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Incremental Lifetime Cancer Risk of $PM_{2.5}$ Bound Polycyclic Aromatic Hydrocarbons (PAHs) Before and After the Wildland Fire Episode

Siwatt Pongpiachan*

NIDA Center for Research & Development of Disaster Prevention & Management, School of Social and Environmental Development, National Institute of Development Administration (NIDA), Bangkok 10240, Thailand

ABSTRACT

In Northern Thailand, wildland fire during cold period releases large amounts of smoke and fine particles into the atmosphere. The fine particles include several persistent organic compounds such as PAHs. In this study, $PM_{2.5}$-bound PAH concentrations in the air of nine administrative provinces, namely Chiang-Mai, Chiang-Rai, Nan, Phayao, Mae Hong Son, Phrae, Lampang, Lamphun, Uttaradit (N Thailand) were determined during the wildland fire and non-wildland fire seasons. The monitoring strategy comprised two campaigns in each season. $PM_{2.5}$ was collected using MiniVolTM portable air samplers (Airmetrics) with quartz fibre filters. Both PAHs and their $B[a]P_{Equivalent}$ concentrations of other urban cities around the world were significantly higher than those of northern provinces for both seasons. The average cancer risks observed at nine administrative provinces were $8.525 \times 10^{-4} \pm 3.493 \times 10^{-3}$ and $2.558 \times 10^{-3} \pm 6.986 \times 10^{-3}$ for ingestion rate of 50 and 100 mg day^{-1}, respectively. The excess cancer risks of world cities for ingestion rate of 50 and 100 mg day^{-1} were much higher than those of Northern Thailand for 851 and 567 times in that order. Dust ingestion was exceedingly critical to non-dietary PAH exposure in comparison with $PM_{2.5}$ inhalation. These results are in good agreement with those of previous studies, underlining the significance of indoor air quality on long-term adverse respiratory diseases in Asian cities.

Keywords: Wildland fire; PAHs; Northern Thailand; $PM_{2.5}$; Incremental lifetime cancer risk.

INTRODUCTION

Physico-chemical behavior of particulate polycyclic aromatic hydrocarbons (PAHs) have been extensively investigated in most cities worldwide during the past few years (Dachs *et al.*, 2002; Bartkow *et al.*, 2004; Ciganek *et al.*, 2004; Dallarosa *et al.*, 2005a, b; Fang *et al.*, 2005; Schnelle-Kreis *et al.*, 2005; Pongpiachan, 2006; Wang *et al.*, 2006; Vasilakos *et al.*, 2007; Pongpiachan, 2013a, b; Pongpiachan *et al.*, 2013a, b). PAHs are a group of organic compounds that are generated during the incomplete burning of lignite (Liu *et al.*, 2012), fuels (Lu *et al.*, 2012), volcanic eruption (Schwandner *et al.*, 2013), biomass (Sanchis *et al.*, 2014), garbage (Chen *et al.*, 2013), or other organic substances, such as tobacco (Chalbot *et al.*, 2012). PAHs are highly toxic organic compounds that can cause a range of adverse health effects (e.g., cancer, endocrine disruption, reproductive and developmental effects) (Hoyer, 2001; Matsui, 2008; Liao *et al.*, 2011; Wickramasinghe *et al.*, 2012). In spite of several studies connected with $PM_{2.5}$-bound PAH contents in different cities, the insight of its fate and behaviour during the episode of wildland fire is severely limited. Early findings apparently focused on the air quality level worsened by agricultural waste burnings, wildland fires, and accidental biomass combustions associated with trans boundary haze pollution from neighbouring countries of Thailand (Pengchai *et al.*, 2009; Oanh *et al.*, 2011; Huang *et al.*, 2013; Shi *et al.*, 2014). Apart from biomass burnings, vehicular exhausts, domestic heating, industrial emissions and other fugitive sources can be acknowledged as important sources of PAHs (Riva *et al.*, 2011; Wei *et al.*, 2012; Amodio *et al.*, 2013; Slezakova *et al.*, 2013; Mu *et al.*, 2014). As a consequence, there is a growing concern for human exposure risk to airborne carcinogens.

Previous epidemiological studies indicate that lung cancer evidences were substantially higher among residents in Northern Thailand, particularly in women (Pisani *et al.*, 2006; Bumroongkit *et al.*, 2008; Kamnerdsupaphon *et al.*, 2008). For instance, a retrospective study used patient's records at the Chiang Mai Cancer registry and Maharaj Nakorn Chiang Mai Hospital from January 2001 to December 2005 revealed a comparatively high lung and breast cancer incidences (Kamnerdsupaphon *et al.*, 2008). Another epidemiological study raised concerns over pesticide exposure or habitation

* Corresponding author.
Tel.: 00 66 2 727 3113; Fax: 00 66 2 732 0276
E-mail address: pongpiajun@gmail.com

in poorly ventilated houses may instead be related to the tumorigenesis of lung cancer patients in upper Northern Thailand (Bumroongkit *et al.*, 2008). On the contrary, a previous study highlighted "tobacco smoking" as a main cause responsible for 96% of male and 64% of female lung cancer incidences found in rural area of Northern Thailand (Pisani *et al.*, 2006).

In spite of some discrepancies over the causes of high lung cancer incidences, there has been no researches aiming on neither chemical characterization nor human exposure risk assessment of $PM_{2.5}$ bound PAHs before and after the wildland-burning episode in Northern part of Thailand. Overall, the major objectives of this study are to (*i*) use three different toxicity equivalent concentration (*TEQ*) equations for assessing the risk of exposure to PAHs, (*ii*) calculate daily exposure of $PM_{2.5}$ bounded PAHs, and (*iii*) conduct incremental lifetime cancer risk of $PM_{2.5}$ bounded PAHs *via* ingestion, inhalation, and dermal contact with dust particles at nine administrative provinces in northern part of Thailand.

