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The influence of the Clean Air Actions on the health risk of atmospheric polycyclic aromatic hydrocarbons

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Abstract

Polycyclic aromatic hydrocarbons (PAHs) are widespread in the atmosphere and pose significant health risks. While China's Clean Air Actions have effectively reduced atmospheric PAH concentrations, the effects of these measures on health risks associated with PAH exposure remain unclear. This study analyzed the temporal trends in the health risks of 15 PAHs in Harbin, China, from June 2014 to May 2019, focusing on benzo[a]pyrene toxicity equivalents (BaP_{eq}) and cancer risks (*CR*). Over five years, the concentrations of total, particle, and gas-phase ΣBaP_{eq} decreased, with halving times of 3.80 ± 1.34, 3.80 ± 1.34, and 2.71 ± 2.67 years, respectively. Unlike the steady decline in total PAH concentrations, BaP_{eq} showed an increase followed by a peak in the third year before decreasing. *CR* values for inhalation and dermal exposure also declined but remained above the safety threshold of 1×10^{-6} for all age groups, indicating potential cancer risks. Multiple linear regression showed only 38% of BaP_{eq} reduction was due to emissions control, compared to 65% for total PAHs, as highly toxic PAHs were less affected. These findings emphasize the need for targeted actions on highly toxic PAHs in the future.

Keywords: *BaP* toxicity equivalent, cancer risk, temporal trend, Monte Carlo Simulation, Clean Air Action

INTRODUCTION

Polycyclic aromatic hydrocarbons (PAHs), characterized by carbon-hydrogen structures containing two or more fused aromatic rings, have garnered significant attention in recent decades[1]. PAHs are ubiquitously present in the atmosphere, primarily originating from the incomplete combustion or pyrolysis of various organic materials, such as forest fires and the inefficient burning of carbon-rich fuels[2]. These compounds are globally recognized for their carcinogenic, mutagenic, and allergenic properties, prompting international regulatory efforts to mitigate their emissions[3,4]. Notably, in 1976, the United States Environmental Protection Agency (U.S. EPA) identified 16 unsubstituted PAHs as priority pollutants for environmental pollution control[5]. Among them, benzo[a]pyrene (BaP), a highly toxic compound, has been designated as a key control pollutant in the Chinese Ambient Air Quality Standards (GB 3095-2012)[6]. Consequently, the occurrence and potential health risks of PAHs have become critical global concerns for public health and environmental protection.

Advances in pollution control technologies have significantly reduced PAH emissions from major anthropogenic sources, such as coal combustion and vehicular emissions, resulting in declining atmospheric concentrations. For example, the halving time of Σ15PAHs was reported to be 5.9 years in the U.K. (1991-2005)[7] and approximately 9 years in Chicago (1998-2003)[8]. In Harbin, China, a shorter halving time of 3.23 ± 0.37 years for Σ15PAHs was observed, reflecting the effectiveness of stringent emissions control measures under the Clean Air Actions[9]. These actions contributed to a 65% reduction in atmospheric PAH concentrations, demonstrating their substantial impact. The decline in PAH concentrations is expected to reduce associated health risks, highlighting the benefits of the Clean Air Actions.

However, assessing health risks from PAHs remains challenging due to the varying toxicities of different congeners. For example, the U.S. EPA classifies benzo[a]pyrene, benzo[a]anthracene, benzo[b]fluoranthene, benzo[k]fluoranthene, chrysene, dibenzo[a,h]anthracene, and indeno[1,2,3-cd]pyrene as probable human carcinogens (Group 2B)[10]. The toxic equivalency factors (TEFs)[11], relative to BaP, vary significantly among PAHs, suggesting that the temporal trends of health risks posed by the PAH mixture may differ from their total concentrations. However, the trends in PAH-related health risks remain unclear, necessitating a comprehensive evaluation of the impact of emissions controls.

This study analyzed the temporal trends in the toxicity equivalence (BaP_{eq}) of 15 PAHs in Harbin, northeastern China, from June 2014 to May 2019. Cancer risks across different age groups were also assessed using monitoring data and Monte Carlo simulations. By examining the contributions of anthropogenic emissions controls and meteorological factors, this research provides critical insights into the effectiveness of the Clean Air Actions in reducing PAH-related health risks and protecting public health.

