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# Human health risk assessment for PAHs, phthalates, elements, PCDD/Fs, and DL-PCBs in PM<sub>2.5</sub> and for NMVOCs in two East-Mediterranean urban sites under industrial influence

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1           **Human health risk assessment for PAHs, phthalates, elements,**  
2           **PCDD/Fs, and DL-PCBs in PM<sub>2.5</sub> and for NMVOCs in two East-**  
3           **Mediterranean urban sites under industrial influence**

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19 **Abstract:**

20 This study evaluates the carcinogenic and the non-carcinogenic health risks related to  
21 non-methane volatile organic compounds (NMVOCs) and elements, dioxins, furans,  
22 dioxin-like polychlorobiphenyls, phthalates and polycyclic aromatic hydrocarbons in  
23 PM<sub>2.5</sub> samples collected during a one-year field campaign in two urban industrial areas  
24 in the East Mediterranean region. The health risk was assessed for the three exposure  
25 pathways (ingestion, inhalation and dermal contact) and for different age categories  
26 (newborns, children, adolescents, and adults). The non-carcinogenic risk calculated for  
27 the different species showed that benzene and n-heptane explained 78 to 94% of the  
28 total hazard index for NMVOCs at both sites. The total HI for NMVOCs varied between  
29 2.9 and 26.8 at ZK and between 0.8 and 6.6 at FA for adults and newborns respectively  
30 exceeding the recommended USEPA limit of 1 for most age categories. PM<sub>2.5</sub>-bound  
31 elements had values higher than the recommended USEPA limit for newborns with Mn,  
32 Pb, V, and Ni as the major contributors. The other species under study presented  
33 moderate risk values. The lifetime cancer risk due only to the exposure to NMVOCs was  
34 170 and 46 times higher than the threshold limit at ZK and FA, respectively. PM<sub>2.5</sub>-bound  
35 PAHs, As, Co, Cr(VI), Ni and V concentrations showed lifetime cancer risk exceeding the  
36 threshold limit of 10<sup>-6</sup> with 58 and 28 additional cancer cases per million habitats at ZK  
37 and FA, respectively. To our knowledge, this assessment is a first evaluating the health  
38 risk of several classes of compounds from both particulate and gas phases.

39 **Keywords:** PM<sub>2.5</sub>, NMVOCs, carcinogenic risk, exposure pathways, non-carcinogenic  
40 risk.

## 41 **1 Introduction**

42 According to the World Health Organization (WHO), air pollution is responsible for  
43 about 11% of the worldwide deaths annually, and therefore considered as the biggest  
44 environmental threat to human health (WHO, 2016). **The international Agency for  
45 Research on Cancer (IARC) classified outdoor air pollution as well as different chemicals  
46 found in the atmosphere (chromium (VI), arsenic, cadmium, nickel, benzo[a]pyrene,  
47 benzene, 1,3-butadiene, 2,3,4,7,8-pentachlorodibenzofuran, and 2,3,7,8-  
48 tetrachlorodibenzo-para-dioxin) as Group I “carcinogenic to humans” (Loomis et al.,  
49 2013).**

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

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

71 The East Mediterranean is the third region with the highest ambient and household air  
72 pollution attributable death rate (age-standardized), scoring an average of 125 per  
73 100,000 population in 2016, after Africa (181) and South East Asia (166), while this  
74 number could tend to rise in the future with the increase of the aerosol concentrations  
75 due to climate change (Lelieveld et al., 2014). Lebanon, an East Mediterranean country,  
76 faces extensive urbanization with heavy traffic, installation of diesel private generators  
77 within neighborhoods with no law enforcement, dust events, open waste burning, along  
78 with emissions from different types of industries (Waked et al., 2012). The air quality in  
79 the country is considered unsafe as exceedances in PM<sub>2.5</sub> concentrations above WHO  
80 guidelines of 25 µg/m<sup>3</sup> are recorded (MoE, 2017) with more than 170 days in 2014 in  
81 Greater Beirut area. In order to evaluate the impact of air quality on human health in  
82 Lebanon, different approaches were tackled. Epidemiological studies were carried out to  
83 determine the association between outdoor air quality and different diseases such as  
84 chronic obstructive pulmonary diseases, chronic bronchitis, cardiovascular diseases and  
85 lung cancer (Aoun et al., 2013; Kobrossi et al., 2002; Nakhlé et al., 2015; Nasser et al.,  
86 2015; Salameh et al., 2012). These studies were based on questionnaires filled by  
87 hospital patients and air quality data. More recently, studies dealing with PM<sub>2.5</sub> from  
88 different areas in Lebanon (Abbas et al., 2019; Badran et al., 2020; Borgie et al., 2015;  
89 Melki et al., 2017) evidenced, on pulmonary cells *in vitro*, toxic effects of PM depending  
90 on the size and the composition of the particles.