EXPERIMENTAL

Descriptions of Observatory Sites

All nine administrative provinces of Northern Thailand are classified as tropical wet and dry climate (Köppen climate classification: Aw). The weather is fairly dry and warm in winters while the temperatures rise until April with the hottest average daily maximum at 34.9°C (94.8°F). The monsoon season runs from late April through October, with heavy rain and somewhat cooler temperatures during the day, although nights remain warm. The geographical positions of each monitoring station were written as below: Chiang-Mai Observatory Site (CMOS): (E: 498805, N: 2077713), Chiang-Rai Province Observatory Site (E: 593783, N: 2258302), Nan Province Observatory Site (NPOS; Thewarat Hotel; E: 687123, N: 2077209), Phayao Province Observatory Site (PYOS; Arunothai Coffee House Homestay; E: 594420, N: 2119215), Mae Hong Son Province Observatory Site (MHOS; Mae Hong Son Provincial Forestry Office; E: 391834, N: 2134869), Phrae Province Observatory Site (PHOS; Nana Charoenmuang Hotel; E: 620935, N: 2006155), Lampang Province Observatory Site (LMOS; Maemoh Training Center; E: 568200, N: 2020017), Lamphun Province Observatory Site (LPOS; Lamphun Provincial Administration Organization Stadium; E: 500441, N: 2052987), Uttaradit Province Observatory Site (UTOS; OUM Hotel; E: 615923, N: 1948269) (See Fig. 1). All air quality observatory sites were situated in the city center of each administrative province of Northern Thailand. The population density vary widely, from the relatively urbanized Chiang-Mai and Chiang-Rai Provinces with the range of 80 to 100 people km^{-2}, to the mostly-forest area of Mae Hon Song Province with less than 40 people km^{-2} (See Fig. 2). It is also worth mentioning that Northern Thailand contrasts remarkably from other regions of Thailand. All monitoring stations are geologically categorized by several mountainous belts, which continue from the Shan Hills in bordering RUM and LPDR, and climatically highlighted by a tropical savanna.

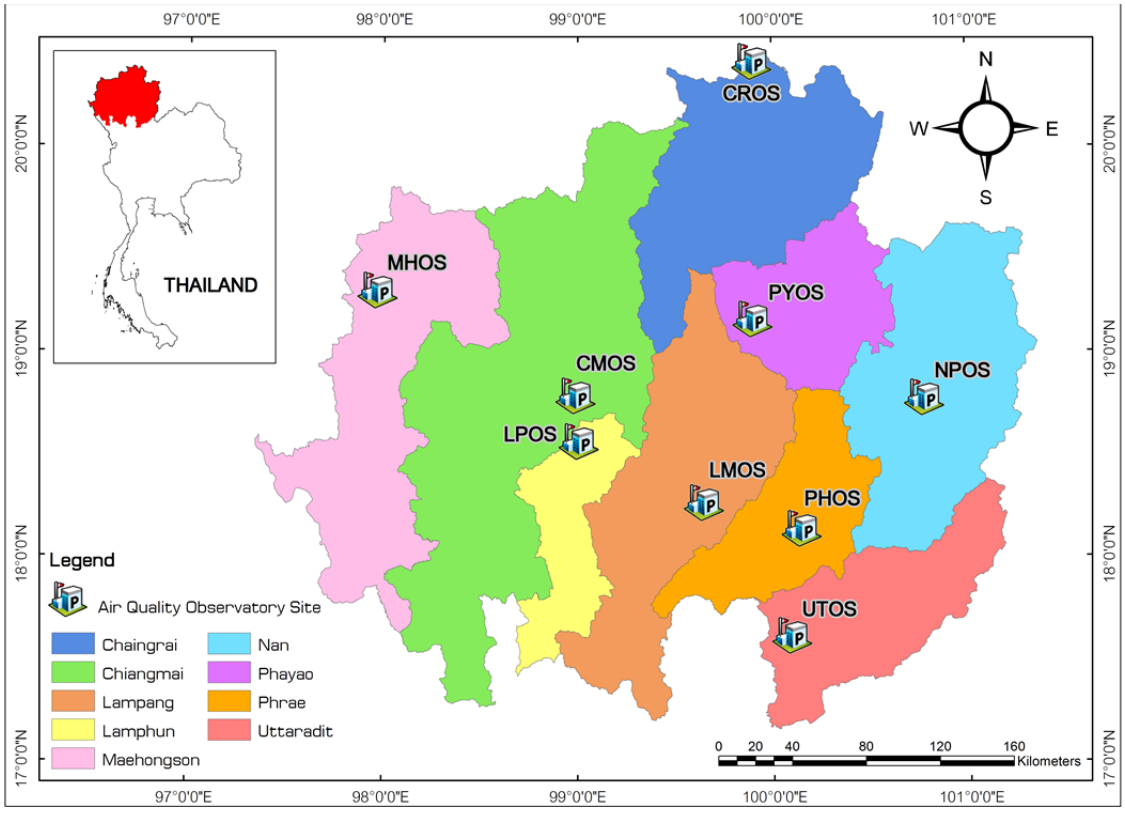

Fig. 1. $PM_{2.5}$ monitoring stations in nine administrative provinces of Northern Thailand.

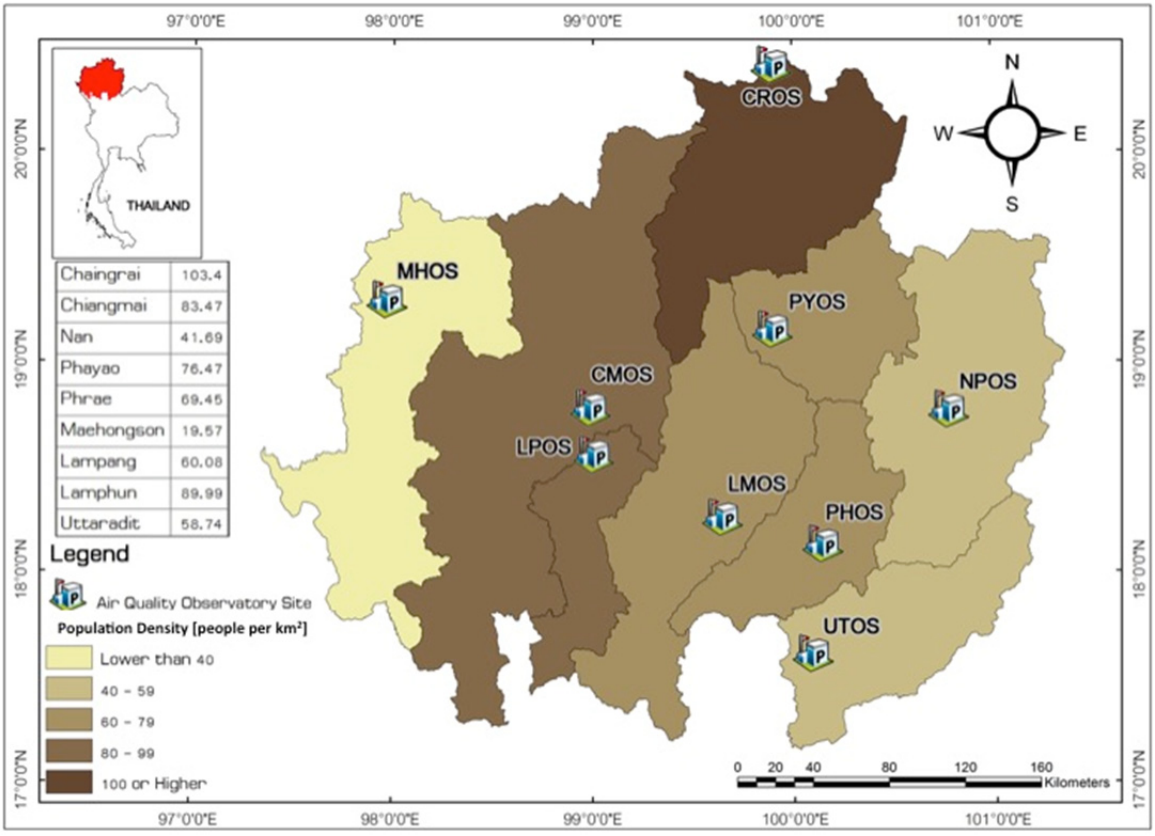

Fig. 2. Population density in nine administrative provinces of Northern Thailand.

