Urinary levels of 1-hydroxypyrene in children residing near a coal-fired power plant

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Abstract

Background: The effects of emissions from coal-fired power plants on children's exposure to polycyclic aromatic hydrocarbons (PAHs) are not well understood.

Objectives: This study aimed to evaluate the sources and the urinary levels of 1-hydroxypyrene (1-OHP), a biomarker of exposure to PAHs, among children living in proximity to a coal-fired power plant.

Methods: Study areas consisted of two high exposure (HE1 and HE2) and two low exposure (LE1 and LE2) communities, at different distances and directions from a large coal-fired power plant in central Taiwan. Study subjects included 369 children aged 1-13 years and randomly selected from each community. Each child's urinary 1-OHP concentration was measured by a high-performance liquid chromatography-fluorescence detector method. Samples of ambient air were analyzed for PAHs using a gas chromatography-mass spectrometry method. Information on important factors was collected by an interview using a structured questionnaire. Multiple regression analysis was used to assess factors significantly associated with urinary 1-OHP levels.

Results: Levels of PAHs in ambient air in HE communities were significantly higher than in LE communities. Children living in HE1 and HE2 had significantly higher mean urinary 1-OHP concentrations than those living in LE1 and LE2 (0.186 and 0.194 v.s. 0.113 and 0.122 μmol/mol-creatinine). The difference in urinary 1-OHP levels between HE1 and HE2, respectively, and LE1 remained significant after adjusting for age, gender, environmental tobacco smoke, dietary exposure, and traffic.

Conclusions: Children living in communities downwind of and in proximity to the coal-fired power plant had significantly increased urinary 1-OHP levels.

Keywords: 1-hydroxypyrene; children; power plant; polycyclic aromatic hydrocarbons; biomarker

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The sponsor was not involved in the study design; in the collection, analysis and interpretation of data; in the writing of the report; and in the decision to submit the paper for publication.

Human subjects review

The study protocol was reviewed and approved, prior to its conduct, by the Institutional Review Board of Chung Shan Medical University Hospital, Taiwan.

Abbreviations

1-OHP - 1-hydroxypyrene;

ETS - Environmental tobacco smoke;

HE - High exposure community;

IQR - Inter-quartile range;

LE - Low exposure community;

PAHs - Polycyclic aromatic hydrocarbons.

1. Introduction

Studies have reported that exposure to polycyclic aromatic hydrocarbons (PAHs) increased cancer incidence among the general population (Baek et al., 1991; Nielsen et al., 1996) and affected neurobehavioral development in children (Perera et al., 2008; Tang et al., 2008). The emission sources of PAHs include residential heating, home cooking, and industrial production activities (Baek et al., 1991). Previous studies have shown that coal-fired power plants (Fang et al., 2006; Tang et al., 2008) and coal-burning activities (Jongeneelen, 1994; Mandalakis et al., 2005; Zhao et al., 1990) are major industrial sources of PAHs.

Urinary 1-hydroxypyrene (1-OHP), a major metabolite of pyrene, has been widely used as a biomarker of occupational and environmental exposure to PAHs in humans (Castano-Vinyals et al., 2004). Previous studies have reported a significant association between urinary 1-OHP levels and occupational exposure to PAHs in traffic police (Burgaz et al., 2002), coke plant workers (Strunk et al., 2002; Wu et al., 1998), steel plant workers (Kang et al., 1995), coal tar workers (Jongeneelen et al., 1985), fire fighters (Caux et al., 2002), restaurant workers (Pan et al., 2008), and boilermaker construction workers at oil-fired power plants (Mukherjee et al., 2004). Results from several studies of the general population have indicated that environmental exposure to PAHs from sources such as traffic emissions (Chang et al., 2009; Ruchirawat et al., 2006), wood burning (Cavanagh et al., 2007), and industrial emissions from steel mills, coke plants and petrochemical manufacturing complexes (Kuo et al., 2004; Lee et al., 2009; Lee et al., 2007; Mucha et al., 2006) significantly increased urinary 1-OHP levels.

However, the effects of emissions from coal-fired power plants on PAH exposure of children living in proximity to the plants have not been investigated. This study aimed to investigate the effects of PAH emissions from a large coal-fired power plant

on ambient air PAH concentrations and on urinary 1-OHP levels of children residing in nearby communities.

2. Materials and Methods

2.1 Study communities and the coal-fired power plant

The power plant investigated in this study is the largest coal-fired power plant in Taiwan and is located in Taichung County of central Taiwan. Taichung County consists of an area of 2,051 km^2 and had an estimated population of about 1.56 million in 2009. The power plant has been in operation since 1990 and currently has ten coal-fired units, which generate 41.9 billion kilowatt-hours per year by burning about 17 million tons of coal and provide over 26% of the electric power supply in Taiwan.

