Health Risk Assessment on Exposure to PM$_{2.5}$-bound PAHs from an Urban-industrial Area in Rayong City, Thailand

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Abstract

BACKGROUND: A city’s industrial area’s air quality has become a major priority. PM$_{2.5}$-bound polycyclic aromatic hydrocarbons (PAHs) are one of the most common pollutants in urban-industrial area and can be linked to health problems.

AIM: This study aims to (1) investigate PM$_{2.5}$ and PAHs emitted from roadside area (RS) and industrial estate (IE) in Rayong city and (2) assess the inhalation of PM$_{2.5}$ and PAHs on the human health of the age group.

METHODS: PM$_{2.5}$-bound PAHs were investigated and their carcinogenic risk was evaluated in this study. PM$_{2.5}$ samples were collected on quartz filters contained in a mini-volume air sampler and analyzed for PAHs by GC–MS.

RESULTS: The average PM$_{2.5}$ concentrations at RS and IE were 43.3 ± 26.8 and 40.4 ± 21.7 µg/m$^3$, respectively. However, it was found that the PM$_{2.5}$ and PAHs values were not significantly different (p > 0.05). The results revealed that the individual lifetime cancer risk demonstrated an increased morbidity and mortality from lung cancer and mutagenesis risk with exposure demonstrated an increased morbidity and mortality from lung cancer and mutagenesis risk with exposure to PM$_{2.5}$ through the inhalation pathway was a negligible (<10$^{-6}$).

CONCLUSIONS: As a result, the PM$_{2.5}$ concentrations have substantial implications for Rayong city’s environmental management and protection, relating to car emissions and coal combustion.

Introduction

Polycyclic aromatic hydrocarbons (PAHs) are released by incomplete combustion or pyrolysis of organic molecules, and they are linked to the resource base of oil, gas, coal, and biomass for energy production. PAHs are formed when semi-volatile organic contaminants with at least two fused aromatic rings are combined. PAHs are widespread environmental pollutant [1]. However, PAHs can be found in both gas and particulate phases, which have associated the PM with carbonaceous core that adsorbs different the strong toxic potential, they are mainly found on submicron-sized particles [2]. Therefore, it is important to clarify the contribution of PAHs to the toxicity of PM. Especially, fine particles (PM$_{2.5}$) are generated from fossil fuel and biomass burning combustion, which size has been influenced by their organic and inorganic composition and toxic potential [3], [4], [5]. Zhang et al. [5] revealed that the cancer risk from exposure to PM$_{2.5}$-bound PAHs in Beijing, China, has been labeled as posing a significant health hazard. They presented that the PAHs concentrations in PM$_{2.5}$ were found 5%–15%.

Road dust, fossil fuel combustion, vehicle exhaust, and a large contribution due to increased coal burning have all been sources of pollution.

Epidemiological studies of PM$_{2.5}$-bound PAHs exposure demonstrated an increased morbidity and mortality from lung cancer and mutagenesis risk with long term using the incremental lifetime cancer risk (ILCR) model. They mentioned that the inhalation pathway ILCR ranging from 10$^{-6}$ to 10$^{-3}$ is implied a high potential cancer risk. Chen et al. [6] reported the estimation of the lung cancer risk exposure to PAHs in PM$_{2.5}$ that was collected from Nanjing, China. The mean ILCR for boys, teenage boys, male adults, elder males, girls, teenage girls, female adults, and elderly females (7.21 × 10$^{-8}$–3.53 × 10$^{-4}$), which the source produced by from vehicle emission, was indicated as a low potential lung cancer risk. However, they reported that children were sensitive to health concerns posed by Nanjing pollution. The evaluation of inhalation exposure to PM$_{2.5}$-bound PAHs of urbanization and industrialization was investigated in five urban cities of Zhejiang Province, China, using the ILCR. The ILCR values for children (1.8 × 10$^{-7}$–4.8 × 10$^{-7}$) and adults (2.6 × 10$^{-7}$–1.57 × 10$^{-7}$) from Jinhua, Lishui, Hangzhou,
Zhoushan, and Ningbo were reported to indicate acceptable cancer risk (<10^{-6}). Zhejiang Province is regarded as one of the China’s most expanding rapidly areas. In the future, controlling atmospheric PAHs, particularly 4–6 rings PAHs, should be a top focus [7]. Hassanvand et al. [8] reported that PM_{2.5}–bound PAHs concentrations were 83–88%, indicating that they might penetrate deep into the lungs’ alveolar regions. In vitro study, the metabolic activation of the very low doses of PAHs in PM_{2.5} onto the inorganic nuclei found that the underlying mechanism of motion carefully concerned in its cytotoxicity in human lung epithelial cells. Especially, benzo(b)fluoranthene (BbF) and benzo(k)fluoranthene (BkF) were highly correlated with damage of epithelial cells, which BbF and BkF released from diesel exhaust particles and atmospheric PM_{2.5} [9]. The dominant of individual PAHs-bound PM_{2.5} emitted from traffic was benzo[a]pyrene (BaP), dibenz[a,h]anthracene (DahA), BbK, and BbF, while BbK, BkF, chrysene (Chr), pyrene (Pyr), and indeno[1,2,3-cd]pyrene (IcdP) were found the industrial region [5], [10]. An epidemiological study reported that the link between traffic-related PM_{2.5}–coated PAHs and odd preterm birth increased by 30% every interquartile rise, which was positively associated and grouped together with the analysis [11].

