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Original Research

Health Risk Assessment of PAHs in Contaminated Soil Based on a Monte Carlo Simulation: a Case of the Guan River Estuary, China

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Abstract

To obtain a scientific and reasonable health risk assessment of polycyclic aromatic hydrocarbons (PAHs) in contaminated soil from the Guan River Estuary, a probabilistic risk assessment model was used and each parameter's sensitivity was analyzed. The results showed that the total carcinogenic risk (TCR) of PAHs in contaminated soil was $2.46\times10^{-3}\pm2.12\times10^{-3}$, with a maximum value of 1.11×10^{-2} and a minimum value of 7.60×10^{-4}. All sampling sites exceeded the upper limit value (10^{-4}) of the acceptable carcinogenic risk recommended by the United States Environmental Protection Agency (USEPA), indicating that the carcinogenic risk of PAHs was very high in the contaminated soil of the Guan River Estuary Industrial Area. The total hazard quotient (THQ) was $4.71\times10^{-1}\pm3.90\times10^{-1}$, with the hazard quotient value at most sampling points being lower than 1. Benzo[a]pyrene(B[a]P) and Dibenz[a,h]anthracene(D[ah]A) were the major contributors to the TCR, with contribution rates of 70.57% and 15.61%, respectively; the main contributing monomers to the THQ were Pyrene(Pyr), Fluoranthene(Fl) and Phenanthrene(Phe), with contribution rates of 34.05%, 29.73%, and 17.20% respectively. The sensitivity of B[a]P and D[ah]A to TCR was the highest, reaching 80.28% and 21.58%, respectively. Body weight had a negative sensitivity (−19.15%). Oral intake and skin contact were the main exposure pathways affecting the TCR and THQ, contributing to exposure at rates of 79.74% and 73.28%, respectively.

Keywords: Guan River Estuary, contaminated soil, PAHs, health risk assessment, Monte Carlo simulation

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Introduction

In recent years, the ecological and health risks caused by polycyclic aromatic hydrocarbons (PAHs) have become an area of academic focus [1-4]. PAHs, a class of persistent organic pollutants, are widely distributed in environmental media such as soil, the atmosphere, plants, and water bodies, and are mainly sourced from the incomplete combustion process of fossil fuels and biomass [5, 6]. Soil is the most important reservoir for these PAHs, with studies finding that in the UK, 90% of PAHs in the environment are stored in surface soil [7]. So far, many studies have been conducted on the pollution characteristics, sources and ecological risks of PAHs in soil [6-10].

As an important environmental management tool, health risk assessment has been used by many researchers to control and manage contaminated soil. Based on the investigation of pollutant concentrations in soil, statistical analysis is often used to determine the potential harm of pollutants to ecology and human health, providing a theoretical basis for policy formulation to control pollutant emissions. Traditional risk assessments use theoretical reference values to calculate the degree of health risk, but these results may over- or underestimate the true values [11]. Therefore, uncertainty should be identified and quantified during risk assessment, which can be achieved using a probabilistic risk model [12]. Probabilistic risk assessment can be used to obtain the distribution of health risks and identify the impact of exposure pathways and parameters on these risks [13]. Toxicity assessments are generally based on the toxicity equivalent factors for each PAH relative to the BaP. Additionally, the 16 carcinogenic slope factors and non-carcinogenic reference doses are used to calculate toxicity assessments, providing results that are more scientific [14]. Based on these methods, a Monte Carlo simulation was used in this study to evaluate the health risks of PAHs to residents and workers in the Guan River Estuary Industrial Area, with an aim to provide data and theoretical guidance for the health risk management of contaminated sites in industrial areas.

Materials and Methods

Data Collection

During April 2017, 30 surface soil samples were collected from the Guan River Estuary Industrial Area. The sampling locations are shown in Fig. 1. The soil collection was based on a multi-point (5 points) mixed method. A stainless-steel shovel was used to collect 0–10 cm of the surface soil, and soil samples were returned to the laboratory for the removal of impurities and freeze-drying prior to analysis. The maximum, minimum, mean, and standard deviation of monomer PAHs were obtained through data processing (Table 1).

