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Estimating population exposure to ambient polycyclic aromatic hydrocarbon in the United States – Part II: Source apportionment and cancer risk assessment

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Abstract

A revised Community Multiscale Air Quality (CMAQ) model was developed to simulate the emission, reactions, transport, deposition and gas-to-particle partitioning processes of 16 priority polycyclic aromatic hydrocarbons (PAHs), as described in Part I of the two-part series. The updated CMAQ model was applied in this study to quantify the contributions of different emission sources to the predicted PAH concentrations and excess cancer risk in the United States (US) in 2011. The cancer risk in the continental US due to inhalation exposure of outdoor naphthalene (NAPH) and seven larger carcinogenic PAHs (cPAHs) was predicted to be significant. The incremental lifetime cancer risk (*ILCR*) exceeds 1×10^{-5} in many urban and industrial areas. Exposure to PAHs was estimated to result in 5704 (608–10,800) excess lifetime cancer cases. Point sources not related with energy generation and the oil and gas processes account for approximately 31% of the excess cancer cases, followed by non-road engines with 18.6% contributions. Contributions of residential wood combustion (16.2%) are similar to that of transportation-related sources (mostly motor vehicles with small contributions from railway and marine vessels; 13.4%). The oil and gas industry emissions, although large contributors to high concentrations of cPAHs regionally, are only responsible of 4.3% of the excess cancer cases, which is similar to the contributions of non-US sources (6.8%) and non-point sources (7.2%). The power generation units pose the most minimal impact on excess cancer risk, with contributions of approximately 2.3%.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at <http://dx.doi.org/10.1016/j.envint.2016.08.024>.

Keywords

Cancer risk assessment; United States; Polycyclic aromatic hydrocarbon; Source apportionment; Benzo[α]pyrene

1. Introduction

Polycyclic aromatic hydrocarbons (PAHs) are organic compounds that contain multiple aromatic rings. Various forms of cancer, such as skin, lung, bladder, liver and stomach, due to PAH exposure have been reported in animal exposure studies (Boström et al., 2002). Occupational and environmental exposure to polycyclic aromatic hydrocarbons (PAH) has also been related with higher human cancer risks in epidemiological studies (Boffetta et al., 1997). The United States Environmental Protection Agency (US EPA) has classified 16 of the PAHs as priority pollutants and designated seven of the PAHs possible human carcinogens (see Table 1 of Zhang et al. (2016)). To properly evaluate the level of population exposure and cancer risk due to PAHs, the spatial and temporal distribution of ambient PAHs need to be determined. Subsequently, to design effective PAH emission control strategies, the contributions of different emission sources to the exposure and cancer risk at different locations and different time of the year need to be quantified. Part I of the two-paper series described the development of an atmospheric chemical transport model (CTM) that is capable of estimating the spatial and temporal distribution of the 16 priority PAHs. The capability of the CTM to estimate daily and monthly concentrations of the PAHs in the United States in 2011 at a spatial resolution of 36-km was demonstrated. In the second part of the study, the CTM was further applied to quantitatively determine the contributions of major sources of PAHs to the estimated population exposure and cancer risk. In the following paragraphs in the introduction, major sources of PAHs as reported by the literature were summarized and typical source apportionment techniques for PAHs and their applications in different countries were critically reviewed.

Atmospheric PAHs are typically formed from fuel combustion sources such as motor vehicle exhaust and wood burning (Ravindra et al., 2008). Biofuel use accounted for a large fraction of annual global PAH emissions, with most of the emissions from developing countries such as India and China (Aramandla et al., 2011). In developed countries, emissions from industries and fossil fuel combustion are considered to be the major sources of PAHs (Aramandla et al., 2011). While the emission studies alone provide emissions rates of PAHs at country or regional level, they cannot be used to directly deduce the contributions of different sources to ambient PAH concentrations due to spatiotemporal variation of the emissions and regional transport and transformation of emitted PAHs in the atmosphere (Inomata et al., 2012).

Traditionally, receptor-oriented source apportionment techniques have been applied to determine contributions of different sources to the observed PAH concentrations. The most commonly applied techniques including Chemical Mass Balance (CMB) (Yang and Chen, 2004), Principle Component Analysis (PCA) (Dong and Lee, 2009; Li et al., 2006; Ma et al., 2011; Mai et al., 2003; McDonough et al., 2014; Mishra et al., 2016), Positive Matrix

Factorization (PMF) (Callen et al., 2014; Xie et al., 2014), and multilinear regression (Harrison et al., 1996; Masiol et al., 2012). Comparison studies showed that these receptor-oriented methods generally give comparable PAH source apportionment results (Larsen and Baker, 2003; Teixeira et al., 2015). In addition, some studies used carbon isotope dating to differentiate contributions from biomass burning to fossil fuel combustion (Mandalakis et al., 2005; Sheesley et al., 2009; Zencak et al., 2007). While the molecular diagnostic ratio (MDR) technique has been applied in several studies (Fu et al., 2010; Schiffman and Boving, 2015; Zhang et al., 2005), many PAH MDRs vary significantly in space and time due to different reaction rates in the atmosphere and the estimates were not valid source markers (Amador-Munoz et al., 2013; Katsoyiannis et al., 2011; Tobiszewski and Namiesnik, 2012). Major sources identified as contributing to ambient PAH concentrations include open burning of agriculture residuals (Chen et al., 2008), gasoline and diesel vehicles (Larsen and Baker, 2003), coal combustion (Chen et al., 2011), wood combustion (Piazzalunga et al., 2013) and industrial sources such as refineries (Motelay-Massei et al., 2007).

