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ORIGINAL ARTICLE

C. Viau · G. Hakizimana · M. Bouchard

Indoor exposure to polycyclic aromatic hydrocarbons and carbon monoxide in traditional houses in Burundi

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Abstract ***Objectives:*** Wood combustion is used as a major energy source in African countries and could result in indoor, pollution-related health problems. This exploratory study was undertaken to estimate polycyclic aromatic hydrocarbon (PAH) and carbon monoxide exposure in individuals living in traditional rural houses in Burundi. ***Methods:*** Standard methods were used to determine indoor air concentrations of 12 PAHs, and carbon monoxide. The urinary excretion of 1-hydroxypyrene (1-OHP) was measured in occupants of traditional houses, and compared with that of individuals living in the town of Bujumbura, the capital of Burundi. ***Results:*** Mean airborne concentration of four volatile PAHs, naphthalene, fluorene, phenanthrene and acenaphthene, exceeded 1 $\mu g/m^3$, and that of benzo(a)pyrene was 0.07 $\mu g/m^3$. Naphthalene was by far the main PAH contaminant, with a mean concentration (± standard deviation) of 28.7 ± 23.4 $\mu g/m^3$, representing on average 60–70% of total PAH concentration. Carbon monoxide mean concentration (± standard deviation) was 42 ± 31 mg/m^3, and correlated with total PAH concentration. Geometric mean urinary 1-OHP excretion (range) in people living in traditional houses was 1.50 (0.26–15.62) μmol/mol creatinine, a value which is on average 30 times higher than that of people living in the capital (0.05 (0.009–0.17) μmol/mol creatinine). ***Conclusions:*** It appears that the substantially high concentrations of the studied contaminants constitute a potential health hazard to the rural population of Burundi.

Key words Polycyclic aromatic hydrocarbons · Indoor air pollution · Environmental exposure · Biological monitoring

C. Viau (✉)
Département de médecine du travail et d'hygiène du milieu, Université de Montréal, P.O. Box 6128, Station Centre-ville, Montréal, Québec, H3C 3J7, Canada
e-mail: claude.viau@umontreal.ca
Fax +1-514-3432200

G. Hakizimana · M. Bouchard
Groupe de recherche en toxicologie humaine and Département de médecine du travail et d'hygiène du milieu, Faculté de médecine, Université de Montréal, Montréal, Québec, H3C 3J7, Canada

Introduction

Wood burning in fireplaces and wood stoves has been recognized as an important source of airborne polycyclic aromatic hydrocarbons (PAHs) (Knight and Humphreys 1985; Lioy et al. 1988; Lioy and Greenberg 1990; Wilson and Chuang 1991). These environmental contaminants have been classified as priority pollutants owing to the carcinogenic potential of several individual PAH compounds and PAH mixtures (IARC 1984, 1985, 1987). In developing countries, since combustion of wood and other organic fuels is used indoors, for cooking, lighting and heating purposes in rural communities, the inhabitants are prone to be exposed to large doses of PAHs. Few studies have, however, been conducted on this type of exposure. In particular, women, who are responsible for most household chores, are probably more exposed than men to the smoke emitted from both fuel burning and cooking itself (He et al. 1991; Liu et al. 1993; Mumford et al. 1993). Raiyani et al. (1993) estimated that the daily benzo(a)pyrene (BaP) dose (914 ng) for the urban poor as a result of a 3-h exposure to cooking fuel smoke emitted from wood burning, was comparable to that of a heavy smoker consuming over 2 packets of cigarettes per day. Furthermore, these authors reported that wood burning produced indoor air concentrations of BaP that were significantly greater than those resulting from the use of coal or kerosene. Ventilation conditions can also strongly influence indoor PAH concentrations (Liu et al. 1993).

To evaluate exposure to airborne PAHs indoors, air sampling has most commonly been used (Chuang et al.

