**A new grid-scale model simulating the spatiotemporal distribution of
PM2.5-PAHs for exposure assessment**

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Abstract

Exposure to polycyclic aromatic hydrocarbons (PAHs) associated with ambient air particulate matter (PM) poses significant health concerns. Several modeling approaches have been developed for simulating ambient PAHs, but no hourly intra-urban spatial data are currently available. The aim of this study is to develop a new modeling strategy in simulating, on an hourly basis, grid-scale PM_{2.5}-PAH levels. PM and PAHs were collected over a one-year time frame through an established air quality monitoring network within a metropolitan area of Taiwan. Multivariate linear regression models, in combination with correlation analysis and PAH source identification by principal component analysis (PCA), were performed to simulate hourly grid-scale PM_{2.5}-PAH concentrations, taking criteria pollutants and meteorological variables selected as possible predictors. The simulated levels of 72-hr personal exposure were found to be significantly ($R=0.729^{**}$, $p<0.01$) correlated with those analyzed from portable personal monitors. A geographic information system (GIS) was used to visualize spatially distributed PM_{2.5}-PAH concentrations of the modeling results. This new grid-scale modeling strategy, incorporating the output of simulated data by GIS, provides a useful and versatile tool in personal exposure analysis and in the health risk assessment of air pollution.

Keywords: PM2.5-PAH, multivariate linear regression model, human health,
grid-scale model, hourly intra-urban spatial simulation

1 Introduction

The impact of air pollution on health and disease has been well recognized, and the adverse effects of particulate matter (PM) and PAHs on cardiovascular mortality and respiratory diseases (e.g., asthma and lung cancer), particularly for the at-risk subsets of the population [1-7], have been well documented. The risk is particularly high when exposed to fine and ultrafine particles (UFP) with an aerodynamic diameter $<2.5\ \mu\text{m}$, PM_{2.5} and $<100\ \text{nm}$, UFP, respectively [8-11]. Also, particle concentration, surface area and particle composition (e.g., organic and inorganic compounds) are important in mediating the effects of PM on human health [12, 13].

PAHs are a group of persistent organic contaminants, some of which have been classified as carcinogenic or probably carcinogenic to humans, such as benzo [a] pyrene and dibenzo [a,h] anthracene [14, 15]. Thus, monitoring PAH exposure is of great importance for emission control and risk assessment, as PAHs can accumulate in the tracheobronchial and alveolar epithelium and enter the circulatory system [16, 17].

In Taiwan, the Environmental Protection Agency (EPA) has long established air quality monitoring stations across different regions of Taiwan, including those for the real-time monitoring of ambient air, traffic, and industrial pollutants. The air quality data are presented and archived on EPA's website (<http://taqm.epa.gov.tw>), providing a risk monitoring system. However, assessment of PAH health effects remains a

1 challenge, as the exposure and risk assessment often rely upon the data generated
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4 from regulatory monitoring stations lacking PAH levels.
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7 In the past decades, modeling approaches have been utilized to simulate ambient
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10 PAH concentrations to resolve the issue of sparse data [18-24]. However, only a
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13 handful of studies related to PAH predictions in the atmosphere have been
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16 demonstrated and no hourly intra-urban spatial data are available. Past studies have
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19 emphasized the importance of considering personal time-spending in daily life
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22 activities in personal PAH exposure estimation [21, 25]. Thus, the development of
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25 hourly-spatial PAH data prediction can be useful and would provide a valuable
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28 database for epidemiological study.
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32 We have previously reported that PM and PAH pollutions were associated with
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35 local microclimate, emission sources, and topography in the Kaohsiung coastal area
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38 [26, 27]. We presented herein the development of a geostatistical model simulating
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41 hourly grid-scale PAH concentrations associated with ambient air pollution. This new
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44 grid-scale model would thus offer an improved monitoring tool with wide
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47 applicability, such as the epidemiological investigation of PM_{2.5}-PAH effects on
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2 Methodology

2.1 Environmental and personal exposure air sample collection

The environmental air samples were collected from four sampling sites in Kaohsiung city, Taiwan (Figure 1). The selection of sampling sites was based on their geographical distribution (north sector, south sector, city center, and coastal areas). The sampling site in the north sector designated FS is located in the most densely populated administrative area in Kaohsiung city. SG is located at the south sector, an area with petrochemical, shipbuilding, fire power plant, iron, and steel industrial activities. The city center site, AG, is characterized by high traffic density and heavy commercial activities, and the coastal area site, designated NSYSU, is located at National Sun Yat-sen university campus. The 24-hour particle samples (twice a month) were collected sequentially at each of the four sampling sites by the Model 310 Universal Air Sampler (UAS-310, MSP Corporation, MN, USA) during the period of December 2012 to November 2013. One time sampling failed at 4 sampling sites in May due to mechanical problems with the sampler. To avoid the interference from road dust, each air sampler was set 15–20 m high on a building above ground level. The sampler has an inlet sampling flow rate of 300 L/min and provided with a virtual impactor for size separation of airborne particles in the 2.5–10 μm (PM_{2.5-10}) and 0–2.5 μm (PM_{2.5}) aerodynamic size ranges. PM_{2.5-10} and PM_{2.5} were collected on

the 62 mm × 165 mm and 200 mm × 250 mm quartz fiber filter (QFF), respectively.

(Figure 1 position)

Personal exposure air samples were also collected, analyzed and compared in the same duration from Dec. 2012 to Nov. 2013, which were conducted through a 72-hr collection time period for PM_{2.5} by a personal environmental monitor (PEM, SKC Model 200) with an air sampling pump (SKC Model AirChek XR5000). The PEM was operated at a flow rate of 4 L/min and protected in the nylon bag to collect PM_{2.5} on a 37 mm QFF. A PEM was hung on a bag strap in the breathing zone of each participant during sampling time. The PEM was left operating in the same space while it was inconvenient for participant to carry (e.g., sleeping or showering time) during the sampling time. To provide enough electricity for AirCheck XR5000, we also requested participants to recharge it before they go to sleep at night every day. Total 62 participants residing in the study area were recruited with informed consent following the approved protocol by the Institutional Review Board, Kaohsiung Medical University. According to the survey of questionnaire, participants with exposure of smoking and incense were excluded. There were 52 personal exposure samples successfully collected. The age of 52 participants ranged from 19 to 77 (51 ± 15) years old with a normal distribution ($p > 0.05$, Shapiro-Wilk Test).