Sampling of $PM_{2.5}$

Samplings of $PM_{2.5}$ were conducted and collected every week on pre-baked (550°C for 12 h) quartz-fiber filters (QFFs; Whatman 47 mm; Article No., 28418542 (US reference)) with the assistance of MiniVolTM portable air samplers (Airmetrics) using the flow rate of 5 litres minute^{-1} through a particle size separator (impactor) and consequently through a 47 mm filter. More details of the $PM_{2.5}$ sampling method was provided in "EPA Quality Assurance Guidance Document: Method Compendium, Field Standard Operating Procedures for the $PM_{2.5}$ Performance Evaluation Program, United States Environmental Protection Agency Office of Air Quality Planning and Standards" (U.S. EPA, 2002). The weight measurement of $PM_{2.5}$ was strictly followed the instructions of US-EPA Quality Assurance Document: Method Compendium, $PM_{2.5}$ Mass Weighing Laboratory Standard Operating Procedures for the Performance Evaluation Program, United States Environmental Protection Agency Office of Air Quality Planning and Standards (U.S. EPA, 1998) with the employment of micro balances (Mettler Toledo, New Classic MF, MS205DU, Switzerland). The sampling campaigns can be separated into two groups according to the monitoring period during which they were conducted: *Campaign-I* was conducted before the "wildland fire episode" in the winter of 2012 (i.e., from the 7th to 22nd of December 2012), while air sampling during *Campaign-II* was done in March 2013 (i.e., from the 4th to 19th of March 2013).

Campaign-I (Nine Northern Provinces-I (NNP-I); Sampling before the Haze Episode)

Observations of $PM_{2.5}$ at *cluster-I* sampling stations (CROS, PYOS and NPOS), *cluster-II* sampling stations (LMOS, PHOS and UTOS) and *cluster-III* sampling stations (MHOS, CMOS and LPOS) was conducted synchronously daily from the 28th of November to the 4th of December 2012, from the 7th to 13th of December 2012 and from the 16th to 22nd of December 2012, respectively.

Campaign-II (Nine Northern Provinces-II (NNP-II); Sampling after the Haze Episode)

Air quality observations at *cluster-I* monitoring stations (CROS, PYOS and NPOS), *cluster-II* monitoring stations (LMOS, PHOS and UTOS) and *cluster-III* monitoring stations (MHOS, CMOS and LPOS) was performed synchronously daily from the 23th of February to the 2nd of March 2013, from the 4th to 11th of March 2013 and from the 13th to 20th of March 2013, respectively.

Since the two campaigns consist of only three weeks durations, it is crucial to carefully investigate seasonal and daily variations, which can be effected by meteorological conditions. The seasonal representativeness of these campaigns should be validated and discussed. As displayed in Fig. 3, the hotspot numbers in northern Thailand provided by the Centre for Remote Imaging, Sensing and Processing (CRISP), the National University of Singapore, were classified into types namely Campaign-I and Campaign-II. The average hotspot number during Campaign-II (i.e., 532

± 543) was significantly ($p < 0.05$) higher than those of Campaign-I (i.e., 96 ± 92), highlighting the importance of biomass burnings in March 2013 and thus the selected sampling period can be used to justify the seasonal representativeness of monitoring campaigns.

Polycyclic Aromatic Hydrocarbons (PAHs)

A mix standard solution of 13 native PAHs [Norwegian Standard (NS 9815: S-4008-100-T): phenanthrene (Phe), anthracene (An), fluoranthene (Fluo), pyrene (Pyr), benz[*a*]anthracene (B[*a*]A), chrysene (Chry), benzo[*b*]fluoranthene (B[*b*]F), benzo[*k*]fluoranthene (B[*k*]F), benzo[*a*]pyrene (B[*a*]P), benzo[*e*]pyrene (B[*e*]P), indeno[1,2,3-*c,d*]pyrene (Ind), dibenz[*a,h*]anthracene (D[*a,h*]A), and benzo[*g,h,i*]perylene (B[*g,h,i*]P) and a mix of recovery internal standard (IS) PAHs [d_{12}-perylene (d_{12}-Per) and d_{10}-fluorene (d_{10}-Fl)] were ordered from Chiron AS (Stiklestadveine 1, N-7041 Trondheim, Norway) and used for this study. The exceptionally low concentrations of PAHs in $PM_{2.5}$ need their pre-concentration through Soxhlet extraction and fractionization prior to their quantitative analysis on a gas chromatograph coupled to a mass spectrometer (Shimadzu GCMS-QP2010 Ultra system with 60 m length × 0.25 mm i.d. capillary column of Agilent JW Scientific DB- 5 GC columns). The analytical procedure used in this study and relevant information has been explained in previous publications (Pongpiachan, 2009, 2011, 2013a, b; Pongpiachan *et al.*, 2013a, b). Briefly, all QFFs samples were chemically extracted with dichloromethane (Fisher Scientific, HPLC grade) by employing the Soxhlet apparatus.