The location of the study communities and the coal-fired power plant are shown in Figure 1. The distance of the community from the power plant and wind direction and wind speed were taken into account in the selection of the study communities. The dominant wind direction for the area near the power plant was from north to south, as shown by the wind rose map in Figure 2. The sequential hourly meteorological data was collected at a meteorological station near the power plant during 2005-2009. Two communities located downwind from the power plant and estimated to be the most affected areas were chosen as the high exposure communities (HE1 and HE2). Two communities located more than 5 kilometers from the plant and which were unlikely to be affected by the plant were chosen as the low exposure communities (LE1 and LE2). The four study communities are located in townships with the same level of urbanization, "newly developing townships" (Liu et al., 2006), and had a population density of 1892 (HE1 and LE2), 1602 (HE2), or 1339 (LE1) person/km^2 in 2008. In the more urbanized townships in Taichung County, the population density ranged from 2720 to 6691

person/km^2.

2.2. Study design and study subjects

This cross-sectional study was part of the Health and Environmental Exposure Study, in which the health status and environmental exposures of 1400 residents aged 1-65 years were evaluated in 2009. These study subjects will be followed periodically to evaluate the long-term health effects of the coal-fired power plant. The household registration data of the four study communities were obtained from the local government and were used to develop a list of all the residents. Eligible study subjects were randomly sampled from the list.

For the present study, children aged 1-13 years were included in the analysis and those who smoked or did not actually live in the four communities were excluded. These children participated in a questionnaire survey and provided a urine sample for measurement of 1-OHP. The study protocol was reviewed and approved, prior to its conduct, by the Institutional Review Board of Chung Shan Medical University Hospital, Taiwan. An informed consent was signed by the parents or guardians of each child before he or she participated in this study.

2.3. Ambient air sampling and analysis for PAHs

The prevailing wind direction in the study area is north to north-northeast during all months, except for June, July and August, in which the main direction is south to south-southwest. Ambient air samples of PAHs were collected during two time periods with distinct wind directions, April and August of 2009. In each of the study communities, we selected two sites (elementary schools or community activity centers) for the collection of ambient air samples. PAHs were sampled using PS-1 high volume

air samplers. Glass fiber was used to collect particle-phase PAHs and polyurethane foam was used to adsorb gas-phase PAHs. The air was sampled continuously for 72 hours at a flow rate of approximately 0.225 m^3/min. The concentrations of the U.S. Environmental Protection Agency's (US EPA) 16 priority pollutant PAHs, including naphthalene, acenaphthylene, acenaphthene, fluorene, phenanthrene, anthracene, fluoranthene, pyrene, benzo[a]anthracene, chrysene, benzo[b]fluoranthene, benzo[k]fluoranthene, benzo[a]pyrene, indeno[1,2,3-cd]pyrene, dibenzo[a,h]anthracene and benzo[ghi]perylene (US EPA, 1998), were analyzed following the National Institute of Occupational Safety and Health method (NIOSH 5515) and using gas chromatography-mass spectrometry. The limits of detection for these 16 species of PAHs were 0.0007, 0.0004, 0.0007, 0.0009, 0.0008, 0.0010, 0.0007, 0.0007, 0.0009, 0.0019, 0.0009, 0.0015, 0.0049, 0.0018, 0.0049 and 0.0015 ng/m^3, respectively.

2.4. Questionnaire survey for important factors

Trained interviewers visited each participating child's home and interviewed his/her parents or guardian using a structured questionnaire. The questionnaire collected information on the child's home characteristics, medical history, demographic factors, semi-quantitative food frequency, food sources, habits, and time-activity profile. The questions were mainly adopted from validated questionnaires used in previous studies and were modified, evaluated by experts, and pre-tested on residents in the study area.

After the interview, the participants were given instructions and a specific date for the collection of a first-morning urine sample. In addition, on the day of urine collection, they answered a short questionnaire regarding the consumption of charbroiled food and cigarette smoking during the previous seven days.

Demographic factors (including gender, age, and socioeconomic status) and factors

reported to be associated with PAH exposures or urinary 1-OHP levels were collected in the questionnaire survey and evaluated in the data analysis. These factors are described as follows. Exposure to environmental tobacco smoke (ETS) was assessed using two questions: frequency of ETS exposure (>=6 days/week, 3-5 days/week, 1-2 days/week, <1 day/week, none) and the places of exposure (home or other specific places). In the data analysis, we dichotomized ETS exposure as: >=3 days/week (exposed) vs. <3 days/week (unexposed). Consumption of charbroiled food was assessed during the seven-day period before the collection of each child's urine sample. In the analysis, we considered charbroiled food intake within 24 hours of urine collection. Questions about the energy sources (wood, straw, coal, gas, or electricity) used for cooking or heating at home were included to define each subject's exposure to PAHs from wood burning at home (yes: using wood, straw or coal; no: not using any of these). For exposure to PAHs from traffic sources, we used the distance from the subject's home to main roads in the study area (yes: next to main roads; no: not next to main roads). Finally, family income (< US$20000 vs >= US$20000 per year) was used as a surrogate measure for the subject's socioeconomic status.