91 PM atmospheric concentration is a widely used indicator of the air quality level.  
92 However, a high share of PM mass consists of compounds with low toxicity such as  
93 ammonium, sulfate, nitrate, sea salt, and crustal dust elements (Borm et al., 2007). On  
94 the other hand, species that barely contribute to PM mass such as transition metals and  
95 organic species can have a major impact on the aerosols' toxicity (Bandowe and Meusel,  
96 2017; Chen and Lippmann, 2009). Among organic species, phthalates and PCDD/Fs are  
97 considered toxic to humans and were identified as endocrine disruptors and  
98 contributing to reproductive toxicity (Marquès and Domingo, 2019; Tsatsakis et al.,  
99 2019). Rengarajan et al. (2015) reported that the long-term exposure to mixtures of  
100 PAHs can increase the risks of mainly lung and skin, but also bladder and  
101 gastrointestinal cancers. That is why it is important to evaluate the risk of exposure to  
102 PM not as whole but based on its chemical composition. Therefore, most of the studies  
103 found in the literature focused on the evaluation of the health risk related to the

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

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

117

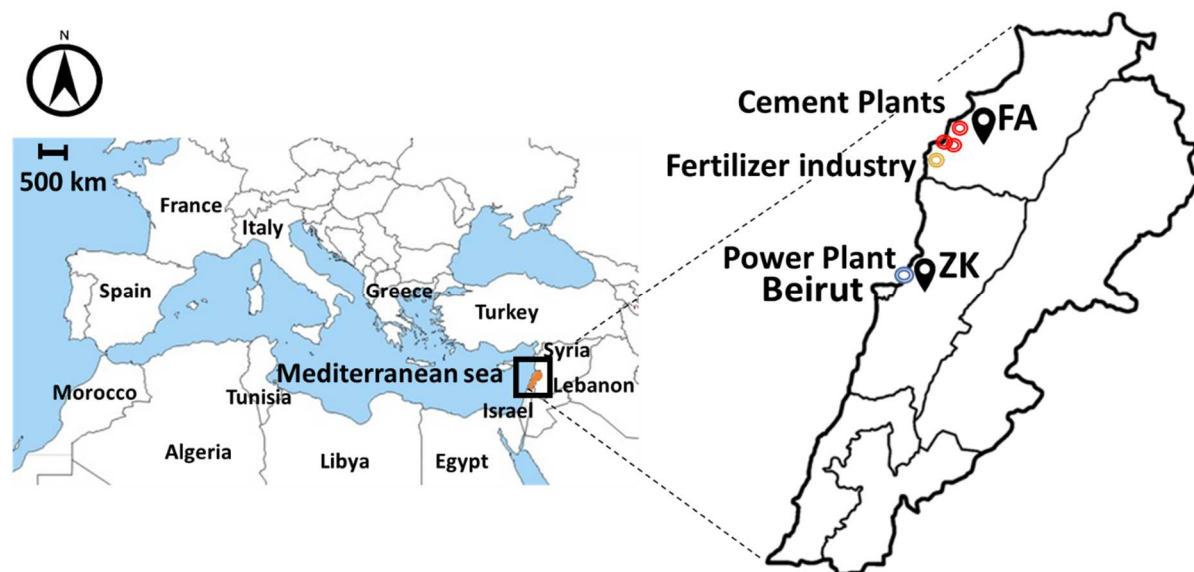
## 118 **2 Materials and methods**

### 119 **2.1 Site description and sample collection**

120 The description of the sampling sites was detailed elsewhere (Fadel et al., 2021) and will  
121 be shortly presented here. PM<sub>2.5</sub> and NMVOC samples were collected in two urban-  
122 industrial sites in Lebanon: Zouk Mikael (33°57'57.07"N 35°37'09.46"E) at a rooftop of a  
123 15 m- residential building, and Fiaa in Chekka region (34°20'47.8"N 35°47'14.0"E)  
124 collocated with the ambient air quality monitoring station of the Lebanese Ministry of  
125 Environment. The locations of the sites were chosen taking into account the topography,  
126 the existence of residents, micro siting (buildings, traffic, etc.) to represent the air  
127 quality background in the study areas.

