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Levels of PM2.5-bound species in Beijing, China: Spatio-temporal distributions and human health risks

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Levels of $PM_{2.5}$-bound species in Beijing, China: Spatio-temporal distributions and human health risks

By

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ABSTRACT

High concentrations of $PM_{2.5}$ and the corresponding health effect in Beijing, China have drawn attention worldwide. This study aims to assess the lifetime health risk of ambient $PM_{2.5}$ bound elements and polycyclic aromatic hydrocarbons (PAHs) from 2013 to 2015 in Beijing. Risk assessment methods of US Environment Protection Agency (USEPA) were applied to the following $PM_{2.5}$ components: six elemental components (Cr, Co, Ni, As, Cd, Pb) and sixteen EPA priority PAHs for cancer risks, and thirteen non- elemental components (Al, Ba, Cr, Mn, Ni, As, Cd, Pb, Co, V, P, Cl, Se) for non-cancer risks. Spatial and temporal variations of health risks were examined across Beijing. Source apportionment was applied to apportion the risks. The estimated lifetime cancer risk due to exposure to ambient $PM_{2.5}$ in Beijing is 2.30E-02. This cancer risk level is two magnitudes higher than EPA upper threshold of 1.00E-04. Thus, remediation is desirable. Lifetime non-cancer hazard quotient in Beijing is 13.7, higher than EPA upper threshold of non-cancer hazard quotient (1.0), indicating that some non-cancer health impacts may also occur. Seasonal cancer risks range 7.47E-03 to 4.78E-03. Summer and winter have higher percentages of 31% and 26% respectively to the total lifetime risk, while lower in spring (23%), and autumn (20%). Seasonal non-cancer hazard quotients in Beijing range 4.5 to 26.2. Winter contributed approximate 52% of total non-cancer hazard quotient, followed by spring (21%), autumn (18%), and summer (9%). Lifetime cancer risk is higher in suburban area (3.53E-02) than in urban area (1.82E-02), while hazard quotient is higher in urban area (14.3) than suburban area (2.6). Overall, both lifetime cancer and non-cancer risks in Beijing are higher than the corresponding USEPA threshold. The health risks in Beijing are all higher than other cities in China, Windsor, and Mexico.

DEDICATION

To my grandfather, who gave so much guidance. May you rest in peace.

To my parents, who were always supportive of my adventurous spirit.

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CHAPTER 1
INTRODUCTION

1.1 Background

Air pollution has become a serious problem worldwide. It is harmful to both human health and the environment. Environmental impacts of air pollution include acid rain and global warming. The hazardous effect on human health depends on the type of air pollutant, its concentration, and the length of exposure.

Human health effects of air pollution vary from nausea and difficulty in breathing or skin irritation, to cancer. Human health effects also include birth defects, serious developmental delays in children, and reduced activity of the immune system, leading to a number of diseases. Epidemiological and animal model data indicate that primarily affected systems are the cardiovascular and the respiratory system. However, the function of several other organs can be also influenced (Cohen et al., 2005; Kunzli and Tager, 2005; Sharma and Agrawal, 2005; Huang & Ghio, 2006). According to the World Health Organization (WHO) (2003), air pollution has caused more than 2.7 million deaths annually.

$PM_{2.5}$ is particulate matter (PM) with diameter of 2.5 um or less. Direct emission of $PM_{2.5}$ comes from combustion sources such as coal-oil-gasoline-diesel-wood combustion, high temperature industrial processes, and biomass burning. Secondary $PM_{2.5}$ comes from reactions of precursor gases such as sulphur dioxide (SO_2), nitrogen oxide (NOx), volatile organic carbons (VOCs), and ammonia (NH_3) (Hodan & Barnard, 2004; Pui et al., 2013).

PM pollution cause a number of health problems, including asthma attacks, heart attack, and lung cancer. It was also linked to premature death (AirNow, 2016). More than 500,000 deaths per year have been reported worldwide due to $PM_{2.5}$ pollution (Nel A., 2005). $PM_{2.5}$ is a better indicator for health threat than PM_{10} (particulate matter with an aerodynamic diameter of 10 um or less) (Schwartz et al., 1996). $PM_{2.5}$ has a smaller diameter, which allows it to penetrate deeper into the lungs to cause respiratory disease such as bronchitis, or worsen existing heart disease (Schwartz et al., 1996). Besides

health effects, $PM_{2.5}$ reduced visibility in large cities. In January 2013, when $PM_{2.5}$ concentration was 500-800 $\mu g/m^3$, the visibility in Beijing was less than 100 meters (Pui et al., 2013).

NOx are one of the major air pollutants. NOx mainly include nitric oxide (NO) and nitrogen dioxide (NO_2) gases. NOx is mainly emitted as NO and rapidly react with ozone or radicals in the atmosphere forming NO_2 (Kampa & Castanas, 2008). The primary sources of NOx are emissions from vehicles, electric utilities, and other industrial, commercial, and residential sources. The emissions from vehicles, power plants, and industries in 1999 accounted for 35%, 27% and 26% in Beijing urban area NOx emissions, respectively (Hao et al., 2005a).

Moreover, NOx can react with VOCs to form Ground-Level Ozone (GLO), which is known to cause a number of deleterious effects on health, including symptoms (e.g., eye discomfort, headache) (Ström et al., 1994), increased reactivity of the airways, inflammation of the lung, and decrements in lung function (Brunekreef, 1995). Chemical reactions of NOx in the atmosphere can cause acid rain, which brings a series of problems such as deterioration of buildings and the acidification of lakes (XRT, 2016). NOx can also be related to global warming, water quality deterioration, and visibility impairment (XRT, 2016).

The adverse impacts of human health caused by $PM_{2.5}$ have drawn attention worldwide. Risk assessment has been conducted to quantify public health risks associated with atmospheric exposure to $PM_{2.5}$. The main approaches used in most of the risk assessment studies are estimation of carcinogenic and non-carcinogenic risks, or endpoints-based studies.

In recent years, China's economic growth has resulted in increases of energy consumption, emissions of air pollutants, and poor air quality days. Air pollution, such as haze, occurs frequently. Haze has become one of the top environmental concerns in China (Chan & Yao, 2008). A regulation "Beijing Meteorological Disaster Warning Signal and Defense Guide" divided the haze level into yellow, orange, and red levels. Beijing is the capital of China with over 215 million residents by the end of 2014

(National Bureau of Statistics of the People's Republic of China, 2016). Air pollution is a major concern for the city residents (Bayraktar et al., 2010). $PM_{2.5}$ mass concentration higher than 35 μg/m^3 which is the polluted hour. A statistical study of Peking University showed that every week there were 119 polluted hours from 2010 to 2015 (Liang et al., 2015).

In order to quantify the risks due to air pollutants, elements, ions, and carbon fractions are often measured to evaluate the adverse effects (Chow et al., 2006; Yang et al., 2011). In China, studies associated with the chemical characterization of the pollutants were mainly conducted in super cities such as Beijing (e.g. Duan et al., 2006) and Shanghai (e.g. Ye et al., 2003).

The limitation of the studies conducted before includes: the carcinogenic and non-carcinogenic elements estimated are little (2 to 8), PAHs were not estimated with elemental components, and health risks were neither analyzed spatially nor temporally, nor connected to different sources in Beijing. In order to reduce the health risks, data support was needed to interpret the distribution of health risks by source, temporal variability, and spatial variability.

In order to solve the concern, $PM_{2.5}$ was selected in the project "Mobile Monitoring of Air Pollution Technology and Environmental Health Risk Modeling" to assess the cancer and non-cancer risks due to long-term non-occupational exposure and to analyze the distributions of health risks in source differences, temporal differences, and spatial differences in Beijing, China. This study is part of the above project, besides Beijing, several temporal health risk assessments were also conducted in Windsor, a city in Ontario, Canada, as Windsor is part of the project. The main reason to conduct health risk in Windsor is to provide a comparison with Beijing. Although some previous studies can provide the health risks in different cities, the elements used and parameters, such as body weight and exposure day, in previous studies were different from in this study. The same element and parameter were applied to Windsor. Thus, comparison between Beijing and Windsor can better reflect the pollution situation. The results from this risk assessment can help policy maker to understand the pollution distribution in Beijing and to make emission control strategies.

1.2 Objectives

The overall objective of this thesis is to quantify the carcinogenic and non-carcinogenic risks due to long-term, non-occupational exposure of ambient $PM_{2.5}$ in Beijing and in Windsor.

The specific objectives are:

(1) To estimate the lifetime cancer and non-cancer risks in Beijing from 2013 to 2015 and to conduct spatial and temporal analysis.
(2) To estimate lifetime cancer and non-cancer risk for Windsor from 2013 to 2015 and to conduct temporal analysis.
(3) To compare the results in Beijing with the results of Windsor and other cities worldwide.

CHAPTER 2
LITERATURE REVIEW

2.1 $PM_{2.5}$ and NOx pollution

There are series of policies and regulations worldwide to divide and classify air pollution level. Details of the guideline levels for $PM_{2.5}$ and NOx are presented in Table 2-1. For annual $PM_{2.5}$ exposure, the new Chinese National Ambient Air Quality Standards (CNAAQS) (GB3095-2012) issued by Chinese government has the higher tolerance than National Ambient Air Quality Standards (NAAQS) by United States Environmental Protection Agency (USEPA) and the WHO AQG. The highest annual $PM_{2.5}$ level is CNAAQS Grade II with 35 μg/m^3, however Beijing is still far above the Grade II level. According to He et al. (2001), annual average $PM_{2.5}$ concentration in Beijing Chegongzhuang sampling site from July 1999 to September 2000 was 115 μg/m^3, more than three times higher than CNAAQS Grade II level. A decreasing trend could be seen for the same sampling site from August 2001 to September 2002, the average $PM_{2.5}$ concentration was reduced to 96.5 μg/m^3, 2.8 times higher than CNAAQS Grade II standards. Nevertheless, the annual average concentration is still too high.

Table 2-1: Concentration limits for $PM_{2.5}$ and NOx in CNAAQS (MEP & AQSIQ, 2012), NAAQS (USEPA, 2016a), and WHO AQG (WHO, 2005).

Pollutants	Averaging time	CNAAQS (μg/m^3)		NAAQS (μg/m^3)	WHOAQG[(a)] (μg/m^3)
		Grade-I	Grade-II		
$PM_{2.5}$	Annual	15	35	12	10
	Daily	35	75	35	25
NO_2	Annual	40	40	53[(d)]/99.6	40
	Daily	80	80		/
	Hourly	200	200	100[(d)]/188	200
NOx	Annual	50	50	/	/
	Daily	100	100	/	/
	Hourly	250	250	/	/

Notes:

(a). These numbers have units of ppb. They were converted from ppb to μg/m3 by times the conversion coefficient 1.88 under 101.325 kPa and 25 °C.

NOx and NO_2 concentrations are usually well correlated, hence NOx concentrations can be estimated through measurements of NO_2 concentrations. Values of annual average NO_2 concentrations in CNAAQS are 40 μg/m^3 as shown in Table 2-1. From 1998 to 2005, the annual averages of NO_2 concentrations in Beijing stayed at a level of 70 μg/m^3± 10% (BEB, 1998-2005), as shown in Figure 2-1. Although annual average NO_2 concentrations have remained almost constant, the annual averages of NO_2 concentrations in Beijing still exceed the CNAAQS Grade-II standard, 40 μg/m^3 (MEP & AQSIQ, 2012).

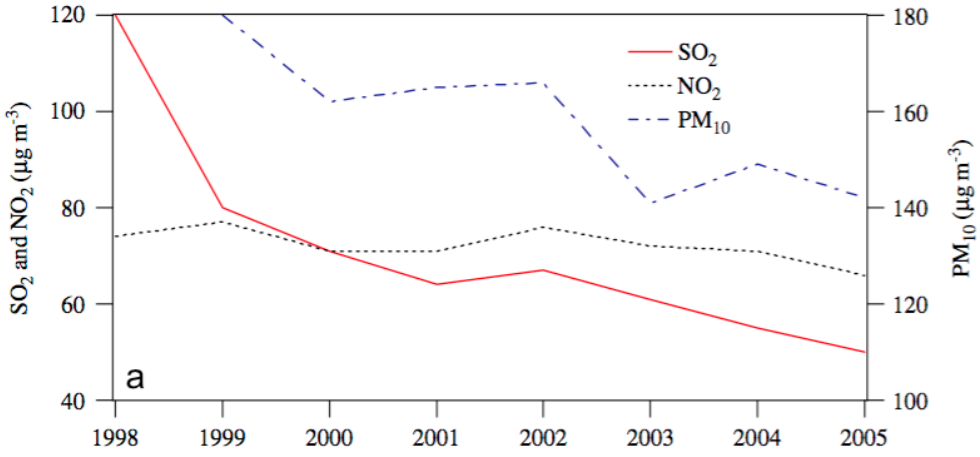


Figure 2-1: Annual variations in selected air pollutants in Beijing from 1998 to 2005 (Graph from: Beijing Environmental Bulltin. (1998-2005). http://www.bjepb.gov.cn.)