2.5. Collection of urine samples and measurement of urinary 1-OHP

The interviewer visited each participating child's home in the early morning on specific days scheduled for each community and collected a first-morning urine sample from each child. The samples were mainly collected on Saturdays from August to mid-December of 2009. The urine sample was collected using a sterile bottle. To prevent contamination, urine samples were not collected from children who were wearing diapers. The samples were stored in –80°C freezers until analysis. We used the following procedures to prepare and analyze urinary 1-OHP: First, the defrosted urine

(10 mL) was adjusted to pH = 5.0 with 10 mL acetate buffer (0.1 M Acetic acid v.s. 0.1 M Sodium acetate = 1:2) and digested with 20 μL of β- glucuronidase/arylsulphatase before being put in an electronically controlled rotary shaking bath (100 rpm for 24 hours) at 37±0.5℃. Second, the 1-OHP in the urine samples was extracted, enriched and purified by cartridges packed with C-18 reversed-phase liquid chromatographic material (Waters SEP-PAK VAC C-18, Waters, Milford, MA, USA) at a loading rate of less than 3 mL/min. The retained solutes in cartridges were eluted with 6 mL of isopropyl alcohol and dried by a laminar flow of nitrogen gas. The processed sample was dissolved in 2 mL of isopropyl alcohol for analysis. Third, chemical analyses were conducted using a high-performance liquid chromatography (Waters 2695) with fluorescence detector (Waters 474) and a 150 mm × 4 mm LiChrosorb RP-18 (5 μm) column (Supelo). A 20 μL sample was injected into the column at room temperature at a flow rate of 1 mL/min. The mobile phase was 65 % methanol (methanol/water = 65:35). The excitation wavelength and emission wavelength of fluorescence were 281 nm and 388 nm, respectively. This method had a limit of detection of 0.028 ng/mL, an average recovery rate of 96.18 %, and a coefficient of variance smaller than 10 % for repeated measurements. Urinary creatinine was used to adjust the measured 1-OHP concentrations, and the levels of 1-OHP were expressed in umol/mol creatinine. Urine samples with creatinine concentrations higher than 1.5-fold of 340 mg/dL or lower than half of 30 mg/mL (Que Hee, 1993) were excluded from the data analysis of this study.

2.6. Statistical analysis

Data analysis started with descriptive statistics for all variables. In the analysis, total PAH concentration represented the sum of 16 PAHs for each collected gas-phase and particle-phase PAH sample. The Mann-Whitney U test was used to compare

ambient air PAH concentrations between HE (HE1 and HE2 combined) and LE (LE1 and LE2 combined) and to compare gas phase and particle phase PAHs in each community. Log_{10} transformation was used to enhance the normality of creatinine-adjusted 1-OHP concentrations. A chi-square test was conducted to compare the frequencies of categorical variables among the four study communities. The Student's t test was used to compare means of urinary 1-OHP between two groups (e.g. girls and boys). Pearson's correlation coefficient was used to assess the linear relationship between log_{10}-transformed 1-OHP and continuous variables. One-way analysis of variance (ANOVA) was applied to compare means of urinary 1-OHP or other continuous variables among the four communities, and, if significant, Tukey's test was used for post-hoc comparisons. Finally, multiple regression analysis was conducted to assess the association between the creatinine-adjusted 1-OHP level (log_{10}-transformed) and ambient air PAHs or the study community, taking into account important factors. The covariates included in the regression models were demographic factors (age, gender, and family income) and factors evaluated in the above bi-variate analysis. The multiple regression results are presented by the back-transformation of the parameter estimate (b) and standard error (SE) and estimated the ratio as well as its 95% confidence interval (95% CI)) for 1-OHP levels between two values of each categorical variable (using equations: ratio = 10^{b} and 95% CI = $10^{b\pm1.96\times SE}$) and by the inter-quartile range (IQR) of each continuous variable (using equations: ratio = $10^{IQR\times b}$ and 95% CI = $10^{IQR\times(b\pm1.96\times SE)}$). A p value of less than 0.05 was considered significant.

3. Results

In total, 408 children participated in the questionnaire survey and 380 of them provided urine samples for 1-OHP analysis, with a response rate of 75% and 70%,

respectively. After excluding 11 urine samples with extreme creatinine concentrations, there were 369 children with both questionnaire data and urinary 1-OHP data included in the data analysis for this study.

The characteristics and urinary 1-OHP levels of the participating children are presented in Table 1. There was a marginally significant difference in family income among the communities ($p = 0.07$). Distributions of gender and frequencies of exposure to ETS were similar among the communities. The majority of the children did not eat any charbroiled food on the day before the collection of their urine samples; therefore, this factor was not evaluated further in the regression analysis. About 37% of the children lived next to main roads in the study area, and there was a marginally significant difference among the communities ($p = 0.07$). None of the families used wood, straw or coal for heating or cooking (data not shown in table). Children in HE1, the community nearest to the power plant, had the highest mean (± SD) urinary concentrations of 1-OHP (0.194 ± 0.143 μmol/mol creatinine). Those in LE1, the community farthest from the plant, had the lowest 1-OHP levels (0.113 ± 0.082 μmol/mol creatinine). Using ANOVA, the means of urinary 1-OHP levels (log10-transformed) were significantly different among the communities. Further comparison using Tukey's test showed that children in HE1 and HE2, respectively, had significantly higher urinary 1-OHP levels than those in LE1 and LE2 (HE1> LE1, HE1>LE2, HE2>LE1, and HE2>LE2).

