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RESEARCH ARTICLE

Quantitative cancer risk assessment and local mortality burden for ambient air pollution in an eastern Mediterranean City

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Abstract Health risks posed by ambient air pollutants to the urban Lebanese population have not been well characterized. The aim of this study is to assess cancer risk and mortality burden of non-methane hydrocarbons (NMHCs) and particulates (PM) based on two field-sampling campaigns conducted during summer and winter seasons in Beirut. Seventy NMHCs were analyzed by TD-GC-FID. $PM_{2.5}$ elemental carbon (EC) components were examined using a Lab OC-EC aerosol Analyzer, and polycyclic aromatic hydrocarbons were analyzed by GC-MS. The US EPA fraction-based approach was used to assess non-cancer hazard and cancer risk for the hydrocarbon mixture, and the UK Committee on Medical Effects of Air Pollutants (COMEAP) guidelines were followed to determine the $PM_{2.5}$ attributable mortality burden. The average cumulative cancer risk exceeded the US EPA acceptable level (10^{-6}) by 40-fold in the summer and 30-fold in the winter. Benzene was found to be the highest contributor to

cancer risk (39–43%), followed by 1,3-butadiene (25–29%), both originating from traffic gasoline evaporation and combustion. The EC attributable average mortality fraction was 7.8–10%, while the average attributable number of deaths (AD) and years of life lost (YLL) were found to be 257–327 and 3086–3923, respectively. Our findings provide a baseline for future air monitoring programs, and for interventions aiming at reducing cancer risk in this population.

Keywords Air pollution · NMHC · Particulate matter · Lebanon · Cancer risk · Mortality burden

Introduction

Health concerns regarding air pollution are increasing worldwide. Ambient air pollution (AAP) is growingly causing a considerable disease burden (Tobollik et al. 2015). The International Agency for Research on Cancer (IARC) has recently classified outdoor air pollution as Group 1 "Carcinogenic to Humans" based on sufficient evidence of carcinogenicity in experimental animals and strong support by mechanistic studies (Loomis et al. 2013).

AAP involves emissions of complex mixtures from various sources. Of particular interest are the ubiquitous non-methane hydrocarbons (NMHCs), which are mainly emitted from different anthropogenic activities in and around urban areas (Shi et al. 2015) such as vehicle exhaust emissions and other combustion sources, hot soak evaporation, and fuel storage facilities (Salameh et al. 2015). NMHCs contribute to the formation of tropospheric ozone and lead to formation of secondary organic aerosols (Salameh et al. 2014). In addition, NHMC emissions may contain organic hazardous air pollutants (HAPS) which have a wide range of sources, effects, and exposure routes (Du et al. 2014). Acute exposure to these

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HAPS may lead to irritation of epithelial tissues in the eyes, skin, and respiratory tract, while chronic exposure may cause severe chronic illnesses ranging from asthma, chronic obstructive pulmonary disease, to cardiovascular disease and different types of cancer (Lewtas 2007).

AAP also includes fine particulate matter ($PM_{2.5}$) that has been widely demonstrated to cause various adverse effects in humans (Du et al. 2016). $PM_{2.5}$ is a respirable complex mixture of fine particles and liquid droplets made up of acids, organic chemicals, and dust particles. Sources of $PM_{2.5}$ include both mobile sources—such as motor vehicles—and stationary sources—such as power plants, industries, construction, and agricultural activities (Anderson et al. 2012). Inhalation of $PM_{2.5}$ over time increases the risk of morbidity and mortality (Hoek et al. 2013; Zhang et al. 2014). Further, the World Health Organization (WHO) estimates that exposure to $PM_{2.5}$ contributes, globally, to about 3% of cardiopulmonary and 5% of lung cancer deaths yearly, ranking it as the 13th leading cause of mortality worldwide (WHO 2013).

Lebanon, a small developing country located on the eastern Mediterranean coast, is characterized by extensive urbanization with heavy traffic and overcrowding in its urban areas. Despite limited availability of air quality data, studies show levels of measured air pollutants to exceed WHO and US Environmental Protection Agency (EPA) guideline values and air quality reference standards (Afif et al. 2009; El-Fadel and Massoud 2000; Salameh et al. 2015). The aim of this study is to assess cancer risk and mortality burden through inhalation based on two intensive field measurement campaigns we previously conducted during summer 2011 and winter 2012 (Salameh et al. 2014, 2015; Waked et al. 2013, 2014).

Material and methods

Sampling and analytical methods

Sampling took place at two seasonal time points within the framework of the Emission and Chemistry of Organic Carbon in the East Mediterranean (ECOCEM) field campaigns, during the periods of July 2–18, 2011 and January 28th to February 12th, 2012. The sampling site was located in Mar Roukoz Mansourieh (33°88/7N, 35°56/E), a semi-urban area situated in the eastern suburbs of the capital city of Beirut −6 km southeast from Downtown and 8 km southwest from Beirut International Airport and at an altitude of 230 m above sea level. The sampling was deemed appropriately positioned for monitoring air masses coming from the Greater Beirut Area—which includes the city of Beirut and its suburbs.

For NMHC analysis, a total of 477 air samples were continuously hourly analyzed by online thermal desorption gas chromatography with a flame ionization detector (TD-GC-

FID). Measurement and analysis procedures are described elsewhere (Salameh et al. 2015). In addition, a total of 52 samples (12-h and 24-h sampling periods) for $PM_{2.5}$ analysis were collected on Pallflex quartz fiber filters using a high-volume sampler. Measurement and analysis procedures are described elsewhere (Waked et al. 2013).