2.2 PAH analytical procedures

For PAH analysis, the QFF samples were extracted with dichloromethane using pressurized fluid extraction with an accelerated solvent extractor (Dionex ASE 300) at a pressure of 1500 psi and 100°C. The extracts were concentrated to 5 mL in a rotary evaporator. The resulting extracts were concentrated to 0.5 mL by evaporation under a nitrogen stream for GC-MS analysis. A capillary gas chromatograph (Agilent 6890N) and a mass spectrometer (Agilent 5973N), operating under the selected ion monitoring mode, were used to identify and quantify PAHs from samples. Prior to analysis, a mixture of perdeuterated PAHs (acenaphthene-d₁₀, phenanthrene-d₁₀, benzo(a)anthracene-d₁₂, and benzo(a)pyrene-d₁₂, and benzo[g,h,i]perylene-d₁₂) was added as an internal standard. Each PAH was identified by retention time relative to the internal standards and quantified by comparing the integrated area of the molecular ion chromatogram to those of the standards. The concentrations of 16 PAHs were determined in this study, including naphthalene (Nap), acenaphthylene (Aceny), acenaphthene (Acen), fluorene (Flu), phenanthrene (Ph), anthracene (An), fluoranthene (Flt), pyrene (Py), chrysene+triphenylene (Chry+TriPhe), benzo[a]anthracene (BaA), benzo[k]fluoranthene (BkF), benzo[b]fluoranthene (BbF), benzo[a]pyrene (BaP), dibenzo[a,h]anthracene (DBA), indeno[1,2,3-cd]pyrene (IP), and benzo[g,h,i]perylene (BghiP). The dominant PAHs found in PM_{2.5} were

associated with high molecular weight compounds, such as BbF, BkF, BaP, DBA, BghiP and IP, (averagely 67.9% among 16 PAHs) in this study (Table S1). These compounds have been identified as having high carcinogenic and mutagenic potential for human health [28]. Considering the effects of PM_{2.5}-PAHs on human health is more severe than other coarse fractions, only the results of hourly PM_{2.5}-PAH simulation are reported in this study.

2.3 Quality assurance and control

For quality control measurements, field and laboratory blanks were extracted and analyzed in the same way as samples. In this study, four perdeuterated PAHs (naphthalene-d₈, fluorene-d₁₀, fluoranthene-d₁₀, and perylene-d₁₂) were utilized as surrogates to determine the efficiency of the sample extraction and analysis. The ranges of mean recoveries in environmental PM_{2.5}-PAH of four sites were $50.7 \pm 9.8\%$ ~ $53.2 \pm 13.3\%$ for naphthalene-d₈, $79.2 \pm 13.2\%$ ~ $80.7 \pm 11.5\%$ for fluorene-d₁₀, $83.8 \pm 15.7\%$ ~ $88.3 \pm 12.8\%$ for fluoranthene-d₁₀, and $73.5 \pm 14.5\%$ ~ $78.7 \pm 11.5\%$ for perylene-d₁₂. In personal exposure assessment of PM_{2.5}-PAH, the ranges of mean recoveries were $74.7 \pm 9.9\%$, $92.7 \pm 11.4\%$, $108 \pm 14.8\%$, and $88.0 \pm 11.4\%$ for naphthalene-d₈, fluorene-d₁₀, fluoranthene-d₁₀, and perylene-d₁₂, respectively.

2.4 Model simulation and GIS mapping approach for epidemiological research

The new hourly grid-scale model was constructed by multivariate linear regression (MLR) for PM_{2.5}-PAH concentration simulation and a GIS mapping approach with ordinary kriging (OK) interpolation method (Figure 2).

(Figure 2 position)

2.4.1 Multivariate linear regression (MLR) model

There are four sampling sites (SG, AG, FS and NSYSU) and 13 EPA monitoring stations, including AG, in the study area (Figure 2). PM_{2.5}-PAH data collected from the four sampling sites covered the urban area of Kaohsiung. The other air pollutant data and meteorological data (e.g., PM₁₀, NO_x, wind speed, and temperature) were collected from EPA monitoring station nearby; the data of rainfall, rainfall strength and atmospheric pressure were obtained from the Central Weather Bureau (CWB, <http://www.cwb.gov.tw>) in Kaohsiung city. All of the data from EPA and CWB were in hourly format. Statistical analysis was performed using Statistical Package for Social Sciences (SPSS) software (version 19.0.0, SPSS, Inc, Chicago, IL, USA). The correlations among routinely monitored criteria pollutant data, meteorological data, and measured PM_{2.5}-PAH concentrations are shown in Supplemental Material, Table S2; parameter selection was mainly based on the correlation coefficients greater than

0.5 and had significant correlations with PM2.5-PAH concentrations.

The equation of the MLR model is as follows:

$$y = \alpha + \beta_1 X_1 + \beta_2 X_2 + \beta_3 X_3 + \beta_4 X_4 \quad (1)$$

where X is the independent variable (the values of four parameters: PM10, NO_x, CO, and TEMP), y is the value of the dependent variable (PM2.5-PAH) for values X₁, X₂, X₃, and X₄ of the independent variables, and $\alpha(1.462)$, $\beta_1(0.001)$, $\beta_2(0.049)$, $\beta_3(0.410)$ and $\beta_4(-0.064)$ are regression coefficients.

2.4.2 Ordinary kriging (OK) interpolation

After MLR model construction, the hourly prediction of PM2.5-PAHs derived from MLR model at the 13 EPA monitoring sites was utilized for optimizing OK interpolation. The GIS mapping approach used the R (version 3.2.2) with gstat package [29] by OK interpolation to illustrate the spatial variation of PM2.5-PAH concentrations. Three variogram models (spherical, exponential and Gaussian) were used to fit the data distribution with no nugget effect for selecting the best parameters (sill and range). The resolution of GIS mapping was 0.001 by 0.001-degree latitude/longitude grid, with coordinates ranging from longitude 120.20 to 120.45 E and latitude 22.45 to 22.78 N. The model performance indicated that spherical model showed better results than the others (Table S3); therefore, only the simulation by

spherical model was presented in this study. Geocoding from 4 sampling sites and 52 participants' home addresses were then used for mapping spatiotemporal PM_{2.5}-PAH distributions from hourly OK output at each grid point.

The accuracy of grid-scale model prediction was assessed by two measures: the RMSE (equation 2) and the mean absolute error (MAE, equation 3).

$$\text{RMSE} = \sqrt{\frac{\sum_{i=1}^n (M_i - P_i)^2}{n}} \quad (2)$$

$$\text{MAE} = \frac{\sum_{i=1}^n |M_i - P_i|}{n} \quad (3)$$

where n is the number of variables, M_i is the actual output, and P_i is the predicted output. When RMSE and MAE values are zero, this indicates that the model prediction is a perfect fit to the actual observation. Thus, smaller values indicate a better prediction of a model [30].