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

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



136  
137 Fig. 1 Location of the two sampling sites: Zouk Mikael (ZK) and Fiaa (FA) and the  
138 position of the nearby industries

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

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

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

153

## 154 2.2 Analysis of NMVOCs

155 NMVOCs were chemically extracted from cartridge by adding 2 mL of CS<sub>2</sub> 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<sup>3</sup>) were calculated  
169 using Eq.1 provided by the manufacturer of Radiello diffusive passive sampler  
170 (Fondazione Salvatore Maugeri- IRCCS, Padova, Italy):

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

172 Where C is the average concentration in µg/m<sup>3</sup>, m is the mass of analyte in µg, t is the  
173 exposure time in minutes, and Q<sub>k</sub> 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<sub>2.5</sub>

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



185 method described in Fadel et al. (2021). Major and trace elements were **extracted by a**  
186 **mixture of HNO<sub>3</sub>/HF/HClO<sub>4</sub> and** quantified using Inductively Coupled Plasma-Atomic  
187 Emission Spectrometry (ICP-AES, iCAP 6000 series, Thermo Scientific, United Kingdom)  
188 for Al, Ba, Fe, Mn, Ni, Sr, Zn and Pb, and ICP-Mass Spectrometry (ICP-MS, Agilent 7900,  
189 Varian, United States of America) for elements such as As, Cd, Co, Sn, Cu, Cr, Sb, V, La, and  
190 Tl. The method is detailed in (Kfoury et al., 2016; Ledoux et al., 2006). The analytical  
191 procedure for elements analysis by ICP-AES and ICP-MS was validated considering  
192 standard reference materials: urban particulate matter NIST-SRM 1648a (National  
193 Institute of Standards and Technology, USA). Recovery rates varied between 89% and  
194 103% with an exception for Cr (76%). Polychlorinated Dibenzo-p-Dioxins (PCDDs),  
195 furans (PCDFs), and Dioxin-Like Polychlorinated Biphenyls (DL-PCBs) were analyzed by  
196 MicroPolluants Technology SA (Saint Julien Les Metz, France) based on the US  
197 Environmental Protection Agency (US EPA) methods 1613 and 1668 (Borgie et al.,  
198 2015). The apparatus used is a high-resolution gas chromatography coupled to a high-  
199 resolution mass spectrometer (HRGC/HRMS).

200

## 201 2.4 Toxic equivalent concentrations for PAHs and PCDD/Fs and DL-PCBs

### 202 2.4.1 2,3,7,8-TCDD equivalents

203 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) is known to be a human carcinogen,  
204 highly persistent and widespread environmental contaminant (NTP, 2016). A toxic  
205 equivalent factor (TEF<sub>i</sub>) was introduced to assign toxicity values for the different  
206 congeners (**PCDD/Fs and DL-PCBs**) based on the toxicity of 2,3,7,8-TCDD reported as 1  
207 (Van den Berg et al., 2006). The toxic equivalent “TEQ” representing an equivalent  
208 concentration of 2,3,7,8-TCDD is calculated following Eq. 2 by summing the product of  
209 the congeners’ concentration (**C<sub>i</sub>**) multiplied by its corresponding TEF values (TEF<sub>i</sub> -  
210 unitless) (**Table S1**).

$$211 \quad \text{TEQ} = \sum_{i=1}^n C_i \times \text{TEF}_i \quad (\text{Eq. 2})$$

### 212 2.4.2 Benzo[a]pyrene equivalents

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

$$217 \quad B[a]P_{eq} = \sum_{j=1}^n C_j \times TEF_j \quad (\text{Eq. 3})$$

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

221

## 222 2.5 Health Risk assessment

223 The health risk assessment was based on the Human health evaluation guidelines  
224 proposed by the United States Environmental Protection Agency (USEPA, 1989, 1991,  
225 2011) and was performed for different age categories: newborns (0 to <1 year), children  
226 (1 to <12 years), adolescents (12 to <18 years) and adults (18 to 70 years), and for three  
227 exposure pathways: inhalation, ingestion, and dermal absorption on exposed skin when  
228 data were available. The average concentrations of the species on the sampling period  
229 were considered for the health risk assessment assuming that they represent a typical  
230 yearly concentration of the pollutants in the atmosphere of the considered sites.