1990; Risner and Conner 1991; Shuguang et al. 1994). This method allows the estimation of respiratory uptake only, and does not take into account other exposure routes. On the other hand, an increasingly popular approach for the evaluation of exposure to PAHs in both occupational and general environments is the use of urinary 1-hydroxypyrene (1-OHP) as a biological indicator of overall PAH exposure (Jongeneelen et al. 1988; Jongeneelen 1994; Zhao et al. 1992a). Since exposure to PAHs can occur through inhalation of ambient air or tobacco smoke, through ingestion of food, in particular smoked, fried or charcoal broiled foodstuffs, or through skin contact, a measurement of the absorbed dose allows us to take into consideration exposure by the different routes (Bowman et al. 1997; Lauwerys and Hoet 1993; Sithisarankul et al. 1997; Van Rooij et al. 1994; Zhao et al. 1995). Background median urinary 1-OHP concentrations from people throughout the world have been found to range from 0.03 to 0.76 μmol/mol creatinine, with the highest values recorded in China (Levin 1995), probably due to the extensive use of coal for both cooking and heating in that country (Zhao et al. 1992a).

Furthermore, since carbon monoxide is probably the most important single contaminant emitted during combustion of wood (Gad and Anderson 1990), determination of its concentration is also of primary importance. In industrialised countries, concentration limits have been set at 10 mg/m^3 (9 ppm) for outdoor ambient air, while occupational exposure limits lie around 29–55 mg/m^3 (25–50 ppm) (ACGIH 1996; Calabrese and Kenyon 1991; OSHA 1994). Carbon monoxide binds to haem-containing proteins such as haemoglobin, and reduces the oxygen transport capacity of the blood, leading to tissue hypoxia (Jaffe 1997). It also interferes with the dissociation of oxyhaemoglobin and removal of carbon dioxide, and causes neurological, cardiac, respiratory, vascular and foetal effects (Jaffe 1997; Hardy and Thom 1994; Norman and Halton 1990; Wattel et al. 1996). Death may occur at exposure levels above 1,375 mg/m^3 (1,200 ppm) (NIOSH 1994).

This exploratory study was therefore undertaken to estimate PAH and carbon monoxide exposure in individuals living in traditional rural houses in Burundi, by the measurements of the concentrations of PAHs and carbon monoxide in the indoor air and of 1-OHP in urine.

Materials and methods

Traditional rural and urban house characteristics

Traditional rural houses in Burundi, as in many other African countries, are constructed directly on the ground, and can be rectangular or circular in shape. Walls are made of bamboo or eucalyptus branches consolidated with mud. The roof, supported by two pillars, is thatched or covered with banana shingles. The inside of the house usually consists of a reception room with an entrance (the door usually stays open during the day and is closed at nightfall), a bedroom and a living/dining room with sometimes a small opening of approximately 1,000 cm^2. The fireplace is located in the centre of the latter and consists of a 30–40 cm diameter hole dug in the ground and surrounded by three stones to support cooking ware. Approximately 1.6 m above this fireplace is suspended a rack, which serves for the preservation of various domestic products. Eucalyptus wood burning constitutes the main source of energy. Both smoke and gases emitted during combustion simply diffuse throughout the house. This setup is used for almost all cooking operations, especially during the wet season, and serves also as a heating and lighting source during the night. The inside walls are often covered with soot and there is a typically permanent smoke odour indoors.

In contrast, urban houses are modern, with electricity being used as a lighting source, and cooking performed outside on portable metal coal-burning stoves.

Subjects

The field study part of this investigation was conducted between June and August 1993. Subjects were recruited from the rural districts of Burundi: Mugamba and Rwibaga, which are located respectively 75 and 40 km from the capital, Bujumbura. It was explained to the people in Kirundi, the Burundi language, the purpose of the study and the types of measurements and collaboration required from them. For obvious reasons, in these remote African locations no written consent could be obtained, but the subjects were free to withdraw from the study at any time. A total of 32 individuals (15 smokers, 17 non-smokers) from 16 houses agreed to provide urine samples. The ages of the participants varied between 16 and 60 years, with a large proportion in the 25–30 year range, but subjects were not always able to give their exact age.

In order to obtain reference data from Burundese people who were not exposed to wood smoke in traditional houses or during cooking, we asked 18 subjects working for the Ministry of Health (mainly laboratory employees) and living in Bujumbura, to provide urine samples. These participants were aged between 22 and 28 years.