The PAH concentrations were determined by gas chromatography-mass spectrometry (GC–MS, Agilent, 7890A/5975C, USA). The gas chromatography column was a DB-5 polysiloxane polymer column (30 m×0.25 mm×0.25 μm). The column warming procedure: the column was held at 55ºC for 2 min, heated to 280ºC at 20ºC/min, and then heated to 310ºC at 10ºC/min for 5 min. The carrier gas was high-purity He, and its velocity was 1 mL/min. The SIM scan mode was used. The limit of detection (LOD) and the limit of quantitation (LOQ) were 0.04-1.18 ng/g and 0.13-3.89 ng/g. The recoveries of the 16 PAHs (Dr Ehrenstorfer GmbH, Germany) were 70.0-109%. The recoveries of the surrogate standards Nap-d8, Ace-d_{10}, Phe-d_{10}, Chry-d_{12}, and Per-d_{12} were 79.6-94.1%,

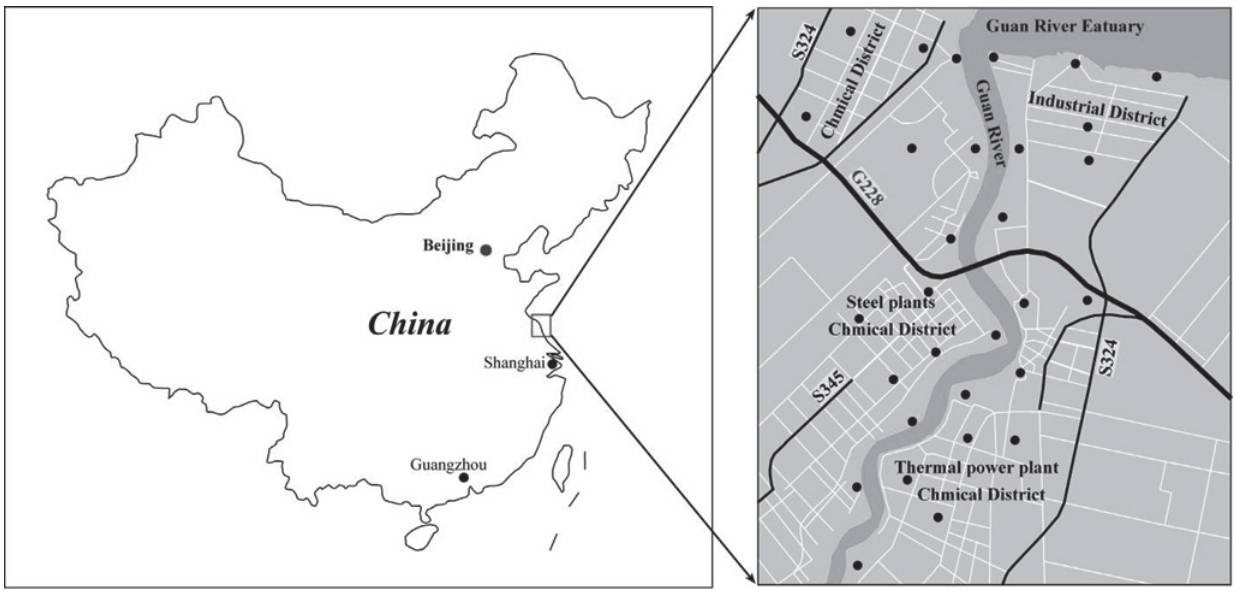

Fig. 1. Distribution of surface soil sampling points in contaminated sites.

Table 1. Concentration characteristics of monomer PAH in soil (μg/g).

