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1 **Human health risk assessment for PAHs, phthalates, elements,**
2 **PCDD/Fs, and DL-PCBs in PM_{2.5} 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_{2.5} 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_{2.5}-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_{2.5}-bound
35 PAHs, As, Co, Cr(VI), Ni and V concentrations showed lifetime cancer risk exceeding the
36 threshold limit of 10⁻⁶ 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_{2.5}, 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 μm , 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_{2.5} concentrations above WHO
80 guidelines of 25 µg/m³ 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_{2.5} 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_{2.5} 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

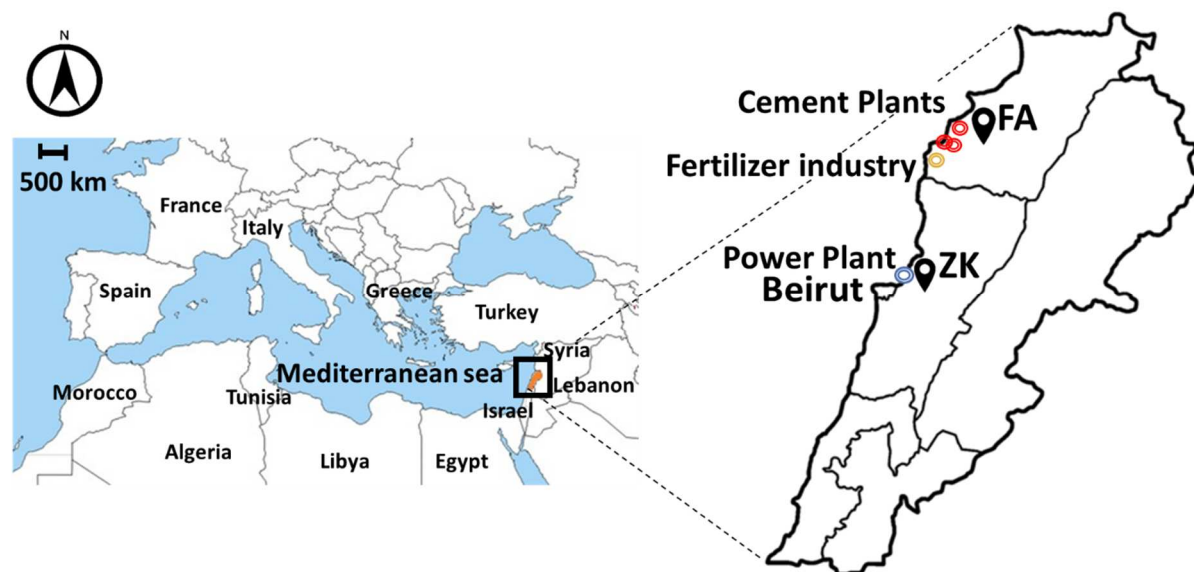
118 **2 Materials and methods**

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_{2.5} 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/km²) 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/km²) 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_{2.5} samples were collected on a 24-hour basis every third day at
140 ZK and FA, respectively from the 13th of December 2018 to the 15th of October 2019. The
141 sampling was carried out using high volume samplers operating at 30 m³/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 CS₂ 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/m³) 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/m³, 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

185 method described in Fadel et al. (2021). Major and trace elements were **extracted by a**
186 **mixture of HNO₃/HF/HClO₄ 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_i) 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_i**) multiplied by its corresponding TEF values (TEF_i -
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 (ng/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 mg/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**.

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

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):

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

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 ³					
Inhalation intake rate	IR_{inh}	m ³ /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 ²	1300	3200	4650	5700	(USEPA, 2002)
Dermal adherence factor	AF	mg/cm ²	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_{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/m ³)	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	
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	
NMVOCs			
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_{2.5}-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/m³). 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/m³). 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/m³ at
320 ZK, while at FA, the concentrations were 66 and 12.4 ng/m³, 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/m³ 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/m³ for Tl to
328 593 ng/m³ for Al, and from 0.03 ng/m³ for Tl to 492 ng/m³ 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_i) and the cancer risk (CR_i) 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_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/m³) in PM_{2.5} samples at Zouk (ZK) and Fiaa (FA)

Compounds (fg/m³)	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/m³ 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/m³) (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 2.5×10^{-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_{2.5}-
387 bound PAHs concentrations (87-608 ng/m³) that were at least 80 times higher than the
388 ones at our study (2.56 ng/m³ at ZK and 0.95 ng/m³ at FA).

389 The lifetime cancer risk values for the exposure to PAHs were 1.4×10^{-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_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 ($7.8 \text{ m}^3/\text{day}$ for a child and
427 $12.8 \text{ m}^3/\text{day}$ for an adult). The lifetime cancer risk from exposure to BEHP was 3.8×10^{-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 $\text{PM}_{2.5}$ -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 4.0×10^{-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 1.2×10^{-6} , 1.9×10^{-6} , 1.7×10^{-6} ,
502 1.5×10^{-6} and 5.2×10^{-5} respectively, exceeding the safe limit of 10^{-6} . Additionally,
503 vanadium showed high cancer risk values of 8.1×10^{-6} and 5.9×10^{-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 (7.1×10^{-6}), and an
508 urban site in Colombia (3.1×10^{-6}) (Galon-Negru et al., 2019; Liu et al., 2018; Ramírez et
509 al., 2020).

