Exposure to some carcinogenic compounds in air, with special reference to wood smoke

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ABSTRACT

The general population is exposed to air pollutants in both indoor and outdoor air from many different sources, including traffic, biomass burning, industries, cigarette smoking, and certain building materials. Air pollutants can cause a variety of health effects such as cancer and respiratory and cardiovascular diseases. The overall aim of this thesis is to increase the knowledge regarding the exposure to some carcinogenic compounds, especially those emitted by domestic wood burning, thereby contributing to risk assessment. The exposure has been assessed by personal sampling in the breathing zone as well as by stationary measurements.

Median personal exposure to formaldehyde was 23 $\mu g/m^3$, which is within the guideline value range of 12-60 $\mu g/m^3$ proposed in Sweden. Bedroom concentration, used as a proxy of personal exposure, accounted for 90% of the variability of personal exposure. Subjects living in single-family houses had significantly higher exposure to formaldehyde compared with subjects living in apartments. The within-individual (day-to-day) source of variability in personal exposure was low.

In a residential area where wood burning for domestic heating is common, significantly higher indoor levels of 1,3-butadiene, benzene, and several PAHs, such as benzo(a)pyrene (BaP), were found in homes using wood-burning appliances compared to homes without. High correlations were found between personal and indoor levels of 1,3-butadiene, benzene, formaldehyde, and acetaldehyde ($r_s > 0.8$). The 1,3-butadiene levels measured personally, indoors, and outdoors were low with respect to risk for cancer. By contrast, benzene and BaP levels in the wood-burning homes (medians 2.6 $\mu g/m^3$ and 0.52 ng/m^3, respectively) were 2 and 5 times higher than their Swedish health-based guideline, which was also exceeded outdoors for BaP.

An experimental set-up of a system for studying human exposure in a chamber to the carcinogenic wood smoke constituents 1,3-butadiene, benzene, formaldehyde, acetaldehyde, and PAHs, as well as fine particles, was developed. Relatively constant particle mass and number concentrations were obtained over each exposure session. Exposure levels were, as expected, clearly higher (5–50 times) during the wood smoke session compared with the clean air session. Stationary measurements could be used to predict the personal exposure in the chamber.

In conclusion, this thesis demonstrates that personal exposure of formaldehyde is well reflected by the residential indoor concentration, which was higher in single-family homes than in apartments, and that a minor part of the general population is exposed to airborne concentrations of formaldehyde at levels associated with sensory irritation. Domestic wood burning can increase the indoor concentration of several PAHs, as well as 1,3-butadiene and benzene in homes with wood-burning appliances. BaP is the largest contributor to the increased cancer risk for people living in those homes. The developed experimental set-up for wood smoke exposure can be used to study effects of such exposure in humans by careful control of the burning process and characterization of the exposure.

Key words: formaldehyde, acetaldehyde, 1,3-butadiene, benzene, polycyclic aromatic hydrocarbons, particulate matter, domestic wood burning, exposure assessment, personal exposure, experimental study

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List of publications

This thesis is based on following publications, which will be referred to in the text by the Roman numerals I-IV:

I. Gustafson P., Barregård L., Lindahl R., Sällsten G. Formaldehyde levels in Sweden: personal exposure, indoor, and outdoor concentrations. *Journal of Exposure Analysis and Environmental Epidemiology* 2005; 15(3):252–260.

II. Gustafson P., Barregard L., Strandberg B., Sällsten G. The impact of domestic wood burning on personal, indoor and outdoor levels of 1,3-butadiene, benzene, formaldehyde and acetaldehyde. *Journal of Environmental Monitoring* 2007; 9(1):23–32.

III. Gustafson P., Östman C., Sällsten G. Indoor levels of polycyclic aromatic hydrocarbons in homes with and without wood burning for heating. *Environmental Science & Technology* 2008; 42(14):5074–5080.

IV. Sällsten G., Gustafson P., Johansson L., Johannesson S., Molnár P., Strandberg B., Tullin C., Barregard L. Experimental wood smoke exposure in humans. *Inhalation Toxicology* 2006; 18(11):855–864.

List of abbreviations

Ant	Anthracene
ATD	Automatic thermic desorption
BaA	Benz(a)anthracene
BaP	Benzo(a)pyrene
BaPeqs	Benzo(a)pyrene equivalents
BbF	Benzo(b)fluoranthene
BeP	Benzo(e)pyrene
BghiF	Benzo(ghi)fluoranthene
BghiP	Benzo(ghi)perylene
BkF	Benzo(k)fluoranthene
Br	Bromide
BS	Black smoke
Ca	Calcium
CcdP	Cyclopenta(cd)pyrene
Chr/Tri	Chrysene/triphenylene
Cl	Chlorine
CO	Carbon monoxide
CO_2	Carbon dioxide
Cor	Coronene
DNPH	2,4-Dinitrophenylhydrazine
EDXRF	Energy dispersive X-ray fluorescence
ELPI	Electric low-pressure impactor
EPA	Environmental Protection Agency
EPE	Estimated personal exposure
Flu	Fluoranthene
GC-MS	Gas chromatograph-mass spectrometer
GC-FID	Gas chromatograph-flame ionization detection
HPLC	High performance liquid chromatography
IARC	International Agency for Research on Cancer
IcdP	Indeno(1,2,3-cd)pyrene
IRIS	Integrated Risk Information System
K	Potassium
MPE	Measured personal exposure
NDIR	Non-dispersive infrared
NO	Nitrogen monoxide
NO_2	Nitrogen dioxide
NOAEL	No observable adverse effect level
Pb	Lead
PE	Perkin Elmer
Phe	Phenanthrene
$PM_{2.5}$	Particulate matter with an aerodynamic diameter below 2.5 µm
PM_1	Particulate matter with an aerodynamic diameter below 1 µm
ppm	parts per million
PUF	Polyurethane foam
Pyr	Pyrene
Rb	Rubidium
SPE	Solid phase extraction
TEF	Toxic equivalent factor
TEOM	Tapered element oscillating microbalance
UFP	Ultra fine particles
WHO	World Health Organization
Zn	Zink

Contents

1 Introduction

A person's exposure to an environmental pollutant is generally defined as any contact between a substance in an environmental medium (e.g., air, water, soil, food) and a surface of the human body (e.g., skin and respiratory tract) (Nieuwenhuijsen, 2003). Exposure is a key element in a chain of events that leads from emission of pollutants into the environment to a concentration in one or more environmental media, to actual human exposure, to internal dose, and in the end, to health effect as illustrated in Figure 1.1 (Sexton et al., 1992). Total exposure is made up of contributions from contaminants in all different media by entry through any of the three major exposure routes: inhalation, ingestion, and dermal contact. This thesis is focused on human exposure to air pollution by inhalation.

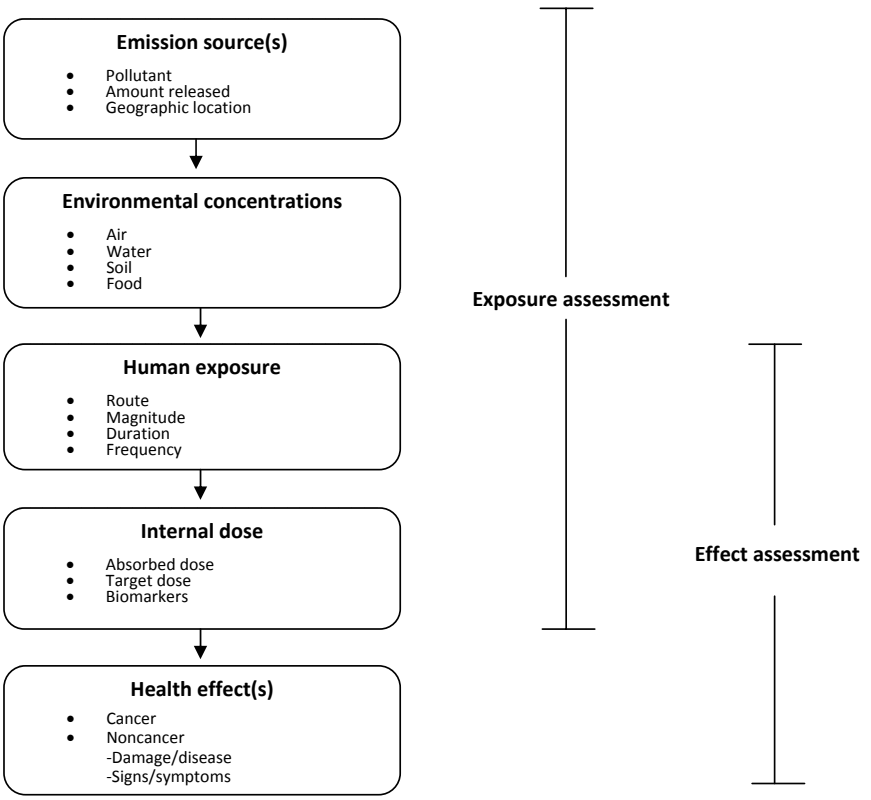


Figure 1.1 *The series of events that serves as the conceptual basis for understanding and evaluating environmental health (modified from Sexton et al. 1992).*

Air pollutants may either be emitted directly into the air (primary air pollutants) or be formed in the atmosphere by chemical reactions (secondary air pollutants). Various air pollutants are released from a number of natural and anthropogenic sources in the forms of gases or particles. Most air pollutants are released from anthropogenic sources, such as combustion of biomass and fossil fuel for generation of energy and transportation, including mobile sources (e.g., cars, trucks, and buses) and stationary sources (e.g., industries, power plants, and domestic heating appliances). Natural emissions include sources such as volcanoes and fires. Most of these sources are found outdoors, but they may also be important for indoor air, due

to infiltration of outdoor air pollutants. Other important indoor sources include heating and cooking appliances, smoking, and emissions from various materials and products.

The concentration in air of a specific pollutant is not only dependent on the emitted amount but also on meteorology, such as air movements, temperature, relative humidity, air pressure, and precipitation, together with atmospheric chemistry processes such as transformation and degradation by oxidation, hydrolysis, and photolysis (Finlayson-Pitts and Pitts, 2000). Indoors, chemistry also affects the fate of an air pollutant and its transportation, since reactions can occur in building products, on surfaces, and in air (Uhde and Salthammer, 2007). In addition, ventilation rate is a crucial factor for the concentration of air pollution indoors (Sax et al., 2004; Tucker, 2001). How these factors affect the air pollutant is dependent on its chemical and physical composition and reaction properties. An air pollutant may also be transported between different environmental media (e.g., air, water, and soil), which includes processes such as adsorption and volatilization.

Consequently, the emission of various pollutants from different sources can result in air pollution concentration showing a substantial spatial and temporal variation. People are therefore exposed to different concentrations of air pollutants as they move from place to place throughout the day. Human exposure is largely dependent upon the concentration in an environment and the time spent there. Thus, high air pollution concentrations in an environment do not necessarily result in high exposure. After uptake of the substance into the body the exposure is referred to as a dose (Nieuwenhuijsen, 2003). Measurements of internal dose are crucial for relating exposure to dose and dose to effects, and can sometimes be accomplished by analyzing biological samples for the particular substance and/or its metabolite(s).

1.1 Exposure assessment

The aim of human exposure assessment is to identify and quantify exposure that may cause health effects and includes identification of sources emitting harmful substances; determination of concentrations; identification of routes of exposure; determination of intensity; duration and frequency of exposure; and dose (Figure 1.1). In addition, estimation of the number of persons exposed and identification of high-risk groups (highly exposed or more susceptible to effects) are an important part of exposure assessment. Exposure assessment data are mainly used in epidemiological studies, risk assessment, and risk management, and for describing actual exposure levels present and trends in exposures (Sexton et al., 1992). Thus, it is necessary to obtain exposure data, as shown in ***Paper I-III***, to evaluate present exposure levels and to detect populations with exposure to high concentrations of air pollutants. For example, persons living in homes heated with wood, or living next door to one of these homes, have a relatively unknown exposure to air pollutants emitted by wood burning. In ***Papers II–III*** these individuals and their home environment are investigated to increase the knowledge of their exposure.

Exposure assessment can be carried out directly or indirectly by different methods (Figure 1.2). The indirect method of measuring stationary ambient air pollution levels for estimating exposure has been used in most air pollution epidemiological studies so far (Nieuwenhuijsen, 2003). However, these data may not reflect the real exposure of a population, since they are often measured at an urban location on a street or at roof level high above ground and represent only one of the many environments where people may spend their time. In fact, many people spend the majority (about 90%) of their time indoors (WHO, 2000). In addition,

studies have shown that indoor exposure to some pollutants can exceed outdoor exposure levels (WHO, 2000). If concentrations were obtained from several different microenvironments (e.g., homes, workplace, shops, cars, and buses), a person's exposure could be calculated or modeled by combining information on the time spent in a particular environment with the concentration in that environment. This indirect method may involve questionnaires and diaries including information on personal and home characteristics, time-activity pattern, and different exposure factors.

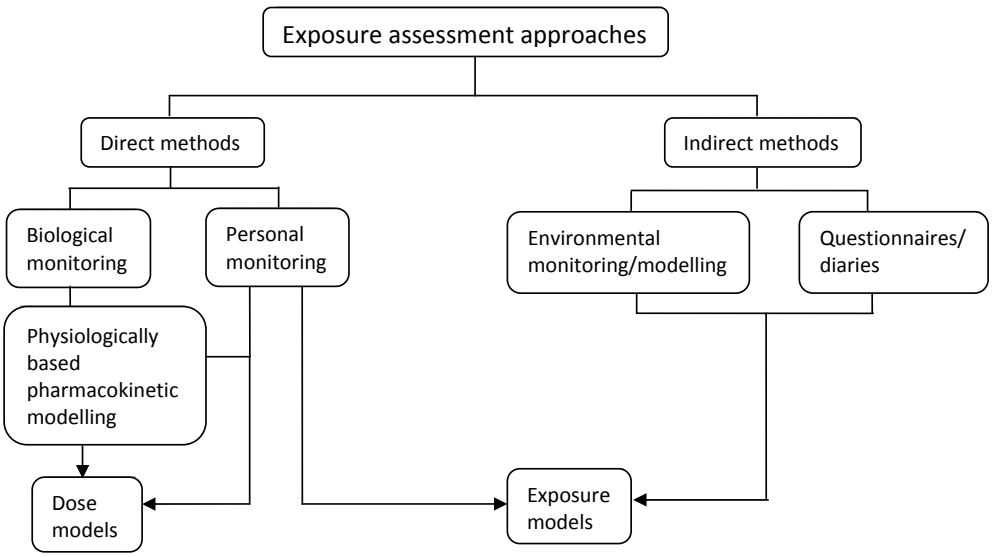


Figure 1.2. *Different approaches to human exposure assessment (adopted from Nieuwenhuijsen, 2003).*

However, a personal sampler carried by an individual close to the breathing zone provides much more realistic information about the exposure by including contributions from all microenvironments, in addition to pollutant-emitting activities performed by the individual (Nieuwenhuijsen, 2003). In ***Paper I*** exposure to formaldehyde measured by personal samplers is compared with the indirect method based on measurements in microenvironments and diary data. Another direct method of assessing exposure is biological monitoring, which measures biological markers in human media (e.g., blood and urine), reflecting the exposure that the person actually inhales (dose).

As a consequence of the temporal and spatial variation in exposure experienced by a person, a range of exposure levels is obtained. The distribution of the obtained exposure levels is generally lognormal and skewed to the right, which is important knowledge for statistical purposes (Rappaport and Kupper, 2008). Exposure generally varies from day to day for any given subject and from subject to subject, often referred to as the within- and between-subject exposure variability, respectively (Rappaport and Kupper, 2008). These components of variance are estimated in ***Paper I***, where repeated measurements of personal exposure to formaldehyde were performed.

Samplers can obtain concentrations continuously or time-integrated over a specific period by active or passive sampling. The choice of sampling method should be based on the objective

of the study. In general, direct-reading instruments would be the best option to obtain information about short-term fluctuations in exposure and reveal the frequency of occasional peaks; whereas, time-integrated samplers are more suitable for assessing the average exposure. The choice between conducting short-term or long-term measurements would depend on which is more important for the health effect: the ability of a specific air pollutant to cause an immediate or a delayed effect (Ayres, 1998). However, cost and convenience for an individual to carry the sampler are important to consider, as well. For example, continuous samplers are generally more expensive than time-integrated samplers, and active samplers are more noisy and heavy to carry, due to the requirements of a pump and battery supply, compared to passive samplers. Passive samplers are generally easy to handle and lightweight, because the measuring technique is based on diffusion.

The choices between personal exposure measurements and fixed site measurements, and between different types of samplers, are some of the factors determining the number of people included in a study. In all cases it is important to select study subjects in a manner that is statistically representative of a larger population (Ott, 1985), either by randomization or stratification.

1.2 Risk assessment and risk management regarding air pollutants

Risk assessment is a process used to estimate the likelihood and magnitude of health effects caused by a pollutant in humans. Risk assessments contain some or all of the following steps: hazard identification, exposure or dose-response assessment, exposure assessment, and risk characterization (National Research Council, 1983). The hazard identification includes a qualitative determination of whether exposure to a pollutant can cause adverse health effects in humans based on human and/or animal studies. Development of an exposure-response relationship, together with evaluation of the mechanism of action and species differences is the next step for making a quantitative evaluation of the health effects. The most relevant estimate of an exposure-response relationship is obtained from epidemiological studies, which are performed under "real life" conditions. In such studies, to detect a risk and determine the dose-response relationship with a high degree of certainty requires an accurate exposure assessment. However, a large number of individuals is generally needed to discover an association between exposure and health outcomes, which may limit the ability to do accurate exposure assessment. The uncertainty in the exposure-response relationship can be estimated by determining the components of variability and then calculating the number of measurements needed per person to achieve an acceptable level of uncertainty. This problem is illustrated in ***Paper I***, where repeated measurements of personal exposure to formaldehyde were taken.

However, attributing an observed health effect to a single air pollutant or a specific exposure can be complex, as air pollutants often occur in mixtures and may interact, causing additive or synergistic health effects. In ***Paper IV***, the set-up of an exposure chamber for studying the impact of wood smoke exposure on different markers of inflammation and coagulation in a controlled human exposure study is described together with a detailed characterization of the exposure. Exposing volunteers in a chamber under controlled conditions with an exactly known exposure allows the effect of single pollutants or a specific mixture to be studied. This design is only suitable for studying health effects caused by short-term exposure and may include only a relatively small number of persons. The exposure is not expected to have either lasting or potentially hazardous consequences. Despite the problem of extrapolating findings from chamber studies to real-life, where individuals with different ages and in different state

of health are exposed to a mixture of air pollutants from different sources, often during longer periods, chamber studies are useful for identifying health effects caused by a specific exposure, in addition to giving insights into the mechanism of these effects (Ayres, 1998).

Exposure assessment determining the exposure or dose in a population constitutes the third step of a risk assessment. Finally, the risk characterization step combines exposure estimates with exposure-response relationships to generate quantitative estimates of risks which provide information such as how many individuals may be affected. Because of different susceptibilities and exposures among a population, the risk will vary within a population. Both low and high risk can be important to consider, since a low risk could have a significant impact on the public health if a large number of people are exposed, whereas relatively uncommon exposure connected to a high risk may have a minor impact on the population level. Variability and uncertainty of a risk assessment are important to consider for subsequent use of the results in risk management.

The main purpose of air quality management is to protect the public from adverse effects of air pollution (WHO, 2006) by minimizing the risk. Risk management involves three basic types of decisions: determination of "unacceptable" risks, selection of the most cost-effective way to prevent or reduce unacceptable risks, and evaluation of the success of exposure and risk reduction-efforts (Sexton et al., 1992). Many activities are included in risk management, such as risk assessment, establishment of guideline values, and air quality and emission standards, exposure- and risk-control measures, and risk communication.

Potential or known carcinogenic air pollutants constitute an important group of air pollutants. Cancer risk estimate is generally based on data from epidemiological studies of occupationally exposed workers or studies of animals exposed to high doses. However, the generally lower exposure encountered among the general population requires extrapolation from high doses to low doses, and/or to conversion from animal to human conditions. In most cases, a linear dose-response relationship is assumed, and a unit risk factor can be calculated (Larsen and Larsen, 1998; WHO, 2000). A unit risk is the probability of developing cancer from, for example, a continuous lifetime inhalation of 1 $\mu g/m^3$ of the airborne chemical. In Sweden, a health-based guideline value exists for many carcinogens, and this value is defined as the exposure experienced during a lifetime that corresponds to an "acceptable" risk of 1 extra cancer case per 100,000 inhabitants (1×10^{-5}) (Victorin, 1998). Because there is a significant uncertainty in the evaluation and interpretation of the underlying dose-response studies, different unit risk estimates have been reported for a specific carcinogen, as shown by Loh et al. (2007) for unit risks reported by the United States Environmental Protection Agency's (U.S. EPA) Integrated Risk Information System (IRIS) and the California Office of Environmental Health and Hazard Assessment. Table 1.1 presents the exposure levels corresponding to a lifetime risk of 1×10^{-5} reported by Sweden, World Health Organization (WHO), and U.S. EPA, together with the International Agency for Research on Cancer (IARC) cancer classification for formaldehyde, acetaldehyde, 1,3-butadiene, benzene, and benzo(a)pyrene (BaP).