Rayong air quality is regarded as a major problem that poses a threat to human health. As a result, the primary goal of this study is to properly determine PM_{2.5} and PAHs in PM_{2.5} concentrations at two sites in Rayong city: The roadside of high-traffic areas and an industrial estate (IE). Second, using the incremental life cancer risk (ILCR), study the cancer and mutagenic risk of the determined PM_{2.5}–bound PAHs on human health.

Materials and Methods

**Sampling sites**

Rayong province is located on the Gulf of Thailand’s east coast. The city is a key part of Thailand’s IE. The Maptaphut IE, which spans 8012.39 acres in Rayong province, is the main IE (Figure 1). A total of 142 petrochemical and chemical product industries, 88 steel and metal industries, 36 machinery and equipment factories, six coal-fired power factories, nine petroleum factories, two oil refineries, and 31 rubber processing facilities are among the 695 industrial plants on the estate [12]. Within two areas of Rayong city, sampling sites were surveyed and selected at random. By community ambient air, air quality samples were randomly collected roadside in metropolitan areas and IE s. The following are the categories of sample sites: (Figure 1).

- **Roadside area (RS):** The sampling sites are situated in Rayong City municipality (12°40′28.38″N and 101°16′41.88″E) in urban area, which was based on criteria such as roadside, residential and business buildings, government office, transportation network and high-traffic intensity, and human activities. The roadside sampling was placed 1.5–2.0 m of the above the ground level.
- **IE:** The selection of sampling locations for the Maptaphut IE was collected from rooftop of a four-storied building at Maptaphut Town municipality (12°43′29.66″N and 101°7′34.78″E). Most of the area is occupied by petrochemical and chemical product industries, steel and metal, machinery and equipment, coal-fired power, petroleum factories, oil refineries, community areas, and transportation networks.

**PM_{2.5} collection**

PM_{2.5} samples were collected on quartz fiber filters (47.0 mm diameter, Whatman) using mini-volumetric air samplers (Air metric, USA) at a flow rate of 5 L/min for 24 h (9:00 am–9:00 am). PM_{2.5} samples were randomly collected during August 2019–February 2020. Both sites were collected samples at the same time. Quartz fiber filters were baked at 800°C for 3 h. To remove organic contamination, place the samples in the oven before collecting them [13]. The filters were pre-weighed by a 6-place microbalance (Mettler Toledo, Switzerland) in a controlled room (24.0 ± 0.3°C, 41.9 ± 1.8% RH). After the sampling, for 48 h, the filters were maintained in a plastic box with aluminum foil wrapped around it inside a desiccator, before reweighed and stored in a freezer (−20.0°C) until analysis.

**Extraction and analysis of PAHs**

The quartz fiber filter samples were extracted for 10 min using an ultrasonicator in 15 mL.
dichloromethane (1:1, v/v) at a regulated temperature (10.0°C). The extracted solutions were then filtered and dried in a rotary evaporator using a 0.45 PTFE syringe filter (Whatman). The solution was added 20 µL of 2 mg/L for acenaphthene-d10 and 20 µL of 5 mg/L for pyrene-d12 with mixture of internal standards. And then, the solvent mixture was used to dissolve the extracted solution to 1 mL. PAHs were measured using a gas chromatography–mass spectrometry (GC–MS, Agilent 7890A, USA) with a 30 m HP-5MS capillary column (0.25 mm inner diameter, 0.25 mm film thickness, Agilent, USA) and a 30 m HP-5MS capillary column (0.25 mm inner diameter, 0.25 mm film thickness, Agilent, USA). The heating program was set for column oven was held at 75°C for 0.5 min, increased to 150°C by a rate of 20°C/min, heated to 150–290°C on rate of 10°C/min, and then hold at 290°C for 3 min. The MS was operated in selective ion monitoring mode (SIM) at 70 Ev and 1 µL for injected value with splitless mode [14]. The 16-PAHs concentration was determined and analyzed consisting of naphthalene (Nap), acenaphthylene (Acy), acenaphthene (Ace), fluorene (Flu), phenanthrene (Phe), anthracene (Ant), fluoranthene (Fla), pyrene (Pyr), benzo[a]anthracene (BaA), chrysene (Chr), BbF, BkF, BaP, IcdP, DahA, and benzo[g,h,i]perylene (BghiP). Detailed pre-treatment and analysis procedures for the quality control/quality assurance (QC/QA) of PAHs could be found in our previous work of Kawichai et al., 2020. Recovery efficiency of 16-PAHs (n = 7) ranged 34.7% (Flu) and averaged 84% from an extraction of 20 mg the urban dust NIST1649b standard reference materials. The results of spiked method (n = 7) showed the recovery values of the low concentrations of 16-PAHs (n = 7) ranged 34.7% (Flu) to 109% (IcdP) and averaged 84% from an extraction of 20 mg the urban dust NIST1649b standard reference materials. The results of spiked method (n = 7) showed the recovery values of the low concentrations of 16-PAHs (4 µg/L) for the extraction method ranged from 71 to 107%, while recoveries of high concentrations of 16-PAHs (4 µg/L) ranged from 75 to 96%. The limit of detection in this study was 0.002–0.006 ng/m³ and limit of quantitation was 0.007–0.021 ng/m³.