3 Results and Discussion

3.1 Spatiotemporal variations of the PM and PAH concentrations

The seasonal concentrations of PM (PM_{2.5} and PM₁₀) at the four sampling sites (SG, FS, AG, and NSYSU) from December 2012 to November 2013 are shown in

Figure 3, which also depicts the total concentrations of sixteen PAHs in PM_{2.5}. The air pollution data (PM and PAHs) showed the same temporal distribution with high concentrations being noted during the spring, autumn, and winter, while the lowest concentrations were found in the summer at the sampling sites, which may be caused by consecutive emissions from traffic activities (Figure 3). The spatiotemporal difference in PM_{2.5}-PAH concentrations, with relatively low concentrations being noted at the NSYSU site among the four sites, suggests that the amount of pollution emissions and meteorological features were important factors. About 70% of PM pollution was found to be attributed to PM_{2.5}, and the average daily and annual concentrations of PM_{2.5} in observation days both exceeded the current 24-hr and annual standards, 35 and 15 $\mu\text{g}/\text{m}^3$, respectively. Thus, it would be useful and valuable to provide PM_{2.5}-PAH data with time series and geographical information for epidemiological research, for instance exposure estimations and the correlation between daily registered patients in the hospital and PM_{2.5}-PAH concentrations.

(Figure 3 position)

3.2 Correlation analysis and source identification

The correlations among the concentrations of PM at the four sampling sites and EPA monitoring data are shown in Supplemental Material, Figure S1. The measured

PM2.5 and PM10 at the sampling sites were all significantly correlated with the data from EPA (PM2.5, $r=0.678^{**}$ and PM10, $r=0.776^{**}$, $p<0.01$). These correlations suggested that the measurement of PM2.5 concentrations at the four sampling sites was representative in the study area.

PM2.5-PAH source identification plays an important role in grid-scale model construction for diminishing prediction errors from the interferences of local sources. Principal component analysis (PCA) was performed to identify the possible sources of PM2.5-PAHs. Supplemental Material, Table S4 lists PCA rotated component loadings of the concentrations for PM2.5-PAHs at the four sampling sites. The major sources of PM2.5-PAHs in the study area could be classified into three categories as petroleum combustion (gasoline and diesel vehicular), incinerator/coke oven emissions, and coal combustion (see Supplemental Material, Table S4 and the description therein)[31-35]. PCA results demonstrate that the sources of PM2.5-PAH with temporal-spatial differences were affected by the characteristics of local emissions and geographical features. This information emphasized the importance in obtaining small-scale hourly PM2.5-PAH concentrations, which may be valuable for further understanding hourly and spatial variations of PAHs, and in applying the data to epidemiological research.

The source characteristics of PM2.5-PAH identified by PCA are useful for

1 predictor selection of MLR simulation to represent different sources and
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3 meteorological factors for the study area. According to the results of correlation
4 analysis and PCA, PM10, NO_x, CO, and TEMP were selected as predictors to
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6 enhance the accuracy of predicted values (Table S2 and Table S4). NO_x and TEMP
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8 were selected to represent nitrogen oxide group and meteorological group,
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10 respectively, although the coefficients of NO, NO₂ and Pressure were higher than 0.5.
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12 Correlation analysis showed that THC and CH₄ were with higher correlation
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14 coefficients than PM10 to PM2.5-PAHs; however, these two pollutant data were not
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16 concerned to be parameters, as the regularly monitoring data were not available for all
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18 EPA stations.
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36 **3.3 Evaluation of measurements and simulation results**

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38 The validation and evaluation of the hourly model predicted concentrations was
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40 carried out in two ways. One was using 24-hour measured data (92 samples) versus
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42 the average of each set of 24 hourly grid-scale model predicted concentrations (Figure
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44 4). The other one was using the 72-hour personal monitoring data (52 cases) versus
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46 average of each set of 72 hourly grid-scale model predicted concentrations (Figure 5),
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55 due to the hourly measurement of PAH is technically not feasible up to date.
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58 Comparisons of the grid-scale model predicted values and the measured data of
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PM2.5-PAHs at the four sampling sites are shown in Figure 4. The results revealed good correlations between the predicted and the measured PM2.5-PAH concentrations at all sites ($n=92$, $r=0.817^{**}$, $p<0.01$) and each site. The values of prediction accuracy measures of RMSE and MAE also demonstrated that the grid-scale model could provide satisfactory simulations of PM2.5-PAHs for the four sampling sites (Table 1).

Figure 5 displays the correlations of 72-hr average PM2.5-PAH data between predicted values and personal exposure measured by the use of personal portable monitors for 52 cases. The results indicated that the predicted values exhibited significant positive correlations ($r=0.729^{**}$, $p<0.01$) with measured concentrations. These results suggest the PM2.5-PAHs predicted values are reliable to be utilized to evaluate the effects of PM2.5-PAHs on respiratory diseases in the epidemiological study.

(Table 1, Figures 4 and 5 position)

3.4 Applications of hourly PM_{2.5}-PAH concentration simulation

Radar plots illustrated the frequency of daily peak and low PM2.5-PAH concentrations' occurrence in a 24 hours period for observing the hourly PM2.5-PAH variation in the study area (Figure 6), and the dynamic changes were demonstrated in Supplemental Material, Figure S2. For the four sampling sites, the highest

PM2.5-PAH concentrations mostly occurred at 8:00 to 9:00 (frequency: 13-25%) and the lowest level was observed at 14:00 to 15:00 (frequency: 13-24%). In addition, there was another time period (23:00 to 1:00) showing around 5-9% occurrence with the highest levels of PM2.5-PAHs. The high frequency observed at 8:00 to 9:00 was associated with a peak traffic period in the study area. As mentioned above, one of the parameters utilized for data simulation was NO_x mainly emitted by automobiles for the study area. Therefore, the high NO_x concentration provided further evidence of traffic's contribution to the high PM2.5-PAH concentrations observed during peak traffic periods (Figure 6).

