sustainability

MDPI

Article

Modeling of Inhalation Health Risk of Volatile Organic Compounds in the Vicinity of Maptaphut Petroleum and Petrochemical Industrial Estate, Thailand

Wissawa Malakan [1], Sarawut Thepanondh [1,2,*] and Akira Kondo [3]

[1] Department of Sanitary Engineering, Faculty of Public Health, Mahidol University, Bangkok 10400, Thailand
[2] Center of Excellence on Environmental Health and Toxicology (EHT), Bangkok 10400, Thailand
[3] Graduate School of Engineering, Osaka University, Osaka 565-0871, Japan
* Correspondence: sarawut.the@mahidol.ac.th

Abstract: The purpose of this research was to explicate a human health risk assessment that can be employed with inhalation risk estimates to provide a screening level of risks. Model input parameters provide reasonable values with the site- and compound-specific values relied on by the Human Health Risk Assessment Protocol (HHRAP). This method uses a generic risk assessment, consisting of air dispersion and deposition modeling followed by risk modeling. An intensive evaluation was conducted in the surrounding area of the largest petroleum and petrochemical estate in Thailand, the Maptaphut industrial area, where a large volume of VOCs was emitted, with an increasing negative health impact on the local population. The potential inhalation health risk assessment showed that the lifetime cancer risk in all residential areas is higher than the health benchmarks. The highest cancer risk was 7.82×10^{-2} in children and 3.91×10^{-1} in adults. The inhalation effects are based on the specific emission rates, the united concentrations and deposition fluxes, and the emission phase. The results revealed that four VOCs (benzene, 1,3-butadiene, vinyl chloride, and 1,2-dichloroethane) should be given priority when controlling for sustainable health risk management through the comprehensive analysis of the integrated analysis of air dispersion and health risk mathematical models.

Keywords: AERMOD; emission inventory; HHRAP; IRAP-h view; Maptaphut; VOCs

check for updates

Citation: Malakan, W.; Thepanondh, S.; Kondo, A. Modeling of Inhalation Health Risk of Volatile Organic Compounds in the Vicinity of Maptaphut Petroleum and Petrochemical Industrial Estate, Thailand. *Sustainability* **2022**, *14*, 12073. https://doi.org/10.3390/su141912073

Academic Editors: Omar Hahad, Katie Frenis and Marin Kuntic

Received: 1 September 2022
Accepted: 19 September 2022
Published: 24 September 2022

Publisher's Note: MDPI stays neutral with regard to jurisdictional claims in published maps and institutional affiliations.

Copyright: © 2022 by the authors. Licensee MDPI, Basel, Switzerland. This article is an open access article distributed under the terms and conditions of the Creative Commons Attribution (CC BY) license (https://creativecommons.org/licenses/by/4.0/).

1. Introduction

Environmental and health perspectives are interrelated nowadays, especially concerning air pollution, resulting in various regulatory bodies regularly assessing the potential human health risks of toxic air pollutant emissions. In most previous studies, human health risk assessment is quantified in terms of emissions; air concentrations obtained from measurements or predictions through air dispersion modeling that either can be compared with air quality standards or be further analyzed for the potential inhalation exposure risk by complex equations and then compared with the health standards. Moreover, the estimated dose from the ingestion of food and water was attributed to atmospheric deposition as well as risks from all exposure pathways for a more comprehensive evaluation, as chemicals can transfer and accumulate in an environmental medium or dietary intake. The risk severity of a given chemical depends upon both physical and chemical properties; two of such properties are the PBT profiler (persistence, bioaccumulation, and toxicity of chemical substances) and individual exposure [1,2]. Thus, risk assessors are typically effort-intensive, with systematic assessments carried out on transportation, dispersion, deposition, uptake, and other systems both concerning land use and in the organism [3–5]. Unfortunately, air emissions risk analyses are often restricted to the inhalation route related to only ambient concentration, and do not include deposition, which could lead to misinterpretation, because of the complexity of

deposition processes and the lack of information acquisition [5]. Consequently, the number of publications on this subject in academic journals is quite limited.

The Maptaphut industrial area (MA) is the largest industrial estate in Thailand, located in Rayong province in the eastern region [6,7]. Several manufacturing industries are located here, such as the petrochemical industry, a coal-fired power plant, the metal industry, a natural gas power plant, a gas separation plant, and an oil refinery [8]. Even though the area was crowded, people still lived around it [9]. Therefore, this area represents a tremendous and renowned potential air pollution problem affecting the health of nearby inhabitants, especially due to volatile organic compounds (VOCs) [10,11]. These are considered the primary and most significant human exposure route, especially via the inhalation pathway [12]. Most of them have toxic and carcinogenic human health effects on the residents and workers exposed to them [13,14]. Epidemiological studies indicate that benzene, a key component in petrochemical processes [15], has the highest cancer risk and can also lead to the development of lymphocyte cell reduction, thrombocytopenia [16], and leukemia [17–19]. Moreover, 1,3-butadiene was related to an increased risk of non-Hodgkin lymphoma [20,21]. Hypothetically, if measures are adequate, the number of people sick and dying in the area due to pollutants should be small, a hypothesis which conflicts with the annual report of the health data center in Maptaphut [22]. Thus, ambient air quality standards to reduce air pollution levels and prevent the disease burden from air pollution might not be sufficient. A comprehensive risk assessment of human inhalation exposure to VOCs should be considered.

In this study, the emission of VOCs from industrial and vehicular sources was utilized as an input on the air dispersion model (AERMOD). The predicted concentrations were further used to assess the potential health impact using a comprehensive IRAP-h View risk assessment model. A human health risk assessment was evaluated for the total calculated cancer risk and hazard quotient for each chemical of potential concern (COPC) for a specific receptor and exposure scenario based on the latest USEPA-OSW Human Health Risk Assessment Protocol (HHRAP) [5,23–25].

2. Methods

2.1. Targeted VOC Emission Characteristics

The distinct VOC emissions in this area come from two primary sources, which consisted of (1) industrial emissions from a variety of sources, namely stack and flare emissions, storage tanks, wastewater, loading and unloading processes (ships and trucks), and fugitive emission as well as slurry, open equipment, and vessels (SOVs), and (2) non-industrial sources from on-road mobile emissions. In addition, this study considered the criteria of prospective priority VOCs applied in Munshed [26] and Thepanondh [27], as shown in Table 1.