Unfortunately, in the African context, all the information usually required for state of the art epidemiological studies could not be collected (such as questionnaire-acquired data on age, gender, smoking habits and PAH dietary intake of each individual, type of work of the exposed subjects, time spent indoors and time exposed to wood smoke during the course of the day). It should however be noted that, in most of the published studies, it has been reported that age and gender did not influence the creatinine-adjusted urinary excretion of 1-OHP in the general population (Ovrebo et al. 1995; Roggi et al. 1997; Van Wijnen et al. 1996; Zhao et al. 1992a).

Air sampling and analysis

Air samples were collected by the use of a sampling train, composed of a polystyrene glass-fibre filter to collect particulate matter (37 mm diameter, 0.3 μm porosity, Millipore, Missisauga, Ontario, Canada) and a styrene-divinylbenzene adsorption tube to capture volatile PAHs (ORBO-43, 100 + 50 mg, Supelco, Oakville, Ontario, Canada). Air was sampled for periods of 8 to 12 h using a Gilair constant flow personal pump (Gilian, distributed by Levitt Safety, Oakville, Ontario, Canada) at a rate of 1.5 l/min as calibrated with an electronic calibrator (SKC, model 712, distributed by Durpro, Brossard, Quebec, Canada). The sampling device was placed either in the bedroom on the bed (0.8 m above ground level), or in the living/dining room suspended from the rack, approximately 1.5 m away from the fire and 1.3 m above ground level (corresponding to the breathing zone of a seated individual). Air from 16 houses in total was sampled. In the first 8 houses, sampling took place between 8 a.m. and 6 p.m., and between 6 p.m. and 8 a.m. in the living/dining room. In the next 8 houses, samples were taken simultaneously in the living/dining room and in the bedroom between 8 a.m. and 6 p.m. The mean sampling temperature was approximately 22 °C. It should be noted that cooking times were usually at noon and at nightfall (around 7 p.m.). For practical

reasons, sampling was performed during the dry season, when people spent most of their days outdoors, although at nightfall family life usually took place around the fire, which burned all night since it was the only lighting and heating source. For comparison, 8 outdoor air samples were also collected between 8 a.m. and 6 p.m., with the same personal sampling devices placed 20 m away from the house at a height of 1.6 m. On two occasions, the pump stopped running after less than 8 h, and the samples were discarded from data analysis. Immediately after collection, both filter cassettes and tubes were sealed with the caps supplied. They were transported to Bujumbura within 12 h, wrapped in Parafilm and kept at −20 °C until their final transfer to our laboratory at the University of Montreal. Filters were extracted with 5 ml of acetonitrile and each section of each tube was desorbed with the same volume of this solvent according to NIOSH standard procedure (NIOSH 1985). The following PAHs were quantified by high performance liquid chromatography (HPLC) with fluorescence detection: naphthalene, acenaphthene, fluorene, phenanthrene, anthracene, fluoranthene, pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, BaP, dibenzo(a,h)anthracene and benzo(g,h,i)perylene. The sum of the concentrations of these individual compounds will be referred to as "total PAHs (ΣPAH) concentration". Standards were obtained from Supelco. The HPLC system consisted of a model AS-100 HPLC automatic sampling system (Bio-Rad, Richmond, Calif.), a model 250 binary pump (Perkin-Elmer, Buckingham, England), a 250 × 4.6 mm Supelcosil LC PAH column (Supelco, Oakville, Ontario, Canada) and a Perkin-Elmer LS-40 fluorescence detector. Excitation and emission wavelengths were programmed and optimised for the detection and quantification of each PAH. The detector signal was recorded and treated with PE Nelson software. Injection volume was 20 µl throughout. Elution was accomplished with a gradient of water and acetonitrile based on the recommendations of the manufacturer of the HPLC column. Carbon monoxide was measured by passive dosimetry (distributed by Levitt Safety, Oakville, Ontario, Canada) by clipping the tube to the PAH sampling system over the same period as that used for PAHs air sampling.

