an enclosed combustion chamber and a chimney for ventilation ranged from 2.99 to 4.78 μg/m^3. High LG concentrations have been found in ambient air collected close to a source of biomass burning, such as in urban Chiang Mai (0.26 μg/m^3) during the dry season, when forest fires occur (Chuesaard et al. 2014), and in Montana (0.9–6.0 μg/m^3) due to wildfires (Ward et al. 2006). The concentrations found in our outside samples when no cooking was taking place were between 1.8 and 2.8 μg/m^3, higher than or comparable with those from the earlier reports. We assume that this was due to the effect of earlier cooking and of wildfires around the village. As noted above, LG is stable in the atmosphere; thus, the effect of a combustion source may be long-term.

The correlation coefficients for PAHs and NPAHs with LG are shown in Table 2.12. PAH and NPAH concentrations showed significant correlations ($p < 0.01$) with the LG concentration, especially, in House 1 during cooking, suggesting that traditional open wood stove combustion for cooking was a major source of PAHs and NPAHs. The concentrations in House 2 during cooking periods were more weakly correlated, due to differences in fuel consumption and house structure. The correlation of outdoor LG with PAHs and NPAHs during cooking periods was less strong than the indoor correlation. Indoor biomass cooking was the major source of outdoor air pollution because the samplers were hung under the eaves of the houses, thus exposing them to air leaking from the interior (Fig. 2.3). In general, NPAHs have a larger level per unit weight in particulates from vehicle exhaust than from biomass burning (Yang et al. 2010; Cochran et al. 2012). However, large-scale biomass burning may contribute to the generation of NPAHs (Chuesaard et al. 2014).

Table 2.12 Correlation coefficient between PAHs and NPAHs with LG in each house during cooking period and in indoor and outdoor during cooking period

Compound	House 1-cooking (n=19)	House 2-cooking (n=15)	Indoor-cooking [a)] (n=22)	Outdoor-cooking [a)] (n=12)
Flu	0.925**	-0.093	0.820**	0.287
Pyr	0.926**	0.339	0.893**	0.322
BaA	0.928**	0.296	0.902**	0.448
Chr	0.875**	0.586*	0.876**	0.329
BbF	0.879**	0.711*	0.876**	0.273
BkF	0.889**	0.639*	0.884**	0.280
BaP	0.867**	0.314	0.833**	0.280
BghiPe	0.870**	0.525*	0.857**	0.252
IDP	0.793**	0.404	0.773**	0.315
Total PAHs	0.947**	0.457	0.929**	0.280
9-NA	0.725**	0.532*	0.678**	0.608*
2-NFR	0.747**	0.739**	0.737**	0.378
1-NP	0.958**	0.568*	0.930**	0.049
2-NP	0.784**	0.625*	0.744**	0.343
Total NPAHs	0.781**	0.596*	0.745**	0.671*

[a)] The correlation coefficients in the columns of indoor-cooking and outdoor-cooking were calculated using the data from both houses.

**Correlation is significant at the 0.01 level (2-tailed)

*Correlation is significant at the 0.05 level (2-tailed)

2.3.4 Carcinogenic risk assessment

Carcinogenic risk was estimated using toxic equivalent factors (TEFs) based on BaP and was calculated according to the following equation (Eq. 1):

$$\text{Carcinogenic risk} = [\sum [\text{PAH}]i \ \text{TEF}_{\text{PAH}i} + \sum [\text{NPAH}]i \ \text{TEF}_{\text{NPAH}i}] \times \text{UR}_{\text{BaP}} \qquad \text{(Eq. 1)}$$

where [PAH]i and [NPAH]i are the atmospheric concentrations of individual PAHs and NPAHs in units of ng/m^3 (Albinet et al. 2008; OEHHA 2011; Bandowe et al. 2014). $\text{TEF}_{\text{PAH}i}$ and $\text{TEF}_{\text{NPAH}i}$ are the TEF values of the individual PAHs and NPAHs, respectively (Table 2.13). UR_{BaP} (unit risk) is defined as the number of people at a risk of contracting cancer from inhalational exposure to 1 ng/m^3 of BaP over a lifetime of 70 years and has a value of 1.1×10^{-6} (ng/m^3)$^{-1}$. For values below the LOQ, we used a value of half the LOQ, as suggested by the US EPA (US EPA 2000).

The total excess lifetime risk of lung cancer from exposure to PAHs and NPAHs was calculated from nine PAHs and three NPAHs. Fig. 2.11 shows the estimated carcinogenic risk in each house and the sampling period. The total risks of indoor exposure during cooking periods were much higher than those in noncooking periods or at outdoor sites. The total risks due to indoor air during cooking in House 1 were much higher than those in House 2 because of the differences in house structure and fuel consumption. The highest risk arose during indoor cooking in House 1 on the first study day with values of 1.9×10^{-3}, suggesting risks 3,800 and 16 times higher than those in noncooking periods and at outdoor sites, respectively. The highest risk found in this study was notably higher than that found in rural Chinese homes using biomass for cooking (7.8×10^{-4}) (Ding et al. 2012) or cooking with wood stoves in rural Sierra Leone (6.8×10^{-4}) (Taylor and Nakai 2012). Furthermore, the carcinogenic risk associated with indoor biomass burning in our study exceeded the WHO recommended threshold of 10^{-5} (Ramirez et al. 2011), which suggests adverse health effects. A carcinogenic risk greater than 1×10^{-4} is classified as a "definite risk," where there is compelling and convincing evidence of significant risks to the general population (Sexton et al. 2007). The results revealed serious health risks from the inhalation of PAHs and NPAHs during cooking with the potential to increase the incidence of cancer in this area.

Table 2.13 Toxic equivalent factors (TEFs) for PAHs and NPAHs

Compound	**TEF** [a)]								
PAHs	**Flu**	**Pyr**	**BaA**	**Chr**	**BbF**	**BkF**	**BaP**	**BghiPe**	**IDP**
	0.001	0.001	0.1	0.01	0.1	0.1	1	0.01	0.1
NPAHs	**1-NP**	**4-NP**	**6-NC**						
	0.1	0.1	10						

[a)] Data from Albinet et al. (2008), OEHHA (2011), and Bandowe et al. (2014)

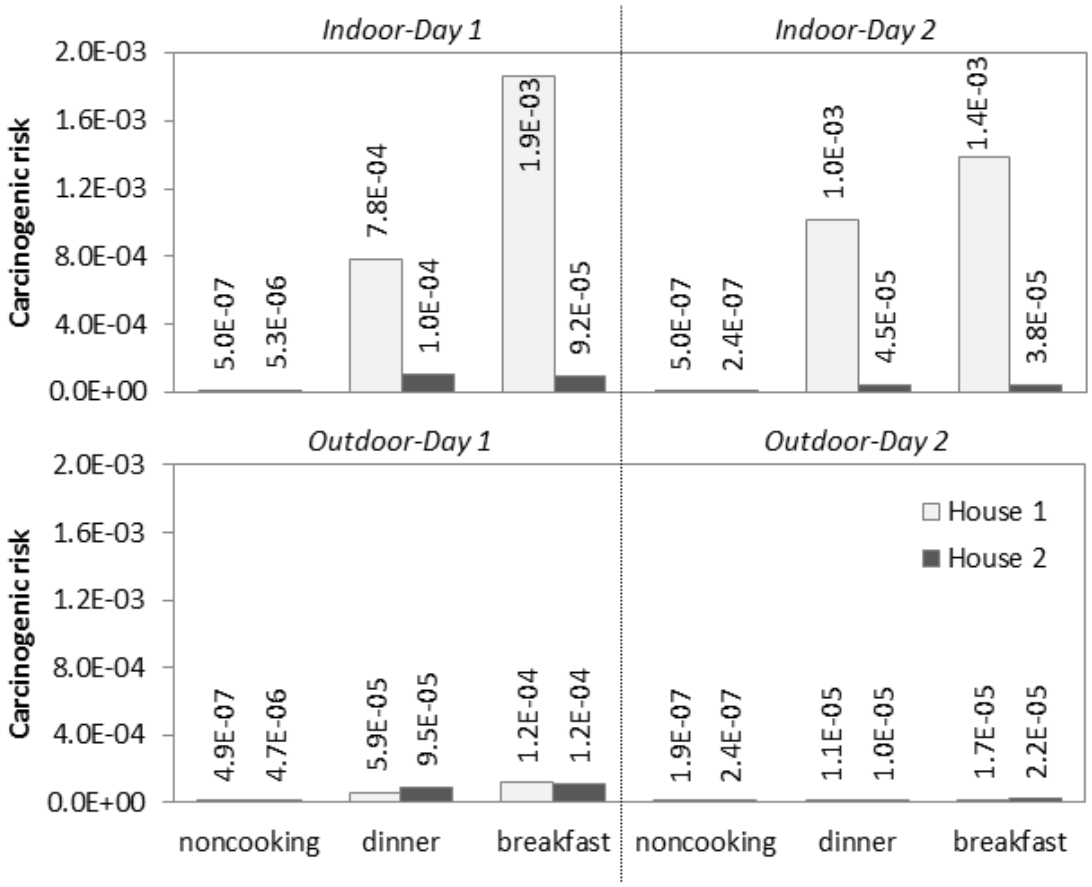


Fig. 2.11 Carcinogenic risk calculated indoors and outdoors at Houses 1 and 2 during cooking (dinner and breakfast) and noncooking periods.

