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Population Health Risks Assessment from Air Pollution Exposure in an Industrialized Residential Area in Greece

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Abstract: Industrial activities nearby residential areas lead to poor local air quality. Therefore, short-term exposure to an aggravated environment and the subsequent health effects should be the subject of further research. The purpose of this study is to estimate the health risks resulting from such exposure in population groups living in an industrialized area. The risk estimation was performed using different approaches suggested in relative literature. Monitoring of the air quality in an industrial zone of Attica was carried out including 24-h measurements of $PM_{2.5}$ and analysis of their chemical composition for Polycyclic Aromatic Hydrocarbons and heavy metals (Pb, Cd, As, Ni, Hg, Cu, Zn). Samples of Volatile Organic Compounds were also collected. Health effects on different population subgroups were estimated for the targeted pollutants through different mathematical approaches provided by the literature, taking into consideration different parameters (e.g., age, gender, exposure duration). Inhalation rate and body weight were important parameters to estimate the exposure dose of people, and they can vary greatly depending on the age, gender, and daily activity of the person under consideration. The results indicated that the risk for potential carcinogenic and non-carcinogenic effects varies depending on the applied methodology. In any case, the acceptable limits for cancer risk provided by the OEHHA, EPA, and WHO were not exceeded.

Keywords: atmospheric pollutants; risk assessment; human health; exposure dose; carcinogenesis

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1. Introduction

Air quality deterioration has become a serious matter of concern due to increased anthropogenic and natural emissions [1,2], leading to increasing cases of acute air pollution episodes and exceedances of the air quality standards on a global scale [3,4]. Public health has been seriously affected by air pollution during the last decades, and the problem is expected to intensify in the future. According to the guidelines of the World Health Organization (WHO), seven million people die early from air pollution every year worldwide, while nine out of 10 people breathe air containing high levels of pollutants [5,6]. Nowadays, there is much more evidence to support these claims, as well as better our understanding of the way the air pollutants affect public health even in lower concentrations, as it is estimated that the number of deaths and the loss of healthy years of life due to air pollution has not been reduced [7].

Suspended Particulate Matter (PM) is considered to be one of the most serious pollutants and has been classified as a 'Group 1' contaminant (according to the International Agency for Research on Cancer, Group 1 includes substances that have sufficient evidence

of carcinogenicity in humans, while Group 2 includes substances for which the degree of evidence of carcinogenicity in humans is almost sufficient) [8,9].

Consequently, an increasing number of studies have focused on providing data about air quality deterioration and the resulting health effects on the population. This relation has been extensively documented [10,11] in studies examining indoor and outdoor exposure to hazardous substances in children and adults (e.g., PAHs & heavy metals) [12–16]. The main target of those studies was to assess the exposure to ambient PM associated with increased mortality and morbidity, bio-accessibility through inhalation exposure and lifetime lung cancer risk [17,18], disability-adjusted life years (DALYs), cardiopulmonary diseases, and risk factors including a wide variety of health effects for people of all ages [4,19,20]. However, there is still uncertainty about the mechanisms through which air pollutants influence human health; for example, it is not clear if health implications are the result of synergistic or individual effects of pollutants, which makes the assessment a complicated process.

The current study aimed to assess the risk of exposure of a population of an industrialized residential area (Elefsina, Greece) to atmospheric pollutants originating mainly from industrial activities. For this scope, estimation of the risk of carcinogenic and non-carcinogenic effects in the human body was carried out, based on a number of different mathematical approaches reported in the literature. A range of impact assessments was achieved and a comparison of their strengths and weaknesses was attempted with the scope of contributing to a better understanding of the population health risk caused by air pollution. The effect of considering various parameters (i.e., age, gender, potential cancer factor, daily exposure) on the risk estimation outcome, was also investigated. To the best of our knowledge, this is the first time that a comparative study on the population health risks assessment from air pollution exposure in an industrialized residential area has been conducted.

2. Materials and Methods

2.1. Location Description

An integrated sampling campaign was carried out at three sites in the wider area of Elefsina (Figures 1 and 2; Table 1) during the winter and summer period of 2019 (February–October). Elefsina is a municipality of the West Attica Prefecture, located at a distance of about 20 km from the center of Athens, Greece. It covers an area of 20 sq km and has a population of 24,910 inhabitants (2011 census). The area is characterized by intense industrial activity (i.e., oil refinery). The sampling sites selection followed EU Directive 2008/50/EC while taking into consideration the characteristics of the surroundings of each area. All sites were selected to be located within the residential area of Elefsina. More specifically, E1 and E2 were located close to the industrial zone (refiners) while the E3 site was near the port (Figure 2).

All sampling and monitoring equipment was installed in schoolyards. For all three locations, meteorological parameters (wind speed and direction) were continuously recorded using a portable anemometer. During the sampling period, the prevailing wind direction was W-NW with wind speed ranging from 0.70 to 4.07 m/s while temperature ranged from 5 to 21 °C (February–May) and 20 to 31 °C (June–October). The relative humidity ranged between 42 and 88%.

Table 1. Description of Sampling and Coordinating Points.

Sampling Point Code	Sampling Point Features	Region	Coordinates	
			X	Y
E1	Primary School Mandra	Elefsina (Mandra)	38°3′7″ B	23°31′35″ A
E2	Primary School I Elefsina	Elefsina	38°3′10″ B	23°31′51″ A
E3	Primary School II Elefsina	Elefsina	38°2′26″ B	23°32′4″ A

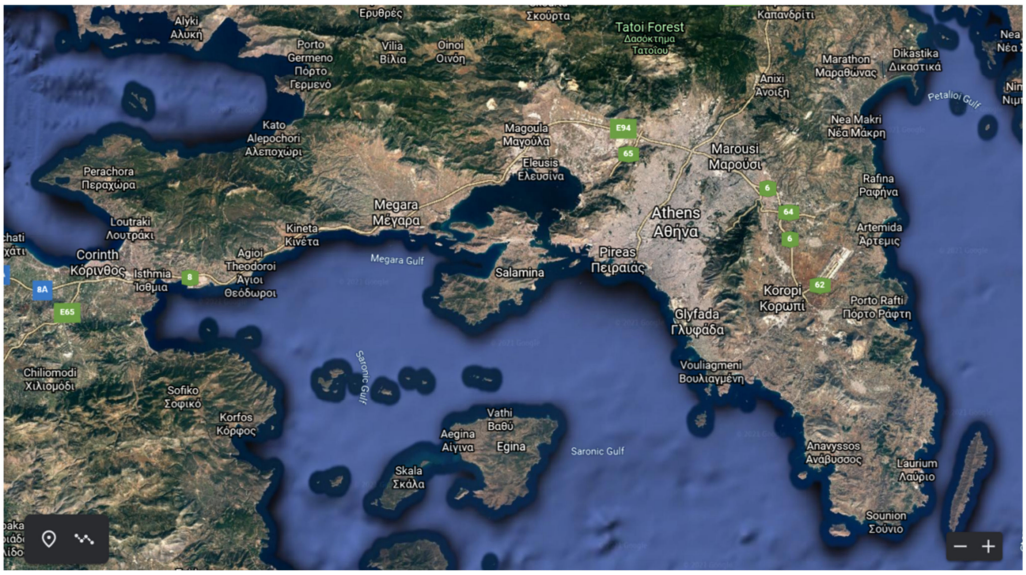


Figure 1. The sampling site and the industrial area in Eleusina nearby Athens (source: Google Maps).

Figure 2. Spatial representation of the sampling sites in the municipality of Elefsina (1, 2, 3, Source: Google Maps).

2.2. Sampling and Chemical Analysis

The 24-h (starting at 8 a.m.) $PM_{2.5}$ samples (n = 180) were collected on 47 mm Tissue Quartz 25000QAO PALL membrane filters with the use of low volume (2.3 m^3/h) samplers (Derenda, Leckel). The determination of the PM mass was conducted according to EN 12,341:2014. After PM mass concentration determination, PM samples were analyzed for 22 PAHs (naphthalene, acenaphthylene, acenaphthene, fluorene, phenanthrene, anthracene, fluoranthene, pyrene, benzo(a)anthracene, chrysene, benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(a)pyrene, indeno(123-cd)pyrene, dibenzo(ah)anthracene, benzo(ghi)perylene, benzo(b)fluorene, benzo(e)pyrene, perylene, 1,2-dimethylnaphthalene, 1-methylnaphthalene, 1-methylphenanthrene, 2-methylnaphthalene) and seven heavy metals (Pb, Hg, Ni, Cr, As, Cd & Be). The determination of heavy metals was based on EN14902:2005 European Standard using an ED-XRF (Energy Dispersive X-Ray Fluorescence) Epsilon 5, Panalytical, analyzer for the analysis of Pb, Hg, Ni, Cr while for As,

Cd, Be an Atomic Absorption Spectrometer (AAS) Varian 220, GTA 110 was used. The sampling and the analysis of the PAHs were performed according to EN15549:2008 using a gas chromatograph (GC Agilent 5975C) coupled to a mass spectrometry detector (Agilent 7890A MS. The values of LOD, LOQ, as well as the expanded uncertainty at 95% confidence level and k = 2 that has been estimated experimentally (Uexp) for each PAH, are summarized in the Supplementary Material, Table S1. The analytical procedures are described elsewhere [16,21].

In parallel, 330 samples of VOCs were collected during the summer and winter periods of 2019 (February–October), covering the factor of seasonal fluctuations in their concentration levels (Table 2). Samples were analyzed using a gas chromatography system (Agilent GC6890) coupled with an FID detector and a Thermal Desorption System (Gerstel TDSA) according to EN 16017:2001 (Table S2). Samples were collected in preconditioned glass tubes filled with Tenax TA (Gerstel) and were analyzed within 1 day after sampling. LOD for VOCs (benzene) analysis was 0.02 ng/l while the expanded uncertainty (% Uexp, 95%, k = 2) of the analytical procedure was 11.7%.

Table 2. Number of samples per sampling site.