In order to find out the sources of air pollution, studies of source apportionment of $PM_{2.5}$ and NOx have been conducted. Previous studies (Cao et al., 2002; Sun et al., 2004) have indicated that, the major emission sources of PM in Beijing are industrial emissions, vehicles exhausts, dust, and coal burning. According to a study of Zheng et al. (2005), the major sources of $PM_{2.5}$ mass in Beijing were determined as dust (20%), secondary sulfate (17%), secondary nitrate (10%), coal combustion (7%), diesel and gasoline exhaust (7%), in 2000. Another study identified six main sources of $PM_{2.5}$ in Beijing Xinzhen sampling site from 2007 to 2013. Five major sources of $PM_{2.5}$ were identified According to 19 elements -- coal burning (29.2 %), vehicle exhaust and waste incineration (26.3 %), construction industry (23.3 %), soil (15.4 %), and industry with chlorine (5.9 %) (Jin et al., 2015).

In 2000, source apportionment of $PM_{2.5}$ was conducted in Beijing metropolitan area. Emissions from combustion sources, such as coal burning and oil burning, and the secondary products from photochemical reactions contributed 30% and 31% of $PM_{2.5}$ in Beijing, respectively (Song et al., 2006). In addition, a study according to 121 daily $PM_{2.5}$ samples collected in Beijing reported that, soil dust, coal combustion, biomass burning, traffic and waste incineration emission, industrial pollution, and secondary inorganic aerosol were the main sources of $PM_{2.5}$ (Zhang et al., 2013).

NO_2 emissions in Beijing were growing between 1990 and 1995 with the transportation sector contributing to the largest increase of 62%. The increases could also be seen in industrial from 187 kilotonnes (kt) to 243 kt. Among the increases created by industry, power and domestic sectors have contributed to growth of 26%, 20%, and 21%, respectively (Streets et al., 2000). In 1999, 67% of the ground NOx was due to vehicular emissions while power plants and industrial sources only contributed 2% and 13%, respectively (Hao et al., 2005b).

2.2 Health impacts of $PM_{2.5}$ and NOx

According to epidemiological studies (Pope et al., 1995a,b, 2002, 2004; Schwartz et al., 1996; Xu et al., 1995a,b; Morgan et al., 2003; USEPA, 2009), PM proves to be a risk factor for serious public health problems including acute and chronic respiratory illnesses, cardiovascular diseases, and premature death. According to WHO, approximate 800,000 premature deaths worldwide each year can be attributed to the effects of urban outdoor air pollution, of which approximate 300,000 occur in China (Cohen et al., 2004, Zhang and Smith, 2007). Globally, approximately 712,000 cardiopulmonary diseases (CPD) and 62,000 lung cancer deaths were attributed to urban $PM_{2.5}$ in 2000 (Cohen et al., 2004). More than 3,500,000 CPD and 220,000 lung cancer mortalities were caused by anthropogenic $PM_{2.5}$ annually (Anenberg et al., 2010).

Using remote sensing data, Evans et al. (2013) found that the global fraction of adult mortality attributed to the component of $PM_{2.5}$ is 8.0 % for CPD and 12.8 % for lung

cancer. Pope III et al. (2002) conducted a health endpoint study and found that each 10 $\mu g/m^3$ elevation in fine particulate air pollution was associated with approximately a 4%, 6%, and 8% increased risk of all-cause, lung cancer, and cardiopulmonary mortality in the United States of America (USA).

In the USA, the Integrated Science Assessment for Particulate Matter (ISAPM) (2009) indicated that, the causal relationship with short-term and long-term $PM_{2.5}$ exposure exists in cardiovascular effects and mortality, while a causal relationship is likely to exist between $PM_{2.5}$ exposure and respiratory effects. However, these conclusions could not be simply extrapolated to developing countries such as China, due to the differences of the characteristics of $PM_{2.5}$ and socioeconomic statues (HEI, 2010).

In China, several epidemiological studies (Chen et al., 2011; Zhang et al., 2012; Li et al., 2013) have established the relationship between PM concentrations and human illnesses associated with mortality and morbidity. Nevertheless, these studies have focused on particular periods of a few years or less, and according to limited ground-based $PM_{2.5}$ and PM_{10} measurements, which may not represent the situation in the whole city region. In order to address these concerns, an estimated linear regression model for aerosol optical depth (AOD) and annual premature mortality from different diseases caused by $PM_{2.5}$ was used to conduct a 12-year study for the period 2001–2012 in Beijing central area. AOD was defined as negative natural logarithm of the fraction of radiation (e.g., light) that is not scattered or absorbed on a path. It is a measure of the extinction of the solar beam by dust and haze (NOAA Earth System Research Laboratory, 2005). And it was based on observations from the Moderate Resolution Imaging Spectroradiometer on NASA's satellite (NASA, 2017). The annual average total mortality due to $PM_{2.5}$ from 2001 to 2012 in Beijing central area estimated 5100 individuals (Zheng et al., 2015).

When estimating the health effects of NO_2, it is often difficult to separate effects of NO_2 from other pollutants, especially PM (Frampton & Greaves, 2009). Epidemiology studies show that increased concentrations of NOx can be linked with reduced lung function (Linn et al., 1996), increased asthma symptoms (Schildcrout et al., 2006), and increased emergency hospital visits (Bates, 1992). ISAPM concluded it is sufficient to infer a likely causal relationship for respiratory effects following short-term NO_2 exposure, but

inadequate to prove the existence of a causal relationship between long-term exposure to NO_2 and associated endpoints (shown in Table 2-2).

Table 2-2: Causality judgments made in the Integrated Science Assessment for endpoints associated with long term NO_2 (USEPA, 2008).

HEALTH OUTCOME	CONCLUSION FROM PREVIOUS NAAQS REVIEW	CONCLUSION FROM 2008 ISA
LONG-TERM EXPOSURE TO NO_2		
Respiratory Morbidity	**No Overall Conclusion**	**"suggestive but not sufficient to infer a causal relationship"**
Respiratory Effects	NO_2 can cause emphysema (meeting the human definition criteria) in animals at high concentrations of NO_2.	Epidemiologic studies observed decrements in lung function growth associated with long-term exposure to NO_2.
Other Morbidity	**No Studies**	**"inadequate to infer the presence or absence of a causal relationship"**
Cancer	No Studies	Limited epidemiologic studies observed an association between long-term NO_2 exposure and cancer; animal toxicological studies have not provided clear evidence that NO_2 acts as a carcinogen.
Cardiovascular Effects	No Studies	Very limited epidemiologic and toxicological evidence does not suggest that long-term exposure to NO_2 has cardiovascular effects.
Birth Outcomes	No Studies	The epidemiologic evidence for an association between long-term exposure to NO_2 and birth outcomes is generally inconsistent, with limited support from animal toxicological studies.
Mortality	**No Studies**	**"inadequate to infer the presence or absence of a causal relationship"**
All Cause and Cardiopulmonary Mortality	No Studies	The results of epidemiologic studies examining the association between long-term exposure to NO_2 and mortality were generally inconsistent.

(Table from: USEPA. (2008). Risk and Exposure Assessment to Support the Review of the NO_2 Primary National Ambient Air Quality Standard. http://www3.epa.gov/ttn/naaqs/standards/nox/data/20081121_NO2_REA_final.pdf.)

2.3 Risk Assessment Methods

Two major approaches were used to estimate the risks in previous studies for $PM_{2.5}$. The first one is the health endpoint studies (including morbidity and mortality). The second one is to estimate carcinogenic and non-carcinogenic risks according to the carcinogenicity of components and corresponding unit risk (UR) and Reference concentration (RfC).

2.3.1 Health endpoint studies

The epidemiological studies assessed the risks of the $PM_{2.5}$ according to corresponding health endpoints. Commonly used epidemiological approaches are time-series analyses and cohort studies (Englert, 2004).

Frequently focused health endpoints are relationship of PM concentrations to mortality (total and cause-specific) and morbidity, hospital admissions due to cardiovascular and respiratory diseases, lung function, and functional endpoints, e.g., heart rate variability (HRV) (Englert, 2004). In epidemiological studies with health endpoints, relative risk (RR) is a measure commonly used to characterize the comparative health effects associated with a particular air quality comparison. According to this study, population data (density, distribution, and age profile), estimated air pollution exposure, and the baselines of the diseases, RRs were usually used when an increment of air pollution yield a different percentage in different populations (Levy., 2002).

Risk estimation

In a recent health endpoint study, mathematical forms of RR functions were selected and fitted for cause-specific mortality in adults: ischemic heart disease, cerebrovascular disease (stroke), chronic obstructive pulmonary disease, and lung cancer (Burnett et al., 2014). These RR functions were used by Colin J.L. et al. (2015) to estimate global mortality attributable to $PM_{2.5}$ exposure. RRs of the pollutants by the time-averaged $PM_{2.5}$ concentration were calculated rely on information on the RRs of mortality at specified $PM_{2.5}$ exposure concentrations from the available literature (Lee et al., 2015; Burnett et al., 2014). The equation is shown below:

$$RR=\begin{cases}1+\alpha\left(1-e^{-\beta(x-X)^{\rho}}\right), & \text{if } x > X\\ 1, & \text{otherwise}\end{cases} \qquad (2\text{-}1)$$

where, x= time-averaged $PM_{2.5}$ concentration at 10 km resolution; X=theoretical minimum risk exposure, which is defined as the concentration below which it is assumed there are no health effects; α , β , ρ=parameters describing the shape of the exposure-response curve, these parameters could be estimated from Monte Carlo simulations as performed by Burnett et al. (2014).

2.3.2 Cancer and non-carcer risks studies

When estimating the cancer and non-cancer risks, the components of $PM_{2.5}$ were classified into groups according to the carcinogenicity they may bring to human health. In 2001, the International Agency for Research on Cancer (IARC) classifies the cancer components into five classes to evaluate the possibilities of an individual for developing any type of cancer from lifetime exposure to carcinogenic hazards: 1. Carcinogenic to humans; 2A.Probably carcinogenic to humans; 2B.Possibly carcinogenic to humans; 3. Not classifiable as to its carcinogenicity to humans; 4. Probably not carcinogenic to humans.

Adapted from IARC classification, USEPA developed Classification System for Categorizing Weight of Evidence for Carcinogenicity from Human and Animal studies in 1986 (USEPA). In the 1986 guideline, the agents were classified into five categories: A. Human Carcinogen; B. Probable Human Carcinogen; C. Possible Human Carcinogen; D. Not classifiable as to Human Carcinogenicity; E. Evidence of Non-carcinogenicity for Humans (USEPA, 1986).

The 1986 guideline was updated in 2005 by USEPA. The 2005 guideline suggests to use narrative statements to express weight of evidence. It also separates the exposure from oral to inhalation routes. The general categories recognized by the 2005 guidelines are: 1. Carcinogenic to Humans; 2. Likely to be Carcinogenic to Humans; 3. Suggestive Evidence of Carcinogenic Potential; 4. Inadequate Information to Assess Carcinogenic Potential (USEPA, 2005).

Choice of classification of $PM_{2.5}$ components varied in different studies. According to the classification of the components, carcinogenic risks were assessed by each of $PM_{2.5}$ component. For carcinogens, absolute risks (ARs) are estimated by using the estimated average concentration of pollutants multiplied by their corresponding UR (Payne-Sturges et al., 2004; Hu et al., 2012; Yang et al., 2013), or according to the contaminant concentration of each component, to calculate the lifetime average daily concentration. Multiply the lifetime average daily concentration by the SF to get cancer risk (Greene et

al., 2006). For non-carcinogens, hazard quotient (HQ) was calculated by a direct comparison of the exposure concentration with a chemical-specific reference concentration (USEPA, 2011a).

As mentioned above, AR calculation can be performed with UR or with SF. UR is the inhalation cancer potency estimated according to the slope of the dose-response curve. The UR represents excess cancer risk over background associated with continuous lifetime exposure to a pollutant and is typically expressed as risk or probability of cancer for a 70-yr exposure per 1 $\mu g/m^3$ pollutant air (USEPA 1986). URs are derived either from occupational studies in humans, typically adult males, when available, or from toxicological studies in animals (Woodruff et al. 2000). A UR according to human data is typically defined as the maximum likelihood estimate representing a "best estimate" of the dose response in the occupational study population and are somewhat less conservative than upper bound estimates (Woodruff et al. 2000). UR and SF can convert to each other.