2.4 Conclusions

This study was the first investigation of indoor air pollution from open-fire cooking with wood as the fuel in a rural area of Chiang Mai, Thailand, although it was short-term monitoring and conducted in only two houses. Biomass burning was shown to be the main source of pollution. Severe $PM_{2.5}$, PAH, and NPAH contamination of the indoor air was observed during cooking periods. Time-dependent changes in $PM_{2.5}$ counts inside both study houses demonstrated that $PM_{2.5}$ level increased during cooking periods. The indoor PAH levels recorded in this study were higher than those found in similar studies of homes using biomass. The indoor to outdoor ratios and diagnostic ratios using PAHs and NPAHs or carbonaceous fractions also demonstrated the large contribution made by biomass burning to indoor air pollution. In all the

NPAHs detected, 9-NA was the most abundant, originating mainly in the incomplete combustion of organic materials. The exceptions were 2-NFR and 2-NP, which were formed as secondary NPAHs in the atmosphere. That wood combustion was the main source of PAHs and NPAHs in the study area, and this was confirmed by the correlation coefficient ($p < 0.01$) between PAHs and NPAHs, with LG as the tracer. The carcinogenic risk from the indoor air exceeded the guidelines for human health, suggesting that inhalation exposure to emissions of biomass burning through open-fire for cooking may increase the risk of lung cancer in this area. Local communities should be informed and educated about risks which come from inhalation exposure to indoor PM, PAHs, and NPAHs originated from biomass combustion for daily cooking. Ways of improving indoor air quality in households that use biomass as fuel should be found to reduce exposure and prevent health problems arising in the future. Residential environments may be improved by the adoption of high-efficiency wood stoves, which can reduce emissions both by controlling air flow and temperature. The installation of ventilation systems and the transition to cleaner fuels such as LPG or electricity are also effective.

CHAPTER 3

Personal exposure of rural residents to polycyclic aromatic hydrocarbons and their nitro derivatives in northern Thailand

3.1 Introduction

Airborne particulate matter (PM) is emitted by the combustion of organic materials and is formed in the atmosphere from gaseous precursors; it has been designated as carcinogenic to humans (Group 1) by the IARC (IARC 2013). The WHO reported that around seven million deaths are caused by air pollution, which equates to approximately one in eight of all deaths (WHO 2014b). Air pollution is composed of various components in gaseous and solid phases. High levels of fine particulate matter, $PM_{2.5}$ (atmospheric particles with a diameter <2.5 μm), are particularly associated with high numbers of deaths from heart disease and stroke as well as respiratory illnesses and cancers (WHO 2017). Furthermore, long-term exposure to $PM_{2.5}$ has potentially increased health risks including premature death (Pope et al. 2002).

In Thailand, dramatic economic growth has caused numerous environmental issues including air pollution problems (He et al. 2010). It was estimated by the World Bank that deaths in Thailand attributable to air pollution have increased from 31,000 in 1990 to approximately 49,000 in 2013 (*World Bank and Institute for Health Metrics and Evaluation* 2016). Previous studies have focused on air pollution from wildland fires, agricultural waste combustion, and accidental biomass burnings related to transboundary haze pollution from Thailand's neighboring countries (Pengchai et al. 2009; Oanh et al. 2011; Huang et al. 2013; Shi et al. 2014). Many provinces in the upper northern area have been annually facing air pollution, especially PM, during a dry season that was generated by open burning (e.g., rice field, agricultural waste, and forest land). This severe air pollution problem in the northern region has impacted the air quality and health of local people (Pengchai et al. 2009; Wiriya et al 2013; Phoothiwat and Junyapoon 2013; Chuesaard et al. 2014). The most severe PM_{10} pollution was observed on 14 March 2007, with the highest concentration of 383 $\mu g/m^3$ exceeding the national standard of Thailand, which is 120 $\mu g/m^3$ (Chantara 2012). This event increased the number of

patients with respiratory problems in March 2007 compared to March 2006 and 2008 (Chuesaard et al. 2014).

Polycyclic aromatic hydrocarbons (PAHs) absorbed on $PM_{2.5}$ are of great concern because they are a human health hazard and a number of them are well-known carcinogenic compounds (Bostrom et al. 2002). PAHs are generally produced through incomplete combustion or pyrolysis of organic materials (Mastral and Callen 2000). Exposure to PAHs by inhalation has been associated with an increased risk of health effects such as respiratory diseases and lung cancer (Jarvis et al. 2014). PAHs are released into the environment as a complex mixture containing their derivatives, such as nitro-polycyclic aromatic hydrocarbons (NPAHs) (Bamford et al. 2003b; Hayakawa et al. 2007). NPAHs are considered to be much more toxic than their corresponding parent PAHs as they are direct-acting mutagens and carcinogens, whereas PAHs require enzymatic activation (Durant et al. 1996; Albinet et al. 2008). NPAHs can be emitted directly from combustion sources or formed via atmospheric reactions between PAHs and atmospheric oxidants including ozone and hydroxyl and nitrate radicals (Atkinson and Arey 2007). Atmospheric PAH and NPAH levels and their sources in northern Thailand were reported in previous studies (Chantara et al. 2009; Pengchai et al. 2009; Chantara et al. 2010; Wiriya et al. 2013; Phoothiwat and Junyapoon 2013; Chuesaard et al. 2014). The most severe ambient PAH concentration in northern Thailand was reported as 29.1 ng/m^3; this was observed in Lampang province during a haze period on March 9–12, 2009 (Phoothiwat and Junyapoon 2013). There are several possible sources of air pollution in Lampang, such as coal-fired power plants, vehicle exhausts, solid waste burning, and biomass burning (Phoothiwat and Junyapoon 2013). Further information about PAHs and NPAHs in microenvironments such as indoor air is required to estimate the exposure of the residents in this area to these chemicals.

Lung cancer has been defined as a major health problem in Thailand (Kamnerdsupaphon et al. 2008). In particular, the incidence of lung cancer is significantly higher in upper northern Thailand than in other regions of the country (Bumroongkit et al. 2008; Kamnerdsupaphon et al. 2008; Wiwatanadate 2011), and the highest incidence of lung cancer was recorded in Lampang province (Wiwatanadate 2011). Pisani et al. (2006) studied the factors associated with lung cancer in Lampang and suggested smoking as an important factor. The results showed that the risk of lung cancer increased with a longer duration of smoking, a greater daily cigarette

consumption, and traditional unfiltered cigarettes. However, the causes of lung cancer in northern Thailand have not been clearly understood.

The highest incidence of lung cancer in Lampang may be attributable to environmental carcinogens derived from air pollution, such as PAHs in ambient air during haze periods (Phoothiwat and Junyapoon 2013). The observed PAH concentrations in Lampang were 1.1–9.3 times higher than those in other northern sites (Pengchai et al. 2009; Chantara et al. 2010; Wiriya et al. 2013; Chuesaard et al. 2014), whereas the concentrations of NPAHs in Lampang have not been reported before. To evaluate the exposure of the residents and important sources of their exposure, personal monitoring of hazardous chemicals is necessary in addition to stationary ambient monitoring, which shows representative data for the neighborhood. However, the actual exposure to air pollutants of the local residents during their daily life in northern Thailand has never been investigated. The present study aimed to investigate personal exposure to $PM_{2.5}$, PAHs, and NPAHs of rural residents who live a typical lifestyle in Lampang province. For more accurate measuring of inhalation exposure, $PM_{2.5}$ samples were collected near the breathing zones of each subject using personal samplers. Moreover, the contribution of combustion sources was evaluated using diagnostic ratios, and the potential cancer risks of the residents were also estimated.

3.2 Materials and methods

3.2.1 Chemicals and reagents

All reagents and chemicals for the determination of PAHs and NPAHs were obtained commercially and used without further purification. They were described in our previous chapter.

3.2.2 Sampling site and procedures

This study was carried out in a rural and an urban area of Lampang province, which is the third largest town in northern Thailand (Fig. 3.1). The rural area was located in Thoen district (17°36′42″ N, 99°12′57″E), which was about 100 km from the central area. Thoen has a population of approximately 60,000. Personal sampling for the residents was conducted for three days between 9th and 12th of March in 2013. Fifteen rural residents were willing to participate in

our study. They were considered with respect to smoking habits and then divided into three groups: smoker (n = 3), passive smoker (n = 4), and nonsmoker (n = 8). All subjects were asked to carry personal air samplers throughout the day except when sleeping, showering, using the restroom, or working in the rice field. During these activities, the subjects were required to keep the samplers close to them. They carried out the sampling three times over 24 h. Moreover, they were required to be interviewed using a questionnaire survey in order to record their activities each day from waking until bedtime. Information on age, gender, occupation, transportation, smoking and cooking activity and frequency, and household fuel types were recorded by questionnaire survey. In the rural area used for sampling, solid fuel such as charcoal is usually burned in traditional open fire stoves, and LPG is also used for daily cooking. Hand-rolled tobacco cigarettes and cigars (Khiyo) are popular among Thai rural residents including the rural subjects who participated in our study. A Khiyo is a cigar made of tobacco mixed with additives. One of the additives is usually tamarind bark. The wrappers are made from sun-dried banana leaves or fresh banana leaves dried rapidly on a hot plate. In addition to personal monitoring, stationary air samples were collected in a central area of the city on 8th March.