Carcinogenic PAHs					Non-narcinogenic PAHs				
PAHs	Min	Max	Mean	Standard Deviation	PAHs	Min	Max	Mean	Standard Deviation
B[a]A	76.8	1457.6	290.4	279.2	Na	1.7	79.0	17.2	15.8
Chry	102.0	1214.7	308.2	260.0	Acy	5.2	519.3	43.7	94.0
B[b+k]F	108.0	986.0	301.5	231.5	Ace	1.3	86.8	21.5	19.2
B[a]P	89.4	1737.6	336.6	326.0	Fluo	6.9	129.9	36.8	35.4
InP	83.0	556.8	199.3	128.7	Phe	56.2	915.6	295.8	235.5
D[ah]A	21.9	287.4	74.4	66.6	An	13.1	250.1	68.8	68.6
					Fl	176.7	2659.6	681.6	677.4
					Pyr	189.2	2173.4	585.5	539.8
					B[ghi]P	93.2	770.2	243.6	174.5

Carcinogenic PAHs: Benzo[*a*]anthracene(B[a]A), Chrysene(Chry), Benzo[*b*+*k*]fluoranthene(B[b+k]F), Benzo[a]pyrene(B[a]P), Indeno[1,2,3-*cd*]pyrene(InP), Dibenz[a,h]anthracene (D[ah]A); **Non-narcinogenic PAHs**: Naphthalene(Na), Acenaphthylene(Acy), Acenaphthene(Ace), Fluorene(Fluo), Phenanthrene(Phe), Anthracene(An), Fluoranthene(Fl), Pyrene(Pyr), Benzo[g,h,i] perylene(B[ghi]P)

78.5-95.6%, 75.3-98.5%, 78.3-106%, and 79.6-101%, respectively.

Exposure Assumptions

The land use type of the Guan River Estuary is mainly industrial, the exposure scenario is set as nonsensitive land, the population is mostly workers and residents, the exposure period of adults is long, and the exposure frequency is high. Therefore, the carcinogenesis risk and hazard quotient of adults were the primary factors evaluated. The carcinogenic risk and hazard quotient represent the level of exposure to carcinogenic and non-carcinogens monomer PAH, respectively. In this study, the exposure pathways of PAHs included oral intake, skin contact, and respiratory intake.

Toxicity Assessment

The harmful effects of PAHs on human health include both carcinogenic and non-carcinogenic effects. Research has shown that the concentration of PAHs in the Guan River Estuary has increased annually, and 16 types of PAHs controlled by the USEPA contribute to the overall risk level; therefore, they are used as the evaluation objects [8]. The carcinogenicity slope factor and non-carcinogenic reference dose of the 16 types of monomer PAHs were obtained by referring to China's guidelines for the risk assessment of contaminated sites. Below, *SF* is the carcinogenic slope factor of PAHs with carcinogenic effect, and *RfD* is the reference dose of PAHs with non-carcinogenic effects (Table 2).

Table 2. *SF* and *RfD* data of 16 kinds of PAHs (kg·d·mg^{-1}).

PAHs	Carcinogenic effect value			PAHs	Non-carcinogenic effect value		
	SF_o	SF_i	SF_d		RfD_o	RfD_i	RfD_d
B[a]A	7.30E-01	3.10E-01	1.46E+00	Na	4.00E-02	8.75E-04	2.00E-02
Chry	7.30E-03	3.10E-03	1.46E-02	Acy	6.00E-02	3.00E-02	3.00E-02
B[b+k]F	4.02E-01	1.71E-01	8.03E-01	Ace	6.00E-02	3.00E-02	3.00E-02
B[a]P	7.30E+00	3.10E+00	1.46E+01	Fluo	4.00E-02	2.00E-02	2.00E-02
InP	7.30E-01	3.10E-01	1.46E+00	Phe	3.00E-02	1.50E-02	1.50E-02
D[ah]A	7.30E+00	3.10E+00	1.46E+01	An	3.00E-01	1.50E-01	1.50E-01
				Fl	4.00E-02	2.00E-02	2.00E-02
				Pyr	3.00E-02	1.50E-02	1.50E-02
				B[ghi]P	3.00E-02	1.50E-02	1.50E-02

Risk Characterization

Based on the "Technical Guidelines for Risk Assessment of Contaminated Sites" issued by the Ministry of Ecology and Environment of China [15], the health risks of PAHs in contaminated sites can be divided into two types: carcinogenic risk (the probability of people to induce carcinogenic diseases or injuries when exposed to carcinogenic pollutants) and hazard quotient (the ratio of daily intake and reference dose of pollutants, which is used to characterize the exposure of the human body to non-carcinogenic pollutants through a single pathway). Their formulas for various exposure pathways are given in what follows [13, 15].