Table 1.1. *The IARC classification, together with the concentrations of the compounds (μg/m^3) producing an excess lifetime cancer risk of 1/100,000, based on the estimated unit risk from Sweden, WHO and U.S. EPA.*

		Concentrations (μg/m^3)		
Compound	**IARC classification[a]**	**Sweden**	**WHO**	**U.S. EPA[b]**
Formaldehyde	1	12-60[c]	100[c]	0.8
Acetaldehyde	2B	NA	NA	5
1,3-Butadiene	1	2.5	NA	0.3
Benzene	1	1.3	1.7	1.3-4.5
Benzo(a)pyrene	1	0.0001	NA	NA

[a]Group 1 = human carcinogen, group 2B = possible human carcinogen
[b]Integrated Risk Information System (IRIS)
[c]Based on irritation effects
Abbreviations: IARC, International Agency for Research on Cancer; WHO, World Health Organization; U.S. EPA, the U.S. Environmental Protection Agency; NA, not available

1.3 Generation of energy as an emission source of air pollutants

The use of fossil fuel as an energy source has been the key factor in the rapid technological, social, and cultural changes seen over the past 250 years. Its use has grown exponentially since the industrial revolution, and today nearly 80% of the human energy use is in the form of oil, gas, and coal (Wilkinson et al., 2007). Emissions from combustion of fossil fuel are responsible for a large fraction of the air pollution problems seen in densely populated regions of the world and are widely considered to be the dominant cause of climate change. Traditional use of biomass fuel (in the form of wood and agricultural residues) for heating and cooking, on the other hand, has occurred during many thousands of years, and wood smoke is for many people considered as natural and harmless. Biomass burning accounts for almost 10% of the world's energy use (Wilkinson et al., 2007). It has been estimated that about half of the world's population relies on biomass (wood, crop residues, and dung) and coal for cooking or heating, especially in the developing countries (Rehfuess et al., 2006), where it poses a large threat to human health (Naeher et al., 2007). However, in many countries with cold winters and good availability of wood, such as Sweden, burning of biomass for heating is also common (Glasius et al., 2006; Hedberg et al., 2002; Hellén et al., 2008). Burning of biomass, mainly wood, is considered to contribute about 24% of the energy consumption for heating of Swedish one-and two-dwelling buildings (Statistics Sweden, 2007). During recent years, the use of pellets has started to increase. About 9% of the one- and two-dwelling buildings are heated exclusively with biomass, although a combination of firewood and electricity is more common (24%) (Statistics Sweden, 2007). Electricity and air heat pumps are the dominant heating systems (40%), while only about 4% are nowadays heated by oil exclusively (Statistics Sweden, 2007). However, the use of different heating systems in different parts of Sweden is not evenly distributed and depends on the availability of firewood and the extension of the local district heating network. Over the past decades, increasing concern regarding the issue of global warming, along with the rising cost of fossil fuel and its limited reserves, have led to increased focus on the use of wood and other biomass fuels as a renewable energy source. The European Union has, within the "Clean Air for Europe" (CAFE) program, estimated that the contribution of emissions from domestic wood burning to primary $PM_{2.5}$ emissions will increase and become the largest source of emissions in 2020 in

the European Union (EU-15) countries (Figure 1.3) (Amann et al., 2005). In contrast, a decline in the share of mobile (vehicular) sources has been predicted.

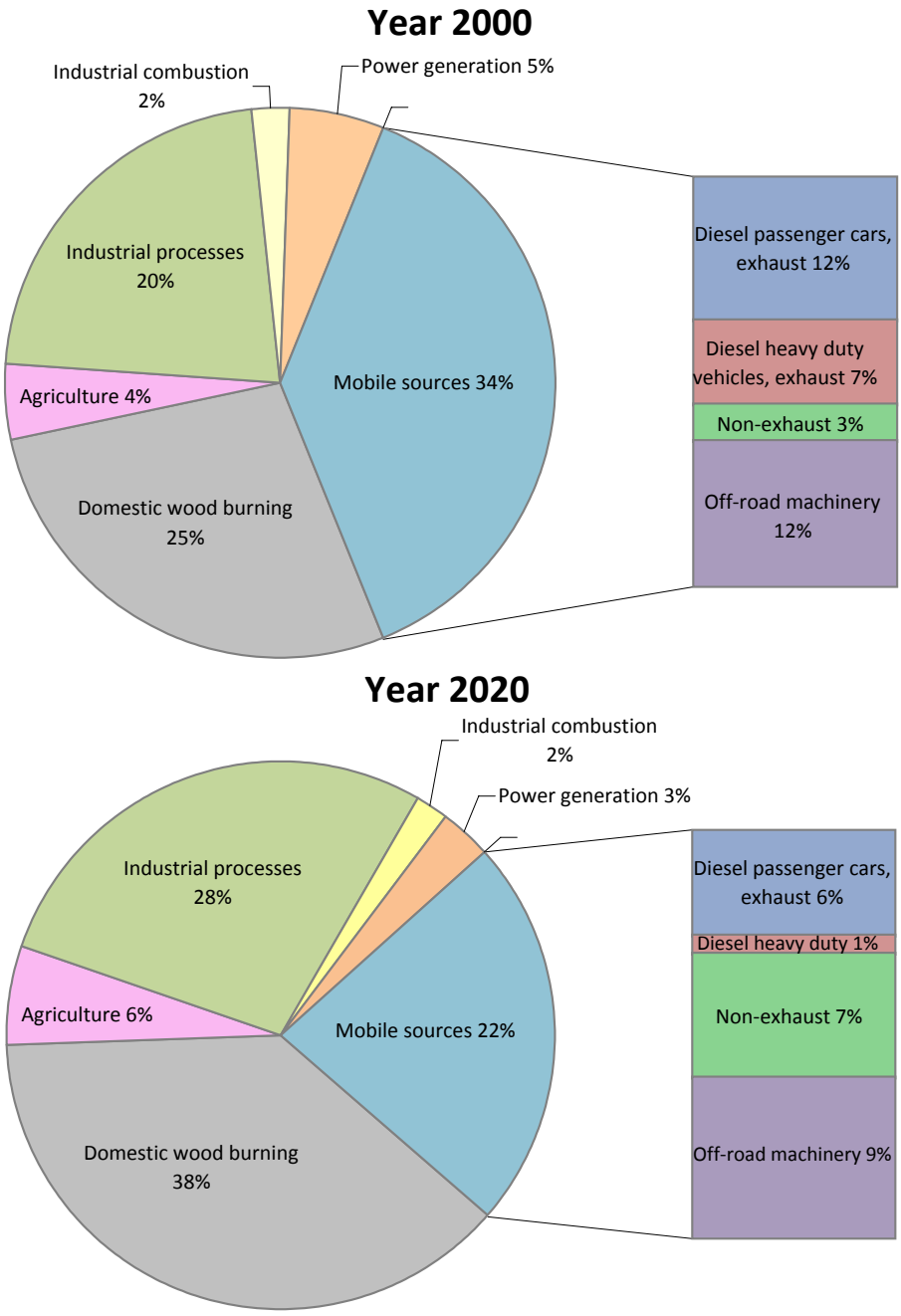


Figure 1.3. *Contribution to primary $PM_{2.5}$ emissions in the EU-15 countries, year 2000 and year 2020 (adopted from Amann, 2005).*

1.4 Specific air pollutants/mixtures measured in this thesis: sources and health effects

This thesis is mainly focused on the exposure to formaldehyde, acetaldehyde, 1,3-butadiene, benzene, and polycyclic aromatic hydrocarbons (PAHs) including BaP, which are known or potential carcinogenic air pollutants among the general population, according to the IARC (Table 1.1).

Another focus has been on exposure to wood smoke, which constitutes a mixture of air pollutants including the above-mentioned compounds, as well as fine particles and many other compounds. Among the organic compounds found in ambient and indoor air, these carcinogenic compounds have most often been implicated in cancer risk assessments performed among the general population. Three recently conducted risk assessments studies (Dodson et al., 2007; Loh et al., 2007; Sax et al., 2006) pointed out 1,3-butadiene, benzene, and formaldehyde among the top-ranking cancer risk contributors within nonsmoking Americans, with cancer risks on the order of 10^{-4}–10^{-5}. The European Commission has also given formaldehyde and benzene the highest priority and acetaldehyde the second highest based on their health risks in indoor air (Koistinen, 2008).

An environmental monitoring program concerning the general population's exposure to carcinogenic compounds, coordinated by the Swedish Environmental Protection Agency (Swedish EPA), was started in the year 2000 to provide data for evaluation of the environmental objective "Clean Air" and remedial actions. The program includes measurements of benzene, 1,3-butadiene, formaldehyde, and PAHs, as well as nitrogen dioxide and fine particles (www.naturvardsverket.se). Measurements are conducted once a year in one of five selected Swedish cities. The formaldehyde measurements conducted in ***Paper I*** were part of the first round of this project. A combination of the health relevance of these pollutants and the availability of sampling and analytical methods underlies the selection of the studied air pollutants in this thesis.

1.4.1 Formaldehyde and acetaldehyde

Formaldehyde and acetaldehyde are the two simplest members of the aldehyde family. They are formed naturally in the troposphere by photo-oxidation of hydrocarbons (Seinfeld and Pandis, 1998). Formaldehyde is a reactive gas with a short atmospheric lifetime in urban areas during daytime, approximately 50 minutes in absence of nitrogen dioxide, and even shorter if nitrogen dioxide is present (WHO, 2000). Formaldehyde is formed by incomplete combustion, and the main sources in populated regions are anthropogenic, for example, exhaust from vehicles without catalytic converters (Larsen and Larsen, 1998; WHO, 2000). Levels of formaldehyde in outdoor air are generally below 1 $\mu g/m^3$ in remote areas and below 20 $\mu g/m^3$ in urban settings (IARC, 2006a). However, indoor levels generally exceed outdoor levels by an order of magnitude or more, due to the predominance of indoor sources (Godish, 2001; WHO, 2000). Formaldehyde is a widely used industrial chemical, with its greatest use in the production of resins based on urea, phenol, and melamine. Formaldehyde-based resins are used as wood adhesives in the manufacture of pressed wood products such as particleboard, plywood, and medium density fiberboard (MDF), finish coatings (acid-cured), textile treatments (permanent-press finishing), and urea formaldehyde foam insulation (UFFI) (Godish, 2001; IARC, 2006a). As a consequence, building materials and interior, furnishing, and consumer products introduce formaldehyde to the indoor air (Kelly et al., 1999; WHO, 2006). Other formaldehyde-containing products that may contribute to indoor levels include paper products, deodorants, fabric dyes, air fresheners, cleaners, pesticides, and preservatives

(Hess-Kosa, 2002; Kelly et al., 1999). In addition, tobacco smoke, heating, and cooking may contribute to elevated indoor levels of formaldehyde (WHO, 2000). The general population is mainly exposed to formaldehyde by inhalation.

As a result of its water solubility and chemical reactivity, formaldehyde and other aldehydes such as acetaldehyde, can cause irritation of the eyes and mucous membranes of the upper respiratory tract on exposure (Godish, 2001; WHO, 2000). Animal experiments have shown that formaldehyde is among the most potent of the aldehydes in causing irritation (e.g., 1,000 times more potent than acetaldehyde) (Godish, 2001). The no-effect level (acute and chronic) is estimated to be at 30 $\mu g/m^3$ as 30-min average (Koistinen, 2008). A significant increase in symptoms of irritation occurs at levels above 100 $\mu g/m^3$ in healthy subjects (WHO, 2000). Higher formaldehyde levels are associated with general discomfort, lacrimation, sneezing, coughing, nausea, and dyspnea, and concentrations above 60 mg/m^3 may be lethal. In Sweden, the occupational exposure limit value for a whole workday (8 hours) is 600 $\mu g/m^3$ (Swedish Work Environment Authority, 2005). In addition, formaldehyde is a skin irritant and can cause allergic contact dermatitis and asthma, at least among occupationally exposed people (IARC, 2006a).

The International Agency for Research on Cancer upgraded formaldehyde from group 2A (a probable human carcinogen) to group 1 (a human carcinogen), based on the epidemiology for nasopharyngeal cancer, in particular (IARC, 2006a). The EU has therefore recently recommended that the exposure to formaldehyde should be as low as possible (Koistinen, 2008). The Swedish guideline value of 12-60 $\mu g/m^3$ is based on the irritative effects and not on the carcinogenic potency of formaldehyde (Victorin, 1998) since a damage to the respiratory tract tissue likely is needed for cancer to develop (WHO, 2000). An uncertainty factor of 10 was applied to 120–600 $\mu g/m^3$, where irritation to the eyes and upper airways may occur in sensitive individuals. In addition, for risk of cancer, this dose-range was used as a low-risk level (Victorin, 1998), which represents a lifetime cancer risk of 1×10^{-5} in the Swedish population. The World Health Organization has likewise based its air quality guideline value of 100 $\mu g/m^3$ (30-minute average) on irritation (WHO, 2000). Thus, this guideline is considered to protect against sensory irritation and therefore also against carcinogenic effects. In contrast, the U.S. EPA (2008d) presents a unit risk estimate of 1.3×10^{-5} per $\mu g/m^3$ which gives a low-risk value of 0.8 $\mu g/m^3$.

Although acetaldehyde is a relatively weak irritant, the exposure and health effects may be important to consider, since acetaldehyde is a known animal carcinogen and is used industrially in the production of chemicals (e.g., acetic acid) and in the manufacture and production of plastics, phenolic and urea resins, photographic chemicals, rubber accelerants, antioxidants, varnishes, dyes, explosives, disinfectants, drugs, perfumes, flavourings, and vinegar (Godish, 2001). Acetaldehyde is also present in vehicle exhaust, wood smoke, and cigarette smoke. However, the main exposure is intake of food products and especially alcoholic beverages since acetaldehyde is a metabolic product of alcohol (Bruinen de Bruin et al., 2005). Acetaldehyde is classified by the IARC (1999) as a possible human carcinogen (group 2B). The levels of acetaldehyde associated with cancer are much higher compared with formaldehyde, thus acetaldehyde may be considered much less potent than formaldehyde, both regarding irritation potential and carcinogenicity. The EU has recently recommended an exposure limit value of 200 $\mu g/m^3$ (Koistinen, 2008). In Sweden, a guideline value for acetaldehyde has not been recommended. However, the U.S. EPA (2008b) has estimated a cancer risk of 1×10^{-5} at a lifetime exposure of 5.0 $\mu g/m^3$.

1.4.2 1,3-butadiene

1,3-Butadiene is a colorless gas whose principal route of environmental exposure is by inhalation (WHO, 2000). 1,3-Butadiene is emitted to the ambient air primarily from vehicle exhaust emissions, but significant emissions also arise from both its manufacture and its use in industry (Dollard et al., 2001). Other important combustion sources include cigarette smoke and wood smoke (WHO, 2000). The most significant use of 1,3-butadiene is in the production of synthetic rubbers and polymers (Grosse et al., 2007). 1,3-Butadiene is highly reactive in the atmosphere and is rapidly transformed by reaction with hydroxyl radicals, ozone, and nitrate. In the daytime during the summer, the residence time of 1,3-butadiene in the atmosphere is estimated to be less than one hour, while in winter on cloudy days it may exceed one day (Larsen and Larsen, 1998). Average concentrations in ambient air have been reported to be below 1 $\mu g/m^3$ with a magnitude lower concentrations at rural sites compared to urban sites (Curren et al., 2006; Dollard et al., 2001). These studies indicate a declining trend of ambient concentrations in response to reduced rates of emissions from mobile sources and point sources.

The IARC recently reassessed the carcinogenicity of 1,3-butadiene from group 2A (probably carcinogenic to humans) to group 1, which means that there is now "sufficient evidence" of an increased risk for leukemia in humans. The epidemiologic evidence is based on two studies including workers in the butadiene rubber industry and in butadiene monomer production (Grosse et al., 2007). In Sweden, a lifetime exposure to 2.5 $\mu g/m^3$ has been estimated to cause one case of leukemia among 100,000 persons (Finnberg et al., 2004). The corresponding unit risk estimate calculated by the U.S. EPA (2008a) results in a much lower low-risk value of 0.3 $\mu g/m^3$, which is similar to the health-based guideline values (0.2–1.0 $\mu g/m^3$) discussed in Sweden, when an uncertainty factor is applied to 2.5 $\mu g/m^3$ (Finnberg 2004). Very high exposure levels of several thousands mg/m^3 may cause respiratory tract and eye irritation in humans (Larsen and Larsen, 1998).

1.4.3 Benzene

Benzene is a colorless liquid with a high vapor pressure, causing it to evaporate rapidly in room temperature (WHO, 2000). Inhalation is the major exposure pathway of benzene (Wallace, 1996). Benzene is a natural component of crude oil, and is therefore found in petrol. During the 1990s benzene in petrol was reduced in many countries and since the year 2000, the content of benzene in petrol must not exceed 1% in the European Union, a reduction from the previous upper limit of 5%. Together with exhaust emissions from motor vehicles, evaporation losses from motor vehicles and those that occur during handling, distribution, and storage of petrol are the main sources of benzene in ambient air (WHO, 2000). Other combustion sources, including cigarette smoke, wood smoke, and fossil fuels used for heating, also emit benzene.

Occupational exposure to benzene can cause bone marrow depression and leukemia (IARC, 1982; Larsen and Larsen, 1998; WHO, 2000). Based on epidemiological evidence, the IARC has classified benzene as a human carcinogen (group 1) (IARC, 1982). WHO (2000) used data from an epidemiological study among workers within the rubber film industry (the updated Pliofilm cohort) to calculate a unit risk corresponding to an excess lifetime risk of 1/100,000 at an exposure to 1.7 $\mu g/m^3$. The Swedish health-based guideline value of 1.3 $\mu g/m^3$ is based on the same data (Victorin, 1998). A similar exposure range was provided by the U.S. EPA within the IRIS program (1.3–4.5 $\mu g/m^3$) (U.S. EPA, 2008c). The EU has recommended that the concentration of benzene in indoor air should be as low as reasonably

achievable and that indoor concentration should not exceed outdoor concentrations (Koistinen, 2008). The cancer risk assessment at low exposure to benzene relies on extrapolation from high-level occupational exposure. This technique may, however, underestimate the risk at low-level exposure (Lin et al., 2007; Rappaport et al., 2002).

1.4.4 Polycyclic aromatic hydrocarbons (PAHs)

PAHs constitute a large class of compounds that contain two or more fused aromatic rings. The smaller PAHs with 2 to 4 rings are more volatile and are found in the gaseous phase in air in a higher degree than the 5- to 7-ring PAHs, which occur mainly or entirely as particles (Finlayson-Pitts and Pitts, 2000). PAHs are products of incomplete combustion or pyrolysis of organic material such as coal, oil, gas, and wood, and are found in emissions from industries, traffic, cigarette smoking, cooking, and residential heating with fossil fuels and biomass (Bostrom et al., 2002; WHO, 2000). About 500 PAHs and related compounds have been detected in air, but most measurements have been made on BaP (WHO, 2000). In the 1960s the annual average concentration of BaP was reported to be higher than 100 ng/m^3 in several European cities (WHO, 2000). In most developed countries, PAH concentrations have decreased substantially during the last 30 years owing to improved combustion technologies, increased use of catalytic converters in motor vehicles, and replacement of coal with oil and natural gas as an energy source (Larsen and Larsen, 1998). However, very high exposure can still occur in workplaces during the conversion of coal to coke and coal tar, and during the processing and use of products derived from coal tar (IARC, 2006b; Straif et al., 2005). In the atmosphere, PAHs undergo chemical and photochemical degradation by reactions with ozone, hydroxyl radicals, and nitrate radicals (Finlayson-Pitts and Pitts, 2000). The natural background level of BaP may be nearly zero (WHO, 2000). Ingestion of food is considered to be the foremost exposure route, due to formation during cooking or from atmospheric deposition (WHO, 2000).