Sampling Site	$PM_{2.5}$	PAH	VOCs	Heavy Metals
E1	93	42	131	43
E2	44	22	115	22
E3	43	27	84	25
Total	180	91	330	90

2.3. Health Risk Assessment (Methodological Approach)

The process to determine if a substance, chemical or not, constitutes a potential risk to human health is a complex procedure. The dose-response assessment refers to the characterization of the correlation between exposure to an agent and the incidence of an adverse health effect in the exposed population. In the present study, a literature review on methods used to evaluate these potential health risks through different mathematical approaches was conducted. Based on this review, different hypothetical scenarios of human exposure were selected to be applied in the case of Elefsina's population. Specifically, the following representative cases were selected

The case of a male and a female living and working at the place of exposure, i.e., 24 h/day.

The case of a male and a female living at the place of exposure (their place of residence) for 14 h/day, and working outside of that region,

The cases of a child in the age groups of 0–2 and 2–16 years old, living at the place of exposure, i.e., 24 h/day.

The exposure period was selected to be 350 days/year in all cases. Furthermore, specific values of the parameters IR and BW (Inhalation Rate & Body Weight) were selected according to literature [22,23], for the residents of Elefsina. Particularly:

For males: IR = 16.4 (m^3/day) and BW = 76 (kg),

For females: IR = 12.6 (m^3/day) and BW = 63 (kg),

For children of age 2–16 years old: IR = 10.8 (m^3/day) and BW = 32.5 (kg),

For children of age 0–2 years old: IR = 4.9 (m^3/day) and BW = 10.3 (kg).

For the calculation of the examined parameters, the average concentration of the considered pollutants given in Table 3 was used.

Table 3. Average concentration of measured pollutants in Elefsina area.

	Chemical Substance	Concentration (ng/m^3)
PAHs	Benzo(a)pyrene	0.059
	Benz(a)anthracene	0.053
	Benzo(b)fluoranthene	0.289
	Benzo(k)fluoranthene	0.079
	Chrysene	0.108
	Dibenzo(a,h)anthracene	0.023
	Indeno(1,2,3-c,d)pyrene	0.135
	1-methylnaphthalene	0.577
Heavy metals	Nickel	4.380
	Lead	8.031
VOC	Benzene	1390

2.3.1. Carcinogenic Risk Assessment Methodological Approaches (Group A)

The probability of developing cancer over a lifetime in the people who live in the Elefsina area was estimated based on methods reported in the literature. In quantitative carcinogenic risk assessment, the dose-response correlation is expressed in terms of a potency slope, which is used to calculate the probability of carcinogenic risk associated with an estimated exposure (95th percent upper confidence limit of the slope of the dose response curve). The cancer risk (CR) value below 10^{-6}–10^{-4} is considered acceptable [13,14,24–27], whereas 10^{-6} is considered the most tolerable risk [28]. In this study, CR characterization was conducted by applying the methodologies described in the following paragraphs (A.I–A.IV). As a final step, the cancer risk of each pollutant was summed up for the calculation of the Total Cancer Risk and then the overall Risk of Cancer was converted to "chances per million"

$$\text{(Total Cancer Risk) } (1 \times 10^6) = \text{Total Cancer Risk in chances per million}$$

Approaches used for the estimation of potential cancer risk

Methodology A.I

In the study of R.M. Maertens (2008) [14] a risk assessment was conducted to evaluate the excess lifetime cancer risk associated with exposure to seven PAHs (Table S3), using the method proposed by [12] Collins et al., 1998. To be more specific, the following equation was used to estimate the lifetime cancer risk for these seven PAHs Equation (1):

$$\text{Lifetime Cancer Risk} = \sum_{i=1}^{n} \left(\frac{(C_i \times PEF_i) \times IR \times EF \times SF \times AF}{BW \times 1000} \right) \quad (1)$$

where C: concentration (μg/g) of each PAH, which are categorized as probable human carcinogens (B2) according to U.S. EPA classifications [14], PEF_i: the potency of each PAH in correlation to benzo[a]pyrene, according to Collins et al., 1998 [12] (Potency equivalency factor), SF: Slope factor for carcinogenic (mg kg^{-1} day^{-1})$^{-1}$, indicating the probability of cancer occurring per unit of inhaled PAHs over a lifetime. Maertens (2008) examined the cancer risk in association with ingestion instead of inhalation, which differentiates the values of SF, IR and BW, so were selected according to Handbook "Guidance Manual for Preparation of Health Risk Assessment" from the OEHHA, 2015 [24]. The PEF value remains the same, while IR (m^3/day) was the same as mentioned previously (M: 16.4, W: 12.6, $Child_{2–16}$: 10.8 & $Child_{0–2}$: 4.9), as well as for BW (Kg), respectively, (M: 76, W: 63, $Child_{2–16}$: 32.5 & $Child_{0–2}$: 10.3), EF: exposure factor, the average proportion of daily exposure of people through inhalation rate (h/day), AF: adjustment factor for exposure

dose modification in early life stages, as young children are more vulnerable to the effects of chemical toxins according to EPA, 2003 & 2005 [29,30].

Specifically:

$AF_{0–2}$: 10-fold adjustment, for children between 0–2 years old
$AF_{2–16}$: 3-fold adjustment, for children between 3–16 years old
$AF_{16–80}$: no adjustment needed for children >16 years old and adults

Methodology A.II

A previous study by R.M. Maertens (2004) [13], also evaluating the excess lifetime cancer risk caused by exposure to the specific PAHs, applied the same methodology without including the adjustment factor (AF). The evaluation of cancer risk was estimated in two steps, as described by Equations (2) and (3):

$$\text{Lifetime average daily exposure dose} = \frac{C \times IR \times EF}{BW} \quad (2)$$

$$\begin{aligned}\text{Lifetime Cancer Risk} = \ & \text{Lifetime average daily exposure dose } (mg/kg/day) \\ & \times \text{Slope Factor } (mg/kg/day)^{-1} \\ & \times \text{Potency Equivalency Factor}\end{aligned} \quad (3)$$

The remaining parameters were selected similarly to the methodology A.I.

Methodology A.III

In the study of M.M. Jackson (2005) [31], the contribution of road traffic to the air pollution levels in Dar-es-Salaam city of Tasmania was examined; a risk assessment was conducted for the people who were in the nearby area. In the present study, the potential cancer risk for the residents in the region Elefsina was estimated for a variety of pollutants, including some heavy metals (Pb & Ni). In addition, according to U.S. EPA risk guidelines from 1986 [32], Pb has been classified, with a group B2 classification, as a potentially carcinogenic substance. The risk for cancer (R) was calculated from the following equations Equations (4) and (5):

$$CDI = \frac{CA \times IR \times ED \times EF \times L}{BW \times AT \times 365} \quad (4)$$

$$R = CDI \times P \quad (5)$$

where P is the cancer potency for the pollutants $(mg{\cdot}kg^{-1}{\cdot}day^{-1})^{-1}$, that were selected according to US-EPA Regional Screening Level (RSL) Summary Supplementary 2019 [33], except for Ni and Pb. The *p*-value for Pb was set equal to 4.2×10^{-2}, according to the study of M. Jackson et al., 2005 [31] complying with OEHHA, 2015 (Appendices) [34]. Regarding Ni, it was set equal to 9.1×10^{-1} [34]. The CDI (chronic daily intake) dose $(mg{\cdot}kg^{-1}{\cdot}day^{-1})$ was calculated from Equation (4), with CA: the concentration of pollutant in the air (mg/m^3), IR the inhalation rate (m^3/h), EF the exposure frequency (days/year), ED the exposure duration per day (h/day), L the length of the exposure (years), BW the average body weight (kg) and AT the averaging time of 80 years length for carcinogens (ICRP, 2002) [22].

Methodology A.IV

Gao, (2019) [5] and Hong et al., 2020 [17], evaluated the potential cancer risk during inhalation for nine PAHs detected in $PM_{2.5}$. In Gao's 2019 study, the bioaccessibility of these PAHs was investigated, by employing a physiological extraction test with simulated lung fluids [Gamble's solution and artificial lysosomal fluid (ALF)]. With the term bioaccessibility (%), we refer to the proportion of the pollutants which are important contributors to the effects on the human body. In the present study, this practical application was not

feasible. The estimated risk for respiratory cancer (CR) was evaluated through the following Equation (6):

$$\text{Cancer Risk} = \sum_{i=1}^{n} (C_{PAHi} \times RPF_i) \times UR_{BaP} \quad (6)$$

where $C_{PAHi:}$ the concentration of each PAH detected in $PM_{2.5}$, RPF: the relative potency factor of each PAH can be determined in correlation to benzo[a]pyrene, according to the U.S. EPA (2010) Integrated Risk Information System [35] (Table 4), which can be found in the supplementary data of Gao et al. 2019 [5], UR_{BaP} (Unit Risk) is the probability of the maximum theoretical limit of the number of people with cancer in the respiratory system caused by inhalation at an equivalent concentration of 1 $\mu g/m^3$ BaP over a lifetime of 70 years. Two different UR_{BaP} values were selected for the inhalation cancer risk assessment according to the California Environmental Protection Agency (CalEPA) (1.10×10^{-6} per ng/m^3 based on the data for respiratory tract tumors from inhalation exposure in hamsters) and the World Health Organization (8.70×10^{-5} per ng/m^3 based on an epidemiological study on coke-oven workers in Pennsylvania) (OEHHA, 1993, 2005; WHO, 2000, 2010) [36–39].

Table 4. Relative potency factor, (EPA, 2010) [35].

PAH	Relative Potency Factor
benzo[a]pyrene	1.000
benz[a]anthracene	0.200
benzo[b]fluoranthrene	0.800
benzo[k]fluoranthrene	0.030
chrysene	0.100
dibenz[a,h]anthracene	10.00
Indeno[1,2,3-c,d]pyrene	0.070
benzo[g,h,i]perylene	0.009
fluoranthene	0.080

It is clear from the description of the different methodologies that there are key differences in the approaches for the evaluation of the potential cancer risk. The first three approaches include common parameters (PEF, SF & P). However, the A.I and A.II differ in the adjustment factor (AF), which modifies the estimated cancer risk for children as a function of age. In the A.III method, the parameters of Daily exposure (hours/day), exposure duration (days per year), and the length of the exposure (years) of how many years the exposure took place were added. On the contrary, the A.IV methodology presents significant differences from the others as exposure time, AF, IR and BW parameters are not included, while UR parameter is included.