The receptor-oriented techniques are useful in that they are based on observations and do not need information on emissions and detailed knowledge of physical and chemical processes of PAHs in the atmosphere. However, as the PAH species are not strictly inert, concentrations of the measured PAH concentrations need to be adjusted to compensate for the differential reactive decay of the PAH species before they can be applied in some of the techniques such as CMB and MDR. Such adjustments are difficult to perform without detailed information on the spatial and temporal history of the PAH species. Another limitation of the receptor-oriented methods is that they can only provide source apportionment information in the location where observations are made. To overcome the data gap, regional chemical transport models (CTMs) can be used. Currently, only a few studies used a regional model to determine source contributions of PAHs (San Jose et al., 2013; Zhang and Ying, 2012). In a recent study, Shen et al. (2014) estimated source contributions to lung cancer risk from Benzo[α]pyrene (BaP) exposure using a global chemical transport model. However, they didn't include other possible carcinogenic PAHs in their calculations.

Few studies have been reported in the peer-reviewed literature on PAH concentrations, sources and population health risk in the US, and most published studies (McDonough et al., 2014; Naumova et al., 2002; Schiffman and Boving, 2015; Simcik et al., 1999) focused on PAH concentrations and sources using local scale models but did not evaluate the regional population health risk. As reported in Part I of this two paper series, concentrations of PAHs are high in many places in the US. This makes it necessary to quantify the contributions of different emission sources to the predicted ambient PAH concentrations so that effective emission control strategies can be formulated.

Thus, the objectives of this study are to provide a regional source apportionment of PAHs using a regional chemical transport model and to evaluate the contributions of different sources to excess cancer risk. To our knowledge, this is the first study to provide a national level prediction of PAH source apportionment information and cancer risk assessment in the continental US.

2. Methods

2.1. Modeling PAHs using the CMAQ model

The Community Multiscale Air Quality (CMAQ) model version 5.0.1 developed by the US EPA was expanded to include treatment of emission, reactions, transport, deposition and gas-to-particle partitioning processes to predict the gas and particle phase concentrations of 16 priority PAH concentrations, including the 7 carcinogenic PAH (cPAH) species, Benzo[*a*]anthracene (BaA), Chrysene (CHRY), Benzo[*b*]fluoranthene (BbF), Benzo[*k*]fluoranthene (BkF), Benzo[*a*]pyrene (BaP), Indeno[1,2,3-*cd*]pyrene (IcdP) and Dibenzo[*ah*]anthracene (DahA), and 9 other PAHs, naphthalene (NAPH), acenaphthylene (ACY), acenaphthene (ACE), fluorene (FLU), phenanthrene (PHE), anthracene (ANT), fluoranthene (FTH), pyrene (PYR) and benzo[*ghi*]perylene (B*ghi*P). A complete list of the 16 PAHs and the kinetics and equilibrium parameters are listed in Table 2 of Zhang et al. (2016).

Details of the gas phase reaction, gas-to-particle partitioning and heterogeneous reactions of the PAHs have been discussed in detail in Zhang et al. (2016) and a short summary is provided below. The gas phase reaction mechanism SAPRC-99 (Carter, 2003) was expanded to include reactions of PAHs with hydroxyl radical (OH) and ozone (O₃). All gas phase reactions were considered as second order reactions and further reactions of the intermediate reaction products were not considered except for NAPH. Reaction products of NAPH were represented using the SAPRC-99 common intermediate products, following that of Zhang and Ying (2012). Heterogeneous reactions of particle-bound PAHs with O₃ were also modeled as second order reactions, with reaction rate coefficients taken from Perraudin et al. (2007). Gas-to-particle partitioning was modeled as equilibrium processes for both adsorption of PAHs on black carbon (BC) and absorption into organic matter (OM). Partitioning coefficients were estimated using the parameterizations proposed by Lohmann and Lammel (2004). Treatment of the temperature dependence of the partitioning coefficients for OM-air was based on the parameterizations for octanol-air (K_{oa}) as suggested by Odabasi et al. (2006). The temperature dependence of the partitioning coefficients for BC-air partitioning was based on the parameters provided by Dachs et al. (2004).