Non-Sensitive Land Exposure Assessment Model

Oral Intake of Soil

The carcinogenic risk of oral exposure is calculated by the following formula:

$$CR_{ois} = OISER_{ca} \times C_{sur} \times SF_o \quad (1)$$

where CR_{ois} is the carcinogenic risk of contaminated soil by oral intake; $OISER_{ca}$ is the soil exposure through oral intake (carcinogenic effect); C_{sur} is the content of PAHs in soil; and SF_o is the carcinogenic slope factor of oral intake. For the carcinogenic effects of a single pollutant, considering the lifetime hazards of adult exposure, the soil exposure corresponding to oral intake is calculated using the following formula:

$$OISER_{ca} = \frac{OSIR \times ED \times EF \times ABS_O}{BW \times AT_{ca}} \times 10^{-6} \quad (2)$$

The hazard quotient of oral exposure is calculated by the following formula:

$$HQ_{ois} = \frac{OISER_{nc} \times C_{sur}}{RfD_O \times SAF} \quad (3)$$

where HQ_{ois} is the hazard quotient of contaminated soil by oral intake; $OISER_{nc}$ is the soil exposure by oral intake (non-carcinogenic effect); and RfD_o is the oral intake reference dose. Regarding the non-carcinogenic effects of a single pollutant, when considering the exposure hazards of adults, the formula for calculating the amount of soil exposure by oral intake is as follows:

$$OISER_{nc} = \frac{OSIR \times ED \times EF \times ABS_O}{BW \times AT_{nc}} \times 10^{-6} \quad (4)$$

Skin Contact with Soil

The carcinogenic risk of skin contact is calculated as follows:

$$CR_{dcs} = DCSER_{ca} \times C_{sur} \times SF_d \quad (5)$$

where CR_{dcs} is the carcinogenic risk of contaminated soil by skin contact; $DCSER_{ca}$ is the soil exposure by skin contact (carcinogenic effect); and SF_d is the carcinogenic slope factor of skin contact. The soil exposure corresponding to skin contact is calculated using the following formula:

$$DCSER_{ca} = \frac{SAE \times SSAR \times ED \times EF \times E_V \times ABS_d}{BW \times AT_{ca}} \times 10^{-6} \quad (6)$$

The hazard quotient of skin contact is calculated using the following formula:

$$HQ_{dcs} = \frac{DCSER_{nc} \times C_{sur}}{RfD_d \times SAF} \quad (7)$$

where HQ_{dcs} is the hazard quotient of contaminated soil by skin contact; RfD_d is the skin contact reference dose; and $DCSER_{nc}$ is the soil exposure by skin contact (non-carcinogenic effect). The formula for calculating the amount of soil exposure by skin contact is as follows:

$$DCSER_{nc} = \frac{SAE \times SSAR \times ED \times EF \times E_V \times ABS_d}{BW \times AT_{nc}} \times 10^{-6} \quad (8)$$

Respiratory Intake of Soil

The carcinogenic risk of respiratory intake is calculated as follows:

$$CR_{pis} = PISER_{ca} \times C_{sur} \times SF_i \quad (9)$$

where CR_{pis} is the carcinogenic risk of contaminated soil by respiratory intake; $PISER_{ca}$ is the soil exposure by respiratory intake (carcinogenic effect); and SF_i is the carcinogenic slope factor of respiratory intake. The soil exposure corresponding to respiratory intake is calculated using the following formula:

$$PISER_{ca} = \frac{PM_{10} \times DAIR \times EF \times PIAF}{BW} \times \frac{EFO \times fsp_o + fsp_i \times EFI}{AT_{ca}} \times 10^{-6} \quad (10)$$

The hazard quotient of respiratory intake is calculated using the following formula:

$$HQ_{pis} = \frac{PISER_{nc} \times C_{sur}}{RfD_i \times SAF} \quad (11)$$

where HQ_{pis} is the hazard quotient of contaminated soil through respiratory intake; RfD_i is the respiratory intake reference dose; and $PISER_{nc}$ is the soil exposure by respiratory intake (non-carcinogenic effect). The formula for calculating the amount of soil exposure by respiratory intake is as follows:

$$PISER_{nc} = \frac{PM_{10} \times DAIR \times EF \times PIAF}{BW} \times \frac{EFO \times fsp_o + fsp_i \times EFI}{AT_{nc}} \times 10^{-6} \quad (12)$$

Total Health Risk Level

The TCR is calculated using the following formula:

$$TCR = \sum_{i=1}^{n} (CR_{ois,n} + CR_{dcs,n} + CR_{pis,n}) \quad (13)$$

where n is 7 types of PAHs with known carcinogenic effects (Table 2).