Individual PAHs and specific PAH mixtures have been classified as carcinogenic by the IARC (IARC, 2006b; Straif et al., 2005). Benzo(a)pyrene, the most widely investigated PAH, has been classified as carcinogenic to humans (IARC, group 1) (IARC, 2006b; Straif et al., 2005). The lung cancer risk from inhalation exposure to a PAH mixture can be estimated by summarizing the individual PAH concentrations, taking into account the toxicity equivalency factors (TEFs) denoting the cancer potency relative to the cancer potency of BaP (Bostrom et al., 2002). WHO has estimated a unit risk of 8.7×10^{-5} per ng/m^3 BaP, based on epidemiological data from studies of coke-oven workers (WHO, 2000). The total carcinogenicity of the PAH mixture in this estimate is represented by BaP. This unit risk corresponds to an excess lifetime cancer risk of 1/100,000 at an exposure of 0.1 ng/m^3 (WHO, 2000), which is used as the health-based guideline value in Sweden (Bostrom et al., 2002). Other adverse health effects, such as immunotoxicity, reproductive toxicity, and the possible influence on development of atherosclerosis have been reported from animal studies after exposure to PAHs (WHO, 2000).

1.4.5 Wood smoke

The characteristic composition of wood smoke is derived from the structure of wood, which can vary between different tree species. However, all wood consists primarily of the two polymers, cellulose and lignin (Simoneit, 2002). In addition, wood also includes several elements, especially Ca, K, Zn, and Fe (McDonald et al., 2006). Due to the typically inefficient combustion of wood, many organic chemicals are produced. Emission from

residential wood burning includes carbon monoxide, particulate matter (inorganic ash material, condensable organic compounds, and carbon-containing particles) and a wide range of gaseous organic compounds (Hedberg et al., 2002; Johansson et al., 2004b; McDonald et al., 2000; Schauer et al., 2001). Formaldehyde, acetaldehyde, benzene, 1,3-butadiene and PAHs were pointed out as significant constituents in the wood smoke emissions in several of the cited studies.

Studies from Sweden, Canada, the United States, and Denmark have shown that residential wood burning for heating is a significant source of ambient PM in rural areas during wintertime (Glasius et al., 2006; Hedberg and Johansson, 2006; Jeong et al., 2008; Ward et al., 2006; Wu et al., 2007). Those studies showed that wood burning can account for 23–82% of the total outdoor $PM_{2.5}$ concentration during winter. Especially during periods with low air temperatures and very stable atmospheric conditions close to the ground, the concentrations can be high (Krecl et al., 2008). The effect of wood burning has been reported to be even more pronounced for organic compounds than for PM (Glasius et al., 2006; Hellén et al., 2008). Benzene and 1,3-butadiene are often considered traffic-generated pollutants, however, wood burning was shown to be the main local source in residential areas studied by Hellén et al. (2008; 2006). In a German study, wood combustion was found to be a significant source of many different organic compounds in $PM_{2.5}$ (Schnelle-Kreis et al., 2007). The contribution of wood combustion to measured levels of PAH was in that study estimated to be 80–95%. Wood burning for residential heating is the major source of PAH emissions in Sweden, with a recent estimate of 280 tonnes annually, according to the Swedish EPA (Todorovic et al., 2007). This amount is much higher than the emission from traffic (Bostrom et al., 2002). However, large variations (orders of magnitude) in the emission data for different wood-burning appliances used for residential heating in Sweden (Hedberg et al., 2002; Johansson et al., 2004b) make this estimation uncertain.

Except for the impact seen on ambient air, domestic wood burning may also affect the indoor air quality in developed countries (Alfheim and Ramdahl, 1984; Daisey et al., 1989; Mandin et al., 2008; Molnar et al., 2005; Traynor et al., 1987), despite the fact that relatively well-ventilated wood-burning appliances have been in use for many years. In Sweden boilers for water-based heating and hot water production are generally used for heating, whereas stoves and fireplaces are often used for pleasure or as a secondary heating source. But also infiltration of ambient air may elevate the indoor levels. In contrast, open fires and unvented or poorly ventilated appliances are still in use for cooking and heating often in combination with use of poor quality fuel such as dung and agricultural residues in many developing countries. The low combustion efficiency achieved under these conditions results in very high indoor levels of incomplete combustion products. Several hundreds of $\mu g/m^3$ of PM have been found, for example, in Mexico, Guatemala, Kenya, and India (Balakrishnan et al., 2002; Balakrishnan et al., 2004; Clark et al., 2007; Cynthia et al., 2008; Naeher et al., 2007), exceeding by far the WHO air quality guidelines (WHO, 2006) and levels found in Swedish households using wood-burning (Molnar et al., 2005).

Numerous epidemiological studies have shown an increased risk of cardiovascular and respiratory morbidity and mortality in association with increased exposure to particulate air pollution in general (WHO, 2006), while relatively few studies examining the health impacts of wood smoke have been conducted in the developed world. This is partly due to the difficulty of disentangling risks due to wood smoke from those associated with other air pollutants also present (Naeher et al., 2007). Those that have been done were reviewed by Naeher et al. (2007), who found that exposure to wood smoke from residential wood burning is associated with a variety of respiratory health effects, which are no different in kind and

show no difference in magnitude of effect from those associated with exposure to other combustion-derived particles. In a discussion of the relative toxicity of wood smoke compared with vehicle exhaust, Boman et al. (2003) reviewed nine epidemiologic studies including health outcomes such as respiratory symptoms, daily mortality rates, and lung function, and came to the same conclusion that PM from wood smoke seems to be at least as harmful as PM derived from other sources.

Recently, IARC classified indoor emissions from household combustion of biomass fuel (mainly wood) as probably carcinogenic to humans (group 2A), based on limited human evidence but with supporting animal and mechanistic evidence (Straif et al., 2006). The human evidence included four epidemiological studies reporting an increased risk of lung cancer in association with wood burning. However, information of any exposure-response relationship could not be examined, since information on duration and intensity of exposure was lacking in these studies (Straif et al., 2006). An association between lung cancer and exposure to ambient air pollution in general has been reported, but the evidence is still uncertain (Boffetta, 2006; Pope et al., 2002; Pope and Dockery, 2006)

In the recent Global Comparative Risk Assessment managed by WHO, indoor smoke from burning of solid fuels (biomass and to a lesser extent coal) was identified as one of the world's ten major causes of morbidity and mortality, accounting for 2.6% of the global burden of disease and 1.6 million premature deaths annually from acute lower respiratory infections, chronic obstructive pulmonary disease, and lung cancer (for coal smoke only), mainly occurring in the developing world (Ezzati et al., 2002; Smith et al., 2004).

2 Aims of the thesis

The main purpose of this thesis is to increase the knowledge regarding the exposure to some carcinogenic compounds, especially those emitted by wood burning, and thereby contributing to risk assessment.

The specific aims were to:

- Characterize personal exposure to formaldehyde and its variability and determinants in the general population, as well as its indoor and outdoor concentrations ***(Paper I)***.

- Investigate the impact of domestic wood burning on personal exposure and/or indoor levels for 1,3-butadiene, benzene, formaldehyde, and acetaldehyde ***(Paper II)*** and PAHs ***(Paper III)*** in a residential area where wood burning for space heating is common.

- Develop and execute the experimental set-up of a system for studying human exposure in a chamber to the carcinogenic wood smoke constituents 1,3-butadiene, benzene, formaldehyde, acetaldehyde, and PAHs, as well as to fine particles ***(Paper IV)***.

3 Materials and methods

3.1 Study population, study areas, and measurement periods

The subjects in ***Papers I–III*** all represent the Swedish general population, although the selection was based on different criteria. In ***Paper I***, one group was randomly recruited from the population register (aged 20–50 years), and the other group consisted of ten volunteers (aged 27–54 years) among the staff of our department in Gothenburg, as shown in Figure 3.1.

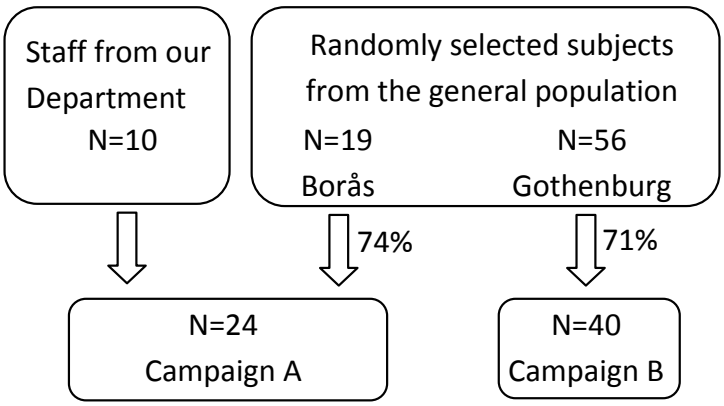


Figure 3.1. *The recruitment process in* ***Paper I*** *with the participation rate (%) among the randomly selected participants shown.*

Gothenburg is the second largest city in Sweden, with about 500,000 inhabitants, and located on the west coast, while Borås is a somewhat smaller city (100,000 inhabitants) located 65 km east of Gothenburg (Figure 3.2). The two sampling campaigns in ***Paper I*** were performed in October–November 1999 (campaign A), and 2000 (campaign B).

In ***Papers II and III*****,** the subjects were selected based on information obtained from the local chimney sweeping register in Hagfors as to the type of heating systems present in the inhabitants' homes. Eighteen households in the area, with daily use of wood-burning appliances, were contacted. In 14 of them one householder each agreed to participate in the study (78%). One home had a boiler located in a shelter outside the house, and therefore, this home was excluded from the wood-burning group when the indoor levels were analyzed. Twelve households from the same area, using electrical heating or heat pumps, were contacted, and ten individuals agreed to participate. To avoid contamination from other possible indoor sources besides burning wood logs, houses heated by combustion of other fuels, such as oil, were not included. Also, possible occupational exposure and smoking by the subjects or other household members were avoided during the sampling period.

The residential area is located in the small Swedish town of Hagfors (5,600 inhabitants in the population center) in the inland region of Sweden (Figure 3.2). The area (400 × 1,500 m)

Figure 3.2. *Locations of the cities where the subjects were living.*

consists of 225 single-family homes with about one-third using wood-burning appliances (boilers or fireplaces) continuously (11%), daily or weekly (10%) or less frequently (16%). The study was carried out during winter, from 10 February to 12 March 2003.

In ***Paper IV***, 13 healthy subjects, six men and seven women aged 20–56 years were recruited from our department. The study was approved by the Ethics Committee at the University of Gothenburg. The exposure to wood-smoke-impacted air took place in a chamber (Figure 3.4), specially designed for this purpose, at the SP Technical Research Institute of Sweden in 2005. The same chamber was used both for the sessions of exposure to wood smoke and for the filtered indoor air sessions. The chamber can hold at least ten subjects.

3.2 Sampling strategy/Study design

The features of the study designs in ***Papers I–III*** and ***Paper IV*****,** together with details of the sampling techniques, are summarized in Tables 3.1 and 3.2, respectively. In ***Paper I***, the personal formaldehyde sampler was worn within the breathing zone and placed close to the bed at night. Simultaneous indoor measurements were taken in the bedroom with the sampler hanging at least 1.5 m above the floor and at least 0.5 m away from lamps, walls, and windows. Repeated personal measurements were performed for a subgroup of the study subjects in both campaigns (campaign A: $n = 10$, campaign B: $n = 20$) in order to study the variability between and within individuals. In addition, repeated measurements were taken in nine bedrooms in campaign A. The interval between measurements was approximately one week in campaign A and two weeks in campaign B. Campaign B also included outdoor measurements on a balcony or a terrace at 20 participants' homes. In addition, stationary outdoor measurements were performed at two places located in the center of Gothenburg, the city in campaign B. The measurements were taken during 24-hour periods in campaign A and during 6-day periods in campaign B. A questionnaire about occupation, age, and type of home was distributed at the start of the sampling. During the sampling period the participants filled in a diary, including time spent in different microenvironments, and any formaldehyde-emitting activities were noted.

Table 3.1. *Features of the study design: type of sampler, sampling time, uptake rate or flow rate, limit of detection (LOD) or for the PAHs limit of quantification (LOQ), and number of samples (N) taken at the different locations in* ***Papers I–III****.*

	Pollutant	Sampler	Sampling time (days)	Uptake rate or flow rate (ml/min)	LOD or LOQ ($\mu g/m^3$ or ng/m^3)[a]	Personal (N)	Living room (N)	Outside home (N)	Ambient site (N)
Paper I									
Campaign A	Formaldehyde	GMD	1	25.2	2.8	24+10[b]	24+10[b]	–	–
Campaign B	Formaldehyde	Modified GMD	6	20.5	0.6	40+20[b]	40	20	10
Paper II									
	1,3-Butadiene	SKC	1	14.9	0.015	24	24	–	9
	1,3-Butadiene	PE	7	0.56	0.03	–	24	24[c]	–
	Benzene	SKC	1	16	0.03	24	24	–	9
	Benzene	PE	7	0.59	0.04	–	24	24	4
	Formaldehyde	UMEx	1	25	1.9	24	24	–	9
	Acetaldehyde	UMEx	1	21	2.3	24	24	–	9
Paper III									
	PAH		1	2,000	PUF: 0.15 Filter: 0.05	–	23	–	8

[a] ng/m^3 for PAH
[b] Repeated measurements
[c] The results of the 1,3-butadiene measurements outside the homes were not reliable and have not been presented
GMD = GMD Model 570 sampler (GMD System Inc., Hendersonville PA, USA)
Modified GMD = GMD Model 570 sampler with extra amount of reagent (GMD System Inc., Hendersonville PA, USA)
PE = Perkin Elmer (PE) sampler (Perkin Elmer, Wellesley MA, USA)
SKC = SKC Ultra Passive Sampler (SKC Inc., Eighty Four PA, USA)
UMEx 100 Passive Sampler (SKC Inc., Eighty Four PA, USA)

Table 3.2. *Features of the study design: type of sampler, uptake rate or flow rate, and number of personal and stationary samples (N) performed in the chamber in* ***Paper IV****.*

Pollutant	**Sampler**	**Pump**	**Uptake rate or flow rate (ml/min)**	**Wood smoke**		**Clean air**	
				Personal (N)	**Stationary (N)**	**Personal (N)**	**Stationary (N)**
Particle size distribution	ELPI		Continous				
$PM_{2.5}$	1400 TEOM		Continous				
$PM_{2.5}$	Cyclone Triplex SCC1.062	BGI 400S	4,000	3	1	1	2
PM_1	Cyclone GK2.05	BGI 400S	3,500	3	1	1	2
Formaldehyde	SEP-PAK	Gilian	200	3	2	1	2
Acetaldehyde	SEP-PAK	Gilian	200	3	2	1	2
Benzene	SKC	*	16	3	2	1	2
1,3-Butadiene	SKC	*	14.9	3	2	1	2
PAH	GFF and PUF	GilAir	2,000	-	2	-	-
Naphtalene, toulene and xylenes	PE	Gilian	200	-	2	-	2
CO	Unor 6N		Continous				
NO, NO_2, and NOx	Chemiluminescence Analyser		Continous				
CO_2	Metrosonic		Continous				
Temperature and relative humidity	Tinytag Ultra THU-1500		Continous				

ELPI (Dekati, Tampere, Finland)
1400 TEOM (Thermo Inc., Waltham MA, USA)
Triplex SCC1.062 (BGI Inc., Waltham MA, USA)
GK2.05 (BGI Inc., Waltham MA, USA)
SEP-PAK (Waters Corp., Milford MA, USA)
SKC = SKC Ultra Passive Sampler (SKC Inc., Eighty Four PA, USA)
GFF = glass fiber filter and PUF = polyurethane foam
PE = Perkin Elmer (PE) sampler (Perkin Elmer, Wellesley MA, USA)
Unor 6N (Maihak AG, Hamburg, Germany)
Chemiluminescence Analyser (MEMonitor Europe Chemiluminescence instrument, ML 9841B, UK)
Metrosonics (NDIR analyzer, aq 5001, Metrosonics, Inc., Rochester NY, USA)
Tinytag Ultra THU-1500 (Gemini Data Loggers, West Sussex, UK)
*Passive sampler

Personal exposure to, and indoor levels of, 1,3-butadiene, benzene, formaldehyde, acetaldehyde ***(Paper II)***, and indoors levels of PAHs ***(Paper III)*** were measured simultaneously during 24 hours (Figure 3.3). In addition, an extended sampling period of 7 days was used for 1,3-butadiene and benzene indoors and outside the participants' homes in ***Paper II***. Indoor measurements were performed in the living room.

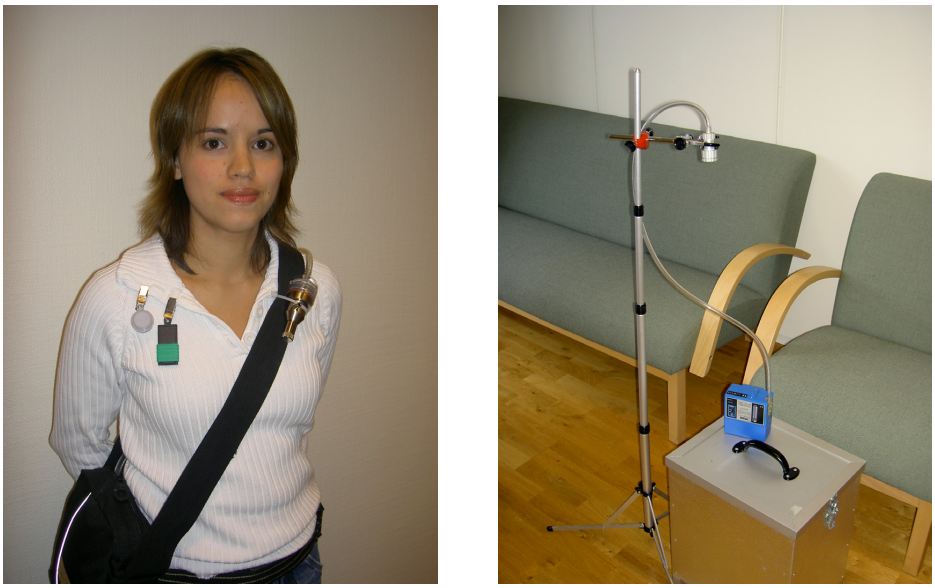


Figure 3.3 *The personal SKC and UMEx samplers (left) and the indoor PAH sampling equipment (right).*

The measurements in wood-burning homes and reference homes in ***Papers II and III*** were performed in parallel (2–3 homes/occasion), except on one and two occasions, respectively. On every occasion, the outdoor air in the area was measured on the roof (4 m above the ground) of a single-car garage attached to a home without a wood-burning appliance. In addition, for benzene and 1,3-butadiene the air outside each home was measured.

A diary of the same type as in ***Paper I*** was distributed to the subjects. The subjects or household member also recorded activities at home in order to collect information about contributions to the indoor air from possible sources of the pollutant of interest. The wood-burning participants also noted when they or someone else in the household had made a fire, how often wood was replenished, and how much, and what type of wood they burned. For the 7-day sampling, participants completed a questionnaire regarding activities in the home that could possibly have affected the concentrations. Information on the age and type of boiler, presence of an accumulator tank (i.e., a water tank used as a heat reservoir), and firewood storage was also collected from each household.

In ***Paper IV***, the subjects were exposed to filtered air for 4 hours on one occasion and then to wood smoke for 4 hours one week later. The filtered air and wood smoke sessions were identical except for the air quality. The sessions started with blood, urine, and breath sampling; then the subject entered the exposure chamber according to a schedule (Barregard et al., 2008; Barregard et al., 2006). The experiment was carried out in two sessions (sessions 1 and 2), with seven and six subjects, respectively. During the exposure sessions, subjective symptoms were registered using a self-administered questionnaire. There were two 25-minute periods of light exercise. After exposure, blood, urine, and breath sampling were performed at regular intervals.

Wood smoke was generated in a small cast iron wood stove (Jotul F400, Jotul AS, Fredriksstad, Norway) placed just outside the chamber, as illustrated in Figure 3.4. A standardized mixture (50/50) of hardwood/softwood (birch/spruce), dried for 1 year (moisture content 15–18%), was used in the wood stove. A partial flow of the generated wood smoke from the stove was mixed with indoor air (filtered through a high efficiency particulate air (HEPA) filter and a charcoal filter). The sidestream wood smoke was continually regulated by a slide valve in ten positions, and a stable concentration in the chamber was obtained by adjusting the ratio of the wood smoke to the filtered air. In the two wood smoke sessions, the smoke was diluted with filtered air 180 and 220 times, respectively. The mixed air inlet is placed in the ceiling of the chamber and the air is distributed through a supply air terminal device. The exhaust ventilation outlet is placed in a corner, near the floor. In the present study, 8–10 L/s per person, corresponding to 1.6–2.0 air exchanges/hour, was used for both the inlet and outlet air. The combustion conditions were followed online by measurements of temperature, CO_2, and CO (Binos 100 nondispersive infrared [NDIR] analyzer; Rosemount GmbH & Co., Germany) before and after mixing of wood smoke and filtered air. At the beginning of the wood smoke session, a larger amount of wood was introduced to give a glow bed. After approximately 1 hour, the concentration in the chamber was stable, and the subjects entered the chamber. The stove was replenished with about 2 kg of wood (four logs) every 40 minutes to maintain a constant concentration of wood smoke in the chamber. At replenishment, the slide valve was closed for a few minutes. The combustion conditions and the $PM_{2.5}$ concentration were controlled online (TEOM) to maintain the target concentration. Before the real wood smoke sessions started, the concentrations of fine particles and gaseous pollutants were measured in test sessions to be able to adjust and optimize the burning conditions in order to reach the target PM concentration. Moreover, different types of samplers and suitable flow rates were evaluated for the sampling of gaseous components. The chamber walls, floor, and ceiling are covered with Teflon-impregnated glass fiber fabric and were cleaned between sessions.