URs according to animal data are the upper 95% confidence bound of the estimated cancer potency. The use of upper bounds is generally considered a health-protective approach for covering the risk to susceptible individuals (USEPA 2003). When needed information was absent, a non-threshold, linear model is assumed for cancer when extrapolating from high dose to low dose and/or from animals to humans (USEPA 1986; 2005).

$PM_{2.5}$ *components analysis*

The composition of the $PM_{2.5}$ varies significantly with geographical location, local climate, season, industry, and traffic (Bhatnagar, 2004). Previous studies have characterized the components of $PM_{2.5}$ to estimate the health effects (Greene & Morris, 2006; IE, 2009; Hu et al., 2012; Yang et al., 2013), and seasonal variation (Yang et al., 2014; Cao et al., 2012; Liu, 2014) caused by different components, or to trace the emission sources (Sun et al., 2004; Yang et al., 2013).

The major $PM_{2.5}$ components can be classified into 3 groups: ions, elements, and carbon fractions. Ions include SO_4^{2-}, NO_3^-, NH_4^+, Na^+, Ca^{2+}, Mg^{2+}, Cl^-, K^+, and F^-. Elements

have 25 species, they are Al, Ba, Ca, Cr, Mn, Ni, Cu, Zn, As, Cd, Pb, Co, Fe, Sr, Ti, Mg, K, V, P, Cl, S, Si, Se, Br, and Na. Carbon fractions include organic carbon (OC) and elemental carbon (EC). PAHs are part of the OC that have been proved to be carcinogenic.

The risk brought by PAHs was calculated by using the Benzo[a]pyrene equivalents based PAH concentration. However, the elemental carbon, ions, and 12 species of the elements (Ca, Fe, Sr, Ti, Mg, K, S, Si, Br, Cu, Zn, and Na) were not used to estimate the risk, because these components were not assessed as carcinogenic or mutagenic under Integrated Risk Information System (IRIS), and the corresponding UR and (or) RfC could not be found from regional screen level (RSL) table either (USEPA, 2015b). The rest of the 13 elements and 16 EPA-priority PAHs (USEPA, 1994) were classified according to the classification mentioned before, and corresponding UR of components and/or RfC were also be found out from IRIS or RSL.

In order to analyze the association between particulate emissions and public health, the components of particulates that individuals are being exposed to and at what level is the exposure must be considered. A series of researches (Yang et al., 2013; Hu et al., 2012) have characterized the fine particulates by ions, elements, and PAHs. Besides, the new NAAQS issued by Chinese government has for the first time included the limits of Cd, Hg, As, Cr (VI) in the reference for local governments to set up local ambient air quality standards.

Previous studies suggest that specific $PM_{2.5}$ components are responsible for the majority of health effects, especially toxic trace metals (Protonotarios et al., 2002). Most of the toxic trace metals in the air are contained in fine particles with a size distribution of 1.0 μm or less, such as nickel (Ni), arsenic (As), and chromium (Cr) (Greene & Morris, 2006). Several studies assessed carcinogenic risks according to the characterization of $PM_{2.5}$ have estimated the health effects caused by these elements (shown in Table 2-3).

Table 2-3: Health risks from $PM_{2.5}$ components.

Author	City	Time	Components	Cancer risk	Hazard quotient
Wang et al., 2008	Beijing	Dec 2005-Jan 2006	16 EPA priority PAHs	4.48E-04	/
Wu et al., 2014	Shanghai	Oct 2011-Mar 2012	16 EPA priority PAHs	4.56E-06	/
Zheng et al., 2014	Hefei	Aug 2013-Jan 2014	16 EPA priority PAHs	3.86E-06	/
Fei et al., 2015	Huzhou	June 2013-March 2014	16 EPA priority PAHs	8.70E-06	/
Zhou et al., 2015	Wuhan	Nov-13	16 EPA priority PAHs	8.06E-05	/
Jiao et al., 2016	Jinan	Jan-Dec 2014	16 EPA priority PAHs	6.80E-06	/
Wang et al., 2016	Xiangtan	Dec 2015-Feb 2016	16 EPA priority PAHs	1.76E-06	/
R.V.Diaz et al., 2009	Mexico	Dec 2004-Mar 2005	Cl, V, Cr, Mn, Ni, Pb	/	0.96
Hu et al., 2012	Nanjing	Apr-Sep 2010	As, Cd, Co, Cr, Ni, Pb for cancer risk/ As, Cd, Co, Cr, Ni, Mn for hazard quotient	8.01E-05	2.96
Yang et al., 2013	Jinan	Dec 2007-Oct 2008	Cr, Mn, Ni, As, Cd, Pb, Co, and V.	/	0.86
Yang et al., 2014	Changsha	Oct-Dec 2012	As, Cd, Pb	8.44E-07	/
Zhuang et al., 2016	Xiamen	Not given	Cr, Ni, Cd for cancer risk/ Cr, Mn, Ni, Cd, Pb for hazard quotient	9.49E-08	7.74E-05
Li et al., 2016	Guangzhou	Jan-Dec 2015	As, Cd, Cr, Ni for cancer risk /Pb, Mn for hazard quotient	1.05E-05	1.97E-08
Zhang et al., 2016	The Pearl River Delta Region	2014-2015	Pb, Ni, Cr, Cd, As for cancer risk /As, Cd, Cr, Ni for hazard quotient	2.24E-04	1.43

Although there have been many recent studies on PAH concentrations and heath effects, most do no simultaneously consider the health risk, spatial variability, and temporal variability, for example, seasonal cancer risks. $PM_{2.5}$-bound 16 EPA priority PAHs were sampled in Beijing from December 2005 to January 2006 (Wang et al., 2008). The BaP_{eq} concentrations were calculated for industrial area, residential area, village, parks and schools, and commercial area. The highest PAHs concentration was observed in commercial area with 90.8 ng/m^3 and the lowest concentration was observed in residential area with 11.6 ng/m^3. Cancer risks were not assessed in Wang et al, the corresponding cancer risks were calculated by the same method used in this study. The health risk in Wang et al is only related with PAHs concentrations. Spatial variability was estimated in Wuhan as well (Zhou et al., 2015). The results shown that the total PAHs concentration was the highest in industrial area, followed by urban area and suburban area. The corresponding cancer risks were not estimated in Zhou et al as well.

The PAHs concentrations in winter, autumn, and summer were sampled in Hefei, except for spring (Zheng et al., 2014). The seasonal PAHs concentrations were in order of winter > autumn > summer from 2013 to 2014. BaP_{eq} concentrations were estimated however, the cancer risks were not calculated. PAHs were also estimated in Huzhou (Fei et al., 2015). Seasonal variations were observed for total PAHs concentrations as winter > autumn > spring > summer. The BaP_{eq} concentrations estimated from June 2013 to March 2014 was 1.14 ng/m^3 with corresponding cancer risks of 8.7E-06 for adults and 6.0E-06 for kids. Seasonal variations were also observed in Jinan (Jiao et al., 2016). The total PAHs concentrations were the highest in winter, followed by spring, summer, and autumn. The lifetime cancer risks caused by PAHs were 6.8E-06 and 4.8E-06 for adults and kids.

The PAHs cancer risks for different gender and age groups were estimated in Xiangtan (Wang et al., 2016) and Shanghai (Wu et al., 2014). The PAHs cancer risks for adults in these two studies are all lower than EPA upper threshold with 1.76E-06 in Xiangtan from 2015 to 2016 and 4.56E-06 in Shanghai from 2011 to 2012. These studies contributed the health risks of PAHs in some cities. However, could have been expanded to consider seasonal and spatial variability

Cancer risks caused by elemental components were estimated in Nanjing (Hu et al., 2012), Changsha (Yang et al., 2014), Xiamen (Zhuang et al., 2016), Guangzhou (Li et al, 2016), and The PRDR (Zhang et al., 2016). Except for Nanjing, the elemental components used to estimate cancer risks are all less than six. There are only three elements used in Changsha and Xiamen. Cancer risks for different ages and genders were estimated in Changsha, Jinan, Guangzhou and Nanjing. Spatial and seasonal variability were calculated in Xiamen and the PRDR.

Non-cancer hazard quotients were estimated in Mexico (R.V.Diaz et al., 2009), Nanjing (Hu et al., 2012), Jinan (Yang et al., 2013), Xiamen (Zhuang et al., 2016), Guangzhou (Li et al., 2016), and the PRDR (Zhang et al., 2016). The non-cancer hazard quotient in Nanjing and the PRDR are 2.96 and 1.43 respectively, higher than EPA upper threshold of non-cancer hazard quotient. Non-carcinogenic elements estimated in these studies ranged from 2 to 8. Spatial and seasonal variability were only assessed in Xiamen and the PRDR.

In these previous studies, there were up to five carcinogenic elements and up to eight non-carcinogenic elements estimated. According to the literature review, there are six carcinogenic elements and 13 non-carcinogenic elements could be used to estimate health risks (Table 2-4). The papers estimated cancer risks of $PM_{2.5}$-bound PAHs did not take the cancer risks of elemental components into consideration. Spatial and temporal variability were not estimated in most of the papers. Besides, the cancer risks estimated were not apportioned into different sources. Thus, future study of the health risks caused by elemental components and PAHs, temporal and spatial variability analyses, and source apportionment of health risk were needed to provide data support for reducing the $PM_{2.5}$ emissions.

As mentioned above, there are thirteen elemental components could be used to estimate health risks according to the available URs and/or RfCs of the elements (Table 2-4). As mentioned above, the URs of the components could be converted to SFs by using the equation (2-2) with unit of $(\mu g/kg*day)^{-1}$.

$$SF = \frac{UR*BW}{IR} \qquad (2\text{-}2)$$

where, BW = body weight (kg), 70kg; IR = inhalation rate (m^3/day), 20 m^3/day. The SFs were listed in the Table 2-4 as well.

Table 2-4: Elemental components of $PM_{2.5}$ and corresponding unit risk, slope factor, and reference concentration.

	Classification [(a)]	Carcinogenic			Non-carcinogenic	
Chemical name		UR $1/(\mu g/m^3)$	SF $(\mu g/kg*day)^{-1}$	Source	RfC $(mg/m^{3)}$	Source
Al (Aluminum)	InI				5.00E-03	USEPA, 2016.
Ba (Barium)	D				5.00E-04	USEPA, 1997.
Cr (Chromium)	CH	1.20E-02	4.20E-02	USEPA, 2016b.	1.00E-04	USEPA, 2016b.
Mn (Manganese)	D				3.00E-04	ATSDR, 2012.
Ni (Nickel)	A	2.40E-04	8.40E-04	USEPA, 2016b.	9.00E-05	ATSDR, 2012.
As (Arsenic)	A	4.30E-03	1.51E-02	USEPA, 2016b..	1.50E-05	Cal EPA, 2000.
Cd (Cadmium)	B1	1.80E-03	6.30E-03	USEPA, 2016b.	1.00E-05	ATSDR, 2012.
Pb (Lead)	B2	8.00E-05	2.80E-04	Cal EPA, 2000.	1.50E-04	USEPA, 2016c.
Co (Cobalt)	2B	9.00E-03	3.15E-02	ATSDR, 2012.	1.00E-04	ATSDR, 2012.
V (Vanadium)					1.00E-04	ATSDR, 2012.
P (Phosphorus)	D				7.00E-05	Cal EPA, 2000.
Cl (Chlorine)					1.50E-04	ATSDR, 2012.
Se (Selenium)	D				2.00E-02	Cal EPA, 2000.

Notes:

(a) Classifications are according to: EPA WOE (2005 Guidelines) = weight of evidence for carcinogenicity under 2005 EPA cancer guidelines: CH-carcinogenic to humans; LH - likely to be carcinogenic; SE-suggestive evidence of carcinogenic potential; InI-

inadequate information to assess carcinogenic potential; NH -not likely to be carcinogenic).

EPA WOE (1986 Guidelines) = weight-of-evidence for carcinogenicity under the 1986 EPA cancer guidelines: A -human carcinogen; B1 – probable carcinogen, limited human evidence; B2 - probable carcinogen, sufficient evidence in animals; C – possible human carcinogen; D - not classifiable; E - evidence of noncarcinogenicity.

IARC WOE = weight-of-evidence for carcinogenicity in humans (1 - carcinogenic; 2A - probably carcinogenic; 2B – possibly carcinogenic; 3 – not classifiable; 4 - probably not carcinogenic).

PAHs components in $PM_{2.5}$

Polycyclic aromatic hydrocarbons (PAHs) in $PM_{2.5}$ are part of the carbon fractions of $PM_{2.5}$. PAHs often show up as complex mixtures of many components with a wide range of the toxic potency (Nisbet, C. et al, 1992). PAHs in the environment attract more and more attention due to their widespread occurrence and their toxic, mutagenic, and carcinogenic potentials (Freitag et al., 1985). Several compounds of this group have been classified by the IARC as probable (2A) or possible (2B) human carcinogens (IARC, 2011).