Urine sampling and analysis

Urine samples were always collected at the end of an air sampling period, from two subjects in each sampled house. Immediately after micturition, urine samples, which were collected over thymol, were placed in a cooler with ice packs and subsequently transported within 12 h to Bujumbura, where they were kept at 4 °C. Tests conducted in our laboratory have shown that 1-OHP under these conditions, is very stable (unpublished). We avoided problems associated with the transport of biological samples from Burundi to Canada by subjecting the urine samples in Bujumbura to enzymatic hydrolysis and cleanup on C18 Sep-Pak cartridges (Millipore) as previously described (Jongeneelen et al. 1987). The C18 cartridges were then wrapped in Parafilm and kept at −20 °C until they were brought to our laboratory in Montreal. It had previously been established in the laboratory that preservation of 1-OHP adsorbed on the C18 cartridges, at temperatures varying between room temperature and −20 °C, caused no loss of the analyte, for a period of several weeks (unpublished). In Montreal, desorption and analysis of 1-OHP was conducted according to Bouchard et al. (1994) by the use of the same HPLC system described above, except that the LC PAH column was replaced with a Supelcosil LC18 column. Urine samples were adjusted for creatinine content. Samples with a creatinine concentration below 0.3 g/l (0.0027 mol/l) were not included in the analysis (Lauwerys and Hoet 1993).

Statistical analysis

Simple linear regression analysis was used to study the relationship between various indicators. Variations in PAH concentrations between daytime and night-time and between the living area and the bedroom for each house were compared by paired *t*-test analysis. Comparison of 1-OHP excretion between the rural group, the Bujumbura group and a reference group in Montreal was performed by one-way analysis of variance carried out on log-transformed data, since 1-OHP concentrations were log-normally distributed. Comparison of 1-OHP urinary concentrations between smokers and non-smokers was made by unpaired *t*-test analysis on log-transformed values.

Results

Outdoor air sampling with the low volume samplers used in this study revealed no measurable concentrations of PAHs. Figure 1 shows the distribution of PAH measurements in all the indoor air samples collected. The mean concentration (± standard deviation) of ΣPAH was 43 ± 34 µg/m^3. Naphthalene was by far the main PAH contaminant with a mean concentration (± standard deviation) of 28.7 ± 23.4 µg/m^3, representing on average 60–70% of ΣPAH, followed by fluorene, phenanthrene and acenaphthene at 8.6 ± 8.0, 3.4 ± 4.4, and 1.0 ± 1.3 µg/m^3. All other PAHs were present in mean concentrations below 1 µg/m^3. Excluding naphthalene from the calculations of ΣPAH gave a mean concentration (± standard deviation) of 14.6 ± 12.3 µg/m^3. In searching for a specific PAH as an indicator of ΣPAH, we examined the correlation between individual PAHs and ΣPAH. As seen in Table 1, a significant correlation was observed for naphthalene, fluorene, anthracene, fluoranthene, acenaphthene, pyrene and phenanthrene. No significant correlation was found for the other PAHs. Excluding naphthalene from ΣPAH and looking at the same correlations did not significantly modify the results (Table 1). Even naphthalene appeared to be very well correlated with [ΣPAH – naphthalene]. It was also observed that BaP was significantly correlated with pyrene, benzo(b)fluoranthene and benzo(k)fluoranthene only. Two- and three-ring PAHs were present mainly (>90%) in the gaseous phase while five- and six-ring PAHs appeared mostly (>85%, except for benzo(b)fluoranthene at 65%) in the solid phase, retained on the filter. The four-ring PAHs, fluoranthene and pyrene, appeared almost equally in both phases with respectively 47% and 57% present in the solid phase.

The semi-quantitative evaluation of carbon monoxide with the passive samplers yielded a mean concentration (± standard deviation) of 42 ± 31 mg/m^3. Since the source of both PAHs and carbon monoxide was the same, correlation between the concentration of the latter and ΣPAH was examined and found to be highly significant ($r = 0.70$, $P < 0.001$, Fig. 2). When the relationship between carbon monoxide and [ΣPAH-naphthalene] was examined, similar results were obtained ($r = 0.61$, $P < 0.001$).