METHODS

Data information

The atmospheric PAH concentrations used for the health risk assessment were cited from our previous study[9]. Briefly, 15 PAHs in both gas and particle phases were measured in Harbin City from June 2014 to May 2019. Detailed information about these 15 PAHs can be found in our previous study and is summarized in Supplementary Table 1 of the Supporting Information. The measured concentrations were used to calculate the BaP equivalent (BaP_{eq}) concentration and assess cancer risks. Meteorological variables, including temperature (TEM, °C), wind speed (WIN, m/s), relative humidity (RHU, %), and precipitation (PRE, mm), were sourced from the NCEP/NCAR Reanalysis Dataset (National Center for Environmental Prediction/National Center for Atmospheric Research Reanalysis 1, 2014)[9]. More detailed information about the data was recorded in Supplementary Texts 1 and 2.

Health risk assessment method

Firstly, the toxicity of the 15 PAHs was evaluated by their corresponding toxicity equivalence (BaP_{eqi}, ng·m^{-3}), which was calculated by the following equation[11]:

$$BaP_{eqi} = C_i \times TEF_i \quad (1)$$

where C_i is the atmospheric concentration of PAH i (ng·m^{-3}); TEF_i is the toxicity equivalence factor corresponding to PAH i [dimensionless, Supplementary Table 1].

The total toxicity equivalent BaP_{eq} (ng·m^{-3}) of the 15 PAHs is calculated by summing the BaP_{eqi} of the 15 PAHs:

$$BaP_{eq} = \sum BaP_{eqi} \quad (2)$$

Secondly, the cancer risk (*CR*) of atmospheric PAHs was evaluated by two exposure ways: inhalation exposure and dermal contact exposure. The *CR* values from the two exposure ways (inhalation exposure: CR_i and dermal contact exposure CR_d) were calculated based on the following equations:

$$CR_i = \frac{BaP_{eq} \times IR \times EF \times ED \times CSF_i \times ADAF}{BW \times AT \times 10^6} \quad (3)$$

$$CR_d = \frac{BaP_{eq}^P \times SA \times AF \times EV \times ABS_d \times EF \times ED \times CSF_d \times ADAF}{BW \times AT \times 10^{-1}} \quad (4)$$

where *IR* is inhalation rate (m^3·day^{-1}); *EF* is the outdoor exposure factor (dimensionless); *BW* is the body weight (kg); BaP^P_{eq} is the sum of BaP_{eqi} for particulate PAHs (ng·μg^{-1}), which was calculated by dividing the concentrations in the particle phase (ng·m^{-3}) by the total suspended particles (TSP, μg·m^{-3}); *SA* is the surface area that can be exposed, including head and hand (m^2); *AF* is the amount of particles attached to the dermal (mg·cm^{-2}·event^{-1}); *EV* is the event frequency (event·day^{-1}); ABS_d is the dermal absorption fraction (dimensionless) of PAHs. *ED* is the exposure duration (year). *AT* is the averaging time for carcinogens (70 years). CSF_i and CSF_d are the cancer slope factors for inhalation exposure and dermal contact exposure, respectively (kg·day·mg^{-1}). *ADAF* is the age-dependent adjustment factor in cancer slope factors (dimensionless).

The total *CR* is calculated as the sum of the *CR* values of the two exposure pathways, as follows:

$$CR = CR_i + CR_d \quad (5)$$

The exposure factors used for the following three age groups, namely adults (18 to 70 years), adolescents (12 to 17 years), and children (1 to 11 years), were cited from the specific exposure factors for the Chinese population[12,13]. The specific exposure factor values for these three age groups are provided in Supplementary Table 2. The reference criteria commonly used in previous studies[14,15] are applied in this study: *CR* value below 1 × 10^{-6} indicates a negligible cancer risk; *CR* value between 1 × 10^{-6} and 1 × 10^{-4} signifies a potential cancer risk, and when the *CR* value exceeds 1 × 10^{-4}, it indicates a higher cancer risk.