PAHs are components of OC on $PM_{2.5}$ and are mutagenic products of incomplete combustion (Zhang et al., 2009; Birch et al., 1996). It was estimated that an annual average of 6.5 per million people in China have lung cancer due to PAH inhalation exposure (Zhang Y et al., 2009). In Beijing residents during 1997, an estimated 1.7% of cancers diagnosed was attributable to PAH pollution in the air (Yu et al., 2008). The 4–6 ring PAHs, namely BaA, Chr, BbF, BkF, BaP, InP, DBA, BghiP are considered to have carcinogenic effects (Li et al., 2006). USEPA listed 16 PAHs as priority-controlled pollutants their information is listed in Table 2-5 (USEPA, 1994).

The method of estimating inhalation cancer risk developed by Nisbet and LaGoy (1992) presented a toxic equivalency factor (TEF) for 17 PAHs to summarize and quantify

existing relative carcinogenicity data on PAHs according to the benzo[a]pyrene equivalent (BaP_{eq}) concentration of the individual PAHs. Along the similar line, the Office of Environmental Health Hazard Assessment (OEHHA) of California Environmental Protection Agency developed a potency equivalency factor (PEF) procedure to assess the relative potencies of PAHs (Collins et al. 1998; OEHHA, 2003). TEFs are used for multiple end points while PEFs are specific for the estimation of cancer risk because almost all PEFs for PAHs are according to cancer bioassay information. PEFs are slightly different. In this study, the cancer risk caused by PAHs according to the TEFs were estimated, because most of the studies conducted in Beijing between 2000 and 2016 were TEF-based. In this way, the consistency of the data calculation can be kept.

BaP was chosen as the primary representative of the PAHs due to three major reasons: (1) the relatively large amount of toxicological data available on BaP. (2) the availability of air-monitoring techniques for BaP, and (3) the known and frequent human exposure to BaP in airborne PAHs (Collins et al., 1998). Besides, BaP is the only PAH for which a complete quantitative risk assessment has been done (OEHHA, 1994; Collins et al., 1991). These 16 priority EPA-PAHs were used to estimate the corresponding cancer risks. The information of 16 priority EPA-PAHs have been collected and listed in Table 2-5.

Table 2-5: Sixteen priority EPA-PAHs and corresponding potency equivalency factor.

Name	Abbreviation	Classification (1)	TEF (2)	Name	Abbreviation	Classification (1)	TEF (2)
Naphthalene	Nap	C	0.001	Benz[a]anthracene	BaA	2A	0.1
Acenaphthylene	Acy	D	0.001	Chrysene	Chr	3	0.01
Acenaphthene	Ace	D	0.001	Benzo[b]fluoranthene	BbF	2B	0.1
Fluorene	Fl	3	0.001	Benzo[k]fluoranthene	BkF	2B	0.1
Phenanthrene	Phe	3	0.001	Benzo[a]pyrene	BaP	2A	1
Anthracene	An	3	0.01	Indeno[1,2,3,-cd]pyrene	InP	2B	0.1
Fluoranthene	Flu	3	0.001	Dibenz[a,h]anthracene	DBA	2A	1
Pyrene	Pyr	3	0.001	Benzo[g,h,i]perylene	BghiP	3	0.01

Notes:

(1) Classification is according to: EPA WOE (1986 Guidelines) = weight-of-evidence for carcinogenicity under the 1986 EPA cancer guidelines: A -human carcinogen; B1 – probable carcinogen, limited human evidence; B2 - probable carcinogen, sufficient evidence in animals; C – possible human carcinogen; D - not classifiable; E - evidence of non-carcinogenicity.

IARC WOE = weight-of-evidence for carcinogenicity in humans (1 - carcinogenic; 2A - probably carcinogenic; 2B – possibly carcinogenic; 3 – not classifiable; 4 - probably not carcinogenic).

(2) Nisbet and LaGoy, 1992.

$PM_{2.5}$ *mass concentration analysis method*

Particulate matter mass concentration is widely analyzed by gravimetric analysis method. This method is almost exclusively used in labs. Gravimetric measures the weight of the filter before and after sampling under the controlled temperature and relative humidity to minimize the potential bias caused by particle volatilization and aerosol liquid water (Chow & Watson et al., 1998).

Elements analysis method

Several widely used elemental analysis methods are Instrumental neutron activation analysis (NAA), atomic absorption spectrophotometry (AAS), inductively coupled plasma with atomic emission spectroscopy (ICP-AES) or with mass spectroscopy (ICP-MS), photon-induced X-ray fluorescence (XRF), and proton induced X-ray emission (PIXE). The comparison of these methods has been listed in the Table 2-6.

Table 2-6: Comparison of elemental analysis method (Geoff, 2016).

Analysis method	Preparation	Advantages	Disadvantages	Detection limits
NAA	None	1.Technically nondestructive 2. Low instrument cost	1.Do not quantify some of the abundant species in ambient particulate matter such as silicon, nickel, tin, and lead 2. Irradiation process makes the filter membrane brittle and radioactive 3.Relatively high cost	Extremely low
AAS	Sample dissolved	1. Useful complement to other methods 2. Relative low cost 3. Easy to use 4. Short operation time	1.Require individual analysis for each element 2. Require destruction of sample	High
ICP-AES	Sample dissolved	1. Multi-element capabilities 2. Acquire a large number of elemental concentrations using small sample volumes	1. Require complete extraction and destruction of the sample 2. High potential of spectral interference	Acceptable
ICP-MS	Sample dissolved (Acid digestion, water extraction)	1. Multi-element capabilities 2. Short operation time	1. Strongly affected by complex matrix effects 2. Destructive analysis	Low
XRF	None	1. Multi-element capabilities 2. Relatively low cost 3. Non-destructive 4. Wide element coverage	1. Possibly affected by matrix effects 2. Unsuitable for analysis of very light elements e.g. H to Ne. 3.Analysis of liquids requires the use of large volumes of He. 4.Fused bead preparation is time consuming and takes a certain amount of practice.	High

PIXE	None	1. Multi-element capabilities 2. Relatively low cost 3. Non-destructive	1. Unsuitable for analysis of very light elements	High

Several inter-comparison studies of these methods were conducted before. XRF was compared with ICP-OES for phosphorus in raw materials and ceramics, similar results were reported in this study (Marina & Lopez, 2001). Close agreement of results were reported for trace and major elements in soils determined with both XRF and ICP-AES (Hannaker et al., 1984). Limited comparative studies on their detection limits, accuracy and precision of determination of a variety of trace elements and heavy metals were available (McComb et al., 2014). No inter-adjustment data could be found to adjust the bias.

PAHs analysis method

The most commonly used analysis method for PAHs is the extraction of filters with suitable organic solvent followed by gas chromatography (GC) combined with mass spectrometry (MS) or other kinds of detectors. Other analysis methods such as combining GC-Fourier transform infrared (FTIR)-MS or high performance liquid chromatography (HPLC)-MS techniques could also be seen in some of the studies (Chow & Watson et al., 1998).

Risk estimation

One typical study (Payne-Sturges et al., 2004) was conducted before, the cancer risk for each VOC and corresponding study participant were estimated by using Equation:

$$E_{ij} * UR_j = R_{ij}, \qquad (2\text{-}3)$$

where, R_{ij} =the estimated risk from pollutant j for study participant i; E_{ij} = the measured exposure (indoor, outdoor, or personal) concentration ($\mu g/m^3$) for pollutant j for study

participant i; UR_j= the inhalation UR for pollutant j. Summary statistics (e.g., mean, median, percentiles) of the cancer risk estimates for each pollutant were calculated across study participants.

Usually, cancer risks are summed across all chemicals of concern and all exposure pathways that contribute to exposure of an individual in a given population. In general, the USEPA considers any cancer risk less than 1E-06 (one in a million) is negligible, and risks above 1E-04 to be sufficiently large that some sort of remediation is desirable. Cancer risks that range between 1E-06 and 1E-04 are generally considered to be acceptable (USEPA, 2015a).

For non-cancer effects, the default assumption is that the dose–response model has a threshold below which no adverse health effects are expected to occur. Non-cancer hazard quotients in this study were measured by a direct comparison of the exposure with a chemical-specific RfC. Non-cancer HQ was calculated as below:

$$HQ_{ij} = E_{ij} * RfC_j^{-1} \qquad (2\text{-}4)$$

where, RfC_j = non-cancer RfC for pollutant j (micrograms per cubic meter); HQ_{ij} =HQ for participant i for pollutant j.

An HQ below 1 indicates that there is no significant risk of non-cancer effects. On the contrary, an HQ above 1 indicates that there is a chance of non-cancer effects occurring, with a probability that tends to increase as the value of HQ increases (USEPA, 2011a).

A similar approach was used to calculate the health risks of arsenic and heavy metals in TSP and $PM_{2.5}$ in Nanjing, China (Hu et al., 2012), in Jinan, China (Yang et al.,2013), and in upper Columbia River/Lake Roosevelt (IE, 2009).

There is no sufficient epidemiologic study to demonstrate the relationship between long-term exposure to NOx and cancer. Besides, NOx is not listed as the carcinogenicity in USEPA. Among the papers reviewed, no studies about the health risk of NOx were conducted. Thus, in this study, cancer risk and non-cancer hazard quotient of NOx was not conducted

2.4 Source apportionment

Receptor models such Principal component analysis (PCA), Positive Matrix Factorization (PMF), and Chemical Mass Balance (CMB) were widely used to conduct source apportionment. Due to the limitation of data in this study, none of the receptor models could be used to do a source apportionment. Hence, a literature review was conducted to find other materials that may be useful in source apportionment.

It is well known that PMF can identify several kinds of possible factors. The results were usually shown in three kinds of formats—percentages of species, concentration, and percentages of factors. According to the results, the major contributors under different factors could be analyzed to find out the possible sources. Source profile can distribute the overall contribution into different sources by species. According to this, nine previous papers with PMF source profiles of $PM_{2.5}$ (exclude PAHs) and two $PM_{2.5}$-bound PAHs in Beijing area were found. The elements found in papers were compared with the elements investigated in this study (Table 2-4; Table 2-5). Missing elements were listed in Table 2-7:

Table 2-7: Missing elements in papers with PMF source profiles for (a) elemental components (b) PAHs.

(a) Elemental components

Author	Missing elements	Sampling time
Wang et al., 2015	Ni, Co, V, P, Cl, Se	Aug 2012 to Jul 2013
Song et al., 2006	Ba, Cd, Co	Jan, Apr, Jul, and Oct 2000
Yang et al., 2016	Ba, Cr, Mn, N, Co, V, P, Cl, Se	Oct, Nov, and Dec 2013
Gao et al., 2016	Al, Co, V, P, Cl	Jul 22nd to Aug 12th, 2014
Zíková et al., 2016	Ba, Cd, P, Se	2012-2013
Song et al., 2007	Ba, Ni, Cd, Co, P,	Jan and Aug 2004
Zhang et al., 2013	P, Cl	Apr, Jul, Oct 2009. Jan 2010
Yu et al., 2013	Cd, Co	2010
Jin et al., 2015	Ba, As, Co, V, Se	2007-2013

(b) PAHs

Author	Missing PAHs	Sampling time
Wu et al., 2014	Nap, Acy, Ace, Fl, DBA	Summer and winter in 2008 and 2009
Yu et al., 2008	None	Nov 2005- Nov 2006

Health risks could be distributed into different sources by using the distributed percentage of each element in each source. The estimated contributions from different sources could help to determine the emission control strategies. However, different numbers of sources, different species of elements, and different emission sources were used in different papers. It is hard to combine the sources in different papers or to take the averages for the same element in the same source. A justification was conducted in chapter 3 to interpret the way to choose which paper to use for source apportionment.

CHAPTER 3
METHODOLOGY

3.1 Data sources

3.1.1 In Beijing

$PM_{2.5}$ mass concentrations

There are three sources providing $PM_{2.5}$ mass concentration in Beijing. One official source is Ministry of Environmental Protection of the People's Republic of China (MEP), two unofficial sources are US Embassy in Beijing (US Embassy) and Qingyue environmental data open platform (QY). Besides providing $PM_{2.5}$ mass concentration directly, MEP also provides Air Quality Index (AQI), which can be used to back calculate the $PM_{2.5}$ mass concentration when the primary pollutant is $PM_{2.5}$ in that day. Details of these data sources have been listed in Table 3-1.