2.2. Incremental lifetime cancer risk

In this study, the incremental lifetime cancer risk (*ILCR*), which represents the probability of developing cancer during a 70-year lifetime continuous exposure, was used as a measure of adverse health effect of PAH exposure. Cancer risk is considered non-negligible where the estimated *ILCR* is $>1 \times 10^{-6}$ – 1×10^{-5} (Health Canada, 2010; US EPA, 2004a), depending on the background cancer risk in the region. The *ILCR* due to inhalation of a mixture of NAPH and cPAHs was considered as additive and was calculated using Eq. (1):

$$ILCR = C_{NAPH} \times UR_{NAPH} + \sum_{i=1}^n (C_{cPAH,i} \times TEF_i) \times UR_{BaP} \quad (1)$$

where n is the number of cPAHs in the mixture ($n = 7$); TEF_i is the toxic equivalence factor (dimensionless) of the i^{th} cPAH in the mixture relative to that of BaP; The product of the concentration of a cPAH and its corresponding TEF value is referred to as the toxic equivalent concentration (TEQ). In this document, the sum of the TEQs of the 7 cPAH species is denoted as TEQ_{cPAH} . C_{NAPH} is the concentration of naphthalene. UR_{NAPH} and UR_{BaP} are the unit risk (UR) (the *ILCR* due to lifetime exposure to 1 unit mass concentration) of inhalation exposure to NAPH and BaP, respectively.

By definition, TEF of BaP is 1.0. The TEF values for the rest of the 6 cPAHs proposed by the US EPA are: 0.1 (BaA), 0.1 (BbF), 0.01 (BkF), 0.001 (CHRY), 1.0 (DahA) and 0.1 (IcdP). It should be noted that these are order of magnitude estimations rather than their actual relative cancer potency (US EPA, 1993). This set of TEF values is referred to as TEF_{EPA1993} in this study. Another set of TEF values were suggested in a separate report by a more recent study of US EPA (2010): 0.2 (BaA), 0.5 (BbF), 0.03 (BkF), 0.1 (CHRY), 6 (DahA) and 0.07 (IcdP). This set of TEFs is referred to as TEF_{EPA2010} in this study. In addition, BghiP and FTH were assigned a TEF value of 0.009 and 0.08, respectively. However, as the relative confidence of these values was low, these two species were not included in the cancer risk assessment.

A wide range of UR_{BaP} has been reported in the literature ($7.7\text{--}98 \times 10^{-5}$ per ng m^{-3} , see WHO (2010) and the references therein). In this study, the UR_{BaP} was taken as 8.7×10^{-5} per ng m^{-3} ($UR_{high,BaP}$), based on the World Health Organization (WHO) recommendations (WHO, 2010), to provide an upper bound estimation of the *ILCR*. The California EPA (CalEPA) uses a lower value of 1.1×10^{-6} (OEHHA, 2009) and the US EPA recommends an even lower value of 0.5×10^{-6} per ng m^{-3} (US EPA, 2013). The average of these two lower bound estimations (8.0×10^{-7} per ng m^{-3} , $UR_{low,BaP}$) was used to provide a lower bound estimation of *ILCR* due to cPAHs. The cancer risk of naphthalene was calculated separately, using a UR value of 3.4×10^{-5} per $\mu\text{g m}^{-3}$ ($UR_{low,NAPH}$), based on the CalEPA recommendation (OEHHA, 2009), to provide a lower bound estimation of the *ILCR* due to NAPH. A higher 95% upper bound estimation of 1.0×10^{-4} per $\mu\text{g m}^{-3}$ ($UR_{high,NAPH}$) was recommended in the draft revision to EPA's risk assessment (US EPA, 2004b) and was used to calculate the upper bound estimation of the *ILCR* due to NAPH in this study.

Excess lifetime cancer cases were calculated by multiplying the *ILCR* at each model grid with the gridded population data. Gridded population data were obtained from Oak Ridge National Laboratory's LandScan database at 1 km (Bright et al., 2009). The 1-km resolution data were re-projected into the same resolution and domain used by the chemical transport model (see Fig. S1).

2.3. Source apportionment of PAHs

Contributions from nine major sectors were determined: 1) point sources associated with electric generation units, 2) motor vehicles, 3) non-road engines, 4) oil and gas processes, 5) residential wood combustion, 6) railway and class 1–3 marine vessels, 7) nonpoint sources, 8) other industrial point sources (point sources excluding power generation units and oil and gas processes), and 9) non-US sources. The non-US sources include emissions from Canada and Mexico as well as emissions from offshore oil and gas production processes. As

contribution of each source to PAH concentrations is generally additive, in each of the source apportionment run, PAH emissions from a single emission sector were included. As the PAH concentrations were low and removing their emissions from other source categories was not expected to significantly affect the oxidant (i.e. O_3 and OH) levels, this brute-force source attribution could represent the actual source contributions of PAHs.