2.3.2. Risk Assessment for Carcinogenesis and non-Cancer Effects Approaches (Group B)

Characterization of the risk of developing cancer and non-cancer effects over a lifetime was estimated for people in Elefsina, using the methods found in our literature review.

Cancer risk (CR) with a value below 10^{-6}–10^{-4} is considered acceptable, as already mentioned. The non-cancer risk was estimated according to U.S.EPA (2004, 2009) [40,41], is expressed as the hazard quotient (HQ) and the hazard index (HI). HQ is calculated for each pollutant and HI corresponds to the sum of the individual HQs [41]. The acceptable values for HI and HQ are considered to be lower than unity (HI & HQ < 1), meaning that the greater the HQ and HI values are, the higher the probability of developing non-cancer effects in humans (U.S. EPA, 1989) [42]. In the final step of risk assessment, modeled concentrations and exposure data, which are determined through exposure evaluation and are combined with potency factors and Reference Exposure Levels (REL's) are developed through a dose-response curve estimated assuming continuous lifetime exposure to substances [40–43].

The following methodologies were applied in order to estimate the potential cancer risk and non-cancer effects.

Methodology B.I

Using Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessment (Guidance Manual) from the Office of Environmental Health Hazard Assessment [24,34] (OEHHA, 2015; OEHHA Appendices, 2015), the evaluation of potential inhalation cancer risk and non-cancer acute hazard quotient (HQ) and hazard index (HI) was carried out. The risk of cancer was calculated from the following Equation (8) and the Dose through inhalation (mg/kg/d) from Equation (7):

$$\text{Dose} - \text{air} = (\text{Cair})\frac{\text{BR}}{\text{BW}}(\text{A})(\text{EF})\left(1 \times 10^{-6}\right) \quad (7)$$

$$\text{Cancer Risk} = \left(\text{InhalationDose}\frac{\text{mg}}{\text{kg} - \text{day}}\right)\left(\text{Cancer Potence}\frac{\text{kg} - \text{day}}{\text{mg}}\right)(\text{ASF})(\text{FAH})\left(\frac{\text{ED}_{\text{years}}}{\text{AT}_{\text{years}}}\right) \quad (8)$$

where Cair: concentration of pollutant ($\mu g/m^3$), BR/BW: daily breathing rate adjusted with body weight (L/kg BW-day), A: inhalation absorption factor (unitless, A = 1 according to OEHHA, 2015) [24], EF: exposure frequency (unit less, days/365 days), and 1×10^{-6}: conversion micrograms to milligrams and liters to cubic meters, Cancer Potency was the cancer potency of a pollutant $(mg \cdot kg^{-1} \cdot day^{-1})^{-1}$, and was selected according to the OEHHA, 2015 [24] (Table S5), the ASF and FAH were variables that were used only when estimating residential cancer risk, so they were not applied, ED: the years of exposure duration and AT: averaging time period over which exposure duration (always 70 years) is averaged, (BR and BW for adults have used the averages values of men and women combined: 14.5 and 69, respectively).

Risk assessment for non-cancer Acute Hazard Indices was estimated. Firstly, the HQ_{Acute} for each pollutant was determined (9) with the 1-h maximum corresponding concentration for each pollutant and the acute reference exposure level (*REL*) for each pollutant correlated with the target organ system(s), according to OEHHA (2015) [24] (Table S6). Then, HQ_{Acute} for each pollutant was summed up for the calculation of the cumulative HI.

$$\text{Acute Hazard Quotient} = \frac{(\text{Maximum 1 h Concentration})}{(\text{Acute REL})} \quad (9)$$

Methodology B.II

In the study by Tianjie Shao et al., 2018 [15], an assessment method of human exposure risk proposed by the U.S. EPA (Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites EPA, 2002; L. Ferreira-Baptista et al., 2005) [44,45], was implemented, to estimate carcinogenic and non—carcinogenic exposure risk. The calculation of the potential cancer risk (Risk) was estimated by using the following Equations (10) and (11):

$$\text{Risk} = \text{LADD} \times \text{SF} \quad (10)$$

$$\text{LADD}_{\text{inh}} = \frac{\text{C} \times \text{EF}}{\text{PEF} \times \text{AT}} \times \left(\frac{\text{InhR}_{\text{child}} \times \text{ED}_{\text{child}}}{\text{BW}_{\text{child}}} + \frac{\text{InhR}_{\text{adult}} \times \text{ED}_{\text{adult}}}{\text{BW}_{\text{adult}}}\right) \quad (11)$$

where SF: the slope cancer factor for each pollutant, $LADD_{inh}$: refers to the daily average exposure for life through the inhalation, C: concentration of pollutant (mg/kg), EF: exposure frequency (days/year), PEF: Particulate emission factor 1.36×10^{-9} (m^3/kg). "This factor represents an estimate of the relationship between soil contaminant concentrations and the concentration of these contaminants in the air as a consequence of particle suspension" [44], AT: mean exposure time (70 years × 365 days), ED: exposure time (years), InhR: Inhalation rate $(m^3 \cdot day^{-1})$ and BW: weight per citizen (kg). The non-carcinogenic risk was evaluated from Equations (12)–(14):

$$\text{HI} = \sum \text{HQ}_{\text{i}} \quad (12)$$

$$\text{HQ} = \text{ADD}/\text{RfD} \quad (13)$$

$$ADD_{inh} = \frac{InhR \times EF \times ED}{PEF \times BW \times AT} \quad (14)$$

where HQ is a non-carcinogenic risk factor that characterizes the non-carcinogenic risk of a single contaminant, ADD_{inh} is a non-carcinogenic risk factor of a single contaminant from a pathway inhalation ($mg \cdot kg^{-1} \cdot day^{-1}$), RfD is the reference dose for the pathway inhalation ($mg \cdot kg^{-1} \cdot day^{-1}$), AT: mean exposure time (ED years × 365 days) and the remaining parameters were the same as those in Equation (11). In this study, the RfD was selected according to EPA, 2002 [44] and non-cancer effects were estimated only for Ni because there were no reference values for other pollutants.

Methodology B.III

In the study of Chalvatzaki et al., (2019) [26], health risk indices caused by inhalation were estimated. Specifically, the cancer risk (CR), non–cancer effects (HQ), and mortality cases (RR) from PM_{10} and $PM_{2.5}$ were calculated. The probability of developing cancer risk (CR) was calculated by using Equation (15):

$$CR = CDI \times CSF \quad (15)$$

where CSF is the cancer slope factor $(mg/kg\ day)^{-1}$ and CDI is the chronic daily intake (mg/kg/day) which were calculated from the following Equations (16) and (17), respectively:

$$CDI = \frac{C_a \times IR \times ET \times EF \times ED}{BW \times AT} \quad (16)$$

$$CSF = IUR \times \left(\frac{BW}{IR_d}\right) \times 10^{-3} \quad (17)$$

C_a is the contaminant concentration (mg/m^3), IR is the inhalation rate (m^3/h), ET is the exposure time (h/day), EF is the exposure frequency (days/year), ED is the exposure duration (years), BW is the body weight (kg) and AT is the averaging time (70 years × 365 days/year), IUR is the inhalation unit risk for each pollutant according to U.S.EPA Regional Screening Level (RSL) Summary Table (2019) [33], except for the case of Pb, where the value IUR_{Pb}: 1.2×10^{-5} proposed by U.S.EPA Regional Screening Level (RSL) Summary Table (2017) [46] was used, and IR_d is the daily inhalation rate (m^3/day). Finally, the number of new cancer cases (I) per lifetime (80 years) in Elefsina was estimated using Equation (18):

$$I = N \times CR \quad (18)$$

CR is the cancer risk probability estimated in this work and N is the number of people in the target city (Elefsina), which according to the latest population census is 29,000 residents. The non-cancer risk assessment was performed using Equations (19) and (20):

$$HQ = CDI/RFD \quad (19)$$

$$RFD = RFC \times (IR_d/BW) \quad (20)$$

where RFD is the reference dose (mg/kg/day), CDI was calculated as in the case of cancer risk Equation (16) with the only difference being the AT calculation (ED years × 365 days/year), RFC is the reference concentration according to Summary Table (2019) [33], except for the case of Pb, where the value RFC_{Pb}: 2×10^{-4} by the U.S. EPA Regional Screening Level Summary Table (2017) [46] was used (Table S7).

The methodology for the estimation of mortality cases was performed according to Ostro et al., 2004 [19]. Relative Risk (RR) for all-cause mortality, for all ages from $PM_{10,}$ was estimated by (21) & (22):

$$RR = \exp[\beta(X - X_0)] \quad (21)$$

$$AF = (RR - 1)/RR \quad (22)$$

where X is the annual mean concentration of PM_{10} ($\mu g/m^3$), and X_0 is the baseline concentration of PM_{10} ($\mu g/m^3$). For the annual mean concentrations (X), the average concentration of $PM_{2.5}$ from E1, E2 and E3 was used (X = 14.4 $\mu g/m^3$) while for the baseline concentration, the average concentration of $PM_{2.5}$ from E3 (X_0 = 3.2 $\mu g/m^3$) was taken into account. Due to the lack of PM_{10} measurements, values of the $PM_{10}/PM_{2.5}$ ratio from the literature were used (for the region of Europe is 0.73; Ostro et al., 2004; Cardaba et al., 2014) [19,47]. Thus, the concentrations for PM_{10} were X = 19.3 and X_0 = 10 $\mu g/m^3$, β is the coefficient of the risk function (0.0008; 95% confidence interval (CI):0.0006–0.0010), AF is the attributable fraction (Equation (22)) which was used to estimate the proportion of deaths from a disease (e.g., lung cancer), and which could have been avoided if PM concentrations were reduced to background concentration, (i.e., concentrations that would exist without any human activities) [19,26]. Finally, the number of attributable deaths (AI) was calculated using Equation (23):

$$AI = AF \times I \quad (23)$$

where I is the total number of deaths in the target population; for the region of Elefsina, this was equal to 222 for the year 2019.