All samples were collected using a personal air sampler with an ATPS-20H impactor (Shibata Sci. Tech., Tokyo, Japan) connected to a portable MP-∑300 pump (Shibata) that provides an air flow of 1.5 L/min. Particles >10 μm in diameter were collected on a metal impaction plate coated with grease immediately downstream of the inlet. Particles that were >2.5 μm and <10 μm passed through the impaction plate and were collected on a 10 mm Fiberfilm filter (heat resistant borosilicate glass fiber coated with fluorocarbon, T60A20, Pall Life Sciences, Ann Arbor, MI, USA) with a 50% cutoff point of 10 μm ($PM_{2.5–10}$). Particles <2.5 μm were collected on a 20 mm Fiberfilm filter with a 50% cutoff point of 2.5 μm ($PM_{2.5}$). Forty-five personal samples were obtained. The PM mass concentrations were obtained by weighing the filters before and after sampling, after a storage period (24 h) in a temperature- and humidity-controlled room (ambient temperature: 23°C ± 0.2°C; relative humidity: 35% ± 5%), with an ultra-microbalance (sensitivity 0.1 μg, UMX-2, Mettler-Toledo, Inc., Columbus, OH, USA) (Miller-Schulze et al. 2010). The filter samples were stored at -20°C until analysis.

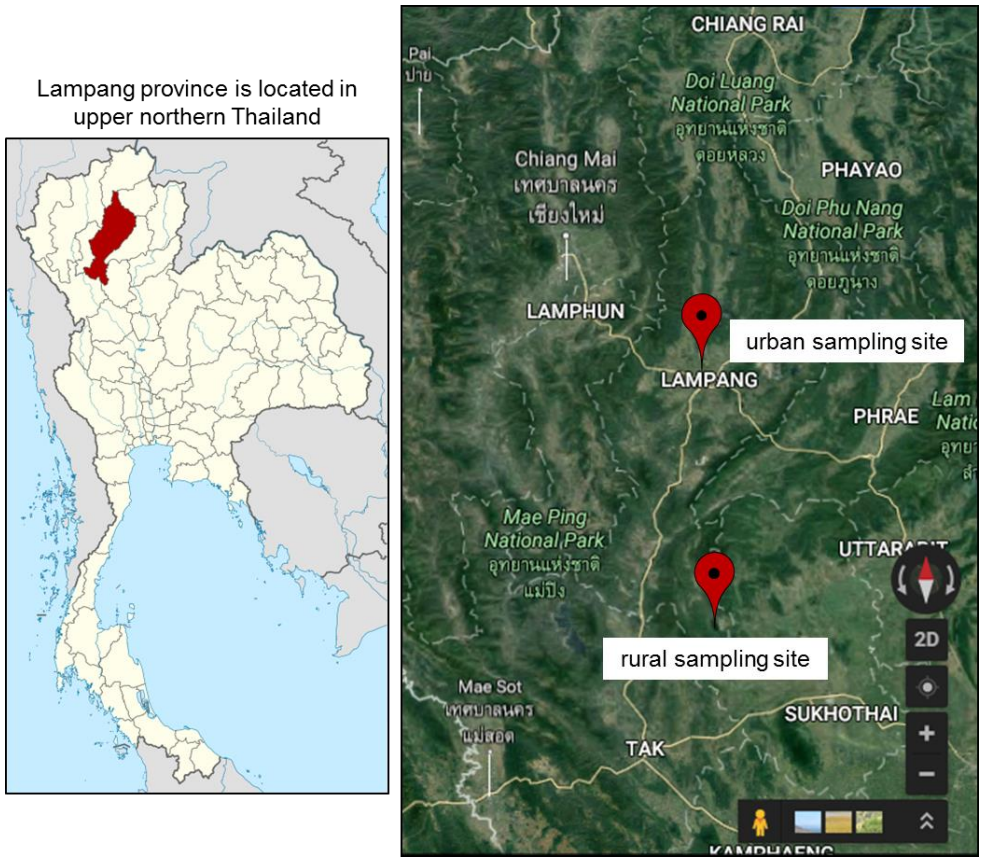


Fig. 3.1 Sampling locations at rural and urban areas in Lampang province, Thailand.

3.2.3 Sample preparation and analysis

The $PM_{2.5}$ filter samples were spiked with internal standards (Pyr-d_{10}, BaA-d_{12}, BaP-d_{12}, 6-NC-d_{11}) prior to extraction with 5 mL of dichloromethane (DCM) under sonication for 15 min. The extraction procedure was repeated three times with a final volume of 15 mL. The extracts were evaporated until completely dry and then redissolved in 450 µL of ethanol. The solutions were filtered through a membrane filter (HLC-DISK 3 with pore a size of 0.45 µm, Kanto Chemical Co., Inc., Tokyo, Japan) and then kept at -20°C until analysis. The extract was injected into an HPLC system with an injection volume of 100 µL for PAH and NPAH determination.

Ten PAHs (Flu, Pyr, BaA, Chr, BbF, BkF, BaP, DBA, BghiPe, and IDP) and eleven NPAHs (9-NPh, 2-NA, 9-NA, 2-NFR, 3-NFR, 1-NP, 2-NP, 4-NP, 6-NC, 7-NBaA, and 6-NBaP) were analyzed by HPLC with a fluorescence detector (HPLC-FL), which was developed based

on previous reports (Tang et al. 2005; Chuesaard et al. 2014). The system comprised four HPLC pumps (LC-20AD), a system controller (CBM-20A), a degasser (DGU-20A5), an auto sample injector (SIL-20AC), a column oven (CTO-20AC), a six port switching valve, and a fluorescence detector (RF-20A xs); all the components were from Shimadzu (Kyoto, Japan). The NPAHs were purified using a clean-up column (Cosmosil 5NPE, 150 × 4.6 mm i.d. 5 μm, Nacalai Tesque, Kyoto, Japan) with a guard column and were then reduced to their amino derivatives by using a reduction column (NPpak-RS, 10 × 4.0 mm JASCO, Tokyo, Japan) at 80 °C. The mobile phase in the clean-up column and reduction column was acetate buffer (pH 5.5)/ethanol (5/95, v/v) with a flow rate of 0.2 mL/min. The mobile phase eluted from the reduction column was mixed with 30 mM ascorbic acid at a flow rate of 1.6 mL/min before entering a concentration column (Spheri-5 RP-18, 30 × 4.6 mm i.d. 5 μm, Perkin Elmer, MA, USA) at the switching valve. A fraction of the amino derivative was trapped on the concentration column using the switching valve with a switching time of 8.2–17.0 min. The concentrated fraction was passed through two separation columns (Inertsil ODS-P, 250 × 4.6 mm i.d. 5 μm, GL, Sciences, Tokyo, Japan) in tandem. All columns were maintained at 20 °C. A gradient elution of the separation columns was performed using 10 mM imidazole buffer (pH 7.6) as eluent A and acetonitrile as eluent B. The gradient conditions were as follows: 0–8.20 min (B conc. 20%, 0.5 mL/min), 8.20–17.00 min (B conc. 70%, 0.5–0.8 mL/min), 17.00–27.00 min (B conc. 70%, 0.8 mL/min), 27.01–47.01 min (B conc. 80%, 0.9 mL/min), 47.01–62.00 min (B conc. 100%, 0.9–1.8 mL/min), 62.00–90.00 min (B conc. 100%, 1.8 mL/min), 90.00–95.10 min (B conc. 20%, 1.0–0.5 mL/min), and 95.10–103.00 min (B conc. 20%, 0.5 mL/min). The eluted fraction from the separation columns was detected with a fluorescence detector. The wavelengths used for PAHs and NPAHs analysis were as follows. Channel 1: 0–35.00 min (Ex. 247 nm, Em. 430 nm), 35.00–37.30 min (Ex. 260 nm, Em. 490 nm), 37.30–40.00 min (Ex. 300, nm Em. 530 nm), 40.00–48.50 min (Ex. 273 nm, Em. 437 nm), 48.50–53.25 min (Ex. 286 nm, Em. 433 nm), 53.25–79.50 min (Ex. 283 nm, Em. 513 nm), and 79.50–90.00 min (Ex. 294 nm, Em. 482 nm) and Channel 2: 0–35.00 min (Ex. 260 nm, Em. 490 nm), 35.0–38.50 min (Ex. 283 nm, Em. 513 nm), 38.50–41.50 min (Ex. 360 nm, Em. 430 nm), 41.50–48.50 min (Ex. 300 nm, Em. 475 nm), 48.50–55.55 min (Ex. 331 nm, Em. 392 nm), and 55.55–90.00 min (Ex. 264 nm, Em. 407 nm).

3.2.4 Quality control and data analysis

Quantitative analysis of PAHs and NPAHs was based on the peak area ratios of the analytes to the deuterated internal standards. Validation of the analytical methods was conducted using spiked $PM_{2.5}$ samples. The spiked amounts were ca three times higher than the concentration observed in the sample. For analytes that were undetectable in the unspiked sample, the spiked concentration was based on the limit of quantification (LOQ). Table 3.1 shows result of method validation of PAHs and NPAHs. The accuracy was 100% ± 20% for all analytes. The precision was favorable with an RSD of 10% or less for all analytes. The recoveries of the deuterated internal standards (Pyr-d_{10}, BaA-d_{12}, BaP-d_{12}, and 6-NC-d_{11}) were between 81% and 117%. The LOQ, calculated with a signal-to-noise ratio of 10:1 was 0.001–0. 021 ng/m^3 for PAHs and 0.58–30.4 pg/m^3 for NPAHs. Data analysis was conducted using SPSS 23 software (IBM Inc., North Castle, NY, USA). The kruskal–Wallis test was used to compare concentrations of $PM_{2.5}$, PAHs, and NPAHs among the three groups, and then pairwise comparisons using the Dunn-Bonferroni approach were carried out for dependent variables for which the Kruskal–Wallis test is significant.