The selection of the target VOCs was based on their level of carcinogenicity, toxicity, possibility of exposure, and standard value. Firstly, cancer classification was based on certain institutions' criteria, categorizing carcinogenicity in a scale ranging from 1(A)–2(B). For instance, those chemicals in the IRIS assessment classified as A and B carcinogens or "Carcinogenic to humans or likely to be carcinogenic to humans" are considered carcinogens. Benzene is designated as a regional cancer risk driver, and 1,3-butadiene is a national cancer risk contributor based on the list of the 2011 National Air Toxics Assessment (NATA) [24]. Secondly, we deliberated on the human toxicity portion of the PBT (persistent, bioaccumulative, and toxic) profiler but did not select persistent and bioaccumulative organic substances because the compounds in question did not tend to bioaccumulate through the food chain and were toxic when ingested. For instance, although benzene is not bioaccumulated and 1,3-butadiene is not persistent, both can reach toxic levels [28,29]. Thus, this study provided toxicity values to assess the endpoint of concern (e.g., cancer or non-cancer). The toxicity of chemical substances, and the relationship between exposure to potentially problematic chemicals and the likelihood and magnitude of developing an adverse health impact are estimated by this toxicity assessment. Toxicity values such as

reference concentration (RfC) for noncancer health effects, inhalation unit risk factor (URF) for carcinogenic potential, and acute inhalation exposure criteria (AIEC) are extracted from studies and experiments conducted on laboratory animals and human epidemiological studies. The toxicity values used to calculate the health risk in this study are given in Table 2. As shown in Figure S1, the toxicity values used in the calculations selected from references follow the U.S. EPA-approved hierarchy [23]. Because the model is designed for screening purposes, using surrogate data for VOCs was considered appropriate. Each VOC chemical is referred to as a COPC under HHRAP. Hierarchical approaches are required because AIECs are COPC-specific, and no single organization or method has developed a list of AIECs for all chemicals [26]. Thirdly, the targeted VOCs are used and potentially released into the atmosphere according to their consumption. Finally, the target VOCs were selected because their observed concentrations exceed the regulation ambient standards. By using the above criteria, the target VOCs were selected and listed in Table 2. This study considered the vapor phase of VOCs. Thus, vapor phase fraction (Fv = 1.0) is modeled for air concentrations as well as dry and wet vapor deposition rates.

Table 1. Concept of selection criteria of prospective priority VOCs based on carcinogenicity, toxicity, possibility of exposure, and standard value.

Carcinogenicity and Toxicity	Possibility of Exposure	Standard and Surveillance Values
- Available toxicity data in reliable institutes like IARC, IRIS(EPA), ACGIH, JSOH, JSOH, and WHO	- High cons. in ambient air - Tend to increase in ambient air if without policy - Demand-supply (source and released and production and imported)	- Both existing and exceeding criteria

Note: World Health Organization (WHO), Integrated Risk Information System (IRIS), International Agency for Research on Cancer (IARC), Environmental Protection Agency (EPA), American Conference of Governmental Industrial Hygienists (ACGIH), The Japan Society for Occupational Health (JSOH).

Table 2. Screening matrix of prospective priority VOCs based on toxicity values, carcinogen classification and standard value used in the risk assessment.

Target VOCs	Carcinogenicity and Toxicity Values			Carcinogen Classification				Std. of Annual Mean Cons.; μg/m^3	24-h Surveillance Cons.; μg/m^3
	RfC; mg/m^3	URF; 1/(μg/m^3)	AIEC; mg/m^3	IARC [1]	EPA [2]	ACGIH [3]	JSOH [4]		
Benzene	0.03	7.80×10^{-6}	1.3	1	A	A1	1	1.7	7.6
1,3-Butadiene	0.002	5.88×10^{-5}	0.66	2A	A	A2	1	0.33	5.3
1,2-Dichloroethane	2.4	2.60×10^{-5}	202	2B	B	A3	2A	0.4	48
Vinyl Chloride	0.1	8.80×10^{-6}	180	1	A	A1	1	10	20

Notes: [1] IARC: The International Agency for Research on Cancer: Toxicity levels are classified into five classes as follows: 1: The agent is carcinogenic to humans; 2A: The agent is probably carcinogenic to humans.; 2B: The agent is possibly carcinogenic to humans.; 3: The agent is not classifiable as to its carcinogenicity to humans, and 4: The agent is probably not carcinogenic to humans. [2] Evaluation under USNTP of USEPA: Toxicity levels are divided as follows: A: The agent is carcinogenic to humans with enough epidemiological evidence, and B: The agent is probably carcinogenic to humans but with limited epidemiological evidence. [3] Evaluation by ACGIH: Toxicity levels are classified into 5 classes as follows: A1: The agent is carcinogenic to humans; A2: Carcinogenesis to humans is suspected with limited epidemiological evidence or animal study; A3: Carcinogenesis is perceived with animal study; A4: The agent is not classifiable as to its carcinogenesis in humans; A5: The agent is not suspected of carcinogenesis to humans. [4] Evaluation by JSOH: Toxicity levels are classified into three classes as follows: 1: The agent is carcinogenic to humans; 2A: The agent is probably carcinogenic to humans with enough evidence, and 2B: The agent is possibly carcinogenic to humans without enough evidence.

2.2. HHRAP

The U.S. Environmental Protection Agency's Human Health Risk Assessment Protocol (HHRAP) for Hazardous Waste Combustion Facilities, which provides regularly used methodologies for multi-pathway exposure and screening-level risk analyses by site-specific data, was applied in this study [23]. This protocol was specially designed

to evaluate risks from hazardous waste combustors; however, the fate and transport algorithms comprised in the functions are involved with any pollutant or emission source once source-specific air dispersion and deposition modeling have been conducted [5]. The HHRAP methodology provides possible parameter values and algorithms relying on the best available science. One of these involves combining high-end and mean parameter values. High-end recommendations use the highest predicted modeled air parameters (air concentrations and deposition rates) at selected exposure scenario locations and assume high-end exposure frequencies and durations [23]. Results from a hypothetical worst-case scenario are displayed at the point of maximum impact as a result of the facility emissions, as well as designated health benchmarks. As mentioned above, each COPC chemical is apportioned to the particle, particle-bound, and vapor phases, which is determined based on the fraction of the substance in the vapor phase (Fv) according to the HHRAP companion database. Although the full US EPA database of COPCs, comprising more than a hundred types, is included in the IRAP-h view, 1,3-butadiene is not in the database. Thus, it is treated specially in the IRAP-h View. Information on its properties for input data modeling was taken from the available information on either the Risk Assessment Screening Spreadsheet (RASS) used in the Minnesota Air Emissions Risk Analysis (AERA) process [30] or Munshed [26], for surrogate data. Thus, we methodically selected model input parameters over the range of appropriate values.

The overall plan for this research involves the following steps: selected VOCs were included in the analysis in accordance with chosen criteria, and then a consistent emission inventory (EI) was developed for industrial and non-industrial sources. Two modeling steps were carried out in the risk assessment process. The Atmospheric dispersion modeling was the first step, and the second step was risk modeling. Concentration and deposition values were conducted by air dispersion modeling using the AERMOD model via the fate and transport calculations of VOCs in media (only air). Then we conducted an inhalation analysis for each modeled source and VOC using HHRAP as implemented in the IRAP-h View, where parameter values were provided appropriately to eliminate bias for exposure scenarios.

However, uncertainty exists in the process even when using the most accurate data and complex models [23]. Finally, we described the uncertainties and limitations of the work, as shown in Figure S2.

2.3. Source Emission Inventory

VOC emissions from industrial sources were obtained from the emission database of the Thai Ministry of Natural Resources and Environment. Meanwhile, the non-industrial sector (on-road transportation) is based on emission factors developed by Thepanondh [31].