Risk assessment methodology

In principle, the health risk assessment process consists of the following steps: hazard identification and characterization, exposure assessment, and risk characterization. Methods and values used in the process integrate estimates of exposure intensity with appropriate toxicological values to determine the likelihood of adverse effects in potentially exposed populations. The typical exposure pathway for both PM and NMHCs is inhalation. In this study, we followed the US EPA fraction-based approach in order to conduct a risk assessment for the measured mixtures of aliphatic and aromatic hydrocarbons (Brewer et al. 2013; MADEP 2003; US EPA 2009). Measured hydrocarbon mixtures were first classified as aliphatic or aromatic hydrocarbons. Each of these two major fractions was then further separated into low-, medium-, or high-carbon range fractions based on the US EPA fraction definition (Table 1). Briefly, the fractions are defined by the number of carbon atoms (C) in the compounds of the fraction and by the compounds' equivalent carbon (EC) number index, which is related to their transport in the environment. In the EPA's approach, the potential health risk of each of the aliphatic or aromatic hydrocarbon fractions is represented in one of three ways: (1) the surrogate method: the toxicity value for a surrogate (similar) aliphatic or aromatic hydrocarbon mixture or compound is integrated with the exposure data for the entire mass of the fraction; (2) the component method: toxicity values for well-studied individual chemicals that make up a large portion of the fraction are combined with their respective exposure estimates under an assumption of dose- or response addition; or (3) the hybrid method: a combination of the above two methods is used for the same fraction, and the results are combined under an assumption of dose or response addition.

The individual fractions and total mixture health hazards for the measured compounds were estimated for all aromatic and aliphatic hydrocarbons by summing fraction specific hazard indices assuming dose addition as follows:

$$HI_m = \sum_{i=1}^{7} HI_i \tag{1}$$

$$HI_i = \frac{E_i}{RfV_i} \tag{2}$$

where:

Table 1 Adapted aliphatic and aromatic fractionation scheme (US EPA 2009; Brewer et al. 2013; MADEP 2003)

Primary fractions	Secondary fractions	Fraction description	Components and surrogates
Aliphatics	Aliph vl	Very low carbon range C2–C4	*All present components*
	Aliph 1	Low carbon range (C5–C8; EC5–EC8)	*Surrogates*: Commercial hexane or *n*-hexane *Components*: *n*-pentane, *n*-hexane, cyclohexane, dimethylbutanes, methylpentanes, cyclopentane, *n*-heptane, *n*-octane, trimethylpentanes, cyclohexane, methylcyclopentane, methylcyclohexane, 1-hexene
	Aliph 2	Medium carbon range (C9–C18; EC > 8–EC16)	*Surrogates*: mid range aliphatic hydrocarbon streams (AHS) *Components*: *n*-nonane, *n*-decane, longer chain *n*-alkanes; some *n*-alkenes (e.g., tridecene); branched chain alkanes and alkenes, alkyl-substituted cycloalkanes
	Aliph 3	High carbon range (C19–C32; EC > 16–EC35)	*Surrogates*: white mineral oil *Components*: longer *n*-alkanes (e.g., eicosane), branched and cyclic alkanes
Aromatics	Arom1	Low carbon range (C6–C8; EC6–EC < 9)	*Components*: benzene, toluene. Ethylbenzene, and *o*-, *m*-, and *p*-xylenes (commonly referred to as BTEX)
	Arom 2	Medium carbon range (C9–C16; EC9–EC < 22)	*Surrogates*: high-flash aromatic naphtha; *Components*: longer chain and multi-substituted ben-
	Arom 3	High carbon range (C17–C32; EC22–EC35)	zenes (e.g., cumene [isopropylbenzene], *n*-propylbenzene, methylethylbenzenes, trimethylbenzenes), indan, methylindans, naphthalenes, lower molecular weight PAHs (e.g., acenaphthylene, acenaphthene, fluorene, phenanthrene, anthracene, and pyrene) *Surrogates*: Fluoranthene *Components*: High-molecular weight PAHs: benzo[a]pyrene, benz[a]anthracene, chrysene, benzo[b]fluoranthene, benzo[k]fluoranthene, dibenz[a,h]anthracene, indeno[1,2,3-c,d]pyrene)

C number of carbon atoms in the compounds of the fraction, *EC* the compounds' equivalent carbon number index

HI_m Hazard index for the whole mixture
HI_i Hazard index for the *i*th fraction
E_i Inhalation intake of the *i*th chemical or fraction ($\mu g/m^3$)
RfV_i Reference value determined as the inhalation reference concentration (RfC) ($\mu g/m^3$) for the *i*th chemical or fraction

$Dose_i$ Determined by the inhalation exposure concentration for the *i*th fraction ($\mu g/m^3$)
CSF_i Cancer slope factor determined as the inhalation unit risk (IUR) ($\mu g/m^3$)$^{-1}$

Similarly, the individual fractions and total mixture cancer risks for the measured compounds were estimated for all aromatic and aliphatic fractions by summing fraction specific risk estimates assuming response addition as follows:

$$R_m = \sum_{i=1}^{7} R_i \qquad (3)$$

$$R_i = Dose_i \times CSF_i \qquad (4)$$

where

R_m Cumulative cancer risk posed by the mixture
R_i Cancer risk function associated with the *i*th aliphatic or aromatic fraction

In order to determine risk apportionment, we compared individual component mean risks as a function of cumulative cancer risk. In addition, hourly average concentrations were determined for individual aliphatic and aromatic components in order to determine patterns of cancer risk and source apportionment to cumulative cancer risk on a typical day of both summer and winter based on the hourly average concentration for individual components and fractions. Source apportionment for NMHC was conducted using the positive matrix factorization source-receptor model; methods and results are described elsewhere (Salameh et al. 2016). In brief, six sources in winter and five sources in summer were identified to contribute to NMHC ambient concentrations in Beirut. Their diurnal profiles were also determined. The identified sources in winter were the combustion related to regional traffic, the combustion related to local traffic, the gasoline evaporation originating from traffic, the

gasoline evaporation coming from fuel storage facilities, the gas leakage, and the background. These sources contribute to 19, 10, 22, 21, 10, and 18% of NMHCs concentrations, respectively. On the other hand, the identified sources in summer were the combustion related traffic, the combustion related to nocturnal traffic, the gasoline evaporation related to traffic, the gasoline evaporation originating from the fuel storage facilities, and the local background which contribute to 17, 27, 30, 16, and 10% of NMHCs concentrations, respectively.