Table 3.1 Result of analytical method validation of PAHs and NPAHs (n = 3)

PAHs (ng/m^3)	Spiked concentration	PAH concentration (mean ± SD)	Accuracy (%)	Precision (%RSD)
Flu	0	0.05 ± 0.002		4.42
	2.71	2.65 ± 0.05	96	1.95
Pyr	0	0.08 ± 0.002		2.93
	1.35	1.45 ± 0.02	101	1.59
BaA	0	0.05 ± 0.000		0.34
	1.36	1.44 ± 0.01	103	0.97
Chr	0	0.06 ± 0.001		1.28
	1.36	1.44 ± 0.02	102	1.17
BbF	0	0.10 ± 0.001		1.34
	2.71	2.67 ± 0.05	95	1.96
BkF	0	0.05 ± 0.000		0.74
	1.36	1.36 ± 0.02	97	1.44
BaP	0	0.07 ± 0.002		2.58
	1.36	1.44 ± 0.004	101	0.26
DBA	0	ND [a]		ND
	2.71	2.85 ± 0.04	105	1.49
BghiPe	0	0.16 ± 0.003		1.73
	2.71	2.95 ± 0.05	103	1.66
IDP	0	0.11 ± 0.004		3.76
	1.36	1.48 ± 0.02	101	1.26

[a] ND = not detected

NPAHs (pg/m3)	Spiked concentration	NPAHs concentration mean ± SD	Accuracy (%)	Precision (%RSD)
9-NPh	0	ND		ND
	10.3	0.76 ± 0.42	112	3.63
2-NA	0	ND		ND
	6.53	0.47 ± 0.08	110	1.07
3-NFR	0	ND		ND
	2.77	0.19 ± 0.04	118	1.27
4-NP	0	ND		ND
	3.01	0.20 ± 0.02	110	0.67
2-NP	0	0.88 ± 0.02		2.47
	3.95	0.29 ± 0.08	102	1.71
6-NC	0	ND		ND
	1.8	0.10 ± 0.03	103	1.78
6-NBaP	0	ND		ND
	2.15	0.12 ± 0.09	108	4.05
9-NA	0	5.53 ± 0.31		5.6
	38.7	3.40 ± 1.41	116	2.73
2-NFR	0	11.2 ± 0.16		1.4
	47	3.54 ± 0.28	102	0.47
1-NP	0	0.76 ± 0.03		3.32
	3.21	0.24 ± 0.05	102	1.13
7-NBaA	0	ND		ND
	2.05	0.17 ± 0.06	94	1.95

3.3 Results and discussions

3.3.1 Personal inhalation exposure levels

The personal inhalation exposure of fifteen rural residents in Lampang was measured over 24 h for three days. The urban ambient air collected in the center of the city was used as a reference. The levels of $PM_{2.5}$, ∑PAHs, and ∑NPAHs in the personal exposure samples and urban ambient air are shown in Fig. 3.2. The details of their concentrations are shown in Tables 3.2 and 3.3. Their concentrations and personal exposure varied considerably among participants due to the differences in personal lifestyle in addition to the levels of air pollution in the residential area. Even for the same subject, the concentrations were different on each sampling day. The level of $PM_{2.5}$ in personal inhalation exposure ranged from 44.4 to 316 μg/m^3. Almost all personal $PM_{2.5}$ levels were 1.0–6.8 times higher than in the sample collected at the urban site (46.2 μg/m^3) (Tables 3.2 and 3.3). The lowest level of personal $PM_{2.5}$ on the 1st day (44.4 μg/m^3) was higher than the maximum levels recommended by the WHO (25 μg/m^3) and the EPA (35 μg/m^3) (US EPA 2013; WHO 2016b).This indicates that rural residents are exposed to $PM_{2.5}$, which can cause adverse health effects. The ΣPAHs and ΣNPAHs in the personal inhalation exposure samples ranged from 4.2–224 ng/m^3 and 120–1,449 pg/m^3, respectively, which were 1.4–76 and 4.0–48 times higher than that in urban ambient air (Tables 3.2 and 3.3). The observed high levels of personal exposure caused by microenvironments in the residents' lives indicated the unreliability of estimating their exposure from the results of stationary sampling alone. Investigations of personal inhalation exposure to residential solid fuel (wood, peat, coal, and honeycomb briquette) combustion in rural populations in Shanxi and Hubei, China show daily means of 2,000 ng/m^3 (Σ16PAHs) (Chen et al. 2016) and 922 ng/m^3 (Σ15PAHs) (Lin et al. 2016). Although gas-phase PAHs having low toxicity account for a large portion of the total concentrations in the reports and only 10 PAHs in the particulate phase were determined in this study, the PAH exposure of the Thai subjects was lower than that of the particulate phase were determined in this study, the PAH exposure of the Thai subjects was lower than that of the Chinese residents. In this study, the use of charcoal open-fires for cooking was influential as an indoor source of PAHs. On the other hand, our ΣNPAH level for all subjects was comparable to reports from China, where a level of 470 pg/m^3 is reported by Chen et al. (2017) and 171 pg/m^3 by Shen et al. (2016).

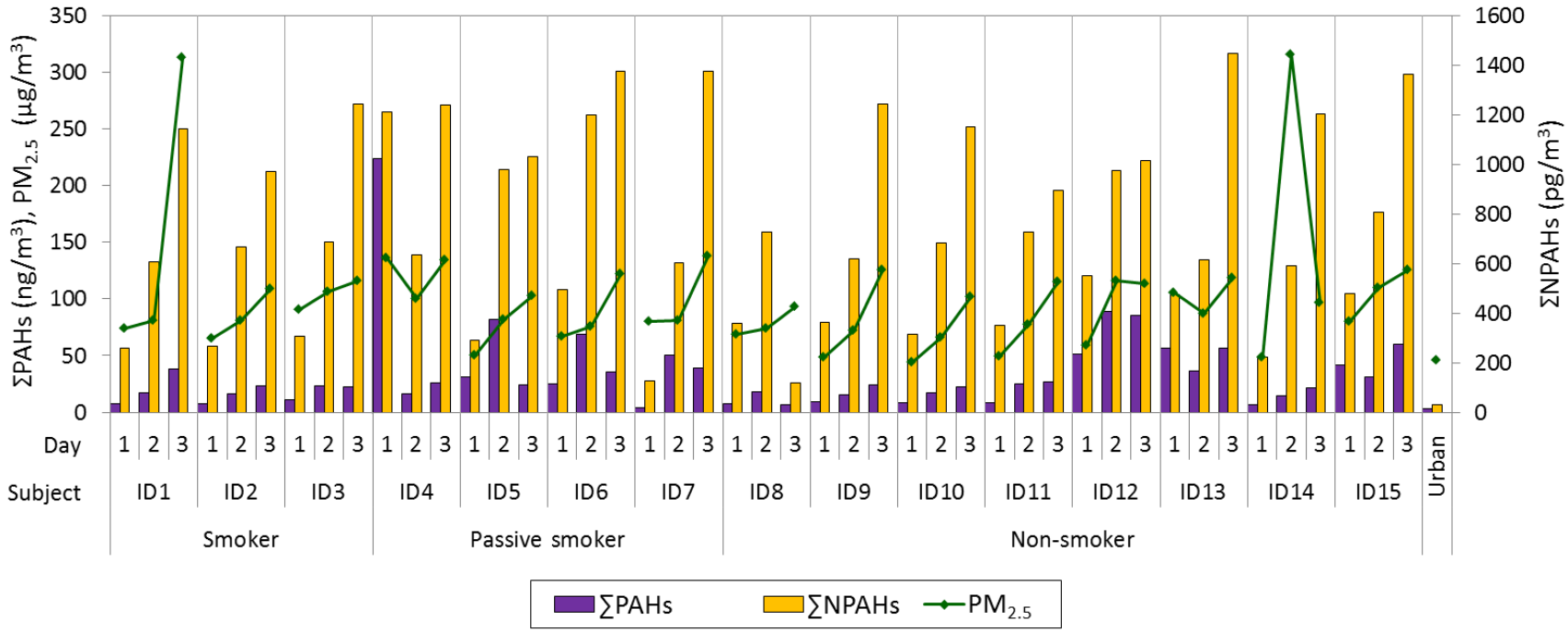


Fig. 3.2 Concentrations of $PM_{2.5}$, ΣPAHs, and ΣNPAHs in personal samples collected by fifteen rural residents for three days and urban ambient air.