The emission factors used in this study were developed based on the average speed and standard Thailand driving cycle using the International Vehicle Emissions (IVE) Model. The emission rates were then calculated as aggregated emission rates using Equation (1) [32].

$$ER_{(i)} = \Sigma(j)\Sigma N_{(j)} \times EF_{(i,j)} \times L \quad (1)$$

where $ER_{(i)}$ is the emissions rate for i pollutant (g/s); $N_{(j)}$ is the number of vehicles of a particular type 'j' (car/day); $EF_{(i,j)}$ is emission factor (g/km) for pollutant 'i' in the vehicle type 'j'; j is a type of vehicle (truck, motorcycle, passenger car, van and pick up and bus); and L is road length (m).

2.4. Air Dispersion and Deposition Modeling

2.4.1. Model Configuration

AERMOD View (version 9.9.0, Lakes Environmental Software, Waterloo, Ontario, Canada) was used as the main tool used in this study to predict ground-level concentrations and spatial distribution of VOCs. The model was configured to cover an area of 12.5 × 12.5 km^2, No. of points: x = 50 m; y = 50 m, and Grid spacing: x = 500 m; y = 500 m. The regulatory modeling options used the default mode of operation for the

urban options of the dispersion coefficient. An analysis by the air dispersion model was performed for the gas phase for each case. The VOCs were calculated hourly, also taking into account the annual average concentration and the deposition rate (wet and dry) on the elevated-terrain-height option. Meteorological data files are employed from on-site meteorological data for 2019. In total, there were 22 stacks, 13 flares, 16 tanks, 9 loading/unloading processes, 34 fugitive areas, 15 wastewater treatment plants, 15 SOV units, and 11 roads used as emission inputs. The spatial distribution of emission sources is illustrated in Figure 1. Discrete cartesian receptors were assigned for every 38 villages and every 16 air monitoring stations located in the study domain, as shown in Figure 1.

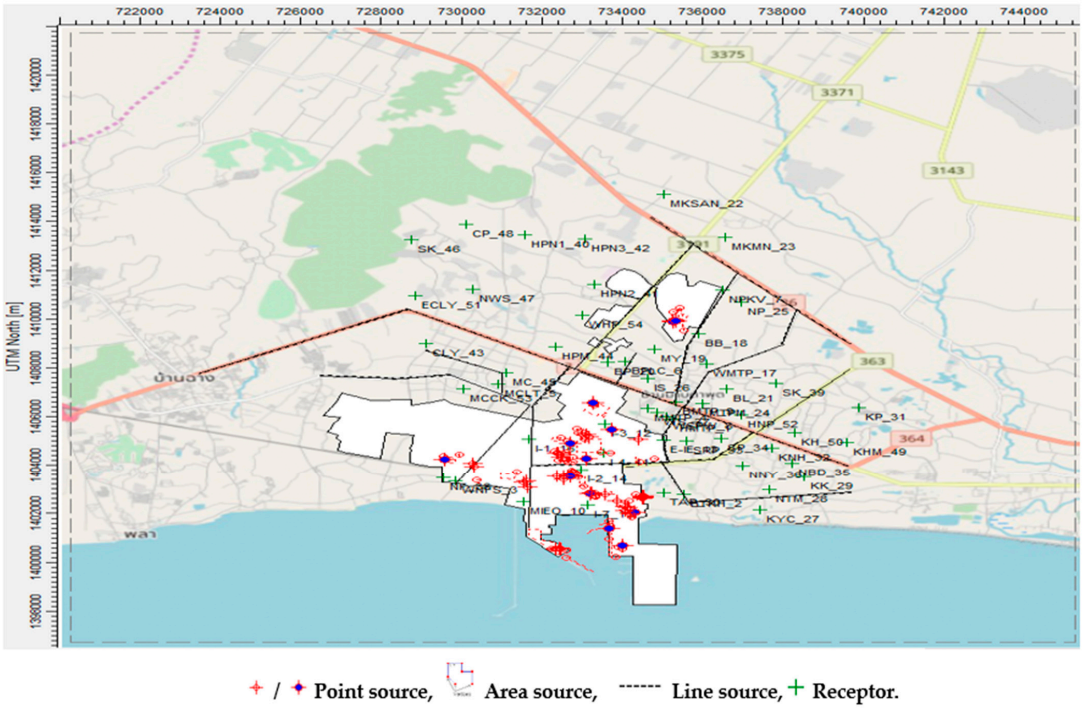


Figure 1. Study domain and location of all sources and all receptors were displayed in AERMOD.

2.4.2. Risk Mode Output

AERMOD was run by unitized emission rates for each source to interpret ambient air concentrations and deposition rates. Annual average values were modeled to evaluate the potential adverse health impacts from long-term exposure (cancer risk). In subsequent analyses, these modeled results were carried forward to calculate the inhalation risk for each substance at each receptor in the risk modeling.

2.5. Risk Modeling

The IRAP-h View (latest version 5.1.0, Lakes Environmental Software, Waterloo, Ontario, Canada) is pervasively applied by international environmental and engineering consultants. This risk model is implemented according to the HHRAP methodology. It is a graphics program capable of simultaneously and rapidly calculating risk values without a traditional process for various chemicals from several sources at different locations. The program can import concentration and deposition values from plot files generated by AERMOD. These plot files contained the necessary air parameter values for the fate and transport algorithms required for the risk assessment. It also allowed us to see the pattern and spatial distribution for each phase at each risk receptor of each substance of each source in the atmosphere. Subsequently, the program provided functions to determine risk receptors, exposure scenarios, and specific land-use areas at the exposure scenario locations [5].

2.5.1. COPC Database

The IRAP-h View program comprises a database with default values, called the COPC database, for the chemical and physical properties and toxicity values for the included chemical substances, though the database does not include 1,3-butadiene. In addition, the inhalation pathway of vapor was designated. Thus, VOC-specific parameters such as diffusivity in air, diffusivity in water, leaf cuticular resistance, and Henry's Law Constant are needed to model the vapor phase air concentrations, dry vapor deposition, and wet vapor depositions of chemicals in each COPC. However, this study used the default chemical and physical properties for the targeted VOCs, except for Henry's Law Constant, the unit of which was changed from atm-m^3/mol to Pa-m^3/mol. Regarding toxicity data, this study used toxicity values selected from prioritized sources as described previously. The characteristics of four of the substances selected as the target VOCs in this study are provided in Table S1.

2.5.2. Exposure Scenarios

Exposure pathways were evaluated in this framework as recommended by the HHRAP [23]. Regarding quantifying exposure, the most significant effect of most substances released on human health will arise from vapor inhalation. In addition, the targeted VOCs were not included in either of the PBT profilers of the EPA and the European Union [33], substances for which inhalation risks are expected to be higher than ingestion risks. Therefore, inhalation exposure scenarios of vapor phase air concentrations without a particle phase were designed to assess the cancer risk for child and adult residents.

In addition, the media concentration in ambient air (C_i) is automatically estimated by a model involving the COPC-specific emission rate, a fraction of the substance, the unitized yearly air concentration, and the hourly air concentration from the vapor phase for chronic and acute exposures, respectively. Then the potential for human exposure was evaluated by aggregating the COPC concentrations in ambient air with human receptor-specific exposure parameter values via exposure concentration (EC), which was calculated using Equation (2); this relies on the exposure frequency, the exposure duration, and the averaging time for quantifying exposure based on HHRAP recommendations [23]. The exposure frequency is 8400 h per year (350 days). This assumption is in accordance with the protective (or conservative) estimate that all receptors spend 2 weeks away from the exposure scenario location. The exposure duration is determined by the hypothetical age of the resident children and adults, 6 and 30 years, respectively. Finally, the averaging time depends on the type of toxic effect being assessed. The averaging time (AT) for noncarcinogenic pollutants is the exposure duration in years multiplied by 365 days; however, for carcinogenic pollutants, the effect may have long latency periods. The recommended averaging time is 70 years.

$$EC_i = \frac{C_i \times EF \times ED}{AT \times 365 \text{ days/year}} \quad (2)$$

Moreover, this calculation of the inhalation risk is not similar to those used in other sources due to the absence of inhalation rate and body weight. The parameters and variables used in the health risk assessment model are shown in Table S2.