Mortality burden evaluation for PM$_{2.5}$ Elemental Carbon fraction

Elemental Carbon (EC) is considered a marker of combustion sources of exposure. In order to estimate the burden of local mortality from chronic exposure to the EC fraction of PM$_{2.5}$, the methods of the UK Committee on Medical Effects of Air Pollutants (COMEAP) were used (COMEAP 2012) as follows:

$$AF = \frac{(RR-1)}{RR} \tag{5}$$

$$RR = 1 + \left(r \times \frac{x}{y}\right) \tag{6}$$

$$AD = AF \times D \tag{7}$$

$$YLL = AD \times AYL \tag{8}$$

where:

AF	The EC attributable fraction of mortality
RR	Relative risk
r	the all-cause mortality percent risk increase corresponding to EC air pollution increment
x	Modeled EC concentration
y	Corresponding increment of EC in the annual concentration
AD	EC attributable total number of deaths in the specified year
D	Total number of deaths in the specified year
YLL	Years of life lost
AYL	Average loss of years of life

The concentration-response function between additional mortality risk and chronic exposure to EC was determined by assuming that an increment of 1 μg/m^3 in the annual concentration will increase the all-cause mortality risk by 6% based on published pooled global data (Hoek et al. 2013). The mortality rate and total number of deaths in the region of Beirut were obtained from the National Health Statistics Report in Lebanon (MOPH 2012). An average loss of 12 years of life (AYL) per attributable death was assumed to estimate loss of life to the local population—inclusion of deaths from all ages in the calculation would make a very negligible difference in the

estimate, considering evidence of a very few attributable deaths under 30 years of age (COMEAP 2012).

Results and discussion

NMHC non-carcinogenic health risk assessment

Applying the US EPA approach to NMHCs to assess AAP health hazards in the Beirut region resulted in hourly mean HI values for each field campaign of less than 1 for all the measured individual fractions and components and for both sampling seasons. Computed mean HI$_i$ values ranged from 0.00005 to 0.19 and 0.00002 to 0.13, while whole mixture HI$_m$ values were 0.53 and 0.33, for summer and winter seasons, respectively. Therefore, we conclude that the additive toxicity of recorded NMHCs is compliant with US EPA limits, and consequently do not pose any non-cancer health risk. However, it is important to note that the worst-hour of a typical day marked HI$_m$ values above 1 (1.22 for summer vs 1.42 for winter, respectively) suggesting a potential health hazard. Calculated HI values recorded for all aliphatic and aromatic fractions and whole mixtures are summarized in Table 2. The highest HI values were observed for 1,3-butadiene both during summer and winter seasons.

NMHC cancer risk assessment

The seasonal cancer risks for NMHCs through inhalation are illustrated in Table 3. Results indicate that the exposed population is at excessive risk with a higher risk in the summer season. Seasonal differences in cancer risk are mainly due to lower air concentrations of all the measured compounds contributing to cancer risk, in the winter compared to the summer season field campaign, except for AHS. The average cumulative cancer risk was found to exceed the US EPA acceptable level of 1 in 1 million (10^{-6}) by 40-fold in the summer and 30-fold in the winter. A worst-case scenario (the hour with the highest observed R$_m$ during the field campaign) revealed a 120-fold and 150-fold higher cumulative cancer risk compared to the US EPA acceptable risk limit, for summer and winter, respectively. In addition, results highlighted aromatic fractions as the main contributors to cancer risk, with benzene having the highest contribution (39–43%), followed by 1,3-butadiene (25–29%) (Fig. 1). Patterns of cancer risk show a progressive increase in cumulative cancer risk starting at 8:00 pm and peaking at midnight on a typical day of summer, while a peak cumulative cancer risk is observed at 10:00 am on a typical day during winter (Fig. 2). In addition to local background, peaking at 80% contribution to cancer risk in summer and a bit over 40% in the winter, main contributors to cancer risk were

Table 2 Estimated inhalation non-cancer health hazards for measured aliphatic and aromatic hydrocarbon fractions during summer and winter seasons