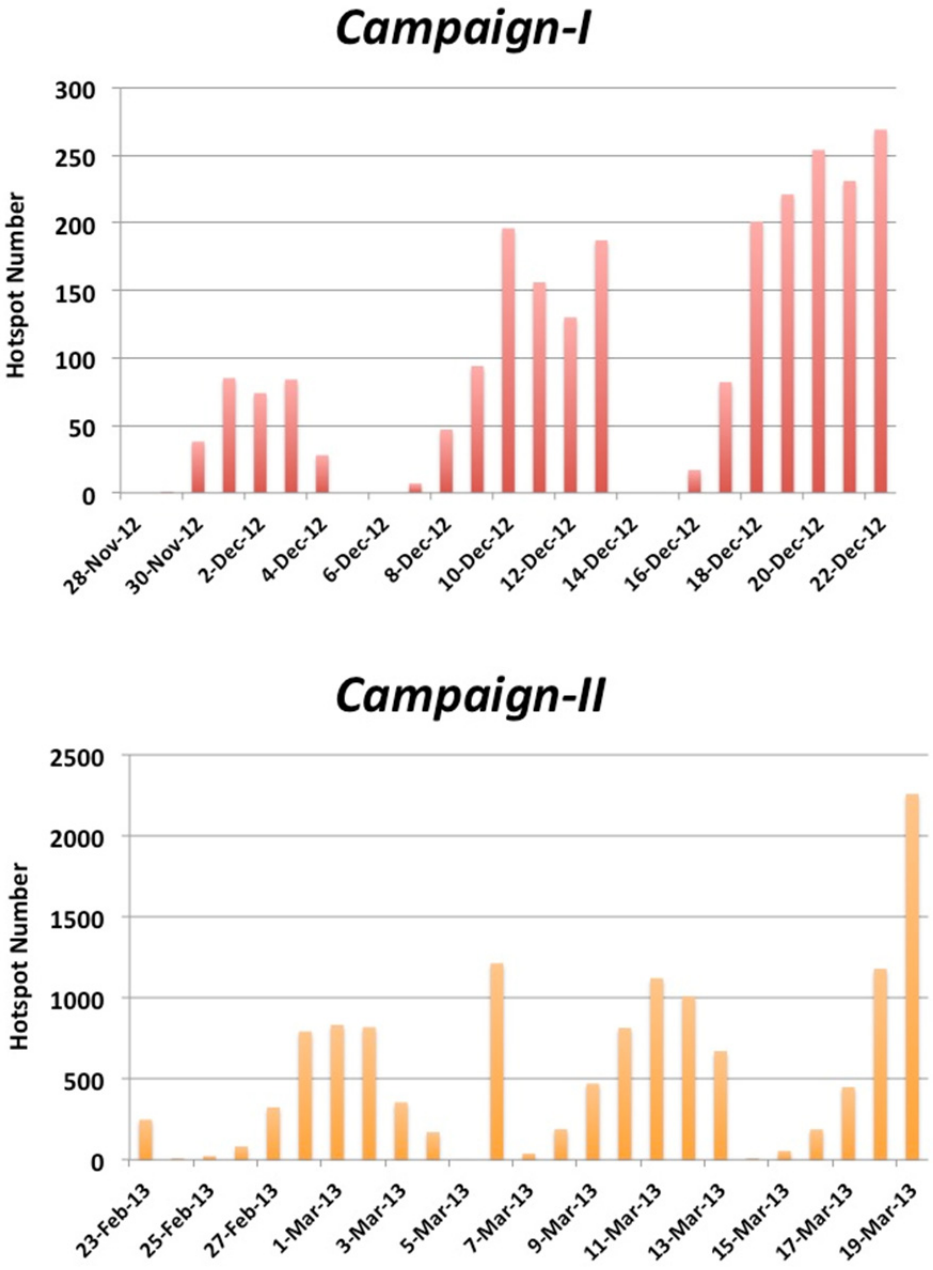

Fig. 3. Hot spot numbers before and after burning episodes in nine administrative provinces of Northern Thailand.

The QFFs samples were spiked with known amount of deuterated internal standards (i.e., d_{10}-fluorene and d_{12}-perylene) prior to the Soxhlet extraction. The fractionization protocol was started with the elution of PAH fraction with15 mL of 4:6 (v/v) toluene:hexane by using silica gel. Subsequently, the organic extract was rotary evaporated and reduced the volume with the assistance of gentle nitrogen stream to almost dryness until the final volume was 100 μL. More details of GC/MS method were provided in previous studies and will not be discussed here (Pongpiachan *et al.*, 2009, 2011). Both precisions and accuracies were quarantined by applying the standard reference material (SRM 1941b: National Institute of Standard and Technology).

Health Risk Assessment of PAHs

Toxicity Equivalent Concentration (TEQ)

The toxicity equivalent concentration (*TEQ*) equation is broadly used for assessing the risk of exposure to PAHs, which can be calculated as described bellows (Yang *et al.*, 2007; Yu *et al.*, 2008; Pongpiachan *et al.*, 2013b):

$$TEQ = \sum_i [C_i \times TEF_i] \quad (1)$$

here, C_i and TEF_i are acronyms for the concentrations of individual PAH and the toxic equivalency factors, respectively. Since the poisonousness of a single PAH congener can differ by orders of degree, it is essential to show the toxicity of PAHs in terms of the most toxic form of PAHs, B[*a*]P. By employing *TEF*, the toxicity of a combination of PAH congeners can be evaluated in a single number, which is generally referred as the toxic equivalency, *TEQ*. It is a specific number causing from the product of the concentration and individual *TEF* values of each PAH, as described in Eq. (1). In this study, three *TEQ* equations were used with the citations of Nisbet and Lagoy (1992) (Eq. (2)), US EPA (1993) (Eq. (3)), and Cecinato (1997) (Eq. (4)). In these three *TEQ* formulas, the acronyms for the PAH congeners represent their concentrations.

$$\begin{aligned} TEQ_{Nisbet\ and\ Lagoy} &= 0.001\left(Phe + Fluo + Pyr\right) \\ &+0.01\left(An + B\left[g,h,i\right]P + Chry\right) \\ &+0.1\left(B\left[a\right]A + B\left[b\right]F + B\left[k\right]F + Ind\right) \\ &+B\left[a\right]P + D\left[a,h\right]A \end{aligned} \quad (2)$$

$$\begin{aligned} TEQ_{US-EPA} &= 0.06\left(B\left[a\right]A\right) + 0.07\left(B\left[b\right]F + B\left[k\right]F\right) \\ &+B\left[a\right]P + 0.08\left(Ind\right) + 0.6\left(D\left[a,h\right]A\right) \end{aligned} \quad (3)$$

$$\begin{aligned} TEQ_{Cecinato} &= 0.01\left(Chry\right) \\ &+0.1\left(B\left[a\right]A + B\left[b\right]F + B\left[k\right]F + Ind\right) \\ &+B\left[a\right]P + D\left[a,h\right]A \end{aligned} \quad (4)$$

In addition, the abbreviations of Σ3,4-rings PAHs and Σ5,6-rings PAHs stand for the sum of Phe, An, Fluo, Pyr, B[*a*]A, Chry+Tri and B[*b*+*k*]F, B[*e*]P, B[*a*]P, Ind, D[*a,h*]A, B[*g,h,i*]P, respectively.