Carcinogenic risk assessment of PM

An estimate of the probability that an individual will develop lung cancer as a result of PM
exposure [15]. Lung cancer risk is calculated using the PHASE lifetime cancer risk (Rc) to evaluate the long-term PM
exposure, as shown in Equation 1.

\[
R_{lc} = \text{LADD} \times \text{SF}
\]

For exposure to air pollutants with carcinogenic or chronic effects, the lifetime average daily dose (LADD) is utilized, which is represented in Equation 2. The inhalation slope factor (SF) can be used to calculate the value of toxicity for health effects, as shown in Equation 3. Rc values can be estimated to seasonal or intermittent exposure pattern over 1 or more years [15]. The value of Rlc less than 10⁻⁶ is considered negligible cancer risk, while the range between 10⁻⁶ and 10⁻⁴ is acceptable cancer risk. Moreover, the ≥10⁻⁴ is significant cancer potency. Table 1 lists the parameter for calculation of LADD as PM
exposure by the US.EPA 2011.

| Receptor | BW (kg) | InhR (m³/day) | EF (hour/day) | ED (day/year) | AT (year) | SF (µg/kg/day) |
|----------|---------|---------------|--------------|---------------|-----------|---------------|
| Children 1–<2 years | 11.4 | 5.4 | 4 | 350 | 1 | 1460 |
| 2–<3 years | 13.8 | 8.9 | 4 | 350 | 1 | 1460 |
| 3–<5 years | 18.6 | 10.1 | 4 | 350 | 3 | 4380 |
| 5–<10 years | 31.8 | 12.0 | 4 | 350 | 5 | 7300 |
| Teenagers 11–<16 years | 56.8 | 15.2 | 4 | 350 | 5 | 7300 |
| 16–<21 years | 71.6 | 16.3 | 4 | 350 | 5 | 7300 |
| Adults ≥21 years | 80.0 | 17.5 | 4 | 350 | 49 | 71,540 |

Growth rate for adults referred to Liu et al. [25]

\[
\text{LADD} = \frac{C \times \text{InhR} \times \text{ED}}{\text{BW} \times 70 \text{ years}}
\]

\[
\text{SF} = \frac{\text{UR} \times \text{InhR}}{\text{BW} \times \text{EF}}
\]

Where, the C is PM
concentrations (µg/m³). InhR is inhalation rate (m³/day). ED is exposure duration (year). BW is bodyweight (kg) and UR is unit risk of PM
which is 0.008 per µg/m³ [15].

Carcinogenic risk assessment of PM
-bound PAHs

Toxicity equivalent concentration (TEQ
) concentrations are commonly used to measure the risk of carcinogenic effectiveness and are linked to the mutagenic potency of each PAH [16], [17]. The calculation of the total of individual concentrations of each PAH can be multiplied by their toxic equivalent factor (TEF) relative to the carcinogenic potency. TEQ
was referred to the carcinogenic substances. The TEQ
was calculated by Nisbet and LaGoy [18] and Durant et al. [19], as shown in Equations 4–5.

\[
\text{TEQ}_{\text{BaP}} = \sum_{i=1}^{n=1} \left( \text{PAH}_i \times \text{TEF}_i \right)
\]

\[
\text{TEQ}_{\text{BaP}} = 0.001(\text{Nap}+\text{Acy}+\text{Ac}+\text{Flu}+\text{Phe}+\text{Fla}+\text{Pyr})
+ 0.01(\text{Ant}+\text{BghiP}+\text{Chr})
+ 0.1(\text{BaA}+\text{BbF}+\text{BkF}+\text{IcdP})
+ \text{BaP} + \text{DahA}
\]

Where, PAH
is concentration of individual PAHs and TEF, is the TEFs, as shown in Equation (5). In addition, the excess cancer risk (ECR) is estimated using Equation 6 to assess the PAHs exposure associated to carcinogenic risk [16], [17], [20].

\[
\text{The UR}_{\text{BaP}} = \text{Inhalation cancer unit risk of PAHs exposure to estimate the number of people who will develop cancer as a result of inhaling 1 ng/m³}
\]
of BaP equivalent concentration over the course of 70 years lifetime. The recommended UR_{BaP} of the World Health Organization (WHO) is $8.7 \times 10^{-5}$ per ng/m$^3$ [1].

$$ECR = TEQ_{BaP} \times UR_{BaP}$$

ECR values ranging from $10^{-6}$ to $10^{-4}$ suggest a potential risk, with a value of $10^{-6}$ indicating no risk of cancer, and a value of $10^{-3}$ indicating a possibility of carcinogenic effect [21].

The ILCR of PAHs in PM$_{2.5}$

Based on the US-EPA standard approach, the ILCR was applied to quantitatively evaluate PAHs exposure for cancer risk [22]. The exposure factor established by the US-EPA: 2011 Edition was used to assess the human health risk of PAHs in the PM$_{2.5}$ exposure pathway [23]. Children, teenagers, and adults were the three age groups whose inhalation pathways were studied (Table 1). Several studies [3], [7], [17], [24] reported the inhalation of ILCR model for this calculation as follows (Equation 7):

$$ILCR = \left( \frac{CSF \times \frac{BW}{70}}{BW} \times TEQ \times InhR \times ET \times EF \times ED \right) \times cf$$

Where, the CSF is inhalation carcinogenic SF corresponded to 3.1 mg/kg-day [3]. BW is body weight (Kg). InhR is inhalation rate (m$^3$/hour). ET is daily exposure time as 4 h/day [3]. EF and ED are exposure frequency (350 days/year) and exposure duration (year). AT is the average time based on 365 days for year, and cf is conversion factor with the value of $1 \times 10^{6}$. The value of ILCR from $10^{-11}$ to $10^{-12}$ indicated a potential risk, while ILCR value $\geq 10^{-1}$ is implied a serious risk and suggested virtual safety as $\leq 10^{-6}$ [25].