The THQ is calculated using the following formula:

$$THQ = \sum_{i=1}^{m} (HQ_{ois,m} + HQ_{dcs,m} + HQ_{pis,m}) \quad (14)$$

where m is 9 types of PAHs with known non-carcinogenic effects (Table 2). The values and meanings of the other parameters in Formulas (1)–(14) are shown in Table 3.

Results and Discussion

Crystal Ball software was used to iteratively calculate the risk associated with PAHs, where the uncertainty parameters are the independent variables, the number of random simulation iterations was set to 10 000, and the confidence level was 95%. Sensitivity analysis results can reflect the degree of influence of each parameter on exposure risk. If the sensitivity is a positive value, risk is present; the larger the value, the greater the impact on the risk result. If the sensitivity is negative, it means that it is negatively correlated with the risk results; the greater the absolute value of the sensitivity, the greater the impact on the risk.

Total Health Risk

The carcinogenic risk and hazard quotient of the three input pathways were calculated according to formulas (1)–(12), and the TCR and THQ of PAHs in contaminated soil according to formulas (13) and (14), with the TCR and the THQ having a normal distribution (Fig. 2). The distribution range of TCR was $2.46\times10^{-3}\pm2.12\times10^{-3}$, its maximum was 1.11×10^{-2}, and its minimum was 7.60×10^{-4}. The acceptable TCR recommended by the USEPA was 1×10^{-6}, and the upper limit of the TCR was 1×10^{-4}. If the TCR is less than 1×10^{-6}, it is acceptable, though if the risk value is greater than 1×10^{-4}, the potential risk is high [13]. The average carcinogenic risk of PAHs in the contaminated soil of the Guan River Estuary is one order of magnitude higher than that recommended by the USEPA, with the minimum value also being higher than the recommended upper limit. This result shows that the PAHs in the study

Table 3. Exposure parameters of adults in the Guan River Estuary Industrial Area.

Parameter	Parameter meaning	Unit	Value	References
OSIR	Daily soil intake	*mg/d*	100	[15]
ED	Exposure period	*a*	25	[17]
EF	Exposure frequency	*d/a*	250	[15]
ABSo	Oral absorption efficiency factor	-	1	[15]
BW	Body weight	*kg*	56.8	[15]
ATca	Mean time of carcinogenic effect	*d*	26280	[15]
ATnc	Mean time of non-carcinogenic effects	*d*	9125	[15]
SAE	Exposed skin surface area	*cm²*	1848	[17]
SSAR	Skin surface soil adhesion coefficient	-	0.2	[15]
Ev	Frequency of daily skin contact events	-	1	[15]
ABSd	Skin contact absorption efficiency factor	-	0.13	[16]
PM_{10}	Content of respirable suspended particulates in air	-	0.15	[15]
DAIR	Daily air respiration	*m³/d*	14.5	[15]
PIAF	Retention ratio of inhaled soil particles	-	0.75	[15]
fspo	Proportion of particulate matter from the outdoor air	-	0.5	[15]
EFO	Outdoor exposure frequency	*d/a*	87.5	[15]
fspi	Proportion of particulate matter from the indoor air	-	0.8	[15]
SAF	Reference dose partition coefficient exposed to soil	-	0.2	[15]
EFI	Indoor exposure frequency	*d/a*	187.5	[15]