231

### 232 2.5.1 Non-cancer risk assessment

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

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

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

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

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

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

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

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

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

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

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

265

## 266 2.5.2 Cancer risk assessment

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

$$274 \quad CR_{i-inhalation} = EC_i \times IUR_i \quad \text{or} \quad CR_{i-ingestion/dermal} = LADD_i \times CSF_i \quad (Eq. 12)$$

275 When considering carcinogenic species, the exposure period during which the dose is  
276 averaged is usually lifetime or 70 years. Accordingly, the lifetime average daily dose  
277 ( $LADD_i$ ) calculation equations for the different exposure pathways take the form of (Eq.  
278 5 to 8) with lifetime (25,500 days) replacing the average timing ( $ED \times 365$  days) (USEPA,  
279 1989, 2011).

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

$$282 \quad Total \ CR_i = CR_{i-inhalation} + CR_{i-ingestion} + CR_{i-dermal} \quad (Eq. 13)$$

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

$$285 \quad CR_{inhalation/ingestion/dermal} = \sum_i CR_{i \text{ inhalation/ingestion/dermal}} \quad (Eq. 14)$$

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

$$289 \quad Cumulative \ CR = CCR = CR_{inhalation} + CR_{ingestion} + CR_{dermal} \quad (Eq. 15)$$

290

291

292

293 Table 1 : Exposure parameters for newborns, children, adolescents, and adults through  
 294 inhalation, ingestion, and dermal contact considered in this study for the health risk  
 295 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/m <sup>3</sup>					
Inhalation intake rate	$IR_{inh}$	m <sup>3</sup> /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	cm <sup>2</sup>	1300	3200	4650	5700	(USEPA, 2002)
Dermal adherence factor	AF	mg/cm <sup>2</sup>	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)

296

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

$$\begin{aligned}
 299 \quad LCR = & \text{Cumulative } CR_{Newborn} \times \frac{1}{70} + \text{Cumulative } CR_{child} \times \frac{11}{70} \\
 300 \quad & + \text{Cumulative } CR_{adolescent} \times \frac{6}{70} + \text{Cumulative } CR_{adult} \times \frac{52}{70} \quad (\text{Eq. 16})
 \end{aligned}$$

301

### 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<sub>2.5</sub> 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<sub>2.5</sub> along with concentrations of non-methane volatile  
 311 organic compounds (NMVOCs) during the entire sampling period

Compounds (ng/m <sup>3</sup> )	ZK site	FA site	Reference
<b>Total PM<sub>2.5</sub>-bound PAHs</b>	2.56 ± 2.5	0.95 ± 1.12	(Fadel et al., 2021)
<b>PM<sub>2.5</sub>-bound phthalates</b>			
dibutyl phthalate	37 ± 29	12.4 ± 11.6	(Fadel et al., 2021)
bis(2-ethylhexyl)phthalate	49 ± 17	66 ± 70	
<b>Major and trace elements</b>			
As	0.53 ± 0.28	0.49 ± 0.24	
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	This study
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	
<b>NMVOCs</b>			
1,3-butadiene	77.7 ± 70	<D.L.	
hexane	419 ± 720	150 ± 137	
tetrachloroethene	291 ± 115	58 ± 9	
benzene	4318 ± 5393	1526 ± 3340	
cyclohexane	398 ± 187	94 ± 200	This study
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	

312

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

321 **Table 3** presents PCDD/Fs, and DL-PCBs congener concentrations observed in ZK and  
322 FA sites in the particulate phase. The total average concentrations of PCDDs and PCDFs  
323 were 164 and 124 fg/m<sup>3</sup> at ZK and FA, respectively. OCDD and 1,2,3,4,6,7,8-HpCDF were  
324 the two dominant congeners among PCDD/Fs contributing to 49% and 46% at ZK and  
325 FA, respectively, followed by 1,2,3,4,6,7,8-HpCDD and OCDF. DL-PCB congeners are  
326 dominated by PCB 105 and PCB 118 accounting for 86% and 83% of total DL-PCBs at ZK  
327 and FA, respectively. Elements' concentrations varied at ZK from 0.03 ng/m<sup>3</sup> for Tl to  
328 593 ng/m<sup>3</sup> for Al, and from 0.03 ng/m<sup>3</sup> for Tl to 492 ng/m<sup>3</sup> for Al at FA (**Table 2**).  
329 Finally, for NMVOCs, the highest concentrations are observed for benzene, toluene,  
330 ethylbenzene and xylenes. Between these compounds, toluene showed the highest  
331 concentrations at both sites and 1,3-butadiene showed the lowest concentrations at ZK  
332 and was not detected at FA (**Table 2**).