Stationary measurements were performed in the center of the chamber during the whole sessions (5.5 hours), while personal measurements (4 hours) were performed on three subjects during the wood smoke sessions and on one subject during the clean air sessions (Table 3.2).

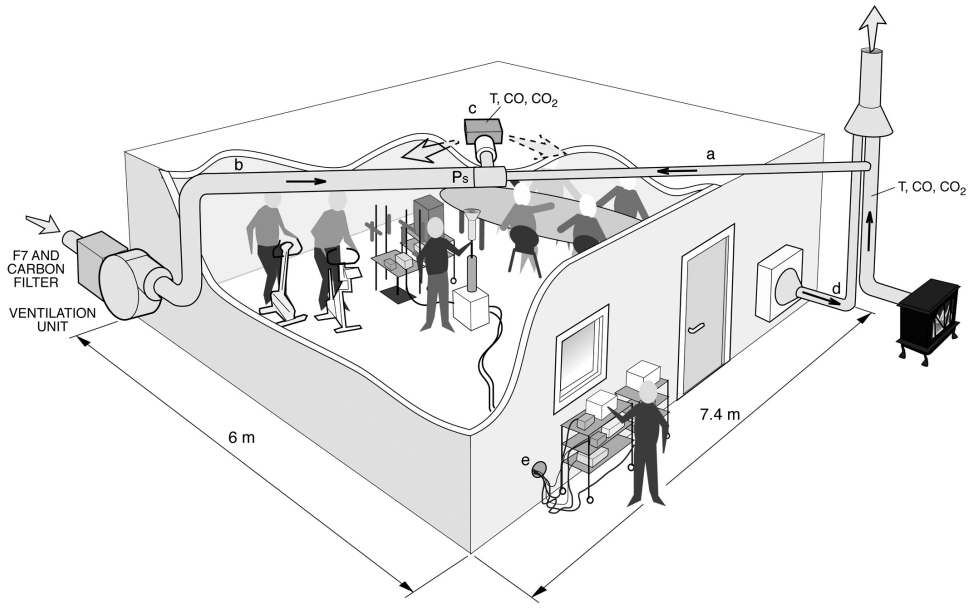


Figure 3.4. *The chamber (dimensions of 7.4 m × 6 m × 2.9 m) used in* ***Paper IV****. Partial flow of flue gas (a), flow of filtered indoor air (b), supply of air terminal device (c), ventilation outlet (d), and connection hole for instruments (e).*

3.3 Sampling equipment & analyses

3.3.1 Formaldehyde and acetaldehyde

In ***Paper I***, the diffusive GMD Model 570 sampler (GMD System Inc., Hendersonville PA, USA) was used to measure exposure to formaldehyde and acetaldehyde (Levin et al., 1988; Lindahl et al., 1996). It consists of a polypropylene housing (20 × 30 × 5 mm) containing two reagent (2,4-dinitrophenylhydrazine (DNPH))-coated filters, one working as the sampling filter and the other as a blank filter. In campaign A, we used the standard sampler (0.7 mg DNPH/filter), whereas, in campaign B the sampler was modified by adding an extra amount of reagent to the filter (3.5 mg DNPH/filter) for the extended sampling time (six days). A modification of the GMD sampler, the UMEx 100 Diffusive Sampler (SKC Inc., Eighty Four PA, USA) was used in ***Paper II***. The uptake rates used and the detection limits are presented in Table 3.1. Active sampling was performed in ***Paper IV*** using pumps (Gilian LFS-113 DC Low flow sampler) with a flow rate of 200 ml/min and Sep-Pak DNPH-impregnated silica cartridges (Waters Corp., Milford MA, USA). Active sampling equipment was always checked for flow rate before and after sampling.

The aldehyde-DNPH hydrazone was eluated from the filter with acetonitrile and analyzed by High Performance Liquid Chromatography (HPLC) with UV detection (Levin et al.,

1988). All samples were corrected for blanks. The analyses were performed at the National Institute of Working Life in Umeå ***(Paper I)*** and at the Department of Occupational and Environmental Medicine at Sahlgrenska University Hospital ***(Paper II and IV)***.

When comparing our acetaldehyde results from the measurements in ***Paper I*** with the acetaldehyde levels reported in France (Gonzalez-Flesca et al., 1999) and Finland (Jurvelin et al., 2001), we found unrealistically low levels of acetaldehyde in our study. Therefore, we initiated a comparison study in cooperation with the National Institute of Working Life in Sweden and Laboratoire d'Hygiène de la Ville de Paris in France in February 2002. The GMD sampler was compared with the Radiello Aldehyde Sampler (Fondazione Salvatore Maugeri, Padova, Italy), the diffusive sampler used in the French study, by measuring formaldehyde and acetaldehyde in 10 Swedish and 10 French homes with both type of samplers in each home for 3 and 6 days. A total of 40 GMD samples and 40 Radiello samples were analyzed at the National Institute of Working Life in Sweden and Laboratoire d'Hygiene de la Ville de Paris in France, respectively. The uptake rates used for formaldehyde and acetaldehyde in the comparison were 20.4 ml/min and 23.9 ml/min with the GMD sampler and 98.8 ml/min and 81.3 ml/min with the Radiello sampler, respectively. The results of the measurements are presented in section 4.1. The conclusion was that the GMD sampler could not measure acetaldehyde, and consequently, the acetaldehyde results are not presented in ***Paper I***. The GMD sampler has been validated for sampling of acetaldehyde (Lindahl et al., 1996) in a chamber, but not in any field study, which has been done for sampling of formaldehyde (Levin et al., 1988). The UMEx sampler has been validated for sampling of formaldehyde (8 hours up to 7 days). Field studies comparing the diffusive UMEx sampler with an active sampler indicated that it is useful and accurate for both 24-hour and 7-day sampling in indoor environments (Levin et al., 2004; Lindahl and Rehn, 2005).

3.3.2 Benzene, 1,3-butadiene, toluene, and xylenes (o-, m-, and p-xylene)

The SKC Ultra Passive Sampler (SKC Inc., Eighty Four PA, USA) was used for 24-hour sampling of benzene and 1,3-butadiene in ***Paper II*** and for the whole sessions (5.5-hour) in ***Paper IV***. It is a plastic, badge-type sampler (diameter: 30 mm; thickness: 15 mm) containing about 600 mg adsorbent, in this case Carbopack X 60–80 mesh (Supelco, Bellefonte PA, USA). For 1-week measurements of benzene and 1,3-butadiene in ***Paper II***, the Perkin Elmer (PE) sampler (Perkin Elmer, Wellesley MA, USA) was used. This sampler consists of a steel tube (90 mm × 6.3 mm outside diameter (o.d.) × 5.0 mm inside diameter (i.d.)) filled with about 300 mg Carbopack X 60–80 mesh. Uptake rates and detection limits are presented in Tables 3.1 and 3.2. Validation has been performed for measurements in ambient air with the SKC Ultra Passive Sampler (Strandberg et al., 2005; Strandberg et al., 2006) and the PE sampler (Levin et al., 2005). 1,3-Butadiene and benzene were analyzed with automatic thermic desorption (ATD) connected to a gas chromatograph-flame ionization detection (GC-FID) (Strandberg et al., 2005).

In ***Paper IV***, toluene and xylenes were measured with active sampling using PE tubes filled with Tenax TA (Scientific Instrument Services, Inc., Ringoes NJ, USA). The analyses were performed with ATD GC-FID (Egeghy et al., 2003; Strandberg et al., 2005, slightly modified). The analyses of benzene, 1,3-butadiene, toluene, and xylenes were performed at the Department of Occupational and Environmental Medicine at Sahlgrenska University Hospital ***(Papers II and IV)***.

3.3.3 Polycyclic aromatic hydrocarbons (PAHs)

In ***Papers III and IV***, an active sampling technique using a glass fibre filter and two polyurethane foam (PUF) plugs connected in series was used for collecting the particulate-associated and the gaseous PAHs, respectively. In ***Paper III***, a cylindrical sampler, previously described by Östman et al. (1993) was used. The sampler used in ***Paper IV*** was somewhat larger (Wingfors et al., 2001). Air was pumped through the samplers with a GilAir5 pump (Sensidyne, Clearwater FL, USA). For naphthalene, the most volatile of the measured PAHs, the sampling and the analyses were performed in the same way as described above for toluene and xylenes ***(Paper IV)***.

Twenty-seven PAH components were analyzed as described in ***Paper III*** at the Department of Analytical Chemistry at Stockholm University. The filter and PUFs from each sampler were extracted separately with ultrasonic-assisted solvent extraction in an ultrasonic bath. The extracts were then cleaned using solid phase extraction (SPE). The PAHs in the PAH-enriched fraction were finally determined by gas chromatograph-mass spectrometer (GC-MS) using electron ionization and selected ion monitoring. Triphenylene elutes, together with chrysene from the GC column, and the sum of the two compounds is reported. Three field blanks, treated in the same way as the real samples, were also analyzed. The real samples were corrected for presence of phenanthrene, 3-methylphenanthrene, 9-methylphenanthrene, fluoranthene, pyrene, and cyclopenta(cd)pyrene, which were found in low concentrations in the blank samples. For the other PAHs, the concentrations in the field blanks were below the LOQ. In ***Paper IV***, the filter and the PUFs were Soxhlet extracted and 14 PAHs were quantified using HPLC with fluorescence detection (Wingfors et al., 2001) at the Swedish Environmental Research Institute.

3.3.4 Particles and other gaseous pollutants

In ***Paper IV***, measurements of $PM_{2.5}$ and PM_1 mass concentrations were performed using cyclones (GK2.05 (KTL) and Triplex SCC1.062) and sampling pumps (BGI 400S), respectively (Molnar et al., 2005). In addition, particle $PM_{2.5}$ mass was measured online using a tapered element oscillating microbalance (TEOM) (1400 TEOM; Thermo Scientific Inc., Waltham MA, USA) instrument. Some of the filters were analyzed for trace elements using an energy dispersive X-ray fluorescence (EDXRF) spectrometer, and for black smoke (BS) using a reflectometer (Molnar et al., 2005). Number concentrations and size distributions of particles (0.007–6.7 μm) were measured by an electric low-pressure impactor (ELPI) from Dekati, Finland.

Concentrations of CO_2 and CO and temperature were measured online with infrared instruments (NDIR analyzer, aq 5001, Metrosonics, Inc., Rochester NY, USA; and Unor 6N; Maihak AG, Germany) and Tinytag Ultra TGU-1500 (Gemini Data Loggers, UK). NOx, NO, and NO_2 were measured using a chemiluminescence instrument (MEMonitor Europe Chemiluminescence Analyser, ML 9841B, West Sussex, UK).

3.4 Statistical methods

Non-parametric methods were usually used since the data were in general not normally distributed. Group comparisons of paired observations, for example, between locations, were assessed using the Student's *t* test ***(Paper I***) or Wilcoxon's signed-rank tests ***(Paper II–III)***. For unpaired observations such as apartments versus single-family houses in ***Paper I*** and wood-burning group versus reference group in ***Paper II–III***, the Wilcoxon rank-sum test was performed. The effect of type of residence on the personal exposure levels was also assessed using a mixed model (Proc Mixed of SAS) on log-transformed levels in order to take account of the within-individual and between-individual variance. Spearman's rank correlation coefficient (r_s) was used to express correlations between the different sampling locations (personal, indoors, and outdoors), between levels of pollutants and between repeated measurements. The tests performed were generally two-sided (P-values <0.05 considered as statistically significant), except for the comparisons between wood-burning group and reference group, which were performed one-sided, since the hypothesis was that the wood-burning group would show higher concentrations.

In ***Paper I***, the within-individual (σ^2_{Within}) and between-individual ($\sigma^2_{Between}$) components of variance were estimated using analysis of variance (Proc Nested of SAS) of log-transformed personal exposure levels. With knowledge of the variance ratio (λ), the number of measurements (n) per subject that would be required to reduce the bias (bias = 1-b) to 10% (b = 0.9) of a true underlying exposure-response relationship was calculated using estimates of the variance components according to equation 1 (Rappaport et al., 1995),

$$b = \frac{\beta_o}{\beta_t} = \frac{1}{1 + \frac{\lambda}{n}} \qquad \text{or} \qquad n = \left(\frac{b}{(1-b)}\right)\lambda \qquad \text{(Equation 1)}$$

β_t = true regression coefficient
β_o = observed regression coefficient
b = the attenuation of the slope of the exposure-response relationship in a simple linear regression model
$\lambda = \sigma^2_{Within}/\sigma^2_{Between}$
n = the number of measurements obtained from each subject

In ***Paper I***, personal exposure was estimated from bedroom and outdoor formaldehyde concentrations and the time spent in these microenvironments. The estimated personal exposure (EPE) was compared with the measured personal exposure. Three different approaches were assigned as models 1, 2, and 3. In model 1 the personal exposure was estimated from the bedroom concentrations (C_{home}). In model 2 a time-weighted EPE was

estimated by multiplying the time the individuals spent at home (t_{home}) and outdoors (t_{out}) by the individual C_{home} and the median outdoor residential concentration (C_{out} = 4 μg/m^3), respectively, and summarizing these contributions. An extended model (model 3) also includes the time subjects spent in other indoor environments than their homes (t_{other}) e.g. workplaces and shops. The concentration (C_{other}) was assumed to be similar to the median bedroom concentration in apartments, 25 μg/m^3.

$$EPE = C_{i,\mathrm{hom}e} \qquad \text{Model 1}$$

$$EPE = \frac{C_{i,\mathrm{hom}e} \times t_{i,\mathrm{hom}e} + C_{out} \times t_{i,out}}{t_{i,\mathrm{hom}e} + t_{i,out}} \qquad \text{Model 2}$$

$$EPE = \frac{C_{i,\mathrm{hom}e} \times t_{i,\mathrm{hom}e} + C_{out} \times t_{i,out} + C_{other} \times t_{i,other}}{t_{i,\mathrm{hom}e} + t_{i,out} + t_{i,other}} \qquad \text{Model 3}$$

i = i:th subject

The ability to predict personal exposure levels of the three different models was investigated with regression analysis and Bland Altman plot (Bland and Altman, 1986). The agreement between the measured and estimated personal exposure levels was also evaluated as the intraclass correlation coefficient (ICC), estimated as the between-pair variance divided by the sum of within- and between-pair variance components.

In ***Papers II–III***, the associations between the levels of the pollutants (log-transformed data) and the characteristics of wood-burning appliances (presence of water storage tank and type of appliance — boiler or fireplace) and wood-burning behavior (frequency of wood replenishments, amount and type of wood (deciduous trees or a mixture of deciduous and coniferous trees), and wood-burning time) were investigated using stepwise backward multiple regression analysis.

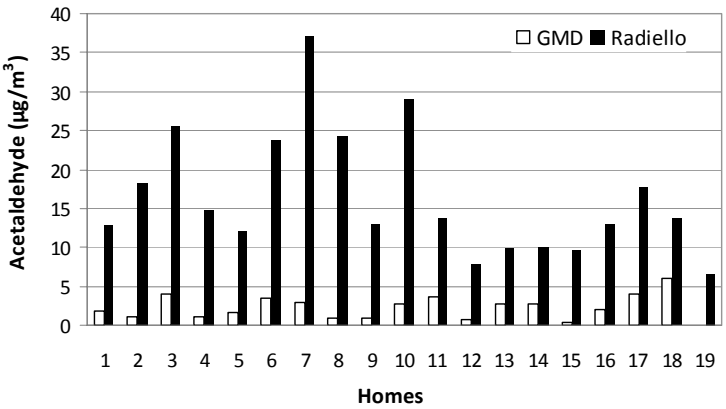
For values below the limit of detection (LOD) or the limit of quantification (LOQ), the calculated values of the LOD or the LOQ divided by the square root of 2 were used in the statistical calculations (Hornung and Reed, 1990). Statistical calculations were made using SAS for Windows, version 9.1 (SAS Statistical Software, SAS Institute, Cary NC, USA).

4 Results

4.1 Paper I

After comparing the acetaldehyde levels measured with the GMD sampler and the Radiello sampler during 6 days in Swedish and French homes, clearly lower levels were found with the GMD sampler (87% lower, based on the median ratio between the GMD and Radiello sampler) as shown in Figure 4.1a. The median indoor acetaldehyde level was 16 µg/m^3 (6.6–37 µg/m^3) measured with the Radiello sampler. Similar results were found for the 3-day sampling period. For formaldehyde, the agreement between the two samplers was much better, with the GMD sampler showing in median 22% higher levels than the Radiello sampler for the 6-day sampling period (Figure 1b).

(a)



(b)

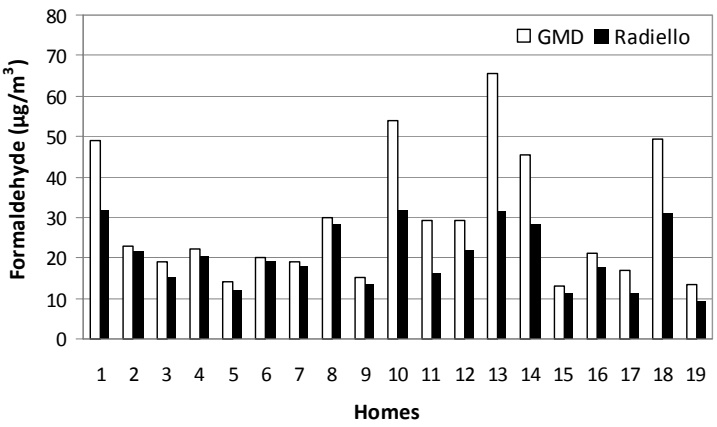


Figure 4.1. *(a) Acetaldehyde and (b) formaldehyde levels measured with the GMD sampler and the Radiello sampler in Swedish and French homes during a 6-day period.*

The median levels were 23 μg/m^3 (13–66 μg/m^3) and 19 μg/m^3 (9–32 μg/m^3) for the GMD and the Radiello sampler, respectively. The differences were largest for high formaldehyde levels (Figure 4.1b). For the 3-day sampling period the agreement was even better (median GMD-to-Radiello ratio of 1.1). Based on these results and further tests at the National Institute of Working Life, the results from the measurements of acetaldehyde were excluded in ***Paper I***.

Personal exposure to formaldehyde (median 23 μg/m^3, all persons from campaign A and B with one occupationally exposed person excluded) was similar to (campaign A) or slightly lower than (campaign B) the bedroom concentrations, but six times higher than the residential outdoor concentrations (Figure 4.2). Much higher exposure (566 μg/m^3) was found for the occupationally exposed person in campaign A.

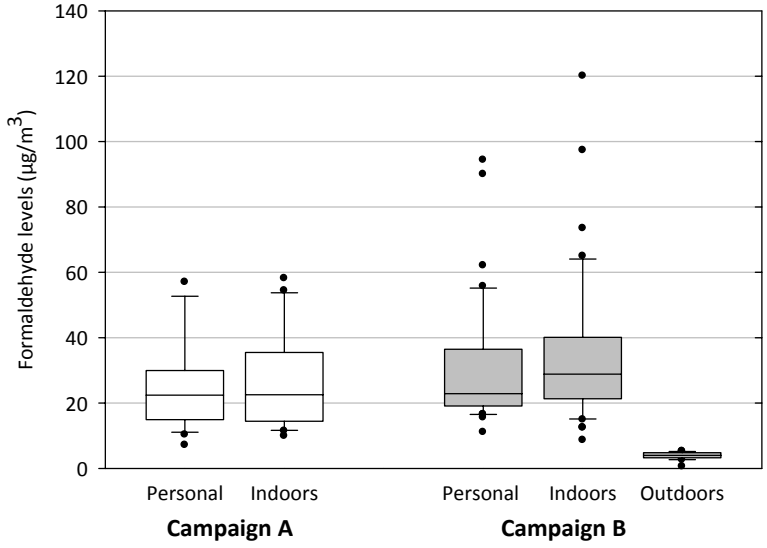


Figure 4.2. *Personal exposure, indoor, and outdoor levels (μg/m^3) of formaldehyde in campaigns A and B. The box plots show the 10th, 25th, 50th (i.e., the median), 75th, and 90th percentiles, and the outliers (the occupationally exposed person in campaign A is not shown).*

As a consequence of the low outdoor concentrations (4 μg/m^3) in relation to the indoor levels and the large fraction of time the subjects spent indoors (Figure 4.3), the personal exposure was well explained by the indoor concentrations as shown in model 1 ($R^2 = 0.90$) for campaign B, whereas the other two models, including also the outdoor environment, showed only minor improvements ($R^2 = 0.91$ and 0.92). All three models showed high ICC values (model 1: 0.86, model 2: 0.88, model 3: 0.91). All differences between sampling locations (personal, indoors, and outdoors) were significant in campaign B, whereas no significant difference was found between the personal and bedroom measurements in campaign A.

