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Human health risk assessment for PAHs, phthalates, elements, PCDD/Fs, and DL-PCBs in $PM_{2.5}$ and for NMVOCs in two East-Mediterranean urban sites under industrial influence

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Abstract:

This study evaluates the carcinogenic and the non-carcinogenic health risks related to non-methane volatile organic compounds (NMVOCs) and elements, dioxins, furans, dioxin-like polychlorobiphenyls, phthalates and polycyclic aromatic hydrocarbons in $PM_{2.5}$ samples collected during a one-year field campaign in two urban industrial areas in the East Mediterranean region. The health risk was assessed for the three exposure pathways (ingestion, inhalation and dermal contact) and for different age categories (newborns, children, adolescents, and adults). The non-carcinogenic risk calculated for the different species showed that benzene and n-heptane explained 78 to 94% of the total hazard index for NMVOCs at both sites. The total HI for NMVOCs varied between 2.9 and 26.8 at ZK and between 0.8 and 6.6 at FA for adults and newborns respectively exceeding the recommended USEPA limit of 1 for most age categories. $PM_{2.5}$-bound elements had values higher than the recommended USEPA limit for newborns with Mn, Pb, V, and Ni as the major contributors. The other species under study presented moderate risk values. The lifetime cancer risk due only to the exposure to NMVOCs was 170 and 46 times higher than the threshold limit at ZK and FA, respectively. $PM_{2.5}$-bound PAHs, As, Co, Cr(VI), Ni and V concentrations showed lifetime cancer risk exceeding the threshold limit of 10^{-6} with 58 and 28 additional cancer cases per million habitats at ZK and FA, respectively. To our knowledge, this assessment is a first evaluating the health risk of several classes of compounds from both particulate and gas phases.

Keywords: $PM_{2.5}$, NMVOCs, carcinogenic risk, exposure pathways, non-carcinogenic risk.

1 Introduction

According to the World Health Organization (WHO), air pollution is responsible for about 11% of the worldwide deaths annually, and therefore considered as the biggest environmental threat to human health (WHO, 2016). The international Agency for Research on Cancer (IARC) classified outdoor air pollution as well as different chemicals found in the atmosphere (chromium (VI), arsenic, cadmium, nickel, benzo[a]pyrene, benzene, 1,3-butadiene, 2,3,4,7,8-pentachlorodibenzofuran, and 2,3,7,8-tetrachlorodibenzo-para-dioxin) as Group I "carcinogenic to humans" (Loomis et al., 2013).

Inhalation, oral ingestion, and dermal absorption are the three major routes of both intentional and accidental exposure to atmospheric chemicals. Vapor-phase and fine respirable particulate matter can travel into the respiratory system by adhering to the walls of the respiratory tract or penetrating deep into the lungs (Díaz and Rosa Dominguez, 2009). However, the non-respirable particulates usually enter the human body via the oral ingestion route. The dermal absorption is possible for different physical forms. Particles - especially fine and ultrafine ones (having an aerodynamic diameter less than 2.5 and 0.1 μm, respectively) - exposure to the respiratory tract has been associated with various health problems such as asthma, chronic obstructive pulmonary disease, cardiovascular diseases and lung cancer (Alkoussa et al., 2020; Cochard et al., 2020; Mark Goldberg, 2008; Thurston et al., 2017).

Health risk assessment studies aim at evaluating the potential health effects resulting from human exposure to any source of risk or particular hazard (DoHA, 2012). This approach can be applied in a qualitative or quantitative way. In order to evaluate the severity of the exposure, it takes generally into account several factors such as the dose or intake, the characteristics of the exposed individuals, the duration and the frequency of the exposure, and other parameters related to the harmfulness of the compounds on human health. The evaluation of the health risks associated with exposure to atmospheric pollutants is of utmost importance as it can lead to developing effective risk management policies and strategies for a population (Chen et al., 2015; Samet and Krewski, 2007).

The East Mediterranean is the third region with the highest ambient and household air pollution attributable death rate (age-standardized), scoring an average of 125 per 100,000 population in 2016, after Africa (181) and South East Asia (166), while this number could tend to rise in the future with the increase of the aerosol concentrations due to climate change (Lelieveld et al., 2014). Lebanon, an East Mediterranean country, faces extensive urbanization with heavy traffic, installation of diesel private generators within neighborhoods with no law enforcement, dust events, open waste burning, along with emissions from different types of industries (Waked et al., 2012). The air quality in the country is considered unsafe as exceedances in $PM_{2.5}$ concentrations above WHO guidelines of 25 μg/m^3 are recorded (MoE, 2017) with more than 170 days in 2014 in Greater Beirut area. In order to evaluate the impact of air quality on human health in Lebanon, different approaches were tackled. Epidemiological studies were carried out to determine the association between outdoor air quality and different diseases such as chronic obstructive pulmonary diseases, chronic bronchitis, cardiovascular diseases and lung cancer (Aoun et al., 2013; Kobrossi et al., 2002; Nakhlé et al., 2015; Nasser et al., 2015; Salameh et al., 2012). These studies were based on questionnaires filled by hospital patients and air quality data. More recently, studies dealing with $PM_{2.5}$ from different areas in Lebanon (Abbas et al., 2019; Badran et al., 2020; Borgie et al., 2015; Melki et al., 2017) evidenced, on pulmonary cells *in vitro,* toxic effects of PM depending on the size and the composition of the particles.

PM atmospheric concentration is a widely used indicator of the air quality level. However, a high share of PM mass consists of compounds with low toxicity such as ammonium, sulfate, nitrate, sea salt, and crustal dust elements (Borm et al., 2007). On the other hand, species that barely contribute to PM mass such as transition metals and organic species can have a major impact on the aerosols' toxicity (Bandowe and Meusel, 2017; Chen and Lippmann, 2009). Among organic species, phthalates and PCDD/Fs are considered toxic to humans and were identified as endocrine disruptors and contributing to reproductive toxicity (Marquès and Domingo, 2019; Tsatsakis et al., 2019). Rengarajan et al. (2015) reported that the long-term exposure to mixtures of PAHs can increase the risks of mainly lung and skin, but also bladder and gastrointestinal cancers. That is why it is important to evaluate the risk of exposure to PM not as whole but based on its chemical composition. Therefore, most of the studies found in the literature focused on the evaluation of the health risk related to the

exposure to a specific family of compounds in PM such as elements, PAHs, and phthalates (Bai et al., 2020; Murari et al., 2020; Zhang et al., 2019) or for Volatile Organic Compounds (VOCs) (Dhaini et al., 2017).

Therefore, the aims of this work were as follow: (1) evaluate the cancer and non-cancer health risks based on a one-year $PM_{2.5}$ and NMVOCs field campaign in two urban industrial areas, (2) compare the risks associated with the different exposure pathways and different age categories, and (3) compare the risks associated with the different classes of compounds such as polycyclic aromatic hydrocarbons (PAHs), phthalates, major and trace elements, non-methane volatile organic compounds (NMVOCs), dioxins, furans, and dioxin-like polychlorobiphenyls. This study will be a first evaluating the health impact of the different classes of compounds (organic and inorganic) from particulate and gas phases for the different exposure pathways and for different age categories.

2 Materials and methods

2.1 Site description and sample collection

The description of the sampling sites was detailed elsewhere (Fadel et al., 2021) and will be shortly presented here. $PM_{2.5}$ and NMVOC samples were collected in two urban-industrial sites in Lebanon: Zouk Mikael (33°57'57.07''N 35°37'09.46''E) at a rooftop of a 15 m- residential building, and Fiaa in Chekka region (34°20'47.8''N 35°47'14.0''E) collocated with the ambient air quality monitoring station of the Lebanese Ministry of Environment. The locations of the sites were chosen taking into account the topography, the existence of residents, micro siting (buildings, traffic, etc.) to represent the air quality background in the study areas.

The Zouk Mikael site (ZK) is characterized by a high residential density (4,200 inhabitants/km^2) along with the biggest power plant in Lebanon which runs on Heavy Fuel Oil (HFO), an important number of private generators, and traffic. The Fiaa site (FA) is less populated (250 inhabitants/km^2) with moderate traffic activity and some private generators. The major industrial activities around FA site are related to three cement industries (distant between 5 and 7 km from the sampling site) in the region along with

their corresponding quarries and a sulfuric acid and phosphate fertilizer industry (15 km from the sampling site) in Selaata **(Fig. 1)**.