The concentrations of both ΣPAH and carbon monoxide were not significantly greater during the day than during the night (paired *t*-test, $P > 0.05$) and there was little difference in the concentrations of the

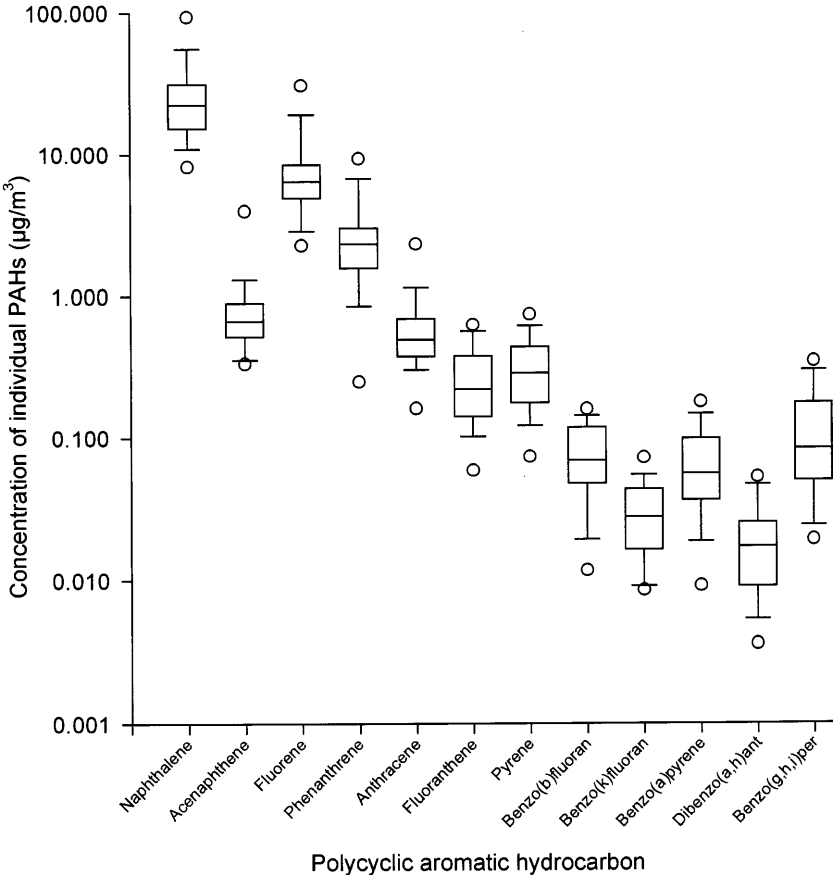

Fig. 1 Box and whisker plot showing the distribution of the concentrations of the 12 polycyclic aromatic hydrocarbon (PAHs) determined in the present study. The *points* outside the box represent the 5th and 95th percentile of the distribution. The *horizontal lines* represent respectively, from top to bottom, the 90th, 75th, 50th, 25th and 10th percentile

contaminants between the living room and the bedroom (paired *t*-test, $P > 0.05$).

Fourteen urine samples out of the 32 collected from people living in traditional houses had to be rejected on the basis of a low creatinine concentration compared to 3 rejected out of the 18 Bujumbura samples. Figure 3 shows the distribution of 1-OHP excretion in people from the rural districts, from Bujumbura, and in a reference group of our laboratory in Montreal. The respective geometric mean concentrations (range) for these three groups were 1.50 (0.26–15.62), 0.05 (0.009–0.17) and 0.08 (0.002–0.57) µmol/mol creatinine, revealing a highly significant difference between people living in traditional houses in Burundi and the other two groups ($P < 0.001$). Urinary concentrations of 1-OHP in smoking and non-smoking subjects living in traditional houses were not significantly different ($P = 0.60$). No significant correlation was found between urinary 1-OHP and either air concentrations of ΣPAH or pyrene itself.

Table 1 Linear correlation coefficients for various relationships among polycyclic aromatic hydrocarbon (PAH) airborne concentrations (*BaP* benzo(a)pyrene)

PAH	vs ΣPAH	vs ΣPAH without naphthalene	vs BaP
Naphthalene	0.982***	0.846***	0.042
Acenaphthene	0.652***	0.804***	0.220
Fluorene	0.969***	0.937***	0.075
Phenanthrene	0.464**	0.654***	0.019
Anthracene	0.850***	0.898***	0.149
Fluoranthene	0.644***	0.721***	0.341
Pyrene	0.529**	0.601***	0.488**
Benzo(b)fluoranthene	0.127	0.055	0.409*
Benzo(k)fluoranthene	0.113	0.199	0.678***
Benzo(a)pyrene	0.063	0.097	–
Dibenzo(a,h)anthracene	0.092	0.022	0.070
Benzo(g,h,i)perylene	0.262	0.312	0.204