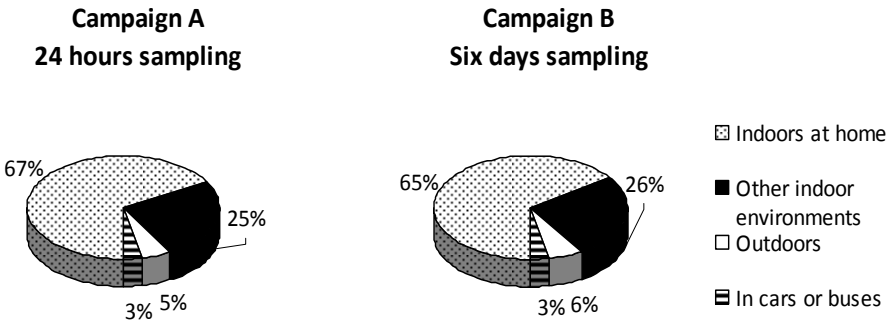


Figure 4.3. *Percentage of sampling time the participants spent in different environments for each of the two measurement campaigns.*

The repeated personal measurements showed similar median concentrations in the first and second rounds with high correlations between the two rounds in both campaigns. The between-individual source of variation dominated. Only 10% of the total variability for the six days' measurements was attributable to the within-individual source of variation in campaign B. It was somewhat higher, 31%, in campaign A, when the sampling period was shorter (24 h). Four measurements per individual are required to reduce the attenuation of the slope of an exposure-response relationship to 10% for a 24-hour sampling period, whereas one measurement is sufficient for 6 days' sampling.

The personal exposure was affected by type of residence with significantly higher levels for persons living in single-family homes compared to persons living in apartments (medians 27 $\mu g/m^3$ and 16 $\mu g/m^3$, P = 0.03 in campaign A and 38 $\mu g/m^3$ and 22 $\mu g/m^3$, P = 0.02 in campaign B). A significant effect of type of residence was also found when the variability in personal exposure levels was taken into account (Proc Mixed, SAS). The estimates of the personal exposure for persons living in single-family homes and apartments were 26 $\mu g/m^3$ and 16 $\mu g/m^3$ (P = 0.02), and 34 $\mu g/m^3$ and 24 $\mu g/m^3$ (P = 0.01), in campaigns A and B, respectively, thus, similar to the medians presented above. However, smoking, exposure to environmental tobacco smoke, or time spent in areas with heavy road traffic were not significant factors in the personal exposure.

4.2 Paper II

Significantly higher indoor levels of 1,3-butadiene (1- and 7-day sampling period) and benzene (7-day sampling period) were found in homes with daily used wood-burning appliances compared to homes without, although all homes were located in the same residential area (Figure 4.4). For 1,3-butadiene, the difference was also reflected in the personal exposure. The personal exposure and indoor levels of 1,3-butadiene and benzene showed a large variation among the wood-burning group: the personal exposure of, and indoor levels for, some subjects were considerably higher than the medians. With regard to formaldehyde and acetaldehyde, median levels obtained from personal and indoor

measurements were similar, although the four most extreme acetaldehyde levels were all found in wood burners (Figure 4.4).

High correlations were found between personal and indoor levels for all substances ($r_s > 0.8$). No correlation was found between indoor and residential outdoor levels of benzene. Personal exposure levels were significantly higher than the indoor levels of 1,3-butadiene and benzene in the two groups combined and in the reference group, and for benzene also in the wood-burning group. The residential outdoor benzene concentration was also significantly lower than the indoor levels in the combined group and also in the two separate groups. For the aldehydes, there was no significant difference between personal exposure and indoor levels.

The subjects spent most of the 24-hour sampling time indoors (mean 91%, range 71–100%). Time in different microenvironments together with information related to the wood burning is shown in Table 4.1. No differences in time activities were found between the wood-burning group and the reference group.

Table 4.1. *Diary information regarding the wood burning and time spent in different microenvironments per 24 hours.*

	Mean	**Range**
Wood-burning characteristics		
Burning time (h)	8	2-20
Number of replenishments (n)	4	1-10
Amount of wood (dm^3)	114	15-360
Age of the boilers (years)	18	3-51
Time spent in different environments		
Indoors at home (%)	77	33-100
Other indoor places (%)	4.7	0-33
Workplace (%)	9.6	0-38
Outdoors (%)	6.1	0-25
Cars or buses (%)	2.8	0-21

In a linear regression model, type of wood-burning appliance (higher for wood boilers), burning time (higher with longer burning time) and number of wood replenishments (higher with few replenishments) were significant factors explaining 64% of the variation in 24-hour indoor levels of 1,3-butadiene. Similar associations were found for acetaldehyde and to some extent for formaldehyde, but not for benzene.

Personal exposure and indoor levels of acetaldehyde were significantly correlated with 1,3-butadiene and formaldehyde in the wood-burning group. In the reference group, 1,3-butadiene was correlated with personal exposure to formaldehyde ($r_s = 0.81$, $P = 0.005$), and indoor levels of benzene ($r_s = 0.79$, $P = 0.006$) and acetaldehyde ($r_s = 0.66$, $P = 0.038$). Outdoors, no correlations were found between the compounds.

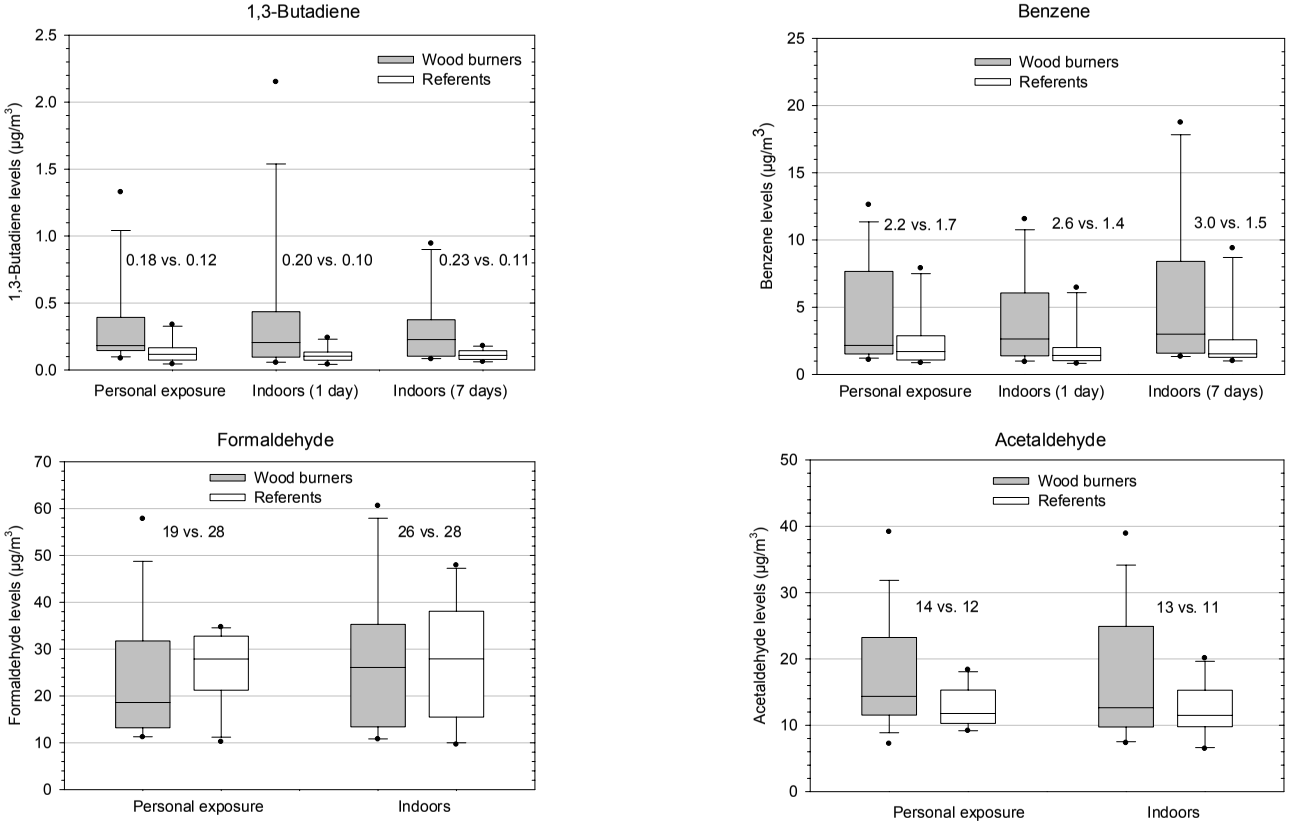


Figure 4.4. *Personal exposure and indoor levels (µg/m^3) of 1,3-butadiene, benzene, formaldehyde, and acetaldehyde for the wood-burning group (grey boxes) and the reference group (unfilled boxes). The box plots show the 10th, 25th, 50th (i.e., the median), 75th, and 90th percentiles, and the dots represent the outliers. The median values are also displayed in the graphs.*

4.3 Paper III

The particle-associated PAHs; BaP, indeno(1,2,3-cd)pyrene (IcdP), benzo(ghi)perylene (BghiP), and coronene (Cor) were significantly higher in homes with wood-burning appliances, compared to homes without (Figure 4.5a). The median indoor level of BaP was more than four times higher in the wood-burning homes compared to the reference homes (0.52 ng/m^3 vs. 0.12 ng/m^3). Also, levels of some of the semi-volatile PAH compounds, such as anthracene (Ant), benzo(ghi)fluoranthene (BghiF), cyclopenta(cd)pyrene (CcdP), benz(a)anthracene (BaA), and chrysene/triphenylene (Chr/Tri), was higher in the wood-burning homes than in the reference homes (Figure 4.5b). The semivolatile PAHs were found both in the gaseous and the particulate phase (Figure 4.6) and the levels are therefore expressed as the sum of these phases.

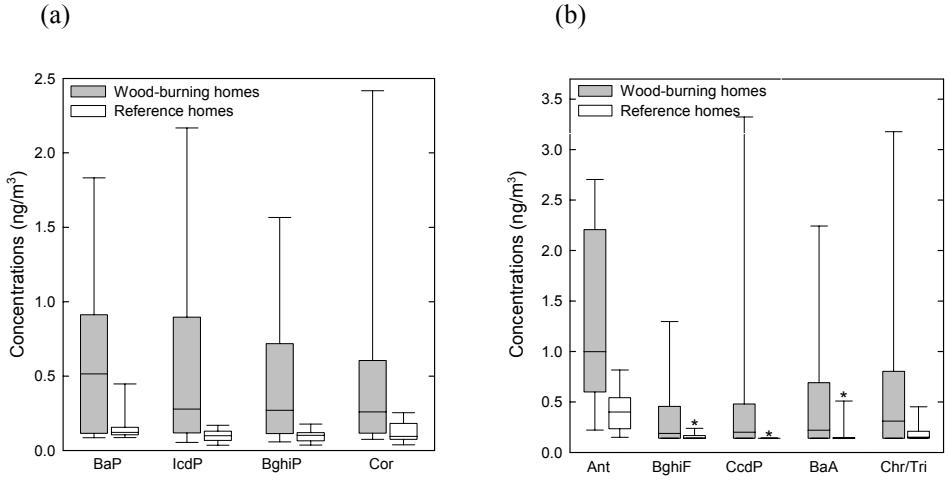


Figure 4.5. *Indoor levels of (a) the particulate (5- to 7-ring) polycyclic aromatic hydrocarbons (PAHs); and (b) the semi-volatile (3- to 4-ring) PAHs. Levels were significantly higher in the wood-burning homes (grey boxes) than in the reference homes (unfilled boxes). The box plots show the 10th, 25th, 50th (i.e., the median), 75th, and 90th percentiles. *The indoor levels in most of the reference homes were below the limit of quantification (LOQ).*

The relations between the gaseous and particulate phases in the wood-burning homes, the reference homes, and outdoors were similar for the 3-ring and the 5- to 7-ring PAHs as shown in Figure 4.6. The gaseous phase accounted for over 90% of all 3-ring PAHs but was absent for the 5- to 7-ring PAHs. Retene, an alkylated phenanthrene, was found in the gaseous phase with somewhat lower fraction outdoors (86%) compared to the other 3-ring PAHs, which all have lower molecular weights. For the 4-ring PAHs, the gaseous fractions were 77–88% in the wood-burning homes. In the reference homes and outdoors, similar relations as in the wood-burning homes were found for fluoranthene and pyrene, while the gaseous fractions of the other 4-ring PAHs (2-metylpyrene to chrysene/triphenylene) could not be estimated due to a large number of samples below LOQ in the reference homes. This was the case also for the

gaseous phase outdoor samples of cyclopenta(cd)pyrene, benzo(a)anthracene, 2-methylpyrene, benzo(ghi)fluoranthene and chrysene/triphenylene, indicating that these PAHs are were more likely found associated to particles.

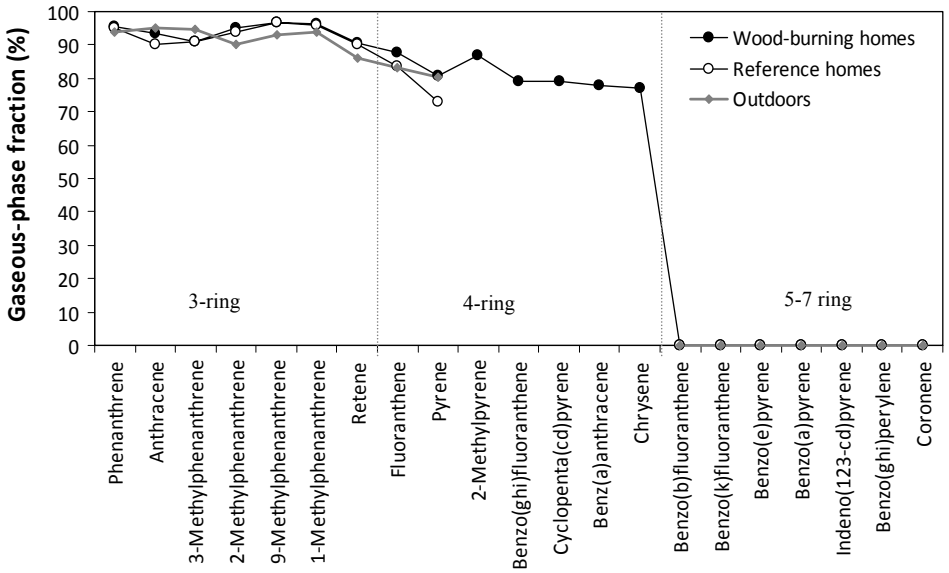


Figure 4.6. *Median fractions (%) of the gaseous phase PAHs (gaseous phase divided by the sum of gaseous and particulate phase) in the wood-burning homes, the reference homes, and outdoors at the rooftop of the garage in the residential area. For the 4-ring PAHs, such as 2-methylpyrene to chrysene/triphenylene, the gaseous fractions could not be estimated in the reference homes (few samples above the LOQ) and outdoors (almost all gaseous samples below the LOQ). A line connecting the points has been added to make the graph clearer and emphasize the differences between the groups.*

Retene, a suggested marker for wood smoke, was only somewhat higher in the wood-burning homes compared with the reference homes. As for benzene and 1,3-butadiene (in ***Paper II***) the indoor levels of the increased PAHs showed a larger variation among the wood-burning homes compared to the reference homes. Type of appliance showed a significant impact on the indoor levels of anthracene, indeno(1,2,3-cd)pyrene, and benzo(ghi)perylene with higher levels for homes with wood boilers. None of the factors were found to significantly influence the other PAHs that were elevated in the wood-burning homes. However, the outdoor levels were significantly higher than the levels inside the reference homes for several PAHs. In the wood-burnings homes, generally, fewer significant differences were found and only benzo(b)fluoranthene was significantly higher outdoors among the 5- to 7-ring PAHs. The reverse relationship with higher levels indoors than outdoors was generally found for the methylated phenanthrenes.

The total PAH cancer potency (sum of BaP equivalents of 15 PAHs) was significantly higher (about 4 times) in the wood-burning homes compared with the reference

homes, with BaP being the largest contributor (Figure 4.7), while phenanthrene made the largest contribution to the total PAH concentration in air. Fluoranthene was important both regarding the concentrations (6–14%), and the cancer potency (18–20%) (Figure 4.7).

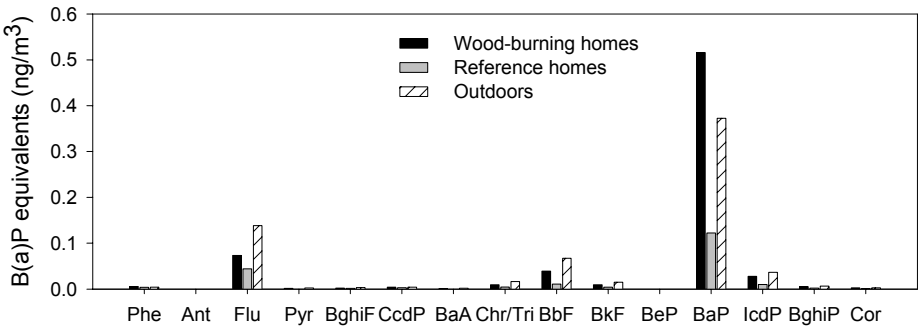


Figure 4.7. *The cancer potency of individual PAHs, expressed as benzo(a)pyrene equivalents BaPeqs (ng/m^3), for the wood-burning homes, the reference homes, and outdoors. The bars show median potencies. BaPeqs have been calculated from the individual PAH indoor concentration, multiplied by its toxic equivalent factor (TEF). For the abbreviations used for the PAH compounds, see Table 1 in* ***Paper III****.*

In the wood-burning homes, relatively high correlations were found between all the semivolatile 4-ring PAHs and also the 3-ring retene (r_s = 0.60–0.97), and between all the particulate 5- to 7-ring PAHs (r_s = 0.77–0.98). In addition, the 3-ring anthracene was correlated with all PAH compounds (e.g., BaP as shown in Figure 4.8) except benz(a)anthracene and the methylated phenanthrenes in the wood-burning homes (r_s = 0.58–0.85), while it was correlated only to 2-methylpyrene in the reference homes. High correlations were found between all the methylated phenanthrenes (r_s > 0.76), both in the wood-burning homes and in the reference homes.

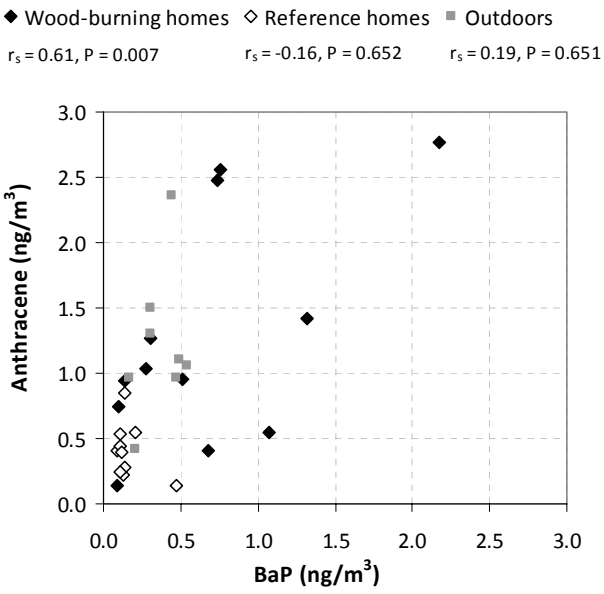


Figure 4.8. *The association between indoor levels of anthracene and BaP in the wood-burning homes, in the reference homes, and outdoors. Spearman's rank correlation coefficient is presented.*

4.4 Paper IV

Personal and stationary measurements of the concentrations of benzene, 1,3-butadiene, and the aldehydes were similar or mostly similar, and are therefore presented as a combined average concentration in Table 4.3. As expected, the concentrations of the four compounds were higher in the wood smoke sessions compared to the clean air sessions (Table 4.3). The concentration of benzene was 10–50 times higher in the wood smoke sessions compared to the clean air sessions. The differences between the wood smoke sessions and the clean air sessions were smaller for 1,3-butadiene and the aldehydes (about 5–25 times). Somewhat higher concentrations were obtained in wood smoke session 1 than 2 (Table 4.3). Toluene, xylenes, and naphthalene levels were higher in the wood smoke compared with the clean air sessions (Table 3 in ***Paper IV***), but the differences were much smaller compared to those for benzene, 1,3-butadiene, and the aldehydes. The toluene/benzene ratios were lower in the wood smoke sessions. The concentrations of individual PAHs were relatively high in duplicate samples from the second wood smoke session (Table 4 in ***Paper IV***). For example, the level of BaP was about 20 ng/m^3 and the level of fluoranthene was about 44 ng/m^3.