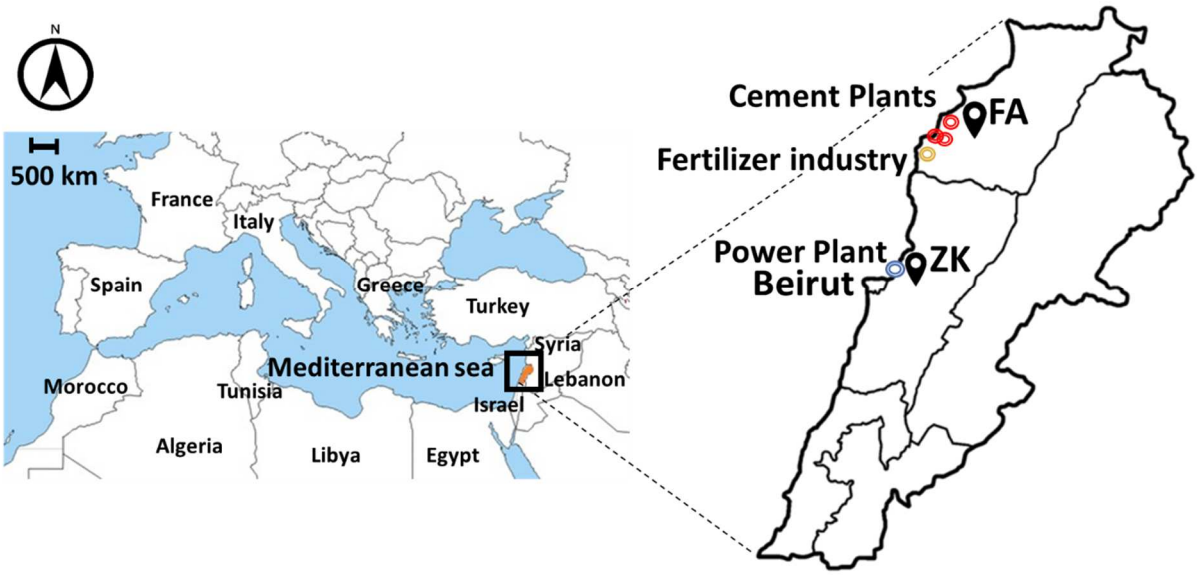

Fig. 1 Location of the two sampling sites: Zouk Mikael (ZK) and Fiaa (FA) and the position of the nearby industries

A total of 98 and 95 $PM_{2.5}$ samples were collected on a 24-hour basis every third day at ZK and FA, respectively from the 13th of December 2018 to the 15th of October 2019. The sampling was carried out using high volume samplers operating at 30 m^3/h, onto 150 mm pure quartz microfibers filters. Prior to sampling, the filters were heated for 12 hours at 550°C to remove the organic impurities.

In addition, non-methane VOCs (NMVOCs) were collected using passive radial diffuse samplers (Radiello– Code 120) and by following the guidelines of the manufacturer of Radiello diffusive passive sampler (Fondazione Salvatore Maugeri, IRCCS, Padova, Italy). The samples were placed for 4 weeks at both sites (11 samples per site). The cartridges were then kept at 4°C away from solvent until the analysis.

As for the 1,3-butadiene, the compound was trapped using Carbopack X diffusion tubes from Gradko International (Winchester, England) also over 4 weeks and a total of 9 samples per site were collected. Once the sampling completed, the tubes were returned to the company for the analysis.

2.2 Analysis of NMVOCs

155 NMVOCs were chemically extracted from cartridge by adding 2 mL of CS_2 and 100 µL of
156 2-fluorotoluene as internal standard in the Radiello glass tube without drawing out the
157 cartridge. The solution was stirred from time to time for 30 min before discarding the
158 cartridge, then filtered using PTFE Whatman filter (0.2 µm). 2 µL of the solution was
159 injected in the GC-MS (ISQ 7000, Thermo Scientific, United States of America) operating
160 in the split mode with a split ratio of 25. The GC was equipped with a 100% dimethyl
161 polysiloxane column (50 m x 0.2 mm x 0.5 µm, Supelco, United States of America). The
162 column temperature program consisted of an injection at 35˚C hold for 5 min, a ramp of
163 temperature corresponding to 1˚C/min up to 135˚C followed by another ramp of
164 temperature of 5˚C/min up to 250 ˚C and finally an isothermal hold step at 250 ˚C for 7
165 min. The GC was interfaced to an ion trap MS with an external electron ionization (EI)
166 source (220 ˚C, 70 eV). The identification of the compounds was made by comparing
167 their retention times and their mass spectrum (full scan mode, range 25-350 m/z) to the
168 authentic standards. The concentrations of the compounds (in ng/m^3) were calculated
169 using Eq.1 provided by the manufacturer of Radiello diffusive passive sampler
170 (Fondazione Salvatore Maugeri- IRCCS, Padova, Italy):

171 $$C = \frac{m}{Q_k \times t} \times 10^6 \quad (Eq.1)$$

172 Where C is the average concentration in µg/m^3, m is the mass of analyte in µg, t is the
173 exposure time in minutes, and Q_k is the sampling rate expressed in mL/min.

174 Field blanks and laboratory blanks were analyzed following the the same procedures as
175 the sampling cartridges. The regression coefficient for the calibration curves was in the
176 range of 0.96 - 0.99 for the different compounds.

177 1,3-butadiene quantification was achieved by the Gradko International company
178 (Winchester, England). The analysis has been carried out in accordance with in-house
179 method GLM 13-6 under UKAS fixed scope accreditation using a thermal desorption-gas
180 chromatography coupled to a mass spectrometer (TD-GC-MS).

181

182 ## 2.3 Chemical characterization of $PM_{2.5}$

183 PAHs and phthalates were extracted by a mixture of acetone/dichloromethane and
184 analyzed by GC/MS (ISQ 7000, Thermo Scientific, United States of America) using the

method described in Fadel et al. (2021). Major and trace elements were extracted by a mixture of $HNO_3/HF/HClO_4$ and quantified using Inductively Coupled Plasma-Atomic Emission Spectrometry (ICP-AES, iCAP 6000 series, Thermo Scientific, United Kingdom) for Al, Ba, Fe, Mn, Ni, Sr, Zn and Pb, and ICP-Mass Spectrometry (ICP-MS, Agilent 7900, Varian, United States of America) for elements such as As, Cd, Co, Sn, Cu, Cr, Sb, V, La, and Tl. The method is detailed in (Kfoury et al., 2016; Ledoux et al., 2006). The analytical procedure for elements analysis by ICP-AES and ICP-MS was validated considering standard reference materials: urban particulate matter NIST-SRM 1648a (National Institute of Standards and Technology, USA). Recovery rates varied between 89% and 103% with an exception for Cr (76%). Polychlorinated Dibenzo-p-Dioxins (PCDDs), furans (PCDFs), and Dioxin-Like Polychlorinated Biphenyls (DL-PCBs) were analyzed by MicroPolluants Technology SA (Saint Julien Les Metz, France) based on the US Environmental Protection Agency (US EPA) methods 1613 and 1668 (Borgie et al., 2015). The apparatus used is a high-resolution gas chromatography coupled to a high-resolution mass spectrometer (HRGC/HRMS).

2.4 Toxic equivalent concentrations for PAHs and PCDD/Fs and DL-PCBs

2.4.1 2,3,7,8-TCDD equivalents

2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) is known to be a human carcinogen, highly persistent and widespread environmental contaminant (NTP, 2016). A toxic equivalent factor (TEF_i) was introduced to assign toxicity values for the different congeners (PCDD/Fs and DL-PCBs) based on the toxicity of 2,3,7,8-TCDD reported as 1 (Van den Berg et al., 2006). The toxic equivalent "TEQ" representing an equivalent concentration of 2,3,7,8-TCDD is calculated following Eq. 2 by summing the product of the congeners' concentration (C_i) multiplied by its corresponding TEF values (TEF_i - unitless) (**Table S1**).

$$TEQ = \sum_{i=1}^{n} C_i \times TEF_i \quad \text{(Eq. 2)}$$

2.4.2 Benzo[a]pyrene equivalents

Among the different PAHs, benzo[a]pyrene has been classified as group I "Carcinogenic to humans" by the IARC. Upon that, the B[a]P equivalent concentration ($B[a]P_{eq}$) is often used to characterize the level of toxicity associated with PAHs. It is calculated as follows:

$$B[a]P_{eq} = \sum_{j=1}^{n} C_j \times TEF_j \quad (Eq. 3)$$

Where C_j is the concentration of the PAHs (ng/m^3) and TEF_j (unitless) is the corresponding Toxic Equivalency Factor for each PAH compared to B[a]P (TEF for B[a]P is 1) proposed by Nisbet and LaGoy (1992) and listed in **Table S2**.

2.5 Health Risk assessment

The health risk assessment was based on the Human health evaluation guidelines proposed by the United States Environmental Protection Agency (USEPA, 1989, 1991, 2011) and was performed for different age categories: newborns (0 to <1 year), children (1 to <12 years), adolescents (12 to <18 years) and adults (18 to 70 years), and for three exposure pathways: inhalation, ingestion, and dermal absorption on exposed skin when data were available. The average concentrations of the species on the sampling period were considered for the health risk assessment assuming that they represent a typical yearly concentration of the pollutants in the atmosphere of the considered sites.

2.5.1 Non-cancer risk assessment

The assessment of the non-carcinogenic risk resulting from exposure to a chemical was based on the comparison between the exposure estimate such as the average daily dose (ADD_i) to the reference dose (RfD_i) both expressed in mg/kg/day or the comparison of the exposure concentration in air (EC_i) to the reference concentration for inhalation (RfC_i) expressed in mg/m^3. These ratios are known as hazard quotients (HQ_i) and are calculated following one of the formulas of Eq. 4 depending on the availability of RfD_i or RfC_i in the literature (USEPA, 1989, 1991, 2011):

240 $$HQ_i = \frac{ADD_i}{RfD_i} \; or \; HQ_i = \frac{EC_i}{RfC_i} \quad (Eq.4)$$

241 With ADD_i for the different exposure pathways and EC_i for inhalation estimated as
242 follows (Eq. 5 to Eq. 8):

243 $$ADD_{i-inhalation} = \frac{C_i \times IR_{inh} \times ED \times EF \times 10^{-6}}{BW \times AT} \quad (Eq.5)$$

244 $$ADD_{i-ingestion} = \frac{C_i \times IR_{ing} \times ED \times EF \times 10^{-6}}{BW \times AT} \quad (Eq.6)$$

245 $$ADD_{i-dermal} = \frac{C_i \times SA \times AF \times ABS \times ED \times EF \times 10^{-6}}{BW \times AT} \quad (Eq.7)$$

246 $$EC_i = \frac{C_i \times ET \times EF \times ED \times 10^{-6}}{AT} \quad (Eq.8)$$

247 Detailed information of each parameter used in the different formulas is given in **Table**
248 **1** for newborns, children, adolescents, and adults. Values for RfD_i and RfC_i are presented
249 in Table **S3** for the different exposure pathways.