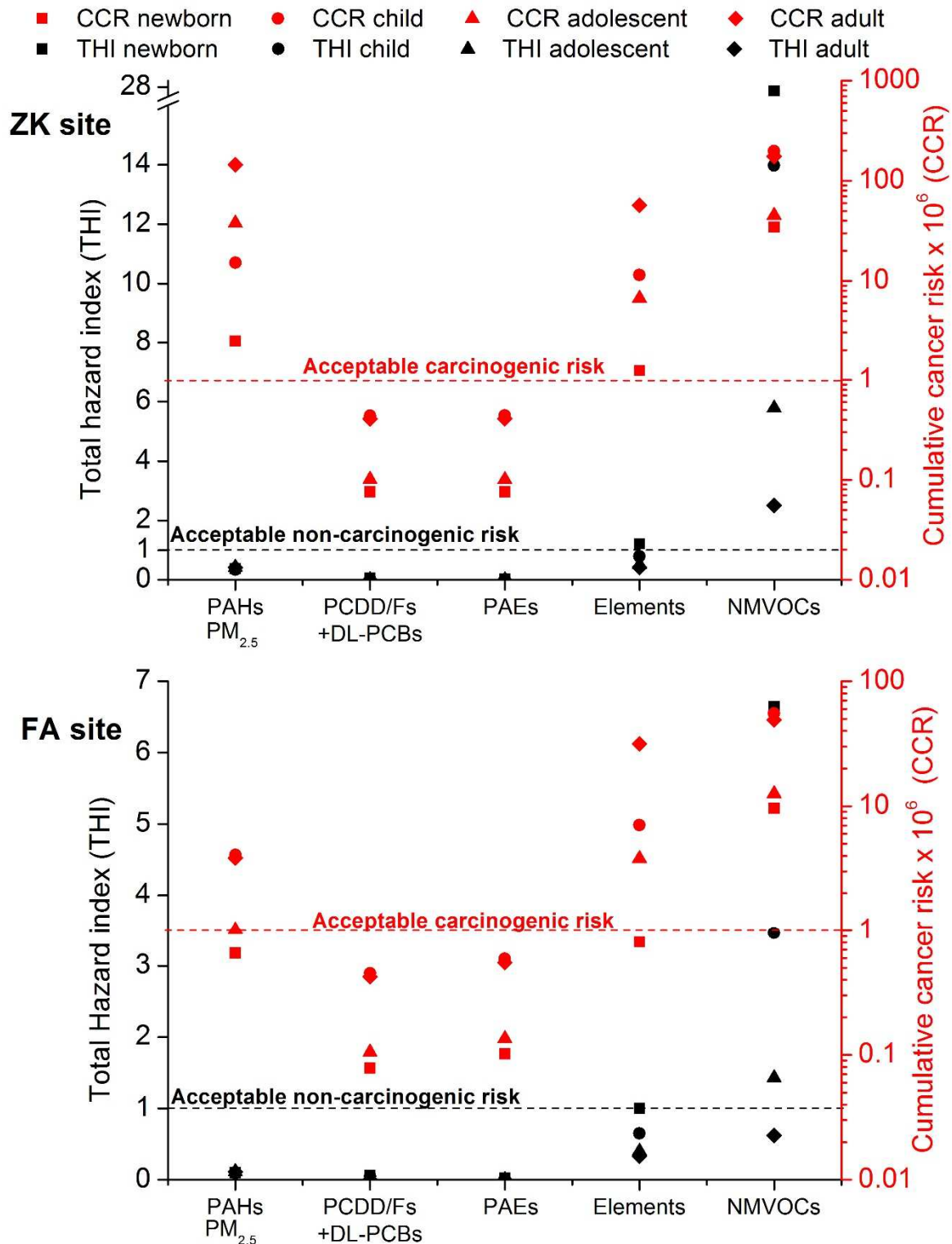
510 The cumulative cancer risk for the three exposure pathways was assessed (**Tables S6**
511 **and S7**) and the problematic species were, from highest to lower values,
512 $V > Cr(VI) > As > Co > Ni > Pb > Cd$ for FA and $V > As > Cr(VI) > Co > Ni > Pb > Cd$ for ZK.
513 Hence, V, As, and Cr(VI) were 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 (5.2×10^{-5} at ZK and 2.6×10^{-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 ng/m^3) and FA (13 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	-	6.4×10^{-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

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	-	1.7×10^{-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 ($2 \times 10^{-2} - 8 \times 10^{-2}$) 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_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

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_{2.5}, 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_{2.5}-B[a]P_{eq}, As, Co, Cr(VI), Ni,
669 and V showed values higher than the threshold limit of 10⁻⁶ 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