Table 3-1: Details of (a) Four methods and (b) Locations of 12 National Air Quality Monitoring Station (NAQMS).
(a) Four data sources

Data sources	MEP, 2016	QY, 2016.	US Embassy, 2016.	MEP, 2016.
Time period	2013-2016	2013-2016	2008-2015	2000-2012
Resolution	Daily/hourly average $PM_{2.5}$ mass concentrations	Daily/hourly average $PM_{2.5}$ mass concentrations	Hourly $PM_{2.5}$ mass concentrations	Daily/hourly average Air Quality Index (AQI)
Sampling sites	12	12	1	12
Strength	1) Official source 2) Free of charge 3) No need to back calculate 4) Continuous time series 5) 12 sampling sites available	1) Root source is official 2) No need to back calculate 3) Continuous time series 3) 12 sampling sites in Beijing	1) Free of charge 2) No need to back calculate 3) No need to back calculate 4) 12 sampling sits available 5) Relatively longer sampling period	1) Official source. 2) Free of charge. 3) 12 sampling sites available
Weakness	1) Historical data is not available 2) Time period relatively short	1) Overall charges 200 RMB 2) Platform is unofficial although the root source is 3) Time period relatively short	1) Not an official source 2) Only one sampling site available 3) Data in 2008 and 2009 have a large number of missing values	1) Need to back calculate 2) Historical data is only available for certain stations 3) Not a continuous time series (a) 4) Time period relatively short

(b) Locations of 12 National Air Quality Monitoring Stations (NAQMS)

Name	Abbreviation	Coordinates[(b)]	Name	Abbreviation	Coordinates	Name	Abbreviation	Coordinates
Wanshouxigong	WS	(116.36, 39.86)	Nongzhanguan	NZG	(116.47, 39.97)	Huairouzhen	HR	(116.64, 40.39)
Gucheng	GC	(116.22, 39.92)	Guanyuan	GY	(116.36, 39.94)	Changpingzhen	CP	(116.23, 40.19)
Dongsi	DS	(116.43, 39.95)	Haidianqu wanliu	HD	(116.31, 39.99)	Shunyixincheng	SY	(116.72, 40.14)
Tiantan	TT	(116.43, 39.87)	Aotizhongxin	ATZX	(116.40, 40.00)	Dingling	DL	(116.17, 40.28)

Note:

(a) $PM_{2.5}$ mass concentration could only be back calculated when $PM_{2.5}$ is the primary pollutant of the day.
(b) Coordinates were used to do closest distance analysis.

Choice of data source for $PM_{2.5}$ mass concentration in Beijing

MEP, the official data source, has the priority. However, in consideration of the difficulty of getting data, the backup data source, QY was chosen as the data source for $PM_{2.5}$ mass concentration in Beijing. The reasons are:

1) This platform can provide three-year historical $PM_{2.5}$ mass concentration.
2) Data are more completed with sampling sites in Beijing.
3) Data have more varieties. Daily-average and hourly-average concentrations are available for 12 sampling sites and each of 12 sites.
4) No need to back calculate, avoid secondary data error happened in calculation.
5) No limitations for data compare to calculate from AQI.
6) The data source is not free but the fee is acceptable.
7) The platform is unofficial but the root source is official. The platform promised didn't make up data.
8) The time period of the platform is relatively short. However Three-year period is still acceptable.

$PM_{2.5}$ elemental components concentrations

Lots of funding support is needed for sampling and analyzing the components of $PM_{2.5}$, hence, elemental component concentrations data source is rare. To date, a known official source that may have $PM_{2.5}$ elemental component concentrations is MEP. However, it is difficult to get from MEP. Another available method is to collect $PM_{2.5}$ elemental component concentrations from previous studies. Details of these two different sources were listed in Table 3-2.

Table 3-2: Data sources of $PM_{2.5}$ elemental component concentrations.

Data sources	MEP, 2016.	Collect from previous studies
Time period	2013-2016	2000-2015
Resolution	Daily average $PM_{2.5}$ elemental components concentrations	Most are daily average $PM_{2.5}$
Sampling sites	12	Research stations
Strength	1) Official source 2) Continuous time series	1) Easier to get data
Weakness	1) Data is not available	1) Component concentrations were estimated. 2) May have secondary error when calculating and estimating $PM_{2.5}$ components concentration.

According to the literature review I conducted before, several previous studies with $PM_{2.5}$ elemental components concentration were discovered. Elemental component concentrations were collected and listed in Table 3-3.

Table 3-3: Elemental component concentrations of $PM_{2.5}$ ($\mu g/m^3$).

Author	Sampling site	$PM_{2.5}$	Al	Ba	Cr	Mn	Ni	As	Cd	Pb	Co	V	P	Cl	Se
He et al., 2001	Chegongzhuang	129.0	0.800			0.097	0.015			0.304				2.05	0.009
Duan et al., 2006	Chegongzhuang	96.5						0.060		0.170				1.64	0.090
Yang et al., 2014	North Fifth-Ring Road	137.6			0.032	0.079	0.005	0.028	0.004	0.355					
Zhao et al., 2013	Beijing	123.5	0.970	0.020	0.020	0.070	0.010	0.030		0.140			0.090		
Yu et al., 2013	Beijing Normal University	55.5	0.491	0.088	0.022	0.062	0.028	0.038		0.117		0.015	0.028	2.20	0.038
Sun et al., 2006	Beijing Normal University	220.0	1.46		0.010	0.160	0.090	0.040		0.440	0.002	0.006			
Sun et al., 2006	Beijing Normal University	36.7	1.00		0.002	0.040	0.020	0.010		0.040	0.007	0.008			
Yu et al., 2012	China University of Geosciences	89.6						0.016	0.003	0.148					
Song et al., 2006	Ming Tombs, the airport, Beijing University, Dong Si EPB, and Yong Le Dian	96.1	0.400		0.020	0.070	0.020	0.030		0.300				1.69	0.010
Cui et al., 2008	North Forth-Ring Road	166.3	0.470		0.003	0.120	0.005	0.029	0.002	0.180	0.001	0.004		4.27	0.005
Tao et al., 2014	Peking University	135.0			0.011		0.004	0.012	0.003	0.143	0.001				0.003
Du et al., 2010	Qinghuayuan Community	110.5	0.606			0.100				0.193					
Zhao et al., 2013	Shangdianzi	71.79	0.570	0.010	0.040	0.040	0.010	0.010		0.070			0.060		
Duan et al., 2006	Tsinghua University	106.9						0.060		0.210				2.09	0.120
He et al., 2001	Tsinghua University	145.0	0.800			0.093	0.012			0.335				2.45	0.010

Sampling information of previous studies

Instruments used to analyze $PM_{2.5}$ mass concentrations and elemental component concentrations were also collected (if available) besides concentrations. $PM_{2.5}$ mass concentrations were analyzed by gravimetric analysis, and 5 out of 11 studies used ICP-MS solely or combined with ICP-AES to analyze elemental components in previous studies. Details are listed in Table 3-4.

Table 3-4: Sampling methods of elemental components from previous studies.

Author	Sampling site	Instrument	Resolution	Chemical analysis
He et al., 2001	Chegongzhuang, Tsinghua University	Low-flow rate sampler, LFS Teflon filter (Ø = 47 mm)	Daily average concentration from Jul 1999 to Sep 2000.	Mass: Gravimetry analysis Elements: XRF
Sun et al., 2006	Beijing Normal University	Medium-volume samplers Flow rate: 77.59 L/min Whatman 41 filters	Hourly average concentration from Dec 4th to Dec 6th, 2004.	Elements: ICP-AES
Duan et al., 2006	Chegongzhuang, Tsinghua University	Low-flow rate samplers Teflon-membrane filter (Ø = 47 mm)	Daily average concentration from Aug 2001 to Sep 2002.	Elements: XRF
Song et al., 2006	Ming Tombs, the airport, Beijing University, Dong Si EPB, and Yong Le Dian.		Six-day average concentration from Jan, Apr, Jul, and Oct 2000.	Elements: XRF
Cui et al., 2008	North Forth Ring Road	Medium-volume samplers Flow rate: 77.36 L/min QR-100 Quartz filters	Daily average concentration from Mar 3rd to 8th, 10th to 14th, 2006.	Elements: ICP-MS and ICP-AES
Du et al., 2010	Qinghuayuan Community	Personal exposure monitor Teflon filters (Ø = 37 mm)	Daily average concentration from Oct 2006 to Nov 2007.	Mass: Gravimetric analysis Elements: NAA for Mn, Al, Ca. ICP-MS for Pb, Fe.

Continued from Table 3-4.

Yu et al., 2012	China University of Geosciences	Mini Volume Sampler Flowrate: 3.33 L/h Whatman filter (Ø = 47 mm	Weekly average concentration from Apr 18th, 2005 to May 26th, 2008. Daily average concentration from Jul 14th to Sep 27th, 2008.	Mass: Gravimetry analyasis Elements: ICP-MS
Zhao et al., 2013	Beijing, Shangdianzi	Medium-volume samplers Flowrate: 100 L/min. Polypropylene filters (Ø = 90 mm)	Daily average concentration from Apr 6th to May 1st, 2009, Jul 9th to 4 Aug 4th, 2009, Oct 11th to 4th Nov 2009, and Jan 14th to Feb 8th, 2010.	Elements: ICP-AES
Yu et al., 2013	Beijing Normal University	Automated Cartridge Collection Unit Flowrate: 16 L/m Teflon filters (Ø = 47 mm)	Daily average concentration from Jan to Dec 2010.	Elements: PIXE
Yang et al., 2014	North Fifth-Ring Road	Low-volume air sampler Flowrate: 16.7 L/min Teflon membrane filters (Ø = 47 mm)	Daily average concentration from Sep 2012 to Aug 2013.	Mass: Gravimetric analysis Elements: ICP-MS
Tao et al., 2014	Peking University	frmOMNITM sampler Flowrate: 5L/min Whatman PTFE Teflon	Daily average concentration from Apr, Jul, Oct 2009 and Jan 2010.	Mass: Gravimetry analysis Elements: ICP-MS

Choice of data source for elements in Beijing

Official data source MEP has the priority to be used in this study. Because data from MEP could not be gotten, data collected from previous studies were used.

$PM_{2.5}$-bound PAHs concentrations

Data sources for $PM_{2.5}$ bound PAHs are from previous studies, similar to data sources for $PM_{2.5}$ elemental components. Details of these two data sources are similar with Table 3-2. Unlike $PM_{2.5}$ elemental components, available data source for $PM_{2.5}$ bound PAHs is limited. The available data are listed in the Table 3-5.

Table 3-5: List of PAHs concentrations (μg/m^3 for $PM_{2.5}$; ng/m^3 for individual PAHs) in Beijing.

Author	Wang et al., 2008.	Liu et al., 2008.	Hou et al., 2006	Yu et al., 2008	Cui et al., 2008	Duan et al., 2009.
Sampling sites	20 sampling sites	6 sampling sites	Beijing Normal University	China University of Petroleum	North Forth-Ring Road	Not mentioned
$PM_{2.5}$		103		95.5	166	
Nap	2.2			0.49	2.16	0.16
Acy	0.25	13.3		0.46	0.83	0.03
Ace	0.53	13.5		0.61	_	0.1
Fl	3.84	5.25		2.03	0.33	0.44
Phe	11.6	3.87		7.87	71.5	7.05
Ant	1.15	0.39		1.43	0.5	1.15
Flu	186	7.61	5.3	7.26	26.1	18.9
Pyr	16.2	8.69	3.65	7.08	25.9	17.1
BaA	12.3	2.03	2.42	4.52	8.15	12.4
Chr	8.27	2.18	3.08	8.76	31.1	16.6
BbF	36.7	5.25	3.68	9.71	8.14	16
BkF	53.1	1.95	1.49	6.37	8.32	12.1
BaP	17.8	2.04	2.41	5.73	3.66	11.5
InP	27.6	3.22	2.16	4.6	2.83	9.59
DBA	6.27	27.3	0.9	7.86	1.5	6.52
BghiP	25.2	16.2	2.7	15.7	7.82	14.1

Sampling information from previous studies

As mentioned before, GC-MS is widely used in analyzing PAHs. Among six research stations from previous studies, five of the sampling stations used GC-MS and only one used HPLC as shown in Table 3-6.

Table 3-6: Sampling methods of $PM_{2.5}$-bound PAHs from previous studies in Beijing.

Author	Sampling sites	Instrument	Resolution	Chemical analysis
Hou et al., 2006	Not mentioned	High volume air samples with quartz fibre filters	Daily averaged individual $PM_{2.5}$ bound PAH concentration from September 2006 to August 2007	GC-MS
Wang et al., 2008.	20 sampling sites	Medium volume samplers with glass fibre filters	Hourly sampling times from 8:00 to 12:00 everyday from December 2005 and January 2006	GC-MS
Liu et al., 2008.	6 sampling sites	Medium volume sampler with fiberglass filters	Daily averaged Individual PAH concentrations from October 2003 to October 2004	HPLC
Yu et al., 2008	China University of Petroleum	High volume air samples with fibre filters	Daily averaged individual $PM_{2.5}$ bound PAH concentration from October 2005 to September 2006	GC-MS
Cui et al., 2008	North Forth-Ring Road	Medium-volume samplers with 90mm filters	Daily averaged concentration collected fro March 3 to 5, March 6 to 8, March 10 to 12, March 12 to 14 of 2006.	GC-MS
Duan et al., 2009.	Not mentioned	Medium-volume samplers with #2500QAT-UP 90mm filters	Daily averaged concentration collected from September 2003 to July 2004.	GC-MS

Choice of data source for PAHs in Beijing

Similarly, official data source MEP has the priority to be used in this study. Because data from MEP could not be collected, the data collected from previous studies was used.