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

336

### 337 3.2 Health risk evaluation

338 The hazard quotient (HQ<sub>i</sub>) and the cancer risk (CR<sub>i</sub>) associated with each species in the  
339 three exposure pathways for the different age categories are reported in **Table S4** and  
340 **Table S5** for ZK and FA, respectively. Furthermore, the total hazard index (THI) and the  
341 cumulative cancer risk (CCR) for PAHs and TCDD equivalents along with total HQ<sub>i</sub> and

342 total CR<sub>i</sub> 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<sup>3</sup>) in PM<sub>2.5</sub> samples at Zouk (ZK) and Fiaa (FA)

<b>Compounds (fg/m<sup>3</sup>)</b>	<b>Abbreviation</b>	<b>ZK site</b>	<b>FA site</b>
<b>PCDDs</b>		<b>81.1 ± 38.7</b>	<b>52.0 ± 21.7</b>
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
<b>TEQ for PCDD</b>		<b>1.03</b>	<b>0.34</b>
<b>PCDFs</b>		<b>82.5 ± 35.1</b>	<b>71.8 ± 36.7</b>
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
<b>TEQ for PCDF</b>		<b>4.45</b>	<b>4.96</b>
<b>DL-PCBs</b>		<b>454.8 ± 202</b>	<b>586.3 ± 128</b>
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
<b>TEQ for PCB</b>		<b>0.02</b>	<b>0.37</b>
<b>Total TEQ</b>		<b>5.49</b>	<b>5.67</b>

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<sub>eq</sub> concentrations can be estimated. This  
355 could be explained by the abundance of the PAHs with high contribution to the B[a]P<sub>eq</sub>  
356 value in the particulate phase. Consequently, only PM<sub>2.5</sub>-bound PAHs were studied.

357 The B[a]P<sub>eq</sub> value for the 16 PAHs in the particulate phase was 2.58 and 0.69 ng/m<sup>3</sup> 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<sub>eq</sub> value at ZK and 99% at FA showing that these compounds were the  
362 main contributors to PM<sub>2.5</sub>-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<sub>2.5</sub>-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<sub>2.5</sub>-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<sup>-6</sup>) except  
373 for newborns at FA with a trend of children (1.5 x 10<sup>-5</sup> at ZK and 4.1 x 10<sup>-6</sup> at FA) >  
374 adults (1.4 x 10<sup>-5</sup> at ZK and 3.9 x 10<sup>-6</sup> at FA) > adolescents (3.8 x 10<sup>-6</sup> at ZK and 1.0 x 10<sup>-6</sup>  
375 at FA) > newborns (2.5 x 10<sup>-6</sup> at ZK and 7 x 10<sup>-7</sup> 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<sup>-9</sup> - 7.7 x 10<sup>-9</sup> for children and 1.3 x 10<sup>-8</sup> - 1.4 x 10<sup>-8</sup> for adults)  
379 having lower B[a]P<sub>eq</sub> concentration (0.06-0.07 ng/m<sup>3</sup>) (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<sup>-6</sup> and 3.8 x 10<sup>-7</sup> at ZK, 5.3 x 10<sup>-7</sup> and 1.0 x 10<sup>-7</sup> at FA,  
382 respectively), which were at least three times higher than the inhalation pathway (9.4 x

383  $10^{-8}$  at ZK and  $2.5 \times 10^{-8}$  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 ( $4 \times 10^{-7} - 8.4 \times 10^{-5}$ ), 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<sub>2.5</sub>-  
387 bound PAHs concentrations (87-608 ng/m<sup>3</sup>) that were at least 80 times higher than the  
388 ones at our study (2.56 ng/m<sup>3</sup> at ZK and 0.95 ng/m<sup>3</sup> at FA).