The last step of a risk assessment is risk characterization. This involved combining the equation of exposure quantities and the toxicity benchmarks to compute the cancer risks and noncancer hazards for each receptor in the inhalation pathway. These risks and hazards were automatically calculated by the IRAP-h view.

2.6. Risk Characterization of Inhalation

2.6.1. Quantitative Estimation of Chronic Cancer Risk

Cancer risk is the probability that a human receptor will develop cancer based on a unique set of exposure, model, and toxicity assumptions [23]. The equation for calculating inhalation risk is presented in Equation (3). Moreover, it is plausible for receptors to be exposed to multiple VOCs within an individual exposure pathway. Thus, the total risk

associated with exposure to all VOCs through direct inhalation exposure was calculated by Equation (4).

$$\text{Cancer Risk}_{\text{Inhalation(i)}} = \text{EC}_i \times \text{URF}_i \quad (3)$$

$$\text{Cancer Risk}_T = \Sigma_i \text{ Cancer Risk}_{\text{Inhalation(i)}} \quad (4)$$

where Cancer $\text{Risk}_{\text{Inhalation(i)}}$ represents the individual lifetime cancer risk through direct inhalation of carcinogen compound i (unitless); Cancer Risk_T is the total cancer risk for all VOCs of inhalation exposure (unitless); EC_i is the exposure concentration of pollutant i; ($\mu g/m^3$); and URF_i is the pollutant-specific inhalation unit risk factor $(\mu g\ m^{-3})^{-1}$.

2.6.2. Cancer Numeric Target Levels

Regarding the health benchmark, the risk threshold values set in the IRAP-h View were based on the U.S. EPA Region 6 Addendum summarized in Table S3 [34]. For the thresholds of cancer risk, if the cancer risk is more remarkable than the designated thresholds, then the probability of an individual developing cancer is possible. Conversely, suppose the cancer risk is calculated to be less than the thresholds. In that case, it is an acceptable risk (or negligible risk), as the probability of an individual developing cancer from exposure to VOC concentration is low. For instance, a risk of 1×10^{-5} means that an individual has up to a one-in-one-hundred-thousand chance of developing cancer during their lifetime from the exposure being evaluated [23].

Moreover, this model can be used to optimize the risks using the risk receptor identification tool to identify sensitive receptors because risk assessments are frequently conducted on the receptor where the unitized air toxic concentrations and deposition fluxes are maximized to define the points of maximum exposure as a hypothetical worst-case scenario as shown in Table S4. These locations are generally located close to the emission sources (factories and roads).

3. Results and Discussion

3.1. Emission Inventory Results

The anthropogenic VOC emission inventory in this study is divided into two categories: industrial and non-industrial sources (Table 3). The total emissions of ethylene dichloride (EDC) and vinyl chloride (VCM) emissions, which are solely emitted from industrial sources, are 7.72 and 37.78 tons/year, respectively. The major sources of their emissions were storage tanks and SOVs. The vehicular (mobile source) causes about 73.78 and 9.96 tons/year of benzene and 1,3-butadiene emissions, respectively. Passenger cars and motorcycles are the largest contributors to these emissions [35].

The emission of EDC and VCM in this study area originated from only industrial sources (manufacturing of the polyvinylchloride plastic) [36,37]. On the other hand, mobile sources are the dominant emission source of benzene and 1,3-butadiene (about 94 and 76% of total emission), respectively.

Table 3. Anthropogenic VOC emission inventory carried out for different levels across study area.

Level I	Level II	EI (Tons/Year)			
		BZ	BD	EDC	VCM
Industrial sources	Stack	0.53	0.74	0.06	0.06
	Flare	0.18	0.02	0	0
	Storage tank	0.06	0	7.11	0
	Loading and unloading	1.85	0.05	0.00	0
	Fugitive	1.40	0.58	0.36	0.44
	Wastewater treatment	1.09	1.79	0.19	0.32
	Slurry, Open equipment, Vessel (SOV)	0	0	0	36.96
	Total	**5.11**	**3.19**	**7.72**	**37.78**

Table 3. *Cont.*

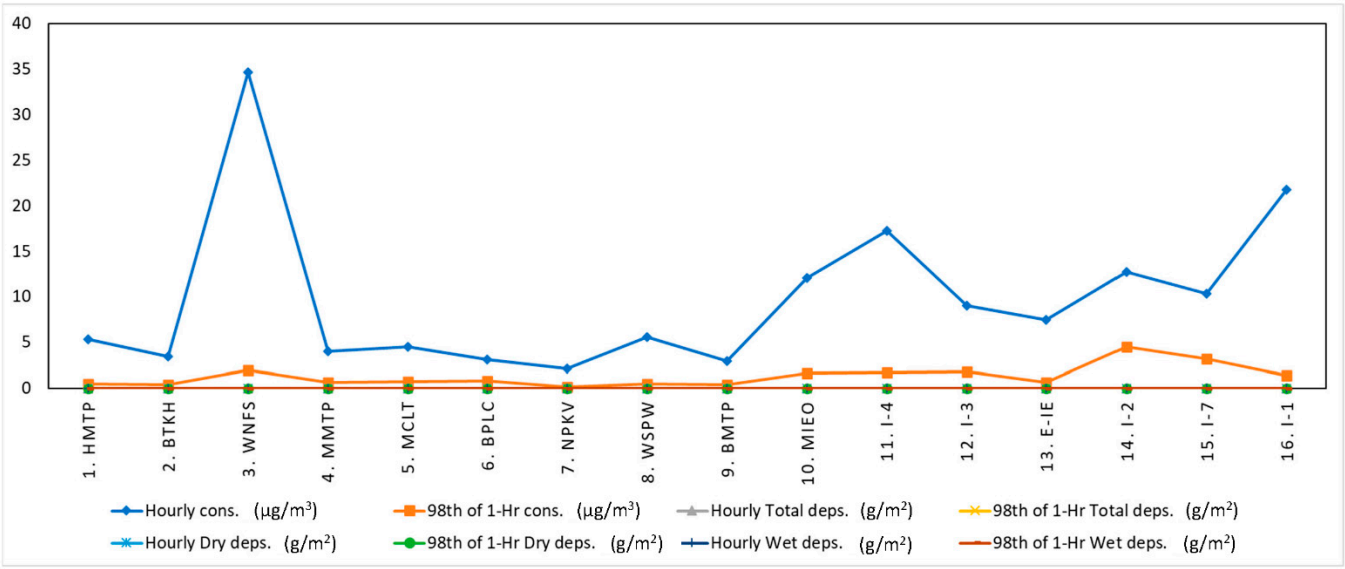
Level I	Level II	EI (Tons/Year)			
		BZ	**BD**	**EDC**	**VCM**
Non-Industrial sources	Motorcycle	5.84	4.56	0	0
	Passenger car	61.60	3.61	0	0
	Van and Pick up	1.02	0.44	0	0
	Truck	2.87	0.96	0	0
	Bus	2.46	0.40	0	0
	Total	**73.78**	**9.96**	**0**	**0**
Total	**12**	**78.89**	**13.15**	**7.72**	**37.78**

Note: BZ; Benzene, BD; 1,3-Butadiene, EDC; Ethylene dichloride or 1,2-Dichloroethane, and VCM; Vinyl chloride monomer.