250 Total HQ_i is defined as the non-carcinogenic risk for a certain compound in the different
251 exposure pathways assuming additive effects (Eq. 9) and calculated as follows:

252 $$Total\ HQ_i = HQ_{i-inhalation} + HQ_{i-ingestion} + HQ_{i-dermal} \quad (Eq.9)$$

253 On the other hand, a receptor may be exposed to a specific family of chemicals
254 associated with non-carcinogenic effects. Accordingly, a hazard index (HI) for a family of
255 chemicals is defined as the total non-carcinogenic hazard for each exposure pathway
256 and calculated as follows (Eq.10):

257 $$HI_{inhalation/ingestion/dermal} = \sum_i HQ_{i-inhalation\ /\ ingestion/\ dermal} \quad (Eq.10)$$

258 Finally, in order to evaluate the total non-carcinogenic risk for a specific family for the
259 combined exposure pathways, the total hazard index (THI) is calculated as follows (Eq.
260 11):

261 $$Total\ HI = THI = HI_{inhalation} + HI_{ingestion} + HI_{dermal} \quad (Eq.11)$$

Generally, the higher the HQ_i, total HQ_i, HI and Total HI values above 1, the greater is the level of concern and the higher the probability of occurrence for hazardous non-carcinogenic effects (USEPA, 2011).

2.5.2 Cancer risk assessment

The risk estimates of carcinogenic chemicals represent the incremental probability that an individual might develop cancer over a lifetime as a result of a specific exposure to a carcinogenic chemical. The risks for inhalation and other pathways are calculated following (Eq. 12). CSF_i is the cancer slope factor for a chemical in a specific exposure pathway expressed in kg·day/mg. IUR_i is the inhalation unit risk for a chemical expressed in m^3/mg used to calculate the cancer risk associated with the inhalation pathway (USEPA, 1997). CSF_i and IUR_i values are presented in **Table S3**.

$$CR_{i-inhalation} = EC_i \times IUR_i \quad or \quad CR_{i-ingestion/dermal} = LADD_i \times CSF_i \quad (Eq.12)$$

When considering carcinogenic species, the exposure period during which the dose is averaged is usually lifetime or 70 years. Accordingly, the lifetime average daily dose ($LADD_i$) calculation equations for the different exposure pathways take the form of (Eq. 5 to 8) with lifetime (25,500 days) replacing the average timing (ED x 365 days) (USEPA, 1989, 2011).

To estimate the carcinogenic risk for a certain compound for the three exposure pathways, total CR_i was calculated as follows (Eq.13):

$$Total\ CR_i = CR_{i-inhalation} + CR_{i-ingestion} + CR_{i-dermal} \quad (Eq.13)$$

The total cancer risk for a specific family of chemicals associated with an exposure pathway is estimated as follows (Eq.14):

$$CR_{\ inhalation/ingestion/dermal} = \sum_i CR_{i\ inhalation/ingestion/dermal} \quad (Eq.14)$$

In order to evaluate the cancer risk for a family of chemicals attributed to the combined three exposure pathways, the cumulative cancer risk was calculated according to (Eq.15):

$$Cumulative\ CR = CCR = CR_{inhalation} + CR_{\ ingestion} + CR_{dermal} \quad (Eq.15)$$

Table 1 : Exposure parameters for newborns, children, adolescents, and adults through inhalation, ingestion, and dermal contact considered in this study for the health risk assessment

Exposure parameters		**Unit**	**Newborn**	**Child**	**Adolescent**	**Adult**	**Reference**
			0 to <1 year	**1 to <12 years**	**12 to <18 years**	**18 to 70 years**	
Concentration in air	C_i	ng/m³					
Inhalation intake rate	IR_{inh}	m³/day	5.4	11.2	15.6	15.5	(USEPA, 2011)
Soil intake rate	IR_{ing}	mg/day	60	100	100	50	(USEPA, 2011)
Body Weight	BW	kg	7.8	25	61	72	(USEPA, 1997, 2011)
Exposure frequency (for residents)	EF	days/year	350	350	350	350	(USEPA, 2004b)
Exposure time	ET	h/day	6	6	8	8	(Dahmardeh Behrooz et al., 2021)
Exposure duration	ED	years	1	11	6	52	(USEPA, 2011)
Average timing	AT	days	AT = ED x 365(days) for non-carcinogens AT = 70 years x 365 for carcinogens				(USEPA, 1989)
Exposed skin surface area	SA	cm²	1300	3200	4650	5700	(USEPA, 2002)
Dermal adherence factor	AF	mg/cm²	0.2	0.2	0.2	0.07	(USEPA, 2002)
Dermal adsorption factor	ABS	-	0.13 for PAHs 0.03 for As 0.001 for Cd 0.01 for other metals				(USEPA, 2002)

Finally, the estimation of the lifetime cancer risk (LCR) for a species or a group of species, the following equation (Eq.16) was used (OEHHA, 2012) :

$$LCR = Cumulative\ CR_{Newborn} \times \frac{1}{70} + Cumulative\ CR_{child} \times \frac{11}{70} + Cumulative\ CR_{adolescent} \times \frac{6}{70} + Cumumative\ CR_{adult} \times \frac{52}{70} \quad (Eq.\ 16)$$

302 # 3 Results and discussion

303

304 ## 3.1 Atmospheric concentrations of the compounds

305 The average concentrations of PAHs, phthalates, and elements in $PM_{2.5}$ and NMVOCs are
306 presented in **Table 2** for ZK and FA sites. The concentrations of individual PAHs as well
307 as phthalates were presented in details in Fadel et al. (2021).

308

309 Table 2: Average atmospheric concentrations and standard deviations of total PAHs,
310 phthalates, and elements in $PM_{2.5}$ along with concentrations of non-methane volatile
311 organic compounds (NMVOCs) during the entire sampling period

Compounds (ng/m³)	**ZK site**	**FA site**	**Reference**
Total $PM_{2.5}$-bound PAHs	2.56 ± 2.5	0.95 ± 1.12	(Fadel et al., 2021)
$PM_{2.5}$-bound phthalates			
dibutyl phthalate	37 ± 29	12.4 ± 11.6	(Fadel et al., 2021)
bis(2-ethylhexyl)phthalate	49 ± 17	66 ± 70	
Major and trace elements			
As	0.53 ± 0.28	0.49 ± 0.24	This study
Cd	0.13 ± 0.10	0.14 ± 0.10	
Co	1.22 ± 2.10	0.61 ± 0.76	
Cr	1.88 ± 1.50	1.97 ± 1.33	
Cu	4.63 ± 3.66	2.53 ± 1.75	
Mn	7.46 ± 8.78	5.61 ± 7.0	
Ni	11.7 ± 12.3	6.29 ± 4.50	
Pb	12.8 ± 20.1	17.5 ± 29.3	
Zn	26.1 ± 16.3	16.7 ± 11.1	
Ba	14.2 ± 13.6	9.1 ± 5.6	
Al	593 ± 1000	492 ± 800	
V	25 ± 27	13 ± 9	
Fe	452 ± 615	298 ± 466	
Sr	5.4 ± 9.0	4.5 ± 5.7	
Sn	0.83 ± 0.60	0.57 ± 0.60	
Sb	1.11 ± 0.75	0.95 ± 0.92	
La	0.54 ± 0.63	0.33 ± 0.44	
Tl	0.03 ± 0.02	0.03 ± 0.03	
NMVOCs			
1,3-butadiene	77.7 ± 70	<D.L.	This study
hexane	419 ± 720	150 ± 137	
tetrachloroethene	291 ± 115	58 ± 9	
benzene	4318 ± 5393	1526 ± 3340	
cyclohexane	398 ± 187	94 ± 200	
heptane	657 ± 170	138 ± 84	
methylcyclohexane	446 ± 93	88 ± 37	
toluene	16910 ± 3428	2740 ± 1280	
ethylbenzene	4210 ± 911	655 ± 354	
m+p-xylene	5730 ± 1293	743 ± 264	

o-xylene	4375 ± 946	605 ± 210

Briefly, the major contributors to the total $PM_{2.5}$-bound PAHs at ZK were chrysene, benzo[b]fluoranthene, benzo[a]pyrene, dibenz[a,h]anthracene, and indeno(1,2,3-c,d)pyrene accounting for 66% of the total PAHs concentration (2.56 ng/m^3). While at FA, fluoranthene, pyrene, benzo[b]fluoranthene, dibenz[a,h]anthracene, and benzo[g,h,i]perylene contribute to 63% of the total PAHs in the particulate phase (0.95 ng/m^3). As for phthalates, the average concentrations of bis(2-ethylhexyl)phthalate (BEHP) and dibutyl phthalate (DBP) over the sampling period were 49 and 37 ng/m^3 at ZK, while at FA, the concentrations were 66 and 12.4 ng/m^3, respectively.