*$P < 0.05$, **$P < 0.01$, ***$P < 0.001$

Discussion

The quantitative determination of PAHs in the indoor air of traditional houses in developing countries has received relatively little attention, yet the observed concentrations can be quite substantial. In the current study, the mean concentration of four volatile PAHs, naphthalene, fluorene, phenanthrene and acenaphthene, exceeded 1 µg/m^3. These concentrations can hardly be compared with those of similar studies conducted in India (Raiyani et al. 1993) or in China (Mumford et al. 1990) where particulate PAHs only, emitted during cooking, were measured. Indeed, Mumford et al. (1990) determined the concentration of a number of PAHs in a fraction of airborne particles of 10 µm (PM_{10}) while

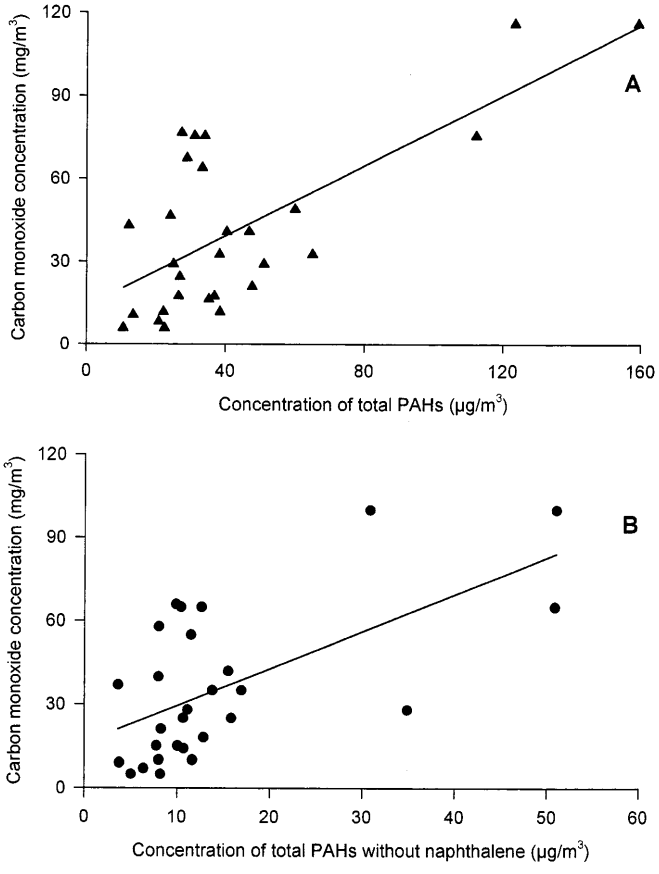

Fig. 2 Correlations between the concentrations of carbon monoxide and the sum of all polycyclic aromatic hydrocarbon (PAHs) (panel A) and between concentrations of carbon monoxide and the sum of all PAHs except naphthalene (panel B)

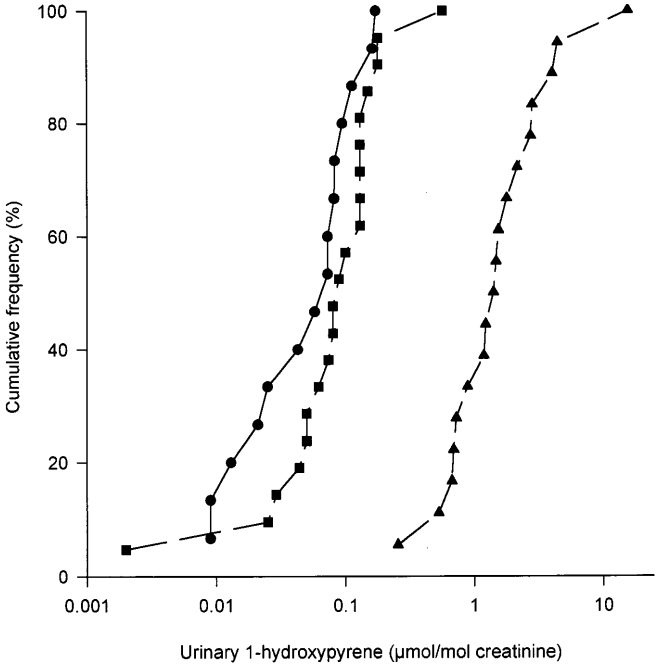

Fig. 3 Cumulative frequency distribution of the urinary excretion of 1-hydroxypyrene in people living in two rural districts of Burundi (▲), in Bujumbura (●) and in a reference group at the University of Montreal (■)