Data analysis

The independent sample t-test was used to analyze the differences between PM$_{2.5}$-bound PAHs concentrations in the RS and the IE. To establish a normal distribution, the PM$_{2.5}$ and PAHs values were log-transformed.

Results and Discussion

Concentration of PM$_{2.5}$

The average concentrations of PM$_{2.5}$ in RS and IE site were 43.3 ± 26.8 µg/m$^3$ (13.9–113.1 µg/m$^3$) and 40.4 ± 21.7 µg/m$^3$ (13.4–105.2 µg/m$^3$), respectively (Table 2). According to Ambient Air Quality Standard of the Pollution Control Department (PCD) in Thailand, the daily average of PM$_{2.5}$ values is 50 µg/m$^3$. During the sampling periods in RS and IE, the percentage of days above the daily PCD limit was 26.7% (12 days) and 28.9% (13 days), respectively. Furthermore, in RS, the proportion of days was 48.9% (22 days), and in IE, it was 53.3% (24 days), which was greater than the US-EPA National Ambient Air Quality Standard (NAAQS) of 35 µg/m$^3$. Apart from that, the daily PM$_{2.5}$ concentrations (43.3 ± 26.8 µg/m$^3$ and 40.4 ± 21.7 µg/m$^3$) found in this study were higher than those found in ambient air quality studies in an urban-industrial area, South Africa in winter (38.2 ± 8.4 µg/m$^3$) and summer (22.3 ± 4.1 µg/m$^3$) [17]. Li et al. [3] reported that the ambient of PM$_{2.5}$ concentration in Beijing (125.7 µg/m$^3$), Jinan (115.9 µg/m$^3$), and Shanghai (85.1 µg/m$^3$), which has influence from massive coal combustion was 2–3 times higher than the daily values in this study. The levels were also lower than the heating period (55.7 ± 35 µg/m$^3$) in Tangshan, China, which were emitted from heavy industries as well as a growth in energy consuming firms such as steel, coal, cement, and ceramics [26]. An average PM$_{2.5}$ concentration in this study was comparable to value obtained from high-traffic site (34.3 ± 24.8–41.5 ± 19.8 µg/m$^3$) and industrial site (60.0 ± 31.3 to 66.2 ± 30.5 µg/m$^3$) in Tehran, Iran [10]. Furthermore, the average PM$_{2.5}$ values measured in both sites were higher than that found in urban area in Chang Mai city in 2017 (6.07–79.9 µg/m$^3$; mean 35.8 ± 16.3 µg/m$^3$), which released from the high biomass burning period [14]. However, t-test was applied to distinguish the average PM$_{2.5}$ concentrations between the two Rayong city sites. There was no significant difference in PM$_{2.5}$ concentrations between the Rs and IE sites (p > 0.05). According to the findings, the two sites’ varied urban areas, IE s, and transportation networks may not significantly affect PM concentrations.

Concentration of PAHs in PM$_{2.5}$

Table 2: Concentration of 16-PAHs-bound PM$_{2.5}$ in RS and IE at Rayong city

| Parameters | RS (n = 45) | IE (n = 45) |
|------------|------------|------------|
| PM$_{2.5}$, µg/m$^3$ | Median ± SD | Max ± SD | Median ± SD |
| BaA | 1.20 ± 0.24 | 2.12 ± 0.33 | 2.10 ± 0.24 |
| BbF | 0.51 ± 0.16 | 0.83 ± 0.12 | 0.83 ± 0.12 |
| BaP | 0.52 ± 0.21 | 0.78 ± 0.21 | 0.78 ± 0.21 |
| BrA | 0.08 ± 0.04 | 0.10 ± 0.04 | 0.10 ± 0.04 |
| Car | 0.04 ± 0.02 | 0.06 ± 0.02 | 0.06 ± 0.02 |
| Chr | 0.07 ± 0.03 | 0.09 ± 0.03 | 0.09 ± 0.03 |
| DBA | 0.03 ± 0.02 | 0.04 ± 0.02 | 0.04 ± 0.02 |
| DBF | 0.02 ± 0.01 | 0.03 ± 0.01 | 0.03 ± 0.01 |
| DHH | 0.01 ± 0.005 | 0.01 ± 0.005 | 0.01 ± 0.005 |
| DHX | 0.01 ± 0.005 | 0.01 ± 0.005 | 0.01 ± 0.005 |
| DiBa | 0.01 ± 0.005 | 0.01 ± 0.005 | 0.01 ± 0.005 |
| DgA | 0.01 ± 0.005 | 0.01 ± 0.005 | 0.01 ± 0.005 |
| DpA | 0.01 ± 0.005 | 0.01 ± 0.005 | 0.01 ± 0.005 |
| FL | 0.01 ± 0.005 | 0.01 ± 0.005 | 0.01 ± 0.005 |
| FTH | 0.01 ± 0.005 | 0.01 ± 0.005 | 0.01 ± 0.005 |
| GbA | 0.01 ± 0.005 | 0.01 ± 0.005 | 0.01 ± 0.005 |
| GbP | 0.01 ± 0.005 | 0.01 ± 0.005 | 0.01 ± 0.005 |
| IcOc | 0.01 ± 0.005 | 0.01 ± 0.005 | 0.01 ± 0.005 |
| IcOH | 0.01 ± 0.005 | 0.01 ± 0.005 | 0.01 ± 0.005 |
| Ind | 0.01 ± 0.005 | 0.01 ± 0.005 | 0.01 ± 0.005 |