Table 3.2 Concentration of PM ($PM_{2.5}$, $PM_{2.5-10}$, and PM_{10}), PAHs and NPAHs in rural resident samples

Subject	Smoker									Passive smoker											
	ID1			ID2			ID3			ID4			ID5			ID6			ID7		
Sampling date	**1st**	**2nd**	**3rd**	**1st**	**2nd**	**3rd**	**1st**	**2nd**	**3rd**	**1st**	**2nd**	**3rd**	**1st**	**2nd**	**3rd**	**1st**	**2nd**	**3rd**	**1st**	**2nd**	**3rd**
PM (μg/m³)																					
$PM_{2.5}$	73.8	80.7	313	65.5	80.8	109	90.8	106	116	136	100	134	50.7	81.8	103	67.4	75.8	122	80.3	80.8	138
$PM_{2.5-10}$	21.6	13.9	28.8	- [a]	11.7	29.6	5.61	11.6	18.2	6.68	15.1	61.1	4.40	17.6	43.2	5.58	19.1	44.0	26.6	23.9	24.4
PM_{10}	95.4	94.5	342	-	92.5	139	96.4	118	135	143	115	195	55.1	99.4	147	73.0	94.9	166	107	105	162
PAHs (ng/m³)																					
Flu	0.26	0.70	3.05	0.28	0.70	0.72	0.48	0.92	0.84	8.02	0.51	0.91	0.76	2.10	0.76	0.86	3.77	1.93	0.17	2.44	1.50
Pyr	0.23	0.63	2.71	0.26	0.61	0.62	0.38	0.89	0.76	16.6	0.42	0.90	0.77	2.84	0.72	0.42	2.99	1.71	0.18	4.11	1.45
BaA	0.27	0.81	4.29	0.32	0.70	1.32	0.61	1.38	1.54	40.8	0.76	1.82	3.93	12.9	2.10	2.93	12.9	4.14	0.23	5.48	4.31
Chr	0.48	1.41	5.19	0.55	1.22	2.24	0.91	2.19	2.48	31.0	1.29	2.77	3.58	10.3	2.61	3.12	12.8	4.59	0.37	4.94	4.80
BbF	0.81	2.00	3.45	0.84	1.89	2.61	1.18	2.47	2.54	15.3	1.91	3.00	3.70	7.83	2.83	2.44	5.32	3.22	0.50	5.44	3.87
BkF	0.41	1.14	1.62	0.46	1.07	1.40	0.63	1.38	1.34	14.8	1.05	1.64	2.33	5.40	1.51	1.53	3.41	1.78	0.24	3.35	2.10
BaP	1.17	2.71	5.83	1.19	2.61	3.85	1.74	3.84	3.69	41.7	2.73	4.44	5.45	14.9	4.21	4.44	10.4	5.72	0.61	8.34	6.54
DBA	NQ [a]	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	ND	NQ	NQ
BghiPe	2.09	4.44	6.79	2.17	4.20	5.60	2.62	5.79	5.30	25.6	4.05	5.59	5.53	13.3	5.27	4.74	9.81	6.63	1.00	8.20	7.64
IDP	1.65	3.13	5.03	1.76	3.16	4.66	2.22	4.43	4.07	29.7	3.14	4.48	5.17	12.2	4.49	4.10	7.74	5.66	0.88	8.09	6.38
Total PAHs	7.36	17.0	38.0	7.84	16.2	23.0	10.8	23.3	22.5	224	15.9	25.5	31.2	81.9	24.5	24.6	69.1	35.4	4.19	50.4	38.6
NPAHs (pg/m³)																					
9-NPh	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	20.1	NQ	NQ	ND	NQ	NQ
2-NA	23.5	34.4	NQ	18.2	35.2	43.2	26.4	46.1	38.0	160	30.6	40.3	24.5	78.4	35.7	30.3	70.8	47.5	10.7	48.0	47.7
9-NA	165	389	883	177	446	595	195	428	835	720	413	834	197	689	724	343	1021	913	76.9	428	891
2-NFR	43.9	117	151	46.5	118	239	60.9	150	267	171	122	261	44.0	134	192	67.9	NQ	300	25.7	77.9	318
3-NFR	NQ	NQ	NQ	ND [c]	NQ	NQ	NQ	ND	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	ND	NQ	NQ
1-NP	1.22	3.90	27.5	1.29	3.44	4.28	1.53	2.54	4.23	31.0	3.15	3.74	2.68	8.71	3.80	4.60	14.7	5.31	1.81	6.10	6.10
2-NP	20.1	54.0	80.9	20.3	54.8	89.6	21.2	60.8	93.8	38.6	53.8	86.6	14.8	44.3	63.6	28.5	93.1	92.8	11.6	30.2	95.2
4-NP	3.47	7.39	NQ	3.64	9.17	NQ	ND	ND	5.21	47.5	9.88	12.1	6.91	23.7	9.97	NQ	NQ	14.3	ND	11.2	15.8
6-NC	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	ND	NQ	NQ
7-NBaA	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	ND	NQ	ND	ND	ND	ND	ND	NQ	ND	ND	ND	ND
6-NBaP	NQ	NQ	NQ	ND	NQ	NQ	NQ	NQ	NQ	41.8	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	ND	NQ	NQ
Total NPAHs	258	606	1142	267	666	971	305	687	1243	1210	633	1238	290	978	1029	494	1199	1373	127	601	1374

[a] NQ = not quantified
[b] Error
[c] ND = not detected
[d] LOQ = lower than limit of detection

Table 3.2 Continue

Subject	Non-smoker																							
	ID8			ID9			ID10			ID11			ID12			ID13			ID14			ID15		
Sampling date	**1st**	**2nd**	**3rd**	**1st**	**2nd**	**3rd**	**1st**	**2nd**	**3rd**	**1st**	**2nd**	**3rd**	**1st**	**2nd**	**3rd**	**1st**	**2nd**	**3rd**	**1st**	**2nd**	**3rd**	**1st**	**2nd**	**3rd**
PM (μg/m^3)																								
$PM_{2.5}$	68.9	73.9	93.0	49.0	72.7	126	44.4	66.5	102	49.5	77.8	116	58.8	116	114	106	87.5	118	48.5	316	96.6	80.0	110	126
$PM_{2.5-10}$	19.5	12.4	17.8	13.0	11.5	21.5	5.17	28.0	24.5	9.22	10.6	19.2	21.2	19.8	36.9	52.6	29.0	31.5	10.3	70.7	17.4	18.2	21.0	14.4
PM_{10}	88.4	86.3	111	62.0	84.2	147	49.6	94.5	127	58.8	88.4	135	79.9	136	151	158	116	150	58.8	386	114	98.1	131	140
PAHs (ng/m^3)																								
Flu	0.28	0.63	0.64	0.28	0.51	0.83	0.25	0.64	0.91	0.25	0.86	1.03	0.87	2.46	4.10	2.05	1.43	2.26	0.23	0.58	0.87	1.51	1.09	2.00
Pyr	0.18	0.63	1.22	0.22	0.52	0.75	0.22	0.66	0.92	0.24	0.88	1.16	1.28	4.68	6.85	1.14	1.09	2.33	0.20	0.50	0.78	2.24	1.10	1.89
BaA	0.19	1.03	0.64	0.41	0.67	1.65	0.36	0.90	1.76	0.29	1.08	2.05	7.42	11.8	12.8	9.06	1.56	6.39	0.21	0.84	1.30	4.89	3.29	6.04
Chr	0.37	1.54	0.79	0.69	1.16	2.61	0.57	1.42	2.49	0.51	1.92	3.12	6.03	10.8	10.0	7.79	2.45	6.32	0.38	1.27	2.18	5.24	4.05	6.73
BbF	0.75	2.09	0.62	1.04	1.72	2.72	0.89	1.86	2.69	0.87	3.20	3.04	5.27	9.80	7.43	4.88	4.66	5.70	0.69	1.63	2.47	4.64	3.64	6.58
BkF	0.39	1.20	0.28	0.56	0.98	1.42	0.51	1.07	1.40	0.49	1.76	1.63	3.60	6.47	4.72	3.12	2.69	3.42	0.35	0.91	1.32	2.59	2.09	3.80
BaP	1.13	2.92	0.58	1.38	2.38	3.99	1.28	2.66	3.61	1.29	4.09	4.15	9.39	14.9	14.1	10.7	7.29	10.6	0.99	2.47	3.45	6.99	4.85	11.1
DBA	NQ	NQ	ND	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ
BghiPe	2.10	4.70	1.29	2.46	4.11	5.54	2.24	4.30	4.91	2.51	6.37	5.73	9.13	13.8	12.3	8.73	8.39	10.3	1.87	3.60	4.87	6.83	6.02	11.2
IDP	1.80	3.52	0.78	1.94	3.05	4.46	1.83	3.18	3.97	1.96	4.55	4.62	7.87	13.8	12.6	8.66	6.67	9.52	1.52	2.85	3.86	6.69	4.93	11.0
Total PAHs	7.17	18.3	6.84	8.97	15.1	24.0	8.16	16.7	22.7	8.40	24.7	26.5	50.9	88.5	85.0	56.1	36.2	56.9	6.46	14.7	21.1	41.6	31.1	60.3
NPAHs (pg/m^3)																								
9-NPh	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	60.8	NQ	NQ
2-NA	20.7	33.4	9.40	21.9	30.5	39.1	19.0	33.8	37.4	19.4	51.9	39.0	43.2	107	90.7	35.7	43.5	64.5	12.8	24.8	36.8	38.8	40.6	61.4
3-NFR	NQ	NQ	6.08	NQ	NQ	NQ	1.72	NQ	NQ	1.76	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	ND	NQ	NQ
4-NP	3.69	6.39	ND	3.96	4.27	LOQ	4.00	7.64	8.89	3.91	9.87	NQ	13.2	36.8	30.8	NQ	20.3	21.2	2.35	6.27	10.1	ND	NQ	NQ
2-NP	24.4	62.2	7.05	27.3	52.7	87.0	22.1	57.9	78.0	25.0	69.6	82.1	25.1	67.2	73.9	53.7	42.7	94.3	17.8	45.0	82.6	32.8	70.3	101
6-NC	ND	NQ	ND	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	ND	NQ	NQ	NQ	NQ	NQ
6-NBaP	5.20	NQ	LOQ [d)]	2.53	NQ	5.51	NQ	NQ	NQ	2.72	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	ND	3.83	NQ	NQ	NQ	NQ
9-NA	214	465	53.6	249	414	841	215	448	800	243	440	531	389	473	548	298	394	917	151	364	836	270	465	844
2-NFR	52.0	156	30.7	56.1	116	267	49.5	133	222	51.5	152	231	77.4	284	266	77.7	107	345	36.9	146	233	72.2	218	350
1-NP	1.35	3.80	12.8	2.06	2.94	4.29	1.63	3.55	3.99	1.82	4.37	10.7	4.46	9.33	6.34	8.34	4.56	6.92	1.58	2.21	4.75	5.63	13.4	6.42
7-NBaA	35.5	NQ	ND	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	NQ	ND	NQ	NQ	ND	ND	ND	NQ	NQ	NQ	NQ	NQ	NQ
Total NPAHs	357	727	120	363	620	1244	313	683	1150	349	728	894	552	976	1016	473	613	1449	223	592	1203	480	808	1362