There were no differences in mass concentrations between $PM_{2.5}$ and PM_1 in any of the two wood smoke sessions or the clean air sessions, and similar results for mass concentrations were obtained for both stationary and personal measurements. Consequently, the particle mass measurements were evaluated together and the results are presented in Table 4.3.

The results of the continuous ELPI measurements (particle number concentrations) confirmed that nearly all particles were <1µm. The average particle number concentrations were almost two times higher in the first wood smoke session compared to the second (Table 4.3). The particle number concentration was clearly higher during the first 2.5 hours in wood smoke session 1, and was dominated by ultra fine particles (UFP) with diameters <100 nm (stages 1-3) as shown in Figure 4.9. The UFP fraction was 65% in the first wood smoke session compared to 28% in the second. Particle size distributions resulted in a geometric mean diameter of 42 nm (σ_g=1.7) and 112 nm (σ_g=1.4) in the first and second wood smoke sessions, respectively. The difference in number concentration observed between the first and second wood smoke sessions was not clearly reflected in the mass concentrations measured with the cyclones and the TEOM instrument (see Table 4.3 and Figure 4.9 for the TEOM mass concentration).

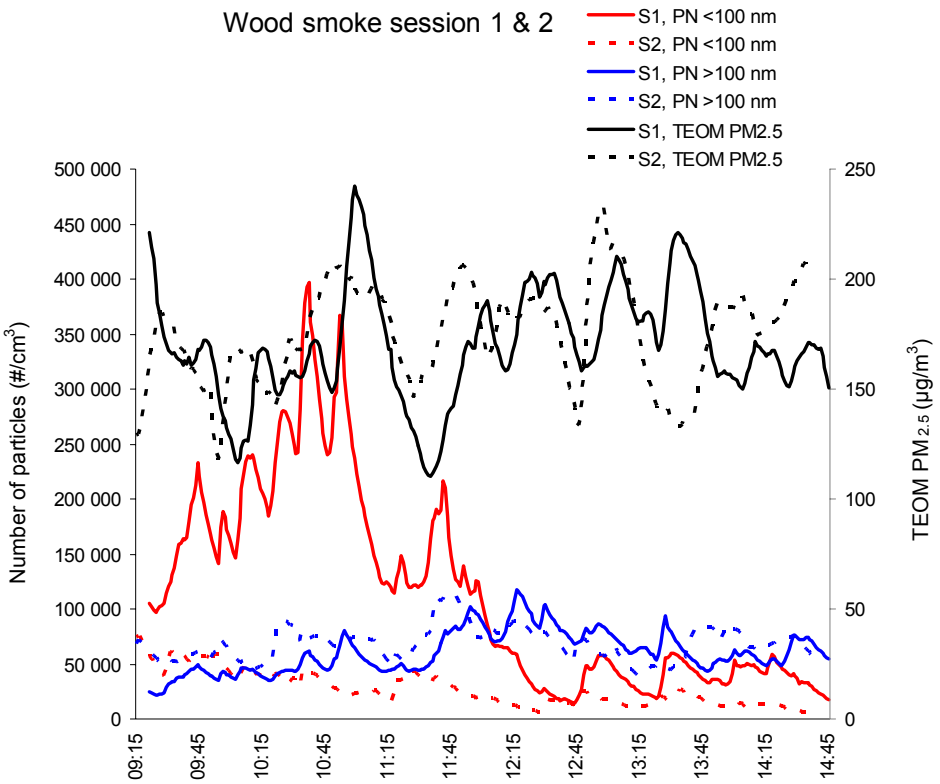


Figure 4.9. *Particle number concentrations and particle mass ($PM_{2.5}$), as measured online using an electric low-pressure impactor (ELPI) and a tapered element oscillating microbalance (TEOM), respectively, during 5.5 h in an exposure chamber with wood smoke*

Table 4.3. *Particle mass concentrations (μg/m^3), particle number concentrations (#/cm^3), black smoke levels (10^{-5}/m), and volatile organic compounds (μg/m^3) during two sessions of experimental exposure to wood smoke and clean air in healthy human subjects measured with time-integrated samplers and continuous instruments*

	Wood smoke						**Clean air**					
	Session 1			**Session 2**			**Session 1**			**Session 2**		
	N	**Mean**	**SD**	**N**	**Mean**	**SD**	**N**	**Mean**	**SD**	**N**	**Mean**	**SD**
Benzene	5	31	2.0	5	20	0.8	3	1.6	0.23	3	0.61	0.12
1,3-Butadiene	5	6.2	0.30	5	3.9	0.29	3	0.27	0.04	3	0.61	0.12
Formaldehyde	5	124	18	5	64	10	3	7.7	3.1	3	5.0	0.00
Acetaldehyde	5	73	8.0	5	40	8.6	3	6.3	3.8	3	7.7	2.1
Particle mass (cyclone)	8	272	21	8	242	8.3	5	<LOQ		6	<LOQ	
Black smoke	8	81	8.6	8	106	11	2	1.7	0.07	3	1.3	0.37
Particle mass (TEOM)	C	170		C	175		C	15		C	11	
Particle number	C	180,000		C	95,000		C	4,400		C	7,500	

In the clean air sessions, the majority (70–80%) of the particles were ultrafine, and the total number concentrations were 4,400 and 7,500/cm^3, respectively (Table 4.3).
In the clean air sessions, low particle mass concentrations (cyclones and TEOM instrument) were obtained (Table 4.3), with six of the cyclone samples below the detection limit and the concentrations in the other five samples varying from 14 $\mu g/m^3$ to 27 $\mu g/m^3$.

As expected, the black smoke levels were much higher in the wood smoke sessions than in the clean air sessions (Table 4.3). In the wood smoke sessions, the concentrations of K and Zn were much higher compared with the clean air sessions. Also, the concentrations of Br, Rb, and Pb were higher (Table 2, ***Paper IV***).

The CO levels were at least 18 times higher during the wood smoke than during the clean air sessions (13 ppm, and 9.1 ppm versus <0.5 ppm). Some influence of replenishing the stove could be seen and the peak concentration of 22 ppm was obtained in connection with one of the replenishments. Wood smoke had an impact also on the concentrations of NO_2 (about 8 times higher), NO (about five times higher), and CO_2 (about 2 times higher).

Subjective symptoms were generally weak, while clear objective signs were found, for example, in biomarkers of inflammation and in factors of coagulation (Barregard et al., 2008; Barregard et al., 2006).

5 Discussion

This thesis reports personal exposure to, and/or indoor and outdoor levels of formaldehyde ***(Papers I–II)***, acetaldehyde, 1,3-butadiene, benzene ***(Paper II)***, and PAHs ***(Paper III)*** found in the Swedish general population. These compounds belong to different chemical classes having different sources and health effects, but all of them are known or potential carcinogens and are ubiquitous in the air. Worldwide, exposure to these pollutants exists in a wide range of concentrations, depending on local sources, topography, and meteorology. Exposure assessment studies have been conducted in several countries to a varying extent and by use of different measures such as personal and/or microenvironment concentrations and modeling data. The results from those studies are first compared with the findings in this thesis (section 5.1), and thereafter the relations between the different types of measures and the variability in exposure levels are discussed (section 5.2). However, comparing results between different studies can often be difficult and caution must be taken, since results are from different seasons and sampling sites, and various sampling times, type of samplers and analytical techniques are used.

Domestic wood burning is gaining increasing focus as an important source of many air pollutants. Its impact on the personal exposure and indoor concentrations of formaldehyde, acetaldehyde, 1,3-butadiene, benzene, and PAHs has been investigated in ***Papers II*** and ***III***. As described in the introduction, the most extensive use of wood burning takes place in the developing world in unvented or poorly ventilated wood stoves, and measurements of PM and to a lesser extent also organic compounds have shown very high exposures in these countries (Naeher et al., 2007). In developed countries such as Sweden, lower exposure can be expected as a result of the use of other types of wood-burning appliances and different needs for heating and cooking, however, very limited data exists for comparison with our finding (section 5.1). The impact of wood burning and factors related to the burning conditions on exposure and indoor levels of pollutants is discussed in section 5.3.

Several of the pollutants emitted from wood burning are also emitted from a wide range of other sources (section 5.4), but specific compounds have been used in the literature for investigating the contribution of wood smoke (section 5.5). The mix of pollutants emitted from different sources makes it difficult to study the health effects of exposure to wood smoke. ***Paper IV*** describes an experimental set-up for studying markers of inflammation and coagulation in humans after exposure to wood smoke. With this approach the contribution from other sources to the exposure is avoided. The levels and composition of the above-mentioned pollutants, together with some other hydrocarbons, fine particles, black smoke, and associated trace elements in the wood smoke sessions were discussed and compared to other studies and findings in ***Papers II*** and ***III*** (sections 5.1 and 5.6) to highlight the significance of the degree of exposure in the chamber. Risk assessment is important in the view of risk management, and the cancer risk related to an exposure to formaldehyde ***(Paper I)*** and wood smoke ***(Papers II*** and ***III)*** found in this thesis has been estimated (section 5.8). However, exposure to wood smoke has also been associated with respiratory and cardiovascular effects (section 5.7).

5.1 Personal exposure and indoor and outdoor concentrations

5.1.1 Formaldehyde and acetaldehyde

While formaldehyde is a widely measured indoor air pollutant, acetaldehyde has been the subject of fewer studies. Unfortunately, the GMD sampler used in ***Paper I*** was unable to measure acetaldehyde, as shown by the comparison between GMD samplers and Radiello samplers. The GMD sampler has been validated in work environments but not in home environments, where longer sampling times often are used (Lindahl et al., 1996). This fact shows the importance of using samplers validated for their specific use.

The personal exposure to formaldehyde was similar in Gothenburg, Borås (***Paper I***), and Hagfors (***Paper II***), which was also the case for the indoor levels. Moreover, no significant difference between the wood-burning group and the reference group could be seen for formaldehyde or acetaldehyde in the personal and indoor measurements. Nor was any impact of wood burning on personal exposure seen in ***Paper I***, but only three subjects reported exposure to wood smoke.

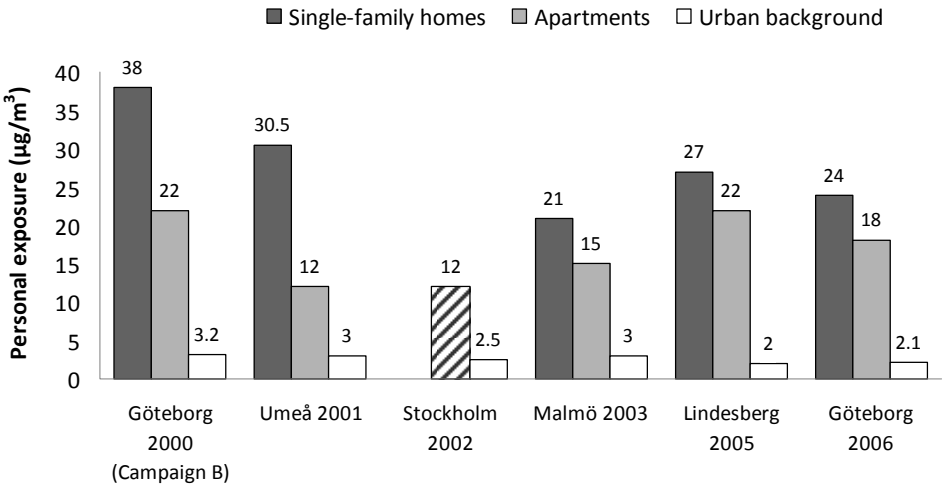


Figure 5.1. *The median personal formaldehyde exposures (µg/m^3) for subjects living in single-family houses and apartments, together with the median urban background reported from the different cities (in years 2000–2006) included in the national project regarding the general population's exposure to carcinogenic compounds, funded by the Swedish EPA. Note that the personal exposure in Stockholm is not stratified by type of home.*

The personal exposure to formaldehyde in ***Papers I*** and ***II*** (medians 23 µg/m^3) was within the range of medians found among the general population in five Swedish cities measured within the Swedish EPA environmental monitoring program (12–27 µg/m^3) (Johannesson et al., 2008). The median levels show some variation between the cities,

which may be partly reflected by variation in the fraction of people living in single-family homes or apartments. Therefore, the median personal exposures are shown in Figure 5.1, stratified by type of residence (except for Stockholm). The effect of house type is further discussed in section 5.4.

The range of median personal exposure observed in Sweden is in agreement with or somewhat higher than studies from countries including the United States, France, Finland, and Mexico (Dodson et al., 2007; Gonzalez-Flesca et al., 1999; Jurvelin et al., 2001; Liu et al., 2007; Sax et al., 2006; Serrano-Trespalacios et al., 2004) (Table 5.1). A review of personal exposure to formaldehyde (six studies) showed a median level of 26 $\mu g/m^3$ (Koistinen, 2008). A recently conducted study showed a mean of pooled personal exposures within four European cities (Brussels, Budapest, Leipzig, and Helsinki) of 17 $\mu g/m^3$ (Bruinen de Bruin et al., 2008). If occupational exposure occurs, the personal exposure could be much higher as illustrated by the subject in ***Paper I***. This subject had been occupationally exposed during spray-painting with acid-curing paint, which emits formaldehyde during the curing process and had a 24-hour average personal exposure of 566 $\mu g/m^3$. In Sweden the occupational exposure limit value for a whole workday is 600 $\mu g/m^3$, and this subject must thus have been exposed above this limit value. The concurrently measured indoor levels also show some variations between the countries, which may reflect seasonal effects and different building characteristics. During the 1970s and 1980s formaldehyde attracted attention, as very high (more than a magnitude higher than nowadays) indoor formaldehyde concentrations were found in schools and homes where occupants reported health complaints (Azuma et al., 2006). As a consequence, many countries established indoor air quality guidelines and standards for regulation of emissions from sources such as particle board and urea-formaldehyde foam insulation (Azuma et al., 2006). Personal exposure and indoor levels of acetaldehyde also showed some variation between countries, but the range was somewhat smaller than for formaldehyde (Table 5.1). As expected from those studies (Dodson et al., 2007; Gonzalez-Flesca et al., 1999; Jurvelin et al., 2001; Liu et al., 2007; Sax et al., 2006; Serrano-Trespalacios et al., 2004), the outdoor levels of formaldehyde and acetaldehyde were several times lower than personal exposure and indoor levels (***Papers I*** and ***II***).

However, the above-mentioned studies did not focus on the impact of wood burning or discuss wood burning as a potential source. Only a few studies have investigated indoor aldehyde levels and domestic wood burning in the developed world. These are from Canada, where Lévesque et al. (2001) found no difference between homes with and without wood-burning appliances regarding indoor formaldehyde levels. The same result was reported by Gilbert et al. (2006; 2005) for formaldehyde and for acetaldehyde (Gilbert et al., 2005), which is consistent with the findings in ***Paper II***. A recently performed study from France reported minor impact on indoor air quality regarding formaldehyde in three homes with a fireplace or a wood stove (Mandin et al., 2008). In developing countries, however, where simple and often poorly ventilated cook stoves are used, levels are much higher. A median level of formaldehyde as high as 652 $\mu g/m^3$ was found inside 20 Indian homes where wood stoves were used for cooking (Raiyani et al., 1993b). Despite the failure to detect a possible impact of wood burning on the levels of formaldehyde and acetaldehyde in Hagfors, the chamber results in ***Paper IV*** (10–20 times

and 5–10 times higher, respectively, for formaldehyde and acetaldehyde levels during exposure to wood smoke compared to clean air) show that wood burning can indeed increase levels of aldehydes. Also, our findings of a few wood burners having elevated acetaldehyde levels, together with the positive association between 1,3-butadiene and acetaldehyde for wood burners, could possibly have been caused by acetaldehyde in wood smoke.

5.1.2 1,3-Butadiene

Although the wood-burning group in the town of Hagfors in ***Paper II*** had significantly higher personal exposure (0.18 μg/m^3) and indoor levels (0.20 μg/m^3) than their reference group (0.12 and 0.10 μg/m^3, respectively), their median personal exposure was about two to three times lower than the medians found within the environmental monitoring program coordinated by the Swedish EPA among the general population in five Swedish cities (except in Gothenburg, where the median personal exposure was lower than in Hagfors) (Johannesson et al., 2008; Modig et al., 2004). Moreover, the median outdoor level in Hagfors (0.11 μg/m^3) was similar to or higher than in urban background, while lower than at street level in these cities (Johannesson et al., 2008; Modig et al., 2004). Median personal exposure and indoor levels reported by two studies from the United States were similar to or (up to four times) higher than for the wood-burning group in our study, while outdoor levels were similar (Dodson et al., 2007; Sax et al., 2006). Indoor and outdoor levels comparable to those in our study were, however, reported in a Canadian study (Zhu et al., 2005). Moreover, ten to twenty times higher median personal exposure and indoor levels, and also higher outdoor levels, were found in Mexico City (Serrano-Trespalacios et al., 2004). The higher traffic intensity in more densely populated cities and the impact of tobacco smoke are factors that may have contributed to the generally higher levels found in other studies compared with ours. In outdoor air, elevated 1,3-butadiene levels have been reported in relation to an extended use of wood burning appliances in an Alpine village (Gaeggler, 2008). The experimental study ***(Paper IV)*** showed clearly that wood burning increased the level of 1,3-butadiene in the chamber (Table 4.3).

5.1.3 Benzene

Significantly higher 7-day median indoor levels of benzene (3.0 μg/m^3) were found in the wood-burning homes compared to the reference homes (1.5 μg/m^3) ***(Paper II)***. Also the personal exposure and 1-day indoor levels were higher, but the differences were not statistically significant. An impact on the indoor levels of benzene was also seen in French homes (Mandin et al., 2008). Sinha et al. (2006) reported much higher indoor levels of benzene for people in India who cook indoors with wood fuels (23–50 μg/m^3). Similar concentrations (19–34 μg/m^3) were found in the chamber during the wood smoke session, which were much higher than during the clean air session (0.54–1.7 μg/m^3) ***(Paper IV)***.

Table 5.1. *Summary of the median personal exposure (P) to, and indoor (In) and outdoor (Out) concentrations of formaldehyde and acetaldehyde (μg/m^3) from some recent international studies.*

			Formaldehyde			Acetaldehyde			
City, country	**Year**	**Season**	**P**	**In**	**Out**	**P**	**In**	**Out**	**Reference**
Gothenburg and Borås, Sweden	2000-01	Autumn	23	27	4.0	-	-	-	Paper I
Hagfors, Sweden	2003	Winter	23	28	3.1	13	12	3.1	Paper II
Mexico City, Mexico	1998-99	Full year	17	16	5.2	11	14	3.9	Seerano-Trespalacios et al. 2004[a]
Nancy, France	1997		15	25	2.9	19	24	2.1	Gonzales-Flesca et al. 1999[b,f]
Helsinki, Finland	1997	Summer + fall	22	40	1.8	13	14	2.2	Jurvelin et al. 2001[c]
Three cities, USA	1999-2001	Full year	21	20	6.4	19	23	5.4	Liu et al. 2006, 2007[d]
New York City, USA	1999	Winter + summer	17	16	3.1	17	13	3.2	Sax et al. 2006[e]
Los Angeles, USA	1999	Winter + summer	21	18	4.0	11	12	3.8	Sax et al. 2006[e]
Boston, USA		Winter + summer	18	16	0.38	10	8.3	1.0	Dodson et al. 2007[f]

[a]Non-ocupationally exposed and non- or light smoking volunteers

[b]Non-smoking volunteers working outdoors

[c]Randomly selected subjects (The EXPOLIS study)

[d]Non-smoking volunteers (The RIOPA study)

[e]Non-smoking students (The TEACH study)

[f]Non-smoking volunteers (The BEAM study)

The median personal exposure in the wood-burning group (2.2 μg/m^3) and the reference group (1.7 μg/m^3) were within the range of median personal exposures found in the general population in five Swedish cities during the years 2000 to 2006 (0.8–2.8 μg/m^3), despite the fact that some of these studies include smokers and that these cities are more densely populated compared to Hagfors, and hence have a higher impact from traffic (Johannesson et al., 2008; Modig et al., 2004). Similar or higher personal exposures are reported within the European study (EXPOLIS) performed in 1996–1997, where the geometric means ranged from 2–3 μg/m^3 in Helsinki, Basel, and Oxford, and 8 and 12 μg/m^3 respectively in Prague and Athens (Saarela et al., 2003). Benzene in petrol was reduced in many countries during the 1990s. However, mean personal exposures measured in 12 European cities showed similar levels (2.0–9.4 μg/m^3) in 2003, with the highest exposures mostly found in southern European cities (Bruinen de Bruin et al., 2008). The European population has been estimated to be exposed to benzene at a median level of 4.2 μg/m^3 (Koistinen, 2008). The levels in our study were also in agreement with median personal exposures (1.6–4.2 μg/m^3) reported in some recently published studies from the United States (Dodson et al., 2007; Sax et al., 2006; Sexton et al., 2004), but lower than those in Mexico City (Serrano-Trespalacios et al., 2004). The outdoor levels in Hagfors were somewhat lower than the personal and indoor levels, which is consistent with results found in many other studies (Bruinen de Bruin et al., 2008; Dodson et al., 2007; Modig et al., 2004; Sax et al., 2006; Sexton et al., 2004). The median outdoor level (1.2 μg/m^3) was similar to concentrations found in urban background air measured within the environmental monitoring program (Johannesson et al., 2008; Modig et al., 2004) and in residential areas where wood burning is common in Finland and Sweden (Finnberg et al., 2004; Hellén et al., 2008). Hellén et al. (2008) reported that the highest benzene levels occur in the afternoon when the fireplaces also are most frequently used.