Further analysis of Table 2 revealed that the concentration of BaA, BbF, BaP, BrA, Car, Chr, DBA, DBF, DHH, DHX, DiBa, DgA, DpA, FL, FTH, GbA, and GbP in PM$_{2.5}$ in RS and IE were similar, while the concentrations of Chr, DBA, DBF, and DHH in RS were significantly higher than those in IE (p < 0.05).

https://oamjms.eu/index.php/mjms/index
detected in this study that excepted Acy and Ace. The carcinogenic PAHs (c-PAHs) were BaA, Chr, BkF, BbF, BaP, IcdP, and DahA, while non-carcinogenic PAHs (nc-PAHs) were Nap, Acy, Ace, Fla, Phe, Ant, Flu, Pyr, and BghiP [27]. The average daily concentrations of t-PAHs, c-PAHs, and nc-PAHs at RS site were 1.68 ± 1.53, 1.41 ± 1.37, and 0.45 ± 0.31 ng/m³, while values of t-PAHs, c-PAHs, and nc-PAHs at IE site were 1.34 ± 1.22, 1.16 ± 1.01, and 0.49 ± 0.46 ng/m³, respectively. The t-PAHs measured in both study sites were higher than an urban-industrial area in winter (0.07–0.92 ng/m³) and summer (0.04–0.88 ng/m³) by Morakinya et al. [17] in South Africa. The t-PAHs values were lower than those reported in a previous study in China [7], in which t-PAHs from urbanization and industrialization in Jinhua, Hangzhou, Lishui, Ningbo, and Zhoushan city in Zhejiang Province were 18.3 ± 16.0, 17.8 ± 15.8, 16.9 ± 15.2, 13.5 ± 10.0, and 7.5 ± 4.0 ng/m³, respectively. Furthermore, the values of t-PAHs obtained from Beijing, Jinan, and Shanghai ranged 23.2–819.8 ng/m³, 25.7–727.1 ng/m³, and 8.5–133.9 ng/m³, respectively [3]. Kongpran et al. [20] investigated that the average concentrations of t-PAHs in Upper North Thailand during the haze episode were 137.7 ± 80.4 ng/m³, which were 69–89 times higher than those both sites in this study. Moreover, the t-PAHs concentrations in Chiang Mai emitted from the high biomass burning period of 2016 from Thepnuan and Chantara [28] (10.23 ± 2.49 ng/m³) and of 2017 from Kawichai et al. [14] (9.22 ± 3.11 ng/m³) were higher than in this study (1.98 ± 1.96 ng/m³ in RS site and 1.50 ± 1.94 ng/m³ in IE site). However, t-test analysis was used to test the difference of the mean of t-PAHs concentrations between both sites. It was found that the values of t-PAHs in RS site were not significantly different in IE site (p > 0.05), while values of c-PAHs at both sites in Rayong city were significantly higher than those of nc-PAHs (p < 0.05). Kawichai et al. [14] reported that the high values of c-PAHs obtained from a busy urban area with the traffic density.

Figure 2 illustrates the values of the daily PM_{2.5} bound PAHs concentrations in both sites and percentage of individual PAHs. The t-PAHs concentrations at RS site in descending order were BaP (24%) > BbF = IcdP = DahA (11%) > BkF = BghiP (9%) > Chr = BaA (7%), while the concentrations of 16-PAHs at IE site in descending order were DahA (15%) > BaP (14%) > IcdP (13%) > BbF (12%) > BkF (11%) > Chr = BaA (7%). The result was same as Zhang et al. [5], the profile of PM_{2.5} in Northwest China emitted from coal combustion was found to be Pyr, BaA, Chr, and BbF, while the oil spill and leakage and volatilization during transportation and production processes in the petrochemical industry were Nap, Flu, Phe, and Ant. Furthermore, Fla, Chr, BkF, BaP, Pyr, and BghiP have been found in diesel engines and gasoline exhaust. Moreover, Kermani et al. [10] revealed that the main of PAHs in the traffic and industrial areas in Tehran, Iran, was BaP, DahA, BbK, and BbF in high-traffic regain, while the BbK, BkF, Chr, Pyr, and IcdP were found the industrial region. Zhang et al. [5] reported that the dominant PAHs in an industrial city in Northwest China were found to be Fla, Pyr, BaA, Chr, BbF, BaP, IcdP, and BghiP, which indicated characteristic of combustion emission. In South Africa, Morakinyo et al. [17] reported that the dominant PAHs in an industrial area in winter were Phe, Acy, Flu, and Flia. However, the average BaP concentrations in RS and IE sites were 0.98 ± 0.88 and 0.61 ± 0.31 ng/m³, respectively (Table 2). BaP is the carcinogenic substance (Group 1) of 16-PAHs, which is created by the International Agency for Research on Cancer [29]. However, the concentrations of BaP at both sites in Rayong city were lower than the US-EPA recommended annual limit value of 1 ng/m³ [26].