Raiyani et al. (1993) simply collected total airborne particulate matter on a glass-fibre filter. For higher molecular weight PAHs such as BaP, the determination of the concentration either in the particulate phase or in both the gaseous and particulate phase should not affect the result, as this compound is exclusively present in the solid phase (mostly adsorbed to airborne particulate matter). However, many three- and four-ring PAHs can exist both in the gaseous and solid phase (Atkinson and Arey 1994). Indeed, Lesage et al. (1987) have shown that at a sampling temperature of 18 °C, pyrene and fluoranthene, for example, will be present exclusively in the solid phase while at 28 °C, pyrene is mostly recovered in the gaseous phase and fluoranthene is found in approximately equal proportions in both phases. Similar observations were made for other PAHs. Our results from samples collected at a mean temperature of 22 °C are consistent with these observations. Consequently, for a proper quantitative determination of some PAHs, a sampling train that retains both forms is required. Furthermore, Raiyani et al. (1993) used benzene as an extraction solvent to recover PAHs from sampled particulate matter, although this solvent has been shown to be somewhat rather inefficient in desorbing PAHs from the surface of carbonaceous particulate matter (Lesage et al. 1987). The comparison of the results of Raiyani et al. (1993) with those obtained in the current study revealed large discrepancies in the concentrations of the two- to four-ring PAHs measured, and closer values for the heavier PAHs, that is certainly largely due to differences in sampling procedures. For example, a mean (range) phenanthrene concentration of 3.4 (0.2–23.8) $\mu g/m^3$ was obtained in this study, with 93% in the gaseous phase, while Raiyani et al. (1993) reported a value of 0.02 (0.005–0.09) $\mu g/m^3$. On the other hand, mean (range) concentration of BaP was 0.07 (0.01–0.27) $\mu g/m^3$, compared with 0.4 (0.07–0.8) $\mu g/m^3$ in the study of Raiyani et al. (1993). It should further be pointed out that these last authors measured PAH concentrations over only 1 h, during cooking periods, whereas our measurements were performed over 10 to 14 h, covering the whole day or night periods, thereby causing a certain degree of "dilution" of the samples with respect to peak concentrations.

The PAH concentrations measured in this report should be considered as high. Indeed, the range of BaP airborne concentrations obtained in this study (0.01–0.27 $\mu g/m^3$) exceeded that reported for cities near aluminium smelters in Canada (0.003–0.049 $\mu g/m^3$) (Meek et al. 1994). These values also largely exceeded the maximum permissible risk level of 0.001 $\mu g/m^3$ BaP in the ambient air, proposed by Slooff et al. (1989) based on the carcinogenic potential of inhaled particulate PAHs. Naphthalene, the main volatile PAH, was also present in large concentrations. Mean value exceeded the reference concentration (RfC) of 3 $\mu g/m^3$ in the

ambient air, proposed by the U.S. Environmental Protection Agency (U.S. EPA) to prevent respiratory tract, ocular, and haemolytic effects of naphthalene, as well as renal, liver and neurological effects which can occur as a consequence of jaundice resulting from naphthalene-induced haemolysis (U.S. Department of Health and Human Services 1995; U.S. EPA 1998).

Carbon monoxide was also present in concentrations sufficiently high to pose a health threat to the rural population. Indeed, the semi-quantitative measurements made in the current study revealed mean 10- (day) or 14- (night) h concentrations reaching 115 mg/m^3, a value greatly above the U.S. National Ambient Air Quality Standard of 10 mg/m^3 or 9 ppm (8-h average) (Calabrese and Kenyon 1991). The organs probably most susceptible to carbon monoxide-induced hypoxia at these concentrations are the central nervous system and the heart (Osterloh and Tarcher 1992; Urbanetti 1981). Clinical manifestations of carbon monoxide poisoning include neurological disturbance, cardiac arrhythmia, respiratory and circulatory failure, all of which are usually reversible. However, long-term neurological manifestations have been observed and can result in function impairment and disability (Hardy and Thom 1994; Wattel et al. 1996). Also, the developing foetus has been identified as being particularly vulnerable to low levels of carbon monoxide (Osterloh and Tarcher 1992), that is probably of primary importance in the African context where women are the main group exposed to wood smoke due to their responsibility for most household chores.