HAP	Summer concentrations		Winter concentrations		Non-cancer toxic critical effect	RfC (µg/m³)	RfC source	Summer health hazard		Winter health hazard	
	Mean (µg/m³)	Max[d] (µg/m³)	Mean (µg/m³)	Max[d] (µg/m³)				Mean HI	Max HI	Mean HI	Max HI
Aliph v/											
1,3-Butadiene	0.39	1.69	0.26	1.83	Ovarian Atrophy	2	US EPA IRIS	0.19	0.85	0.13	0.91
Tetrachloroethene	0.75	19.07	nd	nd	Cognitive Neurotoxicity	40	US EPA IRIS	0.02	0.48	-	-
Aliph 1											
Hexane[a]	1.03	6.68	0.6	4.08	Peripheral Neuropathy	700	US EPA PPRTV; IRIS	2×10^{-3}	0.01	1×10^{-3}	7×10^{-3}
Cyclohexane	0.28	1.51	0.13	0.71	Reduced Weight	6000	US EPA PPRTV	5×10^{-5}	3×10^{-4}	2×10^{-5}	1×10^{-4}
Methyl-cyclohexane	0.33	1.75	0.2	1.14	Kidney Toxicity	3000	US EPA PPRTV	1×10^{-4}	6×10^{-4}	7×10^{-5}	4×10^{-4}
Total Aliph 1 measured components	34.13	280.4	19.7	201.88		600	US EPA (Screening Levels for Superfund sites)	0.06	0.47	0.03	0.34
Aliph 2											
Total Aliph 2 measured components	1.41	7.41	1.57	8.05	Nasal goblet cell hypertrophy and Adrenal hyperplasia	100	US EPA PPRTV	0.01	0.07	0.02	0.08
Arom 1											
Benzene	2	7.55	1.72	7.83	Decreased lymphocyte count	30	US EPA IRIS	0.07	0.25	0.06	0.26
Ethylbenzene	2.29	9.33	1.14	6.83	Developmental toxicity	1000	US EPA IRIS	2×10^{-3}	9×10^{-3}	1×10^{-3}	7×10^{-3}
Toluene	14.6	62	8.09	49.98	Neurological effects	5000	US EPA IRIS	3×10^{-3}	0.01	2×10^{-3}	0.01
Xylenes[b]	10.67	41.76	5.22	33.9	Impaired motor coordination	100	US EPA IRIS	0.11	0.42	0.05	0.34
Total Arom 1								0.18	0.69	0.11	0.62
Arom 2											
Propylbenzene	0.31	1.3	0.19	1.19	Developmental toxicity	1000	US EPA PPRTV	3×10^{-4}	1×10^{-3}	2×10^{-4}	1×10^{-3}
1,3,5-Trimethylbenzene	0.69	2.72	0.31	1.73	Liver toxicity	7	US EPA PPRTV	0.1	0.39	0.04	0.25
Total Aromatics C9-C16	6.98	26.03	3.65	25.21	Maternal body weight depression	100	US EPA (Screening Levels for Superfund sites)	0.07	0.26	0.04	0.25
Mixture hazard indices HI_m								*0.53*	*1.22[c]*	*0.33*	*1.42[c]*

[a] Commercial hexane was used since *n*-hexane was present at ≤53% of fraction based on EPA guidelines

[b] Xylenes refer to the sum of m,p,o—xylenes

[c] HI_m, Max refers to the highest observed non-cancer health hazard for the whole mixture

[d] Max concentrations refer to the observed hourly maximum concentration of each component or fraction for each field campaign

HAP hazardous air pollutant, *nd* not detected, *RfC* inhalation reference concentration, *HI* hazard index, *PPRTV* provisional peer reviewed toxicity values, *IRIS* integrated risk information system

Table 3 Estimated inhalation cancer risk for measured aliphatic and aromatic hydrocarbon fractions during summer and winter seasons

Carcinogenic fractions and components	Summer concentrations		Winter concentrations		Carcinogenicity according to IARC Classification	Cancer outcome	IUR (µg/m³)⁻¹	IUR source	Summer cancer risk		Winter cancer risk	
	Mean (µg/m³)	Max[a] (µg/m³)	Mean (µg/m³)	Max[a] (µg/m³)			$\mathrm{IUR\ (µg/m^3)^{-1}}$		Mean cancer risk	Worst-hour scenario cancer risk[b]	Mean cancer risk	Worst-hour scenario cancer risk[b]
Aliph vl												
Tetrachloroethene	0.75	19.07	nd	nd	Group 2A Probably carcinogenic to Humans	Bladder Cancer	3×10^{-7}	US EPA IRIS	2×10^{-7}	6×10^{-6}	-	-
1,3 Butadiene	0.39	1.69	0.26	1.83	Group 1: Carcinogenic to Humans	Lympho-hematopoeitic cancer, Leukemia	3×10^{-5}	US EPA IRIS	12×10^{-6}	51×10^{-6}	8×10^{-6}	55×10^{-6}
Aliph 1												
Commercial Hexane	1.03	6.68	0.6	4.08	Not Classifiable as to its Human Carcinogenicity; Suggestive	Pituitary Adenoma or Carcinoma	1.9×10^{-7}	US EPA PPRTV	2×10^{-7}	1×10^{-6}	1×10^{-7}	8×10^{-7}
Aliph 2												
Total AHS C9-C18 with <1% Aromatics	1.41	7.41	1.57	8.05		Benign or Malignant Adrenal Pheochromocytoma	4.5×10^{-6}	US EPA PPRTV	6×10^{-6}	33×10^{-6}	7×10^{-6}	36×10^{-6}
Aliphatics cumultative cancer risk									19×10^{-6}	91×10^{-6}	15×10^{-6}	92×10^{-6}
Arom 1												
Benzene	2	7.55	1.72	7.83	Group 1: Carcinogenic to Humans	Leukemia	7.8×10^{-6}	US EPA IRIS	16×10^{-6}	59×10^{-6}	13×10^{-6}	61×10^{-6}
EthylBenzene	2.29	9.33	1.14	6.83	Group 2B: Possibly Carcinogenic to Humans	Leukemia & Liver cancer	2.5×10^{-6}	US EPA (Screening Levels for Superfund sites)	6×10^{-6}	23×10^{-6}	3×10^{-6}	17×10^{-6}
Aromatics Cumulative Cancer Risk									22×10^{-6}	82×10^{-6}	16×10^{-6}	78×10^{-6}
Lifetime cumulative cancer risk (R_m)									4×10^{-5}	12×10^{-5}	3×10^{-5}	15×10^{-5}