697 **References :**

698 Abbas, I., Badran, G., Verdin, A., Ledoux, F., Roumie, M., Lo Guidice, J.-M., Courcot, D.,
699 Garçon, G., 2019. In vitro evaluation of organic extractable matter from ambient PM_{2.5}
700 using human bronchial epithelial BEAS-2B cells: Cytotoxicity, oxidative stress, pro-
701 inflammatory response, genotoxicity, and cell cycle deregulation. *Environ. Res.* 171, 510-
702 522.<https://doi.org/10.1016/j.envres.2019.01.052>

703 Abdallah, C., Afif, C., Masri, N., Öztürk, F., Dağkuş Keleş, M., Sartelet, K., 2018. A first
704 annual assessment of air quality modeling over Lebanon using WRF/Polyphemus.
705 *Atmos. Pollut. Res.* 9.<https://doi.org/10.1016/j.apr.2018.01.003>

706 Akyüz, M., Çabuk, H., 2010. Gas–particle partitioning and seasonal variation of polycyclic
707 aromatic hydrocarbons in the atmosphere of Zonguldak, Turkey. *Sci. Total Environ.* 408,
708 5550-5558.<https://doi.org/10.1016/j.scitotenv.2010.07.063>

709 Alkoussa, S., Hulo, S., Courcot, D., Billet, S., Martin, P., 2020. Extracellular vesicles as
710 actors in the air pollution related cardiopulmonary diseases. *Crit. Rev. Toxicol.*
711 50.<https://doi.org/10.1080/10408444.2020.1763252>

- 712 Aoun, J., Saleh, N., Waked, M., Salamé, J., Salameh, P., 2013. Lung cancer correlates in
713 Lebanese adults: a pilot case-control study. *J Epidemiol Glob Health* 3, 235-
714 244.<https://doi.org/10.1016/j.jegh.2013.06.005>
- 715 Badran, G., Ledoux, F., Verdin, A., Abbas, I., Roumie, M., Genevray, P., Landkocz, Y., Lo
716 Guidice, J.-M., Garçon, G., Courcot, D., 2020. Toxicity of fine and quasi-ultrafine particles:
717 Focus on the effects of organic extractable and non-extractable matter fractions.
718 *Chemosphere* 243, 125440.<https://doi.org/10.1016/j.chemosphere.2019.125440>
- 719 Bai, L., Chen, W., He, Z., Sun, S., Qin, J., 2020. Pollution characteristics, sources and health
720 risk assessment of polycyclic aromatic hydrocarbons in PM_{2.5} in an office building in
721 northern areas, China. *Sustainable Cities Soc.* 53,
722 101891.<https://doi.org/10.1016/j.scs.2019.101891>
- 723 Bandowe, B.A.M., Meusel, H., 2017. Nitrated polycyclic aromatic hydrocarbons (nitro-
724 PAHs) in the environment – A review. *Sci. Total Environ.* 581-582, 237-
725 257.<https://doi.org/10.1016/j.scitotenv.2016.12.115>
- 726 Becagli, S., Sferlazzo, D.M., Pace, G., di Sarra, A., Bommarito, C., Calzolari, G., Ghedini, C.,
727 Lucarelli, F., Meloni, D., Monteleone, F., Severi, M., Traversi, R., Udisti, R., 2012. Evidence
728 for heavy fuel oil combustion aerosols from chemical analyses at the island of
729 Lampedusa: a possible large role of ships emissions in the Mediterranean. *Atmos. Chem.*
730 *Phys.* 12, 3479-3492.<https://doi.org/10.5194/acp-12-3479-2012>
- 731 Borgie, M., Dagher, Z., Ledoux, F., Verdin, A., Cazier, F., Martin, P., Hachimi, A., Shirali, P.,
732 Greige-Gerges, H., Courcot, D., 2015. Comparison between ultrafine and fine particulate
733 matter collected in Lebanon: Chemical characterization, in vitro cytotoxic effects and
734 metabolizing enzymes gene expression in human bronchial epithelial cells. *Environ.*
735 *Pollut.* 205, 250-260.<https://doi.org/10.1016/j.envpol.2015.05.027>
- 736 Borm, P.J.A., Kelly, F., Künzli, N., Schins, R.P.F., Donaldson, K., 2007. Oxidant generation
737 by particulate matter: from biologically effective dose to a promising, novel metric.
738 *Occup. Environ. Med.* 64, 73-74.<http://dx.doi.org/10.1136/oem.2006.029090>
- 739 Chang, M.B., Weng, Y.M., Lee, T.Y., Chen, Y.W., Chang, S.H., Chi, K.H., 2003. Sampling and
740 analysis of ambient dioxins in northern Taiwan. *Chemosphere* 51, 1103-
741 1110.[https://doi.org/10.1016/S0045-6535\(02\)00711-7](https://doi.org/10.1016/S0045-6535(02)00711-7)
- 742 Chen, L.C., Lippmann, M., 2009. Effects of Metals within Ambient Air Particulate Matter
743 (PM) on Human Health. *Inhalation Toxicol.* 21, 1-
744 31.<https://doi.org/10.1080/08958370802105405>
- 745 Chen, P., Bi, X., Zhang, J., Wu, J., Feng, Y., 2015. Assessment of heavy metal pollution
746 characteristics and human health risk of exposure to ambient PM_{2.5} in Tianjin, China.
747 *Particuology* 20, 104-109.<https://doi.org/10.1016/j.partic.2014.04.020>
- 748 Chen, Q., Yang, H., Zhou, N., Sun, L., Bao, H., Tan, L., Chen, H., Ling, X., Zhang, G., Huang, L.,
749 Li, L., Ma, M., Yang, H., Wang, X., Zou, P., Peng, K., Liu, T., Shi, X., Feng, D., Zhou, Z., Ao, L.,
750 Cui, Z., Cao, J., 2017. Phthalate exposure, even below US EPA reference doses, was
751 associated with semen quality and reproductive hormones: Prospective MARHCS study