5.1.4 Polycyclic aromatic hydrocarbons (PAHs)

The indoor levels of PAHs were generally higher in the wood-burning homes compared to the reference homes and the differences were for several PAHs, such as BaP, statistically significant ***(Paper III)***. The median concentration of BaP in the wood-burning homes (0.52 ng/m^3) was within the range (0.34–0.88 ng/m^3) reported for American homes during operation of airtight wood stoves (Daisey et al., 1989; Traynor et al., 1987), while use of a non-airtight wood stove gave rise to much higher levels (2–490 ng/m^3) (Traynor et al., 1987). The BaP concentrations in the reference homes in Hagfors were similar to the indoor concentrations measured by Traynor et al. (1987) on days without wood burning. In addition, higher levels of benzo(ghi)perylene and indeno(1,2,3-cd)pyrene were found on days when a wood stove was operated compared to days without (Daisey et al., 1989; Traynor et al., 1987), which is in agreement with the findings in ***Paper III***. An impact of wood burning on concentrations of particulate PAHs was also found in three French homes (Mandin et al., 2008). In a Norwegian study, a single house was studied, and wood heating was found to increase the indoor concentrations of PAHs (Alfheim and Ramdahl, 1984). The effect was more pronounced when an open fireplace was in use compared with an airtight stove, with BaP levels of 18 and 13 ng/m^3 measured close to the fireplace. These levels can be compared with the BaP concentrations found in the chamber during the wood smoke session (19 and 21 ng/m^3) ***(Paper IV)***. High levels of BaP and other PAHs have been measured in rural homes where wood is burned for cooking and heating in unvented fireplaces in India and Burundi

(Bhargava, 2004; Raiyani et al., 1993a; Viau et al., 2000). The BaP levels in the reference homes in Hagfors were similar to personal exposures (medians about 0.1 ng/m^3) found in the Swedish general population (Johannesson et al., 2008). The levels of BaP were higher in the wood-burning homes in the present study than in non-smoking homes in Germany, the Netherlands, and the United States (Fischer et al., 2000; Fromme et al., 2004; Naumova et al., 2002), despite the fact that the homes in these other studies were located in densely populated cities affected by traffic and, in some cases, also large industries.

The outdoor BaP levels (median 0.37 ng/m^3) in Hagfors ***(Paper III)*** were higher than the outdoor BaP concentrations at street level (medians 0.08–0.28 ng/m^3) and in the urban background (medians 0.04–0.31 ng/m^3) during late fall or winter in five Swedish cities (Johannesson et al., 2008). In areas where wood burning for residential heating is common, average outdoor BaP levels of 0.18–1.7 ng/m^3 have been reported in Sweden, Canada, Finland, and the United States (Hawthorne et al., 1992; Hellén et al., 2008; Johansson et al., 2004a; Ward et al., 2006), with the highest concentrations on cold days (Johansson et al., 2004a). Similar or higher outdoor concentrations (about 1–4 ng/m^3) have been reported in a Swedish city with an aluminum production plant in 2000–2004 (Hanberg et al., 2006). The outdoor levels in Hagfors are relatively high also in comparison with studies from the United States, Japan, and Germany (Fromme et al., 2004; Naumova et al., 2002; Ohura et al., 2004).

5.2 Relations between personal exposure and indoor and outdoor concentrations

Personal exposure reflects the time spent in different microenvironments (e.g., home, workplace, cars or buses, shops, and restaurants) and the concentration within the specific microenvironment. The high correlations ($r_s > 0.8$ and $P < 0.001$) between the residential indoor concentrations and personal exposure found for formaldehyde in campaign B ***(Paper I)*** and for formaldehyde, acetaldehyde, 1,3-butadiene, and benzene among the wood-burning group and the reference group ***(Paper II)*** are a result of the large proportion of the sampling time the subjects spent at home in the two studies (65% and 77%, respectively) and for formaldehyde and acetaldehyde, the much higher concentrations indoors than outdoors. Similar correlations have been reported in other studies for formaldehyde (Dingle, 1993; Jurvelin et al., 2001) and acetaldehyde (Jurvelin et al., 2001) and also to some extent for benzene (Phillips et al., 2005). The indoor levels of formaldehyde were similar to or significantly higher than the personal exposure in ***Paper I***. Hence, the concentration of formaldehyde in the home is likely to make an important contribution to personal exposure of people not occupationally exposed. As expected, when using the bedroom concentration in campaign B ***(Paper I)*** as a proxy of personal exposure, we were able to account for 90% of the variation in personal exposure (R^2). An extended model including the outdoor contribution was also tested, since spending time outdoors where the concentration is significantly lower would decrease the personal exposure. The small improvement (R^2 increased to 91%) is consistent with the short time spent outdoors (about 6%), although for some subjects spending a large proportion of the sampling time outdoors, the model will indeed improve the estimate. In a third model, we included other indoor environments by assuming the concentration to be in the same range as in the apartments in our study. This assumption is supported by studies reporting lower concentrations indoors at workplaces compared to home

environments (Bruinen de Bruin et al., 2008; Jurvelin et al., 2001). However, Loh et al. (2006) reported relatively high formaldehyde levels in certain store types, such as housewares stores (GM of 53 μg/m^3). This third model gave the best result and explained 92% (R^2) of the variation of measured personal exposure. However, all models had an ICC value close to 1.00, which indicated very good agreements between the measured and estimated personal exposure. Since the purpose of modeling personal exposure is to reduce effort for the researcher and the participants, indoor measurements for assessing the personal exposure could be recommended for most purposes, if no occupational exposure occurs (Dingle, 1993; Jurvelin et al., 2001; Liu et al., 2007). These three studies also showed similar results for acetaldehyde. However, making human exposure estimates based on only ambient air levels of formaldehyde may cause great underestimations as illustrated by the low mean exposure estimate (1.2 μg/m^3) by Bostrom et al. (1994) for the Swedish population.

Using indoor and outdoor measurements for estimating personal exposure to other air pollutants with significant outdoor sources, such as benzene, may cause an underestimation of the exposure (Gonzalez-Flesca et al., 1999). For benzene, filling the car with petrol and spending time in areas with traffic have shown to be important contributors to personal exposure (Edwards et al., 2001; Horton et al., 2006). Personal exposure to benzene was statistically significantly higher in both the wood-burning and the reference group compared to indoor and outdoor air ***(Paper II)***, consistent with other studies (Serrano-Trespalacios et al., 2004; Wallace, 1996). For 1,3-butadiene, there was no statistically significant difference between personal exposures and indoor levels for wood burners, while the referents had higher personal exposure compared with indoor levels. These facts may suggest that the personal exposure for the wood-burning group had a dominant indoor source.

The outdoor concentrations of PAHs measured in the center of the residential area were generally higher than indoor concentrations ***(Paper III)***. An exception was the methylated phenanthrenes, which were higher indoors. Especially, the particulate PAHs in the reference homes were lower than outdoors, consistent with the fact that particulate PAHs are reported to predominantly originate from outdoor sources, whereas 2- to 3-ring PAHs may be produced indoors (Naumova et al., 2002; Ohura et al., 2004). However, the indoor levels were more similar to the outdoor levels in the wood-burning homes, suggesting impact of wood burning in those homes.

5.3 Impact of domestic wood burning

The elevated indoor levels of 1,3-butadiene, benzene, and some of the PAHs can result from intrusion of outdoor air and/or leakage from the wood-burning appliance. Consequently, exposure to wood smoke can occur both outdoors and indoors. As discussed in the previous section 5.1, very few studies have focused on the impact of wood burning on personal exposure and indoor levels, while more studies have investigated the contribution of wood smoke to ambient air (Glasius et al., 2006; Hedberg and Johansson, 2006; Jeong et al., 2008; Ward et al., 2006; Wu et al., 2007).

A large variation was found in personal exposure and indoor concentrations of 1,3-butadiene, benzene and some of the PAHs measured in the wood-burning homes, while not in the reference homes (Figures 4.4 and 4.5). There is a wide range of

emission factors for these compounds found by different studies characterizing wood smoke in controlled burning experiments (Hedberg et al., 2002; Johansson et al., 2004b; McDonald et al., 2000; Schauer et al., 2001) ***(Papers II*** and ***III)***. Emission factors can vary widely with many factors related to the type of wood (e.g., tree species, water content), the wood-burning appliance (e.g., burner, stove, fireplace) and operational practice (e.g., small or large batches of wood) (Hedberg et al., 2002; Johansson et al., 2004b; McDonald et al., 2000). These factors may show an even higher variation in field studies; however, they reflect the real situation.

Information on possible factors influencing the emissions, and hence the indoor and outdoor concentrations, was provided by the wood-burning households and was included in a multiple regression model investigating the impact on 24-hour indoor levels of 1,3-butadiene, benzene, formaldehyde, acetaldehyde, and the significantly elevated PAHs in the wood-burning homes ***(Papers II*** and ***III)***. Use of a boiler was found to increase the indoor levels of 1,3-butadiene, acetaldehyde, and some of the PAHs (anthracene, indeno(1,2.3-cd)pyrene and benzo(ghi)perylene). Type of wood-burning appliance was also found to be important in the study by McDonald et al. (2000), where emission rates of 350 elements, inorganic compounds, and organic compounds, among them 1,3-butadiene, benzene, formaldehyde, acetaldehyde, and PAHs from residential wood combustion were quantified, with higher emission rates for wood stoves than for fireplaces. Moreover, a long burning time was associated with higher indoor levels of 1,3-butadiene, while a large number of wood replenishments was associated with lower levels ***(Paper II)***. Johansson et al. (2004b) showed that lower emissions can be achieved with frequent replenishments because of more complete combustion obtained with a smaller amount of wood. At poorly optimized burning conditions with low oxygen and low temperature, the emission of incomplete combustion products can be considerable (Johansson et al., 2004b). Also, connecting the wood boiler to a heat storage tank can reduce the emissions (Johansson et al., 2004b). In addition to a large number of replenishments, a heat storage tank connected to the boiler was associated with lower acetaldehyde levels. Higher emissions have also been found among fireplaces burning hardwood (deciduous trees) compared with softwood (coniferous trees) (McDonald et al., 2000). In Sweden birch and spruce are the main types of wood burned and most of the subjects in our study used a mixture of these; therefore, no effect could be shown for this factor. The variation in the number of wood replenishments performed by the subjects may have contributed to the wide range of personal exposure levels.

In addition to the factors included in the model, the age of the boiler is important for the emissions. Old boilers are known to emit larger amounts of air pollutants compared to new wood-burning appliances (Johansson et al., 2004b). The boilers in Hagfors were relatively old (mean age 18 years, range 3–51 years), which reflect the situation in other parts of Sweden and Norway (Johansson et al., 2004a; Kocbach et al., 2006). Only two households had a boiler with an environmental certification from the Swedish National Testing and Research Institute. The water content of the wood is another factor known to be important, and the storage time varied within the wood-burning group (from 4 months to 3 years). It was not possible to include all these parameters in the model due to the limited number of homes investigated. The layout of the house is an additional factor that may affect the presence of indoor wood smoke.

Correlations between compounds may indicate similar sources and sinks. Since benzene, 1,3-butadiene, formaldehyde, acetaldehyde, and the PAHs are all constituents of wood smoke as well as of motor vehicle exhaust and other combustion processes, a correlation between these compounds is expected. We found an association between benzene and 1,3-butadiene both for personal exposure and for indoor levels. These results are in agreement with other studies (Kim et al., 2001; Modig et al., 2004). High correlations between formaldehyde and acetaldehyde were found, both for personal and for indoor measurements, which is in agreement with the high correlation found inside Canadian homes (Gilbert et al., 2005). By contrast, Jurvelin et al. (2001) found a correlation between these two aldehydes for outdoor levels, but not for personal exposure or indoor levels.

5.4 Other sources, influencing factors, and effect of variability

As discussed in section 5.2, the indoor environment is important for the observed personal formaldehyde exposure in ***Paper I***. Type of home was found to have significant effect on the indoor concentrations, and hence, on the personal exposure. People living in apartments were found to have significantly lower exposure to formaldehyde compared to people living in single-family houses. A difference by type of residence has been reported in several other Swedish studies (Lindahl et al., 1999; Norlén and Andersson, 1993; Sakai et al., 2004). Elevated residential indoor formaldehyde levels have been reported to be dependent on many factors, such as increased temperature and relative humidity, newly built or refurbished home, presence of particle board flooring, smoking, and low air-exchange rate (Clarisse et al., 2003; Gilbert et al., 2005; Raw et al., 2004; Sakai et al., 2004; Sax et al., 2004). The relative significance of these factors on the indoor concentrations observed is not easily resolved, as illustrated by the following discussion. A study of Swedish housing (Norlén and Andersson, 1993) indicates that apartments have higher ventilation rates compared to single-family houses resulting in lower indoor levels, but also higher temperatures, which in contrast lead to increased levels due to higher emission rate from materials (Godish and Rouch, 1986). In the same study, single-family houses were found to have higher relative humidity, which leads to increased levels (Godish and Rouch, 1986). The fact that the emission rate decreases with age would indicate that the age of the building would be important. The apartments in ***Paper I*** were indeed slightly older than the single-family houses; no effects of the age of buildings were, however, observed. Nowadays, the emission of formaldehyde from material is regulated in standards and the emission rate from materials in older buildings has declined. However, renovations may introduce new materials in both old and new homes. It is worth mentioning that the highest bedroom concentration (120 $\mu g/m^3$) was recorded in a single-family house in Gothenburg with particleboard as a building material in subfloor and partition walls.

In the residential area studied in ***Papers II–III***, wood burning constitutes an important source for 1,3-butadiene, benzene, formaldehyde, acetaldehyde, and PAHs. However, these compounds are also emitted from all types of incomplete combustion of organic material, not only wood burning, in addition to consumer products, construction materials, and furnishings. Thus, a number of different sources can have contributed to the observed personal exposure and indoor and outdoor concentrations. The influence of house type was not an issue in ***Paper II-III*****,** since all subjects lived in single-family homes. However, it is possible that indoor sources such as furnishings,

construction materials (e.g., particleboard and insulation), and consumer products may have overshadowed the impact of wood burning on the indoor levels of formaldehyde.

Traffic is a well-known source of all the measured compounds, since they are produced from incomplete combustion. Time spent in areas with heavy traffic was not associated with elevated personal exposure to formaldehyde in ***Paper I***, but the time spent in these was low. The impact of traffic on the indoor concentration in the wood-burning area is considered to have been low and evenly distributed for all the homes, since they are located in a small town and within the same residential area ***(Papers II*** and ***III)***.

Another incomplete combustion process that generates formaldehyde, acetaldehyde, 1,3-butadiene, benzene, and PAHs is cigarette smoking (IARC, 2004; WHO, 2000). Almost twice as high indoor levels of 1,3-butadiene and benzene were reported in homes with smokers compared with non-smoking homes in the United Kingdom (Kim et al., 2001; Kim et al., 2002). High levels of benzene and 1,3-butadiene have also been reported in pubs and restaurants where smoking is prevalent (Edwards et al., 2001; Kim et al., 2001). The interference of smoking was avoided in ***Papers II–III*** by selecting non-smokers and not allowing smoking inside the homes during the sampling period, although non-exposure status was not verified with any biological or indoor measurements of cotinine or nicotine. Environmental tobacco smoke (ETS) is not always avoidable and was noted in the diaries of two wood burners (duration of exposure 20 minutes and 1 hour, respectively). However, in ***Paper I*** no significant impact of active smoking or exposure to ETS on either the personal exposure or the indoor levels of formaldehyde was found.

In a study from China, where oil is frequently used for frying, cooking has been pointed out as a significant indoor source of PAHs (Zhu and Wang, 2003). However, information on the amount of time the household members in Hagfors spent cooking during the sampling time showed no difference between the wood-burning group and the reference group ***(Paper III)***.

Outdoor concentrations are dependent on many factors, not only the sources. Meteorological factors such as wind speed and direction, and mixed boundary layer height may be important, especially in wintertime during cold periods with stagnant weather conditions, when the vertical mixing is low. This may have a deleterious effect on the local air quality in residential areas where wood smoke is released at relatively low heights from chimneys and trapped close to the ground (Krecl et al., 2008). In addition, the need for heating also increases when temperature is decreasing. In Hagfors, higher levels of the pollutants of interest can be expected in wintertime due to the need for heating, and at the same time, inversions occur more frequently. A seasonal effect on indoor concentrations is also possible, due to differences in factors related to ventilation affecting the infiltration of outdoor air. In wintertime, homes are often sealed by keeping the doors and windows closed (windows and doors were only open for an average of 0.1 hours during the 24-hour sampling period in ***Papers II–III***), resulting in reduced air flow in the homes. Air pollution levels emitted by indoor sources can therefore build up inside the home and may be higher in winter than in summer. The measurements in ***Papers I***, ***II***, ***and III*** were only conducted during the late fall or winter, and measurements during the whole year would be needed to evaluate the effect of season in Hagfors.

Defining an exposure-response relationship requires an accurate exposure estimate. If there is a large variation in exposure from day to day (i.e., if the difference in time and activity patterns and microenvironments concentrations is large), one measurement per individual provides an uncertain estimate of the true average exposure. In ***Paper I***, the repeated personal measurements among the individuals were used for estimating the day-to-day variability (the within-individual component of variance) and the variation between individuals (the between-individual component of variance). The between-individual source of variation dominated the total variation observed in personal exposure to formaldehyde, while the opposite is generally found in occupational settings (Kromhout et al., 1993). In industry, production factors considerably influence within-individual variability, whereas, in the general population residential indoor concentration dominates for formaldehyde. The low within-individual variability in our study reflects the large percentage of time the subjects spent at home, where the day-to-day variation was low. As could be expected (Rappaport et al., 1995), the within-individual source of variation was reduced when the sampling period was extended from 24 hours to six days. Only one sample per individual would then be required to reduce the attenuation of a true exposure-response relationship to 10%.

5.5 Wood smoke markers

As discussed in section 5.4, PAH compounds can be found in emissions from many combustion sources (Bostrom et al., 2002; Srogi, 2007; WHO, 2000). Consequently, PAH emission profiles are not very specific for each source, but rather reflect efficiency in combustion and fuel quality in general (Bostrom et al., 2002). Kocbach et al. (2006) reported similar PAH profiles for vehicle exhaust samples and wood smoke emissions. However, the levels of total PAHs were found to be much higher in wood smoke emissions than in vehicle exhaust samples. Wood smoke emissions are dominated by lighter molecular weight PAHs, such as naphthalene, acenaphthene, fluorene, phenanthrene, anthracene, fluoranthene, and pyrene, (Hedberg et al., 2002; Lee et al., 2005; McDonald et al., 2000), but also BaP and benzo(e)pyrene (Khalili et al., 1995). Consistent with these studies, the indoor and outdoor air in Hagfors, as well as the air in the chamber during the wood smoke session, was dominated by these lightest molecular weight PAHs. In Hagfors, anthracene was the only 3-ring PAH found in significantly increased indoor levels in the wood-burning homes. In addition, anthracene was also correlated to the 4- to 7-ring PAHs in contrast to the other, 3-ring PAHs. Anthracene has been found in the highest concentrations in samples affected by wood smoke compared with samples taken in a tunnel, close to a coke plant, or in parking garages (Khalili et al., 1995). Retene has been proposed as a tracer for emissions from combustion of softwood (Ramdahl et al., 1984), but it was only somewhat higher in the wood-burning homes. Retene is more abundant in softwood compared with hardwood (Fine et al., 2001; McDonald et al., 2000) and in Hagfors, the households used either a mix of these two or as in some of the homes only hardwood. In Hagfors, instead, anthracene seems to be a marker for wood smoke. Also, particulate organic compounds such as levoglucosan (a degradation product of cellulose) have been used as specific tracers for wood smoke (Simoneit, 2002). Unfortunately, content of levoglucosan in the particle mass has been shown to be highly dependent on combustion conditions, making it unreliable to use as a quantitative tracer under real-world burning conditions (Hedberg et al., 2002).