389 The lifetime cancer risk values for the exposure to PAHs were  $1.4 \times 10^{-5}$  at ZK and  $3.7 \times$   
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<sub>i</sub> or RfD<sub>i</sub>  
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 ( $3.9 \times 10^{-9}$  at ZK and  $5.2 \times 10^{-9}$  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 ( $8.9 \times 10^{-10}$  -  $7.4 \times 10^{-9}$ ). 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 ( $54.8 \text{ ng/m}^3$ ) 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 ( $7.8 \text{ m}^3/\text{day}$  for a child and  
427  $12.8 \text{ m}^3/\text{day}$  for an adult). The lifetime cancer risk from exposure to BEHP was  $3.8 \times 10^{-7}$   
428 for ZK and  $5.1 \times 10^{-7}$  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  $\text{PM}_{2.5}$ -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  $4.0 \times 10^{-7}$  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  $1.2 \times 10^{-6}$ ,  $1.9 \times 10^{-6}$ ,  $1.7 \times 10^{-6}$ ,  
502  $1.5 \times 10^{-6}$  and  $5.2 \times 10^{-5}$  respectively, exceeding the safe limit of  $10^{-6}$ . Additionally,  
503 vanadium showed high cancer risk values of  $8.1 \times 10^{-6}$  and  $5.9 \times 10^{-6}$  for children and  
504 adolescents, respectively. While at FA, values exceeded the limit for As, Cr(VI) and V for

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

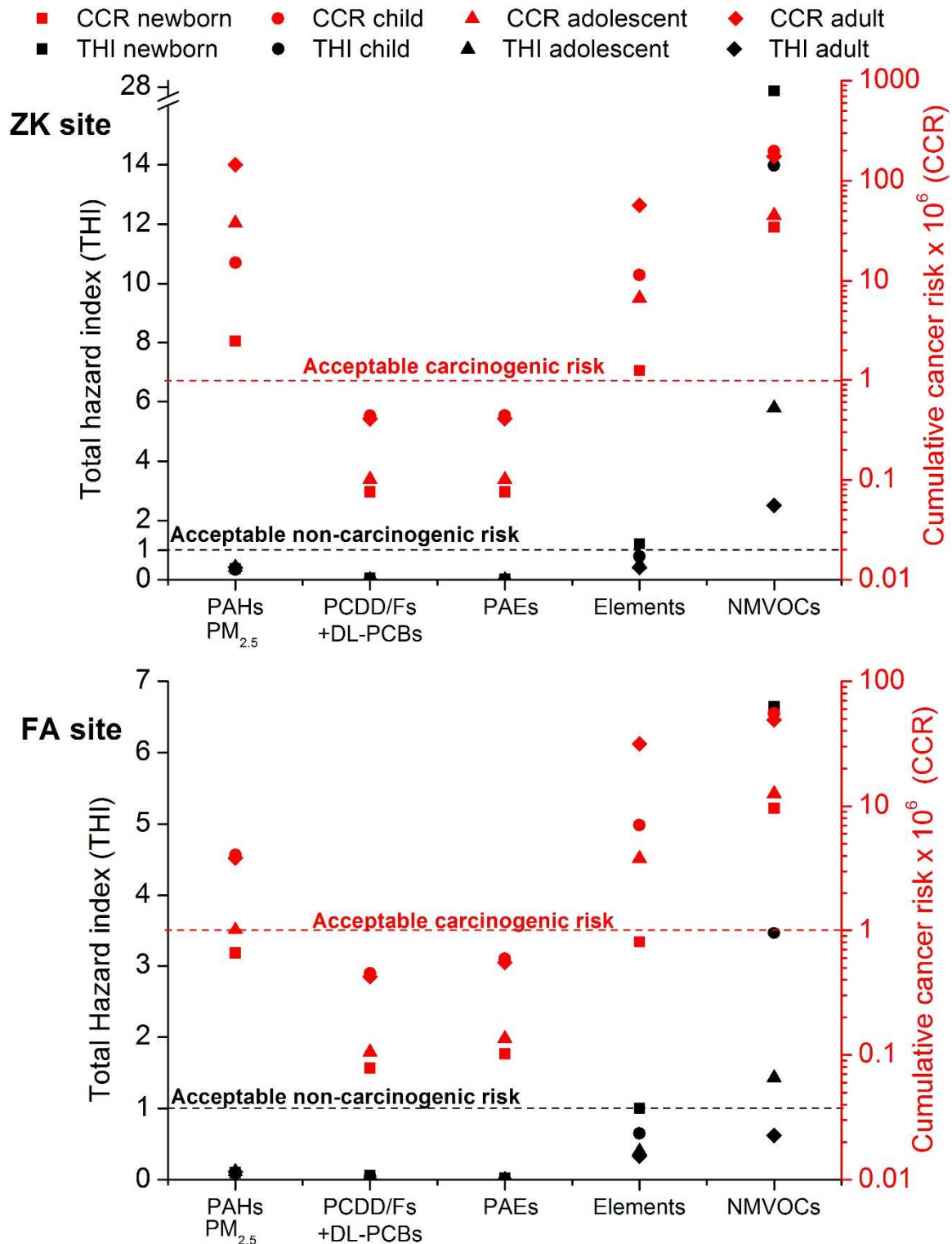
510 The cumulative cancer risk for the three exposure pathways was assessed (**Tables S6**  
511 **and S7**) and the problematic species were, from highest to lower values,  
512  $V > Cr(VI) > As > Co > Ni > Pb > Cd$  for FA and  $V > As > Cr(VI) > Co > Ni > Pb > Cd$  for ZK.  
513 Hence, V, As, and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