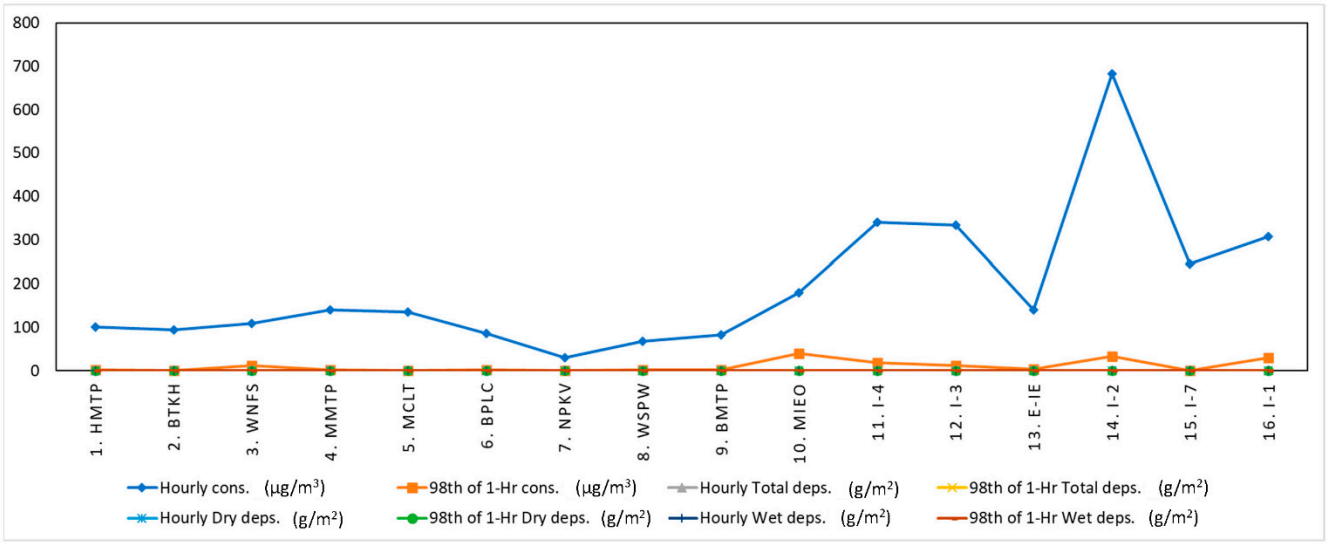
3.2. Air Modeling Results

The air dispersion modeling predicted concentration and deposition values for hourly, daily, and annual values and the 98th percentile of the hourly and daily mean for the vapor phase of all targeted VOCs at each receptor. The modeled results indicated that only annual 1,3-butadiene concentrations exceeded the Thai ambient air quality standard (0.33 $\mu g/m^3$). However, monitoring data from the intensive surveillance of VOCs in ambient air for 24-h average showed that there were some monitoring stations where benzene, 1,3-butadiene, and vinyl chloride exceeded the 24-h ambient guideline values. The peak concentrations of chemicals might be caused by the upset/abnormal operation of the industrial source, which is the limitation of using a constant emission rate in the air dispersion model. The analysis of the source contributions to the ambient concentration of benzene, 1,3-butadiene, and vinyl chloride was further intensively evaluated. Benzene concentrations at the receptor were mostly contributed by fugitive sources and wastewater treatment plant emissions (Figure S3). Although the maximum 1-h average concentration has no typical ambient standard value, it can be compared with inhalation health benchmarks for further risk assessment. This study gives two examples of benzene and vinyl chloride monomers in the average hourly maximum concentration and its 98th percentile, as well as deposition at the air monitoring stations, as shown in Figure 2. Two additional substances can be found in Figure S4. Figure 2 illustrates the significant difference between the maximum and the 98th percentile predicted concentrations. However, the 98th percentile concentrations were further used for the health risk analysis.

As shown in Figure S5, annual deposition results found that although the dry deposition was minimal (maximum annual dry deps. = 4.2×10^{-4} g/m^2), it was predominant in the total deposition. Indeed, the deposition is zero, and then the total risk equals the inhalation risk. In fact, the wet deposition was decreased at a greater distance away from the source because wet scavenging can emerge before the plume reaches ground level and very near the source; before full plume touchdown, the wet deposition value is greater than the dry deposition value [5]. Furthermore, wet deposition is the washout of both the vapour phase and the particulate bound chemicals during precipitation [38,39]. To achieve maximum benefit, this study conducted individual source analyses for each contour plot file. The modeled results attributed to the vapor phase for the air concentration and dry deposition values that were plotted followed analogous patterns, increasing with distance to a maximum and then decreasing, especially in the cases of stack, flare, and tank emissions. The elevated sources have more influence than the source with a low release height, especially area sources, because of the low concentration and deposition values near the source since the plume centerline does not yet wholly reach the ground level for all targeted VOCs [5].



(**a**) benzene



(**b**) vinyl chloride

Figure 2. Hourly and 98th percentile of air concentration, total deposition, dry deposition, and wet deposition for (**a**) benzene; (**b**) vinyl chloride monomer at each air station.

3.3. Health Risk Modeling Results

The results are inferred from the unitized concentrations (µg-s/g-m^3) acquired from the AERMOD dispersion modeling results, with an assumption that the vapor phase is the most critical factor affecting inhalation risk. Thus, we further infer that the vapor phase is the most crucial factor affecting the concentration. Moreover, its phase does not require particle size distribution and, therefore, rarely affects deposition.

As described earlier, the risk model required the unitized air toxic concentrations and deposition fluxes for the vapor phase in air parameters to produce risk results. To confirm the results from the sensitivity analysis, those values were added to the risk model. Results indicated that an overall inhalation risk attributed to deposition fluxes hardly changed, but that deposition can significantly affect the risk of ingestion. According to statistical relationships, the inhalation risk is a linear function of concentration, and ingestion risk is an albeit nonlinear function of deposition [5], although the risk mode is forced to read the entire met data file, for both dry and wet depositions. Therefore, whether or not

deposition is included in the risk calculation does not change the inhalation risk. Other possible reasons for the slight variation in the inhalation risk attributed to deposition are that the surface met data provide less reliable estimates for predicted deposition because it contained inappropriate deposition data, such as the precipitation amount (zero) and the designated phase partitioning for each substance to compute deposition in AERMOD. Therefore, in this case, this reliability is also based on surface met data.