Temporal trends analysis method

In this study, a harmonic regression method[16,17] was used to express the temporal trends and calculate the halving times of the concentrations of BaP_{eq}. The natural logarithms of the concentrations of BaP_{eq} were used in the following equation:

$$\ln C = a_0 + a_1 sin(zt) + a_2 \cos(zt) + a_3 t \quad (6)$$

where C is the concentrations of BaP_{eq}, $z = 2\pi/365.25$, t is Julian days starting from 1 January 2014, a_0 is an intercept that rectifies the units, a_1 and a_2 are harmonic coefficients, and the seasonal variations of the concentrations can be expressed as $(\sqrt{(a_1^2 + a_2^2)})$, and a_3 is a first-order rate constant, and can be used to calculate halving time ($t_{1/2} = -\ln(2)/365.25a_3$). These values of a_0, a_1, a_2, and a_3 were derived through the application of the regression method utilizing the MATLAB software.

Uncertainty and sensitivity analysis method

In the present study, to evaluate the uncertainty of the exposure factors, the probability distributions for exposure factors are applied [Supplementary Table 3]. Subsequently, the Monte Carlo Simulation is employed to quantitatively predict the cumulative density function of *CR* values through probabilistic simulations. This process is iterated 10,000 times to estimate cancer risk based on the distribution functions of input factors. Furthermore, the sensitivity of each variable was evaluated by determining rank correlation coefficients between inputs and outputs throughout the simulations (*CR*). Both the Monte Carlo Simulation and sensitivity analysis are performed using the Crystal Ball software (Version 11.1).

Quantification of contribution to the temporal trend

To distinguish and quantify the respective contribution of the anthropogenic emissions control and the meteorological factors to the temporal trends of BaP_{eq}, a stepwise multiple linear regression (MLR) model was applied. Initially, deseasonalized and detrended time series were derived by subtracting the three-month moving averages from the monthly mean data. This approach emphasizes synoptic-scale variability while minimizing interference from typical seasonal patterns and long-term trends across variables[18,19].

$$y_d = \sum \beta_i x_{d,i} + b \quad (7)$$

where y_d is the deseasonalized and detrended monthly BaP_{eq} mean time series, and $x_{d,i}$ is the deseasonalized and detrended meteorological variable i. The corresponding regression coefficient and the intercepts were obtained from the regression fitting, stepwise, adding and deleting terms based on their independent statistical significance to obtain the best model fit[19]. Here, the MLR model was used to remove the effect of meteorological variability from the BaP_{eq} trends, not only including the monthly synoptic-scale variability but also concluding any inter-annual variability and 5-year trends. This makes the standard assumption that the same factors that drive synoptic-scale variability also drive inter-annual variability[20]. To calculate the meteorological anomalies $x_{a,i}$, the three-month moving averages from the monthly mean time series were calculated first. Then, the mean concentrations of the three-month moving averages for each season during the five years were calculated. Finally, the meteorological anomalies were obtained by removing these means from the monthly mean time series. The anomalies calculated in this manner are deseasonalized but not detrended. The meteorology-driven BaP_{eq} anomalies can be obtained:

$$y_m = \sum \beta_i x_{a,i} + b \quad (8)$$

Then, the residual (y_r) that cannot be explained by the MLR meteorological model can be calculated by removing meteorological influence from the MLR model:

$$y_r = y_a - y_m \quad (9)$$

where y_a is the BaP_{eq} anomalies obtained by deseasonalizing but not detrending the PAHs data like $x_{a,i}$.