3. Model application

The updated CMAQ model with PAH gas and particle schemes was applied to simulate source contributions to gaseous and particulate PAHs in the US. Details of the model setup and input data preparation can be found in Part I of the paper series (Zhang et al., 2016) and only a short summary is provided below. The model domain used in this study is 36×36 km² in horizontal resolution with 18 stretching vertical layers that reach a model top of approximately 20 km above surface. The meteorological inputs were generated using the Weather Research and Forecasting (WRF) model version 3.6. Meteorological data from the North American Regional Reanalysis (NARR) and soil moisture data from the North American Land Data Assimilation System (NLDAS) were used to provide initial and boundary conditions for the WRF simulations. The gridded monthly anthropogenic emissions were generated based on the National Emission Inventory (NEI) 2011. PAH emissions were generated from volatile organic compounds (VOCs) and PM_{2.5} emissions using source specific PAH speciation profiles assembled from the US EPA's SPECIATE and other profile data bases. Biogenic emissions were generated using the Model of Emissions of Gases and Aerosols from Nature (MEGAN) (Guenther et al., 2006, 2012).

Base case simulations were conducted for January, April, July, and October 2011. These four months were chosen to represent typical emission and meteorological conditions in winter, spring, summer and fall, respectively. The average concentrations of the four months were considered as the annual average concentrations. To test whether the four-month average can represent the annual average concentrations, observations of PAHs at all 12 months in 2011 were extracted. Only CHRY and BbF concentrations at selected sites were complete enough (at least four observations were above method detection limit per month, and all 12 month averages were available) to calculate an annual average. As shown in Fig. S2, the 4-month and 12-month average concentrations show strong correlations ($R^2 = 0.842$ and slope = 0.851). Removing two data points from an Illinois site leads to much stronger correlation ($R^2 = 0.952$, slope = 1.011). This suggests that it is viable to use the four representative months to estimate the annual average in long term simulations.

Source apportionment simulations were conducted for January and July only in order to reduce the number of simulations. For a given PAH species, annual source contributions from the nine source sectors were estimated by linear combination of the monthly source apportionment results. The weighting factor w for each source in January and July was determined by minimizing the objective function Q as shown in Eq. (2):

$$Q = \sum_i^{N_x} \sum_j^{N_y} \left[C_{\text{annual}}(i, j) - \sum_{k=1}^{N_s} \sum_{m=1}^2 w_{m,k} C_{m,k}(i, j) \right]^2 \quad (2)$$

where $C_{\text{annual}}(i, j)$ is the predicted annual concentration at grid cell (i, j) from the base case simulations. N is the total number of sources ($N=9$); k is the source index; m is the month index (1 = January and 2 = July); $C_{m,k}$ represents the predicted monthly average concentration for month m and source k from the source apportionment simulations; and w 's are month and source specific weighting factors to be determined. N_x and N_y are number of grid cells in the east-west and north-south directions in the model domain, respectively. There are $2 * N_s$ unknown weighting factors in Eq. (2), and can be determined using a linear least-squares solver (e.g. the `lsqin` function in MATLAB), under the constraints that $w > 0$. Once the weighting factors were determined, $C_{\text{annual},k}$, the annual source contributions to source type k in grid cell (i, j) were determined by Eq. (3):

$$C_{\text{annual},k}(i, j) = \sum_{m=1}^2 w_{m,k} C_{m,k}(i, j) \quad (3)$$

4. Results and discussion

The capability of the model in reproducing the observed PAH concentrations has been evaluated in Part I of the paper series, and is briefly summarized in the following. Predicted daily PAH concentrations at 61 air toxics monitoring sites generally agreed with observations. The best model performance was obtained at rural sites, with an average mean fractional bias (MFB) of -0.03 . Concentrations at suburban and urban sites were underestimated with overall $\text{MFB} = -0.57$. The under-prediction of PAH in urban area due to coarse grid resolution has been noticed in other studies as well. For individual species, the performance of the cPAHs is generally better than the performance of the other PAHs. The following discussion focuses on source contributions to PAH concentrations and their cancer risk in the United States.

4.1. ILCR and excess cancer cases

Fig. 1 shows the gridded ILCR due to NAPH and cPAHs based on Eq. (1), and the predicted excess number of lifetime cancer cases. A country mask file was used to select only grid cells in the continental US in the analyses. The *ILCR* was calculated using the average of the UR_{high} and UR_{low} , and annual average concentrations from the base case simulation. For the cPAHs, the two sets of TEQs were calculated using the $\text{TEF}_{\text{EPA1993}}$ and $\text{TEF}_{\text{EPA2010}}$, and the average TEQ values were used in the calculation for the *ILCR* due to cPAHs. The overall TEQ_{cPAH} values estimated by the $\text{TEF}_{\text{EPA2010}}$ are on average 70% higher than the overall TEQ values estimated by $\text{TEF}_{\text{EPA1993}}$.