752 in general population. *Environ. Int.* 104, 58-
753 68.<https://doi.org/10.1016/j.envint.2017.04.005>

754 Chen, R., Jia, B., Tian, Y., Feng, Y., 2021. Source-specific health risk assessment of PM_{2.5}-
755 bound heavy metals based on high time-resolved measurement in a Chinese megacity:
756 insights into seasonal and diurnal variations. *Ecotoxicol. Environ. Saf.* 216,
757 112167.<https://doi.org/10.1016/j.ecoenv.2021.112167>

758 Cochard, M., Ledoux, F., Landkocz, Y., 2020. Atmospheric fine particulate matter and
759 epithelial mesenchymal transition in pulmonary cells: state of the art and critical review
760 of the in vitro studies. *Journal of Toxicology and Environmental Health, Part B, Critical*
761 *reviews* 23, 293-318.<https://doi.org/10.1080/10937404.2020.1816238>

762 Dahmardeh Behrooz, R., Kaskaoutis, D.G., Grivas, G., Mihalopoulos, N., 2021. Human
763 health risk assessment for toxic elements in the extreme ambient dust conditions
764 observed in Sistan, Iran. *Chemosphere* 262,
765 127835.<https://doi.org/10.1016/j.chemosphere.2020.127835>

766 Deng, Y., Wang, M., Tian, T., Lin, S., Xu, P., Zhou, L., Dai, C., Hao, Q., Wu, Y., Zhai, Z., Zhu, Y.,
767 Zhuang, G., Dai, Z., 2019. The Effect of Hexavalent Chromium on the Incidence and
768 Mortality of Human Cancers: A Meta-Analysis Based on Published Epidemiological
769 Cohort Studies. *Front Oncol* 9, 24-24.<https://doi.org/10.3389/fonc.2019.00024>

770 Dhaini, H.R., Salameh, T., Waked, A., Sauvage, S., Borbon, A., Formenti, P., Doussin, J.-F.,
771 Locoge, N., Afif, C., 2017. Quantitative cancer risk assessment and local mortality burden
772 for ambient air pollution in an eastern Mediterranean City. *Environ Sci Pollut Res Int* 24,
773 14151-14162.<https://doi.org/10.1007/s11356-017-9000-y>

774 Díaz, R.V., Rosa Dominguez, E., 2009. Health risk by inhalation of PM_{2.5} in the
775 metropolitan zone of the City of Mexico. *Ecotoxicol. Environ. Saf.* 72, 866-
776 871.<https://doi.org/10.1016/j.ecoenv.2008.09.014>

777 DoHA, 2012. Environmental health risk assessment : guidelines for assessing human
778 health risks from environmental hazards. Department of Health and Ageing and
779 enHealth Council.
780 [https://www1.health.gov.au/internet/main/publishing.nsf/content/A12B57E41EC9F3](https://www1.health.gov.au/internet/main/publishing.nsf/content/A12B57E41EC9F326CA257BF0001F9E7D/$File/Environmental-health-Risk-Assessment.pdf)
781 [26CA257BF0001F9E7D/\\$File/Environmental-health-Risk-Assessment.pdf](https://www1.health.gov.au/internet/main/publishing.nsf/content/A12B57E41EC9F326CA257BF0001F9E7D/$File/Environmental-health-Risk-Assessment.pdf)

782 Fadel, M., Ledoux, F., Farhat, M., Kfoury, A., Courcot, D., Afif, C., 2021. PM_{2.5}
783 characterization of primary and secondary organic aerosols in two urban-industrial
784 areas in the East Mediterranean. *J Environ Sci* 101, 98-
785 116.<https://doi.org/10.1016/j.jes.2020.07.030>

786 Fang, B., Zhang, L., Zeng, H., Liu, J., Yang, Z., Wang, H., Wang, Q., Wang, M., 2020. PM_{2.5}-
787 Bound Polycyclic Aromatic Hydrocarbons: Sources and Health Risk during Non-Heating
788 and Heating Periods (Tangshan, China). *Int. J. Environ. Res. Public Health*
789 17.<https://doi.org/10.3390/ijerph17020483>

790 Gaga, E.O., Ari, A., 2019. Gas-particle partitioning and health risk estimation of polycyclic
791 aromatic hydrocarbons (PAHs) at urban, suburban and tunnel atmospheres: Use of