[a] Max concentrations refer to the observed hourly maximum concentration of each component or fraction for each field campaign

[b] Worst-hour scenario refers to the highest observed cumulative cancer risk for the entire mixture

PPRTV provisional peer reviewed toxicity values, *IRIS* integrated risk information system, *AHS* mid-range aliphatic hydrocarbon streams, *nd* not detected
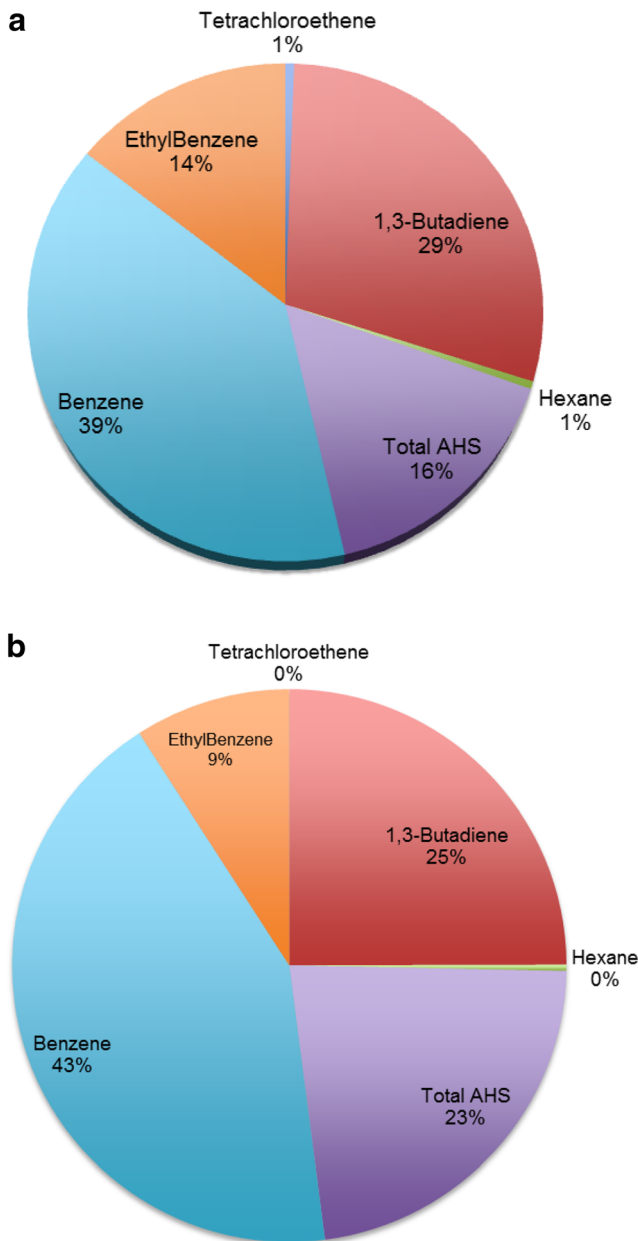
a

b

Fig. 1 Average fraction and/or components apportionment to cumulative cancer risk both in summer (**a**) and winter (**b**) seasons

identified as traffic gasoline evaporation and combustion (Fig. 3).

## PM$_{2.5}$ elemental carbon-fraction mortality burden

The average fraction of mortality attributable to elemental carbon (AF) was found to be 10% in summer and 7.8% in winter, while the average attributable number of deaths (AD) and years of life lost (YLL) in the Beirut area were found to be 327 and 3923 in summer and 257 and 3086 in winter, respectively (Table 4).

Discussion

Our study is one of the very few air pollution cancer risk assessment reports in Lebanon and the Eastern Mediterranean Region (EMR). Lebanon is characterized by a narrow coastal strip in the west, with a Mediterranean climate marked by a land-sea breeze circulation in the coastal area. Cancer registries in the country marked a rise in reported cancer incidence since the 1990s with unusual patterns, particularly for bladder cancer in males and breast cancer in females (Dhaini and Kobeissi 2014). In this study, we evaluated the association between NMHC exposure through inhalation and lifetime cancer risk in the coastal Capital city of Beirut. Our results show an excess cumulative cancer risk with clearly identified patterns where, on a typical day, cancer risk increases starting at 20:00 and peaks at 12:00am during summer, while in the winter, we observe the opposite; cancer risk decreases starting 8:00pm all the way past midnight, and increases again with sunrise, typically peaking at 10:00am of the day. This can be explained by differences in seasonal NMHC emissions since Salameh et al. (2015) demonstrated that NMHC concentrations in Beirut are driven by emissions and meteorological conditions rather than by photochemistry.

Several studies have previously reported a positive association between air pollution and chronic diseases, as well as mortality (Hoek et al. 2013). Effects have been seen at very low levels of exposure, with unclear threshold concentrations below which no health effects are likely (Brunekreef and Holgate 2002). In a study of six US cities and 8111 patients cohort—known as the Harvard Six Cities study—mortality was found to be strongly associated with fine particulate pollution (Dockery et al. 1993). In another study on ambient air pollution from 151 US cities and a cohort of 552,138 adult residents, particulate air pollution was associated with cardiopulmonary and lung cancer mortality (Pope et al. 1995). Similar results were reported in other parts of the world in Asia and in Europe (Beelen et al. 2008; Tobollik et al. 2015; Zhang et al. 2014). Other studies also reported similar positive association between air particulate pollution and risk of respiratory and cardiovascular morbidity, in addition to mortality (Daniels et al. 2000). More specifically, an increase in PM$_{2.5}$ exposure by 10 μg/m^3 was reported in several studies to be associated with 6 and 11% average increase in the relative risk of all-cause mortality and cardiovascular mortality risk, respectively (Hoek et al. 2013; Holloman et al. 2004). In addition, the attributable burden of lung cancer becomes higher than cardiovascular mortality when particulate matter exposure increases further (Fajersztajn et al. 2013). Studies estimate that 7% of lung cancer cases are associated with PM$_{2.5}$ exposure. However, a study examining an Italian cohort found a stronger association with respiratory mortality than with cardiovascular mortality (Carey et al. 2013), which is in contradiction with most US-based studies (Crouse et al. 2016). Within the EMR, a recent study reported health outcomes ranging from allergies to lung cancer and mortality to be associated with air pollution (Abdo