The 16-PAHs were classified as Nap, Acy, Ace, Flu, Phe, and Ant based on the number of aromatic rings in the low molecular weight (LMW; 2–3 rings), and Fla, Pyr, Chr, and BaA based on the number of aromatic rings in the medium molecular weight (MMW; 2–3 rings) (MMW; 4 rings). BbF, BkF, BaP, IcdP, DahA, and BghiP were classified using the high molecular weight (HMW; 5–6 rings) system. It was found that the predominant PAHs in RS and IE site were 5–6 rings (HMW). The component of PM_{2.5}-bound PAHs emitted from RS sites in descending order was HMW (69%) > MMW (15%) > LMW (8%), while values of PAH in IE site in descending order were HMW (66%) > MMW (16%) > LMW (9%). The study about PM_{2.5}-bound PAHs emitted from the economic and financial center of Shanghai in China revealed that the dominant of PAHs concentrations at was HMW (51%), MMW (36%), and LMW (13%), which the city is near the sea [3]. Coincidently, the study from Lui et al. [25] revealed that the dominant of street dust sample was MMW- and HMW-PAHs. A large proportion of 4–6 rings PAHs (84.0–91.6%) was the dominant PM_{2.5}-bound PAHs collected from five cities of urbanization and industrialization in Zhejiang Province, China [7].
The diagnostic ratios are values ratios of some PAHs as use for identifying pollution emission source from sample collection in different environmental compartments [30]. The properties of the specific source were explained using PAH isomer pair ratios. To identify the source of PAHs, the Fla/(Fla+Pyr) and IcdP/(IcdP+BghiP) ratios were evaluated. Biomass and coal combustion were predicted by a ratio >0.5, while fossil fuel combustion or petroleum input was predicted by a ratio <0.5 [31]. Ratio of BaA/(BaA+Chr) implied petrogenic source biomass and coal combustion (<0.20), (0.20–0.35) and vehicular emission to coal combustion or pyrogenic source (>0.35). The ratio value of Ant/(Ant+Phe) <0.10 was represented to petrogenic or vehicular combustion, while a ratio >0.10 indicated pyrogenic source [31]. The obtained average values of the diagnostic ratios in at both sites in Rayong city were applied to identify sources of PAHs, as shown in Figure 3. It was found that the ratio of Ant/(Ant+Phe) and BaA/(BaA+Chr) presented a different main source of PM$_{2.5}$ in Rayong city. The values of BaA/(BaA+Chr) ratios were 0.40–0.73 from roadside (RS) and 0.38–1.00 from IE, it implied vehicular emission to coal combustion or pyrogenic source, while the ratio values of Ant/(Ant+Phe) were higher than 0.10 indicating pyrogenic source (0.17–1.00 in RS and 0.12–1.00 in IE). Furthermore, the previous studies of BaA/(BaA+Chr) ratios can display source of PM$_{2.5}$-bound PAHs which were <0.49 from gasoline emission and 0.49–0.73 from diesel emission [3]. The diagnostic ratios in this study were consistently resulted with the reference sources for emission from vehicular emission and coal combustion, which was same the several studies by Li et al. [3], Morakinyo et al. [17], Fang et al. [26], and Kongpran et al. [20]. The presence of six coal-fired power plants in Rayong city [32]. The air mass backward trajectories presented that air mass arriving at both sites in Rayong city during August 2019–February 2020 using cluster analysis for air mass movement (Figure 4). The 3-day (72 h) reverse trajectory was computed using the National Oceanic and Atmospheric Administration’s Hybrid Single-Particle Lagrangian Integrated Trajectory (HYSPLIT) model at altitudes of 10 m and 1000 m above ground level [33]. In August and September 2019, the southwest monsoon season in Thailand absorbed air mass from the Andaman Sea and Indian Ocean at both 10 m and 1000 m altitudes (14.8–66.7%). From October 2019 to February 2020, the northeast monsoon in Thailand with a dry season originated from distribution air mass (8.9-67.2 %) from the South China Sea and China. However, the biomass open burning season in the northern and northeastern parts of Thailand occurs in January and February 2020, coinciding with the pollution season. It was discovered that the air mass from the Gulf of Thailand had a cluster pattern (14.8–51.6 %). Therefore, the results of ratio analysis indicated that source of PM$_{2.5}$-bound PAHs concentration measured at both sites might be a significant source of vehicle emission and coal combustion.