792 measured EC and OC in model calculations. *Atmospheric Pollution Research* 10, 1-
793 11.<https://doi.org/10.1016/j.apr.2018.05.004>

794 Galon-Negru, A.G., Olariu, R.I., Arsene, C., 2019. Size-resolved measurements of PM_{2.5}
795 water-soluble elements in Iasi, north-eastern Romania: Seasonality, source
796 apportionment and potential implications for human health. *Sci. Total Environ.* 695,
797 133839.<https://doi.org/10.1016/j.scitotenv.2019.133839>

798 Hogarh, J.N., Seike, N., Kobara, Y., Carboo, D., Fobil, J.N., Masunaga, S., 2018. Source
799 characterization and risk of exposure to atmospheric polychlorinated biphenyls (PCBs)
800 in Ghana. *Environ Sci Pollut Res Int* 25, 16316-16324.[10.1007/s11356-018-2090-3](https://doi.org/10.1007/s11356-018-2090-3)

801 Huang, M., Chen, X., Zhao, Y., Yu Chan, C., Wang, W., Wang, X., Wong, M.H., 2014. Arsenic
802 speciation in total contents and bioaccessible fractions in atmospheric particles related
803 to human intakes. *Environ. Pollut.* 188, 37-
804 44.<https://doi.org/10.1016/j.envpol.2014.01.001>

805 Hubaux, R., Becker-Santos, D.D., Enfield, K.S.S., Rowbotham, D., Lam, S., Lam, W.L.,
806 Martinez, V.D., 2013. Molecular features in arsenic-induced lung tumors. *Molecular*
807 *Cancer* 12, 20.<https://doi.org/10.1186/1476-4598-12-20>

808 IARC, 2004. IARC monographs on the evaluation of carcinogenic risks to humans.

809 Jaakkola, J.J., Knight, T.L., 2008. The role of exposure to phthalates from polyvinyl
810 chloride products in the development of asthma and allergies: a systematic review and
811 meta-analysis. *Environ. Health Perspect.* 116, 845-
812 853.<https://doi.org/10.1289/ehp.10846>

813 Kfoury, A., Ledoux, F., Roche, C., Delmaire, G., Roussel, G., Courcot, D., 2016. PM_{2.5} source
814 apportionment in a French urban coastal site under steelworks emission influences
815 using constrained non-negative matrix factorization receptor model. *J Environ Sci* 40,
816 114-128.<https://doi.org/10.1016/j.jes.2015.10.025>

817 Kobrossi, R., Nuwayhid, I., Sibai, A.M., El-Fadel, M., Khogali, M., 2002. Respiratory health
818 effects of industrial air pollution on children in North Lebanon. *Int J Environ Health Res*
819 12, 205-220.<https://doi.org/10.1080/09603/202/000000970>

820 Kumar, A., Ambade, B., Sankar, T.K., Sethi, S.S., Kurwadkar, S., 2020. Source identification
821 and health risk assessment of atmospheric PM_{2.5}-bound polycyclic aromatic
822 hydrocarbons in Jamshedpur, India. *Sustainable Cities Soc.* 52,
823 101801.<https://doi.org/10.1016/j.scs.2019.101801>

824 Ledoux, F., Laversin, H., Courcot, D., Courcot, L., Zhilinskaya, E.A., Puskaric, E., Aboukaïs,
825 A., 2006. Characterization of iron and manganese species in atmospheric aerosols from
826 anthropogenic sources. *Atmos Res* 82, 622-
827 632.<https://doi.org/10.1016/j.atmosres.2006.02.018>

828 Lelieveld, J., Hadjinicolaou, P., Kostopoulou, E., Giannakopoulos, C., Pozzer, A., Tanarhte,
829 M., Tyrllis, E., 2014. Model projected heat extremes and air pollution in the eastern
830 Mediterranean and Middle East in the twenty-first century. *Regional Environmental*
831 *Change* 14, 1937-1949.<http://dx.doi.org/10.1007/s10113-013-0444-4>

832 Li, P.-h., Jia, H.-y., Wang, Y., Li, T., Wang, L., Li, Q.-q., Yang, M.-m., Yue, J.-j., Yi, X.-l., Guo, L.-
833 q., 2019. Characterization of PM_{2.5}-bound phthalic acid esters (PAEs) at regional
834 background site in northern China: Long-range transport and risk assessment. *Sci. Total*
835 *Environ.* 659, 140-149.<https://doi.org/10.1016/j.scitotenv.2018.12.246>

836 Liu, J., Chen, Y., Chao, S., Cao, H., Zhang, A., Yang, Y., 2018. Emission control priority of
837 PM_{2.5}-bound heavy metals in different seasons: A comprehensive analysis from health
838 risk perspective. *Sci. Total Environ.* 644, 20-
839 30.<https://doi.org/10.1016/j.scitotenv.2018.06.226>