The *ILCR* values for NAPH are generally lower than 5×10^{-6} but the *ILCR* values for cPAHs are higher. The entire eastern US and majority of the west coast have *ILCR* values $>5 \times 10^{-6}$, and many urban and industrial areas exceed 1×10^{-5} . The excess cancer cases are highest in the major metropolitan areas: in the west coast, the Greater Los Angeles area and the San Francisco Bay area; in the southern US, Dallas-Fort Worth, Houston and New Orleans; in the east coast, the New York-Philadelphia-Washington areas and in the north, Minneapolis and Chicago. While regional *ILCR* values show a much wider spatial distribution, population density plays an extremely important role in accessing the overall population health impacts. In some areas such as the Mid-West, while the *ILCR* values are high in some places, the low population density leads to low number of excess cancer cases compared to urban areas with similar *ILCR* values. There are approximately 1030 (lower and upper bound estimations: 523–1573) and 4674 (85–9263) cases of excess cancer in the continental US due to exposure to NAPH and cPAHs, respectively, and the overall number of excess cancer cases is 5704 (607–10,800). The lower and upper bound values were estimated using the UR_{low} and UR_{high} values, respectively. The mean, upper and lower bound estimations are likely underestimated because the PAH concentrations were shown to have been underestimated in the current study, especially in urban areas.

Contributions of different cPAHs to *ILCR* and excess cancer cases were also estimated. BaP is the most significant contributor, accounting for ~50% of the *ILCR* and excess cancer cases due to cPAHs, followed by BaA, BbF and DahA, each accounting for ~13–15%. This estimation was based on the average of the $TEF_{EPA2010}$ and TEF_{EP1993} . The contributions of BaP to *ILCR* and excess cancer cases were ~40% and ~70% when $TEF_{EPA2010}$ and $TEF_{EPA1992}$ were used, respectively. Overall cancer risk of cPAH exposure would be underestimated by approximately 30–60% if only BaP was considered. However, using BaP alone would give a reasonable order-of-magnitude estimation of the cancer risk of the seven cPAHs.

4.2. Source contributions to PAHs, *ILCR* and excess cancer cases

Monthly contributions of different sources to individual PAH species and total TEQ of cPAHs are shown in Tables 1 and 2, for January and July of 2011, respectively. To better illustrate the source impact on population health, the source contributions were calculated using population-weighted concentrations of each grid cell.

In January, residential wood combustion is the most significant source of PAHs, with population-weighted contribution of 60.1% for NAPH, 41.3% for cPAHs and 52.1% of the total mass of 15PAH (the sum of the concentrations of all the PAHs except NAPH). It accounts for approximately 30% of the TEQ_{cPAH} , regardless of the TEF values used. The other industrial point sources are also another equally important source of PAHs, especially for the ones associated with cancer risk. Its contribution to NAPH was only 5.3% but it accounted for ~30% of the population-weighted TEQ_{cPAH} . Another important source of PAHs is motor vehicles. It is equally important for overall PAHs (16.7% for 15PAH, 12.9% for NAPH) and toxic PAHs (~18–19% for TEQ_{cPAH}). Non-road engines account for a small fraction (4.1%) of NAPH but higher fraction of toxic PAHs (10–11% of TEQ_{cPAH}). Contributions from sources outside US to concentrations in the US are small, accounting for

only ~3% of the total TEQ_{cPAH} . The fractional contributions of the non-local sources are higher near the border region in the north with Canada, and in the south with Mexico (see Fig. 3). In July, the importance of residential wood combustion is greatly reduced. Based on the toxicological effects, the other industrial point sources become the most important source, accounting for 40% of $cPAHs$ and approximately half (46–50%) of TEQ_{cPAH} . The non-road engines are the second most important sources of $PAHs$ in July, accounting for approximately 20% of most of the $PAHs$ and TEQ_{cPAH} (~19–20%). In July, contributions of the motor vehicle sources to smaller $PAHs$ are higher (~20%) than large $PAHs$ (~10%). It accounts for ~11–12% of the TEQ_{cPAH} . Contributions of oil and gas processes to PAH exposure is approximately 6% in both months. Regional distributions of the TEQ_{cPAH} concentrations are shown in Fig. S3.

To estimate the annual source contributions to $ILCR$ and excess cancer cases, Eqs. (2) and (3) were applied for TEQ_{cPAH} . Two sets of calculations were done for $TEQs$ using $TEF_{EPA1993}$ and $TEF_{EPA2010}$, respectively. The resulted TEQ_{cPAHs} were averaged to calculate the $ILCR$. As low concentrations of TEQ_{cPAH} pose low cancer risk, only points with annual average concentration $> 1 \times 10^{-2} \text{ ng m}^{-3}$ (which leads to an $ILCR$ of $< 1 \times 10^{-6}$) were included in the linear regression. Fig. 2 shows that the fitted annual average concentrations of TEQ_{cPAH} using the weighting factors from Eq. (2) agree well ($R^2 = 0.935$) with the annual average concentrations even though the concentrations cover a range of more than two orders of magnitude. Similar calculation was done for $NAPH$ (cut off concentration is $0.01 \mu\text{g m}^{-3}$) and good agreement between fitted and base case results was also obtained ($R^2 = 0.845$). The weighting coefficients were used to estimate annual source contributions to TEQ_{cPAH} and $NAPH$ using Eq. (3).