Table 3.3 Mean concentrations of PM ($PM_{2.5}$, $PM_{2.5-10}$, and PM_{10}), PAHs, and NPAHs at the urban site

PM ($\mu g/m^3$)	Mean ± SD	PAHs (ng/m^3)	Mean ± SD	NPAHs (pg/m^3)	Mean ± SD
$PM_{2.5}$	46.2 ± 5.8	Flu	0.17 ± 0.02	9-NPh	ND
$PM_{10-2.5}$	10.9 ± 4.3	Pyr	0.23 ± 0.02	2-NA	LOQ
PM_{10}	57.1 ± 4.8	BaA	0.14 ± 0.02	9-NA	LOQ
		Chr	0.20 ± 0.02	2-NFR	22.0 ± 1.7
		BbF	0.30 ± 0.02	3-NFR	ND
		BkF	0.20 ± 0.01	1-NP	3.66 ± 0.6
		BaP	0.34 ± 0.04	2-NP	4.69 ± 0.9
		DBA	ND	4-NP	ND
		BghiP	0.74 ± 0.1	6-NC	ND
		IDP	0.63 ± 0.1	7-NBaA	ND
		Total PAHs	2.94 ± 0.3	6-NBaP	ND
				Total NPAHs	30.3 ± 2.5

ND = not detected
LOQ = less than limit of quantification

3.3.2 Effect of atmospheric environment on the residential area

The daily variations in monitored PM levels for almost all subjects showed a tendency to increase with time (Fig. 3.3). The observed PM_{10} tendency was similar to that of ambient PM_{10} levels (March 9–10th: 47 $\mu g/m^3$, 10–11th: 55 $\mu g/m^3$, 11–12th: 88 $\mu g/m^3$) obtained from the monitoring station of the Pollution Control Department (PCD) at a central area of Lampang (PCD 2013). Our urban PM_{10} data (57.1 $\mu g/m^3$) was consistent with the PCD data (March 8th: 66 $\mu g/m^3$), suggesting that our PM_{10} data can bear comparison with the PCD data. The personal exposure of the residents would be affected by ambient air pollution, especially severe haze during the dry season in northern Thailand. However, mean levels of PM_{10} during each day of personal exposure were markedly higher than the PCD data and our data at the urban site (Fig. 3.3). These results indicate that the rural residents are constantly exposed to PM that is derived from microenvironments through their individual activities, such as agricultural residue burning, smoking, and cooking activity in addition to ambient air pollution. PAHs showed no change in daily concentrations, whereas NPAH concentrations increased daily similarly to $PM_{2.5}$ or PM_{10} levels (Fig. 3.3). The relationships among PAHs, NPAH, and PM concentrations suggested that

different sources contributed to PAHs and NPHs and may indicate that PAHs were strongly affected by individual exposure from microenvironments such as indoor air, whereas NPAHs were affected by daily variations in the atmospheric environment in the residential area.

3.3.3 Effect of smoking behavior

Smoking behavior is an important factor with respect to personal inhalation exposure. In the present study, subjects were divided into three groups according to smoking activity: smokers, passive smokers, and nonsmokers. Fig. 3.3 shows the differences in the levels of monitored components between the above groups. The $PM_{2.5}$ concentrations around smokers and passive smokers were not significantly higher than those for nonsmokers. In general, environmental tobacco smoke (ETS) contributes to $PM_{2.5}$ levels in the surrounding microenvironment (Georgiadis et al. 2001). Handmade traditional cigarettes and cigars may undergo incomplete combustion and generate much more PM.

However, the smoking behavior of the subjects did not contribute significantly to their exposure levels of $PM_{2.5}$. The median values of ΣPAHs (17.0 ng/m^3) and ΣNPAHs (666 pg/m^3) for smokers were not significantly higher than those for non-smokers (ΣPAHs 8.28 ng/m^3; ΣNPAHs 353 pg/m^3). Their levels for passive smokers were also not significantly higher than those for nonsmokers, suggesting that passive smokers were exposed to them at almost the same levels as smokers. Exposure to PAHs through cigarette smoke is well-known (Grimmer et al. 1987; Salomaa et al. 1988; Georgiadis et al. 2001), whereas there are a few reports about NPAHs in cigarette smoke (Havey et al. 2009). Since the cigarette consumption of the subjects or their family members was limited to 4−7 cigarettes or cigars, the effects of smoking behavior may be relatively small. Moreover, passive smoking or secondhand smoking is also important in addition to active smoking with respect to inhalation exposure. In general, PAH concentrations in sidestream smoke are higher than those in mainstream smoke (Schick and Glantz 2005; Valenti et al. 2011). Some passive smoking subjects who cooked with a charcoal fire for a long time (ID4-1st, ID5, ID6, and ID7-2nd and 3rd) showed markedly higher concentrations of monitored chemicals than those observed for passive smokers who did not cook and several smokers; thus, we excluded those who cooked from the passive smoking group. The smoking behavior of the subjects in this study was not significantly reflected in their exposure to PAHs and NPAHs compared to other sources.

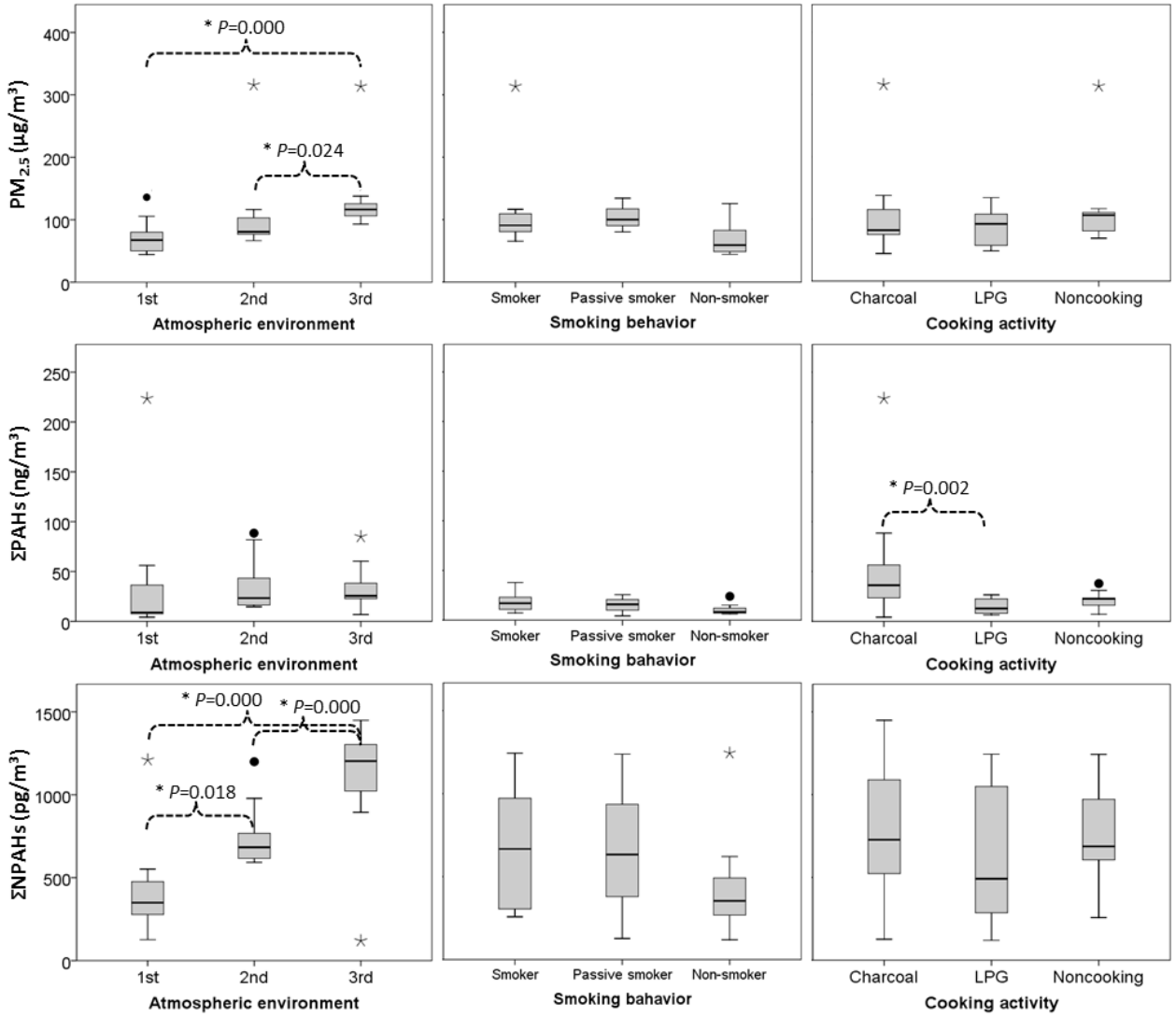


Fig. 3.3 Comparison of concentrations of $PM_{2.5}$, ΣPAHs, and ΣNPAHs among rural resident groups divided by factors related to their exposure: atmospheric environment, smoking, and cooking.