Table 3 presents PCDD/Fs, and DL-PCBs congener concentrations observed in ZK and FA sites in the particulate phase. The total average concentrations of PCDDs and PCDFs were 164 and 124 fg/m^3 at ZK and FA, respectively. OCDD and 1,2,3,4,6,7,8-HpCDF were the two dominant congeners among PCDD/Fs contributing to 49% and 46% at ZK and FA, respectively, followed by 1,2,3,4,6,7,8-HpCDD and OCDF. DL-PCB congeners are dominated by PCB 105 and PCB 118 accounting for 86% and 83% of total DL-PCBs at ZK and FA, respectively. Elements' concentrations varied at ZK from 0.03 ng/m^3 for Tl to 593 ng/m^3 for Al, and from 0.03 ng/m^3 for Tl to 492 ng/m^3 for Al at FA **(Table 2).** Finally, for NMVOCs, the highest concentrations are observed for benzene, toluene, ethylbenzene and xylenes. Between these compounds, toluene showed the highest concentrations at both sites and 1,3-butadiene showed the lowest concentrations at ZK and was not detected at FA **(Table 2).**

These species have been used for the evaluation of the carcinogenic and the non-carcinogenic risk due to their adverse health effects and the availability of reference values (RfD, RfC, CSF, and IUR) in the literature **(Table S3).**

3.2 Health risk evaluation

The hazard quotient (HQ_i) and the cancer risk (CR_i) associated with each species in the three exposure pathways for the different age categories are reported in **Table S4** and **Table S5** for ZK and FA, respectively. Furthermore, the total hazard index (THI) and the cumulative cancer risk (CCR) for PAHs and TCDD equivalents along with total HQ_i and

342 total CR_i for phthalates, elements, and NMVOCs were summarized for ZK in **Table S6**
343 and for FA in **Table S7**.

344

345

346 Table 3: Dioxins (PCDD), furans (PCDF) and dioxin-like polychlorobiphenyls (DL-PCBs)
347 average concentrations (fg/m^3) in $PM_{2.5}$ samples at Zouk (ZK) and Fiaa (FA)

Compounds (fg/m^3)	**Abbreviation**	**ZK site**	**FA site**
PCDDs		**81.1** ± 38.7	**52.0** ± 21.7
2,3,7,8-tetrachlorinated dibenzo-p-dioxin	2,3,7,8 TCDD	<D.L.	<D.L.
1,2,3,7,8-pentachlorinated dibenzo-p-dioxin	1,2,3,7,8 PeCDD	0.43 ± 0.20	<D.L.
1,2,3,4,7,8-hexachlorinated dibenzo-p-dioxin	1,2,3,4,7,8 HxCDD	0.51 ± 0.30	<D.L.
1,2,3,6,7,8-hexachlorinated dibenzo-p-dioxin	1,2,3,6,7,8 HxCDD	1.77 ± 0.93	1.69 ± 0.06
1,2,3,7,8,9-hexachlorinated dibenzo-p-dioxin	1,2,3,7,8,9 HxCDD	1.26 ± 0.95	<D.L.
1,2,3,4,6,7,8-heptachlorinated dibenzo-p-dioxin	1,2,3,4,6,7,8 HpCDD	23.18 ± 7.50	16.13 ± 3.07
octachlorinated dibenzo-p-dioxin	OCDD	53.98 ± 28.81	34.20 ± 18.6
	TEQ for PCDD	***1.03***	***0.34***
PCDFs		***82.5*** ± 35.1	**71.8** ± 36.7
2,3,7,8 tetrachlorinated dibenzofuran	2,3,7,8 TCDF	1.31 ± 1.20	1.57 ± 0.96
1,2,3,7,8 pentachlorinated dibenzofuran	1,2,3,7,8 PeCDF	2.43 ± 2.28	1.95 ± 0.75
2,3,4,7,8 pentachlorinated dibenzofuran	2,3,4,7,8 PeCDF	4.49 ± 3.32	7.35 ± 7.13
1,2,3,4,7,8 hexachlorinated dibenzofuran	1,2,3,4,7,8 HxCDF	7.76 ± 3.99	6.51 ± 4.52
1,2,3,6,7,8 hexachlorinated dibenzofuran	1,2,3,6,7,8 HxCDF	6.61 ± 3.29	5.37 ± 3.13
2,3,4,6,7,8 hexachlorinated dibenzofuran	2,3,4,6,7,8 HxCDF	8.79 ± 1.52	8.30 ± 3.0
1,2,3,7,8,9 hexachlorinated dibenzofuran	1,2,3,7,8,9 HxCDF	2.79 ± 0.77	2.60 ± 1.02
1,2,3,4,6,7,8-heptachlorinated dibenzofuran	1,2,3,4,6,7,8 HpCDF	26.43 ± 8.62	23.04 ± 9.7
1,2,3,4,7,8,9-heptachlorinated dibenzofuran	1,2,3,4,7,8,9 HpCDF	3.16 ± 0.35	2.63 ± 2.4
octachlorinated dibenzofuran	OCDF	18.75 ± 9.76	12.46 ± 4.11
	TEQ for PCDF	***4.45***	***4.96***
DL-PCBs		**454.8** ± 202	**586.3** ± 128
3,4,4',5-tetrachlorobiphenyl	PCB 81	<D.L.	<D.L.
3,3′,4,4′-tetrachlorobiphenyl	PCB 77	21.66 ± 6.64	8.39± 8.0
2,3',4,4',5'-pentachlorobiphenyl	PCB 123	20.20 ± 23.52	55.51± 7.67
2,3',4,4',5-pentachlorobiphenyl	PCB 118	278.2 ± 112.6	325.2 ±50.7
2,3,4,4',5-pentachlorobiphenyl	PCB 114	<D.L.	4.59 ± 5.0
2,3,3',4,4'-pentachlorobiphenyl	PCB 105	111.4 ± 45.0	158.6 ± 37.1
3,3',4,4',5-pentachlorobiphenyl	PCB 126	<D.L.	<D.L.
2,3',4,4',5,5'-hexachlorobiphenyl	PCB 167	8.37 ± 8.0	<D.L.
2,3,3',4,4',5-hexachlorobiphenyl	PCB 156	15.08 ± 6.33	13.40 ± 6.0
2,3,3',4,4',5'-hexachlorobiphenyl	PCB 157	<D.L.	<D.L.
3,3',4,4',5,5'-hexachlorobiphenyl	PCB 169	<D.L.	11.66 ± 6.2
2,3,3',4,4',5,5'-heptachlorobiphenyl	PCB 189	<D.L.	8.95 ± 7.0
	TEQ for PCB	***0.02***	***0.37***
	Total TEQ	***5.49***	***5.67***

348

349

350 3.2.1 Polycyclic Aromatic Hydrocarbons

351 The PAHs in this study were only analyzed in the particulate phase. Even though PAHs
352 concentrations are usually higher in the gas phase according to the literature in other
353 Mediterranean sites such as in Turkey and Greece (Akyüz and Çabuk, 2010; Gaga and
354 Ari, 2019; Vasilakos et al., 2007), low B[a]P_{eq} concentrations can be estimated. This
355 could be explained by the abundance of the PAHs with high contribution to the B[a]P_{eq}
356 value in the particulate phase. Consequently, only $PM_{2.5}$-bound PAHs were studied.

357 The B[a]P_{eq} value for the 16 PAHs in the particulate phase was 2.58 and 0.69 ng/m^3 at
358 ZK and FA, respectively. The 7 PAHs classified by the IARC as group 1, 2A or 2B
359 (benz[a]anthracene, chrysene, benzo[b]fluoranthene, benzo[k]fluoranthene,
360 benzo[a]pyrene, dibenz[a,h]anthracene, and indeno(1,2,3-c,d)pyrene) accounted for
361 98% of the B[a]P_{eq} value at ZK and 99% at FA showing that these compounds were the
362 main contributors to $PM_{2.5}$-bound PAHs toxicity.

363 The non-cancer risk for PAHs was evaluated for ingestion and inhalation pathways.
364 Exposure to PAHs via inhalation exhibited the highest risk for the different age
365 categories contributing to more than 80% of the total HI **(Tables S4 and S5)**. At both
366 sites, the total HI value related with the exposure to the $PM_{2.5}$-bound PAHs was <1
367 (acceptable risk), with a higher value for adolescents (0.43 at ZK and 0.11 at FA) then
368 adults (0.42 at ZK and 0.10 at FA), then come the newborns (0.37 at ZK and 0.10 at FA)
369 and the children (0.34 at ZK and 0.09 at FA) **(Tables S6 and S7).**

370 The cumulative cancer risk associated with exposure to $PM_{2.5}$-bound PAHs via ingestion,
371 inhalation and dermal contact were evaluated **(Tables S6 and S7)**. The results showed
372 that the values exceeded the potential risk value recommended by USEPA (10^{-6}) except
373 for newborns at FA with a trend of children (1.5 x 10^{-5} at ZK and 4.1 x 10^{-6} at FA) >
374 adults (1.4 x 10^{-5} at ZK and 3.9 x 10^{-6} at FA) > adolescents (3.8 x 10^{-6} at ZK and 1.0 x 10^{-6}
375 at FA) > newborns (2.5 x 10^{-6} at ZK and 7 x 10^{-7} at FA), showing a potential cancer risk at
376 these PAHs concentration exposure levels **(Fig. 2).** The cumulative cancer risk values
377 reported for children and adults at both sites were higher than those reported for a site
378 in Thailand (6.1 x 10^{-9} - 7.7 x 10^{-9} for children and 1.3 x 10^{-8} – 1.4 x 10^{-8} for adults)
379 having lower B[a]P_{eq} concentration (0.06-0.07 ng/m^3) (Pongpiachan, 2015). In terms of
380 exposure pathways, ingestion and dermal contact were the main exposure routes
381 leading to cancer risk (2.0 x 10^{-6} and 3.8 x 10^{-7} at ZK, 5.3 x 10^{-7} and 1.0 x 10^{-7} at FA,
382 respectively), which were at least three times higher than the inhalation pathway (9.4 x

383 10^{-8} at ZK and 2.5 x 10^{-8} at FA) **(Table 4, Table 5).** The CR values from inhalation were
384 lower than those reported for a site characterized by high traffic density and industrial
385 activities (4 x 10^{-7} – 8.4 x 10^{-5}), but also an urban Indian site with values in the range of
386 10^{-6} (Fang et al., 2020; Kumar et al., 2020). These differences are mostly related to $PM_{2.5}$-
387 bound PAHs concentrations (87-608 ng/m^3) that were at least 80 times higher than the
388 ones at our study (2.56 ng/m^3 at ZK and 0.95 ng/m^3 at FA).