The occurrence of high PM2.5-PAH concentrations was found to be related to anthropogenic activities directly, and the times with a high frequency of low PM2.5-PAH and the second highest frequency of high PM2.5-PAH occurrence were associated with meteorological conditions from geographic features in the study area as further discussed below. The study area is susceptible to land-sea circulation, in particular for the coastal site (NSYSU) [27]. Figure 6 shows that the higher frequency of low PM2.5-PAH concentration occurred at NSYSU site (28%) during 11:00-13:00 time period than the other sites. This difference may be related to the location of sampling sites and the influence of traffic intensity. The NSYSU site is located in the direction of the prevailing wind during the daytime. The westerly or northwesterly

1 can directly bring clean air from the sea to the sampling sites and favor air pollutant
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4 dispersion, resulting in low PM_{2.5}-PAH concentrations at the time, while the
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7 dispersion effect lessens at the other sites because of their location. In the case of high
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10 PM_{2.5}-PAH concentrations occurred at midnight, this could be caused by the
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13 interaction between meteorological conditions and industrial emissions. At night, a
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16 low inversion layer may suppress the dispersion of air pollutants emitted from
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19 factories in nearby areas. Moreover, the wind blows from the easterly or northeasterly
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22 direction toward the sea, carrying polluted air from the inland area to the coastal area
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25 at night. This hourly modeled data presented the effects of the interaction of land-sea
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28 circulation on diurnal variations of PM_{2.5}-PAHs at the sampling sites.
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32 (Figure 6 position)
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39 Figure 7 shows the spatial variations of seasonal average of PM_{2.5}-PAH
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42 concentrations presented by contour map, according to hourly simulated data from the
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45 grid-scale model for the study area, covering thirteen EPA air quality monitoring sites.
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48 Analysis of seasonal variations indicated that the highest PM_{2.5}-PAH season in the
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51 study area was winter, while the lowest PM_{2.5}-PAH season was noted in summer.
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54 During the winter, the source of PM_{2.5}-PAHs was mainly attributed to pollutants
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57 transported from neighboring areas and local pollution (e.g., a power plant in the
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south monitoring site). In the summer, two local sources were observed: one at the EPA western monitoring site, and the other at the northern monitoring site. The sources of PM_{2.5}-PAHs at both sites are related to industrial activities. These contour maps also showed how meteorological factors, such as wind direction, affect the spatial variations of PM_{2.5}-PAH concentrations. This phenomenon indicated that the land-sea circulation plays an important role in the air quality of Kaohsiung urban area. These maps illustrate the possible effects of the interaction between meteorological conditions and local emission sources on PM_{2.5}-PAH spatial distributions. The model is also applicable for specific PAH compound, for example BaP. In Figure S3, contour maps of PM_{2.5}-BaP seasonal concentration are presented with two representative study cases, one of which residing in the southern area of the city, while another in the northern region. These two cases are indicated, respectively, as “Case A” and “Case B” in Figure S3. Hourly PM_{2.5}-BaP concentrations derived from grid-scale model combined with the unit risk (UR) of BaP ($UR = 8.7 \times 10^{-5}$) [36] were used to estimate the lifetime risk of lung cancer of PM_{2.5}-BaP inhalation exposures. The lifetime lung cancer risks of the case A in the southern area were higher than that for the case B in the northern area with annual average risks of 7.4×10^{-6} and 5.4×10^{-6} , respectively. The estimation of lifetime lung cancer risk by the data from this approach was more representative than other studies [37-39], as the

1 data can be used to present grid-scale PM_{2.5}-BaP exposure risk and to provide
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4 exposure risks different time period. For instance, the lifetime lung cancer risks in
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7 winter period were higher than those in other seasons (Figure S3).
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16 **Conclusions**

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18 A new grid-scale model for simulating hourly PM_{2.5}-PAH concentrations was
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20 developed through the correlation between criteria air pollutants and meteorological
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22 conditions obtained from air quality monitoring network data near the sampling sites.
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29 PM_{2.5}-PAH source identification demonstrated the necessity of small-scale hourly
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33 PM_{2.5}-PAH concentrations in epidemiological research as the sources with
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36 spatiotemporal differences in the study area. Model performance suggests that the
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39 prediction results are acceptable for grid-scale hourly concentrations. From the
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42 evaluation test of portable personal monitor data, moderate correlations between
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45 predicted values and measured concentrations were found, suggesting that hourly
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48 simulated data may be applicable to personal exposure risk assessment to reduce the
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51 inconvenience of personal exposure sampling.
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54 The applications of a grid-scale model could demonstrate the effects of
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57 microclimate and the sources on the distribution of PM_{2.5}-PAHs, the diurnal and
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seasonal PM_{2.5}-PAH concentrations varied with both meteorological phenomena (land-sea circulation) and anthropogenic activities (traffic and industrial emissions). Visualization of hourly grid-scale data by GIS can also be used to understand the spatiotemporal distribution of PM_{2.5}-PAHs in the study area. Overall, the new grid-scale model is believed to be the best approach so far for providing hourly PM_{2.5}-PAH concentration, as the hourly measurement of PM_{2.5}-PAH is technically not feasible up to date disregarding the intensive labor and cost involved.

Acknowledgements

Financial support from the Ministry of Science and Technology (MOST 100-2611-M-110-010), and the Ministry of Education of Taiwan (DOE 01C030203) is gratefully acknowledged. This study was also supported in part by Kaohsiung Medical University “Aim for the Top University Grant” (Grant No. KMU-TP103A27), NSYSU-KMU Joint Research Project (NSYSUKMU104-P030) and Academia Sinica (BM104010120).