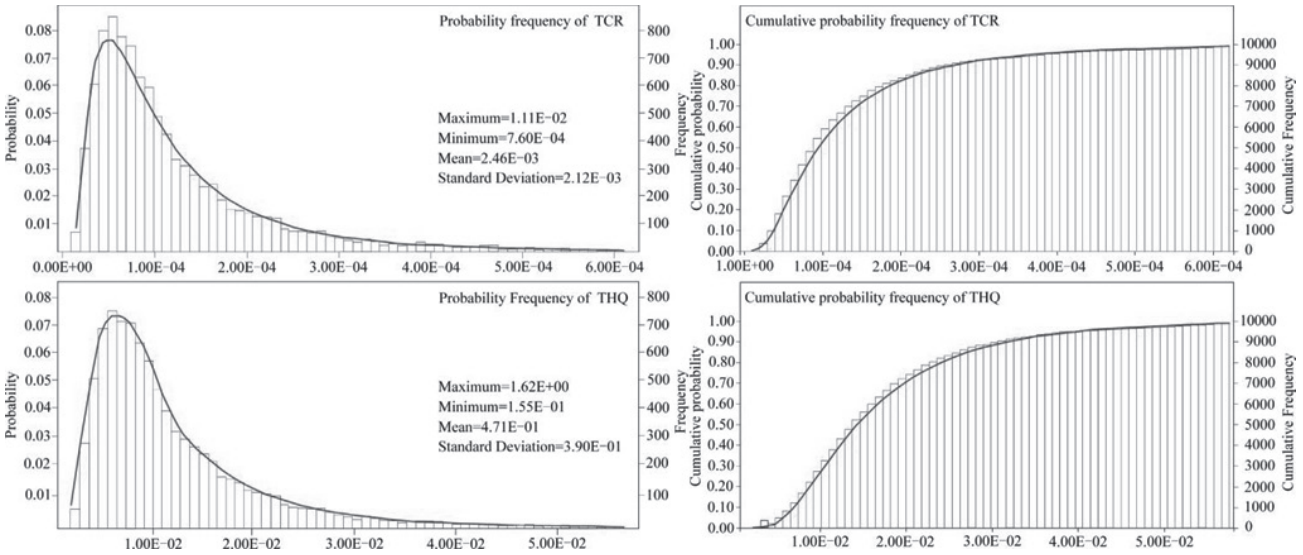

Fig. 2. The total carcinogenic risk and total hazard quotient of PAHs.

area have a significant carcinogenesis risk, and poses a significant danger to the health of workers and residents; therefore, corresponding protective measures should be taken to remediate contaminated soil. Compared with the TCR in other studies in China, the carcinogenic risk of PAHs in the Guan River Estuary was higher than that in Beijing (4.02×10^{-5}) [18], Shanghai (3.43×10^{-5}) [13], Dalian (2.79×10^{-5}) [19], and Lanzhou (4.46×10^{-5}) [20]. The process of industrialization, combustion of coal and petroleum, in the Guan River Estuary has resulted in heavy PAH pollution.

The distribution range of THQ was 4.71×10^{-1} $\pm3.90\times10^{-1}$, with a maximum value of 1.62, a minimum value of 1.55×10^{-1}, and an average value of 4.71×10^{-1}. Values of THQ of less than 1 are acceptable; if the THQ is greater than 1, it may cause toxicity [13]. In this study, the THQ of most sampling locations were lower than 1, but the simulation results showed that 10% of them were higher than 1, indicating that PAHs will not cause obvious health hazards in the short term, but may result in potential risks to the population in the long term.

Sensitivity Analysis

Crystal Ball software was used to explore the sensitivity of various parameters. The results of sensitivity analysis on human exposure parameters show that ED, BW, ATca and EF have the highest sensitivity to the TCR, with sensitivities of 20.13%, −19.15%, −18.60%, and 18.07%, respectively (Fig. 3). In terms of the THQ, ATnc, BW, SAF, and EF were the most sensitive, with sensitivities of −24.48%, −22.17%, 21.88%, and 21.15%, respectively. In terms of PAHs, B [a]P (80.28%) and D [ah]A (21.58%) have the largest impact on TCR, followed by B [a]A (8.57%), InP (6.59%), and B [b+k]F (2.54%), and the smallest is