840 Loomis, D., Grosse, Y., Lauby-Secretan, B., Ghissassi, F.E., Bouvard, V., Benbrahim-Tallaa,
841 L., Guha, N., Baan, R., Mattock, H., Straif, K., 2013. The carcinogenicity of outdoor air
842 pollution. *Lancet Oncol.* 14, 1262-1263.[https://doi.org/10.1016/S1470-](https://doi.org/10.1016/S1470-2045(13)70487-X)
843 [2045\(13\)70487-X](https://doi.org/10.1016/S1470-2045(13)70487-X)

844 Ma, B., Wang, L., Tao, W., Liu, M., Zhang, P., Zhang, S., Li, X., Lu, X., 2020. Phthalate esters
845 in atmospheric PM_{2.5} and PM₁₀ in the semi-arid city of Xi'an, Northwest China: Pollution
846 characteristics, sources, health risks, and relationships with meteorological factors.
847 *Chemosphere* 242, 125226.<https://doi.org/10.1016/j.chemosphere.2019.125226>

848 Mark Goldberg, 2008. A systematic review of the relation between long-term exposure
849 to ambient air pollution and chronic diseases. *Rev. Environ. Health* 23, 243-
850 298.<https://doi.org/10.1515/REVEH.2008.23.4.243>

851 Marquès, M., Domingo, J.L., 2019. Concentrations of PCDD/Fs in Human Blood: A Review
852 of Data from the Current Decade. *Int. J. Environ. Res. Public Health* 16,
853 3566.<https://doi.org/10.3390/ijerph16193566>

854 Melki, P.N., Ledoux, F., Aouad, S., Billet, S., El Khoury, B., Landkocz, Y., Abdel-Massih, R.M.,
855 Courcot, D., 2017. Physicochemical characteristics, mutagenicity and genotoxicity of
856 airborne particles under industrial and rural influences in Northern Lebanon. *Environ*
857 *Sci Pollut Res Int* 24, 18782-18797.<https://doi.org/10.1007/s11356-017-9389-3>

858 Miri, M., Rostami Aghdam Shendi, M., Ghaffari, H.R., Ebrahimi Aval, H., Ahmadi, E., Taban,
859 E., Gholizadeh, A., Yazdani Aval, M., Mohammadi, A., Azari, A., 2016. Investigation of
860 outdoor BTEX: Concentration, variations, sources, spatial distribution, and risk
861 assessment. *Chemosphere* 163, 601-
862 609.<https://doi.org/10.1016/j.chemosphere.2016.07.088>

863 MoE, 2017. Lebanon's National Strategy for Air Quality Management for 2030.
864 [http://www.databank.com.lb/docs/MoE-\(2017\)-National-Air-Quality-Management-](http://www.databank.com.lb/docs/MoE-(2017)-National-Air-Quality-Management-Strategy-EN.pdf)
865 [Strategy-EN.pdf](http://www.databank.com.lb/docs/MoE-(2017)-National-Air-Quality-Management-Strategy-EN.pdf).

866 Murari, V., Singh, N., Ranjan, R., Singh, R.S., Banerjee, T., 2020. Source apportionment and
867 health risk assessment of airborne particulates over central Indo-Gangetic Plain.
868 *Chemosphere* 257, 127145.<https://doi.org/10.1016/j.chemosphere.2020.127145>

869 Nakhlé, M.M., Farah, W., Ziadé, N., Abboud, M., Salameh, D., Annesi-Maesano, I., 2015.
870 Short-term relationships between emergency hospital admissions for respiratory and
871 cardiovascular diseases and fine particulate air pollution in Beirut, Lebanon. *Environ.*
872 *Monit. Assess.* 187, 196.<https://doi.org/10.1007/s10661-015-4409-6>

873 Nasser, Z., Salameh, P., Dakik, H., Elias, E., Abou Abbas, L., Levêque, A., 2015. Outdoor air
874 pollution and cardiovascular diseases in Lebanon: a case-control study. *J Environ Public*
875 *Health* 2015, 810846.<https://doi.org/10.1155/2015/810846>

876 Nisbet, I.C., LaGoy, P.K., 1992. Toxic equivalency factors (TEFs) for polycyclic aromatic
877 hydrocarbons (PAHs). *Regulatory toxicology and pharmacology : RTP* 16, 290-
878 300.[https://doi.org/10.1016/0273-2300\(92\)90009-X](https://doi.org/10.1016/0273-2300(92)90009-X)

879 NTP, 2016. National Toxicology program, Report on carcinogens, fourteenth edition,
880 Research Triangle Park, NC: U.S. Department of Health and Human Services, Public
881 Health Service. <https://ntp.niehs.nih.gov/go/roc14>.

882 OEHHA, 2012. Technical Support Document for Exposure Assessment and Stochastic
883 Analysis. Office of Environmental Health Hazard Assessment, California Environmental
884 Protection Agency, Sacramento, [Sacramento, Calif.]