389 The lifetime cancer risk values for the exposure to PAHs were 1.4 x 10^{-5} at ZK and 3.7 x
390 10^{-6} and FA, exceeding the threshold limit of 10^{-6}. PAHs can be metabolized and can react
391 as electrophilic intermediates capable of forming PAH-DNA adduct, considered as
392 biomarker of DNA damage and that has been linked to cancer (Veglia et al., 2003). The
393 observed concentrations of these species at both sites were mainly attributed to
394 combustion processes such as vehicular emissions, biomass burning, and power plants
395 (Fadel et al., 2021). These results show that exposure to these PAHs emitted from
396 various anthropogenic activities could be responsible for 14 and 4 additional cancer
397 cases per million habitats at ZK and FA, respectively. Reducing PAHs concentrations,
398 especially from these combustion sources can largely reduce the CCR attributed to PAHs
399 exposure for the inhabitants.

400

401 3.2.2 Phthalates

402 Among the analyzed phthalates, BEHP was classified by the IARC as possibly
403 carcinogenic to humans (group 2B) due to sufficient evidence for carcinogenicity in the
404 liver of animals but little evidence of possible association between BEHP and liver
405 cancer in humans (Rusyn and Corton, 2012). On the other hand, butyl benzyl phthalate
406 (BBP) was classified as non-carcinogenic (group 3), and other phthalate esters such as
407 DBP were not assessed (IARC, 2004). As for the non-carcinogenic effects, asthma and
408 allergies were reported to be linked to exposure to BEHP and BBP, respectively
409 (Jaakkola and Knight, 2008). In this study, the cancer risk of BEHP and the non-cancer
410 risk of DBP and BEHP were studied.

411 The total HI values, corresponding here to the HI value for ingestion as no RfC_i or RfD_i
412 were found for these phthalates for inhalation or dermal contact, ranged between 10^{-2}

413 and 10^{-3} for the different age categories at both sites resulting in a negligible non-cancer
414 risk (<1).

415 As for the cancer risk, the only contributor was BEHP. CR values were higher for the
416 ingestion pathway compared to inhalation: 6 times higher for an adult and 18 times
417 higher for a newborn. Conversely, CR for inhalation, ingestion, and cumulative CR
418 (inhalation + ingestion; no value for dermal) presented low cancer risk with values
419 below 10^{-6}. The CR values for inhalation at both sites (3.9×10^{-9} at ZK and 5.2×10^{-9} at
420 FA) **(Table 4, Table 5)** were in the interval of estimated carcinogenic risk reported by Li
421 et al. (2019) for a regional background site (8.9×10^{-10} - 7.4×10^{-9}). The cumulative CR
422 values for BEHP found in this study were higher than the ones reported by Ma et al.
423 (2020) for a Chinese city (in the range of 10^{-10}). Although the concentration of BEHP
424 (54.8 ng/m^3) in the latter study was close to those reported in ZK and FA, the exposure
425 parameters were different especially for body weight that was reported as 15 kg for a
426 child and 58.6 kg for an adult along with the inhalation rate (7.8 m^3/day for a child and
427 12.8 m^3/day for an adult). The lifetime cancer risk from exposure to BEHP was 3.8×10^{-7}
428 for ZK and 5.1×10^{-7} for FA.

429 Phthalates are generally released into the atmosphere during open burning of plastics
430 (Simoneit et al., 2005). Based on the total HI and the cumulative CR values, the risk
431 associated with human exposure to $PM_{2.5}$-bound phthalates were relatively low.
432 However, these compounds should be monitored by reducing open burning of plastics
433 because the exposure to these species even at doses lower than the RfD might be linked
434 to adverse health problems (Chen et al., 2017).

435

436 3.2.3 Dioxins, furans and dioxin-like polychlorobiphenyls

437 TEQ values were calculated for every class of compounds along with total TEQ (**Table**
438 ***3*).** Although contributions in terms of concentrations differ between the two sites,
439 PCDFs were the largest contributors to the total TEQ accounting for 81% (ZK) and 87%
440 (FA) of the total TEQ values. 2,3,4,7,8-PeCDF contributed mostly to total TEQ with a
441 percentage of 23.7% at ZK and 38.9% at FA. These findings were similar to those done in
442 other industrial sites (Chang et al., 2003). DL-PCBs in ZK only contribute to 0.3% of total

443 TEQ while in FA, they contribute to 6.5%. The main difference between the two sites was
444 PCB 169 that was not detected in ZK and highly contributes to the PCB-TEQ at FA.

445 The total HI value calculated using the total TEQ shows that the non-cancer risk due to
446 exposure to PCDD/Fs and DL-PCBs is negligible at both sites **(Fig. 2).** As for the cancer
447 risk, both $CR_{inhalation}$ and $CR_{ingestion}$ have values below 10^{-6} with a higher $CR_{ingestion}$ for
448 adults (7-fold higher) and newborns (100-fold higher).

449 PCDD/Fs were generally emitted from the incineration of hospital, municipal, and
450 hazardous waste as well as during the manufacturing of chlorinated pesticides (Zubair
451 and Adress, 2019). As for DL-PCBs, they were produced as industrial chemicals or
452 generated unintentionally during incineration of waste (Hogarh et al., 2018). The
453 lifetime cancer risk was found to be 4.0×10^{-7} for both sites. These values can be
454 decreased by reducing waste incineration. LCR values found in our study were lower
455 than the ones reported near different factories in China such as sintering, aluminum and
456 copper plants (Sun et al., 2017; Yang et al., 2017). This might be mainly due to reported
457 values for both particulate and gas phase in the above-mentioned studies and reported
458 equivalent TEQ that were at least 10 times higher compared to our study.

459

460 3.2.4 Major and trace elements

461 The populations living around the study sites are considered the main receptors of
462 airborne elements. It is worth noting that the toxicity of Cr was mainly reported to the
463 hexavalent state Cr(VI). Therefore, the Cr atmospheric concentration was scaled by a
464 factor of 1/7, given the assumed Cr(VI)/Cr(III) concentration ratio of 1 to 6 (USEPA,
465 2004a). It was also assumed that the measured arsenic is totally inorganic (Huang et al.,
466 2014). The HQ_i values for all the identified elements (As, Cd, Co, Cr, Cu, Mn, Ni, Pb, Zn, Ba,
467 Al, V, Fe, Sr, Sn, Sb, La and Tl) were calculated for the different exposure pathways and
468 age categories (**Tables S4** and **S5)**.

469 As and Zn exhibit higher HQ_i values for dermal contact and ingestion while for Sb, the
470 main health risk is due to ingestion. For this latter route, As, Zn and Sb were more likely
471 to have potential hazard for newborns and children **(Table S5):** these elements can be
472 accumulated in soil and dust and transferred to children through ingestion (Xue et al.,
473 2007). The total HQ_i value for these elements were far less than 1 for the different age

474 categories at both sites; the potential carcinogenic risks were negligible and considered
475 not harmful (**Tables S6** and **S7**).

476 Fe, Sr, Sn, La and Tl were only considered via the ingestion pathway and exhibited HQ_i
477 lower than the safe limit value of 1. For Ni, the health risk was mainly explained by
478 dermal contact and inhalation. For the other elements, i.e., Cd, Co, Cr(VI), Cu, Mn, Pb, Ba,
479 Al and V, the main risk was associated with the inhalation route at both sites.

480 Considering the three routes, i.e., total HQ_i values, the non-carcinogenic risk, from the
481 highest to the lower value, was observed for Mn (highest risk), then Pb, Ni, Co, and V
482 (lowest risk) ; for FA site, the order was as follows: Pb, then Mn, Ni, Co, and V. Even for
483 these elements, Total HQ_i values were less than 1 for the different age categories
484 implying that these elements do not seem to pose any non-carcinogenic health risk.

485 Mn and Pb contributed to more than 50% of the total HI. This is mainly due to their
486 relatively high concentrations in air. When comparing the three exposure pathways,
487 inhalation contributed the most to the non-cancer risk for the different age categories
488 (77-93%).

489 However, when considering the total HI for elements, the value is close to 1 for children
490 at ZK and FA and exceeding 1 for newborns while lower values were reported for
491 adolescents and adults **(Fig. 2)**. This could possibly be explained by the higher rate of
492 exposure and the lower body weight of children and newborns compared to adolescents
493 and adults. The total HI for elements in this study was higher than those presented by
494 Murari et al. (2020) for an urban city in India (10^{-4}- 10^{-3}). The differences are mainly due
495 to the choice made for exposure parameters and the variations in the concentrations of
496 the elements specifically Pb, Mn and Co.