Methodology B.IV

In the study of Megido et al., (2017) [27], a different approach for the assessment of human exposure risk (cancer and non—cancer effects) was proposed (U.S. EPA, 2003, 2009) [35,41]. The determination of cancer risk through inhalation (CR_{inh}) was conducted from the following Equations (24) and (25):

$$CR_{inh} = IUR \times EC_{inh} \quad (24)$$

where IUR is the Inhalation Unit Risk $(\mu g/m^3)^{-1}$ [33], while EC_{inh} is the exposure concentration through inhalation ($\mu g/m^3$), which was estimated by using the following Equation (25):

$$EC_{inh} = C \times ET \times \frac{EF \times ED}{AT_n} \quad (25)$$

C is the concentration of pollutant ($\mu g/m^3$), ET is the exposure time (h/day), EF is the exposure frequency through the year (days/year), ED is the exposure duration (years) and AT_n is the averaging time (70 years × 365 days/year).

The non-cancer risk assessment was evaluated through the hazard quotient (HQ_{inh}):

$$HQ_{inh} = \frac{EC_{inh}}{RfC_i \times 1000 \times \mu g/mg} \quad (26)$$

RfC_i is the inhalation reference concentration (mg/m^3) for each pollutant [33], and EC_{inh} is the exposure concentration through inhalation as estimated from Equation (25) with the only difference of changing the calculation of parameter AT (ED years × 365 days/year).

There are key differences in the above-mentioned approaches (group B) for the risk assessment for carcinogenesis and non-cancer effects. The B.I methodology differs (a) on the values used for the parameters BR, BW, ED, and CR (which were set according to OEHHA) and (b) on the estimation of the cancer risk, as no differentiation in the gender, as well as adjustment of the daily exposure (hours/day), occurs. Additionally, in methodologies B.II, B.III, and B.IV, the parameters for the cancer potency factor and non-cancer factor are different. Method B.I is the only one in which the estimation of the Acute Hazard Quotient for each Target Organ System(s) is feasible. Contrary to B.I, the BII method includes alternative types ($LADD_{inh}$, *PEF*, *RfD*), but lacks the distinction of age and gender. The B.III method is very similar to the A.III methodology for cancer risk assessment, while the only parameter missing in both is the AF parameter. In addition, with this methodology (B.III), the number of new cases of cancer for humans (I) per lifetime (80 years) can be estimated. Contrary to B.III, the parameters AF, daily exposure (IR), body weight (BW),

and people's gender are missing from the B.IV method. However, the outcomes from the two methods are similar (Tables 5 and 6), possibly due to erroneous validation of these parameters in the B.III method.

Table 5. Potential cancer risk assessment for Elefsina area.

Methodology	Lifetime Cancer Risk Cases/10^6						
	Children 0–2 Years	Children 2–16 Years	Women 16–80 Years		Men 16–80 Years		$\Sigma_{0–80\ Years}$ (24 h/Day)
			14 h/Day	24 h/Day	14 h/Day	24 h/Day	
A.I	3.15	0.66	0.08	0.13	0.08	0.14	3.95 [1]
A.II	0.31	0.22	0.08	0.13	0.08	0.14	0.68 [1]
A.III	5.2		7.2	12.4	7.8	13.4	18.6 [1]
A.IV			0.59 (UR $_{OEHHA,\ 2005}$) 46.66 (UR $_{WHO,\ 2010}$)				0.59 46.66 [3]
B.I	1.64	8.0			23.2		33.84 [2]
B.II			0.05				0.05 [3]
B.III	2.3		5.4	9.3	5.4	9.3	11.7 [1]
B.IV	2.3		–		5.4	9.3	11.7 [2]
C.I	68.44	47.80	16.78	28.77	18.11	31.04	147.3 [1]
C.II	68.44	47.80	16.78	28.77	18.11	31.04	147.3 [1]

[1] Calculation of total inhalation lifetime cancer risk were used the estimated cancer risk from all pollutants for ages intervals children$_{0–2}$, children$_{2–16}$ (or children$_{0–16}$) and Men$_{16–80}$ (exposure duration 24 h/day). [2] According to these methodologies, there were not differences to estimations for the gender of adults. [3] These methodologies did not include any separation for the age of the people.

Table 6. Non-cancer risk assessment for Elefsina area.

Methodology	Hazard Quotient						
	Children 0–2 Years	Children 2–16 Years	Women 16–80 Years		Men 16–80 Years		Hazard Index (24 h/Day)
			14 h/Day	24 h/Day	14 h/Day	24 h/Day	
B.I	HQ for each Target organ System(s) & Acute HI (OEHHA, 2015)						
B.II	3.59×10^{-6}			2.33×10^{-6}			5.92×10^{-6} *
B.III	0.5269		0.3074	0.5269	0.3074	0.5269	1.05 **
B.IV	0.5269		–		0.3074	0.5269	1.05 **

* In this methodology there was no separation of gender and duration of daily exposure people were exposed. ** For the calculation of total inhalation lifetime non-cancer risk, the estimated non-cancer risk from all pollutants for ages intervals Children$_{0–2,}$ Children$_{2–16}$ (or Children$_{0–16}$) and Men$_{16–80}$ (exposure duration 24 h/day) was used.

2.3.3. Risk Assessment Approach Based on Methodologies Combination

In the present study, a combination of methodologies, which were presented in the previous paragraphs, was attempted in order to apply a different approach for estimating the risk of developing cancer over a lifetime for the residents of the Elefsina area (Table S4).

Methodology C.I

From a combination of the methodologies described in the studies of Xu et al., 2018 and Farris et al., 2014 [25,48], the potential cancer effect caused by exposure to air pollution was estimated. In particular, in Xu et al., 2018, the daily exposure dose (D_w) by air pollutants was calculated according to the following Equation (27):

$$D_w = \sum_{i=1} C_i \times \sum_{j=1} T_{ij} \times IR_j \quad (27)$$

C: the concentration of pollutant (in $\mu g/m^3$), Tij is the exposure time (in h/h), IR is the inhalation rate adjusted with the bodyweight of people (m^3/day-kg) [23]. Secondly, the potential cancer effects (Risk) from the study of Farris et al., 2014 [25] were based on the third edition of the book "Encyclopedia of Toxicology (2014)", describing the cancer

potency factor parameter (CPF) for quantifying the risk of chemical factors which are evaluated as carcinogenic (Equation (28)). 'Dose' is the exposure dose-adjusted with BW (mg/kg-day) and CPF is the cancer potency factor for each pollutant according to OEHHA, 2019 [43] (Table S3).

$$\text{Risk} = \text{Dose} \times \text{CPF} \quad (28)$$

Methodology C.II

In this case, the potential cancer effect was estimated based on the combination of the methodologies of Maertens et al., 2004 and Farris et al., 2014, [13,24]. The difference from the previous methodology is the estimation of the Exposure Dose (*or DW*) of people to air pollutants, which was calculated through Equation (29), instead of Equation (27), *Maertens, 2004*. Subsequently, the probability of developing cancer *(Risk)* was calculated similarly to the previous methodology (C.I) [25,48].

$$\text{Lifetime average daily exposure dose} = \frac{\text{C} \times \text{IR} \times \text{EF}}{\text{BW}} \quad (29)$$

$$\text{Risk} = \text{Dose} \times \text{CPF} \quad (30)$$

3. Results and Discussion

The potential cancer and non cancer risk assessments for each methodology are presented in Tables 5 and 6, and the estimated risks for each pollutant are presented in the Appendix A (Tables A1–A14).

3.1. Cancer Risk Assessment

The cancer risk (through inhalation) was estimated, using the methodologies described in the previous paragraphs, for each pollutant and then summed to estimate the total lifetime cancer risk, for the residents of Elefsina (Table 5). The results were different according to the methodology and the formulas applied in each case; the examined pollutants and the parameters are taken into account in each case (e.g., the age, the parameters of Exposure Duration, Cancer Potency).

Risk estimation using approaches A.I and A.II [13,14] resulted in low potential risks values (Tables A1 and A2). The results for adults (men versus women) were similar differing to those for children ($0–2_{\text{years}}$ vs. $2–16_{\text{years}}$ group) which were different due to the presence of the adjustment factor (AF). As a result, the calculated risk included the AF parameter, which was higher for every PAH for both age groups (Tables 5 and 6). It was also observed that the risk of developing cancer increases as age decreases due to IR and BW factors. The higher value was calculated for benzo(a)pyrene (1.09×10^{-6}) and dibenz[a,h]anthracene (1.95×10^{-6}) (due to high PEF and C) and the lower one for chrysene (9.09×10^{-12}).

On the contrary, the estimation performed using the methodology A.III, [31] showed conflicting results. The estimated cancer risk was higher for adults, compared to children as the length of the exposure time for adults (L) was larger, a parameter that is absent from approaches A.I and A.II. Furthermore, in the A.III method, the parameter of exposure frequency (EF) was included and the risk was not estimated only for PAHs. As a result, the total lifetime cancer risk (18.6 cases per 10^6 residents) and the individual risks for each pollutant were higher (Table A3). The higher potential risk was estimated for benzene (1.27×10^{-5}) and the lower one for chrysene (6.88×10^{-12}), similarly to AI and AII methods.

According to approach A.IV, the total lifetime cancer risk was estimated for two different UR cases (1.1×10^{-6}, OEHHA [37] and 8.7×10^{-5}, WHO [39]), while there were no differences for estimations related to age or gender of the residents (Table A4). Moreover, the parameters for daily exposure duration, exposure frequency, and the years of exposure (duration) are missing from this methodology. It is worthy to be mentioned that the RPF_i parameter had a significant influence on the estimation of risk. Specifically, DBahA had the lowest concentration (0.02 ng/m^3) among PAHs, but the corresponding estimated

cancer risk was the second highest, because DBahA had a high value of the parameter RPF_i compared to the other PAHs. Additionally, in the A.IV approach, the parameter (RPF_i) for the cancer potency is different for each PAH [35]. In contrast, in the A.III method, it is the PEF_i parameter that is used for the cancer risk estimation, leading to different results.