Fig. 2 Patterns of cancer risk in the capital city of beirut on a typical day both in summer (**a**) and winter (**b**) seasons

a

b
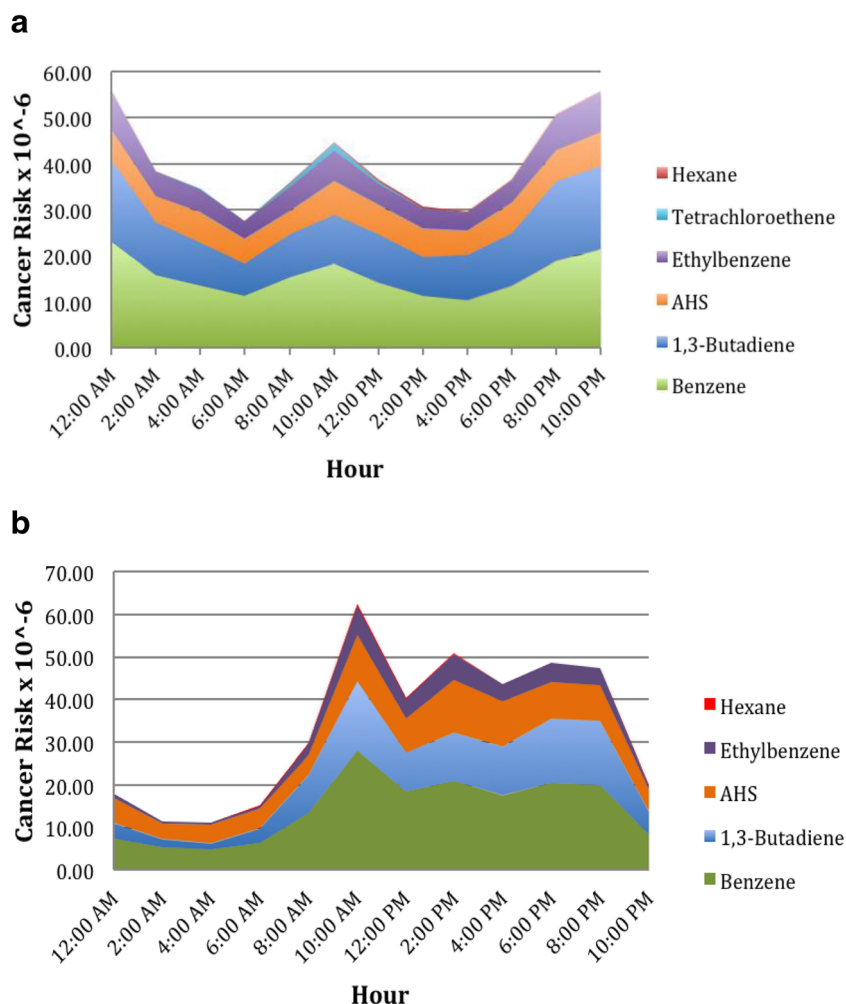
et al. 2016). Further, a study investigating PM concentrations in the city of Beirut and daily emergency hospital admissions reported an association between total respiratory admissions and PM$_{2.5}$ levels (1.016; 95% CI 1.000–1.032) (Nakhle et al. 2015).

In our study, the major contributors to cancer risk were benzene and 1,3-butadiene (BD). The main contributors to benzene in summer are local background and the combustion related to nocturnal traffic, while in winter, these are the background and the combustion related to local traffic. On the other hand, the same sources contribute mostly to 1,3-butadiene in summer whereas in winter, these are the combustion related to regional traffic as well as to local traffic. Our results are consistent with an occupational personal monitoring study in traffic policemen in Beirut, which reported significant exposure to benzene and BD (Borgie et al. 2014). In addition, both benzene and BD are classified as Group 1 by the International Agency for Research on Cancer based on evidence derived from studies in humans and experimental animals, and from mechanistic and other relevant data (Manini et al. 2010; Tabish et al. 2012; Vacek et al. 2010). BD is emitted outdoors by vehicles and may be transported indoors, while benzene, and the rest of the aromatic

fraction 1 components, may be emitted from a wider variety of outdoor sources, including industrial emissions and vehicle exhausts (Du et al. 2014). This is consistent with our findings on patterns of source apportionment where we identified traffic gasoline evaporation and combustion as significant contributors to cumulative cancer risk.