3.1.2 Windsor, Ontario, Canada

$PM_{2.5}$ mass concentrations and elemental components concentrations

$PM_{2.5}$ mass concentrations and elemental component concentrations are downloaded from Environment Canada (EC, 2016). In order to keep consistency of the datasets in Beijing and Windsor, $PM_{2.5}$ mass concentrations, elemental component concentrations from 2013 to 2015 in Windsor were downloaded.

EC used two analysis methods to analyze elements, ICP-MS and XRF. ICP-MS includes two preparation methods, acid digestion and water extraction. ICP-MS provides 11 kinds of elements needed (Al, Ba, Cr, Mn, Ni, As, Cd, Pb, Co, V, Se) for both preparation methods, without P and Cl. XRF only provide 9 kinds of elements needed (Al, Ba, Cr, Mn, Ni, Cd, Pb, V, Se), without P, Cl, Co, As.

Choice of data for elemental concentrations in Windsor

In Beijing, 5 out of 11 research stations from previous studies used ICP-MS to do element analysis, 3 out of 11 used ICP-AES. In order to keep consistency of methods in Beijing and Windsor, elemental concentrations analyzed by ICP-MS were used in Windsor. There are two methods used for ICP-MS, acid digestion and water extraction. In order to keep the consistency of data, ICP-MS with acid digestion was used in Windsor.

PAHs concentrations

PAHs concentrations were available in EC website from year 2000 to 2012, except 2004, 2005 in Windsor. PAHs concentrations were not provided after 2012. The numbers of samples available and months of data available from 2000 to 2012 are listed in Table 3-7 below:

Table 3-7: Numbers of data and months available in Windsor.

Year	Numbers of sample available	Months of data available
2000	30	Jan-Dec
2001	24	Jan-Dec
2002	19	
2003	10	Jan, Feb, May-Sep
2004	0	Jan-Apr, Jul, Aug
2005	0	0
2006	14	0
2007	23	Aug-Dec
2008	12	Jan-Dec
2009	8	Jan-Mar, Aug-Dec
2010	17	Jan-Mar
2011	8	Jul-Dec
2012	5	Jan-Mar,
2013	0	Mar-Apr
2014	0	0
2015	0	0

PAHs concentrations in Beijing are estimated by previous studies. The time range for previous studies in Beijing are from 2003 to 2007. Considering the consistency of time period in Beijing and Windsor, the first estimation was conducted by using PAHs concentrations in Windsor for the same time period as in Beijing. The second estimation of PAHs concentrations in Windsor was conducted by using the PAH concentrations from 2010 to 2012. Because when analyzing time series of the PAHs (Figure 3-1), PAH concentrations overall decreased annually except for the peak showed up in 2009. The time period from 2010 to 2012 may better present the PAH concentrations from 2013 to 2015.

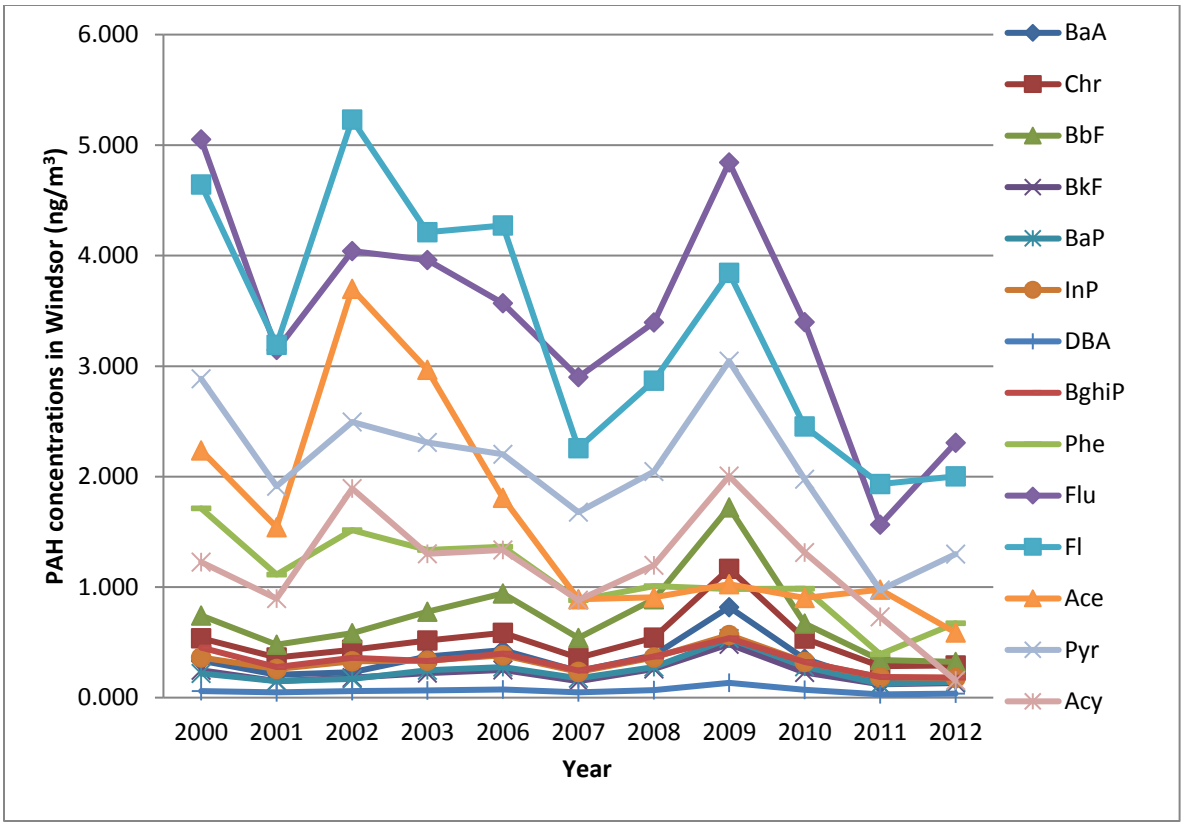


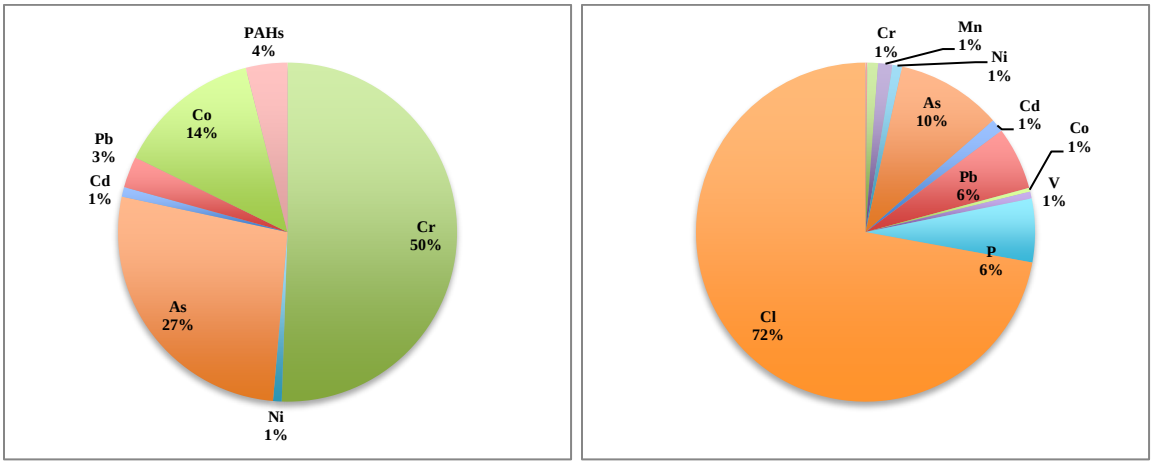
Figure 3-1: Time series of PAH concentrations in Windsor.

3.1.3 Source profiles in Beijing

Health risks calculated were used to conduct a source apportionment to distribute the total health risks into different potential sources. A source apportionment can help us understand the role of each element, to attribute where the risks come from, and the importance of sources. Because in this study components concentrations were estimated, no receptor model could be used to conduct source apportionment. Thus, source profiles from previous studies in Beijing were used.

All the available papers were listed in Table 2-7, justification was conducted according to the tables. According to Table 2-7 (a), all the papers have missing elements compare to the 13 elements needed in this study. Missing elements could only be the elements that account for minor percentages of the total risks (<3%). In order to justify which elements have minor percentages in total risks, a preliminary study was conducted to estimate the

three-year average cancer and non-cancer risks. The percentages of each element in total cancer or non-cancer risks were shown in Figure 3-2:



(a) Cancer (b) non-cancer

Figure 3-2: Contributions of elemental components and PAHs in three-year (a) cancer (b) non-cancer.

After considering the distributions and Table 2-7, only the results from Yu et al., (2013) could be used to do a source apportionment because Cd accounts 1% of total cancer risk and Co accounts for 14% of total cancer risk. Cd and Co account for 1% total non-cancer hazard quotient, respectively. However, it is hard to neglect the contribution of Co because it contributes 14% of total cancer. Two papers with source profile of Co--Zíková et al. (2016) and Zhang et al. (2013) were thus analyzed. In these two papers, over 90% of the Co was from soil dust and the rest were either from biomass burning or coal combustion. Thus, soil dust was regarded as potential source of Co.

Different from Co, Cd accounts for only 1% of the total cancer and non-cancer risks. Potential sources of Cd vary in different papers. Therefore, when conducting source apportionment, Cd was excluded. The source profile of elemental components of $PM_{2.5}$ was interpreted in Table 3-8:

Table 3-8: Source profile of elemental components of $PM_{2.5}$ (%).

	Factor 1	Factor 2	Factor 3	Factor 4	Factor 5	Factor 6	Factor 7
	Secondary sulphur	Vehicle exhaust	Soil dust	Road dust	Biomass burning	Fossil fuel combustion	Metal Processing
Al	6	0	40	30	3	0	21
Ba	3	92	0	5	0	0	0
Cr	0	4	0	66	7	0	23
Mn	5	34	0	0	2	0	59
Ni	0	4	0	28	27	31	10
As	7	19	0	39	0	35	0
Cd	0	0	0	0	0	0	0
Pb	0	27	0	42	25	6	0
Co	0	0	100	0	0	0	0
V	7	8	5	0	6	54	20
P	26	16	21	3	0	34	0
Cl	3	7	0	0	3	79	8
Se	0	0	19	21	4	49	7

As shown in Table 2-7 (b), Wu et al. (2014) have five missing PAHs while Yu et al. (2008) have all the PAHs needed in this study. Cancer risks of PAHs composed of LADI, TEF, and $UR_{[bap]}$, $UR_{[bap]}$ is constant for all PAHs. Compare to LADI, differences of TEFs between PAHs are two to three orders of magnitude higher. Thus, TEF is important when estimating PAHs cancer risks. Among five missing PAHs in Wu et al., 2014 DBA has the largest TEF among 16 PAHs in this study. This means DBA may have important contribution in cancer risks. To neglect the contribution of DBA in PAHs is not wise. Therefore, Yu et al. (2008) was used to estimate sources of PAHs percentages of PAH species for different sources are presented in Table 3-9.

Table 3-9: Source profile of $PM_{2.5}$-bound PAHs (%) (Yu et al., 2008).

	Vehicle exhaust	Biomass emission	Coal combustion
Nap	10.7	3.6	85.7
Acy	26.3	0	73.7
Ace	42.9	0	57.1
Fl	3.8	19.2	76.9
Phe	17.8	37.4	44.8
An	43.8	56.3	0
Flu	78.1	18.2	3.6
Pyr	54.3	26.1	19.6
BaA	75	8.3	16.7
Chr	90.9	1	8.1
BbF	94.8	1.7	3.4
BkF	100	0	0
BaP	77.9	0.4	21.8
InP	99	0	1
DBA	85.1	0	14.9
BghiP	100	0	0

Unlike seven sources of elements described previously, there are only four PAH sources available in Yu et al. (2008), gasoline emission, diesel emission, biomass emission, and coal combustion. According to the introduction of sources in Yu et al., (2008), gasoline emission and diesel emission both belong to vehicle emission. Thus, gasoline emissions and diesel emissions were summed together to present part of the vehicle exhaust. Coal combustion was summed to fossil fuel combustion and biomass burning has the same Factor 5 in sources for elements.