As a result, the estimated risks were higher with the A.IV methodology, with BkF (2.01×10^{-5}) and DBahA (1.74×10^{-5}) pollutants being the highest. Furthermore, in A.IV higher cancer assessment was estimated with the UR according to WHO (2010) for all PAHs, except for the case where UR was used according to OEHHA (2005).

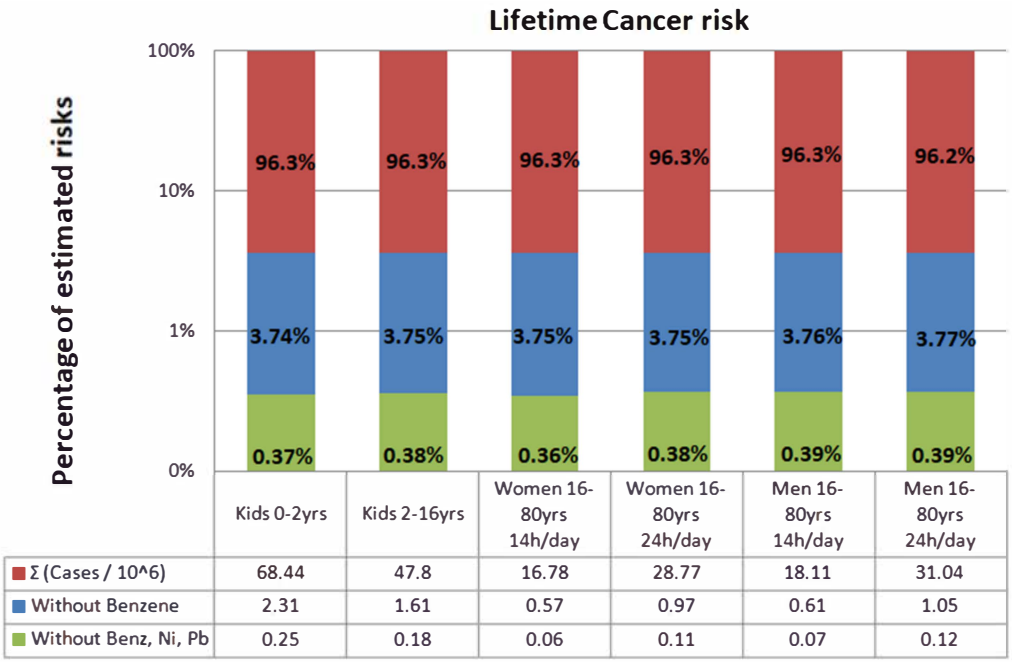
According to the B.I approach, the total inhalation cancer risk as a result of an 80-year exposure to a variety of pollutants was estimated to be 33 chances per million people (Table 6). The risk increased with increasing age, while cancer risk for adults was higher in contrast to children (Table A5). The same assessment was conducted based on the methodologies B.II, B.III, and B.IV and the increase in the estimated risk was proportional to the increase in human age [15,27,28]. In the B.I methodology, the highest cancer risk of all pollutants was estimated for benzene (2.24×10^{-5}, for Adults) and the lower one for chrysene (4.81×10^{-11}, for $Children_{0–2\ years}$), (Table A5). With the application of methodology B.II, the lower total lifetime cancer risk was estimated (0.05 chances per million, Table A7). A significant difference between the two methodologies was the absence of age and gender parameters, resulting in no differentiation in the potential estimated risks between women, men, and children. Furthermore, the B.I method lacks the parameter of the daily exposure dose. The highest cancer risk was estimated for Ni (4.47×10^{-8}) and the lowest for chrysene (9.78×10^{-12}). In general, a parameter that significantly differentiates the outcomes of the several approaches is the Cancer Potency of each pollutant.

According to the B.III methodology, the Total Cancer Risk was estimated to be 11.7 cases per 10^6 humans and the risk assessment from long-term exposure are presented in Table A10. For men and women, the cancer risk was the same (9.3 cases per million) while for $children_{0–16}$ was 2.3 cases per million (Tables 5 and A8). The potential risk for men and women calculated for all pollutants gave the same results. It is important to mention that in the B.III methodology, the parameters IR_d and BW were included in the equation for the Chronic daily intake dose Equation (16) and in the equation for the Cancer slope factor Equation (17). As a result, the fraction of BW and IR did not differentiate the estimated risk for women and men (Table A8). Similar results were observed for the assessment of cancer and non-cancer effects, which means that the parameters BW and IR were not properly expressed. For example, as shown in Tables 5 and 6 or Tables A8 and A9, the estimated values for men and women are the same, even though the parameter of gender is included. It is well known that the IR clearly constitutes a crucial parameter for the calculation of the daily intake dose, and consequently for the risk estimation.

Furthermore, the duration of the daily exposure was proven to be a critical parameter for potential cancer risk calculation (Table A8). Moreover, the number of new cancer cases (I) per lifetime (80 years) in Elefsina was found to be low (0.28 cases for adults and 0.07 for children), due to the limited number of residents (29,900). However, methodology B.IV presented significant differences (IR,BW) from B.III, the results of the cancer risks assessment were found to be the same as those of B.III methodology. In contrast to B.III, the parameters of IR and BW were absent from the B.IV methodology (Table A11). Nevertheless, the results among these two methodologies were similar, verifying that in the B.III methodology the parameters IR and BW do not lead to differentiation in the estimated risks, because they were used for the calculation of both CDI and CSF did not differentiate the assessment. In addition, in B.IV the parameter IUR was included, instead of the CSF parameter from the B.III methodology, in which the CSF Equation (17) was calculated from the IR, BW and IUR. For both methodologies B.III and B.IV the highest cancer risk was found for benzene (8.32×10^{-6} for adults) and the lowest for chrysene (1.24×10^{-11} for $children_{0–16\ years}$).

The combination of the methods (C.I & C.II) leads to the highest total cancer risk, i.e., 147.3 cases per million people (Table 5). The potential cancer risk was observed to increase as the age-range of people decreases, due to IR and BW factors. The daily exposure

duration was proved to be an important parameter with 58% fewer cases (18.11 cases per million) of cancer estimated for men having 14 h of exposure per day (Table A14) compared to those men who were exposed for 24 h per day (31.04 cases per million). The comparison of the results from the cancer risk assessment as presented in the following Figure 3 and the difference of these estimated risks between benzene and other pollutants is noteworthy.



	Kids 0-2yrs	Kids 2-16yrs	Women 16-80yrs 14h/day	Women 16-80yrs 24h/day	Men 16-80yrs 14h/day	Men 16-80yrs 24h/day
Σ (Cases / 10^6)	68.44	47.8	16.78	28.77	18.11	31.04
Without Benzene	2.31	1.61	0.57	0.97	0.61	1.05
Without Benz, Ni, Pb	0.25	0.18	0.06	0.11	0.07	0.12

Figure 3. Percentage of cancer risk assessment (C.I method).

Specifically, if the potential cancer risk related to benzene is removed from the total cancer risk of all pollutants, then the cancer chances decrease significantly (children$_{0–2\ Years}$: 68.44 cases per million with Benzene, 2.31 cases without Benzene, 0.25 without Benzene, Ni and Pb) and if these three pollutants are removed from the total assessment, the estimated cancer risks are also significantly lower. This result is independent of the range of age of the people (kids & adults), which could be also concluded from Table 5 or Table A13. It should be noted that the parameter of the length of the exposure duration (years) of humans was not included in the C.I methodology.

3.2. Non-Cancer Effects

The non-cancer risk (through inhalation) for the residents of Elefsina was estimated and compared, using the methodologies, for each pollutant (HQ) and then summed for the total lifetime non-cancer effects (HI), (Tables 6 and 7). There were no exceedances of the acceptable limits of HI and HQ, for all the methodologies used (HI & HQ < 1).

The results indicate several differences depending on the applied methodology (Table 8). Firstly, according to methodology B.I [24] the risk for non-cancer acute health impacts of each pollutant was estimated and expressed as a Hazard Quotient (individual substances) or a Hazard Index (multiple substances) for the target organ system(s). As shown in Table 6, the non-cancer risk was low, due to low concentrations of the pollutants. As a result, the HI values for every target organ system were low (Table A6), especially for the Respiratory, Nervous Systems, and Eyes (2.28×10^{-4}), while the higher risk was calculated for Immune, Hematologic Systems, and Reproductive/Development (1.72×10^{-2}). In this methodology, the non-cancer risk did not differentiate between age groups and gender, while the parameters of exposure duration were absent. On the other hand, by using method B.II, it was possible to estimate the total non-cancer risk for children and adults, based on their exposure to Ni. The risk was estimated to be 3.59×10^{-6} for children and 2.33×10^{-6} for adults (Table 6). According to the methodology B.III, the non-cancer effects

were assessed for several pollutants, for different age ranges and gender of people, as well as for various exposure periods (Daily exposure, Exposure days/year & length of years exposure duration). The Index for the total non-cancer risk (HI) for the daily exposure time of 24 h per day was estimated to be 1.05, while the estimated HQ separately for children, women, and men was the same, 0.5269 (Table A9). As previously mentioned, IR and BW parameters did not affect the estimated risk value, as they are included in the equation for the calculation of reference dose (RFD) for each pollutant. In addition, for the case of children, the results coincide with those of adults, despite the fact that the duration of the exposure is practically different. This can be attributed to the parameter AT (ED years × 365 days/year), since for the calculation of the non-cancer effects, in AT parameter was included the ED (31), while in the calculation of cancer assessment the AT parameter was not included (80 years × 365 days/year).

Equation (16), Non-cancer: AT= ED years × 365 days/year, Cancer: AT= 80 years × 365 days/year.

The daily exposure duration proved to be a critical parameter for assessing the non-cancer impacts. The higher potential risk among the pollutants (HQ) was calculated for nickel (0.21) for children and the lower one for toluene (3.99×10^{-4}) for adults. In the assessment for the non-cancer risk according to the methodology by B.IV [27] (Table A12), while there were significant differences in the parameters included in both methodologies, the results of the assessment were the same as the results by B.III [26]. In this methodology (B.IV), the parameters IR, BW, and RFD were not included in the equation. A situation that confirms the lack of influence of these parameters in B.III Equations (17) and (20), whether included or not. As a result, the highest and the lowest non-cancer risk for B.III & B.IV was estimated for Nickel (0.21) for children and for Toluene (3.99×10^{-4}) for adults (women & men).