Using the estimated annual average concentrations of TEQ_{cPAH} from each source, the regional distributions of $ILCR$ due each source were calculated using the average of the UR_{high} and UR_{low} , and the $ILCRs$ due to $NAPH$ and TEQ_{cPAH} were added together to give the total $ILCR$ of each source. The resulting regional distributions of $ILCR$ from all the sources are shown in Fig. 3. The residential wood smoke contributions are mostly located in the Northeast with the high $ILCR$ of $\sim 1\text{--}1.5 \times 10^{-5}$ in Massachusetts, Connecticut and southeast part of Pennsylvania. Transportation and non-road engines have similar spatial distributions with higher $ILCR$ values occurring in major urban areas. Other industrial point sources also have similar distributions as the transportation and non-road engines. However, most of the point sources are located in industrial regions near the urban centers and their contributions to $ILCR$ were much higher, with a maximum $ILCR$ of $\sim 1.0 \times 10^{-4}$. Coastal regions of southeast Texas and Louisiana are one of the areas with high $ILCR$. Impact of the oil and gas production processes covers a very wide spatial region with $ILCR$ much $> 1.5 \times 10^{-5}$ and the maximum $ILCR$ is approximately 6.4×10^{-5} . The areas where $ILCR$ values are high match well with the oil and gas production regions. Impact of PAH emissions from Canada and Mexico can be as high as 1.1×10^{-4} but are usually limited to border regions. The largest impact is in the northeast region with significant emissions from Canada, but the Canadian impact might have been overestimated since emissions were based on a 2005 inventory. The contribution to $ILCR$ due to point sources related with energy generation units is low in most areas except a location in central Texas. An examination of the emission inventory reveals high emissions of the particulate $cPAH$ emissions in that grid cell while

emissions of other particulate species appeared normal. It appears that a speciation profile associated with that should have unusual high fractions of PAHs in it. More detailed examination of the speciation profiles for the energy generation units should be carried out to further investigate this.

Table 3 shows the estimated source contributions to excess cancer cases in the US from exposure to NAPH and cPAHs. Other industrial point sources top the source contributions with 1768 (102–3425) cases (~ 31%) attributed to them, among which 142 (72–212) cases are attributed to NAPH and 1626 (30–3223) cases are due to cPAHs. Non-road mobile engines are the second largest contributor to excess cancer cases, with 1061 (63–2059) excess cancer cases (18.6%). Contributions of residential wood combustion (925 cases, 148–1702) are similar to that of transportation related sources (767 cases, 108–1426). The PAH emissions from oil and gas production processes, although contributing to large *ILCR* regionally, are only responsible of 4.3% (413 cases, 53–773) of the excess cancer cases, which is similar to the contributions of non-US sources (6.8%) and non-point sources (7.2%). The power generation units have the minimal impact on excess cancer risk, with contributions of approximately 2.3%.

The importance of using population-weighted exposure for source apportionment of PAH health effect is most obvious for the oil and gas processes. As shown in Fig. 3, emissions from oil and gas processes accounted for a very significant fraction of the *ILCR* due to cPAHs in both January and July (30–35%), and cover a very wide spatial region where oil and gas productions are located. However, as the population density in these regions is typically low, the excess cancer cases due to oil and gas processes were much lower than residential wood combustion, other industrial point sources, motor vehicles and non-road engines. However, the lower contributions should not be interpreted as a negligible impact since emissions from oil and gas processes still pose significant health risk to local communities near these industries.

5. Conclusions

The cancer risk in the continental US due to inhalation exposure of outdoor NAPH and seven cPAHs was predicted to exceed 1×10^{-5} of *ILCR* in many areas. It was estimated that exposure to PAH led to 5704 (608–10,800) excess cancer cases. Major sources that contributions to *ILCR* and excess cancer cases are industrial point sources, on-road vehicles, residential wood combustion and oil and gas industries. Point sources not related with energy generation and the oil gas industry account for approximately 31% of the excess cancer cases, followed by non-road engines with 18.6% contributions. Contributions of residential wood combustion (16.2%) are similar to that of transportation (mostly motor vehicles with small contributions from railway and marine vessels) related sources (13.4%). The oil and gas industry emissions, although contributing to high concentrations of cPAHs regionally, are only responsible of 4.3% of the excess cancer cases, which is similar to the contributions of non-US sources (6.8%) and non-point sources (7.2%). The power generation units pose a minimal impact on excess cancer risk, with contributions of approximately 2.3%.