Exposures to benzene and BD, as well as ethylbenzene, were found to be associated with an increased childhood leukemia risk in many studies (Smith 2010). A nationwide case-control study in France found a significant association between acute leukemia and residence next to gasoline stations and automotive repair garages (OR = 1.6; 95% CI 1.2–2.2) (Brosselin et al. 2009). Further, in a study in Houston, Texas, census tracts with the highest benzene concentrations and BD levels, estimated by EPA models, had elevated rates of all types of leukemia, with RR = 1.37 (95% CI 1.05–1.78) and RR = 1.4 (95% CI, 1.07–1.81), respectively (Whitworth et al. 2008). Although alkanes were the dominant components of total NMHCs in Beirut (46% in summer and 59% in winter), which is similar to global pattern urban areas, however, NMHC levels, particularly those of the C5–C8 alkane

Fig. 3 Patterns of source apportionment to cumulative cancer risk on a typical day both in summer (**a**) and winter (**b**) seasons
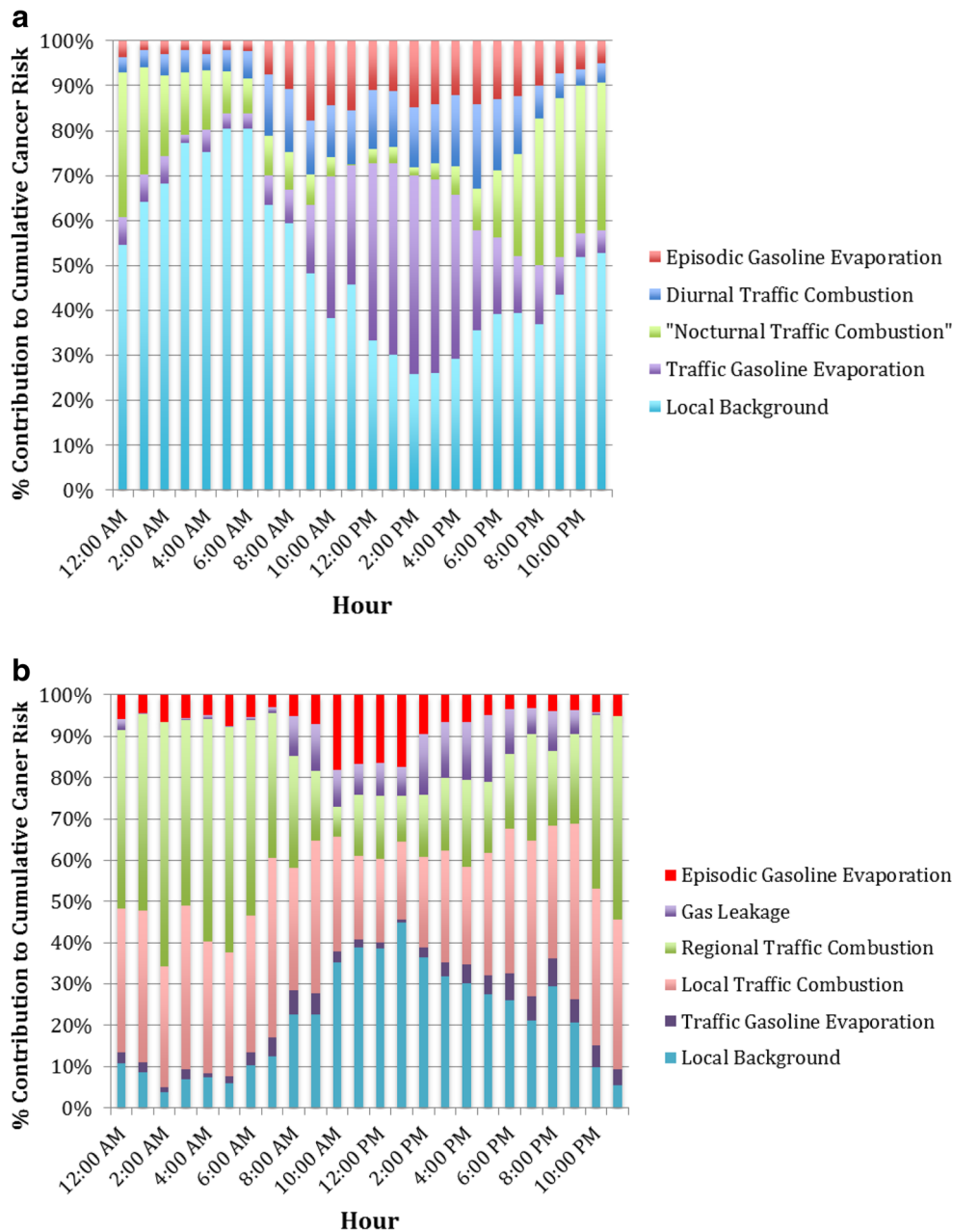
fraction, were two-fold higher compared to western megacities like Paris and Los Angeles, despite the relatively small surface area and city population (Salameh et al. 2015). However, the aliphatic C5–C8 fraction has a very negligible carcinogenic effect, including that of hexane, hence is not significant in terms of contribution to cancer risk in Beirut.

In addition to the heavy traffic in Beirut, power outages occurring daily for hours are compensated by privately owned small diesel generators in every residential and commercial building of the city (Waked et al. 2012). Because these generators are located in dense urban areas, their emissions may significantly impact ambient air quality and health risk. These power generators are emitting aliphatic and aromatic hydrocarbons

with potential carcinogenicity. According to a recent study, power generators account for 24% of $PM_{2.5}$ emissions in Beirut for an average power outage of 3 h and 23% of organic carbon in $PM_{2.5}$ concentrations (Waked et al. 2012, 2015). Another recent study on diesel generator emissions in Hamra—a residential/commercial block within the city of Beirut—reports a 60% increase in particle-bound polycyclic aromatic hydrocarbons (PAHs) above baseline for an average power outage of 3 hours (Shihadeh et al. 2013). In our study, we targeted 16 PAHs; however, only five were detected. Surprisingly, all the components of the Arom3 high-molecular weight carcinogenic PAHs, which are present mostly in the particle phase, were found to be below detection limit in the area of measurement. In contrast,