Daily Exposure of $PM_{2.5}$ Bounded PAHs

The calculations of daily exposure of $PM_{2.5}$ bounded PAHs were conducted by using three different *TEQ* values as previously described in section 2.3.1.

$$M_{PDI-ingestion} = \sum_{13} \frac{TEQ_{\mathrm{PM2.5}} \times M_{DID}}{1{,}000} \quad (5)$$

M_{DID} (mg day^{-1}) represents the daily intake of dust by either adults or children. In this study, the M_{DID} values of 4.16–100 and 55–200 mg day^{-1} were applied for adults and children, respectively (U.S. EPA, 1997). As a consequence, 4.16 and 55 mg day^{-1} for adults and children were assumed as comparatively low exposure situation whilst 100 and 200 mg day^{-1} were taken into account as relatively high exposure situation, correspondingly. A similar concept was applied to the calculation of $PM_{2.5}$ bounded *TEQs* daily exposure via inhalation ($M_{\mathrm{PDI\text{-}inhalation}}$; ng day^{-1}), which is based on Eq. (6):

$$M_{PDI-inhalation} = IR_{inhalation} \sum_{13} TEQ_{\mathrm{PM2.5}} \quad (6)$$

where, $\sum_{13} TEQ_{\mathrm{PM2.5}}$ (ng m^{-3}) is the sum of the *TEQ* contents of the 13 PAHs measured in $PM_{2.5}$. The inhalation rate ($IR_{\mathrm{inhalation}}$) used in this research was 10 and 20 m^3 day^{-1} for children and adults respectively (SFT, 1999).

Incremental Lifetime Cancer Risk of $PM_{2.5}$ Bounded PAHs

Assuming that (*i*) there are only ingestion, inhalation, and dermal contact with dust particles, which can be considered as three major pathways for human exposure; (*ii*) the total carcinogenic risk could be evaluated by using *TEQ* values as previously mentioned in section 2.3.1; (*iii*) intake rates and particle emission can be estimated by those developed for soil particles, the exposure risk for environmental PAHs can be quantitatively evaluated by using the concept of incremental lifetime cancer risk (*ILCR*) (U.S. EPA, 1991; Chen and Liao, 2006; Peng *et al.*, 2011). The subsequent equations were extensively applied to estimate the *ILCR* in terms of ingestion, dermal contact and inhalation:

$$ILCRs_{Ingestion} = \frac{CS \times \left(CSF_{Ingestion} \times \sqrt[3]{\left(\frac{BW}{70}\right)} \right) \times IR_{Ingestion} \times EF \times ED}{BW \times AT \times 10^6} \quad (7)$$

$$ILCRs_{Dermal} = \frac{CS \times \left(CSF_{Dermal} \times \sqrt[3]{\left(\frac{BW}{70}\right)} \right) \times SA \times AF \times ABS \times EF \times ED}{BW \times AT \times 10^6} \quad (8)$$

$$ILCRs_{Inhalation} = \frac{CS \times \left(CSF_{Inhalation} \times \sqrt[3]{\left(\frac{BW}{70} \right)} \right) \times IR_{Inhalation} \times EF \times ED}{BW \times AT \times PEF} \quad (9)$$

where

$ILCRs_{Ingestion}$: Incremental lifetime cancer risk in terms of ingestion.

$ILCRs_{Dermal}$: Incremental lifetime cancer risk in terms of dermal.

$ILCRs_{Inhalation}$: Incremental lifetime cancer risk in terms of inhalation.

CS: The sum of converted PAHs concentrations based on *TEQ* values as proposed by Nisbet and Lagoy (1992) (Eq. (2)), US EPA (1993) (Eq. (3)), and Cecinato (1997) (Eq. (4)).

$CSF_{Ingestion}$: Carcinogenic slope factor for ingestion of B[*a*]P (7.3 mg kg^{-1} day^{-1})$^{-1}$ (Peng *et al.*, 2011).

CSF_{Dermal}: Carcinogenic slope factor for dermal of B[*a*]P (25 mg kg^{-1} day^{-1})$^{-1}$ (Peng *et al.*, 2011).

$CSF_{Inhalation}$: Carcinogenic slope factor for inhalation of B[*a*]P (3.85 mg kg^{-1} day^{-1})$^{-1}$ (Peng *et al.*, 2011).

BW: Body weight of child (15 kg) and adult (61.5 kg) (Shi *et al.*, 2011).

EF: Exposure frequency of child (180 d a^{-1}) and adult (180 d a^{-1}) (Ferreira-Baptista and De Miguel, 2005).

ED: Exposure duration of child (6 a) and adult (24 a) (U.S. EPA, 2001).

$IR_{Inhalation}$: Inhalation rate of child (10 m^3 day^{-1}) and adult (20 m^3 day^{-1}) (SFT, 1999).

$IR_{Ingestion}$: Dust ingestion rate of child (200 mg day^{-1}) and adult (100 mg day^{-1}) (U.S. EPA, 2001).

SA: Dermal exposure area of child (2,800 cm^2) and adult (5,700 cm^2) (UE-EPA, 2001).

AF: Dermal adherence factor of child (0.2 mg cm^{-2}) and adult (0.07 mg cm^{-2}) (U.S. EPA, 2001).

ABS: Dermal adsorption fraction of child (0.13) and adult (0.13) (U.S. EPA, 2001).

AT: Averaging life span of child (25,550 d) and adult (25,550 d) (Ferreira-Baptista and De Miguel, 2005).

PEF: Particle emission factor of child (1.36 × 10^9 m^3 kg^{-1}) and adult (1.36 × 10^9 m^3 kg^{-1}) (U.S. EPA, 2001).

Note that the model for children (1–6 years old) and adults (7–31 years old) were based on the Risk Assessment Guidance of U.S. EPA and related publications.

RESULTS AND DISCUSSION

The average mass concentrations of particulate PAHs (pPAHs, reported in pg m^{-3}) of $PM_{2.5}$ samples collected at NNP-I (i.e., the average values of CROS, PYOS, NPOS, LMOS, PHOS, UTOS, MHOS, CMOS, and LPOS before the haze episode), at NNP-II (i.e., the average values of CROS, PYOS, NPOS, LMOS, PHOS, UTOS, MHOS, CMOS, and LPOS after the haze episode) and at world cities (WCs) are given in Table 1, in terms of their mean values with the corresponding standard deviations. The mean values of Fluo, Pyr, B[*b*+*k*]F, B[*a*]P, B[*g,h,i*]P, Σ3,4-rings PAHs and B[*a*]$P_{Equivalent}$ concentrations (i.e., B[*a*]$P_{Nisbet\text{-}Lagoy}$, B[*a*]$P_{US\text{-}EPA}$, B[*a*]$P_{Cecinato}$) at WCs (Fluo: 1,948 ± 2,395 pg m^{-3}, Pyr: 2,867 ± 2,845 pg m^{-3}, B[*b*+*k*]F: 5,363 ± 8,476 pg m^{-3}, B[*a*]P: 1,794 ± 2,389 pg m^{-3}, B[*g,h,i*]P: 3,057 ± 4,777 pg m^{-3}, Σ3,4-rings PAHs: 9,923 ± 13,628 pg m^{-3}, B[*a*]$P_{Nisbet\text{-}Lagoy}$: 7,345 ± 8,812 pg m^{-3}, B[*a*]$P_{US\text{-}EPA}$: 3,018 ± 3,759 pg m^{-3}, B[*a*]$P_{Cecinato}$: 3,667 ± 4,456 pg m^{-3}) were significantly ($p < 0.05$) much higher than those of samples collected at NNP-I and NNP-II (Table 1).