### Health risk assessment of PM$_{2.5}$-bound PAHs

Carcinogenic risk assessment of PM$_{2.5}$

Figure 5 displays the individual lifetime cancer risk ($R_{ic}$) to evaluate the long-term PM$_{2.5}$ exposure. The average $R_{ic}$ values of children in RS site were $3.81 \times 10^{-5}$ (1–<2 years), $2.60 \times 10^{-5}$ (2–<3 years), $4.29 \times 10^{-5}$ (3–<6 years), and $2.45 \times 10^{-5}$ (6–<11 years), while the values in IE site were $3.56 \times 10^{-5}$ (1–<2 years), $2.43 \times 10^{-5}$ (2–<3 years), $4.01 \times 10^{-5}$ (3–<6 years), and $2.28 \times 10^{-5}$ (6–<11 years). The $R_{ic}$ values of children were higher than $1 \times 10^{-5}$ indicated potential risk. The $R_{ic}$ levels of teenager in RS and IE site were $7.67 \times 10^{-5}$ (11–<16 years) to $4.83 \times 10^{-5}$ (16–<21 years) and $7.16 \times 10^{-6}$ (11–<16 years) to $4.51 \times 10^{-6}$ (16–<21 years), respectively. The $R_{ic}$ values less than $1 \times 10^{-5}$ are represented a negligible cancer risk of teenager. Moreover, the $R_{ic}$ values of adults were $3.79 \times 10^{-5}$ in RS and $3.54 \times 10^{-5}$ in IE site, which indicated potential risk ($>1 \times 10^{-5}$). The societal lifetime cancer risk (cases/million population) was estimated by multiplying the $R_{ic}$ by 1 million citizens [15]. The societal $R_{ic}$ values of long-term PM$_{2.5}$ exposure of children, teenager, and adults in Rayong city were 23–40, 5–7, and 40 cases/people, respectively. However, the characterization of anatomy/physiology for children did not fully. Thus, the inhalation rate of children from the behavior and activity levels differs from those of adults. Children may have a faster metabolic rate and oxygen demand rate per unit of body weight than adults due to their rapid growth and substantially larger lung surface area per unit of body weight. Thus, the fine particles deposition interacts with respiratory tract may be greater in children. In addition, the rate of fine particle inhalation can be significantly associated with overweight [23]. Another factor influencing inhaled particulate matter is associated with the diameter of the airway with a generally inverse relationship [34].

### Carcinogenic risk assessment of PAHs

Table 3 shows the values of TEQ$_{Bap}$ of both sampling sites. Both TEQ$_{Bap}$ values obtained from
RS and IE sites in Rayong city were 0.55 ± 0.78 and 0.29 ± 0.42 ng/m$^3$, respectively. Comparing the TEQ$_{BaP}$ value of this study with the previous study conducted in urban and industrial area in South Africa by collecting PM$_{2.5}$ samples, it was found that the values of TEQ$_{BaP}$ in our study (0.32–0.59 ng/m$^3$) were similar to the previous work (0.17–0.43 ng/m$^3$) [17]. In comparison with the study in Zhejiang province, China [7], where PM$_{2.5}$ samples were collected from five cities of urbanization and industrialization, the TEQ$_{BaP}$ value in this study was lower than the value obtained from China (1.2 ± 0.7–3.1 ± 2.6 ng/m$^3$). The TEQ$_{BaP}$ values observed in this study were substantially lower than the European recommendation (1 ng/m$^3$) but greater than the recommended value by governments of the UK and Sweden which are 0.25 and 0.1 ng/m$^3$, respectively [35]. The ECR calculated based on TEQ$_{BaP}$ obtained from both sites in Rayong city, as shown in Table 4. The average ECR values of TEQ$_{BaP}$ in RS site were 1.11 × 10$^{-5}$, while the values of ECR in IE site were 5.74 ×

Figure 4: The cluster analysis of 3 days backward trajectories in Rayong city, Thailand; (a). August 2019; (b). September 2019; (c). October 2019; (d). November 2019; (e). December 2019; (f). January 2020; and (g). February 2020

Figure 5: The estimation of the individual lifetime cancer risk (R$_{ic}$) from PM$_{2.5}$ exposure in Rayong city
10^6. However, the ECR values of cancer risk at both sites in Rayong city were potential risk. Thus, if 1 million individual people were exposed to PAHs for 70 years in RS and IE site may develop 11 and six people to be cancer risks, respectively. In general, the values of ECR in Rayong city were found to be higher at RS site than IE site. Tafhvaee et al. [36] assessed the lung cancer risk exposure to PAHs in PM_{2.5} that was collected from Central Tehran, Iran. The mean ECR outdoor exposure from the petrogenic sources and petroleum residues, natural gas and biomass burning, industrial emissions, diesel exhaust emissions, and gasoline exhaust was 1.08 × 10^{-6}, 6.60 × 10^{-7}, 1.75 × 10^{-6}, 2.46 × 10^{-6}, and 4.60 × 10^{-7}, respectively. As a result, diesel exhaust and industrial pollution may be the largest contributors to total cancer and mutagenic risk.