RESULTS AND DISCUSSIONS

Temporal trends of ΣBaP_{eq}

The concentrations of ΣBaP_{eq} in different phases are shown in Figure 1 and summarized in Supplementary Table 3. The mean concentration of ΣBaP_{eq} in the total phase was 7.83 ± 12.2 ng·m^{-3} over the five-year period from June 2014 to May 2019 in Harbin City. The concentration of ΣBaP_{eq} was at the same level as the results of 11 cities in China in 2008 (mean values: 8.45 ± 14.1 ng·m^{-3})[21]. Furthermore, similar to the decreasing temporal trend of the total concentration of Σ_{15}PAHs[9], the concentrations of ΣBaP_{eq} in the total phase also exhibited a declining trend from May 2014 to June 2019 [Figure 1A]. A decreasing but not significant trend ($P > 0.05$) of ΣBaP_{eq} was observed in Harbin City, with the halving time of 3.80 ± 1.34 years. As summarized in Supplementary Table 3, an increasing and then decreasing trend with the peak in the third year (June 2016 to May 2017) was observed in the annual mean concentrations of ΣBaP_{eq} in the total phase, with values of 8.20 ± 12.3, 9.61 ± 15.7, 9.60 ± 12.8, 7.88 ± 12.2, and 3.54 ± 3.90 ng·m^{-3} for the five years, respectively. In addition, the halving time of ΣBaP_{eq} in Harbin City was longer than that of Σ15PAHs, which was calculated in our previous study as 3.23 ± 0.370 years[9]. These results suggest different temporal trends between the concentration and toxicity of the total PAHs, which could be attributed to the different toxicities (or TEFs) of the PAHs. The concentrations of Σ15PAHs continuously decreased, while the concentrations of ΣBaP_{eq} exhibited a delayed response. Furthermore, the longer halving time of ΣBaP_{eq} compared to Σ15PAHs might be attributed to the fact that some PAHs with high toxicity (or TEF) have longer halving times. For example, some PAHs with high toxicity had long halving times in the total phase, such as BaP (TEF = 1, 3.92 ± 0.605 years), BbF (TEF = 0.1, 5.03 ± 0.901 years), BkF (TEF = 0.1, 3.69 ± 0.533 years), and IcdP (TEF = 0.1, 4.18 ± 0.659 years) [Supplementary Table 1][9]. Therefore, it is imperative to pay more attention not only to the concentrations of PAHs but also to their toxicities.

Similar to the decreasing trend of ΣBaP_{eq} in the total phase, clearly decreasing trends of ΣBaP_{eq} were also observed in the gas and particle phases over the five-year period. As shown in Figure 1B and C, a significant decreasing trend was observed for ΣBaP_{eq} in the gas phase ($P < 0.001$) and a non-significant decreasing trend was observed in the particle phases ($P > 0.05$), with halving times of 2.71 ± 2.67 and 3.80 ± 1.34 years, respectively. In addition, similar to the trend in the total phase, an increasing and then decreasing trend of the annual mean concentrations was also observed for ΣBaP_{eq} in the particle phase. As summarized in Supplementary Table 3, the annual mean concentrations of ΣBaP_{eq} (mean ± SD) in the particle phase during the five-year period were 7.99 ± 12.2, 9.39 ± 15.6, 9.47 ± 12.7, 7.71 ± 12.1, and 3.46 ± 3.85 ng·m^{-3}, respectively. Furthermore, the mean concentration of ΣBaP_{eq} in the particle phase (7.67 ± 12.1 ng·m^{-3}) was significantly (Wilcoxon signed rank test, $P < 0.001$) higher than in the gas phase (0.164 ± 0.157 ng·m^{-3}) over the five-year period. The higher carcinogenic potency in the particulate phase compared to the gas phase, which was opposite to the concentration trends between the two phases, should be attributed to the fact that high-molecular-weight (HMW) PAHs with high carcinogenic potency are more easily associated with particles in the atmosphere[22]. For example, the particulate proportion of BaP and DahA, both with TEF = 1, are higher than 0.9[23]. However, the particulate proportion of some low-molecular-weight (LMW) PAHs (e.g., Acy, Ace, Flu, and Phe) is lower than 0.2[23], and their TEFs are quite small (0.0001). The results suggested that the decrease in the particulate PAHs dominated the decrease in BaP_{eq} of the atmospheric PAHs, with the