The box boundaries indicate the 25th and 75th percentiles. The horizontal line in the box indicates the median value. Whiskers extending to the highest and lowest levels indicate the 5th and 95th percentiles. The black circle and the asterisk are outliers >1.5 box lengths from the 75th percentile and >3 box lengths from the 75th percentile.

3.3.4 Effect of cooking

In term of cooking as a factor, personal exposure to $PM_{2.5}$ of household members who used a charcoal open fire was comparable with that of the subjects who used an LPG stove and non-cooking subjects (Fig. 3.3). The use of an open fire inside an inadequately ventilated room considerably raises exposure to PM and that is a serious issue in developing countries as mentioned above (Bhargava et al. 2004). The PM generation from a charcoal open fire should be less than that from fires fueled by wood, crop residues, and coal (Titcombe and Simcik 2011; Taylor and Nakai 2012; Taylor et al. 2015). On the other hand, we observed that the median values of ΣPAHs (36.3 ng/m^3) and ΣNPAHs (727 pg/m^3) for charcoal open fire users were higher than those for LPG stove users (ΣPAHs: 12.9 ng/m^3 and ΣNPAHs: 492 pg/m^3) with significance for the PAHs levels (Fig. 3.3), suggesting that charcoal as a combustion source significantly contributed to PAH exposure. The generation of PAHs and NPAHs from an LPG stove is quite small compared to that from other home sources, such as wood, coal, honeycomb briquette, and peat burned in household stoves (Lin et al. 2016; Chen et al. 2016, 2017). Therefore, rural people who are using charcoal as a cooking fuel are faced with a potential increase in their inhalation exposure risk, although this is at least better than open biomass burning using wood, crop residues, and coal. On the other hand, the use of LPG as a cleaner fuel can reduce exposure to air pollutants at home. The ΣPAHs and ΣNPAHs for non-cooking subjects were higher than those for subjects who cooked with LPG, presumably due to the existence of many smokers among the non-cooking subjects. The use of charcoal for indoor cooking seems to be lower risk when compared to wood in the chapter 2. This is consistent with Taylor and Nakai (2012).

3.3.5 Source evaluation using diagnostic ratios

PAH and NPAH ratios have been used as a tool for identification of emission sources. The 1-NP/Pyr ratio can be used as an indicator of vehicle exhaust fumes (Tang et al. 2005). The 1-NP/Pyr ratio of urban ambient air (0.016) is higher than the ratios for all of the rural subjects (Fig. 3.4), indicating that the urban area had a larger contribution from vehicle exhaust than rural residents exposed to emissions from other sources. A BaP/BghiPe ratio of less than 0.6 indicates a contribution from traffic as an emission source (Pandey et al. 1999). The BaP/BghiPe ratio in

urban ambient air (0.46) also indicated a contribution from traffic as an emission source, whereas the corresponding ratios for almost all rural residents were higher than 0.6 in addition to their low 1-NP/Pyr ratios, indicating reduced effects of traffic emission compared to that in the urban area. Phoothiwat and Janyapoon (2013) and Chuesaard et al. (2014) observed BaP/BghiPe ratios of 0.84 and 0.79 during haze periods in March 2009 and 2010, respectively, in northern Thailand. The ratios for residents were similar to those from the stationary samplings, which indicated that ambient air pollution, such as the haze emitted from open burning and forest fires during the dry season in the area, might be an underlying factor in the exposure of residents. Some of the residents had a BaP/BghiPe ratio of more than 0.8, suggesting that they were affected by sources relating to their personal lifestyle in addition to those in the surrounding ambient air. There is a negative correlation between the 1-NP/Pyr and BaP/BghiPe ratios (r = -0.498, p < 0.01) (Fig. 3.4). Two different indicators developed by different groups appear to be effective in evaluating the effects of vehicle exhaust and their combination may be useful for more accurate source evaluation.

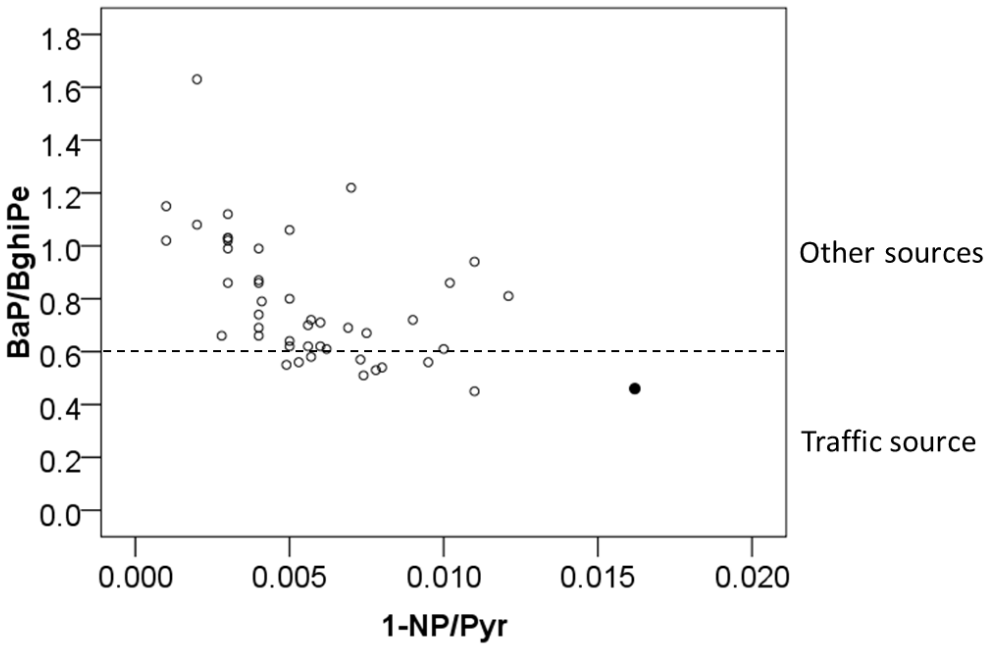


Fig. 3.4 Scatter plot of 1-NP/Pyr and BaP/BghiPe ratios of the samples for all subjects and for the urban site.

The black circle in the figure represents urban ambient air.

3.3.6 Cancer risk of personal inhalation exposure

The estimated excess cancer risk was calculated from the concentrations of nine PAHs and two NPAHs (1-NP and 4-NP) and the unit risk (UR_{BaP}) according to the method developed by Albinet et al. (2008), which is explained the detail in the previous chapter.

Fig. 3.5 shows the cancer risks of rural residents for each sampling day. The observed range of cancer risks in all subjects ($1.0 \times 10^{-6} – 6.0 \times 10^{-5}$) exceeded the USEPA guideline value of 10^{-6} (Ramirez et al. 2011). The highest risk was observed in ID4-1st day with a value of 6.0×10^{-5}, which was 9–13 times higher than the risk on days two and three for the subject. ID12 and ID13 showed a high level of cancer risk within the range $1 \times 10^{-5} – 1 \times 10^{-4}$, defined as "probable risks" (Sexton et al. 2007), throughout the study period. Subject ID12 was the owner of a restaurant. Charcoal and LPG were used as cooking fuel. Charcoal was burned continuously for cooking noodles for approximately 6–7 h a day, while LPG was used sometimes for cooking some foods. ID13 cooked daily with a charcoal open fire at home. These high risks probably resulted from cooking with a charcoal open fire as recorded in the questionnaire. However, there were some subjects who also cooked with a charcoal fire but did not have high risk levels. The emission from cooking with biomass burning depends on many factors, such as fuel consumption, time spent on cooking, fuel and cooking stove quality, and house structure and ventilation. The results revealed that charcoal burning might be a factor related to the high lung cancer incidence observed in Lampang. The cancer risks of subject ID4 on days two and three were lower than that on day one, probably because the subject changed their cooking fuel type from charcoal to LPG. This suggests that cooking with biomass (charcoal) is probably an important factor affecting this subject in terms of cancer risk due to inhalation exposure. The cancer risk associated with urban ambient air (9.5×10^{-7}) was lower than those for all rural residents. The risk associated with urban air was similar to or slightly higher than the risk associated with urban ambient air in Chiang Mai, a large city in northern Thailand, during the same season with a value of 7.0×10^{-7}, reported by Chuesaard et al. (2014). These cancer risks do not exceed the USEPA guideline value of 10^{-6} (Ramirez et al. 2011), which is classified as "uncertain risk" (Sexton et al. 2007). This indicated that the evaluation of personal exposure to PAHs and NPAHs is important in estimating the actual health risks of residents.