References

- [1] J.W. Holloway, S. Savarimuthu Francis, K.M. Fong, I.A. Yang, Genomics and the respiratory effects of air pollution exposure, *Respirology*, 17 (2012) 590-600.
- [2] J. Kaiser, Mounting Evidence Indicts Fine-Particle Pollution, *Science*, 307 (2005) 1858-1861.
- [3] D.M. Krewski, R.T. Jerrett, R. Burnett, E. Ma, Y. Hughes, M.C. Shi, C. Turner, G. Arden Pope III, E.E. Thurston, Calle, M.J. Thun, Extended Follow-Up and Spatial Analysis of the American Cancer Society Study Linking Particulate Air Pollution and Mortality, in: HEI Report 140 2009.
- [4] F. Laden, J. Schwartz, F.E. Speizer, D.W. Dockery, Reduction in Fine Particulate Air Pollution and Mortality: Extended Follow-up of the Harvard Six Cities Study, *American Journal of Respiratory and Critical Care Medicine*, 173 (2006) 667-672.
- [5] C.A. Pope III, R.T. Burnett, M.J. Thun, E.E. Calle, D. Krewski, K. Ito, G.D. Thurston, Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution, *JAMA*, 287 (2002) 1132-1141.
- [6] C.A. Pope III, R.T. Burnett, G.D. Thurston, M.J. Thun, E.E. Calle, D. Krewski, J.J. Godleski, Cardiovascular Mortality and Long-Term Exposure to Particulate Air Pollution: Epidemiological Evidence of General Pathophysiological Pathways of Disease, *Circulation*, 109 (2004) 71-77.
- [7] S.-S. Tsai, H.-F. Chiu, S.-H. Liou, C.-Y. Yang, Short-Term Effects of Fine Particulate Air Pollution on Hospital Admissions for Respiratory Diseases: A Case-Crossover Study in a Tropical City, *Journal of Toxicology and Environmental Health, Part A*, 77 (2014) 1091-1101.
- [8] M.L. Block, L. Calderón-Garcidueñas, Air Pollution: Mechanisms of Neuroinflammation & CNS Disease, *Trends in neurosciences*, 32 (2009) 506-516.
- [9] L. Calderón-Garcidueñas, A. Mora-Tiscareño, E. Ontiveros, G. Gómez-Garza, G. Barragán-Mejía, J. Broadway, S. Chapman, G. Valencia-Salazar, V. Jewells, R.R. Maronpot, C. Henríquez-Roldán, B. Pérez-Guillé, R. Torres-Jardón, L. Herrit, D. Brooks, N. Osnaya-Brizuela, M.E. Monroy, A. González-Maciel, R. Reynoso-Robles, R. Villarreal-Calderon, A.C. Solt, R.W. Engle, Air pollution, cognitive deficits and brain abnormalities: A pilot study with children and dogs, *Brain and Cognition*, 68 (2008) 117-127.
- [10] S. Genc, Z. Zadeoglulari, S.H. Fuss, K. Genc, The Adverse Effects of Air Pollution on the Nervous System, *Journal of Toxicology*, 2012 (2012).
- [11] C. Kim, S.H. Jung, D.R. Kang, H.C. Kim, K.T. Moon, N.W. Hur, D.C. Shin, I. Suh, Ambient Particulate Matter as a Risk Factor for Suicide, *The American Journal of Psychiatry*, 167 (2010) 1100-1107.

- [12] C.C. Daigle, D.C. Chalupa, F.R. Gibb, P.E. Morrow, G. Oberdörster, M.J. Utell, M.W. Frampton, Ultrafine Particle Deposition in Humans During Rest and Exercise, *Inhalation Toxicology*, 15 (2003) 539-552.
- [13] J.S. Lighty, J.M. Veranth, A.F. Sarofim, Combustion Aerosols: Factors Governing Their Size and Composition and Implications to Human Health, *Journal of the Air & Waste Management Association*, 50 (2000) 1565-1618.
- [14] IARC, Overall Evaluations of Carcinogenicity : An updating of IARC Monographs Volumes 1 to 42, in: *Monographs on the Evaluation of the Carcinogenicity of Chemicals to Humans*, 1987.
- [15] IARC, Some non-heterocyclic polycyclic aromatic hydrocarbons and some related exposures, in: *Monographs on the Evaluation of the Carcinogenicity of Chemicals to Humans*, 2010, pp. 1-853.
- [16] C.-E. Boström, P. Gerde, A. Hanberg, B. Jernström, C. Johansson, T. Kyrklund, A. Rannug, M. Törnqvist, K. Victorin, R. Westerholm, Cancer risk assessment, indicators, and guidelines for polycyclic aromatic hydrocarbons in the ambient air, *Environmental Health Perspectives*, 110 (2002) 451-488.
- [17] P. Gerde, B.A. Muggenburg, M.D. Hoover, R.F. Henderson, Disposition of Polycyclic Aromatic Hydrocarbons in the Respiratory Tract of the Beagle Dog: I. The Alveolar Region, *Toxicology and Applied Pharmacology*, 121 (1993) 313-318.
- [18] C. Gariazzo, M. Lamberti, O. Hänninen, C. Silibello, A. Pelliccioni, D. Porta, A. Cecinato, M. Gherardi, F. Forastiere, Assessment of population exposure to Polycyclic Aromatic Hydrocarbons (PAHs) using integrated models and evaluation of uncertainties, *Atmospheric Environment*, 101 (2015) 235-245.
- [19] A.B. Lobscheid, T.E. McKone, D.A. Vallero, Exploring relationships between outdoor air particulate-associated polycyclic aromatic hydrocarbon and PM_{2.5}: A case study of benzo(a)pyrene in California metropolitan regions, *Atmospheric Environment*, 41 (2007) 5659-5672.
- [20] E.M. Noth, S.K. Hammond, G.S. Biging, I.B. Tager, A spatial-temporal regression model to predict daily outdoor residential PAH concentrations in an epidemiologic study in Fresno, CA, *Atmospheric Environment*, 45 (2011) 2394-2403.
- [21] T. Ohura, T. Noda, T. Amagai, M. Fusaya, Prediction of Personal Exposure to PM_{2.5} and Carcinogenic Polycyclic Aromatic Hydrocarbons by Their Concentrations in Residential Microenvironments, *Environmental science & technology*, 39 (2005) 5592-5599.
- [22] R. San José, J.L. Pérez, M.S. Callén, J.M. López, A. Mastral, BaP (PAH) air quality modelling exercise over Zaragoza (Spain) using an adapted version of WRF-CMAQ model, *Environmental Pollution*, 183 (2013) 151-158.
- [23] O. Sáňka, L. Melymuk, P. Čupr, A. Dvorská, J. Klánová, Dispersion modeling of

selected PAHs in urban air: A new approach combining dispersion model with GIS and passive air sampling, *Atmospheric Environment*, 96 (2014) 88-95.

[24] A. Zalel, Yuval, V. Svecova, R.J. Sram, A. Bartonova, D.M. Broday, Modeling airborne benzo(a)pyrene concentrations in the Czech Republic, *Atmospheric Environment*, 101 (2015) 166-176.

[25] C.C. Tonne, R.M. Whyatt, D.E. Camann, F.P. Perera, P.L. Kinney, Predictors of personal polycyclic aromatic hydrocarbon exposures among pregnant minority women in New York City, *Environmental Health Perspectives*, 112 (2004) 754-759.

[26] I.C. Lai, Y.-C. Chang, C.-L. Lee, G.-Y. Chiou, H.-C. Huang, Source identification and characterization of atmospheric polycyclic aromatic hydrocarbons along the southwestern coastal area of Taiwan – with a GMDH approach, *Journal of Environmental Management*, 115 (2013) 60-68.