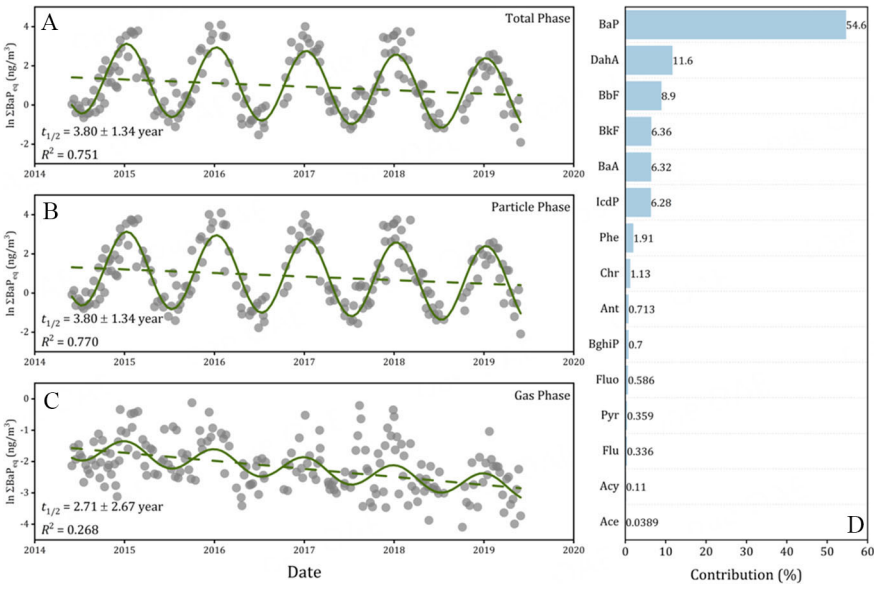

Figure 1. The temporal trends of the total atmospheric concentration of BaP_{eq} for the 15 PAHs in Harbin City during the five-year period for the total phase (gas phase + particle phase) (A), particle phase (B), and gas phase (C) (Note: Solid line represents harmonic regression fitting line; dashed line represents time trend), and the contributions of each PAHs to the total concentration of BaP_{eq} (D).

mean contribution of ΣBaP_{eq} in the particle phase being 82.3%. The dominant role of particulate PAHs, which showed an increasing and then decreasing trend in ΣBaP_{eq}, may also explain the lagging of ΣBaP_{eq} in the total phase.

As shown in Figure 1D, the concentrations of BaP_{eqi} corresponding to each PAH and their contributions to ΣBaP_{eq} were quite different. The mean concentrations of BaP_{eqi} for each PAH in the total phase ranged from 0.00265 ± 0.00432 ng·m^{-3} (Ace) to 4.76 ± 7.61 ng·m^{-3} (BaP). Among the 15 PAHs, BaP (TEF = 1) dominated the total ΣBaP_{eq} of the 15 PAHs (mean contribution: 55%), followed by DahA (TEF = 1, mean contribution: 12%), BbF (TEF = 0.1, mean contribution: 9%), BkF (TEF = 0.1, mean contribution: 6%), and BaA (TEF = 0.1, mean contribution: 6%). Together, these PAHs contributed more than 94% of the total ΣBaP_{eq} and were the main contributors to the carcinogenic efficacy of the 15 PAHs. The main contributions of these HMW PAHs to ΣBaP_{eq} can be attributed to their relatively high toxicity. These PAHs also dominated the temporal trends of the toxicities of the 15 PAHs.

Health risk assessment

The cancer risk was calculated based on ΣBaP_{eq} for the three age groups: child (1-11), adolescent (12-17), and adult (18-70). The trends in cancer risk over the five-year period were consistent with the concentrations of BaP_{eq}. As shown in Figure 2A-C, the annual mean values of the total *CR* showed an increasing and then decreasing trend for the three age groups. For example, the annual mean values of the total cancer risk for children were 3.26 × 10^{-5}, 5.06 × 10^{-5}, 7.53 × 10^{-5}, 4.94 × 10^{-5}, and 2.90 × 10^{-5} during the five-year period, respectively. Although the total *CR* decreased a lot, especially during the last year, the mean values of *CR* during the last year were still higher than the threshold value of 1 × 10^{-6} for all three age groups, indicating a potential cancer risk. Among the different age groups, children had the highest total *CR* values (five-year mean value: 4.66 × 10^{-5}), followed by adults (1.45 × 10^{-5}) and adolescents (5.30 × 10^{-6}). This finding underscores the particular vulnerability of children to atmospheric PAHs, likely due to their low body weight and high *ADAF*. In addition, the cancer risks from dermal contact were higher than those from inhalation, which was consistent with previous studies[14,24]. For example, the mean value of *CR* from dermal