Comparison of the cancer risks of the subject groups divided by the source factors of environmental air and smoking status did not show clear differences as with the PAH

concentrations of the groups. Cooking activity was the most important factor with respect to inhalation exposure risk in this study. As shown in Fig. 3.6, the subjects who used a charcoal open fire for cooking has the most severe cancer risk level (1.2×10^{-5}), defined as "probable risk" (Sexton et al. 2007), and the value was three times higher than that for cooking with LPG (3.8×10^{-6}). Our observation was similar to the reported lung cancer risk of 6.4×10^{-5} for residents using charcoal as a cooking fuel (Taylor and Nakai 2012), and the risk of 8.4×10^{-6} for LPG users (Lin et al. 2016). This indicates that cooking with charcoal is a major factor in the exposure and may be related to the lung cancer incidence in this study area. A report suggests that household biomass combustion for cooking may be related to the high incidence of lung cancer in this area (Wiwatanadate 2011). The use of clean energy such as LPG as a cooking fuel can reduce the exposure of the residents to PAHs and NPAHs and consequently may decrease their cancer risk.

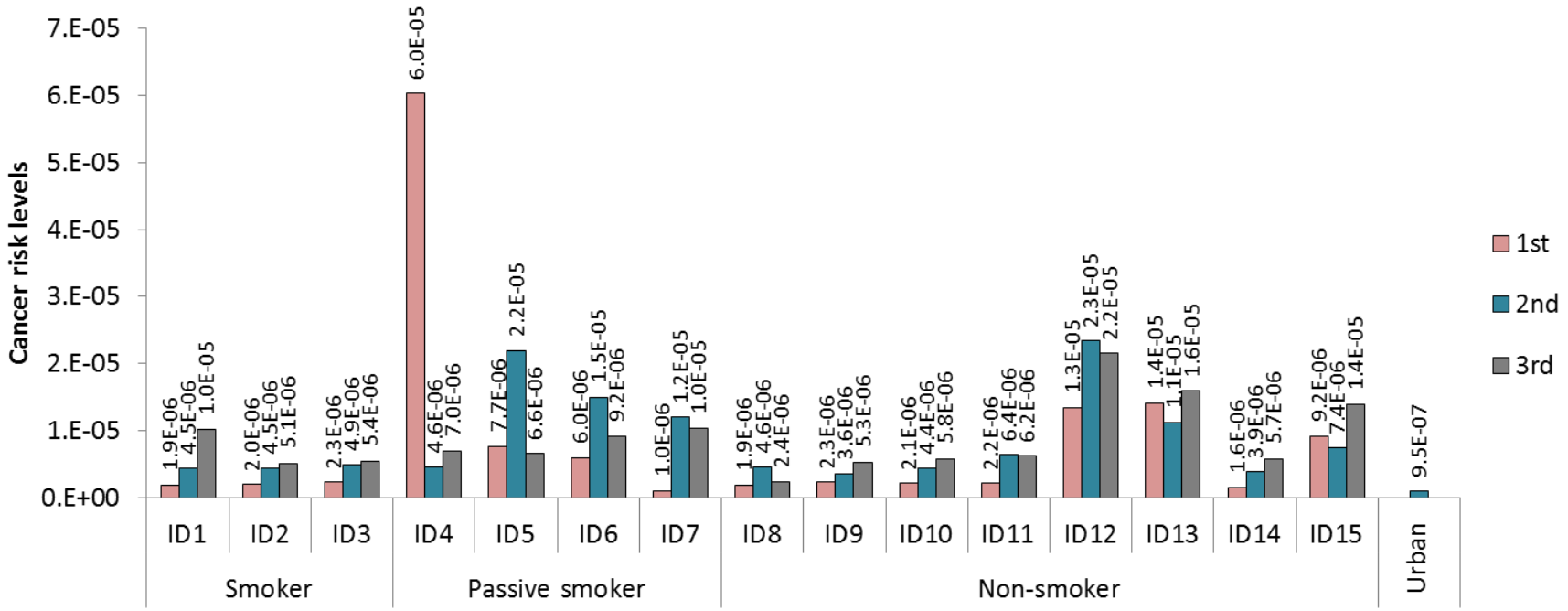


Fig. 3.5 Individual cancer risk assessment of rural subjects for the first to third days of the study period.

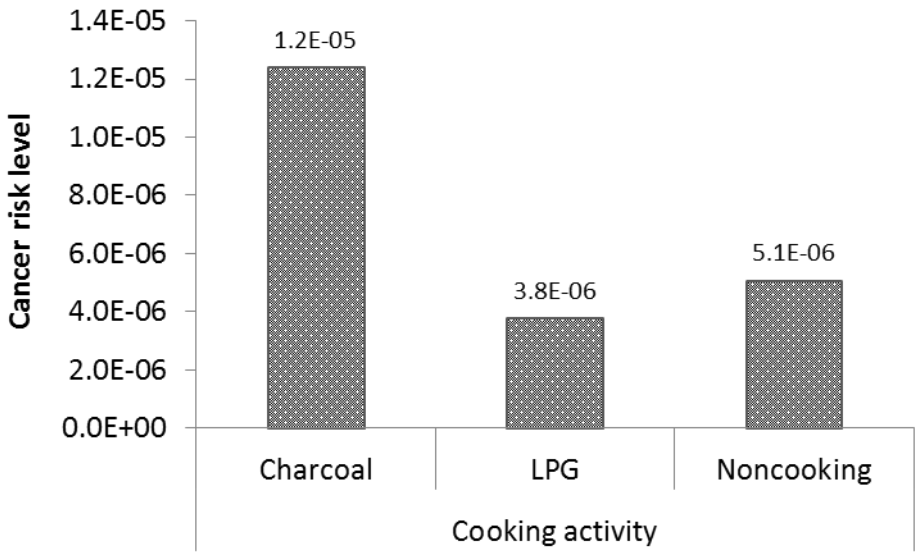


Fig. 3.6 Comparison of cancer risks of personal inhalation exposure for rural subjects who cooked with charcoal and LPG stoves and those who did not take part in cooking activities.

3.4 Conclusion

It is a new challenge to find important factors related to the highest incidence of lung cancer in Lampang, Thailand. This study reports the characterization of personal inhalation exposure to PM2.5, PAHs and NPAHs, and the cancer risk assessment of rural residents in Lampang for the first time. The levels of the monitored components for the subjects were higher than those from stationary samplings, suggesting the unreliability of estimating personal exposure from microenvironments in subjects' lives using only the results of stationary sampling. The atmospheric environment in the residential area contributed less to PAH concentrations because these were strongly affected by individual exposure from microenvironments such as indoor air. The smoking behavior of the residents was not reflected in their exposure to PAHs and NPAHs compared to other sources. Cooking activity was the most important factor concerning exposure to PAHs. The diagnostic ratios for PAHs and NPAHs, 1-NP/Pyr and BaP/BghiPe, were used to identify the combustion sources. Urban ambient air was dominated by vehicle exhaust, whereas exposure to residents was affected by sources related to their personal lifestyle in addition to the atmospheric environment during haze periods. Personal

inhalation cancer risks for all rural subjects during the study period exceeded the guideline value set by the USEPA, suggesting that the residents have a potentially increased cancer risk. In particular, the subjects who cooked using charcoal open fires showed the highest cancer risk. A reduction in exposure to air pollutants in the area could be achieved by encouraging rural residents to use clean fuel such as LPG or electricity for their daily cooking.

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Acknowledgments

This Ph.D. would not have been successful without the support and guidance that I received from many people.

I would like to express my special appreciation and thanks to my supervisor, Assoc. Prof. Dr. Akira Toriba, for giving me an opportunity to do Ph.D. in Hygienic Chemistry, Kanazawa University. He is an excellent supervisor. I appreciate all his contributions of time, suggestions, and ideas. It was a pleasure working with him. Thank you also for his kind supporting me these past three years of my Ph.D.

Besides my supervisor, thank you Prof. Dr. Kazuichi Hayakawa and Assoc. Prof. Dr. Ning Tang for help, good advices, and valuable comments which improved my research throughout my study period.

For this dissertation I am grateful to my committee members: Prof. Dr. Akira Odani, Prof. Dr. Masami Furuuchi, and Prof. Dr. Kazuko Kimura for their time, insightful questions, brilliant comments and suggestions.

A special thanks to Asst. Prof. Dr. Thaneeya Chetiyanukornkul for introducing me to Assoc. Prof. Dr. Akira Toriba, her kind support, suggestion, encouragement, and inspiration. I also thank Dr. Thanyarat Chuesaard, who is my lovely senior, for being a good role model, encouragement, suggestions, and collecting air samples.

I greatly appreciate all laboratory members of Hygienic Chemistry, for their kind help, research cooperation and providing all the needed information during my Ph.D. study. I especially thank my Thai lab mate, Yaowatat Boongla, for a good friendship, help, and encouragement.

I gratefully acknowledge the funding source that made my Ph.D. work possible. I was funded by a Grant-in-Aid for Scientific Research from the Ministry of Education, Culture, Sports, Science and Technology, Japan for three years during my study in Japan.
I'm grateful to students from Chiang Mai University and rural residents who participated in the air sampling.

Lastly, I would like to thank my parents for all their love, support, and encouragement. I also thank my friends for a lovely time we have had together in Japan, help, love, and advices.