[27] I.C. Lai, C.-L. Lee, K.-Y. Zeng, H.-C. Huang, Seasonal variation of atmospheric polycyclic aromatic hydrocarbons along the Kaohsiung coast, *Journal of Environmental Management*, 92 (2011) 2029-2037.

[28] WHO, Polynuclear Aromatic Hydrocarbons (PAH). In: *Air Quality Guidelines for Europe.*, in, World Health Organization Regional Office for Europe, Copenhagen, 1987, pp. 105-117.

[29] E.J. Pebesma, Multivariable geostatistics in S: the gstat package, *Computers & Geosciences*, 30 (2004) 683-691.

[30] D.N. Moriasi, J.G. Arnold, M.W.V. Liew, R.L. Bingner, R.D. Harmel, T.L. Veith, *Model Evaluation Guidelines for Systematic Quantification of Accuracy in Watershed Simulations*, 50 (2007).

[31] H.-C. Huang, C.-L. Lee, C.-H. Lai, M.-D. Fang, I.C. Lai, Transboundary movement of polycyclic aromatic hydrocarbons (PAHs) in the Kuroshio Sphere of the western Pacific Ocean, *Atmospheric Environment*, 54 (2012) 470-479.

[32] S. Kong, J. Shi, B. Lu, W. Qiu, B. Zhang, Y. Peng, B. Zhang, Z. Bai, Characterization of PAHs within PM10 fraction for ashes from coke production, iron smelt, heating station and power plant stacks in Liaoning Province, China, *Atmospheric Environment*, 45 (2011) 3777-3785.

[33] P. Kulkarni, C. Venkataraman, Atmospheric polycyclic aromatic hydrocarbons in Mumbai, India, *Atmospheric Environment*, 34 (2000) 2785-2790.

[34] K. Ravindra, L. Bencs, E. Wauters, J. de Hoog, F. Deutsch, E. Roekens, N. Bleux, P. Berghmans, R. Van Grieken, Seasonal and site-specific variation in vapour and aerosol phase PAHs over Flanders (Belgium) and their relation with anthropogenic activities, *Atmospheric Environment*, 40 (2006) 771-785.

[35] S. Sarkar, P.S. Khillare, Profile of PAHs in the inhalable particulate fraction: source apportionment and associated health risks in a tropical megacity,

Environmental Monitoring and Assessment, 185 (2013) 1199-1213.

[36] WHO, Air Quality Guidelines for Europe. 2nd ed., in, World Health Organization Regional Office for Europe, Copenhagen, 2000.

[37] N. Ramírez, A. Cuadras, E. Rovira, R.M. Marcé, F. Borrull, Risk Assessment Related to Atmospheric Polycyclic Aromatic Hydrocarbons in Gas and Particle Phases near Industrial Sites, Environmental Health Perspectives, 119 (2011) 1110-1116.

[38] E.O. Gaga, A. Ari, T. Dogeroglu, E. Emel Cakrca, N.E. Machin, Atmospheric polycyclic aromatic hydrocarbons in an industrialized city, Kocaeli, Turkey: study of seasonal variations, influence of meteorological parameters and health risk estimation, Journal of Environmental Monitoring, 14 (2012) 2219-2229.

[39] Y. Jia, D. Stone, W. Wang, J. Schrlau, S. Tao, S.L. Massey Simonich, Estimated Reduction in Cancer Risk due to PAH Exposures If Source Control Measures during the 2008 Beijing Olympics Were Sustained, Environmental Health Perspectives, 119 (2011) 815-820.

Table 1 The prediction accuracy of grid-scale model on PM2.5-PAH for four sampling sites

Index	SG (n=23)	AG (n=23)	FS (n=23)	NSYSU (n=23)	All (n=92)
RMSE	0.638	0.468	0.573	0.583	0.569
MAE	0.514	0.340	0.439	0.458	0.438

Figure Captions

Figure 1. Map of the study area, Kaohsiung city in southwest Taiwan.

Figure 2. GIS mapping framework of PM_{2.5}-PAH model simulation procedure (solid line: validation of PM_{2.5}-PAH between measured and grid-scale model predicted data; dot line: evaluation of PM_{2.5}-PAH between personal exposure measured and grid-scale model predicted data).

Figure 3. Temporal variations in the concentration of PM_{2.5}, PM₁₀, and PM_{2.5}-PAHs at four sampling sites. (A) SG, (B) AG, (C) FS, and (D) NSYSU.

Figure 4. Correlation analysis of 24-hr average concentration of the grid-scale model predicted and measured PM_{2.5}-PAHs for the four sampling sites in the study period.

Figure 5. The correlation of 72-hr average of ambient PM_{2.5}-PAH concentrations between grid-scale model predicted and personal exposure measured.

Figure 6. The comparison of frequency for (A) PAH concentrations and (B) NO_x concentrations in a 24hr period at the four sampling sites for the study period.

Figure 7. Seasonal variations of predicted PM_{2.5}-PAH concentrations. (A) Winter, (B) Spring, (C) Summer, and (D) Autumn.

Figure 1

Figure 2

Figure 3

Figure 4

Figure 5

Figure 6

Figure 7

Table S1. The proportion of 16-PAHs in PM2.5

PM2.5-PAHs (%)	Sampling site			
	SG	AG	FS	NSYSU
NaP	0.0	0.5	0.6	0.9
Aceny	0.5	0.6	0.5	0.7
Acen	0.3	0.3	0.3	0.4
Flu	1.1	0.8	0.7	1.2
Ph	7.1	6.0	6.1	7.9
An	1.2	1.0	0.9	1.2
Flt	6.8	5.9	6.4	9.3
Py	6.0	5.1	5.5	7.9
BaA	2.4	2.3	2.5	2.9
Chry+TriPhe	5.7	5.5	5.9	7.2
BbF	16.3	15.7	17.2	15.0
BkF	8.7	8.3	8.5	7.9
BaP	5.0	4.6	5.0	5.7
IP	19.1	20.5	19.2	16.1
DBA	2.1	2.0	2.1	1.8
BghiP	17.6	20.8	18.7	13.8
LMW-PAHs	10.3	9.3	9.2	12.4
MMW-PAHs	20.9	18.8	20.2	27.3
HMW-PAHs	68.8	71.9	70.6	60.3

LMW-PAHs: sum of NaP, Aceny, Acen, Flu, Ph and An

MMW-PAHs: sum of Flt, Py, BaA and Chry+TriPhe

HMW-PAHs: sum of BbF, BkF, BaP, DBA, BghiP and IP

Table S2. The correlation between PM2.5-PAHs concentration of all sampling sites and EPA air monitoring parameters