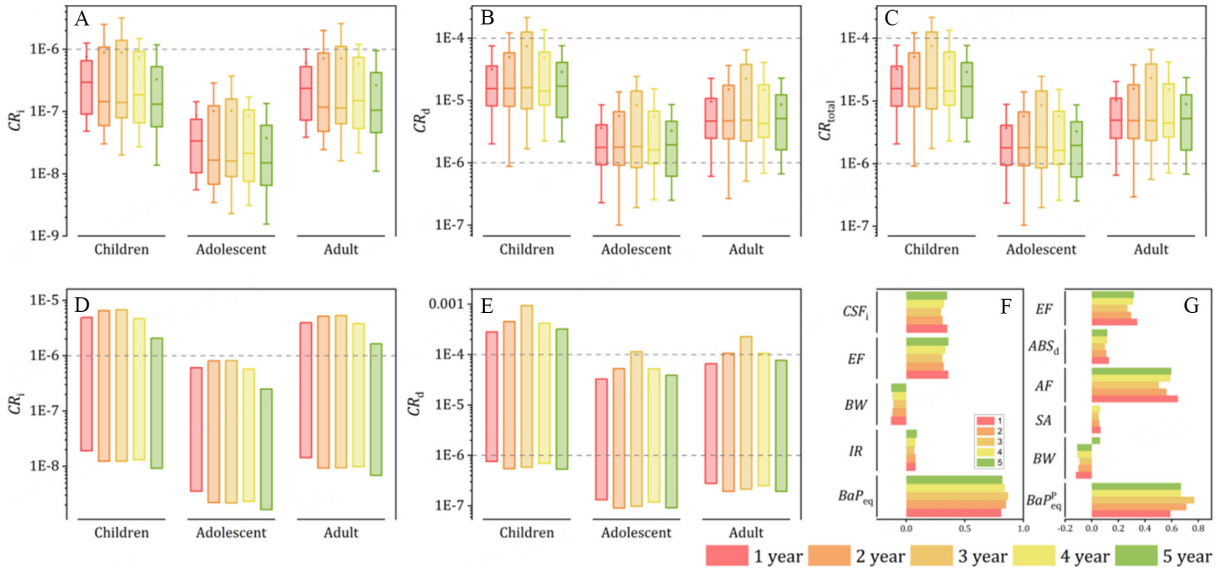

Figure 2. The total *CR* of the 15 PAHs [Note: the *CR* values with inhalation (A), dermal contact (B), and total (C) were calculated based on monitoring data; the *CR* values were estimated based on the Monte Carlo Simulation for inhalation (D) and dermal contact (E), and the sensitivity analysis results of the simulation inhalation cancer risk (F) and dermal contact (G)].

contact for adolescents during the five-year period was 5.22×10^{-6}, nearly two orders of magnitude higher than that from inhalation (8.30×10^{-8}). The higher dermal risk could be attributed to the higher exposure dose. These results suggest that targeted interventions, such as improving personal hygiene practices, reducing direct contact with contaminated surfaces, and enhancing public awareness, could help mitigate dermal exposure risks, especially for vulnerable populations like children. In summary, although a general decreasing trend was observed in the *CR* values for the different age groups, the potential cancer risk associated with atmospheric PAHs still warrants attention, particularly regarding dermal exposure.

Considering the inherent uncertainties of the factors used for calculating *CR*, Monte Carlo Simulation was applied to obtain more statistically robust results. The 5% and 95% values of the *CR* from the two exposure ways for each year, estimated using the Monte Carlo Simulation, are shown in Figure 2D and E. Similar temporal trends to those of the concentrations of ΣBaP_{eq} were also observed in these simulated results. Compared with the calculated results (mean values), the simulated results (mean values) were all higher, with a multiple difference between 1.67 and 12.8, except for dermal contact for adults (where the multiple difference was lower than 0.91). In addition, the difference between the calculated *CR* and the simulated *CR* from dermal contact was larger than that from inhalation. The result might be attributed to more variables in the calculation of dermal contact than inhalation, indicating that to increase the accuracy of the results, efforts should focus on a better definition of probability distributions for these variables. For inhalation, the influence of the concentration of BaP_{eq} was much greater than that of other factors [Figure 2F], which was followed by the variables of CSF_i and *EF*. However, for dermal contact, the influence of the *AF* was comparable to that of BaP^{P}_{eq} in the simulation of *CR* [Figure 2G], which was followed by variables of *EF*. The most influential roles of BaP_{eq} and BaP^{P}_{eq} can be attributed to the fact that the range of these variables is larger than the other variables. Fortunately, the probability distribution functions of atmospheric concentrations were derived from our measurements. Therefore, the result indicated that the probability distributions and variations of the other factors (*EF*, *AF* and CSF_i) need to be carefully considered in order to increase the accuracy of the output *CR*.