Table 4 Mortality burden for PM$_{2.5}$ elemental carbon (EC) fraction

		EC (μg/m^3)	Mortality per 1000 8.7	D 3283	RR	AF	AD	YLL
Summer Scenario								
	Mean EC concentration scenario	1.8			1.11	10%	327	3923
	EC worst-case scenario	3.8			1.25	19.9%	652	7825
Winter Scenario								
	Mean EC concentration scenario	1.4			1.08	7.8%	257	3086
	EC worst-case scenario	3.6			1.23	18.9%	621	7455

EC elemental carbon, *D* total number of deaths in the region of Beirut in the targeted year, *RR* relative risk, *AF* attributable fraction, *AD* attributable number of deaths, *YLL* years of life lost

regionally, a recent study of the atmospheric concentrations of particulate-bound PAHs in the city of Tehran reported up to 16 times increased cancer risk, in excess of the US EPA acceptable limit, and traced it back to traffic-related gasoline combustion and emission of Benzo[a]pyrene (Hoseini et al. 2016). In addition, analysis of PM$_{10}$ particles collected in urban Cordoba, Argentina, showed a lower PAHs attributable cancer risk compared to Tehran, but still in excess of the US EPA acceptable limit (Amarillo et al. 2014). Therefore, further studies in the future should target the aromatic fraction of high-molecular weight carcinogenic PAH components in a larger geographical area in Beirut in order to confirm the levels of high-molecular weight PAHs and to obtain a more accurate attributable cancer risk. On the other hand, we did analyze for carbon components of PM$_{2.5}$ and computed the mortality burden fraction (7.8–10%) and years of lost life attributable to elemental carbon—a biomarker of fuel combustion. Our assessment was at the city-scale and did not account for small-scale variations related to proximity to major roads. Elemental carbon is considered representative of the traffic-related particulate matter causally associated with mortality (Hoek et al. 2013).

High uncertainty normally accompanies risk assessment, particularly when only single point values are used to estimate the risk for a population (Evans et al. 1994). In our study, we applied the principle of additivity for mixture components according to the US EPA mixture guidance and methodology for quantifying non-cancer hazard and cancer risk within and across fractions (US EPA 1986, 1989, 1993, 2000). Considering challenges in substantiating assumptions for complex mixtures, especially when whole mixture toxicity data is not available for a specific site, the methodology we used is considered a default approach to evaluate potential health risks from exposure to aliphatic and aromatic hydrocarbon mixtures. At the mechanistic level, this approach is limited by the assumption of an independence of toxic action within and across fractions and the absence of interactions. However, it is important to note that synergistic or potentiating toxicological interactions among and within fractions are not likely to happen at the observed concentration levels. Another limitation in this

study relates to the phenomenon of weathering. Once released in the environment, the composition of a hydrocarbon fraction will change due to differential fate and transport of its components. Partitioning of the mixture will occur, such that, depending on reactivity, the more volatile compounds will migrate to other locations, while leaving the non-mobile components (the weathered product) at the original location. Thus, the actual aliphatic and aromatic hydrocarbon mixture to which a receptor population is exposed will vary with location, time, and environmental medium. In addition, an uncertainty exists in developing a quantitative estimate of cancer risk from inhalation of some of the components, such as hexane and the mid-range aliphatic hydrocarbon stream (AHS), since the weight-of-evidence indicates only a "Suggestive Evidence for the Carcinogenic Potential" for these components/fractions. However, the impact of hexane on our cumulative cancer risk estimate is minimal since hexane's contribution to cumulative cancer risk was found not to exceed 1%, while AHS had a larger contribution to total cancer risk (16–23%).

On the other hand, although human exposure and inhalation models may be used to model linkages between adverse health outcomes and air pollution and estimate individual exposure, however, in the absence of time-activity data that estimate amount of time spent in different microenvironments, this was not possible for this study (McCurdy et al. 2000). Instead, we followed an environmental concentration fraction-based model in conjunction with inhalation unit risk standards in order to estimate human health risk (Fryer et al. 2006). Alternatively, cohort designs may be more suitable and are needed in future studies to measure short- and long-term health effects and compare exposure levels and health responses in different geographical locations.

Conclusion

The presented data set is of high importance for Lebanon. The only available information on NMHC levels and their contributions to health risks are presented in this report. Our study

provides insight not only into the prevalence of air pollutants in urban Beirut, but also into the associated health risks for aliphatic and aromatic fractions and individual components. An excessive cancer risk is estimated to be mainly due to benzene, BD, and ethylbenzene exposure. Although generated risk estimates should not be interpreted as expected rates of disease due to uncertainties associated with the process of risk assessment, however, they do represent estimates of potential risk and can be used as a metric to prioritize health concerns. At the same time, a better mechanistic understanding of the interactive effects of mixtures is important in order to refine the predictive assessment tools and to reduce uncertainties associated with cancer risk estimates.

Our data do provide decision-makers with reliable guidance in order to mitigate pollution levels and develop appropriate management programs and policies at the national level, particularly aiming to reduce traffic-related air pollution in order to decrease cancer risk. Our results seem to suggest a significant contribution of air pollution to an excessive leukemia risk that needs to be investigated further, and an unclear contribution to other more incident cancers in the country, such as that of the bladder. However, this hypothesis could not be accepted with confidence unless large-scale comprehensive air monitoring programs are launched, covering all aliphatic and aromatic fractions, multiple seasons, and different urban agglomerations.

Compliance with ethical standards

Conflict of interest The authors decalre that they have no conflict of interest.

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