As for the comparisons of $\log_{10}$-transformed 1-OHP levels between groups of children with different characteristics (results of t test not shown in table), the mean 1-OHP level was not significantly different between children who had and had not consumed charbroiled food ($t = -0.49$, $df = 367$, $p = 0.63$). Residential proximity to main roads did not influence 1-OHP levels ($t = 0.17$, $df = 367$, $p = 0.86$). Children with ETS

exposure had similar 1-OHP levels as those without exposure (t = 0.85, df = 367, p = 0.40). Furthermore, the level of urinary 1-OHP was significantly correlated with age (r = -0.28, n=369, $p < 0.0001$) and duration of residency in the study area (r = -0.18, n=369, $p = 0.0004$). Moreover, age was highly correlated with duration of residency (r = 0.83, n=369, $p < 0.0001$). Therefore, duration of residency was not evaluated further in the regression model.

The means of ambient air PAH concentrations in each study community are presented in Table 2. The concentrations of total and gas-phase PAHs, respectively, in the high exposure communities (HE1 and HE2 combined) were significantly higher than those in the low exposure communities (LE1 and LE2 combined). For HE1, HE2, and LE1, the concentrations of gas-phase PAHs were significantly higher than those of particle-phase PAHs. We further analyzed the wind direction and wind speed data during the ambient air sampling periods (one week in April and one week in August) and categorized each PAH air sample into a down-wind or up-wind group according to the main wind direction during that period. The mean concentrations of gas-phase, particle-phase, and total PAHs were 7.794 ng/m^3, 1.086 ng/m^3, and 8.879 ng/m^3, respectively, for the down-wind group and were 5.320 ng/m^3, 1.067 ng/m^3, and 6.388 ng/m^3 for the up-wind group. There was a significant difference in total PAHs between the down-wind and up-wind groups (p values = 0.081, 0.949, and 0.033 for gas-phase, particle-phase, and total PAHs, respectively, Mann-Whitney U test) (data not shown in table).

After adjusting for age, gender, family income, proximity to main roads, and ETS exposure in the multiple regression model, urinary 1-OHP concentrations of children in HE1 and HE2, respectively, were significantly higher than those of children in LE1 (Table 3, model I). The mean 1-OHP level in HE1 was 1.85 times (95% CI: 1.43-2.40)

the mean level in LE1. The variables in model I explain the 17.4% variability in urinary 1-OHP levels. Total PAHs was significantly associated with urinary 1-OHP levels (model II). The mean 1-OHP level at the 75th-percentile of total PAHs was 1.45 times (95% CI: 1.26-1.66) the level at the 25th-percentile of total PAHs. Age was significantly associated with decreased urinary 1-OHP levels in both models. Children with ETS exposures had marginally significant higher urinary 1-OHP levels than those without ETS exposure (estimated ratio = 1.14, 95% CI = 0.98-1.32, Model I). In both models, children in higher income families (>= US$20000/year) had marginally significant lower urinary 1-OHP levels than those in lower income families (<US$20000/year). Furthermore, gas-phase PAHs (estimated ratio = 1.41, 95% CI = 1.23-1.62), but not particle-phase PAHs (estimated ratio = 0.99, 95% CI = 0.85-1.16), were significantly associated with urinary 1-OHP levels (results of multiple regression models not shown in table).

4. Discussion

In the present study, children living in proximity and downwind of the coal-fired power plant had significantly higher urinary concentrations of 1-OHP than those living farther from the plant after taking other factors into account. This result was consistent with the findings of significantly higher concentrations of total and gas-phase PAHs in the ambient air, respectively, in the high exposure communities than in the low exposure communities. Our findings are comparable to a previous Ukrainian study, which showed that children living within three miles of a steel mill and coke oven had the highest urinary 1-OHP concentrations compared with those in areas far away from the plants (Mucha et al., 2006).

Children participating in this study did not smoke cigarettes, a major source of

PAH exposure in people without occupational PAH exposure (Hansen et al., 2008), and had no occupational exposure to PAHs or other chemicals. Moreover, none of the families used coal or wood for heating or cooking, one of the major sources of indoor PAH exposure (Beak et al., 1991). Therefore, these factors could not confound the observed associations between urinary 1-OHP and environmental exposure to PAHs.

As to the effect of age on urinary 1-OHP, our study result was consistent with previous findings of a significant decreasing trend in 1-OHP concentrations with increasing age (Heudorf and Angerer, 2001; Huang et al., 2004). Such a difference is understandable because children tend to have higher PAH exposure from soil and dust through hand-to-mouth activities and have higher inhalation rates per unit body-weight than adults (Committee on Environmental Health, 1999; Landrigan et al., 1998).

Previous studies have shown inconsistent findings with regard to gender differences in urinary 1-OHP concentrations. Some studies found that males had higher 1-OHP concentrations than females (Jongeneelen, 1994; Siwinska et al., 1998), while another study reported higher concentrations in females (Lee et al., 2007). In our study, there was no significant difference in urinary 1-OHP concentrations between boys and girls (Table 3), which was consistent with the finding by Mucha et al. (2006).