885 Pongpiachan, S., 2015. Incremental Lifetime Cancer Risk of PM_{2.5} Bound Polycyclic
886 Aromatic Hydrocarbons (PAHs) Before and After the Wildland Fire Episode. *Aerosol and*
887 *Air Quality Research* x, 1-13.<https://doi.org/10.4209/aaqr.2015.01.0011>

888 Ramírez, O., Sánchez de la Campa, A.M., Sánchez-Rodas, D., de la Rosa, J.D., 2020.
889 Hazardous trace elements in thoracic fraction of airborne particulate matter:
890 Assessment of temporal variations, sources, and health risks in a megacity. *Sci. Total*
891 *Environ.* 710, 136344.<https://doi.org/10.1016/j.scitotenv.2019.136344>

892 Rengarajan, T., Rajendran, P., Nandakumar, N., Lokeshkumar, B., Rajendran, P., Nishigaki,
893 I., 2015. Exposure to polycyclic aromatic hydrocarbons with special focus on cancer.
894 *Asian Pacific Journal of Tropical Biomedicine* 5, 182-
895 189.[https://doi.org/10.1016/S2221-1691\(15\)30003-4](https://doi.org/10.1016/S2221-1691(15)30003-4)

896 Rondini, E.A., Walters, D.M., Bauer, A.K., 2010. Vanadium pentoxide induces pulmonary
897 inflammation and tumor promotion in a strain-dependent manner. *Part Fibre Toxicol* 7,
898 9-9.<https://doi.org/10.1186/1743-8977-7-9>

899 Rusyn, I., Corton, J.C., 2012. Mechanistic considerations for human relevance of cancer
900 hazard of di(2-ethylhexyl) phthalate. *Mutat. Res.* 750, 141-
901 158.<https://doi.org/10.1016/j.mrrev.2011.12.004>

902 Salameh, P., Salame, J., Khayat, G., Akhdar, A., Ziadeh, C., Azizi, S., Khoury, F., Akiki, Z.,
903 Nasser, Z., Abou Abbass, L., Saadeh, D., Waked, M., 2012. Exposure to outdoor air
904 pollution and chronic bronchitis in adults: a case-control study. *Int J Occup Environ Med*
905 3, 165-177

906 Salameh, T., Sauvage, S., Afif, C., Borbon, A., Léonardis, T., Brioude, J., Waked, A., Locoge,
907 N., 2015. Exploring the seasonal NMHC distribution in an urban area of the Middle East
908 during ECOCEM campaigns: Very high loadings dominated by local emissions and
909 dynamics. *Environ. Chem.*10.1071/EN14154

910 Salameh, T., Sauvage, S., Afif, C., Borbon, A., Locoge, N., 2016. Source apportionment vs.
911 emission inventories of non-methane hydrocarbons (NMHC) in an urban area of the

912 Middle East: local and global perspectives. *Atmospheric Chemistry and Physics*
913 *Discussions* 15.<https://doi.org/10.5194/acp-16-3595-2016>

914 Samet, J., Krewski, D., 2007. Health Effects Associated With Exposure to Ambient Air
915 Pollution. *J. Toxicol. Environ. Health, Part A* 70, 227-
916 242.<https://doi.org/10.1080/15287390600884644>

917 Simoneit, B.R.T., Medeiros, P.M., Didyk, B.M., 2005. Combustion Products of Plastics as
918 Indicators for Refuse Burning in the Atmosphere. *Environ. Sci. Technol.* 39, 6961-
919 6970.[10.1021/es050767x](https://doi.org/10.1021/es050767x)

920 Sulaymon, I.D., Mei, X., Yang, S., Chen, S., Zhang, Y., Hopke, P.K., Schauer, J.J., Zhang, Y.,
921 2020. PM_{2.5} in Abuja, Nigeria: Chemical characterization, source apportionment,
922 temporal variations, transport pathways and the health risks assessment. *Atmos Res*
923 237, 104833.<https://doi.org/10.1016/j.atmosres.2019.104833>

924 Sun, J., Tang, J., Chen, Z., Nie, J., Zhang, S., Li, J., 2017. PCDD/Fs profile in ambient air of
925 different types factories and human health risk assessment in Suzhou of Jiangsu
926 province, China. *Atmos. Pollut. Res.* 8, 74-79.<https://doi.org/10.1016/j.apr.2016.07.010>

927 Thurston, G.D., Kipen, H., Annesi-Maesano, I., Balmes, J., Brook, R.D., Cromar, K., De
928 Matteis, S., Forastiere, F., Forsberg, B., Frampton, M.W., Grigg, J., Heederik, D., Kelly, F.J.,
929 Kuenzli, N., Laumbach, R., Peters, A., Rajagopalan, S.T., Rich, D., Ritz, B., Samet, J.M.,
930 Sandstrom, T., Sigsgaard, T., Sunyer, J., Brunekreef, B., 2017. A joint ERS/ATS policy
931 statement: what constitutes an adverse health effect of air pollution? An analytical
932 framework. *European Respiratory Journal* 49, 1600419.[10.1183/13993003.00419-2016](https://doi.org/10.1183/13993003.00419-2016)

933 Tsatsakis, A.M., Katsikantami, I., Kalantzi, O.-I., Sevim, Ç., Tsarouhas, K., Sarigiannis, D.,
934 Tzatzarakis, M.N., Rizos, A.K., 2019. Phthalates: Exposure and Health Effects, in: Nriagu, J.
935 (Ed.), *Encyclopedia of Environmental Health (Second Edition)*. Elsevier, Oxford, pp. 163-
936 173.