Results of PAH source apportionment and risk assessment can be greatly affected by the accuracy of the base case model results. While it will be ideal to be able to estimate the uncertainty in the predicted PAH concentrations, it is not possible in this study because limited data for detailed, speciated PAH measurements from emission sources. Other sources of uncertainty in the cancer risk estimation are the uncertainty in the UR and the TEQ of other the cPAHs. The UR of BaP have two orders of magnitude variations and TEQ of other cPAHs also have a factor of 10 difference in the reported values. These uncertainties are possibly larger than uncertainties in the predicted emissions and concentrations, as shown by the detailed model vs. prediction comparisons described in Part I. The uncertainty in the predicted concentrations might not greatly change the range of the estimated health risk as uncertainties in UR and TEQ were considered in the calculation. Thus the numbers reported in this study can provide useful information for policy makers and stakeholders on the potential risk of PAH exposure in the US.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Fig. 1. Incremental lifetime cancer risk (*ILCR*) due to (a) NAPH, (b) toxic equivalent concentration (TEQ) of seven cPAHs (TEQ_{cPAH}) and (c) NPAH + TEQ_{cPAH} , and their corresponding excess cancer cases (d–f). Note that in all panels the upper limits in the color bar were chosen to better illustrate spatial distributions.

Fig. 2. Fitted annual average concentrations of TEQ_{cPAH} (a) and NAPH (b) using January and July concentrations from nine sources. The dashed lines represent 1.5:1, 1:1, and 0.5:1 ratios.

Fig. 3. Source contributions to incremental lifetime cancer risk. Note that the upper limit in the color bar was chosen to better illustrate spatial distributions. EGU (point sources of electric generation units), NPT (non-point sources), NRD (non-road engines), OGS (oil and gas processes), OPT (other industrial point sources), RWC (residential wood combustion) and OTH (non-US sources; also includes all non-US C3 CMV and offshore oil production processes). The TRP (transportation) source is the sum of motor vehicles, railways and class 1–3 marine vessels.

Table 1

Population-weighted source contributions to PAHs in the continental United States for January 2011.

	C123	EGU	MVH	NPT	NRD	OGS	OPT	RWC	OTH
NAPH	1.1%	0.3%	12.9%	7.0%	4.1%	0.1%	5.3%	60.1%	9.1%
ACE	1.3%	0.8%	17.2%	2.0%	8.4%	1.7%	27.3%	34.6%	6.8%
ACY	0.4%	0.0%	10.9%	0.2%	4.2%	0.3%	5.7%	75.8%	2.4%
FLU	1.5%	0.1%	19.8%	0.3%	9.9%	0.2%	5.4%	51.1%	11.7%
PHE	1.3%	0.1%	15.1%	0.6%	5.9%	1.1%	11.4%	55.2%	9.4%
ANT	1.6%	0.1%	14.3%	0.8%	6.9%	0.2%	8.7%	64.2%	3.2%
FTH	2.2%	0.2%	15.7%	2.0%	8.1%	0.2%	12.9%	53.5%	5.2%
PYR	3.0%	0.1%	20.9%	1.3%	10.7%	2.0%	12.2%	44.9%	4.8%
CHRY	2.0%	0.2%	15.6%	3.0%	8.8%	0.5%	14.1%	52.0%	3.8%
BaA	2.5%	0.1%	16.0%	1.5%	9.2%	2.4%	30.5%	35.1%	2.6%
BbF	0.6%	0.4%	16.5%	3.4%	9.5%	0.0%	14.2%	52.0%	3.5%
BkF	0.1%	0.3%	17.2%	1.7%	9.5%	0.0%	17.9%	49.0%	4.3%
BghiP	0.0%	0.1%	27.1%	0.5%	16.6%	13.0%	26.4%	14.3%	2.0%
BaP	1.2%	0.1%	19.4%	1.4%	11.3%	7.7%	31.0%	25.9%	2.0%
IcdP	0.2%	0.2%	30.6%	1.1%	18.1%	0.4%	9.1%	37.4%	3.0%
DahA	1.4%	1.3%	14.6%	25.9%	9.6%	0.0%	39.6%	0.0%	7.5%
15PAH	1.5%	0.1%	16.7%	0.9%	7.9%	1.3%	12.3%	52.1%	7.2%
cPAH	1.4%	0.2%	18.1%	2.3%	10.4%	2.0%	21.2%	41.3%	3.2%
TEC _{cPAH} (low)	1.3%	0.2%	19.1%	2.5%	11.1%	5.8%	29.0%	28.5%	2.5%
TEC _{cPAH} (high)	1.3%	0.4%	17.6%	5.3%	10.3%	3.6%	27.1%	31.0%	3.3%

C123: (locomotives and class 1–3 marine vessels), EGU (point sources of electric generation units), MVH (motor vehicles), NPT (non-point sources), NRD (non-road engines), OGS (oil and gas processes), OPT (other industrial point sources), RWC (residential wood combustion) and OTH (non-US sources; also includes all non-US C3 CMV and offshore oil production processes). 15PAH is the sum of the concentrations of all the PAHs except NAPH. cPAH is the sum of the concentrations of the seven carcinogenic PAHs (cPAHs).