Drivers on the temporal trend

The MLR model[9,19] was applied to separate and quantify the contributions of the anthropogenic emissions control and the meteorological factors to the decreasing trend in the concentration of ΣBaP_{eq}. In our previous study, the model was successfully used to analyze their contributions to the concentrations of Σ15PAHs[9]. Specifically, 35% of the decrease in PAH concentration can be attributable to meteorological factors, while the remaining 65% can be attributed to anthropogenic emissions control, demonstrating the effect of the Clean Air Actions on atmospheric PAHs. In the present study, the MLR model was also used to separate and quantify their contributions to the temporal trends of the monthly mean concentrations of Σ BaP_{eq}.

As shown in Figure 3, all three lines (monthly mean ΣBaP_{eq} anomalies in the deseasonalized but not detrended data (*y*a, blue), the meteorological contribution to the ΣBaP_{eq} decreasing trend calculated from the meteorological factors (*y*m, orange blue), and the residual being considered to be caused by anthropogenic emissions control (*y*r, green) showed a decreasing trend, indicating that both anthropogenic emissions control and the meteorological factors had the positive influence on the decrease of ΣBaP_{eq}. The decreasing trend in the concentration of ΣBaP_{eq} in the total phase was 8.46 ± 11.4 $ng \cdot m^{-3} \cdot y^{-1}$, with 5.26 ± 3.79 and 3.19 ± 10.8 $ng \cdot m^{-3} \cdot y^{-1}$ attributed to meteorological factors and anthropogenic emissions control, respectively. The result indicated that 62% of the decrease in ΣBaP_{eq} can be attributed to meteorological factors, while 38% of the decrease can be attributed to anthropogenic emissions control. The different results from that of Σ15PAHs[9] might be due to the fact that the HMW PAHs were lightly affected by the Clean Air Actions, while they were the major composition of ΣBaP_{eq}. As mentioned above, these PAHs had longer halving times (or lower decreasing rates) than that of LMW PAHs[9]. Therefore, it can be concluded that the Clean Air Actions had a greater impact on the decrease of atmospheric Σ15PAHs than on the decrease of ΣBaP_{eq}.

The comparison results indicated the different influences of the Clean Air Actions on the concentrations and the toxicity of atmospheric PAHs, suggesting that more attention should be focused on the PAH congeners with high toxicity. However, other factors could also influence the atmospheric concentrations of PAHs and their associated health risks, such as variations in degradation rates over the sampling period and additional contributing factors. Therefore, a deeper understanding of the temporal trends of atmospheric PAHs should be explored in future studies.

CONCLUSION

To quantify the influence of the Clean Air Actions on the toxicity of the atmospheric PAHs, the temporal trends of BaP_{eq} of the 15 PAHs from June 2014 to May 2019 in Harbin City, China, were conducted. During the five-year period, overall decreasing trends were observed for the concentrations of ΣBaP_{eq} in the total, particle, and gas phases, with halving times of 3.80 ± 1.34, 3.80 ± 1.34, and 2.71 ± 2.67 years, respectively. However, due to the different TEF values of the 15 PAHs, an increasing and then decreasing trend with a peak in the third year (June 2016 to May 2017) was observed for the annual mean concentrations of ΣBaP_{eq}, which differed from the continuously decreasing trend observed in the concentrations of Σ15PAHs. Similar decreasing trends were also observed in *CR* from both inhalation and dermal contact exposure pathways. However, the mean values of the total *CR* remained higher than the threshold value of $1 \times 10^{-6}$for all three age groups, indicating a potential cancer risk. Furthermore, the results from the MLR model indicated that 38% of the decrease in ΣBaP_{eq} could be attributed to anthropogenic emissions control. The differing contributions of anthropogenic emissions control to the concentration of Σ15PAHs and ΣBaP_{eq} might be due to the fact that the HMW PAHs, which make up the majority of ΣBaP_{eq}, were less affected by the Clean Air Actions. In conclusion, the Clean Air Actions had different impacts on the temporal trends of