Even though the data are incomplete with concern to the wet deposition results in AERMET, the COPC database in the IRAP-h view contained chemical-specific parameters that caused model vapor phase dispersion and wet and dry deposition with unitized emissions values. In any given receptor location, the result of dry deposition was always higher than wet deposition. To put it simply, effects on wet deposition were smaller than on dry deposition; however, both had a relatively small or almost no impact on breathing in this study. The results coincided with Pratt and Dymond's [5] findings that the vapor-phase results predicted the smallest deposition values compared to the particle and particle-bound phases. In addition to all targeted VOCs, the highest concentration occurred at the representative receptor of air stations near Map Ta Phut Industrial Estate. Hence, the risk assessment was carried out based on the village location but not the location of the monitoring station, to reduce the bias mentioned above. The resulting inhalation risks for child and adult residents were automatically calculated based on the concentration and deposition in potential exposure routes of the specific pollutants and sources, and these results were then exported from the risk model for each community alongside the data on sensitive receptors in terms of the lifetime cancer risk.

Assessment of the Carcinogenic Risk

The lifetime cancer risks are calculated for all targeted VOCs by IRAP and sorted in ascending order for each receptor. An intensive analysis was carried out for its impact on the children and adults who resided in the communities separately (Table S5). Results indicated that at every receptor, the carcinogenic risks exceeded their carcinogenic risk benchmark (1×10^{-5}) for both children and adult population. These results were consistent with the previous study by Pinthong et al. [40]. The highest cancer risk of 7.82×10^{-2} in children and 3.91×10^{-1} in adults was at Mab Ya receptor. It should be noted that the risk values of adults were higher than children. Total cancer risk was decreased at the father distance from the industrial complex. 1,3-butadiene was evaluated as the key compound threated to the carcinogenic risk, while the carcinogenic risk of ethylene dichloride was lowest among the targeted chemicals. Therefore, 1,3-butadiene should be given the first priority and foremost to reduce the risk of cancer in this industrial area. An example of this analysis at Mab Ya receptor is also displayed in Figure 3.

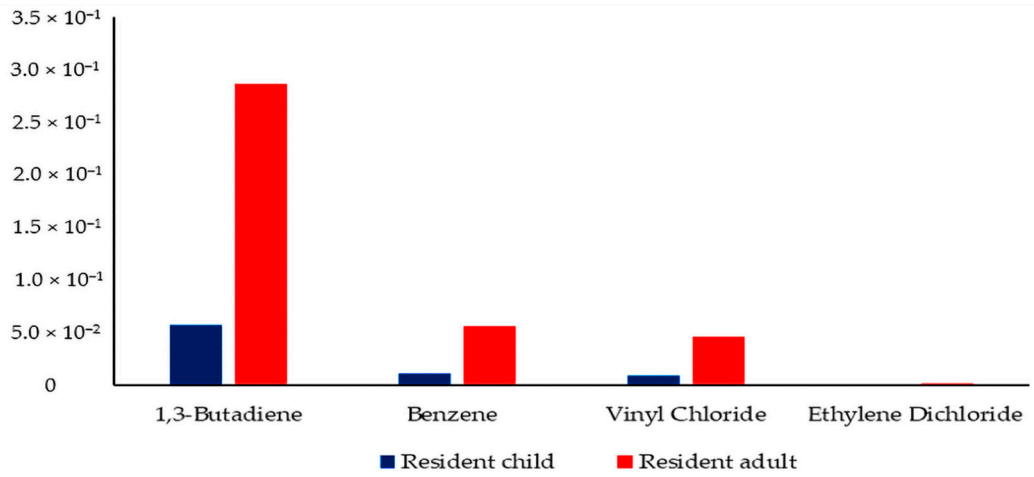


Figure 3. Total cancer risk of resident for individual VOC at Mab Ya receptor.

The source contribution to the ambient concentration analysis at Mab Ya indicated that fugitive sources were responsible for about 98% of 1,3-butadiene emissions when considering the percentage contribution of an individual pollutant (Figure 4). This analysis clearly denoted the necessity of controlling VOC emissions from fugitive sources as the first priority of pollution and health management in this study area.

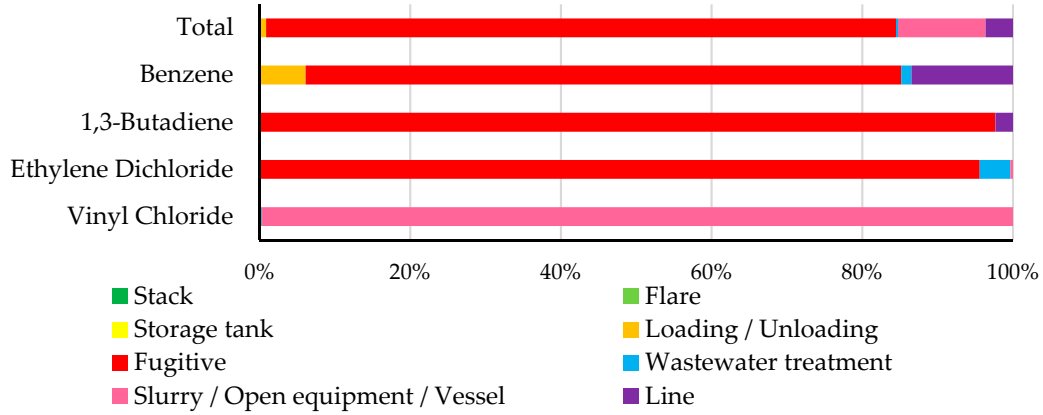


Figure 4. Percentage of source contribution of cancer risk in child and adult at the highest risk.

4. Uncertainties and Limitations

The calculated risk value exceeds the health standard, but this does not mean the proposed operation is unsafe or unacceptable. Instead, calculating risk beyond the norm raises further considerations both surrounding scientific bases and advances, and the uncertainty associated with calculating risk [26]. We contend that these artificially elevated risks should be neglected because of the dominance of combining each pollutant in each source. The results are quite different when compared to ambient standards for specific pollutants. Hence, risk results should consider whether it exceeds the acceptable standard of health. Moreover, there may be other uncertainties resulting from constant emissions and the meteorological data files in the model and the accuracy of emission inventories.

5. Conclusions

Current practices do not go beyond assessing the critical air concentrations of VOC source air toxins. This present study takes a giant leap forward and estimates the cumulative human health risk posed by the aggregated exposures of VOC sources using human health risk assessment software. This study is a reasonable guideline for screening tools that assess inhalation risk. Emissions from both industrial and vehicular sources were intensively analysed to model the ambient ground-level concentrations of each VOC species. The targeted VOCs were systematically selected and consisted of benzene, 1,3-butadiene, 1,2-dichloroethane, and vinyl chloride. The model output plot files consisted of unitized concentration and deposition values that were interpolated from AERMOD via fate and transport algorithms and were further used as risk modeling inputs. The cancer risks were evaluated using the IRAP-h view following specific parameters set by the HHRAP to determine the potential adverse health risks of exposure to the targeted VOCs. The potential inhalation risks showed that the cancer risk for all targeted VOCs was higher than the acceptable standard (1×10^{-5}) in all residential areas. The highest cancer risk was 7.82×10^{-2} in children and 3.91×10^{-1} in adults. Therefore, government agencies should consider human health risk assessments as a regulatory scheme for health benchmarks rather than simply comparing with ambient concentration standards. More stringent emission regulations and control engineering measures as well as the emission management of 1,3-butadiene, benzene, vinyl chloride, and 1,2-dichloroethane, should be systematically implemented for the sustainable management of the industrial sector.