Table 2

Population-weighted source contributions to PAHs in the continental United States for July 2011.

	C123	EGU	MVH	ARE	NRD	OGS	OPT	RWC	OTH
NAPH	3.0%	0.9%	23.9%	15.1%	13.9%	0.2%	13.5%	3.7%	25.8%
ACE	1.9%	1.0%	14.2%	1.9%	15.5%	1.6%	43.1%	1.2%	19.5%
ACY	2.0%	0.1%	24.1%	0.5%	19.8%	1.2%	25.1%	5.9%	21.2%
FLU	2.7%	0.2%	20.3%	0.3%	23.3%	0.2%	9.2%	2.6%	41.2%
PHE	2.8%	0.1%	20.1%	0.7%	15.5%	1.4%	22.5%	2.7%	34.2%
ANT	4.7%	0.2%	25.7%	1.4%	26.0%	0.4%	22.8%	4.6%	14.1%
FTH	4.6%	0.3%	22.2%	1.9%	24.6%	0.3%	24.9%	3.3%	17.8%
PYR	5.2%	0.2%	23.4%	1.3%	26.0%	2.8%	23.0%	2.4%	15.6%
CHRY	4.7%	0.4%	19.4%	3.2%	25.9%	0.7%	28.6%	4.0%	13.1%
BaA	4.1%	0.2%	15.2%	1.3%	20.1%	3.0%	46.3%	2.1%	7.7%
BbF	1.5%	0.8%	14.8%	3.4%	26.7%	0.0%	31.4%	4.6%	16.8%
BkF	0.2%	0.7%	11.5%	1.7%	22.0%	0.0%	36.6%	3.8%	23.4%
BghiP	0.1%	0.1%	9.7%	0.4%	24.9%	10.5%	49.7%	0.6%	4.0%
BaP	1.7%	0.2%	10.3%	1.0%	18.0%	7.6%	53.9%	1.2%	6.1%
IcdP	0.4%	0.3%	18.6%	1.4%	43.5%	0.6%	19.1%	3.0%	13.2%
DahA	1.8%	1.3%	8.0%	8.8%	14.5%	0.0%	45.5%	0.0%	20.2%
15PAH	3.1%	0.3%	19.5%	1.1%	20.7%	1.8%	25.4%	2.7%	25.5%
cPAH	2.8%	0.4%	14.9%	2.0%	23.4%	2.6%	39.8%	2.8%	11.4%
TEQ _{cPAH} (low)	2.0%	0.3%	11.3%	1.6%	19.3%	5.9%	50.2%	1.5%	7.8%
TEQ _{cPAH} (high)	2.2%	0.5%	11.8%	3.0%	19.6%	3.8%	46.2%	1.8%	11.1%

C123: (locomotives and class 1–3 marine vessels), EGU (point sources of electric generation units), MVH (motor vehicles), NPT (non-point sources), NRD (non-road engines), OGS (oil and gas processes), OPT (other industrial point sources), RWC (residential wood combustion) and OTH (non-US sources; also includes all non-US C3 CMV and offshore oil production processes). 15PAH is the sum of the concentrations of all the PAHs except NAPH. cPAH is the sum of the concentrations of the seven carcinogenic PAHs (cPAHs).

Table 3

Excess lifetime cancer cases in the United States due to NAPH and cPAH. Transportation source (TRP) is the sum of C123 and MVH sources.

Source	NAPH			cPAH			Total		
	UR _{low}	Mean	UR _{high}	UR _{low}	Mean	UR _{high}	UR _{low}	Mean	UR _{high}
EGU	54.1	106.5	159.0	0.5	27.5	54.5	54.6	134.0	213.5
NPT	46.7	92.0	137.3	5.8	320.8	635.8	52.5	412.8	773.1
NRD	45.0	88.7	132.3	17.7	972.1	1926.5	62.7	1060.8	2058.9
OGS	8.2	16.1	24.0	4.2	231.6	458.9	12.4	247.7	483.0
OPT	72.1	142.2	212.2	29.6	1626.4	3223.2	101.8	1768.6	3435.3
RWC	135.5	267.0	398.5	12.0	657.5	1303.0	147.5	924.5	1701.5
TRP	97.3	191.7	286.2	10.5	575.0	1139.5	107.8	766.7	1425.7
OTH	63.8	125.8	187.7	4.8	263.1	521.5	68.6	388.9	709.2
Total	522.7	1030.0	1537.3	85.2	4674.0	9262.8	607.9	5704.0	10,800.1