Table 7. HQ for each Target organ System(s) & Acute HI (OEHHA, 2015).

Substance	C (μg/m^3)	Total HQ$_{Acute}$	Immune System	Reproductive/ Development	Hematologic System	Respiratory System	Eyes	Nervous System
Benzene	1.39	5.1×10^{-2}	1.72×10^{-2}	1.72×10^{-2}	1.72×10^{-2}	–	–	–
Toluene	3.56	9.62×10^{-5}	–	2.41×10^{-5}	–	2.41×10^{-5}	2.41×10^{-5}	2.41×10^{-5}
Xylenes 1	3.08	1.40×10^{-4}	–	–	–	4.67×10^{-5}	4.67×10^{-5}	4.67×10^{-5}
Xylenes 2	2.76	1.25×10^{-4}	–	–	–	4.18×10^{-5}	4.18×10^{-5}	4.18×10^{-5}
Xylenes 3	7.53	3.42×10^{-4}	–	–	–	1.14×10^{-4}	1.14×10^{-4}	1.14×10^{-4}
Total Acute Hazard Index			1.72×10^{-2}	1.72×10^{-2}	1.72×10^{-2}	2.27×10^{-4}	2.27×10^{-4}	2.28×10^{-4}

Table 8. Table of methodologies including parameters.

Methodology	Gender	Ages	AF	IR & BW	Exposure (h/Day)	Exposure (Day/Years)	Exposure (Years)
A.I	✓	✓	✓	✓	✓	**X**	**X**
A.II	✓	✓	**X**	✓	✓	**X**	**X**
B.I	**X**	✓	**X**	✓	✓	✓	✓
B.II	**X**	**X**	**X**	✓	✓	✓	✓
A.III	✓	✓	**X**	✓	✓	✓	✓
B.III	✓	✓	**X**	✓	✓	✓	✓
B.IV	**X**	✓	**X**	✓	**X**	✓	✓
A.IV	**X**	**X**	**X**	**X**	**X**	**X**	**X**
C.I	✓	✓	**X**	✓	✓	**X**	**X**
C.II	✓	✓	**X**	✓	✓	**X**	**X**

4. Conclusions

This study aimed to estimate the health risks caused by exposure to air pollution for people living in an urban, industrialized area of Greece. Health effects on different population subgroups were estimated for targeted pollutants ($PM_{2.5}$, PAHs, heavy metals and VOCs) through different mathematical approaches provided by the literature and taking into consideration different parameters, such as age, gender and daily average exposure time. The main conclusions of the study are summarized in the following:

- The estimation of the risk for potential cancer and non-cancer effects varies depending on the applied methodology; however, the acceptable limits for cancer risk provided by OEHHA, EPA and WHO were not exceeded in any case except for one approach, as well as only one case (C.I & C.II) which were found to be in excess of the acceptable limits of cancer risk (10^{-6}–10^{-4}) for the total lifetime.
- Several methodologies lack critical parameters (e.g., IR, BW, AF) which are important for the estimation of the exposure dose of people, depending on their age, gender, and the daily activity of people. In six approaches in which there was the possibility to estimate the exposure specifically for women, the results were lower compared to those for men (with the exception of methods B.III & B.IV). The main reason was the existence of IR and BW parameters.
- The duration of the exposure to air pollution (i.e., hours per day, days per year and number of years) is a major factor influencing the estimated risks and was differentiated as a function of the time the people were exposed to the pollutants, regardless of people gender, age or other parameters.
- Age adjustment factor (AF) proves to be a critical parameter for cancer risk assessment as, during the early stages of life, people are more vulnerable to the effects of chemical toxins.
- Benzene comprises a critical factor as its estimated cancer risk was significant, mainly due to its high concentration and its high cancer slope factor, as well. Additionally, pollutants with low concentrations (DBahA, BaP) have been associated with high potential risks, due to their high value of parameters for the corresponding risk (CSF,P or RFD, HQ).
- In the majority of the methodologies used, the younger the age of people who were studied, the lower the estimated risk for cancer. In cases A.I, A.II, A.IV, C.I & C.II where the duration of exposure was not taken under consideration, the risk for children was equal to or higher than that of adults.

Limitations—Future Study

The assessment was performed according to literature references, as certain parameters used in risk calculation formulas were not available for all examined pollutants (i.e., cancer slope factor, reference dose). This could comprise a limitation of the present study but at the same time a challenge for future research. Moreover, it is important that there are no widely accepted values for all these parameters and as a result, different values of the parameters are selected in every study and consequently, lead to different assessments. Additionally, the collection of air pollution data of higher temporal (i.e., daily and seasonal variation) and spatial resolution (network of sampling sites) would contribute to a more robust risk estimation, as the influence of environmental and human activity factor variability would be taken into account. For this purpose, the scientific community needs to focus on more comprehensive mathematical models and approaches, which will take into consideration all the parameters of the relation between exposure to air pollution and population health.

The limited number of sampling sites, seasonal influences, changes in population vulnerabilities, and influences of lifestyle characteristics.

Supplementary Materials: The following supporting information can be downloaded at: https://www.mdpi.com/article/10.3390/atmos13040615/s1; Table S1. Values of the limit of detection (LOD), the limit of quantitation (LOQ) and the expanded uncertainty at 95% confidence level and k = 2 that has been estimated experimentally (Uexp) for each detected PAH; Table S2. Description of gaseous pollutant recording methods; Table S3. PAHs, PEF & SF (OEHHA, 2015 & 2019); Table S4. Studies were used for risk assessment of cancer and non cancer effects; Table S5. Cancer potency factor, OEHHA (2015 & 2019); Table S6. Concentrations, Acute RELs & Target Organ System(s) (OEHHA Appendices, 2015); Table S7. Parameters for non – cancer risks.

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Glossary

PM	Particulate Matter
IARC	International Agency for Research on Cancer
PAHs	Polycyclic Aromatic Hydrocarbons
DALYs	Disability Adjusted Life Years
VOCs	Volatile Organic Compounds
SVOCs	Semivolatile Organic Compounds
IR	Inhalation Rate
BW	Body weight
CR	Cancer Risk
OEHHA	Office of Environmental Health Hazard Assessment
PEF	Potency Equivalency Factor
SF	Slope Factor
EF	Exposure Factor
AF	Adjustment Factor
ED	Exposure Duration
AT	Averaging Time
RPF	Relative Potency Factor
ASF	Age Sensitivity Factor
FAH	Fraction At Home
RfD	Reference Dose
RR	Relative Risk
CDI	Chronic Daily Intake
IUR	Inhalation Unit Risk
EC	Exposure Concentration

Appendix A. Tables with Results of Assessment

Table A1. Cancer risk assessment A.I method (Maertens, 2008).

PAHs	C (ng/m^3)	Lifetime Cancer Risk:					
		Children 0–2 Years	Children 2–16 Years	Women 16–80 Years		Men 16–80 Years	
				14 h/Day	24 h/Day	14 h/Day	24 h/Day
BaP	0.059	1.09×10^{-6}	2.29×10^{-7}	2.68×10^{-8}	4.60×10^{-8}	2.90×10^{-8}	4.97×10^{-8}
BaA	0.053	9.83×10^{-9}	2.06×10^{-9}	2.41×10^{-10}	4.13×10^{-10}	2.60×10^{-10}	4.46×10^{-10}
BbF	0.289	5.36×10^{-8}	1.12×10^{-8}	1.31×10^{-9}	2.25×10^{-9}	1.42×10^{-9}	2.43×10^{-9}
BkF	0.079	1.47×10^{-8}	3.07×10^{-9}	3.59×10^{-10}	6.16×10^{-10}	3.88×10^{-10}	6.65×10^{-10}
CHRY	0.108	2.00×10^{-10}	4.20×10^{-11}	4.91×10^{-12}	8.42×10^{-12}	5.30×10^{-12}	9.09×10^{-12}
DBahA	0.02	1.95×10^{-6}	4.09×10^{-7}	4.78×10^{-8}	8.20×10^{-8}	5.16×10^{-8}	8.85×10^{-8}
I123cdP	0.135	2.50×10^{-8}	5.25×10^{-9}	6.14×10^{-10}	1.05×10^{-9}	6.63×10^{-10}	1.14×10^{-9}
Σ		3.15×10^{-6}	6.60×10^{-7}	7.72×10^{-8}	1.32×10^{-7}	8.33×10^{-8}	1.43×10^{-7}
Cases/10^6		3.15	0.66	0.08	0.13	0.08	0.14

Table A2. Cancer risk assessment A.II method (Maertens, 2004).

PAHs	C (ng/m^3)	Lifetime Cancer Risk					
		Children 0–2 Years	Children 2–16 Years	Women 16–80 Years		Men 16–80 Years	
				14 h/Day	24 h/Day	14 h/Day	24 h/Day
BaP	0.059	1.09×10^{-7}	7.65×10^{-8}	2.68×10^{-8}	4.60×10^{-8}	2.90×10^{-8}	4.97×10^{-8}
BaA	0.053	9.83×10^{-10}	6.87×10^{-10}	2.41×10^{-10}	4.13×10^{-10}	2.60×10^{-10}	4.46×10^{-10}
BbF	0.289	5.36×10^{-9}	3.75×10^{-9}	1.31×10^{-9}	2.25×10^{-9}	1.42×10^{-9}	2.43×10^{-9}
BkF	0.079	1.47×10^{-9}	1.02×10^{-9}	3.59×10^{-10}	6.16×10^{-10}	3.88×10^{-10}	6.65×10^{-10}
CHRY	0.108	2.00×10^{-11}	1.40×10^{-11}	4.91×10^{-12}	8.42×10^{-12}	5.30×10^{-12}	9.09×10^{-12}
DBahA	0.02	1.95×10^{-7}	1.36×10^{-7}	4.78×10^{-8}	8.20×10^{-8}	5.16×10^{-8}	8.85×10^{-8}
I123cdP	0.135	2.50×10^{-9}	1.75×10^{-9}	6.14×10^{-10}	1.05×10^{-9}	6.63×10^{-10}	1.14×10^{-9}
Σ		3.15×10^{-7}	2.20×10^{-7}	7.72×10^{-8}	1.32×10^{-7}	8.33×10^{-8}	1.43×10^{-7}
Cases/10^6		0.31	0.22	0.08	0.13	0.08	0.14

Table A3. Cancer risk assessment A.III method (Jackson, 2005).