Supplementary Materials: The following supporting information can be downloaded at: https://www.mdpi.com/article/10.3390/su141912073/s1, Figure S1: Hierarchical approaches for (a) General toxicity values; (b) Acute toxicity values for all VOCs, Figure S2: VOC-Human Health Risk Assessment Process, Figure S3: Sources contribution of annual benzene concentrations at air monitoring stations, Figure S4: Hourly and 98th percentile of air concentration, total deposition, dry deposition, and wet deposition for (c) 1,3-butadiene; (d) 1,2-dichloroethane in each air station, Figure S5: Spatial distribution of predicted annual deposition of (a) benzene; (b) 1,3-butadiene; (c) 1,2-dichloroethane; (d) vinyl chloride monomer; Table S1: Chemical-specific parameters required for calculating vapor phase dispersion and deposition, Table S2: Exposure parameters for resident adults and children used in the health risk assessment software, Table S3: Cancer and noncancer numeric target levels used in IRAP-h view, Table S4: Location and reference center distance of sensitive receptors identified in the study domain, Table S5: Total lifetime cancer risk contributed from all VOCs for resident children and adults at all receptors.

Author Contributions: Conceptualization, project administration, resources, acquisition, validation, writing-review and editing, S.T.; software, investigation, formal analysis, visualization, literature review, and writing-original draft preparation, W.M.; validation, A.K. All authors have read and agreed to the published version of the manuscript.

Funding: This research project is supported by National Research Council of Thailand (NRCT) [No. NRCT5-RGJ63012-136].

Institutional Review Board Statement: The aforementioned project has been reviewed and approved according to the Standard Operating Procedures of the Ethical Review Committee for Human Research, Faculty of Public Health, Mahidol University. This protocol complies with a "Research with Exemption" category (No. 6/2565).

Informed Consent Statement: Not applicable.

Data Availability Statement: The data presented in this study are available in supplementary material.

Acknowledgments: The authors greatly appreciate the support with monitoring data from the Pollution Control Department (PCD) and the Industrial Estate Authority of Thailand (IEAT), the community boundaries from Maptaphut Municipality. This study was partially supported for publication by the China Medical Board (CMB), Faculty of Public Health, Mahidol University, Bangkok, Thailand.

Conflicts of Interest: The authors declare no conflict of interest.

Abbreviations

$\mu g/m^3$	Microgram per cubic meter
AERMOD	American Meteorological Society/Environmental Protection Agency Regulatory Model
AIEC	Acute Inhalation Exposure Criteria
COPC	Chemical of Potential Concern
EI	Emission Inventory
Fv	Fraction of Volatilization
HHRAP	Health Risk Assessment Protocol
IARC	International Agency for Research on Cancer
IRIS	Integrated Risk Information System
IRAP-h	Industrial Risk Assessment Program-Human Health
PBT	Persistence, Bioaccumulation, Toxicity
URF	Inhalation Unit Risk Factor
VOC	Volatile Organic Compound
SOV	Slurry, Open Equipment, Vessel

References

1. United States Environmental Protection Agency. PBT Profiler. Office of Pollution Prevention and Toxics. 2006. Available online: http://www.pbtprofiler.net/ (accessed on 12 January 2021).
2. Vallero, D.A. Chapter 35-Waste management accountability: Risk, reliability, and resilience. In *Waste*; Academic Press: Cambridge, MA, USA, 2019; pp. 693–740. [CrossRef]

3. National Research Council. Environmental transport and exposure pathways of substances emitted from incineration facilities. In *Waste Incineration & Public Health*; National Academies Press: Washington, DC, USA, 2000. Available online: https://www.ncbi.nlm.nih.gov/books/NBK233615/ (accessed on 15 February 2021).
4. Sijm, D.T.; Rikken, M.; Rorije, E.; Traas, T.; Mclachlan, M.; Peijnenburg, W. Transport, accumulation and transformation processes. In *Risk Assessment of Chemicals*; Springer: Dordrecht, The Netherlands, 2007; pp. 73–158. [CrossRef]
5. Pratt, G.C.; Dymond, M. Multipathway screening factors for assessing risks from ingestion exposures to air pollutants. *J. Air Waste Manag. Assoc.* **2009**, *59*, 419–429. [CrossRef] [PubMed]
6. Chusai, C.; Manomaiphiboon, K.; Saiyasitpanich, P.; Thepanondh, S. NO_2 and SO_2 dispersion modeling and relative roles of emission sources over Map Ta Phut industrial area, Thailand. *J. Air Waste Manag. Assoc.* **2012**, *62*, 932–945. [CrossRef] [PubMed]
7. Boonpeng, C.; Polyiam, W.; Sriviboon, C.; Sangiamdee, D.; Watthana, S.; Nimis, P.L.; Boonpragob, K. Airborne trace elements near a petrochemical industrial complex in Thailand assessed by the lichen Parmotrema tinctorum (Despr. ex Nyl.) Hale. *Environ. Sci. Pollut. Res.* **2017**, *24*, 12393–12404. [CrossRef] [PubMed]
8. Office of Natural Resources and Environmental Policy and Planning. *Emission Sources Data in Map ta Phut Area for Air Modeling*; ONEP: Bangkok, Thailand, 2016. Available online: https://www.onep.go.th/ (accessed on 5 January 2021).
9. Soytong, P.; Perera, R. Spatial analysis of the environmental conflict between state, society and industry at the Map Ta Phut-Rayong conurbation in Thailand. *Environ. Dev. Sustain.* **2017**, *19*, 839. [CrossRef]
10. Asa, P.; Jinsart, W. Effects of Air Pollution Related Respiratory Symptoms in Schoolchildren in Industrial Areas Rayong, Thailand. *Environmentasia* **2016**, *9*, 116–123. [CrossRef]
11. Soytong, P.; Perera, R. Use of GIS tools for environmental conflict resolution at Map Ta Phut Industrial Zone in Thailand. *Sustainability* **2014**, *6*, 2435–2458. [CrossRef]
12. Mirrezaei, M.A.; Orkomi, A.A. Gas flares contribution in total health risk assessment of BTEX in Asalouyeh, Iran. *Process. Saf. Environ. Prot.* **2020**, *137*, 223–237. [CrossRef]
13. Mihajlović, V.; Grba, N.; Suđi, J.; Eichert, D.; Krajinović, S.; Gavrilov, M.B.; Marković, S.B. Assessment of Occupational Exposure to BTEX in a Petrochemical Plant via Urinary Biomarkers. *Sustainability* **2021**, *13*, 7178. [CrossRef]
14. Tsai, J.H.; Gu, W.T.; Chung, I.I.; Chiang, H.L. Airborne air toxics characteristics and inhalation health risk assessment of a metropolitan industrial complex. *Aerosol Air Qual. Res.* **2019**, *19*, 247–2489. [CrossRef]
15. Chen, M.J.; Lin, C.H.; Lai, C.H.; Cheng, L.H.; Yang, Y.H.; Huang, L.J.; Yeh, S.H.; Hsu, H.T. Excess lifetime cancer risk assessment of volatile organic compounds emitted from a petrochemical industrial complex. *Aerosol Air Qual. Res.* **2016**, *16*, 1954–1966. [CrossRef]
16. Jung, J.H.; Choi, B.W.; Kim, M.H.; Baek, S.O.; Lee, G.W.; Shon, B.H. The characteristics of the appearance and health risks of volatile organic compounds in industrial (Pohang, Ulsan) and non-industrial (Gyeongju) areas. *Environ. Toxicol. Chem.* **2012**, *27*, e2012012. [CrossRef] [PubMed]
17. Hsu, C.Y.; Chiang, H.C.; Shie, R.H.; Ku, C.H.; Lin, T.Y.; Chen, M.J.; Chen, N.T.; Chen, Y.C. Ambient VOCs in residential areas near a large-scale petrochemical complex: Spatiotemporal variation, source apportionment and health risk. *Environ. Pollut.* **2018**, *240*, 95–104. [CrossRef] [PubMed]
18. Snyder, R. Leukemia and benzene. *Int. J. Environ. Res. Public Health* **2012**, *9*, 2875–2893. [CrossRef] [PubMed]
19. Kirkeleit, J.; Riise, T.; Bråtveit, M.; Moen, B.E. Increased risk of acute myelogenous leukemia and multiple myeloma in a historical cohort of upstream petroleum workers exposed to crude oil. *Cancer Causes Control* **2008**, *19*, 13–23. [CrossRef]
20. International Agency for Research on Cancer. *Chemical Agents and Related Occupations. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans*; IARC: Lyon, France, 2012; Volume 100, pp. 225–248. Available online: https://www.ncbi.nlm.nih.gov/books/NBK304416/ (accessed on 5 March 2021).
21. Sathiakumar, N.; Delzell, E.; Hovinga, M.; Macaluso, M.; Julian, J.A.; Larson, R.; Cole, P.; Muir, D. Mortality from cancer and other causes of death among synthetic rubber workers. *Occup Environ. Med.* **1998**, *55*, 230–235. [CrossRef]
22. Ministry of Public Health. Health Data Center (HDC). 2019. Available online: https://hdcservice.moph.go.th/hdc/main/index.php (accessed on 7 September 2021).
23. United States Environmental Protection Agency. *Human Health Risk Assessment Protocol (HHRAP) for Hazardous Waste Combustion Facilities (Final), EPA530-R-05-006*; OSWER: Washington, DC, USA, 2005. Available online: https://archive.epa.gov/epawaste/hazard/tsd/td/web/html/risk.html#hhrad (accessed on 22 December 2020).
24. United States Environmental Protection Agency. 2011 NATA: Assessment Results. 2011. Available online: https://www.epa.gov/national-air-toxics-assessment/2011-nata-assessment-results (accessed on 18 December 2021).
25. Minnesota Pollution Control Agency. Minnesota Air Toxics Emission Inventory. 2008. Available online: http://www.pca.state.mn.us/air/toxics/toxicsinventory.html (accessed on 14 April 2021).
26. Munshed, M. Mobile Toxics Human Health Risk Assessment Framework. Master's Thesis, University of Waterloo, Waterloo, ON, Canada, 2018.
27. Thepanondh, S. Establishment of VOCs Emission Inventory in Thailand: A Report on Methods and Early Results. In *Seminars on Development of Environment and Emission Standard VOC*; PCD: Bangkok, Thailand, 2006. Available online: http://infofile.pcd.go.th/air/VOC_sarawut1.pdf (accessed on 19 November 2020).