The estimated ILCR values of carcinogenic PAHs for human health risk are presented in Table 4. The average values of inhalation cancer risk for children, teenager, and adults in RS site were 4.26 × 10^{-7} to 6.87 × 10^{-7}, 4.44 × 10^{-7} to 5.79 × 10^{-7}, and 3.77 × 10^{-7}, while values of IE site were 2.20 × 10^{-7} to 3.19 × 10^{-7}, 1.95 × 10^{-7} to 2.12 × 10^{-7}, and 1.94 × 10^{-7}, respectively. However, the ILCR values for human health risk in Rayong city were lower than 10^{-6}, thus expressing virtual safety. In comparison with the study site, where the ILCR values were collected IE, the values were higher than the values obtained from roadside site (RS) in this study. Kermani et al. [10] reported that the mean ILCR values of inhalation PM_{2.5}-bound PAHs through high-traffic and industrial region for children and adults in Iran were 1.34 × 10^{-11} to 1.72 × 10^{-10} for high-traffic region and 2.89 × 10^{-11} to 3.71 × 10^{-10} for industrial region, which lower than in this study. Moreover, some previous studies reported that the ILCR model analysis of PAHs inhalation was negligible human health risk [7], [17], [25], [37], which could be explained by cancer risk levels for ingestion and dermal contact route including the different dermal exposure area, exposure duration, and ingest a variety of contaminated dusts as a result of hand-to-mouth actions [38]. Childhood, on the other hand, is an especially sensitive stage of development, with cancer risks up to an order of magnitude higher per year of exposure than in adults [39]. Children’s inhalation of PAHs per unit of body weight of the same volume pollutant was considered higher than that of adults due to their lower body weight [25].

## Conclusions

The concentrations of PM_{2.5} and total PAHs were measured at RS and IE in Rayong city, Thailand. The average PM_{2.5} concentrations at RS and IE site were 43.3 ± 26.8 and 40.4 ± 21.7 µg/m³, while the values of t-PAHs in both sites were 1.68 ± 1.53 and 1.34 ± 1.22 ng/m³, respectively. However, it was found that the PM_{2.5} and t-PAHs values in IE site were not significantly different those in RS site (p > 0.05). The correlation between concentrations of PM_{2.5} and t-PAHs was not significantly correlated (p > 0.01). The observed decline in PAHs concentrations can be explained by the breakdown or degradation of PAHs caused by a photochemical reaction in the presence of increased temperatures and sunlight.

Moreover, the PM_{2.5} values were higher than the standard levels regulated by various organizations concerned with air quality and health. The percentage of days that exceed the daily PCD standard (50 µg/m³) during the sampling periods in RS and IE was 26.7% and 28.9%, while the percentage of days higher than the value of the NAAQS and US-EPA (35 µg/m³) was 48.9% and 53.3%, respectively.

The carcinogenic risk assessment of PM_{2.5} and PAHs in PM_{2.5} concentrations was calculated based on the individual lifetime cancer risk (R_{50}), the toxicity equivalent (TEQ_{equiv}), the ECR, and the ILCR. The results revealed that R_{50} for PM_{2.5} values for children and adults at both sites in Rayong city was indicated acceptable cancer risk (10^{-6}–10^{-4}). Exposure to PAHs in PM_{2.5} through the inhalation pathway in Rayong city was obviously potential risk of cancer risk (10^{-6}–10^{-4}) based on calculate by the ECR model. Moreover, the ILCR values of PAHs for different age groups were less than 10^{-6}, which were negligible cancer risk in Rayong city. Therefore, the result of PM_{2.5} concentrations has important recommendation for the environment management and protection in Rayong city that distribution from vehicle emission and coal combustion.

| Parameters | RS (n = 45) | IE (n = 45) |
|------------|------------|-------------|
|            | Range      | Mean        | Median     | Range      | Mean        | Median     |
| TEQ (ng/m³) | 1.53 × 10^{-3.76} | 0.55 ± 0.78 | 0.06       | 5.20 × 10^{-1.31} | 0.29 ± 0.42 | 0.03       |
| ECR        | 3.07 × 10^{-7.54 × 10^4} | 1.11 × 10^3 | 1.16 × 10^4 | 1.04 × 10^{-3.64 × 10^4} | 5.74 × 10^{-3} | 5.27 × 10^{-7} |
| ILCR       | Children   | 1–<2 years | 1.18 × 10^{-1.89 × 10^4} | 4.26 × 10^7 | 4.43 × 10^8 | 4.00 × 10^{-1.01 × 10^4} | 2.20 × 10^7 | 2.02 × 10^4 |
|            | 2–<3 years | 1.71 × 10^{-1.20 × 10^4} | 6.18 × 10^7 | 6.43 × 10^8 | 5.80 × 10^{-1.13 × 10^4} | 3.19 × 10^7 | 2.93 × 10^4 |
|            | 3–<6 years | 1.59 × 10^{-1.30 × 10^4} | 5.75 × 10^7 | 5.98 × 10^8 | 5.39 × 10^{-1.36 × 10^4} | 2.97 × 10^7 | 2.72 × 10^4 |
|            | 6–<11 years | 1.90 × 10^{-1.67 × 10^4} | 6.87 × 10^7 | 7.15 × 10^8 | 4.48 × 10^{-1.13 × 10^4} | 2.47 × 10^7 | 2.26 × 10^4 |
| Teenagers  | 11–<16 years | 1.60 × 10^{-1.93 × 10^4} | 5.79 × 10^7 | 6.03 × 10^8 | 3.86 × 10^{-1.76 × 10^4} | 2.12 × 10^7 | 1.95 × 10^4 |
|            | 16–<21 years | 1.23 × 10^{-1.30 × 10^4} | 4.44 × 10^7 | 4.62 × 10^8 | 3.54 × 10^{-1.87 × 10^4} | 1.95 × 10^7 | 1.79 × 10^4 |
| Adults     | ≥21 years  | 1.04 × 10^{-2.56 × 10^4} | 3.77 × 10^7 | 3.92 × 10^8 | 3.53 × 10^{-2.84 × 10^4} | 1.94 × 10^7 | 1.79 × 10^4 |

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