Previous studies have also indicated that environmental tobacco smoke, one of the important sources of indoor air pollution, raised urinary 1-OHP concentrations (Freire et al., 2009; Jongeneelen, 2001; Siwinska et al., 1999; Tsai et al., 2003). Our study result showed a marginally significant effect of ETS exposure on urinary 1-OHP levels after taking other factors into account. In this study, the information for ETS exposure at home or in other places was collected by interviewing the children's parents or guardians. Although the interviewers were well-trained, non-differential misclassification of the children's ETS exposure due to incorrect recall could have

biased the association between ETS exposure and urinary 1-OHP toward the null.

Diet has been considered a primary source of PAH exposure in non-occupationally exposed people (Fiala et al., 2001; Viau et al., 2002; Vyskocil et al., 2000). Previous studies have reported that the half-life of 1-OHP ranges from 6 to 35 hours (Jongeneelen et al., 1990) or about 29 hours (Huang et al., 2007). Considering the short half-life of 1-OHP, we only evaluated the effects of charbroiled food intake the day before the collection of urine samples. There was no significant difference in the mean urinary 1-OHP level between children who had and had not consumed charbroiled food, and only 18 (4.88%) of the participating children were reported to have eaten charbroiled food during the specified time period. Therefore, the effects of charbroiled food on urinary 1-OHP levels could not be further evaluated in our multiple regression analysis. Since the majority of the participating children did not eat charbroiled food, PAH exposure from this food source had limited effects on the observed association between community of residency and urinary 1-OHP in this study.

Traffic sources have long been recognized as major contributors to PAHs and have effects on urinary 1-OHP levels (Beak et al., 1991; Tsai et al., 2004). However, in our multiple regression analysis, close proximity to main roads in the study area was not significantly related to urinary 1-OHP concentrations. One possible reason for the limited influence of traffic on the children's PAH exposure is that all four study communities are located in rural areas where the traffic is not heavy.

In this study, the high exposure communities located downwind and near the power plant had significantly higher total and gas-phase PAH concentrations in the ambient air than did communities farther from the plant. Moreover, children living in the high exposure communities had significantly elevated urinary 1-OHP levels than those in the low exposure communities, after controlling for potential confounding factors in the

study design or in the multiple regression analysis. These findings imply that exposure to gas-phase PAHs in the ambient air from the power plant might be associated with higher urinary 1-OHP levels in the participating children. However, caution is needed in interpretation of the association because of the limitations of this study. First, the air samples for PAH concentrations were only collected during two seasons with distinct wind directions and from eight sampling locations. The measured concentrations might not be able to represent the long-term PAH levels in the ambient air in these communities. Nonetheless, other than the measured ambient air PAH concentrations, we have used the community of residence as a surrogate for the children's long-term environmental exposure to PAHs in the analysis. Second, the contribution of emissions from the power plant to ambient air PAH concentrations in the study communities was not clear, although distance and wind direction/speed were used to identify the study communities. Based on 2007 data from the Taiwan Emission Data System, the studied power plant was the main contributor of sulfur oxides (93.50 %), nitrogen oxides (91.02 %), and particulate matter with an aerodynamic diameter less than or equal to 10 μm (49.90%) among all major industrial emission sources within 10 kilometers of the plant (Taiwan Environmental Protection Administration, 2007). The relative roles of the power plant and other emission sources to the ambient air gas-phase PAH concentrations in our study communities needs to be further evaluated through the application of source apportionment methods.

This study also had several strengths. First, in addition to distance and wind direction/speed, the level of urbanization was taken into account in selecting low exposure communities. The study communities were more comparable; therefore, the results were unlikely to be confounded by other unmeasured community-related factors. Second, this study evaluated urinary 1-OHP levels and PAH exposures on a

representative sample of the general population from the study communities. The observed association between urinary 1-OHP levels and environmental PAH exposure was less likely to be affected by selection bias. Third, potential confounding effects of PAH exposure from other important sources, such as smoking, ETS exposure, traffic, home heating/cooking, and diet, were controlled in the study design or in the data analysis and were not likely to bias the findings.

5. Conclusions

Children living downwind of and in proximity to the coal-fired power plant had significantly increased urinary 1-OHP levels. The potential health effects of increased 1-OHP levels in these children merit further investigation.

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Disclosure Statement

The authors have no actual or potential conflict of interest.

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Table legends

Table 1 Characteristics and urinary 1-OHP levels of participating children

Table 2 Ambient air concentrations of 16 US EPA's priority PAHs in the study communities

Table 3 Results of the multiple regression analysis for factors associated with urinary 1-OHP levels

Figure legends

Figure 1 Location of the coal-fired power plant and the study communities

HE–High exposure community; LE–Low exposure community.