It is obvious that the measured PAH congeners in the present study fell within relatively low range, in comparison with other studies around the world (Table 2). Several factors such as unique tropical wet and dry climate (Köppen Aw) characterized by hot and rainy weather coupled with low industrial emission sources of northern provinces might have influenced the comparatively low PAH contents observed in

Table 1. Statistical descriptions of PAHs [pg m^{-3}] in $PM_{2.5}$ collected from nine administrative provinces before (NNP-I) and in the middle (NNP-II) of biomass burning episode coupled with the average of world cities.

	NNP-I (*n* = 9)	NNP-II (*n* = 9)	WCs (*n* = 39)
PAH Congener			
Ph	182 ± 355	185 ± 465	1,484 ± 3,477
An	57 ± 68	18 ± 15	396 ± 1,073
Fluo	84 ± 184	81 ± 191	1,948 ± 2,395
Pyr	182 ± 467	260 ± 696	2,867 ± 2,845
B[a]A	461 ± 1,372	337 ± 990	1,088 ± 1,220
Chry	333 ± 986	478 ± 1,387	2,140 ± 2,618
B[*b*+*k*]F	13 ± 25	83 ± 142	5,363 ± 8,476
B[*e*]P	1,767 ± 3,246	967 ± 1,855	686 ± 1,555
B[*a*]P	17 ± 27	7 ± 22	1,794 ± 2,389
Ind	5 ± 14	7 ± 21	2,975 ± 5,096
D[*a,h*]A	N.D.	N.D.	909 ± 1,292
B[*g,h,i*]P	7 ± 20	8 ± 24	3,057 ± 4,777
B[*a*]P-Equivalent			
B[*a*]$P_{Nisbet\text{-}Lagoy}$ (Eq. (2))	70 ± 144	55 ± 112	7,345 ± 8,812
B[*a*]$P_{US\text{-}EPA}$ (Eq. (3))	46 ± 80	34 ± 61	3,018 ± 3,759
B[*a*]$P_{Cecinato}$ (Eq. (4))	69 ± 143	55 ± 111	3,667 ± 4,456

Table 2. Aerosol observatory sites from cities around the world (WCs) for PAHs analysis.

Country	City	Source Type	References
Taiwan	Taichung	Industrial park	Fang *et al.*, 2005
Taiwan	Southern Taiwan	Carbon Black Industrial	Tsai *et al.*, 2002
Taiwan	Taichung	Urban	Fang *et al.*, 2004
Taiwan	Taichung	Rice Straw Burning	Yang *et al.*, 2006
Taiwan	Taichung	Non- Burning	Yang *et al.*, 2006
China	Guangzhou	Urban	Tan *et al.*, 2006
China	Guangzhou	Urban	Bi *et al.*, 2002
China	Nanjing	Urban	Wang *et al.*, 2006
Taiwan	Taichung	Industrial park	Fang *et al.*, 2005
China	Guangzhou	Urban Traffic	Li *et al.*, 2005
China	Nanjing	Urban	Wang *et al.*, 2003
China	Shantu	Urban	Deng *et al.*, 2006
China	nanjing	Urban	Wang *et al.*, 2006
Hong Kong	Hung hom	Urban	Guo *et al.*, 2003
Singapore	NUS	Urban	Karthikeyan *et al.*, 2006
Singapore	NUS	Canteen	Karthikeyan *et al.*, 2006
Belgium	Antwerp	Urban	Ravindra *et al.*, 2006
Germany	Augburg	Urban	Schnelle-Kreis *et al.*, 2005
Sweden	Gothenberg	Urban	Wingfors *et al.*, 2001
Italy	Naples	Urban	Caricchia *et al.*, 1999
Greece	Athens	Urban	Vasilakos *et al.*, 2007
Greece	Athens	Urban	Mantis *et al.*, 2005
England	Berminghan	Traffic	Pongpiachan, 2006
England	Berminghan	Campus	Pongpiachan, 2006
England	Whitbourne	Rural	Pongpiachan, 2006
Sweden	Gothenberg	Urban	Wingfors *et al.*, 2001
Czech	Brno	Urban	Ciganek *et al.*, 2004
USA	Baltimore	Urban	Dachs *et al.*, 2002
USA	Texus	Urban	Park *et al.*, 2001
USA	Florida	Urban	Poor *et al.*, 2004
Brazil	Rio Grande do Sul	Urban	Dallarosa *et al.*, 2005a
Brazil	Rio Grande do Sul	Urban	Dallarosa *et al.*, 2005b
Brazil		Rural	Dallarosa *et al.*, 2005b
Brazil		Industry	Dallarosa *et al.*, 2005b
Algeria	Algiers	Industry	Yassaa *et al.*, 2001a
Algeria	Algiers	Urban	Yassaa *et al.*, 2001b
Australia	Melbourne	Urban	Bartkow *et al.*, 2004
Australia	Brisbane	Urban	Bartkow *et al.*, 2004
Australia	Springwood	Rural	Lim *et al.*, 2005

NNP-I and NNP-II. While using the Σ3,4-rings PAHs level at NNP-I as the background level, the magnitude of Σ3,4-rings PAHs at the WCs increased by factors of 7.6, and that of $B[a]P_{Nisbet\text{-}Lagoy}$, $B[a]P_{US\text{-}EPA}$, and $B[a]P_{Cecinato}$ were roughly enhanced by factors of 105, 66, and 53, respectively. Previous studies have indicated that vehicular traffic was the main sources of $PM_{2.5}$ bounded PAHs in urban cities (Slezakova *et al.*, 2011; Martellini *et al.*, 2012; Slezakova *et al.*, 2013). These findings underline the prominence of traffic emissions as the main contributors of $PM_{2.5}$ bounded PAHs, and it might have been exceeded numerous emission sources involving wild land fire and biomass burnings. As there were no significant differences detected at NNP-I and NNP-II (Table 1), it seems plausible to mention that the wild land fire episode plays a minor role on variations of $PM_{2.5}$ bounded PAHs in nine administrative provinces of Northern Thailand.