937 USEPA, 1989. General Quantitative Risk Assessment Guidelines for Noncancer Health
938 Effects. External Review Draft. Risk Assessment Forum Technical Panel on Risk
939 Assessment Guidelines for Noncancer Health Effects, U.S. Environmental Protection
940 Agency, Washington, DC.

941 USEPA, 1991. Risk Assessment Guidance for Superfund: Volume I - Human Health
942 Evaluation Manual (Part B, Development of Risk-based Preliminary Remediation Goals)
943 Publication 9285.7-01B. Office of Emergency and Remedial Response, Washington, DC.
944 NTIS PB92-963333.

945 USEPA, 1997. Exposure factors handbook. Washington DC: Office of research and
946 development, national center for environmental assessment.

947 USEPA, 2002. Supplemental guidance for developing soil screening levels for Superfund
948 sites. U.S. Environmental Protection Agency, Washington, DC.

949 USEPA, 2004a. Region 9, Preliminary remediation goals, air-water calculations. U.S.
950 Environmental Protection Agency, Washington, DC.

951 USEPA, 2004b. Risk assessment guidance for superfund volume I. Human Health
952 Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment), Final.
953 EPA/540/R/99/005. Office of Solid Waste and Emergency Response, Washington, DC.
954 PB99-963312.

955 USEPA, 2011. Exposure factors handbook : 2011 edition (Final Report). U.S.
956 Environmental Protection Agency, Washington, DC (EPA/600/R-09/052F).

957 Van den Berg, M., Birnbaum, L.S., Denison, M., De Vito, M., Farland, W., Feeley, M., Fiedler,
958 H., Hakansson, H., Hanberg, A., Haws, L., Rose, M., Safe, S., Schrenk, D., Tohyama, C.,
959 Tritscher, A., Tuomisto, J., Tysklind, M., Walker, N., Peterson, R.E., 2006. The 2005 World
960 Health Organization reevaluation of human and Mammalian toxic equivalency factors
961 for dioxins and dioxin-like compounds. *Toxicol. Sci.* 93, 223-
962 241.<https://doi.org/10.1093/toxsci/kfl055>

963 Vasilakos, C., Levi, N., Maggos, T., Hatzianestis, J., Michopoulos, J., Helmis, C., 2007. Gas-
964 particle concentration and characterization of sources of PAHs in the atmosphere of a
965 suburban area in Athens, Greece. *J. Hazard. Mater.* 140, 45-
966 51.<https://doi.org/10.1016/j.jhazmat.2006.06.047>

967 Veglia, F., Matullo, G., Vineis, P., 2003. Bulky DNA adducts and risk of cancer: a meta-
968 analysis. *Cancer epidemiology, biomarkers & prevention : a publication of the American*
969 *Association for Cancer Research*, cosponsored by the American Society of Preventive
970 *Oncology* 12, 157-160

971 Waked, A., Afif, C., Seigneur, C., 2012. An atmospheric emission inventory of
972 anthropogenic and biogenic sources for Lebanon. *Atmos Environ* 50, 88-
973 96.<https://doi.org/10.1016/j.atmosenv.2011.12.058>

974 WHO, 2016. Ambient air pollution : a global assessment of exposure and burden of
975 disease. Switzerland, Geneva.
976 <https://apps.who.int/iris/bitstream/10665/250141/1/9789241511353-eng.pdf>.

977 Xue, J., Zartarian, V., Moya, J., Freeman, N., Beamer, P., Black, K., Tolve, N., Shalat, S., 2007.
978 A Meta-Analysis of Children's Hand-to-Mouth Frequency Data for Estimating Nondietary
979 Ingestion Exposure. *Risk Anal.* 27, 411-420.<https://doi.org/10.1111/j.1539-6924.2007.00893.x>

981 Yang, L., Liu, G., Zheng, M., Jin, R., Zhu, Q., Zhao, Y., Zhang, X., Xu, Y., 2017. Atmospheric
982 occurrence and health risks of PCDD/Fs, polychlorinated biphenyls, and polychlorinated
983 naphthalenes by air inhalation in metallurgical plants. *Sci. Total Environ.* 580, 1146-
984 1154.<https://doi.org/10.1016/j.scitotenv.2016.12.071>

985 Zhang, X., Wang, Q., Qiu, T., Tang, S., Li, J., Giesy, J.P., Zhu, Y., Hu, X., Xu, D., 2019. PM_{2.5}
986 bound phthalates in four metropolitan cities of China: Concentration, seasonal pattern
987 and health risk via inhalation. *Sci. Total Environ.* 696,
988 133982.<https://doi.org/10.1016/j.scitotenv.2019.133982>

989 Zubair, M., Adress, A., 2019. Dioxins and Furans: Emerging Contaminants of Air, in:
990 Olvera, J.D.R. (Ed.), *Air Pollution - Monitoring, Quantification and Removal of Gases and*
991 *Particles*. IntechOpen.

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