497 The cancer risk for the different exposure pathways was calculated for As, Cd, Co, Cr(VI),
498 Ni, Pb and V. As shown in **Table S4** and **Table S5**, no values exceeded the average risk
499 acceptance of 10^{-6}/year for dermal contact and ingestion pathways at both sites.
500 However, for the inhalation route, adults were subjected to more carcinogenic risk from
501 As, Co, Cr(VI), Ni and V at ZK since their CR values were 1.2 x 10^{-6}, 1.9 x 10^{-6}, 1.7 x 10^{-6},
502 1.5 x 10^{-6} and 5.2 x10^{-5} respectively, exceeding the safe limit of 10^{-6}. Additionally,
503 vanadium showed high cancer risk values of 8.1 x 10^{-6} and 5.9 x 10^{-6} for children and
504 adolescents, respectively. While at FA, values exceeded the limit for As, Cr(VI) and V for

adults and also V for children and adolescents. The cancer risks through the inhalation pathway for the elements were either in the same range or higher than the ones reported for a Romanian site (6.5 - 7.2 x 10^{-6}), an urban site in China (7.1 x 10^{-6}), and an urban site in Colombia (3.1 x 10^{-6}) (Galon-Negru et al., 2019; Liu et al., 2018; Ramírez et al., 2020).

The cumulative cancer risk for the three exposure pathways was assessed **(Tables S6 and S7)** and the problematic species were, from highest to lower values, V > Cr(VI) > As > Co > Ni > Pb > Cd for FA and V > As > Cr(VI) > Co > Ni > Pb > Cd for ZK. Hence, V, As, and Cr(VI) were the major carcinogenic toxic elements among these elements. Specifically, V was the top contributor to the cumulative CR with a contribution varying between 40 - 90% according to the different age categories and the sites. The cancer risk associated to V was higher than 10^{-6} at ZK and FA for all age categories, excepted newborn. The cancer risk values associated with adults were the highest between the different age categories (5.2 x 10^{-5} at ZK and 2.6 x 10^{-5} at FA). V, along with Ni, were generally considered as important tracers of HFO combustion, Cr and Co were mainly associated with industrial emissions and As was emitted from coal combustion (Becagli et al., 2012; Chen et al., 2021). The exposure to high concentrations of V, Cr(VI), and As can lead to an increased risk of lung cancer according to epidemiological studies(Deng et al., 2019; Hubaux et al., 2013; Rondini et al., 2010).

When considering the cumulative cancer risk for the elements as a whole, it exceeded the average risk acceptance of 10^{-6} per year for all age categories at ZK and the risk is higher for adults, followed by children, adolescents and finally newborns **(Fig. 2).** At FA, cumulative CR exceeded the limit of 10^{-6} for all age categories except newborns and the risk occurs on the same order as ZK.

This shows that adults were subjected to more carcinogenic risks than the other age categories at both sites. The cumulative cancer risk (1.3 x 10^{-6} – 5.9 x 10^{-5} for ZK and 8.0 x 10^{-7} 3.0 x 10^{-5} for FA) was mostly found higher than the values presented for a dusty site in Nigeria for children and adults (Sulaymon et al., 2020).

These differences were observed due to the higher concentrations of V (highest contributor to cumulative CR) recorded at ZK (25 ng/m^3) and FA (13 ng/m^3) compared to the Nigerian site (5-8 ng/m^3). The lifetime cancer risk for 70 years of exposure

exceeded the safe limit of 10^{-6}. Per million habitats, 44 and 25 additional cases of cancer were found based on the LCR calculations at ZK and FA, respectively. In order to decrease these numbers, the concentrations of the major contributor to the CCR, namely Vanadium, should be reduced. This can be done by replacing the energy production based on the combustion of heavy fuel oil used by cleaner energy.

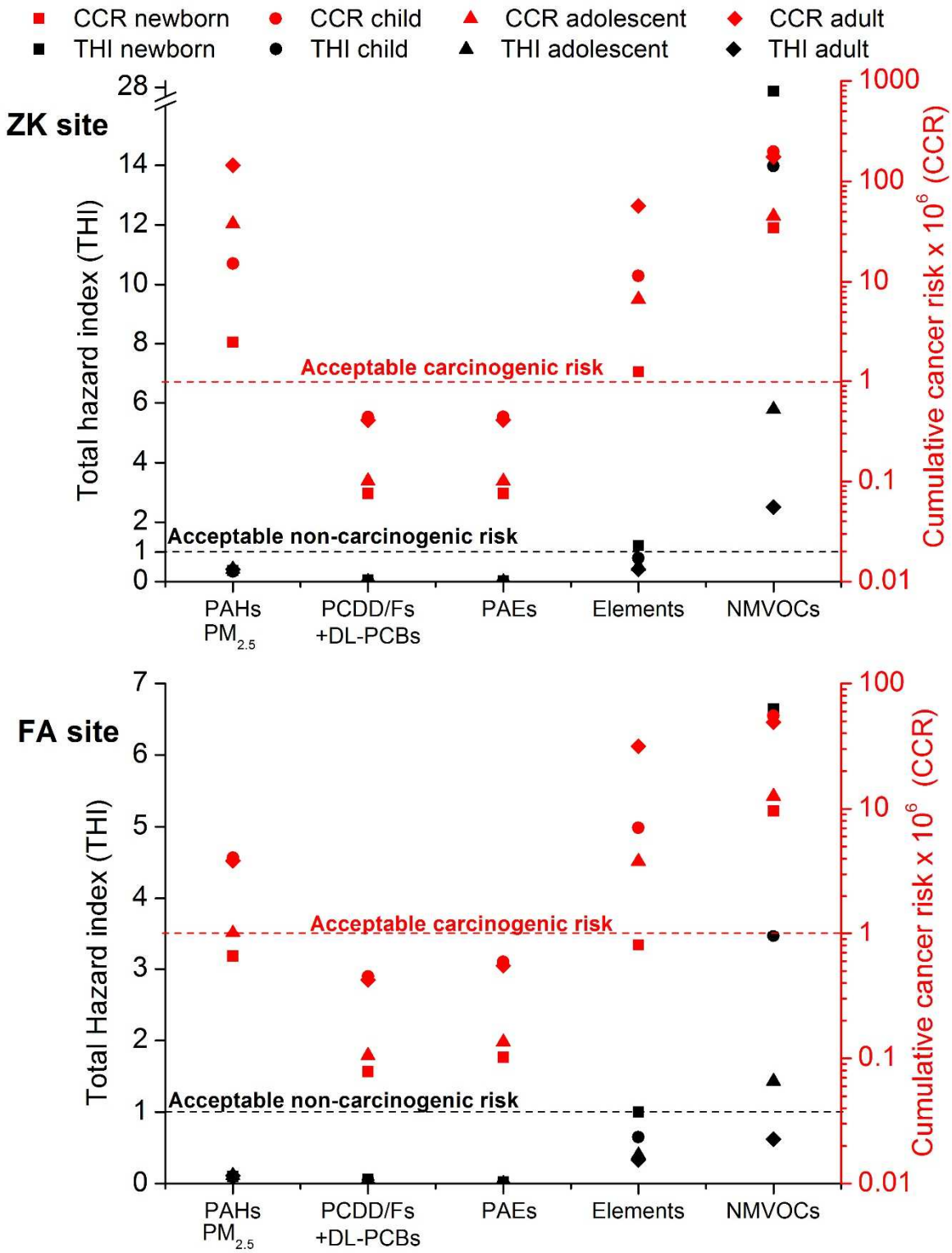

Fig. 2: Cumulative Cancer Risk (CCR) and Total hazard index (THI) values for polycyclic aromatic hydrocarbons (PAHs) in particulate phase, total PCDD/Fs and DL-PCBs, phthalates (PAEs), elements, and non-methane volatile organic compounds (NMVOCs) for the different age categories (newborns, children, adolescents, and adults) at Zouk (ZK) and Fiaa (FA) (HI values and Cumulative CR below 1 means acceptable risk).

547 Table 4: Hazard Index (HI) and cancer risk values (CR) for dermal, ingestion and inhalation pathways for the different age categories and
548 family of compounds at Zouk site