As an alternative to using source-specific PAHs, source indicator ratios of selected PAHs have been used to distinguish different sources. The ratio of fluoranthene to fluoranthene plus pyrene (Flu/Flu+Pyr), and the ratio of indeno(1,2,3-cd)pyrene to indeno(1,2,3-cd)pyrene plus benzo(ghi)perylene (IcdP/IcdP+BghiP) can be used to distinguish biomass or coal from liquid fossil fuel combustion (Yunker et al., 2002). Based on previous studies, ratios of Flu/Flu+Pyr and IcdP/IcdP+BghiP of 0.4–0.5 and 0.20–0.50, respectively, are considered characteristic for liquid fossil fuel combustions, while ratios above 0.5 are more characteristic for grass, wood, and coal combustion (Yunker et al., 2002). The calculated median Flu/Flu+Pyr and IcdP/IcdP+BghiP ratios in the wood-burning homes (0.50 and 0.52, respectively) and reference homes (0.51 for both ratios), as well as outdoors (0.54 and 0.56, respectively), are in agreement with this (Figure 5.2). The ratios were similar in the chamber as in the wood-burning homes.

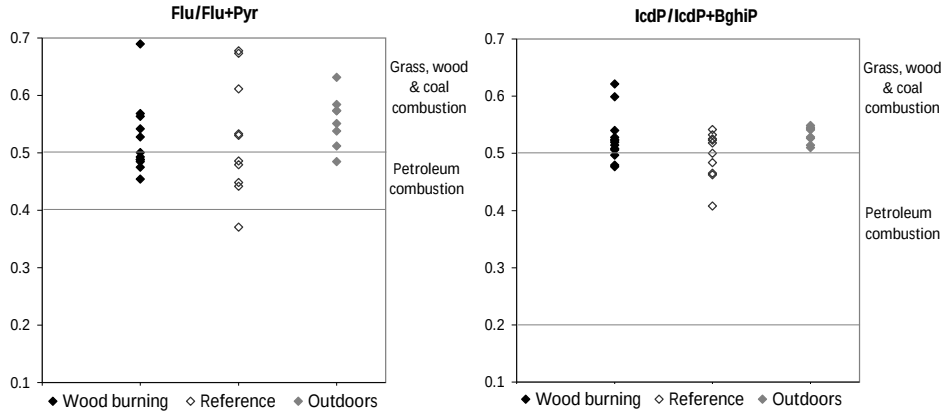


Figure 5.2. *Source-specific ratios of fluoranthene to fluoranthene plus pyrene (Flu/Flu+Pyr), and indeno(1,2,3-cd)pyrene to indeno(1,2,3-cd)pyrene plus benzo(ghi)perylene (IcdP/IcdP+BghiP) in the wood-burning homes, reference homes, and outdoors in the residential area.*

5.6 Levels and composition of the wood smoke exposure in the chamber

A constant and well-characterized exposure is required in an experimental exposure study to obtain a correct evaluation of the relationship between dose and health outcome. As discussed in section 5.3, the wood smoke emissions are strongly dependent on many factors, and a careful control of the combustion process is therefore needed. By using the same mixture of wood logs and by arranging them in a similar way when loading the wood stove, fairly constant burning conditions were achieved over each exposure session ***(Paper IV)***. The predetermined target mass concentrations of particles was relatively easy to obtain, since the PM mass concentration could be roughly determined online using the TEOM equipment, and regulated by adjusting the ratio between the wood smoke and the filtered air.

The particle numbers and size distributions, but also the morphology, of the particles in the emissions from wood log stoves have been shown to differ between different phases within the combustion cycle (Hedberg et al., 2002; Hueglin et al., 1997;

McDonald et al., 2006). However, in our study, the subjects were exposed to wood smoke from the whole burning cycle. Both the particle number and mass concentration were relatively stable over the whole session, except at the beginning of session 1, when the number of the smallest particles (particles with a mean diameter of 14 nm, stage 1) was high (Figure 4.9), which resulted in a smaller geometric diameter compared to session 2. This was probably due to a high fraction of wood smoke in the wood smoke/air mixture in the chamber prior to the exposure. As expected from earlier studies (Hedberg et al., 2002; Kocbach et al., 2005), practically all particles in the wood smoke sessions were <1 μm, and most of them (in terms of number concentration) were <150 nm in size.

The average PM mass and BS concentrations, as well as the benzene, 1,3-butadiene, formaldehyde, and acetaldehyde concentrations showed no differences between the personal and the stationary measurements. Consequently, generation of wood smoke in the chamber resulted in similar exposures for all individuals during the wood smoke exposure session, and stationary measurements in the chamber can be used to predict personal exposure. The levels of formaldehyde, acetaldehyde, 1,3-butadiene, benzene, and PAHs were much higher in the chamber ***(Paper IV)*** than inside the homes in Hagfors ***(Papers II*** and ***III)*** discussed earlier. Also the mass concentrations, about 250 $\mu g/m^3$, were much higher than the contribution of 1–10 $\mu g/m^3$ to personal and indoor $PM_{2.5}$ concentrations found in Hagfors for the subjects who used wood for space heating (Molnar et al., 2005). The knowledge about indoor PM levels in wood-burning households is relatively scarce. However, levels may be higher during shorter periods, especially when operating non-airtight stoves. The same is true when wood-burning appliances are fired in unfavorable ways, or during certain weather conditions in areas where wood burning is common, as discussed earlier. In households using biomass burning for cooking and/or indoor heating in unvented wood-burning appliances or open fires, as generally occurs in many developing countries, daily average concentrations of $PM_{2.5}$ of 100–3500 $\mu g/m^3$ have been reported (Balakrishnan et al., 2002; Balakrishnan et al., 2004; Clark et al., 2007; Cynthia et al., 2008; Naeher et al., 2007). In these countries, the exposure time is often several hours a day, especially for women, children, and elderly people (Balakrishnan et al., 2002; Balakrishnan et al., 2004). The wood smoke level (250 $\mu g/m^3$) and exposure time (4 hours) used in our experimental study ***(Paper IV)*** are therefore not unrealistic compared to those found among many people throughout the world. In addition, the PM dose received by the subjects in the chamber corresponds to an exposure of 40 $\mu g/m^3$ for 24 hours, assuming a similar rest/activity ratio and a similar breathing pattern as in the chamber. Such a PM concentration is often found in ambient air, as well as in personal exposure (Götschi et al., 2005; Qing et al., 2005).

In the wood smoke sessions of our experimental chamber study, the elements K, Zn, and Cl dominated. Kocbach et al. (2005) found that particles from wood smoke–impacted ambient air contained more K, as compared with outdoor vehicle exhaust particles, which were characterized by higher levels of Si and Ca. K, together with Zn and Ca, was also often found in higher concentrations in wood smoke particles in outdoor, indoor, and personal samples in Hagfors compared with particles in ambient air in general (Molnar et al., 2005). Also, McDonald et al. (2006) found that K, Zn, and Ca were the most abundant elements in wood smoke emission.

5.7 Effects of exposure to wood smoke in the chamber

The experimental set-up for studying the exposure to wood smoke is not well suited for quantifying the carcinogenicity. The aim of this study ***(Paper IV)*** was rather to investigate effects on markers of inflammation, coagulation, and lipid peroxidation (Barregard et al., 2008; Barregard et al., 2006). Exposure to wood smoke increased the alveolar nitric oxide, indicating inflammation at the distal part of the airways, and also the serum amyloid A, an acute-phase protein and a cardiovascular risk factor (a systemic inflammation marker). Moreover, airway inflammation was also supported by the increase of serum Clara Cell protein, probably reflected by an increase in permeability of the air-blood barrier. A slight effect on the balance of coagulation factors such as factor VIII in plasma and factor VIII/von Willebrand factor ratio were also observed. Moreover, there was some indication of increased urinary excretion of free 8-iso-Prostaglandin$_{2\alpha}$, a major F_2-isoprostane and marker of free radical–mediated lipid peroxidation. Higher levels of malondialdehyde in exhaled breath condensate also reflected increased lipid peroxidation, possible in the airways. The results found by Barregard et al. (2008; 2006) are consistent with the health effects found in epidemiological studies of PM in ambient air (WHO, 2006).

The particle characteristics responsible for the observed effects are still not known, but size mode, number and mass concentration, and chemical composition are all examples of important factors to consider (WHO, 2006). The observed effects were generally more pronounced in session 1 than in session 2. As discussed above, the PM number concentration was higher in session 1 than in session 2, while the PM mass concentrations were similar. This could possibly indicate that the number concentration was more important than mass concentration for the effects found. There is some toxicological evidence that ultra-fine particles (UFPs) have a more pronounced capacity to cause oxidative stress and proinflammatory effects than have the larger particles (Donaldson et al., 2001; Gilmour et al., 2004). However, the subjects were not exposed to PM alone, but also to gaseous components of the smoke, some of which may cause adverse health effects. Unfortunately, the semi-volatile and non-volatile PAH levels were only determined in session 2, but then the BaP level was high. However, we do not know whether there was a difference in PAH levels between wood smoke sessions, although the levels of BS were similar. The elemental composition of the particles may also be important, as for example, the 50-fold increase in zinc levels during the wood smoke session compared to clean air may be interesting in light of the associations between zinc and proinflammatory changes in rats exposed to concentrated ambient particles (CAPs) reported by Kodavanti et al. (2005) and an increase in blood fibrinogen in one of the human CAP studies (Huang et al., 2003). In comparison with the estimated cancer risk (discussed in section 5.8) attributable to wood smoke exposure, effects on the respiratory and cardiovascular system may be of greater concern for the Swedish general population.

5.8 Risk assessment and risk management of carcinogenic air pollutants

The personal exposure to formaldehyde in the general population (median 23 μg/m^3) ***(Papers I–II)*** was within the guideline value range of 12–60 μg/m^3 (0.01–0.05 ppm) recommended in Sweden (Victorin, 1998) and lower than the no observable adverse effect level (NOAEL) of 30 μg/m^3 (Koistinen, 2008). The general population is here represented by persons living in three Swedish cities with different population sizes:

Gothenburg (large), Borås (medium), and Hagfors (small). The subjects in Hagfors were not randomly selected and may not be representative of the whole city, since they were non-smokers, non-occupationally exposed, living in the same residential area, and selected based on heating system present in their home. However, wood burning was not found to increase the personal exposure or indoor levels of formaldehyde in Hagfors, and neither did smoking in Gothenburg and Borås. The upper limit of the guideline value range (60 μg/m^3) was exceeded by 3 of the 40 non-occupationally exposed subjects in Gothenburg. The risk of developing cancer in the Swedish general population is estimated to be low, based on the health-based guideline range, which is regarded to be protective against irritative effects and thereby also against cancer (Victorin, 1998). By contrast, the U.S. EPA (2008d) presents a low-risk value of 0.8 μg/m^3 (now being reassessed) which would result in about 30 extra cancer cases per year in Sweden at an average lifetime exposure of 23 μg/m^3. Thus, cancer risk assessment is associated with great uncertainty based on the uncertainty in the unit risk estimates, which was also pointed out by Loh et al. (2007). No guideline value for acetaldehyde has been recommended in Sweden, but the personal exposure levels in Hagfors are much lower than the exposure limit of 200 μg/m^3 recommended by the European Commission (Koistinen, 2008), while almost three times higher than the lifetime exposure corresponding to a cancer risk of 1/100 000 (5 μg/m^3) calculated by the U.S. EPA (2008b).

The personal exposure to 1,3-butadiene for wood burners and referents in ***Paper II*** is well below the Swedish low-risk value of 2.5 μg/m^3 (Finnberg et al., 2004). The corresponding unit risk estimate calculated by the U.S. EPA (2008a) results in a much lower low-risk value, of 0.3 μg/m^3, and is within the range of health-based guideline values (0.2–1.0 μg/m^3) discussed in Sweden when applying an uncertainty factor to the low-risk value (Finnberg et al., 2004). Only one wood burner exceeded this range. For benzene, both the wood burners and the referents had a median personal exposure higher than the low-risk guideline of 1.3 μg/m^3 used in Sweden (Victorin, 1998). The U.S. EPA (2008c) and WHO (2000) have similar risk estimates. Considering the risk estimates, and also the practical possibility of limiting the benzene exposure, the European Commission has adopted an ambient air quality limit value for benzene of 5 μg/m^3 to be met by the member countries by January 1, 2010 (Directive 2000/69/EC). As mentioned in the introduction, risk assessment based on extrapolation from high-level exposure may lead to an underestimation of the cancer risk at low-level exposure. It has been shown that the formation of protein adducts of reactive metabolites like benzene oxide and 1,4-benzoquinone is much higher per unit of exposure at low-level exposure than it is at high-level exposure (Lin et al., 2007; Rappaport et al., 2002).

When estimating the cancer risk from exposure to PAHs, the individual carcinogenicity of the different PAHs needs to be considered. However, the estimated unit risk is expressed per ng/m^3 BaP. The indoor BaP levels found in the reference homes ***(Paper III)*** were similar to the health-based guideline value of 0.1 ng/m^3 used in Sweden, while the median indoor BaP level in the wood-burning homes was five times higher. The outdoor concentration measured during this winter period exceeded the guidance value on all days, but summer concentrations are expected to be lower, due to the faster chemical reactions of PAHs and to smaller emissions at that time of year. However, the median outdoor BaP concentration (0.37 ng/m^3) was below the ambient air quality target value of 1 ng/m^3, as an annual average, which has been

adopted by the European Commission, and which has to be met by January 1, 2012, (Directive 2004/107/EG), but above the Swedish interim target level of 0.3 ng/m^3. Consistent with the use of BaP as an indicator of the carcinogenicity of the PAH mixture, the total cancer potency in Hagfors was dominated by BaP, with a contribution of about 60% in the wood-burning homes, the reference homes, and outdoors. The more volatile and abundant fluoranthene was the second largest contributor to the total cancer potency after BaP and has been suggested as a supplementary indicator of carcinogenicity, with a guideline value of 2 ng/m^3 (Bostrom et al., 2002). This value was exceeded outdoors in the residential area (median 2.8 ng/m^3), but not in most of the wood-burning homes (median 1.5 ng/m^3).

Based on the carcinogenic potential of these wood smoke constituents discussed above, an additional contribution from the wood smoke to the personal exposure would increase the cancer risk for individuals living in homes with wood-burning appliances. The point estimates of the difference in mean personal exposure or indoor levels of 1,3-butadiene, benzene, and BaP between the wood-burning group and the reference group were used together with the established exposure-response estimates usually used in Sweden, for estimating the extra cancer risk that might be caused by exposure to wood smoke. The mean exposure to wood smoke (1,3-butadiene, benzene, BaP) would contribute to about six extra cancer cases per 100,000 individuals exposed during a lifetime. BaP (or exposure to PAHs) made up for the largest fraction of this risk. The total cancer risk is probably somewhat overestimated, since the concentrations of these pollutants are most likely to be lower in summer, when the need for heating is less. The indoor concentrations of PAHs might not reflect the true personal exposure, since people also spend time in other microenvironments with lower or higher concentrations, and the measured levels do not capture exposure experienced from different activities. The manner of adding cancer risks of individual carcinogens is also associated with a large uncertainty, since the effect can be synergistic or antagonistic. In addition, wood smoke contains several other pollutants that can be important when estimating the cancer risk. As pointed out in the introduction, exposure to ambient air pollution has been associated with an increased lung cancer risk. These studies indicate that the cancer risk associated with exposure to wood smoke as measured by exposure to PM (assuming that wood smoke is at least as harmful as the ambient air in cities) might result in a considerably higher cancer risk, but substantial uncertainty still remains regarding the effect of ambient air pollution on the risk for lung cancer (Pope et al., 2002; Pope and Dockery, 2006). The cancer risk associated with domestic wood burning is probably significantly higher in developing countries, where a larger number of people are exposed, often at much higher levels.

Since the general population in Sweden spends the majority of their time indoors at home, measures should be taken to reduce wood smoke emissions, which may enter the building from outside or leak directly to the indoor environment from the boiler or stove. For risk management, supporting the substitution of old combustion appliances with modern low-emission appliances, providing training in the operation of wood-burning appliances, and supporting development of low-emission combustion appliances and filter technologies would be efficient measures to lower the emissions, in combination with applying emission limits and testing standards for the appliances (Boman et al., 2008).

5.9 Future needs

Today, the majority of the Swedish general population has a personal exposure to formaldehyde within the recommended guideline value range (Johannesson et al., 2008). However, continuing screening studies are needed to follow future trends and to discover potential new sources. Since the year 2000 this has been accomplished within the environmental monitoring program coordinated by the Swedish EPA. This program includes measurements of personal exposure by use of diffusive samplers. However, ***Paper I*** has shown that fixed measurements in bedrooms are nearly as good as personal measurements to assess personal exposure to formaldehyde. An appropriate technique for including a larger number of subjects would be to use self-administered diffusive samplers (Lindahl et al., 1999).

Few studies have investigated the impact of domestic wood burning on personal exposure to, or indoor levels of, pollutants in the developed world. To verify the impact found in ***Papers II*** and ***III*****,** more studies are needed. Personal exposure and indoor concentrations measured in the wood-burning homes in ***Papers II*** and ***III*** showed a large variation in exposure. This can be expected, since these homes reflect the reality in which many factors related to the wood-burning appliance and individual wood-burning management may differ. This thesis suggests some important factors influencing the air pollution levels that need to be investigated further (e.g., wood boiler or fireplace, number of loadings). Larger studies including a greater number of homes and also repeated measurements would be valuable in order to better assess the influence of wood-burning factors, both for epidemiological studies and for reducing the emissions. Most of the wood-burning appliances in ***Papers II*** and ***III*** are relatively old, and only a couple of them are environmentally certified. New boilers have much lower emissions, and therefore studies including newer wood-burning appliances are needed. The use of pellets has increased during the past years, and in ***Papers II*** and ***III*****,** homes with pellet boilers were not included. Their impact on the personal exposure and indoor air is not known today but is expected to be lower due to lower emissions.

The human experimental set-up for studying effects of wood smoke developed and executed in ***Paper IV*** is the first published study of this kind. However, more experimental studies are needed, and several are at the moment being carried out in Sweden (by our research group), Denmark, and the United States. By varying the combustion with respect to stove, fuel, firing behavior, and dilution ratio of the wood smoke, different exposure scenarios can be achieved, and thus, knowledge can be obtained about which properties of particles and gaseous compounds are crucial for the effects observed. In addition to airways and systemic inflammation, it would be interesting to examine effects of wood smoke on endothelial function (Bräuner et al., 2008; Mills et al., 2005). An advantage of the set-up described in ***Paper IV*** is that a relatively large number of subjects can be exposed at the same time, with similar exposure conditions. This makes the studies less costly and time-consuming.

6 Conclusions

- Indoor concentrations of formaldehyde accurately reflect personal exposure levels, whereas fixed outdoor sampling cannot be used for personal exposure assessment. The within-individual source of variability in personal exposure was low. Living in single-family houses contributed to higher personal exposure compared to living in apartments. A minor part of the general population is exposed to airborne concentrations of formaldehyde at levels associated with sensory irritation.

- Domestic wood burning increased indoor levels of 1,3-butadiene, benzene, and several PAHs, such as BaP. The 1,3-butadiene levels measured personally, as well as indoors and outdoors, were at or below the Swedish low-risk level for cancer. By contrast, BaP levels in the wood-burning homes were five times higher than the Swedish health-based guideline, which was also exceeded outdoors, and comparable to levels in urban areas with heavy traffic. No clear effect of wood burning on the formaldehyde and acetaldehyde levels was found.

  Based on the unit risk estimates, the cancer risk from the contribution of domestic wood burning to exposure to 1,3-butadiene, benzene, and BaP was about six times higher than 1×10^{-5}, and this is higher than what is considered as "acceptable" in Sweden.

- An experimental set-up using wood smoke exposure in humans was developed. It may be used as a model for human exposure to gaseous pollutants and PM. With careful control of the combustion process, relatively constant mass and number concentrations were obtained over each exposure session. By varying the combustion with respect to stove, fuel, firing behavior, and dilution ratio of the wood smoke, different exposure scenarios can be achieved, and thus, knowledge can be obtained about which properties of particles and gaseous compounds are crucial for the effects observed. Exposure levels of 1,3-butadiene, benzene, formaldehyde, acetaldehyde, PAHs, and PM were, as expected, clearly higher (by 5–50 times) during the wood smoke session compared with the clean air session.

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