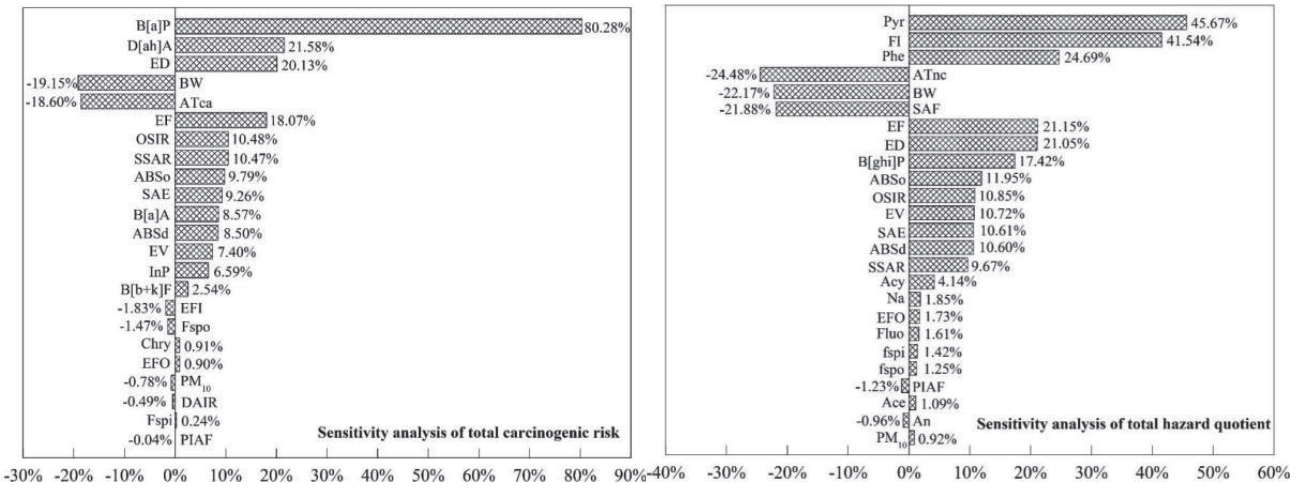

Fig. 3. Sensitivity analysis of the total carcinogenic risk and total hazard quotient.

Table 4. Contribution ratio of different exposure pathways.

Exposure pathways	TCR		THQ	
	Sum	Proportion(%)	Sum	Proportion(%)
Oral intake	5.88E-02	79.74	1.03E+01	73.28
Skin contact	1.47E-02	19.96	3.48E+00	24.65
Respiratory intake	2.25E-04	0.30	2.92E-01	2.07
Total	7.37E-02		1.41E+01	