	PM2.5-PA Hs	SO ₂	CO	O ₃	PM10	NO _x	NO	NO ₂	THC ^a	NMHC ^b	CH ₄	WS ^c	WD ^d	TEMP ^e	RH ^f	P	Rainfal l	RS ^g
PM2.5-PAHs	1																	
SO ₂	.280**	1																
CO	.616**	.310**	1															
O ₃	.031	.055	.071	1														
PM10	.582**	.279**	.598**	.371**	1													
NO _x	.810**	.531**	.615**	-.117	.564**	1												
NO	.553**	.333**	.356**	-.473**	.103	.761**	1											
NO ₂	.799**	.535**	.638**	.084	.700**	.947**	.514**	1										
THC ^a	.642**	.364**	.878**	.178	.667**	.653**	.334**	.700**	1									
NMHC ^b	.597**	.400**	.887**	.020	.514**	.647**	.410**	.652**	.943**	1								
CH ₄	.619**	.270**	.761**	.345**	.754**	.572**	.197	.662**	.932**	.764**	1							
WS ^c	-.322**	-.201	-.612**	-.110	-.273**	-.314**	-.153	-.341**	-.530**	-.554**	-.424**	1						
WD ^d	.055	-.059	.151	.203	.327**	.003	-.109	.059	.182	.096	.250*	-.081	1					
TEMP ^e	-.681**	-.043	-.485**	-.085	-.592**	-.619**	-.314**	-.665**	-.532**	-.383**	-.632**	.133	-.083	1				
RH ^f	-.386**	-.308**	-.116	-.234*	-.390**	-.272**	.057	-.384**	-.177	-.163	-.181	-.038	-.096	.164	1			
P	.615**	.143	.516**	.167	.676**	.559**	.192	.650**	.590**	.454**	.663**	-.286**	.168	-.749**	-.330**	1		
Rainfall	-.052	.004	-.092	-.110	-.207*	.008	.081	-.030	-.075	.000	-.149	-.023	-.224*	.066	.342**	-.255*	1	
RS ^g	-.055	.022	-.069	-.034	-.192	.012	.090	-.029	-.030	.038	-.087	-.073	-.198	.098	.194	-.160	.745**	1

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

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

THC^a: Total hydrocarbons, NMHC^b: Non-methane hydrocarbons, WS^c: Wind speed, WD^d: Wind direction,TEMP^e: Temperature, RH^f: Relative humidity, RS^g: Rainfall strength

Table S3. The performance of Ordinary Kriging variogram model fitted between grid-scale model predicted and environmental measured PM2.5-PAH

Model	MAE	RMSE	r ²
Spherical (n=92)	0.438	0.569	0.667
Exponential (n=92)	0.437	0.572	0.663
Gaussian (n=92)	0.634	0.873	0.337

Table S4. Results of principal component analysis for PAHs in PM2.5

Sampling site	SG			AG			FS			NSYSU		
Phase	PM2.5			PM2.5			PM2.5			PM2.5		
Component	1	2	3	1	2	3	1	2	3	1	2	3
NaP	0.040	0.884	-0.348	0.175	-0.132	0.795	0.027	-0.004	0.880	-0.095	0.199	-0.808
Aceny	-0.064	0.903	-0.118	0.120	-0.056	0.817	-0.112	0.252	0.798	-0.069	0.576	-0.656
Acen	0.445	0.569	-0.644	0.836	-0.161	0.166	0.161	0.585	0.487	-0.080	0.767	-0.472
Flu	0.461	0.536	-0.673	0.933	0.116	0.137	0.041	0.939	0.096	0.028	0.871	-0.191
Ph	0.547	0.467	-0.685	0.853	0.315	0.147	0.280	0.860	-0.039	0.414	0.639	0.026
An	0.416	0.563	-0.695	0.833	-0.136	0.314	-0.095	0.923	0.257	0.069	0.877	0.040
Flt	0.929	-0.117	0.170	-0.033	0.920	-0.093	0.721	0.005	-0.432	0.936	-0.096	0.026
Py	0.971	0.096	-0.171	0.154	0.883	-0.213	0.791	0.200	-0.473	0.797	-0.051	0.234
BaA	0.075	-0.698	0.558	-0.470	0.419	-0.649	0.214	-0.368	-0.694	0.404	-0.234	0.661
Chry+TriPhe	0.245	-0.226	0.901	-0.165	0.880	-0.053	0.872	-0.190	-0.067	0.550	-0.052	0.106
BbF	-0.328	-0.805	0.159	-0.587	0.235	-0.560	0.036	-0.479	-0.463	0.120	-0.739	0.430
BkF	-0.492	0.011	0.660	-0.191	-0.023	0.030	0.031	-0.144	0.148	-0.329	-0.403	0.039
BaP	-0.248	-0.454	0.694	-0.391	0.071	-0.419	0.165	-0.268	-0.174	0.131	-0.060	0.167
IP	-0.802	-0.400	0.249	-0.309	-0.748	-0.151	-0.860	-0.165	-0.026	-0.881	-0.131	0.200
DBA	-0.812	-0.476	0.149	-0.532	-0.542	-0.553	-0.786	-0.198	-0.409	-0.563	-0.065	0.698
BghiP	-0.859	0.087	0.435	0.221	-0.773	0.375	-0.513	0.003	0.489	-0.892	-0.094	-0.083
Variance (%)	32.6	28.5	27.2	26.7	26.7	18.1	23.3	21.7	20.6	26.3	23.2	16.2
Cumulative (%)	32.6	61.0	88.3	26.7	53.3	71.5	23.3	45.1	65.7	26.3	49.4	65.7
Main Sources	1	3	2	2	1	3	1	2	3	1	2	3

Only factors with eigenvalue > 1.0 and variance > 10.0% are included. Factor loading values in bold indicated those values greater than 0.5.

Main sources: 1. Petroleum combustion (gasoline and diesel vehicular) 2. Incinerator /coke oven emissions 3. Coal combustion

Figure S1. Correlation between sampling site measurement data and EPA monitoring data of (A) PM_{2.5} and (B) PM₁₀. The straight-line represents the linear trends.

<https://drive.google.com/file/d/0B0LMfreuEDlUZEdhYnpIWlgwSnM/view?usp=sharing>

Figure S2. Animated demonstration of 24-hr spatiotemporal PM_{2.5}-PAH variations at the four sampling sites.