Pollutants	C (ng/m^3)	Lifetime Cancer Risk				
		Children 0–16 Years	Women 16–80 Years		Men 16–80 Years	
			14 h/Day	24 h/Day	14 h/Day	24 h/Day
BaP	0.059	3.76×10^{-9}	5.28×10^{-9}	9.05×10^{-9}	5.70×10^{-9}	9.77×10^{-9}
BaA	0.053	3.38×10^{-10}	4.74×10^{-10}	8.13×10^{-10}	5.12×10^{-10}	8.77×10^{-10}
BbF	0.289	1.84×10^{-9}	2.59×10^{-9}	4.43×10^{-9}	2.79×10^{-9}	4.78×10^{-9}
BkF	0.079	5.03×10^{-11}	7.07×10^{-11}	1.21×10^{-10}	7.63×10^{-11}	1.31×10^{-10}
CHRY	0.108	6.88×10^{-12}	9.67×10^{-12}	1.66×10^{-11}	1.04×10^{-11}	1.79×10^{-11}
DBahA	0.02	1.27×10^{-9}	1.79×10^{-9}	3.07×10^{-9}	1.93×10^{-9}	3.31×10^{-9}
I123cdP	0.135	8.60×10^{-10}	1.21×10^{-9}	2.07×10^{-9}	1.30×10^{-9}	2.23×10^{-9}
1Methyl	0.577	1.07×10^{-9}	1.50×10^{-9}	2.57×10^{-9}	1.62×10^{-9}	2.77×10^{-9}
Ni	4.38	2.54×10^{-7}	3.57×10^{-7}	6.12×10^{-7}	3.85×10^{-7}	6.60×10^{-7}
Pb	8.03	2.15×10^{-8}	3.02×10^{-8}	5.17×10^{-8}	3.26×10^{-8}	5.58×10^{-8}
Benzene	1390	4.87×10^{-6}	6.84×10^{-6}	1.17×10^{-5}	7.38×10^{-6}	1.27×10^{-5}
Σ		5.83×10^{-6}	1.10×10^{-5}	1.89×10^{-5}	1.15×10^{-5}	1.98×10^{-5}
Cases/10^6		5.2	7.2	12.4	7.8	13.4

Table A4. Cancer risk assessment A.IV method (Gao, 2019).

Pollutants	C (ng/m^3)	Lifetime Cancer Risk	
		UR (OEHHA. 2005)	UR (WHO. 2010)
BaP	0.059	6.49×10^{-8}	5.13×10^{-6}
BaA	0.053	1.17×10^{-8}	9.22×10^{-7}
BbF	0.289	2.54×10^{-7}	2.01×10^{-5}
BkF	0.079	2.61×10^{-9}	2.06×10^{-7}
CHRY	0.108	1.19×10^{-8}	9.40×10^{-7}
DBahA	0.02	2.20×10^{-7}	1.74×10^{-5}
I123cdP	0.135	1.04×10^{-8}	8.22×10^{-7}
BghiP	0.222	2.20×10^{-9}	1.74×10^{-7}
FLA	0.136	1.20×10^{-8}	9.47×10^{-7}
Σ		5.90×10^{-7}	4.67×10^{-5}
Cases/10^6		0.59	46.66

Table A5. Cancer risk assessment B.I method (OEHHA, 2015).

Pollutants	C (ng/m^3)	Lifetime Cancer Risk			
		Children 0–2 Years	Children 2–16 Years	Adults 16–80 Years	Total Cancer Risk 80 Years
BaP	0.059	2.63×10^{-9}	1.28×10^{-8}	3.71×10^{-8}	5.26×10^{-8}
BaA	0.053	2.36×10^{-10}	1.15×10^{-9}	3.34×10^{-9}	4.73×10^{-9}
BbF	0.289	1.29×10^{-9}	6.29×10^{-9}	1.82×10^{-8}	2.58×10^{-8}
BkF	0.079	3.52×10^{-10}	1.72×10^{-9}	4.97×10^{-9}	7.04×10^{-9}
CHRY	0.108	4.81×10^{-11}	2.35×10^{-10}	6.80×10^{-10}	9.63×10^{-10}
DBahA	0.02	9.36×10^{-10}	4.58×10^{-9}	1.32×10^{-8}	1.87×10^{-8}
I123cdP	0.135	6.01×10^{-10}	2.94×10^{-9}	8.50×10^{-9}	1.20×10^{-8}
Nickel	4.38	4.55×10^{-8}	2.23×10^{-7}	6.43×10^{-7}	9.11×10^{-7}
Lead	8.03	3.85×10^{-9}	1.88×10^{-8}	5.44×10^{-8}	7.71×10^{-8}
Benzene	1390	1.59×10^{-6}	7.76×10^{-6}	2.24×10^{-5}	3.18×10^{-5}
Σ		1.64×10^{-6}	8.03×10^{-6}	2.32×10^{-5}	3.29×10^{-5}
Cases/10^6		1.64	8.0	23.2	33

Table A6. HQ for each Target organ System(s) & Acute HI. B.I method (OEHHA, 2015).

Substance	C (μg/m^3)	Total HQ_{Acute}	Immune System	Reproductive/ Development	Hematologic System	Respiratory System	Eyes	Nervous System
Benzene	1.39	5.1×10^{-2}	1.72×10^{-2}	1.72×10^{-2}	1.72×10^{-2}	–	–	–
Toluene	3.56	9.62×10^{-5}	–	2.41×10^{-5}	–	2.41×10^{-5}	2.41×10^{-5}	2.41×10^{-5}
Xylenes 1	3.08	1.40×10^{-4}	–	–	–	4.67×10^{-5}	4.67×10^{-5}	4.67×10^{-5}
Xylenes 2	2.76	1.25×10^{-4}	–	–	–	4.18×10^{-5}	4.18×10^{-5}	4.18×10^{-5}
Xylenes 3	7.53	3.42×10^{-4}	–	–	–	1.14×10^{-4}	1.14×10^{-4}	1.14×10^{-4}
Total Acute Hazard Index			1.72×10^{-2}	1.72×10^{-2}	1.72×10^{-2}	2.27×10^{-4}	2.27×10^{-4}	2.28×10^{-4}

Table A7. Risk assessment B.II method (Tianjie, 2018).

Pollutants	C (mg/kg)	Lifetime Cancer Risk		Non Carcinogenic Risk			
				$ADD_{0–16\ Years}$	$ADD_{16–80\ Years}$	$HQ_{0–16\ Years}$	$HQ_{16–80\ Years}$
Ni	315.7	4.47×10^{-8}		7.40×10^{-8}	4.80×10^{-8}	3.59×10^{-6}	2.33×10^{-6}
BaP	4.46	4.14×10^{-11}					
BaA	3.91	4.81×10^{-10}					
BbF	21.59	2.66×10^{-9}	NO RfD				
BkF	5.93	7.30×10^{-11}					
CHRY	7.95	9.78×10^{-12}			NO RfD		
DBahA	1.49	1.83×10^{-9}					
I123cdP	10.20	1.26×10^{-9}					
Σ		5.11×10^{-8}					
Cases/10^6		0.05					

Table A8. Cancer risk assessment B.III method (Chalvatzaki, 2019).

Pollutants		C (ng/m^3)	Children 0–16 Years	Lifetime Cancer Risk			
				Women 16–80 Years		Men 16–80 Years	
				14 h/Day	24 h/Day	14 h/Day	24 h/Day
	BaP	0.059	6.79×10^{-9}	1.58×10^{-8}	2.72×10^{-8}	1.58×10^{-8}	2.72×10^{-8}
	BaA	0.053	6.10×10^{-10}	1.42×10^{-9}	2.44×10^{-9}	1.42×10^{-9}	2.44×10^{-9}
	BbF	0.289	3.33×10^{-9}	7.76×10^{-9}	1.33×10^{-8}	7.76×10^{-9}	1.33×10^{-8}
PAH	**BkF**	0.079	9.09×10^{-11}	2.12×10^{-10}	3.64×10^{-10}	2.12×10^{-10}	3.64×10^{-10}
	CHRY	0.108	1.24×10^{-11}	2.90×10^{-11}	4.97×10^{-11}	2.90×10^{-11}	4.97×10^{-11}
	DBahA	0.02	2.30×10^{-9}	5.37×10^{-9}	9.21×10^{-9}	5.37×10^{-9}	9.21×10^{-9}
	I123cdP	0.135	1.55×10^{-9}	3.62×10^{-9}	6.21×10^{-9}	3.62×10^{-9}	6.21×10^{-9}
Heavy	**Ni**	4.38	2.18×10^{-7}	5.10×10^{-7}	8.74×10^{-7}	5.10×10^{-7}	8.74×10^{-7}
metals	**Pb**	8.03	1.85×10^{-8}	4.31×10^{-8}	7.39×10^{-8}	4.31×10^{-8}	7.39×10^{-8}
VOC	**Benzene**	1390	2.08×10^{-6}	4.85×10^{-6}	8.32×10^{-6}	4.85×10^{-6}	8.32×10^{-6}
Σ			2.33×10^{-6}	5.44×10^{-6}	9.32×10^{-6}	5.44×10^{-6}	9.32×10^{-6}
Cases/10^6			2.3	5.4	9.3	5.4	9.3
I (Elefsina) *			0.07	0.16	0.28	0.16	0.28

* Cases cancer risk for Elefsina (29900 residents).

Table A9. Non-cancer risk assessment B.III method (Chalvatzaki, 2019).