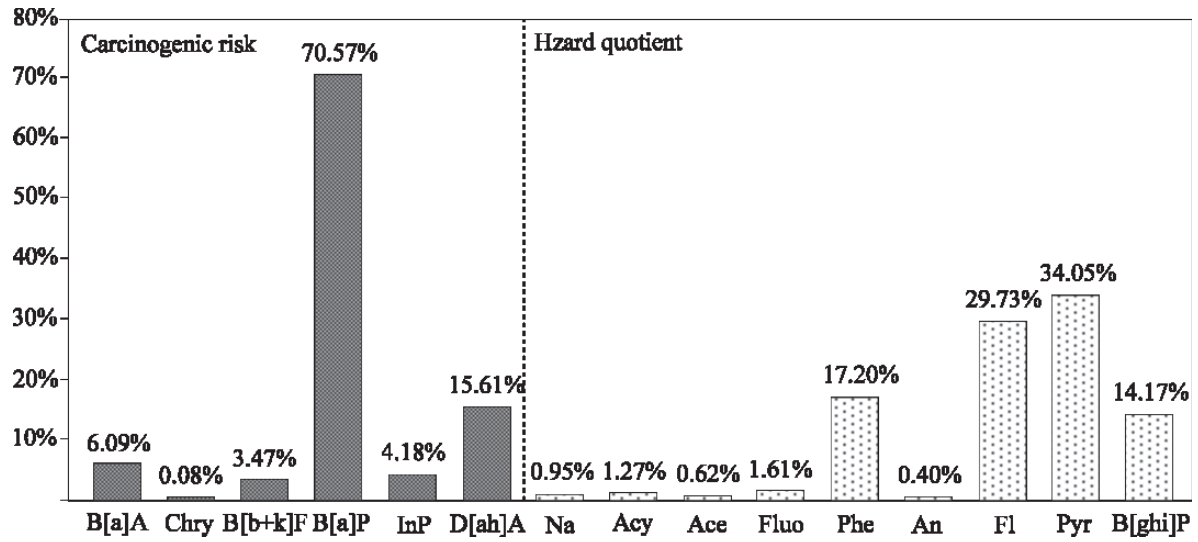

Fig. 4. Contribution rate of monomer PAH to the arcinogenic risk and hazard quotient.

Chry (0.91%). In the sensitivity analysis for the THQ, Pyr and Fl have a the largest impacts on the hazard quotient, with sensitivities of 45.67% and 41.54%, respectively, followed by Phe (24.69%) and B [ghi] P (17.42%). Similar to the results of other studies, B [a]P and D [ah]A are the main contributors to the carcinogenic risk of soil in Shanghai, with sensitivities of 60.41% and 26.84%, respectively. For THQ, the main sensitive monomers for PAH were Pyr (31.65%) and Fl (27.06%) [18].

Contribution Rate Analysis

Oral intake refers to the intake of PAH-contaminated soil through the mouth; skin contact refers to contact with PAH-contaminated soil on the skin; and respiratory intake refers to the inhalation of PAH-contaminated soil particles. The cancer risk and hazard quotient of each PAH contamination pathway were calculated according to formulas (1)–(14), and the distribution of the 3 exposure pathways and the contribution rate of 16 PAHs are shown in Table 4 and Fig. 4.

The contribution rate of the three exposure routes of the TCR and THQ are very similar. Oral intake has the highest contribution rate, accounting for more than 70% of both (TCR: 79.74%, THQ: 73.28%), followed by skin contact, accounting for about 20% of both (TCR: 19.96%, THQ: 24.65%), while the contribution rate of respiratory intake is the lowest (TCR: 0.30%, THQ: 2.07%). Oral intake is the primary exposure route considered by the United States and Canada in establishing soil environmental standards for the protection of human health, followed by skin contact [21]. In China, other related studies have found that the contribution rate of oral intake in Beijing is 88.70% [22], and in Shanghai it is 94% [23], while the contribution rate of exposure caused by skin contact is relatively low.

For TCR, the contribution rate of B [a]P is as high as 70.57%, followed by D [ah]A (15.61%), and B [a]A (6.09%), while Chry (0.08%) has the lowest contribution rate. For THQ, Pyr has the highest contribution rate of 34.05%, followed by Fl (29.73%), and the lowest is An, with a contribution rate of 0.40%. A study on the health risk assessment of soil in Shanghai found that the contribution rate of B [a]P and D [ah]A for TCR were 60.41% and 26.84%, respectively, and that Pyr and Fl had the largest contributions to THQ, with rates of 36.56% and 33.18%, respectively, which are similar to the results of this study [23].

Conclusions

1. The TCR of PAHs was $2.46\times10^{-3}\pm2.12\times10^{-3}$, and the carcinogenic risk of all sampling points was greater than 10^{-4}, indicating that there was a high risk to human health; the THQ was $4.71\times10^{-1}\pm3.90\times10^{-1}$, and 10% of the simulation results were greater than 1, indicating that there is an unacceptable risk associated with living or working in the study area for a long time.
2. B [a]P (80.28%) and D [ah]A (21.58%) had the greatest sensitivity to TCR, while the sensitivity of Pyr and Fl to the THQ was 45.67% and 41.54%, respectively. BW had negative sensitivities to the TCR and THQ, of −19.15% and −24.48%, respectively.
3. The contribution rates of oral intake to the TCR and THQ were 79.74% and 73.28%, respectively. The contribution rates of B [a]P and D [ah]A to the TCR were 70.57% and 15.61%, respectively, while the contribution rates of Pyr and Fl to the THQ were 34.05% and 29.73%, respectively.

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Conflicts of Interest

The authors declare no conflict of interest.

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