28. Gordian, M.E.; Frazier, R.; Hill, A.; Schreiner, I.; Siver, D.; Stewart, A.; Morris, S. Health Effects of Indoor-Air Benzene in Anchorage Residences: A Study of Indoor-Air Quality in Houses with Attached Garages. In *Institute of Social and Economic Research*; UAA: Anchorage, AK, USA, 2009. Available online: http://hdl.handle.net/11122/4330 (accessed on 10 March 2021).
29. Hughes, K.; Meek, M.E.; Walker, M.; Beauchamp, R. 1,3-Butadiene: Human health aspects. In *Concise International Chemical Assessment Document 30*; WHO: Geneva, Switzerland, 2001. Available online: https://apps.who.int/iris/handle/10665/42367 (accessed on 15 June 2021).
30. Minnesota Pollution Control Agency. Air Emissions Risk Analysis (AERA). 2007. Available online: http://www.pca.state.mn.us/air/aera.html (accessed on 16 April 2021).
31. Thepanondh, S. *Development of Emission Factors for Air Pollutants and Greenhouse Gases from Vehicle for Establishment of Appropriate Mitigation Policy and Measure in the Transportation Sector in Thailand*; Mahidol University: Bangkok, Thailand, 2012.
32. Gulia, S.; Shrivastava, A.; Nema, A.K.; Khare, M. Assessment of Urban Air Quality around a Heritage Site Using AERMOD: A Case Study of Amritsar City, India. *Environ. Model. Assess.* **2015**, *20*, 599–608. [CrossRef]
33. EU Regulations. Directive 2006/11/EC of the European Parliament and of 15 February 2006 on pollution caused by certain dangerous substances discharged into the aquatic environment of the Community. *Off. J. Eur. Union* **2006**, *64*, 52–59.
34. United States Environmental Protection Agency. Region 6 Risk Management Addendum—Draft Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities, EPA-R6-98-002. 1998. Available online: http://www.epa.gov/region06 (accessed on 18 March 2021).
35. Jin, J.X.; Sun, S.D.; Wang, P.; Lin, Y.C.; Wang, T.; Wu, L.; Wei, N.; Chang, J.Y.; Mao, H.J. Vehicle emission inventory and scenario analysis in Liaoning from 2000 to 2030. *Huan Jing Ke Xue* **2020**, *41*, 665–673. [CrossRef]
36. Garcia-Herrero, I.; Margallo, M.; Laso, J.; Onandía, R.; Irabien, A.; Aldaco, R. Measuring the Vulnerability of an Energy Intensive Sector to the EU ETS under a Life Cycle Approach: The Case of the Chlor-Alkali Industry. *Sustainability* **2017**, *9*, 837. [CrossRef]
37. Randall, P.M. Pollution prevention strategies for the minimizing of industrial wastes in the VCM-PVC industry. *Environ. Prog.* **1994**, *13*, 269–277. [CrossRef]
38. Xia, W.; Liang, B.; Chen, L.; Zhu, Y.; Gao, M.; Chen, J.; Wang, F.; Chen, Y.; Tian. Atmospheric wet and dry depositions of polycyclic aromatic compounds in a megacity of Southwest China. *Environ. Res.* **2022**, *204*, 112151. [CrossRef] [PubMed]
39. Barber, J.L.; Thomas, G.O.; Kerstiens, G.; Jones, K.C. Current issues and uncertainties in the measurement and modelling of air–vegetation exchange and within-plant processing of POPs. *Environ. Pollut.* **2004**, *128*, 99–138. [CrossRef] [PubMed]
40. Pinthong, N.; Thepanondh, S.; Kondo, A. Source Identification of VOCs and their Environmental Health Risk in a Petrochemical Industrial Area. *Aerosol Air Qual. Res.* **2022**, *22*, 210064. [CrossRef]