Age category	Class of compounds	Hazard Index (HI)			Cancer risk (CR)		
		Dermal	Ingestion	Inhalation	Dermal	Ingestion	Inhalation
Newborns	PAHs	-	6.4×10^{-2}	3.1×10^{-1}	3.8×10^{-7}	2.0×10^{-6}	9.4×10^{-8}
	PCDD/Fs + DL-PCBs	-	5.8×10^{-2}	3.3×10^{-5}	-	7.5×10^{-8}	7.1×10^{-10}
	PAEs	-	1.8×10^{-2}	-	-	7.2×10^{-8}	3.9×10^{-9}
	Elements	5.7×10^{-2}	2.2×10^{-1}	9.3×10^{-1}	3.6×10^{-8}	1.1×10^{-7}	1.1×10^{-6}
	NMVOCs	-	2.7×10^{1}	7.2×10^{-2}	-	3.4×10^{-5}	1.6×10^{-7}
Children	PAHs	-	3.3×10^{-2}	3.1×10^{-1}	3.2×10^{-6}	1.1×10^{-5}	6.7×10^{-7}
	PCDD/Fs + DL-PCBs	-	3.0×10^{-2}	3.3×10^{-5}	-	4.3×10^{-7}	7.9×10^{-9}
	PAEs	-	9.5×10^{-3}	-	-	4.1×10^{-7}	2.8×10^{-8}
	Elements	4.4×10^{-2}	1.1×10^{-1}	6.3×10^{-1}	3.0×10^{-7}	6.3×10^{-7}	1.1×10^{-5}
	NMVOCs	-	1.4×10^{1}	7.2×10^{-2}	-	2.0×10^{-4}	1.8×10^{-6}
Adolescents	PAHs	-	1.4×10^{-2}	4.1×10^{-1}	1.1×10^{-6}	2.5×10^{-6}	2.1×10^{-7}
	PCDD/Fs + DL-PCBs	-	1.2×10^{-2}	4.4×10^{-5}	-	9.6×10^{-8}	5.7×10^{-9}
	PAEs	-	3.9×10^{-3}	-	-	9.2×10^{-8}	8.6×10^{-9}
	Elements	2.6×10^{-2}	4.7×10^{-2}	4.4×10^{-1}	9.9×10^{-8}	1.4×10^{-7}	6.5×10^{-6}
	NMVOCs	-	5.7×10^{0}	9.6×10^{-2}	-	4.4×10^{-5}	1.3×10^{-6}
Adults	PAHs	-	5.7×10^{-3}	4.1×10^{-1}	3.4×10^{-6}	9.5×10^{-6}	1.6×10^{-6}
	PCDD/Fs + DL-PCBs	-	5.2×10^{-3}	4.4×10^{-5}	-	3.6×10^{-7}	5.1×10^{-8}
	PAEs	-	1.7×10^{-3}	-	-	3.5×10^{-7}	6.5×10^{-8}
	Elements	9.5×10^{-3}	2.0×10^{-2}	3.9×10^{-1}	3.2×10^{-7}	5.2×10^{-7}	5.6×10^{-5}
	NMVOCs	-	2.4×10^{0}	9.6×10^{-2}	-	1.6×10^{-4}	1.1×10^{-5}

549

Table 5: Hazard Index (HI) and cancer risk values (CR) for dermal, ingestion, and inhalation pathways for the different age categories and family of compounds at Fiaa site

Age category	Class of compounds	Hazard index (HI)			Cancer risk (CR)		
		Dermal	Ingestion	Inhalation	Dermal	Ingestion	Inhalation
Newborns	PAHs	-	1.7×10^{-2}	8.3×10^{-2}	1.0×10^{-7}	5.3×10^{-7}	2.5×10^{-8}
	PCDD/Fs + DL-PCBs	-	6.0×10^{-2}	3.4×10^{-5}	-	7.8×10^{-8}	7.4×10^{-10}
	PAEs	-	2.4×10^{-2}	-	-	9.7×10^{-8}	5.2×10^{-9}
	Elements	4.0×10^{-2}	1.7×10^{-1}	7.9×10^{-1}	3.6×10^{-8}	1.1×10^{-7}	6.6×10^{-7}
	NMVOCs	-	6.6×10^{0}	1.6×10^{-2}	-	9.5×10^{-6}	4.7×10^{-8}
Children	PAHs	-	8.8×10^{-3}	8.3×10^{-2}	8.6×10^{-7}	3.0×10^{-6}	1.8×10^{-7}
	PCDD/Fs + DL-PCBs	-	3.1×10^{-2}	2.0×10^{-5}	-	4.4×10^{-7}	8.1×10^{-9}
	PAEs	-	1.3×10^{-2}	-	-	5.5×10^{-7}	3.7×10^{-8}
	Elements	3.1×10^{-2}	8.9×10^{-2}	5.3×10^{-1}	3.1×10^{-7}	6.2×10^{-7}	6.1×10^{-6}
	NMVOCs	-	3.4×10^{0}	1.6×10^{-2}	-	5.5×10^{-5}	5.1×10^{-7}
Adolescents	PAHs	-	3.6×10^{-3}	1.1×10^{-1}	2.8×10^{-7}	6.8×10^{-7}	5.6×10^{-8}
	PCDD/Fs + DL-PCBs	-	1.3×10^{-2}	4.5×10^{-5}	-	9.9×10^{-8}	5.9×10^{-9}
	PAEs	-	5.2×10^{-3}	-	-	1.2×10^{-7}	1.2×10^{-8}
	Elements	1.8×10^{-2}	3.7×10^{-2}	3.5×10^{-1}	1.0×10^{-7}	1.4×10^{-7}	3.6×10^{-6}
	NMVOCs	-	1.4×10^{0}	2.2×10^{-2}	-	1.2×10^{-5}	3.7×10^{-7}
Adults	PAHs	-	1.5×10^{-3}	1.1×10^{-1}	9.0×10^{-7}	2.5×10^{-6}	4.2×10^{-7}
	PCDD/Fs + DL-PCBs	-	5.4×10^{-3}	4.5×10^{-5}	-	3.7×10^{-7}	5.2×10^{-8}
	PAEs	-	2.2×10^{-3}	-	-	4.6×10^{-7}	8.6×10^{-8}
	Elements	6.6×10^{-3}	1.5×10^{-2}	3.1×10^{-1}	3.2×10^{-7}	5.2×10^{-7}	3.1×10^{-5}
	NMVOCs	-	6.0×10^{-1}	2.2×10^{-2}	-	4.6×10^{-5}	3.3×10^{-6}

553 3.2.5 Non-methane Volatile organic compounds (NMVOCs)

554 The non-carcinogenic risk related with exposure to the eleven NMVOCs was
555 investigated. Detailed results are shown in **Table S4** and **Table S5**. The species were
556 studied for inhalation and oral ingestion pathways since no RfD_i or CSF_i were found for
557 dermal contact. The ingestion pathway appears as the major exposure route for the
558 different species, contributing to more than 80% of the total HI values at both sites.
559 Heptane and cyclohexane show respectively the highest and the lowest non-
560 carcinogenic risk.

561 A close examination considering the three routes together revealed that benzene is the
562 second contributor to total HI. Benzene and heptane explain at least 78 to 94% of the
563 total HI values for NMVOCs at both sites. The total HQ_i value for benzene and n-heptane
564 exceeded the threshold limit of 1 for non-carcinogenic risk for all age categories at ZK
565 and for newborns and children at FA **(Table S6** and **S7).** Additionally, toluene gave total
566 HQ_i value exceeding the limit at ZK for newborns (1.56). Other species such as
567 tetrachloroethene, ethylbenzene and xylenes recorded total HQ_i values under the
568 threshold limit meaning that these compounds could be associated with acceptable non-
569 carcinogenic effects. The total HI for the NMVOCs class of compounds showed values
570 higher than 1 for all the age categories at ZK and for newborns, children, and
571 adolescents at FA. It ranged between 26.8 (newborns) and 2.9 (adults) at ZK, and
572 between 6.6 (newborns) and 0.8 (adults) at FA **(Fig. 2).** Up to our knowledge, most of
573 the studies focused mainly on BTEX and there have been no studies reporting hazard
574 index values for both ingestion and inhalation pathways despite the higher contribution
575 of the ingestion compared to the inhalation pathway as shown in this study. Considering
576 NMVOCs measured as a whole, the HI values presented in this study for the inhalation
577 pathway (2×10^{-2} – 8×10^{-2}) were similar to the ones reported in Tehran, a Middle
578 Eastern city (in the order of 10^{-2} - 10^{-3}) with a population of approximately 9 million
579 (Miri et al., 2016).

580 5 NMVOCs (1,3-butadiene, n-hexane, tetrachloroethene, benzene and ethylbenzene)
581 were considered for evaluating the cancer risk through two exposure pathways:
582 inhalation and oral ingestion **(values in Tables S4** and **S5)**. Among these compounds,
583 1,3-butadiene and benzene were classified as carcinogenic to humans according to the
584 IARC causing leukemia. While tetrachloroethene was also classified by IARC as potential

585 human carcinogen based on limited evidence and might cause bladder cancer. The
586 carcinogenic risk values from the ingestion pathway were at least ten times higher than
587 the inhalation route. Additionally, benzene was associated with the highest total CR_i
588 with values in the order of 10^{-5} - 10^{-4} at ZK and 10^{-6} - 10^{-5} at FA. These cancer risk values
589 exceeded the acceptable USEPA threshold limit for both sites and for the different age
590 categories. Furthermore, 1,3-butadiene and ethylbenzene also exhibited values higher
591 than 10^{-6} at ZK site with total CR_i values for the different age categories in the following
592 order: child (2.8 x 10^{-5} for 1,3-butadiene and 2.6 x 10^{-5} for ethylbenzene)> adult (2.4 x
593 10^{-5} for 1,3-butadiene and 2.4 x 10^{-5} for ethylbenzene)> adolescent (6.3 x 10^{-6} for 1,3-
594 butadiene and 6.0 x 10^{-6} for ethylbenzene)> newborn (4.9 x 10^{-6} for 1,3-butadiene and
595 4.5 x 10^{-6} for ethylbenzene). Ethylbenzene gave values higher than the threshold limit at
596 FA for children and adults only (~4 x 10^{-6}). The lifetime cancer risk values at ZK for
597 benzene (1.2 x 10^{-4}), 1,3-butadiene (2.3 x 10^{-5}) and ethylbenzene(2.2 x 10^{-5}) reported in
598 this study were higher than the values reported for a suburban site in Lebanon during
599 winter (1.3 x 10^{-5}, 1.2 x 10^{-5}, 6 x 10^{-6}, for the same compounds respectively) and summer
600 (1.6 x 10^{-5}, 8 x 10^{-6}, 3 x 10^{-6})(Dhaini et al., 2017). However, n-hexane and
601 tetrachloroethene showed values lower than the acceptable threshold implying a
602 moderate cancer risk for these species. The cumulative cancer risk for all NMVOCs
603 measured exceeded the permissible risk level at both sites and for the different age
604 categories and varied from 3.5 x 10^{-5} for newborns to 1.8 x 10^{-4} for adults and from 1 x
605 10^{-5} for newborns to 6 x 10^{-5} for children at ZK and FA, respectively. The lifetime cancer
606 risk was 170 (1.7 x 10^{-4}) and 46 (4.6 x 10^{-5}) times higher than the USEPA limit at ZK and
607 FA, respectively. This is mainly due to high concentrations of benzene and ethylbenzene
608 at both sites, and additionally 1,3-butadiene at ZK. Benzene and 1,3-butadiene were
609 known to be mainly generated from combustion sources leading to the assumption that
610 fuel combustion emissions were considered as significant contributors to the cumulative
611 cancer risk calculated for NMVOCs (Salameh et al., 2015; 2016). At least 70% of the
612 additional cancer cases (120 at ZK and 42 at FA per million habitats based on LCR
613 calculations) were caused by the exposure to high concentrations of benzene at both
614 sites, might be highly emitted from traffic gasoline combustion. These numbers can be
615 reduced by implementing strict vehicle emission control measures.