Pollutants	C (ng/m^3)	Hazard Quotient				
		Children 0–16 Years	Women 16–80 Years		Men 16–80 Years	
			14 h/Day	24 h/Day	14 h/Day	24 h/Day
BaP	0.059	0.0283	0.0165	0.0283	0.0165	0.0283
Ni	4.38	0.2100	0.1225	0.2100	0.1225	0.2100
Pb	8.03	0.0385	0.0225	0.0385	0.0225	0.0385
Benzene	1390	0.0444	0.0259	0.0444	0.0259	0.0444
Toluene	3563	6.83×10^{-4}	3.99×10^{-4}	6.83×10^{-4}	3.99×10^{-4}	6.83×10^{-4}
Trimethylben	2300	3.68×10^{-2}	2.14×10^{-2}	3.68×10^{-2}	2.14×10^{-2}	3.68×10^{-2}
m-Xylene	3077	2.95×10^{-2}	1.72×10^{-2}	2.95×10^{-2}	1.72×10^{-2}	2.95×10^{-2}
o-Xylene	2760	2.65×10^{-2}	1.54×10^{-2}	2.65×10^{-2}	1.54×10^{-2}	2.65×10^{-2}
p-Xylene	7533	7.22×10^{-2}	4.21×10^{-2}	7.22×10^{-2}	4.21×10^{-2}	7.22×10^{-2}
Cyclohexane	7187	1.15×10^{-3}	6.70×10^{-3}	1.15×10^{-3}	6.70×10^{-3}	1.15×10^{-3}
Nonane	773	3.71×10^{-2}	2.16×10^{-2}	3.71×10^{-2}	2.16×10^{-2}	3.71×10^{-2}
Hazard Index		0.5269	0.3074	0.5269	0.3074	0.5269

Table A10. Risk assessment from long-term exposure to PM, B.III method (Chalvatzaki, 2019).

Parameters	All Cause Mortality (PM_{10}) (β = 0.0008)	Cardiopulmonary Mortality ($PM_{2.5}$) (β = 0.15515)	Lung Cancer Mortality ($PM_{2.5}$) (β = 0.23218)
ER (95% CI)	0.008	0.223	0.352
AF (95% CI)	0.008	0.183	0.260
Deaths (95% CI)	5.76×10^{-5}	1.36×10^{-3}	1.93×10^{-3}

Table A11. Cancer risk assessment B.IV method (Megido, 2017).

Pollutants		C (ng/m^3)	Lifetime Cancer Risk: Children 0–16 Years	Adults 16–80 Years 14 h/Day	Adults 16–80 Years 24 h/Day
PAH	BaP	0.059	6.79×10^{-9}	1.58×10^{-8}	2.72×10^{-8}
	BaA	0.053	6.10×10^{-10}	1.42×10^{-9}	2.44×10^{-9}
	BbF	0.289	3.33×10^{-9}	7.76×10^{-9}	1.33×10^{-8}
	BkF	0.079	9.09×10^{-11}	2.12×10^{-10}	3.64×10^{-10}
	CHRY	0.108	1.24×10^{-11}	2.90×10^{-11}	4.97×10^{-11}
	DBahA	0.02	2.30×10^{-9}	5.37×10^{-9}	9.21×10^{-9}
	I123cdP	0.135	1.55×10^{-9}	3.62×10^{-9}	6.21×10^{-9}
Heavy metals	Ni	4.38	2.18×10^{-7}	5.10×10^{-7}	8.74×10^{-7}
	Pb	8.03	1.85×10^{-8}	4.31×10^{-8}	7.39×10^{-8}
VOC	Benzene	1390	2.08×10^{-6}	4.85×10^{-6}	8.32×10^{-6}
	Σ		2.33×10^{-6}	5.44×10^{-6}	9.32×10^{-6}
	Cases/10^6		2.3	5.4	9.3

Table A12. Non-cancer risk assessment B.IV method (Megido, 2017).

Pollutants		C (ng/m^3)	Hazard Quotient: Children 0–16 Years	Adults 16–80 Years 14 h/Day	Adults 16–80 Years 24 h/Day
PAH	BaP	0.059	0.0283	0.0165	0.0283
Heavy metals	Ni	4.38	0.2100	0.1225	0.2100
	Pb	8.03	0.0385	0.0225	0.0385
VOC	Benzene	1390	0.0444	0.0259	0.0444
	Toluene	3563	6.83×10^{-4}	3.99×10^{-4}	6.83×10^{-4}
	Trimethylben	2300	3.68×10^{-2}	2.14×10^{-2}	3.68×10^{-2}
	m-Xylene	3077	2.95×10^{-2}	1.72×10^{-2}	2.95×10^{-2}
	o-Xylene	2760	2.65×10^{-2}	1.54×10^{-2}	2.65×10^{-2}
	p-Xylene	7533	7.22×10^{-2}	4.21×10^{-2}	7.22×10^{-2}
	Cyclohexane	7187	1.15×10^{-3}	6.70×10^{-3}	1.15×10^{-3}
	Nonane	773	3.71×10^{-2}	2.16×10^{-2}	3.71×10^{-2}
	Hazard Index		0.5269	0.3074	0.5269

Table A13. Cancer risk assessment C.I method (Xu, 2018 / Farris, 2014).

		Lifetime Cancer Risk					
	C (ng/m^3)	Children 0–2 Years	Children 2–16 Years	Women 16–80 Years		Men 16–80 Years	
				14 h/Day	24 h/day	14 h/Day	24 h/Day
BaP	0.059	1.09×10^{-7}	7.65×10^{-8}	2.68×10^{-8}	4.60×10^{-8}	2.90×10^{-8}	4.97×10^{-8}
BaA	0.053	9.83×10^{-9}	6.87×10^{-9}	2.41×10^{-9}	4.13×10^{-9}	2.60×10^{-9}	4.46×10^{-9}
BbF	0.289	5.36×10^{-8}	3.75×10^{-8}	1.31×10^{-8}	2.25×10^{-8}	1.42×10^{-8}	2.43×10^{-8}
BkF	0.079	1.47×10^{-8}	1.02×10^{-8}	3.59×10^{-9}	6.16×10^{-9}	3.88×10^{-9}	6.65×10^{-9}
CHRY	0.108	2.00×10^{-9}	1.40×10^{-9}	4.91×10^{-10}	8.42×10^{-10}	5.30×10^{-10}	9.09×10^{-10}
DBahA	0.02	3.90×10^{-8}	2.72×10^{-8}	9.57×10^{-9}	1.64×10^{-8}	1.03×10^{-8}	1.77×10^{-8}
I123cdP	0.135	2.50×10^{-8}	1.75×10^{-8}	6.14×10^{-9}	1.05×10^{-8}	6.63×10^{-9}	1.14×10^{-8}
Ni	4.38	1.90×10^{-6}	1.32×10^{-6}	4.65×10^{-7}	7.97×10^{-7}	5.02×10^{-7}	8.60×10^{-7}
Pb	8.03	1.60×10^{-7}	1.12×10^{-7}	3.93×10^{-8}	6.75×10^{-8}	4.25×10^{-8}	7.28×10^{-8}
Benzene	1390	6.61×10^{-5}	4.62×10^{-5}	1.62×10^{-5}	2.78×10^{-5}	1.75×10^{-5}	3.00×10^{-5}
Σ		6.84×10^{-5}	4.78×10^{-5}	1.68×10^{-5}	2.88×10^{-5}	1.81×10^{-5}	3.10×10^{-5}
Cases/10^6		68.44	47.80	16.78	28.77	18.11	31.04
Without Benzene		2.31	1.61	0.57	0.97	0.61	1.05
Without Benz, Ni, Pb		0.25	0.18	0.06	0.11	0.07	0.12

Table A14. Cancer risk assessment C.II method (Maertens, 2004/ Farris, 2014).

		Lifetime Cancer Risk:					
	C (ng/m^3)	Children 0–2 Years	Children 2–16 Years	Women 16–80 Years		Men 16–80 Years	
				14 h/Day	24 h/day	14 h/Day	24 h/Day
BaP	0.059	1.09×10^{-7}	7.65×10^{-8}	2.68×10^{-8}	4.60×10^{-8}	2.90×10^{-8}	4.97×10^{-8}
BaA	0.053	9.83×10^{-9}	6.87×10^{-9}	2.41×10^{-9}	4.13×10^{-9}	2.60×10^{-9}	4.46×10^{-9}
BbF	0.289	5.36×10^{-8}	3.75×10^{-8}	1.31×10^{-8}	2.25×10^{-8}	1.42×10^{-8}	2.43×10^{-8}
BkF	0.079	1.47×10^{-8}	1.02×10^{-8}	3.59×10^{-9}	6.16×10^{-9}	3.88×10^{-9}	6.65×10^{-9}
CHRY	0.108	2.00×10^{-9}	1.40×10^{-9}	4.91×10^{-10}	8.42×10^{-10}	5.30×10^{-10}	9.09×10^{-10}
DBahA	0.02	3.90×10^{-8}	2.72×10^{-8}	9.57×10^{-9}	1.64×10^{-8}	1.03×10^{-8}	1.77×10^{-8}
I123cdP	0.135	2.50×10^{-8}	1.75×10^{-8}	6.14×10^{-9}	1.05×10^{-8}	6.63×10^{-9}	1.14×10^{-8}
Ni	4.38	1.90×10^{-6}	1.32×10^{-6}	4.65×10^{-7}	7.97×10^{-7}	5.02×10^{-7}	8.60×10^{-7}
Pb	8.03	1.60×10^{-7}	1.12×10^{-7}	3.93×10^{-8}	6.75×10^{-8}	4.25×10^{-8}	7.28×10^{-8}
Benzene	1390	6.61×10^{-5}	4.62×10^{-5}	1.62×10^{-5}	2.78×10^{-5}	1.75×10^{-5}	3.00×10^{-5}
Σ		6.84×10^{-5}	4.78×10^{-5}	1.68×10^{-5}	2.88×10^{-5}	1.81×10^{-5}	3.10×10^{-5}
Cases/10^6		68.44	47.80	16.78	28.77	18.11	31.04
Without Benzene		2.31	1.61	0.57	0.97	0.61	1.05
Without Benz, Ni, Pb		0.25	0.18	0.06	0.11	0.07	0.12

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