It is of interest that all two- to four-ring PAHs that were measured in this study were individually correlated with ΣPAH as well as with [ΣPAH-naphthalene]. The latter parameter was introduced because naphthalene represented such a large proportion of ΣPAH that it could by itself have explained the largest portion of the correlations without any strong relationship to other PAHs present. These correlations therefore indicate that any of the lower molecular weight PAHs can serve as an indicator of the overall PAH exposure. The correlation studies, however, indicated that pyrene is the only lower molecular weight PAH that is correlated with BaP. When studying the correlation between the airborne concentrations of the lower molecular weight PAHs and the sum of the concentrations of six carcinogenic PAHs, Hansen et al. (1991) found that naphthalene, fluorene, phenanthrene, anthracene, fluoranthene, and pyrene were individually well correlated with the sum of benzo(a)anthracene, benzo(b)fluoranthene, benzo(k)fluoranthene, BaP, dibenzo(a,h)anthracene, and indeno(1,2,3-c,d)pyrene present in the air sampled in smokehouses. On the other hand, the current study revealed significant correlations between carbon monoxide concentrations and both ΣPAH and [ΣPAH-naphthalene] concentrations. This suggests that for a given constant combustible source, it is possible to define this relationship, and subsequently to use a simple, low-cost passive monitoring technique to estimate the exposure to ΣPAH, that might be a particularly interesting approach in developing countries.

This study also suggests that indoor air concentrations of PAHs and carbon monoxide in traditional houses in Burundi appear relatively constant throughout the diurnal cycle and throughout the house, implying that subjects spending most of their time indoors will potentially be exposed to high doses of both PAHs and carbon monoxide. This might be especially critical during the wet season, for women, aged people and young babies.

Concerning biological monitoring results, we found that examination of the distribution of urinary 1-OHP concentrations in people living in traditional houses and in the city of Bujumbura revealed a spectacular difference between the two groups, the former displaying much higher excretion values. In fact, it was impressive to note that the geometric mean (range) excretion values observed in the rural districts (1.50 (0.26–15.62) μmol/mol creatinine) were comparable to values previously published in workers occupationally exposed to creosote in a wood treatment plant (1.63 (0.18–10.47) μmol/mol creatinine) (Viau et al. 1993). It is also noteworthy that the distribution of values observed in the Bujumbura group and in a reference group taken from our laboratory in Montreal were quite similar. Furthermore, as mentioned previously, 14 urine samples out of 32 (44%) from people living in traditional houses had to be discarded because of low creatinine concentrations. This fraction appears rather significant but seems to be in accordance with the observation that these subjects consumed large amounts of liquid during the day.

In the current study, a lack of correlation was observed between the atmospheric concentrations of PAHs (including pyrene alone) in traditional houses, and the urinary excretion of 1-OHP in people living in such houses. These results are in contrast with those of Zhao et al. (1990, 1992b) who compared outdoor PAH air concentrations and 1-OHP excretion in people in China. This can perhaps first be explained by the fact that the time spent indoors can be highly variable between subjects, with the consequence that indoor air concentration of PAHs may not adequately reflect actual inhalation exposure. Additionally, dietary intake (Van Rooij et al. 1994) and skin uptake (Van Rooij et al. 1993; Viau and Vyskocil 1995) can also contribute to the absorbed dose as estimated by 1-OHP urinary excretion. Indeed, it was observed that the inner walls of traditional houses were often covered with soot, making skin contact with PAHs highly probable.

In conclusion, we found that people living in traditional houses in Burundi are exposed to high doses of both PAHs and carbon monoxide from indoor air pollution generated by unventilated burning of wood. These observations indicated that it would be appropriate to conduct a large scale epidemiological study to assess the potentially deleterious health effects (pulmonary cancer, heart disease, central nervous system disease) related to

such exposures. Simple measures should be taken at least to vent the smoke adequately to the outside of the houses, while the use of cleaner energy sources is awaited.

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