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

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

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

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



541

542 Fig. 2: Cumulative Cancer Risk (CCR) and Total hazard index (THI) values for polycyclic  
543 aromatic hydrocarbons (PAHs) **in particulate phase**, total PCDD/Fs and DL-PCBs,  
544 phthalates (PAEs), elements, and non-methane volatile organic compounds (NMVOCs)  
545 for the different age categories (newborns, children, adolescents, and adults) at Zouk  
546 (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	-	$6.4 \times 10^{-2}$	$3.1 \times 10^{-1}$	$3.8 \times 10^{-7}$	$2.0 \times 10^{-6}$	$9.4 \times 10^{-8}$
	PCDD/Fs + DL-PCBs	-	$5.8 \times 10^{-2}$	$3.3 \times 10^{-5}$	-	$7.5 \times 10^{-8}$	$7.1 \times 10^{-10}$
	PAEs	-	$1.8 \times 10^{-2}$	-	-	$7.2 \times 10^{-8}$	$3.9 \times 10^{-9}$
	Elements	$5.7 \times 10^{-2}$	$2.2 \times 10^{-1}$	$9.3 \times 10^{-1}$	$3.6 \times 10^{-8}$	$1.1 \times 10^{-7}$	$1.1 \times 10^{-6}$
	NMVOCs	-	$2.7 \times 10^1$	$7.2 \times 10^{-2}$	-	$3.4 \times 10^{-5}$	$1.6 \times 10^{-7}$
Children	PAHs	-	$3.3 \times 10^{-2}$	$3.1 \times 10^{-1}$	$3.2 \times 10^{-6}$	$1.1 \times 10^{-5}$	$6.7 \times 10^{-7}$
	PCDD/Fs + DL-PCBs	-	$3.0 \times 10^{-2}$	$3.3 \times 10^{-5}$	-	$4.3 \times 10^{-7}$	$7.9 \times 10^{-9}$
	PAEs	-	$9.5 \times 10^{-3}$	-	-	$4.1 \times 10^{-7}$	$2.8 \times 10^{-8}$
	Elements	$4.4 \times 10^{-2}$	$1.1 \times 10^{-1}$	$6.3 \times 10^{-1}$	$3.0 \times 10^{-7}$	$6.3 \times 10^{-7}$	$1.1 \times 10^{-5}$
	NMVOCs	-	$1.4 \times 10^1$	$7.2 \times 10^{-2}$	-	$2.0 \times 10^{-4}$	$1.8 \times 10^{-6}$
Adolescents	PAHs	-	$1.4 \times 10^{-2}$	$4.1 \times 10^{-1}$	$1.1 \times 10^{-6}$	$2.5 \times 10^{-6}$	$2.1 \times 10^{-7}$
	PCDD/Fs + DL-PCBs	-	$1.2 \times 10^{-2}$	$4.4 \times 10^{-5}$	-	$9.6 \times 10^{-8}$	$5.7 \times 10^{-9}$
	PAEs	-	$3.9 \times 10^{-3}$	-	-	$9.2 \times 10^{-8}$	$8.6 \times 10^{-9}$
	Elements	$2.6 \times 10^{-2}$	$4.7 \times 10^{-2}$	$4.4 \times 10^{-1}$	$9.9 \times 10^{-8}$	$1.4 \times 10^{-7}$	$6.5 \times 10^{-6}$
	NMVOCs	-	$5.7 \times 10^0$	$9.6 \times 10^{-2}$	-	$4.4 \times 10^{-5}$	$1.3 \times 10^{-6}$
Adults	PAHs	-	$5.7 \times 10^{-3}$	$4.1 \times 10^{-1}$	$3.4 \times 10^{-6}$	$9.5 \times 10^{-6}$	$1.6 \times 10^{-6}$
	PCDD/Fs + DL-PCBs	-	$5.2 \times 10^{-3}$	$4.4 \times 10^{-5}$	-	$3.6 \times 10^{-7}$	$5.1 \times 10^{-8}$
	PAEs	-	$1.7 \times 10^{-3}$	-	-	$3.5 \times 10^{-7}$	$6.5 \times 10^{-8}$
	Elements	$9.5 \times 10^{-3}$	$2.0 \times 10^{-2}$	$3.9 \times 10^{-1}$	$3.2 \times 10^{-7}$	$5.2 \times 10^{-7}$	$5.6 \times 10^{-5}$
	NMVOCs	-	$2.4 \times 10^0$	$9.6 \times 10^{-2}$	-	$1.6 \times 10^{-4}$	$1.1 \times 10^{-5}$