Figure S3. Seasonal variations of predicted PM_{2.5}-BaP concentrations. (A) Winter, (B) Spring, (C) Summer, and (D) Autumn. The lifetime lung cancer risks of the case A in the southern area were 1.0×10^{-5} for the winter period, 7.0×10^{-6} for the spring period, 4.0×10^{-6} for the summer period and 8.1×10^{-6} for the autumn period; the lifetime lung cancer risks of case B in the northern area were 8.1×10^{-6} for the winter period, 5.0×10^{-6} for the spring period, 2.7×10^{-6} for the summer period and 5.9×10^{-6} for the autumn period.

Table S1. The proportion of 16-PAHs in PM2.5

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	SG	AG	FS	NSYSU
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Flt	6.8	5.9	6.4	9.3
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RH ^f	-.386**	-.308**	-.116	-.234*	-.390**	-.272**	.057	-.384**	-.177	-.163	-.181	-.038	-.096	.164	1			
P	.615**	.143	.516**	.167	.676**	.559**	.192	.650**	.590**	.454**	.663**	-.286**	.168	-.749**	-.330**	1		
Rainfall	-.052	.004	-.092	-.110	-.207*	.008	.081	-.030	-.075	.000	-.149	-.023	-.224*	.066	.342**	-.255*	1	
RS ^g	-.055	.022	-.069	-.034	-.192	.012	.090	-.029	-.030	.038	-.087	-.073	-.198	.098	.194	-.160	.745**	1

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

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

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TEMP^e: Temperature, RH^f: Relative humidity, RS^g: Rainfall strength

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Aceny	-0.064	0.903	-0.118	0.120	-0.056	0.817	-0.112	0.252	0.798	-0.069	0.576	-0.656
Acen	0.445	0.569	-0.644	0.836	-0.161	0.166	0.161	0.585	0.487	-0.080	0.767	-0.472
Flu	0.461	0.536	-0.673	0.933	0.116	0.137	0.041	0.939	0.096	0.028	0.871	-0.191
Ph	0.547	0.467	-0.685	0.853	0.315	0.147	0.280	0.860	-0.039	0.414	0.639	0.026
An	0.416	0.563	-0.695	0.833	-0.136	0.314	-0.095	0.923	0.257	0.069	0.877	0.040
Flt	0.929	-0.117	0.170	-0.033	0.920	-0.093	0.721	0.005	-0.432	0.936	-0.096	0.026
Py	0.971	0.096	-0.171	0.154	0.883	-0.213	0.791	0.200	-0.473	0.797	-0.051	0.234
BaA	0.075	-0.698	0.558	-0.470	0.419	-0.649	0.214	-0.368	-0.694	0.404	-0.234	0.661
Chry+TriPhe	0.245	-0.226	0.901	-0.165	0.880	-0.053	0.872	-0.190	-0.067	0.550	-0.052	0.106
BbF	-0.328	-0.805	0.159	-0.587	0.235	-0.560	0.036	-0.479	-0.463	0.120	-0.739	0.430
BkF	-0.492	0.011	0.660	-0.191	-0.023	0.030	0.031	-0.144	0.148	-0.329	-0.403	0.039
BaP	-0.248	-0.454	0.694	-0.391	0.071	-0.419	0.165	-0.268	-0.174	0.131	-0.060	0.167
IP	-0.802	-0.400	0.249	-0.309	-0.748	-0.151	-0.860	-0.165	-0.026	-0.881	-0.131	0.200
DBA	-0.812	-0.476	0.149	-0.532	-0.542	-0.553	-0.786	-0.198	-0.409	-0.563	-0.065	0.698
BghiP	-0.859	0.087	0.435	0.221	-0.773	0.375	-0.513	0.003	0.489	-0.892	-0.094	-0.083
Variance (%)	32.6	28.5	27.2	26.7	26.7	18.1	23.3	21.7	20.6	26.3	23.2	16.2
Cumulative (%)	32.6	61.0	88.3	26.7	53.3	71.5	23.3	45.1	65.7	26.3	49.4	65.7
Main Sources	1	3	2	2	1	3	1	2	3	1	2	3

Only factors with eigenvalue > 1.0 and variance > 10.0% are included. Factor loading values in bold indicated those values greater than 0.5.

Main sources: 1. Petroleum combustion (gasoline and diesel vehicular) 2. Incinerator /coke oven emissions 3. Coal combustion

Figure S1. Correlation between sampling site measurement data and EPA monitoring data of (A) PM2.5 and (B) PM10. The straight-line represents the linear trends.

<https://drive.google.com/file/d/0B0LMfreuEDlUZEhYnpIWlgwSnM/view?usp=sharing>

Figure S2. Animated demonstration of 24-hr spatiotemporal PM_{2.5}-PAH variations at the four sampling sites.

Figure S3. Seasonal variations of predicted PM_{2.5}-BaP concentrations. (A) Winter, (B) Spring, (C) Summer, and (D) Autumn. The lifetime lung cancer risks of the case A in the southern area were 1.0×10^{-5} for the winter period, 7.0×10^{-6} for the spring period, 4.0×10^{-6} for the summer period and 8.1×10^{-6} for the autumn period; the lifetime lung cancer risks of case B in the northern area were 8.1×10^{-6} for the winter period, 5.0×10^{-6} for the spring period, 2.7×10^{-6} for the summer period and 5.9×10^{-6} for the autumn period.

Abstract

Exposure to polycyclic aromatic hydrocarbons (PAHs) associated with ambient air particulate matter (PM) poses significant health concerns. Several modeling approaches have been developed for simulating ambient PAHs, but no hourly intra-urban spatial data are currently available. The aim of this study is to develop a new modeling strategy in simulating, on an hourly basis, grid-scale PM_{2.5}-PAH levels. PM and PAHs were collected over a one-year time frame through an established air quality monitoring network within a metropolitan area of Taiwan. Multivariate linear regression models, in combination with correlation analysis and PAH source identification by principal component analysis (PCA), were performed to simulate hourly grid-scale PM_{2.5}-PAH concentrations, taking criteria pollutants and meteorological variables selected as possible predictors. The simulated levels of 72-hr personal exposure were found to be significantly ($R=0.729^{**}$, $p<0.01$) correlated with those analyzed from portable personal monitors. A geographic information system (GIS) was used to visualize spatially distributed PM_{2.5}-PAH concentrations of the modeling results. This new grid-scale modeling strategy, incorporating the output of simulated data by GIS, provides a useful and versatile tool in personal exposure analysis and in the health risk assessment of air pollution.

Keywords: PM2.5-PAH, multivariate linear regression model, human health, grid-scale model, hourly intra-urban spatial simulation

Environmental PM2.5-PAH exposure

Personal PM2.5-PAH exposure