Health Risk Assessment

In this study, non-dietary exposure is defined as human exposure to $PM_{2.5}$ bounded PAHs via both household air and dust (Wilford *et al.*, 2005). Table 3 displays the excess cancer risks associated with house dust exposure in NNP-I, NNP-II, and WCs, ranged from $3.732 \times 10^{-4} \pm 7.929 \times 10^{-4}$ (NNP-II-$TEQ_{US\text{-}EPA}$ with ingestion rate of 50 mg day^{-1}) to 1.451 ± 1.741 (WCs-$TEQ_{Nisbet\text{-}Lagoy}$ with ingestion rate of 100 mg day^{-1}), depending on *TEQ* equations and ingestion rate (*IR*) as displayed in Eq (5). The mean cancer risks of NNP were $8.525 \times 10^{-4} \pm 3.493 \times 10^{-3}$ and $2.558 \times 10^{-3} \pm 6.986 \times 10^{-3}$ for ingestion rate of 50 and 100 mg day^{-1}, respectively. The cancer risks of WCs for ingestion rate of 50 and 100 mg day^{-1} were greatly higher than those of

NNP for 851 and 567 times in that order. Although the cancer risks of NNP were much lower than those of WCs, its average values ranged from 10^{-4} to 10^{-3} (i.e., one cancer incidence case per million), which can be considered as unacceptable cancer risks (Maertens *et al.*, 2008). These values are comparable to those of foundry workers (9.06×10^{-4} and 1.09×10^{-3}) in Taiwan (Liu *et al.*, 2010), but higher than those predicted as occupational exposure for sinter metal workers (3.18×10^{-5} and 4.98×10^{-5}) (Lin *et al.*, 2008; Chiang *et al.*, 2009) and the Canadian maximum acceptable level of risk (1×10^{-5}) (Maertens *et al.*, 2008). The current study indicates there is some concerns on adverse health impact related with $PM_{2.5}$ bounded PAHs exposure for preschool children via non-dietary exposure in home environment, based on the calculation of Eqs. (1)–(5).

The highest average values of *TEQ* daily exposure doses via house dust ($M_{PDI\text{-}ingestion}$; ng day^{-1}) for adults and children was $7.345 \times 10^4 \pm 8.813 \times 10^4$ and $1.469 \times 10^5 \pm 1.763 \times 10^5$ ng day^{-1} under high exposure condition (with ingestion rate of 100 and 200 mg day^{-1} for adults and children) with the application of WCs-$TEQ_{Nisbet\text{-}Lagoy}$, while 4.715 ± 12.08 and 62.33 ± 159.8 ng day^{-1} under low exposure condition (4.16 and 55 mg day^{-1} for adults and children) with the employment of NNP-II-$TEQ_{US\text{-}EPA}$, respectively (Table 4). It is also important to note that $M_{PDI\text{-}ingestion}$ values of NNP-II-$TEQ_{US\text{-}EPA}$ were almost similar to those of Guangzhou for adults and children with the values of 4.31 and 57.0 ng day^{-1}, respectively (Wang *et al.*, 2013). For *TEQs* daily exposure via inhalation ($M_{PDI\text{-}inhalation}$; ng day^{-1}), WCs-$M_{PDI\text{-}inhalation\text{-}Nisbet\text{-}Lagoy}$ shows the highest values for both adults and children with the values of 147 ± 176 and 735 ± 881 ng day^{-1}, in that order (Table 5). Since the values of $M_{PDI\text{-}ingestion}$ were two to three orders of magnitude exceed those of $M_{PDI\text{-}inhalation}$, it would be safe to assume that dust ingestion was extremely crucial to non-dietary PAH exposure in comparison with $PM_{2.5}$ inhalation. These findings in NNP

Table 3. The cancer risks associated with non-dietary ingestion of PAHs in house dust for preschool children as estimated by using Eq. (5) (U.S. EPA, 1997; Maertens *et al.*, 2008).

	NNP-I (n = 9)		NNP-II (n = 9)		WCs (n = 39)	
Ingestion Rate [mg day^{-1}]	50 Aver	50 Stdev	50 Aver	50 Stdev	50 Aver	50 Stdev
Cancer Risk-$_{Nisbet\text{-}Lagoy}$	2.305E-03	6.774E-03	6.037E-04	1.456E-03	7.256E-01	8.707E-01
Ingestion Rate [mg day^{-1}]	100 Aver	100 Stdev	100 Aver	100 Stdev	100 Aver	100 Stdev
Cancer Risk-$_{Nisbet\text{-}Lagoy}$	4.610E-03	1.355E-02	1.207E-03	2.912E-03	1.451E+00	1.741E+00

Table 4. The calculation of TEQs daily exposure via ingestion ($M_{PDI\text{-}ingestion}$; ng day^{-1}) as displayed in Eq. (6) (U.S. EPA, 1997).

M_{DID} (mg day^{-1})	Children		Children		Adult		Adult	
	55		200		4.16		100	
	NNP-I (n = 9)		NNP-I (n = 9)		NNP-I (n = 9)		NNP-I (n = 9)	
	Aver	Stdev	Aver	Stdev	Aver	Stdev	Aver	Stdev
$M_{PDI\text{-}ingestion\text{-}Nisbet\text{-}Lagoy}$	1.283E+02	3.771E+02	4.667E+02	1.371E+03	9.707E+00	2.853E+01	2.333E+02	6.857E+02
	NNP-II (n = 9)		NNP-II (n = 9)		NNP-II (n = 9)		NNP-II (n = 9)	
$M_{PDI\text{-}ingestion\text{-}Nisbet\text{-}Lagoy}$	1.008E+02	2.933E+02	3.667E+02	1.067E+03	7.627E+00	2.219E+01	1.833E+02	5.333E+02
	WCs (n = 39)		WCs (n = 39)		WCs (n = 39)		WCs (n = 39)	
$M_{PDI\text{-}ingestion\text{-}Nisbet\text{-}Lagoy}$	4.040E+04	4.847E+04	1.469E+05	1.763E+05	3.056E+03	3.666E+03	7.345E+04	8.813E+04

Table 5. Calculation of *TEQs* daily exposure via inhalation ($M_{PDI\text{-}inhalation}$; ng day^{-1}) is based on Eq. (6) (SFT, 1999)

	NNP-I (n = 9) Adult		NNP-II (n = 9) Adult		WCs (n = 19) Adult	
Inhalation Rate [m^3]	20 Aver	20 Stdev	20 Aver	20 Stdev	20 Aver	20 Stdev
$M_{PDI\text{-}inhalation\text{-}Nisbet\text{-}Lagoy}$	1.400E+00	2.880E+00	1.100E+00	2.240E+00	1.469E+02	1.763E+02
Inhalation Rate [m^3]	10 Aver	10 Stdev	10 Aver	10 Stdev	10 Aver	10 Stdev
$M_{PDI\text{-}inhalation\text{-}Nisbet\text{-}Lagoy}$	7.000E-01	1.440E+00	5.500E-01	1.120E+00	7.345E+01	8.813E+01

are consistent with those of Guangzhou (Wang *et al.*, 2013), highlighting the importance of indoor air quality on long-term adverse respiratory diseases in Asian cities.