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

552

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

### 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<sub>i</sub> or CSF<sub>i</sub> 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<sub>i</sub> 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<sub>i</sub> value exceeding the limit at ZK for newborns (1.56). Other species such as  
567 tetrachloroethene, ethylbenzene and xylenes recorded total HQ<sub>i</sub> 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 ( $2 \times 10^{-2} - 8 \times 10^{-2}$ ) 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<sub>i</sub>  
588 with values in the order of 10<sup>-5</sup> - 10<sup>-4</sup> at ZK and 10<sup>-6</sup> - 10<sup>-5</sup> 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<sup>-6</sup> at ZK site with total CR<sub>i</sub> values for the different age categories in the following  
592 order: child (2.8 x 10<sup>-5</sup> for 1,3-butadiene and 2.6 x 10<sup>-5</sup> for ethylbenzene)> adult (2.4 x  
593 10<sup>-5</sup> for 1,3-butadiene and 2.4 x 10<sup>-5</sup> for ethylbenzene)> adolescent (6.3 x 10<sup>-6</sup> for 1,3-  
594 butadiene and 6.0 x 10<sup>-6</sup> for ethylbenzene)> newborn (4.9 x 10<sup>-6</sup> for 1,3-butadiene and  
595 4.5 x 10<sup>-6</sup> for ethylbenzene). Ethylbenzene gave values higher than the threshold limit at  
596 FA for children and adults only (~4 x 10<sup>-6</sup>). The lifetime cancer risk values at ZK for  
597 benzene (1.2 x 10<sup>-4</sup>), 1,3-butadiene (2.3 x 10<sup>-5</sup>) and ethylbenzene(2.2 x 10<sup>-5</sup>) reported in  
598 this study were higher than the values reported for a suburban site in Lebanon during  
599 winter (1.3 x 10<sup>-5</sup>, 1.2 x 10<sup>-5</sup>, 6 x 10<sup>-6</sup>, for the same compounds respectively) and summer  
600 (1.6 x 10<sup>-5</sup>, 8 x 10<sup>-6</sup>, 3 x 10<sup>-6</sup>)(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<sup>-5</sup> for newborns to 1.8 x 10<sup>-4</sup> for adults and from 1 x  
605 10<sup>-5</sup> for newborns to 6 x 10<sup>-5</sup> for children at ZK and FA, respectively. The lifetime cancer  
606 risk was 170 (1.7 x 10<sup>-4</sup>) and 46 (4.6 x 10<sup>-5</sup>) 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<sub>eq</sub>, Mn, Pb, V and Ni were the major  
626 contributors despite their values of total HQ<sub>i</sub> 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<sub>2.5</sub>-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<sup>-6</sup> 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<sub>2.5</sub>-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

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

657

#### 658 **4 Conclusions**

659 This study provided for the first time an extensive health risk assessment considering  
660 different classes of compounds such as phthalates, dioxins, furans, dioxin-like  
661 polychlorobiphenyls, PAHs and elements in PM<sub>2.5</sub>, as well as NMVOCs-according to age  
662 categories and different exposure pathways.

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

677

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696

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