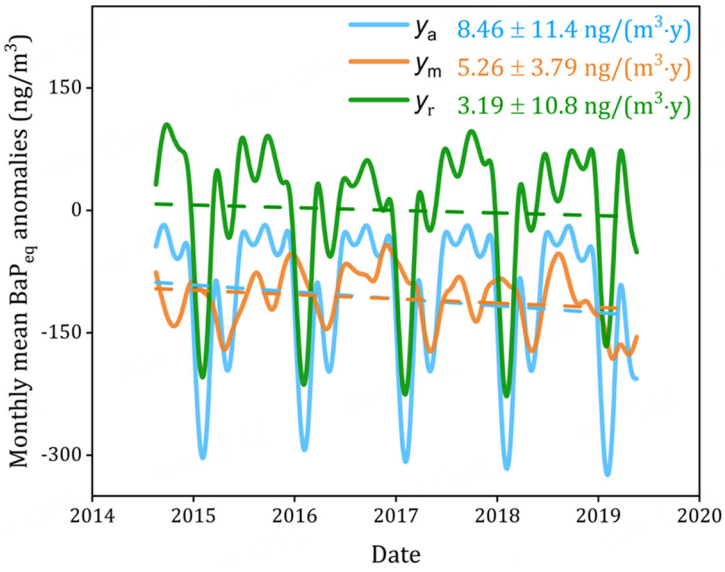

Figure 3. Time series of BaP_{eq} in the atmosphere in Harbin from June 2014 to May 2019 (blue line: monthly mean anomalies, orange line: meteorological contribution to the BaP_{eq} decreasing anomalies, green line: the meteorology-corrected residual).

concentrations and toxicity of atmospheric Σ15PAHs, suggesting that more attention should be focused on the PAHs with high toxicity in future studies.

While Clean Air Actions have led to a decrease in the overall concentration of PAHs, the findings emphasize the need for targeted measures to control high-toxicity PAHs, especially the HMW PAHs. These compounds, which contribute significantly to BaP_{eq}, remain less affected by current emissions control strategies, suggesting a gap in the regulation and reduction of the most harmful pollutants. In addition, it is also recommended that public health campaigns educate communities about the potential risks associated with PAH exposure. By raising awareness and promoting actions such as reducing outdoor air pollution exposure, urban planning that limits exposure near high-emission sources, and encouraging cleaner technologies, long-term health risks can be mitigated. Therefore, while the Clean Air Actions have led to notable reductions in PAH concentrations and associated health risks, further policies should target the most toxic PAHs, ensuring a more comprehensive approach to reducing the cancer risk and protecting public health. More focus should be directed toward emissions from sources that release HMW PAHs, and efforts should be made to enhance the effectiveness of current air quality regulations in reducing these pollutants.

DECLARATIONS

Authors' contributions

Designed the study, and conducted the original draft writing and revising, data curation and analysis: Zhu FJ

Conducted data curation and analysis: Qu LZ

Designed the study, and conducted the original draft revising: Ma WL

Availability of data and materials

The data are included in the Supplementary Materials. Further data are available from the corresponding authors upon reasonable request.

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Conflicts of interest

Ma WL is the Guest Editor of the Special Issue "Global Perspectives on Air Pollution Exposure and Human Health Implications" and an Editorial Board member of *Journal of Environmental Exposure Assessment*. Zhu FJ is a Junior Editorial Board member of *Journal of Environmental Exposure Assessment*. Ma WL Ma and Zhu FJ were not involved in any steps of editorial processing, notably including reviewer selection, manuscript handling, and decision making. The other author declared that there are no conflicts of interest.

Ethical approval and consent to participate

Not applicable.

Consent for publication

Not applicable.

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