Incremental Lifetime Cancer Risk

This is the first attempt that evaluated the potential cancer risk of human exposure to $PM_{2.5}$ bounded PAHs of different exposure routes before and after the haze episode by using three different *TEQ* methods. The potential cancer risks of adults and children can be assessed by using *ILCR* for exposure routes of ingestion, dermal contact and inhalation as displayed in Eq. (7), Eq. (8), and Eq. (9), respectively (Table 6) (Chen and Liao, 2006; Peng *et al.*, 2011; Wang *et al.*, 2011). It is important to note that $ILCR_{Dermal}$ and $ILCR_{Ingestion}$ varied from 10^{-10} to 10^{-8} in both NNP-I and NNP-II samples, while those of WCs was 10^{-7} to 10^{-6}; approximately two to three orders of magnitude higher than those of NNP. This indicates that human activities in urban world cities play an important role in governing potential cancer risk of human exposure to $PM_{2.5}$ bounded PAHs. It is also worth mentioning that both $ILCR_{Dermal}$ and $ILCR_{Ingestion}$ were significantly higher than $ILCR_{Inhalation}$, denoting that respiration of $PM_{2.5}$ bounded PAHs through mouth and nose was practically unimportant, when compared with the other exposure pathways. These findings were in good agreement with studies of exposure to heavy metals in street dust of Angola (Ferreira-Baptista and De Miguel, 2005) and dust samples collected from Guangzhou, a typical urban center in South China (Wang *et al.*, 2011). In the case of children, the $ILCR_{Ingestion}$ was significantly eight times higher ($p < 0.05$) than those of $ILCR_{Dermal}$, suggesting that the hand-to-mouth activity prominently contributed to the cancer risk for children. This finding is also in consistent with the investigation of potential risk of children exposure on heavy metal distribution in dust from elementary schools in Hermosillo, Sonora, Mexico (Meza-Figueroa *et al.*, 2007) and potential cancer risk of children exposure to urban dust bounded PAHs in Guangzhou, China (Wang *et al.*, 2011). Therefore, it appears reasonable to conclude that the hazard health risk for children exposed to $PM_{2.5}$ bounded PAHs through direct ingestion is considered to be noticeably greater than that of dermal contact and inhalation.

Under most guidelines for carcinogen risk assessment, the values of *ILCR* range from 10^{-6} to 10^{-4} implied potential cancer risk, while the practical safety was expressed with an *ILCR* of 10^{-6} or less and a potential high risk was evaluated by an *ILCR* of higher than 10^{-4} (Chen and Liao, 2006; Wang *et al.*, 2011). Furthermore, U.S. EPA Risk Assessment Guidance for Superfund Program adopts a cancer risk level of 10^{-6} to suggest the point at which risk management decisions should be taken. By applying the same guidance, the chance of adverse cancer-causing effects in northern provinces of Thailand was considerably lower in comparison to world urban cities. In this study, the average values of *ILCR* of children as estimated by three different *TEQs* for NNP for both monitoring campaigns were $5.371 \times 10^{-9} \pm 1.518 \times 10^{-8}$, $6.696 \times 10^{-10} \pm 1.893 \times 10^{-9}$ and $1.042 \times 10^{-14} \pm 2.944 \times 10^{-14}$, for ingestion, dermal contact, and inhalation, respectively; these risk levels are 1.862×10^{2}, 1.493×10^{3},

Table 6. Calculations of *ILCR* in terms of ingestion (Eq. (7)), dermal contact (Eq. (8)) and inhalation (Eq. (9)) due to human exposure to PAHs via $PM_{2.5}$

	NNP-I ($n = 9$) Adult		NNP-I ($n = 9$) Children		NNP-II ($n = 9$) Adult		NNP-II ($n = 9$) Children		World Cities ($n = 19$) Adult		World Cities ($n = 19$) Children	
	Aver	Stdev	Aver	Stdev	Aver	Stdev	Aver	Stdev	Aver	Stdev	Aver	Stdev
ILCRs-Ingestion												
Nisbet-Lagoy	1.371E-08	4.030E-08	6.857E-09	2.015E-08	1.078E-08	3.135E-08	5.388E-09	1.567E-08	4.317E-06	5.180E-06	2.159E-06	2.590E-06
ILCRs-Dermal												
Nisbet-Lagoy	2.436E-09	7.159E-09	8.548E-10	2.512E-09	1.914E-09	5.568E-09	6.716E-10	1.954E-09	7.669E-07	9.201E-07	2.691E-07	3.229E-07
ILCRs-Inhalation												
Nisbet-Lagoy	1.064E-13	3.126E-13	1.330E-14	3.907E-14	8.357E-14	2.431E-13	1.045E-14	3.039E-14	3.348E-11	4.017E-11	4.185E-12	5.022E-12

and 9.602 × 10^7 times lower than that of US EPA (1991) baseline in that order. In addition, the average *ILCR* of adults living in NNP as estimated by three different *TEQs* for three various exposure pathways was 256 and 237 times lower than the WCs and US EPA baseline, further indicating that the cancer risk of NNP falls into the ''acceptable level'' range.

CONCLUSIONS

In this study, PAHs analysis in $PM_{2.5}$ collected in nine administrative provinces of Northern Thailand from December 2012 to March 2013 did not show any significant differences between wildland fire and non-wildland fire seasons. These findings underline the influences of traffic emissions as regular contributors of $PM_{2.5}$-bound PAHs. While the excess cancer risks of $PM_{2.5}$-bound PAHs in Northern Thailand were greatly lesser than those of other world cities, its average values ranged from 10^{-4} to 10^{-3}, which was higher than the Canadian maximum acceptable level of risk (1×10^{-5}) and other previous occupation exposure studies. The average *ILCR* values of adults living in Northern Thailand as predicted by three different *TEQs* for ingestion, dermal contact, and inhalation exposure pathways were much lower than the US EPA baseline, further denoting that the cancer risk of NNP falls into the ''acceptable level'' range.

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