Figure 2 The rose histogram of wind direction (one-hour average data) for the period 2005-2009

Table 1 Characteristics and urinary 1-OHP levels of participating children

Variables	All subjects	HE1	HE2	LE1	LE2
Sample size	369	88	146	49	86
Age (yr), mean±SD	8.73 ± 3.16	8.96 ± 3.21	8.33 ± 3.24	9.04 ± 3.06	8.99 ± 3.02
Gender, n (%)					
boy	198 (53.66)	48 (54.55)	70 (47.95)	29 (59.18)	51 (59.30)
girl	171 (46.34)	40 (45.45)	76 (52.05)	20 (40.82)	35 (40.70)
Education, n (%)					
pre-school	114 (30.89)	25 (28.41)	50 (34.25)	13 (26.53)	26 (30.23)
elementary school	255 (69.11)	63 (71.59)	96 (65.75)	36 (73.47)	60 (69.77)
Family income, n (%)					
< US$ 20,000/yr	258 (69.92)	61 (69.32)	103 (70.55)	40 (81.63)	54 (62.79)
>= US$ 20,000/yr	111 (30.08)	27 (30.68)	43 (29.45)	9 (18.37)	32 (37.21)
Eating charbroiled food during the day before urine collection, n (%)					
no	351 (95.12)	84 (95.45)	138 (94.52)	48 (97.96)	81 (94.19)
yes	18 (4.88)	4 (4.55)	8 (5.48)	1 (2.04)	5 (5.81)
Living next to the main roads in study area, n (%)					
no	231 (62.60)	64 (72.73)	89 (60.96)	25 (51.02)	53 (61.63)
yes	138 (37.40)	24 (27.27)	57 (39.04)	24 (48.98)	33 (38.37)
Duration of residency in study area (yr), mean±SD	7.94 (3.51)	8.15 (3.74)	7.60 (3.30)	8.70 (3.28)	7.88 (3.70)
Exposure to environmental tobacco smoke, n (%)					
no (<3 days/week)	201 (54.47)	47 (53.41)	86 (58.90)	22 (44.90)	46 (53.49)
yes (>= 3 days/week)	168 (45.53)	41 (46.59)	60 (41.10)	27 (55.10)	40 (46.51)
Body mass index (kg/m^2), mean±SD	17.99 ± 3.81	18.18 ± 4.91	17.95 ± 3.40	17.37 ± 3.25	18.23 ± 3.50
Urinary 1-hydroxypyrene (μmol/mol-creatinine),					
mean±SD	0.163 ± 0.131	0.194 ± 0.143***	0.186 ± 0.148	0.113 ± 0.082	0.122 ± 0.089
geometric mean±geometric SD	0.123 ± 2.188	0.153 ± 2.025***	0.141 ± 2.195	0.085 ± 2.278	0.096 ± 2.014

HE–high exposure community; LE–low exposure community.

$^{***}p < 0.001$, ANOVA.

Table 2 Ambient air concentrations of 16 US EPA's priority PAHs in the study communities

	High exposure communities (HE)				Low exposure communities (LE)			
	HE1 (n = 6)		HE2 (n = 4)		LE1 (n = 4)		LE2 (n = 2)	
PAHs (ng/m^3)	gas-phase	particle-phase	gas-phase	particle-phase	gas-phase	particle-phase	gas-phase	particle-phase
Naphthalene	0.2750[a]	0.1070	0.3959	0.0438	0.0522	0.1526	0.3779	0.0842
Acenaphthylene	0.0481	0.0049	0.0413	0.0031	0.0088	0.0043	0.0468	0.0016
Acenaphthene	0.0081	0.0033	0.0068	0.0021	0.0082	0.0380	0.0102	0.0017
Fluorene	0.1122	0.0131	0.0802	0.0021	0.0640	0.0125	0.1001	0.0069
Phenanthrene	3.5623	0.1018	3.0025	0.0591	2.0465	0.1117	1.9650	0.0520
Anthracene	0.2097	0.0040	0.1535	0.0022	0.0751	0.0031	0.1367	0.0005
Fluoranthene	1.8219	0.1524	2.4774	0.0737	1.1107	0.1542	1.6512	0.0625
Pyrene	1.3297	0.1380	2.2191	0.0685	1.0255	0.1472	1.4339	0.0639
Benzo[a]anthracene	0.0516	0.0603	0.0693	0.0307	0.0351	0.0414	0.0679	0.0490
Chrysene	0.1119	0.1116	0.1259	0.0737	0.0751	0.0805	0.1213	0.0805
Benzo[b]fluoranthene	0.0449	0.1988	0.0589	0.1439	0.0526	0.1380	0.0576	0.1735
Benzo[k]fluoranthene	0.0117	0.0519	0.0180	0.0370	0.0068	0.0507	0.0169	0.0469
Benzo[a]pyrene	0.0048	0.0579	0.0025	0.0216	0.0107	0.0588	0.0042	0.0541
Indeno[1,2,3-cd]pyrene	0.0009	0.1184	0.0009	0.0369	0.0102	0.0871	0.0009	0.0729
Dibenzo[a,h]anthracene	0.0024	0.0094	0.0024	0.0046	0.0024	0.0051	0.0024	0.0024
Benzo[ghi]perylene	0.0042	0.1560	0.0007	0.0921	0.0159	0.1150	0.0024	0.1490
Sum of PAHs	7.5993*	1.2889	8.6553	0.6951	4.5998	1.2001	5.9955	0.9017
Total PAHs	8.8882*		9.3504		5.7999		6.8972	

PAHs–Polycyclic aromatic hydrocarbons.