616

617 3.3 Health risk comparison between the classes of compounds and limitations of the
618 study

619 As observed in **Fig. 2,** total HI values related to the non-cancer risk were significantly
620 higher than the acceptable limit of 1 for NMVOCs for the different age categories at both
621 sites (except for adults at FA), and for elements for adults. These compounds might pose
622 non-carcinogenic health problems to humans like neurological effects, liver and kidney
623 toxicities, lymphocyte count decrease, etc. (Dhaini et al., 2017). The major compounds
624 contributing to the non-carcinogenic risk were mainly n-heptane, benzene and toluene
625 for NMVOCs. While in the particulate phase, $B[a]P_{eq}$, Mn, Pb, V and Ni were the major
626 contributors despite their values of total HQ_i less than 1.Conversely, PAHs, dioxins,
627 furans, dioxin-like polychlorobiphenyls, and phthalates have an acceptable non-cancer
628 risk since their total HI values were lower than the USEPA limit. When comparing the
629 different families of compounds, NMVOCs exhibited the highest values of total HI for the
630 different age categories, followed by elements, $PM_{2.5}$-bound PAHs, phthalates, and finally
631 PCDD/Fs and DL-PCBs (**Fig. 2)**.

632 For the cancer risk assessment, cumulative CR values for PAHs in the particulate phase,
633 elements and NMVOCs exhibited values higher than the threshold limit of 10^{-6} proposed
634 by the USEPA. The major contributors to cancer risk were benzene, ethylbenzene and
635 1,3-butadiene (at ZK site) in the gas phase, while PAHs, Ni, Cr(VI), Co, and As were the
636 main components in the particulate phase. Additionally, a similar order to the total
637 hazard index was observed for the cumulative cancer risk of the different families of
638 compounds except for $PM_{2.5}$-bound PAHs that showed CCR values higher than elements
639 **(Fig. 2)**. Species that have CCR values higher than the threshold limit were mainly
640 associated to combustion sources such as vehicular emissions, biomass burning, and
641 HFO combustion leading to additional cancer cases for the different families of species.
642 This emphasizes on the relevance of reducing the concentrations of the species by
643 adopting an effective air quality management plan, implementing a national
644 transportation system in urban areas to reduce traffic emissions (Abdallah et al., 2018)
645 and shifting to cleaner energy instead of traditional biomass and fuel combustion. This
646 study used a comprehensive and well-defined calculation method for the carcinogenic
647 and non-carcinogenic risk assessment due to exposure to different classes of compounds
648 considered individually. However, the cancer and non-cancer risks for the classes of

compounds were not summed since there were no available methods for the calculation of health risk hazards due to exposure to total air contaminants. These were considered as limitations of the health assessment studies which assume independence between toxic actions within and across compounds. Actually, the interactions between the different contaminants on health, defined as cocktail effect reflecting synergism or antagonism action is not well defined as it still remains unsolved. Additionally, uncertainties accompany risk assessment studies especially in the choice of exposure parameters and reference values.

4 Conclusions

This study provided for the first time an extensive health risk assessment considering different classes of compounds such as phthalates, dioxins, furans, dioxin-like polychlorobiphenyls, PAHs and elements in $PM_{2.5}$, as well as NMVOCs-according to age categories and different exposure pathways.

The non-carcinogenic risk estimated for the different components showed a risk from benzene and n-heptane exposure. Additionally, this was also observed for the sum of elements where Mn, Pb, V and Ni were the major contributors to the total hazard index. PAHs, phthalates, PCDD/Fs, and DL-PCBs exhibited lower values and present acceptable non-carcinogenic health risks. As for the carcinogenic risk, CR for different species such as benzene, 1,3-butadiene (at Zouk site), ethylbenzene, $PM_{2.5}$-B[a]P_{eq}, As, Co, Cr(VI), Ni, and V showed values higher than the threshold limit of 10^{-6} meaning that these compounds present a carcinogenic risk to different age categories. These compounds were mainly associated to different combustion sources such as vehicular emissions, biomass burning and HFO combustion. The results from this study are of major importance to assess the impact of the different contaminants on the human health. Additionally, health risk assessment results should be combined to source apportionment studies in order to better relate health impact of the different sources of pollutants released in the atmosphere.

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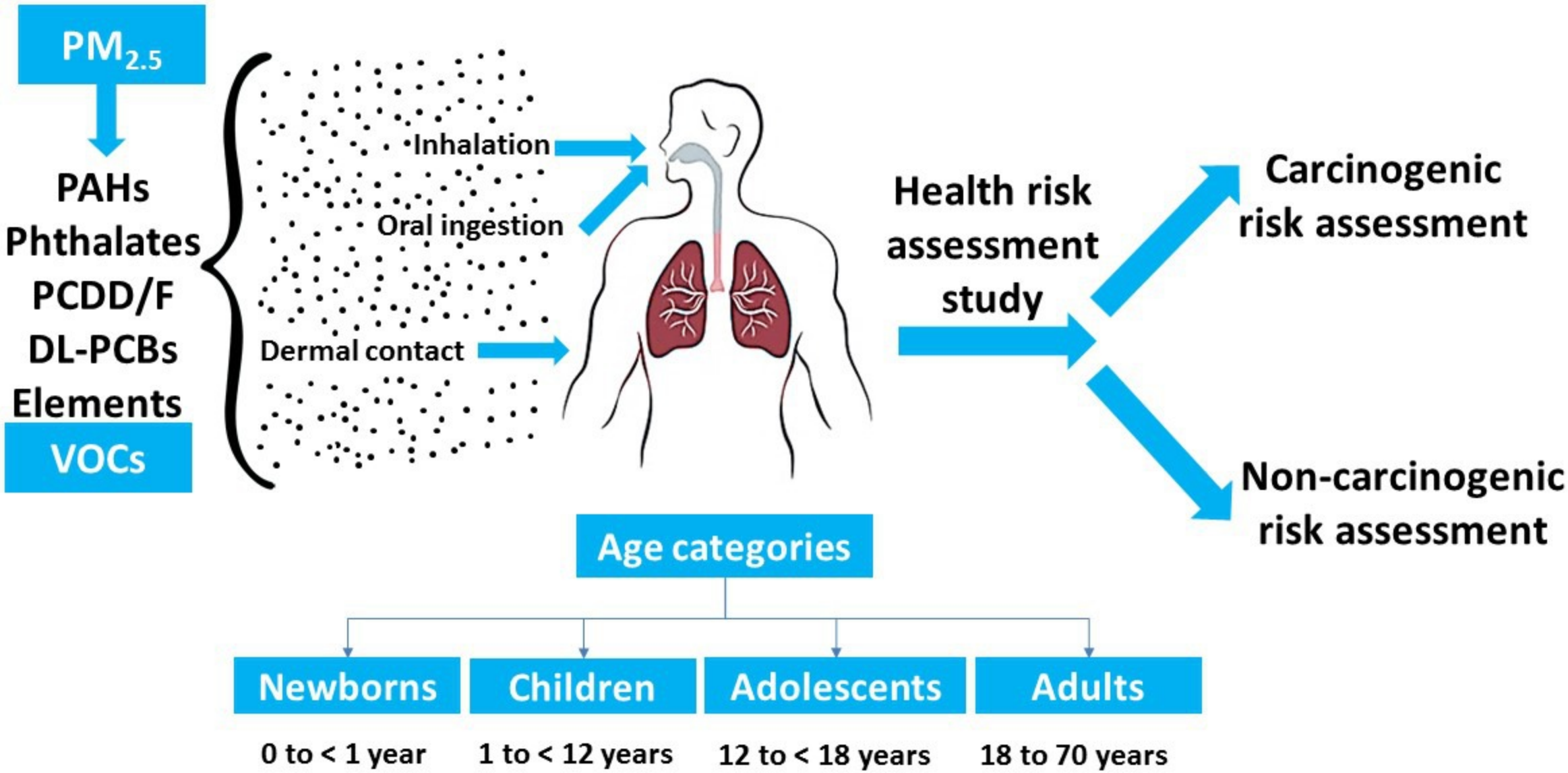
PM2.5
PAHs
Phthalates
PCDD/F
DL-PCBs
Elements
VOCs
Inhalation
Oral ingestion
Dermal contact
Health risk assessment study
Carcinogenic risk assessment
Non-carcinogenic risk assessment
Age categories
Newborns
Children
Adolescents
Adults
0 to < 1 year
1 to < 12 years
12 to < 18 years
18 to 70 years