3.2 Major assumptions of health risks

Heath risk assessment studies were according to several assumptions. In this study, assumptions made include:

1. Three-year average concentration is equal to the lifetime average daily exposure concentration.

   Justification: The health risks were calculated based on lifetime average daily

exposure concentration, however the lifetime daily exposure concentration is not available because there is no data source can provide longer $PM_{2.5}$ concentrations. Thus, three-year average concentration was used to present the lifetime average daily exposure concentration.

2. Lifetime cancer risks of different time periods are equal to summation of seasons and hours of days, respectively. Lifetime non-cancer hazard quotients of different time periods were averages by seasonal and hours of day non-cancer hazard quotients.
3. Ambient concentration is equal to the personal exposure concentrations.

   Justification: Uncertainty of personal exposure concentration exits. However, there is no personal exposure concentration available. Thus, ambient concentration was used as the personal exposure concentrations.
4. Exposure frequency with uncertainties such as wearing mask will not influence health risk.

   Justification: Personal habits could influence the exposure. Take wearing mask for example, if a person wears the mask, the exposure concentration will decrease.
5. Exposure frequency, exposure duration, body weight, and average time collected from EPA Exposure Factors Handbook (USEPA, 2011b) are the actual parameters of health risk.

   Justification: No actual parameters could be found. Therefore, parameters from Exposure Factors Handbook were used.
6. Average percentages of element and PAH concentrations in different seasons equal to seasonal average percentages of corresponding elements and PAHs collected from previous studies.

   Justification: Concentrations of seasonal elemental components and PAHs could not be found for this study, so the percentages from previous studies were used.
7. Average percentages of element and PAH concentrations in three-year average, annual average, hours of day all equal to overall average percentages from previous studies.

   Justification: Same as in 6.
8. Source profile collected from previous studies is equal to the source

apportionment results in this study.

Justification: A source profile could be used to distribute the health risks. However, the concentrations in this study cannot be used to conduct a source apportionment. Thus, a source profile from previous studies in Beijing will be used.

9. Multi-elements will not cause accumulated hazard to the same target organs.

   Justification: Multi-elements could cause accumulated hazard quotient in the same organ. Due to time limit, accumulated hazard will not be considered in this study.

3.3 Data processing

Statistical analysis were conducted by Minitab ® Release 17 (Minitab Inc., State College, Pennsylvania, USA) and Microsoft® Office Excel®.

3.3.1 Missing data in $PM_{2.5}$ mass concentration

Raw $PM_{2.5}$ data were kept in order of public time (day of each month * 24 hours a day) and 12 stations in comma-separated values (CSV) files by month. Missing data caused by equipment failures, power outages, and communication failure were shown as "0" or negative values (MEP, 2016).

The objectives here are:

1) to remove missing data;
2) to reformat data for statistical analysis;
3) to delete data with more than 30% missing data.

Statistical analyses were conducted by Minitab ® Release 17. Five steps were used:

1) Collect three-year data into one worksheet.
2) Public time were originally stored in column of "pubtime" with format of " YYYY/mm/dd 00:00:00". "Extract to numeric" function under "data" menu was

used to extract time and date from column "pubtime" to two columns —"Date" and "Time", respectively.

3) "Replace" function was used to replace missing data with blank cells.
4) Use "display descriptive statistics" under "stat" menu to get daily averages by variables "Date". Choose "Mean", "N total", and "N missing" in "statistics" for data statistics, and choose "standard deviation", "Maximum", "Minimum", "Medium" for future analysis.gb
5) Use "sort" function by column " N total" in increasing order. Delete rows have total number less than 252 (24 hours *12 stations *70%).

Missing number accounts for less than 30% of the total numbers was required in all data set when calculating the means.

Repeat datasets in research stations

Research stations such as Tsinghua University, Beijing Normal University, and Chegongzhuang were found more than once from previous studies. To avoid the same sampling site showed up more than once when calculating the average percentages of elements, factors such as sampling time, sampling method and extreme events were removed. For example: When both datasets in the same sampling site have the same elements, the study conducted earlier was removed. Thus, (1) the study conducted by Sun et al. (2006) with 219.96 μg/m^3 $PM_{2.5}$ mass concentration in Beijing Normal University was removed because the data presents the haze-fog period in Beijing. (2) The study conducted by He et al. (2001) in Chegongzhuang and Tsinghua University were removed because the sampling period was from September 1999 to June 2000, which was beyond my time range set for collecting data. (3) Two studies in Beijing Normal University, Sun et al. (2006) with 36.73 μg/m^3 $PM_{2.5}$ mass concentration and Yu et al. (2013), were averaged for each element to get the average percentage. After data processing, the studies eventually used to estimate the average percentages were listed in Table 3-10:

Table 3-10: Previous studies used to estimate average percentages of elemental components of $PM_{2.5}$ (a) Overall and (b) Seasonal ($\mu g/m^3$).

(a) Overall

Author	Sampling site	$PM_{2.5}$	Al	Ba	Cr	Mn	Ni	As	Cd	Pb	Co	V	P	Cl	Se
Duan et al., 2006	Chegongzhuang	96.5						0.060		0.170				1.640	0.090
Yang et al., 2014	North Fifth-Ring Road	137.6			0.032	0.079	0.005	0.028	0.004	0.355					
Zhao et al., 2013	Beijing	123.5	0.970	0.020	0.020	0.070	0.010	0.030		0.140			0.090		
Yu et al., 2013	Beijing Normal University	55.5	0.491	0.088	0.022	0.062	0.028	0.038		0.117		0.015	0.028	2.204	0.038
Sun et al., 2006	Beijing Normal University	36.7	1.000		0.002	0.040	0.020	0.010		0.040	0.007	0.008			
Yu et al., 2012	China University of Geosciences	89.6						0.016	0.003	0.148					
Song et al., 2006	Ming Tombs, the airport, Beijing University, Dong Si EPB, and Yong Le Dian	96.1	0.400		0.020	0.070	0.020	0.030		0.300				1.690	0.010
Cui et al., 2008	North Forth-Ring Road	166.3	0.470		0.003	0.120	0.005	0.029	0.002	0.180	0.001	0.004		4.270	0.005
Tao et al., 2014	Peking University	135.0			0.011		0.004	0.012	0.003	0.143	0.001				0.003
Du et al., 2010	Qinghuayuan Community	110.5	0.606			0.100				0.193					
Zhao et al., 2013	Shangdianzi	71.8	0.570	0.010	0.040	0.040	0.010	0.010		0.070			0.060		
Duan et al., 2006	Tsinghua University	106.9						0.060		0.210				2.090	0.120

(b) Seasonal

Author	Sampling site	Seasons	$PM_{2.5}$	Al	Ba	Cr	Mn	Ni	As	Cd	Pb	Co	V	P	Cl	Se
Yang et al., 2014	North Fifth Ring Road	Spring	91.2			0.025	0.069	0.005	0.029	0.004	0.288					
Duan et al., 2006	Chegongz huang	Spring	76.4						0.060		0.170				1.13	0.060
Tao et al., 2014	Peking University	Spring	135.0			0.000		0.000	0.000	0.000	0.001	0.000				0.000
Cao et al., 2012	Beijing	Summer	131.6				0.060		0.020		0.180					
Sun et al., 2004	Beijing Normal University	Summer	77.3	0.530		0.020	0.030	0.060	0.010	0.004	0.110	0.004	0.019			
Sun et al., 2004	Capital steel company	Summer	82.2	0.490		0.030	0.080	0.060	0.040	0.003	0.200	0.002	0.059			
Sun et al., 2004	Yihai Garden	Summer	75.4	0.680		0.030	0.050	0.070	0.020	0.002	0.100	0.001	0.057			
Yang et al., 2014	North Fifth-Ring Road	Summer	84.5			0.094	0.037	0.003	0.008	0.002	0.158					
Duan et al., 2006	Chegongz huang	Summer	89.0						0.040		0.120				0.320	0.010
Tao et al., 2014	Peking University	Summer	135.0			0.008	0.000	0.004	0.019	0.003	0.118				0.000	0.004

Continued from Table 3-10 (b)

Yang et al., 2014	North Fifth-Ring Road	Autumn	78.5			0.012	0.120	0.007	0.028	0.005	0.513					
Duan et al., 2006	Chegongz huang	Autumn	79.8						0.050		0.160				0.880	0.130
Tao et al., 2014	Peking University	Autumn	135.0			0.017	0.000	0.002	0.008	0.003	0.147	0.001				0.003
Cao et al., 2012	Beijing	Winter	115.6				0.070		0.040		0.280					
Sun et al., 2004	Beijing Normal University	Winter	135.7	1.110		0.020	0.080	0.080	0.050	0.011	0.310	0.004	0.002			
Sun et al., 2004	Capital steel company	Winter	140.8	0.850		0.030	0.100	0.060	0.050	0.008	0.270	0.001	0.023			
Sun et al., 2004	Yihai Garden	Winter	182.2	0.990		0.030	0.100	0.080	0.060	0.011	0.320	0.003	0.013			
Yang et al., 2014	North Fifth-Ring Road	Winter	143.4			0.010	0.091	0.007	0.045	0.005	0.454					
Duan et al., 2006	Chegongz huang	Winter	122.1						0.070		0.210				3.930	0.120
Tao et al., 2014	Peking University	Winter	135.0			0.010	0.000	0.004	0.007	0.002	0.163	0.002	0.000	0.000	0.000	0.003

The same procedures were conducted for $PM_{2.5}$ bound PAH concentrations in Beijing for both overall and seasonal. Studies eventually used to estimate average percentages of $PM_{2.5}$-bound PAHs did not change compare to Table 3-5. Thus, Table 3-5 was used to estimate average percentages of $PM_{2.5}$ bound PAHs. Seasonal $PM_{2.5}$ bound PAHs concentrations were collected and justified, results were shown in Table 3-11.

Table 3-11: Previous studies used to estimate seasonal average percentages of $PM_{2.5}$-bound PAHs (μg/m3 for $PM_{2.5}$ mass, ng/m3 for PAHs).

Author	Yu et al., 2008	Duan et al., 2009.	Yu et al., 2008	Duan et al., 2009.	Yu et al., 2008	Duan et al., 2009.	Yu et al., 2008	Duan et al., 2009.
Sampling site	China University of Petroleum	Beijing	China University of Petroleum	Beijing	China University of Petroleum	Beijing	China University of Petroleum	Beijing
Season	Spring	Spring	Summer	Summer	Autumn	Autumn	Winter	Winter
$PM_{2.5}$	165.81		52.1		69.4		94.7	
Nap	0.46	0.11	0.1	0.08	0.61	0.17	0.08	0.19
Acy	0.33	0.08	0.09	0.01	0.53	0.01	0.89	0.02
Ace	0.46	0.03	0.11	0.01	0.67	0.04	1.18	0.19
Fl	1.05	0.2	0.23	0.04	2.08	0.15	4.77	0.84
Phe	5.87	2	2.96	0.6	9.73	1.26	12.91	14.19
Ant	0.99	0.6	0.39	0.05	1.48	0.15	2.84	2.11
Flu	4.92	4.67	2.75	1.58	7.09	2.67	14.26	39.49
Pyr	4.99	3.82	3.06	1.45	8.24	2.54	12.04	35.84
BaA	3.06	3.35	1.37	1	5.06	2.57	8.59	24.96
Chr	6.21	5.48	3.28	2.25	10.97	4.94	14.57	31.83
BbF	7.42	5.44	6.36	4.28	9.26	5.2	15.79	30.52
BkF	4.77	4.5	2.52	3.27	6.94	4.5	11.25	21.82
BaP	5.14	3.03	2.48	1.37	5.88	3.06	9.4	22.43
InP	3.26	4.24	2.09	4.36	4.12	4.27	8.94	17.49
DBA	5.86	1.55	3.09	0.69	8.17	0.81	14.32	13.43
BghiP	12.28	5.99	7.47	5.07	15.55	5.44	27.48	22.99

3.3.2 Average $PM_{2.5}$ mass concentrations

Hourly average $PM_{2.5}$ mass concentrations (HACs) in Beijing from 2013 to 2015 in 12 sampling sites were used to calculate $PM_{2.5}$ mass concentrations according to different temporal and spatial groups. Results of these daily average $PM_{2.5}$ mass concentrations (DACs) were listed in appendix A. These $PM_{2.5}$ mass concentrations were used in future studies as interpreted below:

(1) HACs from 2013 to 2015 in 12 sampling sites of Beijing were averaged to get DAC from 2013 to 2015.

(2) HACs in 12 sampling sites of Beijing in 2013 were averaged to get DAC in 2013. Similarly, DACs in 2014 and 2015 were calculated by HACs in 2014 and 2015, respectively.