[a]Data are mean values in ng/m^3. *$p < 0.05$ HE (HE1 and HE2 combined) compared to LE (LE1 and LE2 combined), Mann-Whitney U test.

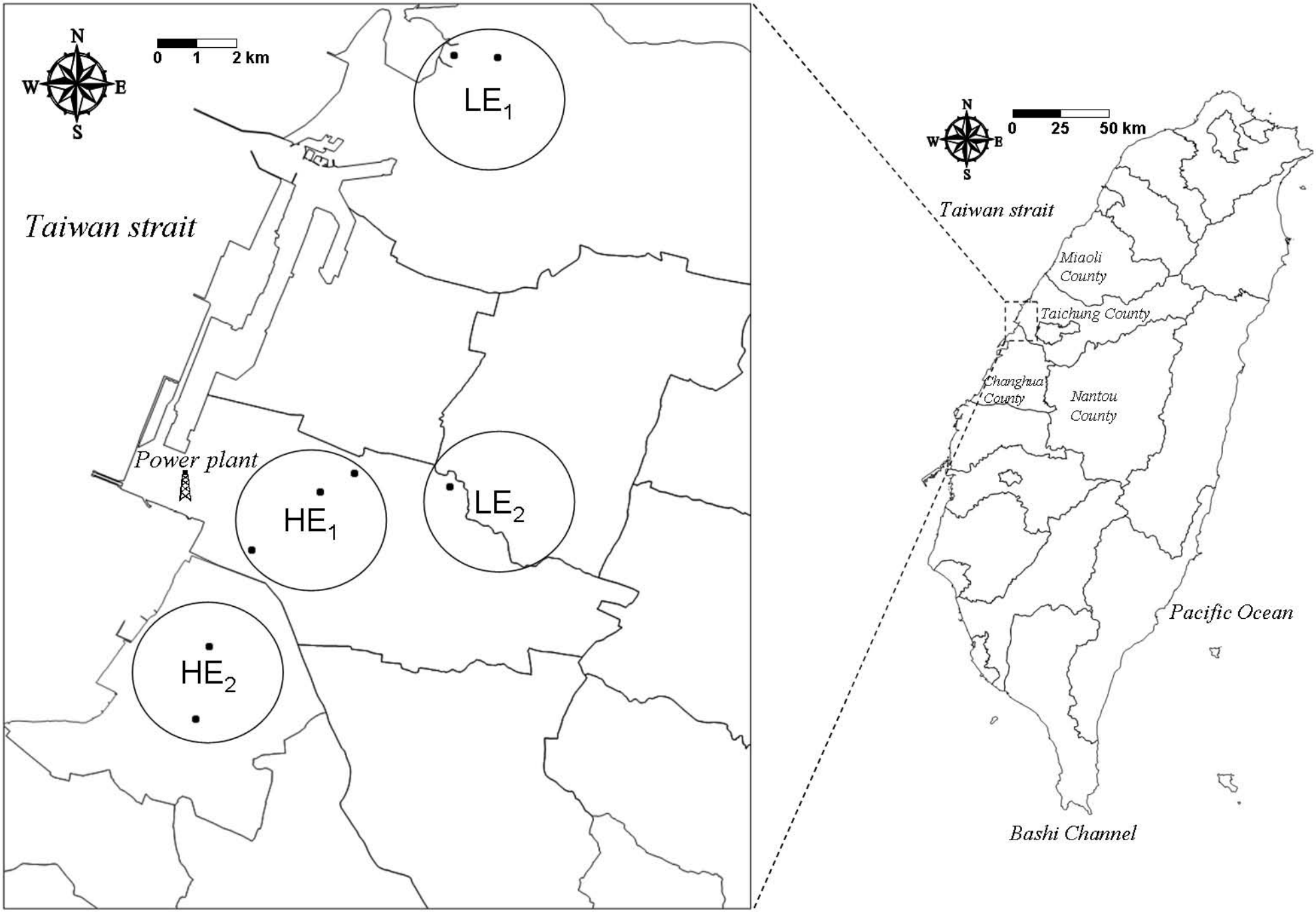
Table 3 Results of the multiple regression analysis for factors associated with urinary 1-OHP levels

	Estimated ratio (95% confidence interval)[a]	
	Model I	Model II
R-square	0.174	0.160
Adjusted R-square	0.153	0.146
Age (yr)	0.78 (0.63, 0.95)***	0.72 (0.64, 0.80)***
Gender		
girl	reference	reference
boy	1.16 (0.88, 1.53)	0.92 (0.79, 1.07)
Study communities		
LE1	reference	—
LE2	1.16 (0.90, 1.51)	—
HE1	1.85 (1.43, 2.40)***	—
HE2	1.65 (1.30, 2.09)***	—
Total PAHs (ng/Nm^3)	—	1.45 (1.26, 1.66)***
Family income		
< US$ 20,000/yr	reference	reference
>= US$ 20,000/yr	0.88 (0.75, 1.03)	0.88 (0.74, 1.03)
Exposure to environmental tobacco smoke		
no (<3 days/week)	reference	reference
yes (>= 3 days/week)	1.14 (0.98, 1.32)	1.12 (0.86, 1.30)
Living next to the main roads in study area		
no	reference	reference
yes	1.06 (0.91, 1.24)	1.05 (0.90, 1.22)

HE–high exposure community; LE–low exposure community.

[a] The parameter estimates were back-transformed and presented as the estimated ratio (95% confidence interval) for creatinine-adjusted 1-OHP levels between two values of each categorical variable and by the inter-quartile range of each continuous variable (age and total PAHs).

***$p < 0.001$.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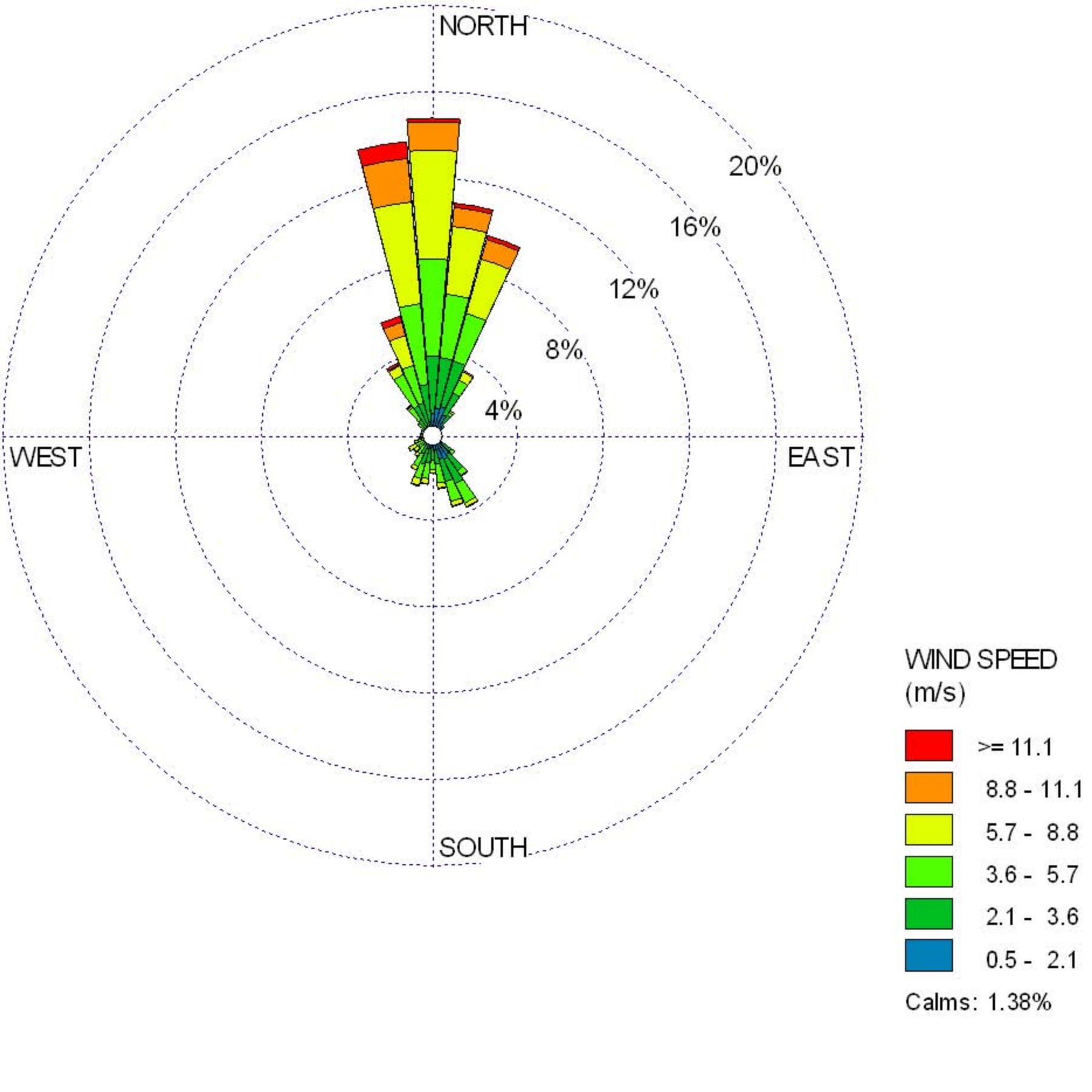
Taichung County
Changhua County
Nantou County
Pacific Ocean
Bashi Channel


NORTH
EAST
SOUTH
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4%
8%
12%
16%
20%
WIND SPEED
(m/s)
>= 11.1
8.8 - 11.1
5.7 - 8.8
3.6 - 5.7
2.1 - 3.6
0.5 - 2.1
Calms: 1.38%