(3) HACs from 2013 to 2015 in 12 sampling sites of Beijing were grouped by seasons: spring (March to May), summer (June to August), fall (September to November), winter (December to February). HACs in different seasons were averaged to get DACs in corresponding groups.

(4) HACs from 2013 to 2015 in 12 sampling sites of Beijing were grouped into 24 groups from 00:00 to 23:00 according to their corresponding time. HACs for these 24 groups were averaged to get DACs for hours of day, respectively.

(5) According to coordinates, twelve sampling sites in Beijing were divided into two groups: urban group and suburban group. Details of grouping were introduced in Table 3-13. HACs from 2013 to 2015 in 12 sampling sites of Beijing were grouped into urban and suburban groups. HACs in these two groups were averaged to get DACs for urban and suburban groups, respectively.

(6) According to coordinates, twelve sampling sites in Beijing were divided into three ring groups. Details of grouping were introduced in Table 3-13. HACs from 2013 to 2015 in 12 sampling sites of Beijing were grouped into three ring groups. HACs in each group were averaged to get DACs for three ring groups, respectively.

(7) According to geographical locations, each of twelve sampling sites in Beijing were analyzed by using percentages of components in nearest station. Details of

grouping were introduced in Table 3-13. HACs from 2013 to 2015 in Beijing were averaged according to sampling sites to get DACs for each sampling site.

3.3.3 Average percentages of elemental components in Beijing

According to components concentrations listed in Table 3-3, the estimated average percentages of $PM_{2.5}$ elemental components were calculated as follow, results of average percentages were listed in Appendix B:

(1) Calculate percentage (P_j) of each component:

$$Pj = \frac{AAj}{AAM} \times 100\% \quad (3\text{-}1)$$

where, AA_j =annual average concentration of component j (μg/m^3) in research stations; AAM= corresponding annual average $PM_{2.5}$ mass concentration (μg/m^3) in research stations.

(2) Calculate average percentage (P_{aj}) in research station of the each component by arithmetic means of all available Pj:

$$Paj = \frac{\sum Pj}{n} \quad (3\text{-}2)$$

where, n= numbers of available concentrations in research stations for each component.

(3) By using percentages, daily average components concentrations were calculated by using Equation 3-3. For example, three-year daily average concentration (DAC_j) (μg/m^3) of each component were calculated:

$$\mathrm{DACj} = \mathrm{Paj} \times DAC \quad (3\text{-}3)$$

3.3.4 Average percentages of $PM_{2.5}$-bound PAHs concentrations in Beijing

The method to estimate concentrations of $PM_{2.5}$-bound PAHs concentrations is the same as the way to estimate the average $PM_{2.5}$ elemental components concentrations. Results were shown in Appendix.

3.4 Calculation

Health risks were quantified separately for cancer and non-cancer effects in Beijing and Windsor, respectively. Different DACs in Beijing were used here according to temporal and spatial groups divided.

3.4.1 Cancer risk

For carcinogenic components (Table 2-4; Table 2-5), potential lifetime cancer risks (R_p) in Beijing and Windsor were calculated as follow:

(1) Calculate the lifetime average daily intake ($LADI_j$) of elements and PAHs according to corresponding daily average concentration, which indicates the amount inhaled in a daily level with the units of (μg/kg*day) (USEPA, 2011b):

$$LADI_j = (DAC_j \times IR \times EF \times ED) / (BW \times AT) \quad (3\text{-}4)$$

where, DAC_j = daily average concentration of each component (μg/m^3); EF = exposure frequency (day/year), 365 days for annual exposure, 91 days for seasonal exposure; ED = exposure duration (years), 70 (years) for lifetime adults exposure, etc; BW = body weight (kg), 70kg; Average time = number of days over which the exposure is averaged (days), here is assumed to be 365 (days) for all.

Values of IR, ED, BW, and AT used in this study, are from EPA Exposure Factors Handbook (USEPA, 2011b).

(2) Calculate potential individual lifetime cancer risks (R_{ic}) from all carcinogenic elements (USEPA, 2011b):

$$Ric = LADIj \times SFj \quad (3\text{-}5)$$

where, SF_j =Inhalation Slope Factor (μg/kg*day)-1 of a specific component (Table 2-4)

(3) Add all R_{ic} together to get the potential overall lifetime cancer risk (R_1) from elements:

$$R_1 = \sum Ric \quad (3\text{-}6)$$

(4) Calculate potential lifetime cancer risk (R_2) from PAHs (OEHHA. 2003):

$$R_2 = \sum_{i=1}^{16} (LADI_i \times TEF) \times UR_{[BaP]} \quad (3\text{-}7)$$

where, i= specific PAH; $C_{[PAHi]}$ =the concentration of the specific PAH; TEF, shown in Table 2-5; $UR_{[BaP]}$ = the inhalation UR of exposure to BaP, $UR_{[BaP]}=1.1*10^{-3}$ $(\mu g/m^3)^{-1}$.

(5) Calculate overall potential lifetime cancer risk for $PM_{2.5}$ (CR):

$$CR = R_1 + R_2 \quad (3\text{-}8)$$

3.4.2 Non-cancer hazard quotient

For non-carcinogenic $PM_{2.5}$ components, DACs were also calculated using Equation (3-4). According to DACs, HQs of each component were calculated by using Equation 3-9 as shown (USEPA, 2011a):

$$HQ_j = DAC_j / RfC_j \quad (3\text{-}9)$$

where, RfC_j=inhalation reference concentration($\mu g/m^3$), this parameter could also be found from Table 2-4.

The Hazard index (HI) was calculated to assess the overall potential for non-cancer effects posed by all the contaminants:

$$HI = \sum HQ \quad (3\text{-}10)$$

3.4.3 Contribution factor (CF)

CF was employed to present the contribution of each element when DACs were not available. According to the equations shown before, cancer risk of each element could be expressed as follow:

$$Ric = CF \times DAC = \frac{Paj \times IR \times EF \times ED \times SFj}{BW \times AT} \times DAC \quad (3\text{-}11)$$

Similarly, non-cancer hazard quotient of each element could be expressed as:

$$HQ = DAC \times \mathrm{CF} = DAC \times Paj \times (1/RfC) \quad (3\text{-}12)$$

First of all, CF could be calculated under different temporal and spatial groups to estimate the influence of each element before conducting the real estimation. Second, CF values could explain the trend of each element in different groups in some extend because differences of DACs are small between groups. Third, CF could help to determine whether the estimation is needed or not according to the trend observed. Details of using CF were introduced in later subsections.

3.5 Data analysis

3.5.1 Cancer risk and non-cancer hazard quotient levels

According to USEPA (2011a), for carcinogens, there are ranges and corresponding classifications for cancer risks:

$$R = \begin{cases} < 1\mathrm{E}-06 & negligible; \\ [1\mathrm{E}-06, 1\mathrm{E}-04] & \text{acceptable;} \\ > 1\mathrm{E}-04 & \text{sufficiently large that some sort of remediation is desirable} \end{cases}$$

Two kinds of risks were analyzed, overall risk and individual risk of each element. Individual risks of different contaminants help to allocate major health risk and to trace

major sources that cause health risks to the human. Overall cancer risks give us an overall risk caused by long-term exposure to $PM_{2.5}$.

For non-carcinogens, there are also thresholds for HQ values as shown below (USEPA, 2011a):

$$HQ\ (or\ HI)\begin{cases} < 1\ there\ is\ no\ appreciable\ or\ adverse\ risk; \\ = 1 \text{ an individual or community is exposed equals the RfC;} \\ > 1\ there\ is\ a\ possibility\ that\ some\ noncancer\ effects\ may\ occur. \end{cases}$$

3.5.2 Target organs for non-cancer hazard quotients

Non-cancer hazard quotients of different elements are targeted in different organs. The primary endpoints (target organs) should be considered in risk assessment. Primary target organs are presented in Table 3-12.

Table 3-12: Primary endpoints of elemental components.

	Carcinogenic		Non-carcinogenic		
Chemical name	UR $1/(\mu g/m^3)$	Source	RfC $(mg/m^{3)}$	Target organ	Source
Al (Aluminum)			5.00E-03	Nervous	USEPA, 2016.
Ba (Barium)			5.00E-04	Fetus	USEPA, 1997.
Cr (Chromium)	1.20E-02	USEPA, 2016b.	1.00E-04	Respiratory	USEPA, 2016b.
Mn (Manganese)			5.00E-05	Nervous	ATSDR, 2012.
Ni (Nickel)	2.40E-04	USEPA, 2016b.	9.00E-05	Respiratory	ATSDR, 2012.
As (Arsenic)	4.30E-03	USEPA, 2016b..	1.50E-05	Reproductive	Cal EPA, 2000.
Cd (Cadmium)	1.80E-03	USEPA, 2016b.	1.00E-05	Renal	ATSDR, 2012.
Pb (Lead)	8.00E-05	Cal EPA, 2000.	1.50E-04	Nervous	USEPA, 2016c.
Co (Cobalt)	9.00E-03	ATSDR, 2012.	1.00E-04	Respiratory	ATSDR, 2012.
V (Vanadium)			1.00E-04	Respiratory	ATSDR, 2012.
P (Phosphorus)			7.00E-05	Respiratory	Cal EPA, 2000.
Cl (Chlorine)			1.50E-04	Respiratory	ATSDR, 2012.
Se (Selenium)			2.00E-02	Alimentary	Cal EPA, 2000.

After estimating non-cancer hazard quotients of each element, elements with the same target organ were summed together. Al, Pb, and Mn influence nervous system, Cr, Ni, Co, V, P, and Cl primarily target respiratory system. These two systems are influenced by multi-elements. Ba is harmful for fetus, As influences reproductive system, Cd targets renal system, and Se is hazardous to alimentary system. These four systems are influenced by single element.

3.5.3 Temporal variability

Three-year average analysis

In order to estimate an overall lifetime health risks, the three-year average was used to present a long-term exposure in Beijing and in Windsor. The health risks were estimated in both cities according to three-year average $PM_{2.5}$ mass concentration from 2013 to 2015. $PM_{2.5}$ component concentrations were calculated in both cities as well. Health risks were analyzed for both cities.

Annual variation

Annual average concentrations of $PM_{2.5}$ mass concentration were calculated for the year 2013, 2014, and 2015 in Beijing and Windsor. $PM_{2.5}$ component concentrations were calculated in both cities as well. Health risks were analyzed for both cities.

Seasonal variation

Health risks were estimated from seasonal perspective estimated According to the average percentages calculated by seasonal concentrations. Seasonal components concentrations were estimated by the average percentages calculated from previous studies. Unlike the each-year variation, data from previous studies are collected throughout the year. For seasonal variations, components concentrations were collected in seasonal order. By using the components concentrations in seasonal order, risks for different season were analyzed. Health risks in four different seasons were added together to present the overall lifetime health risks. Components with obvious seasonal variations and the commonalities and differences were analyzed. The distributions of the health risks were calculated. Potential sources and corresponding health risks were calculated. Non-cancer hazard quotients were distributed according to the target organs.

Hours of day variation

The assumption that components concentrations are the same with the percentages calculated from previous studies were also made to estimate the hours of day variations due to lack of data support. The risks variations throughout the day were analyzed. The

peak hours and lowest hours were found out, and the major contributors in these time slots were compared.

3.5.4 Spatial variability

Several methods were tried by Minitab ® Release 17 and Google maps to divide governmental sampling sites. Corresponding research sites for each group were distributed according to the results. As mentioned before, CF values to an extent expressed the trend of cancer and non-cancer health risks. Spatial analyses were conducted for urban-suburban groups, ring groups, and nearest distance groups because clear differences of CF could be observed within groups. Details of grouping were introduced in following subsections one by one. Results of grouping were listed in Table 3-13:

Table 3-13: Groups of spatial variability for (a) governmental stations and (b) research stations.

(a) Governmental stations

Stations	Government groups by MEP	Urban-suburban groups by Cluster	Ring groups by geographical locations (1)	Groups by ANOVA (2)	Nearest distance groups (3)
WS	Urban	Urban	R<3	A	Chegongzhuang
DS	Urban	Urban	R<3	A	Beijing Normal University
GY	Urban	Urban	R<3	A	Chegongzhuang
TT	Urban	Urban	R<3	A	Chegongzhuang
GC	Urban	Urban	R3-5	A	Chegongzhuang
HD	Urban	Urban	R3-5	A	Peking University
NZG	Urban	Urban	R3-5	A	North Forth-Ring Road
ATZX	Urban	Urban	R3-5	B	North Rorth-Ring Road
SY	Suburban	Suburban	R>5	B	North Fifth-Ring Road
HR	Suburban	Suburban	R>5	C	Shangdianzi
CP	Suburban	Suburban	R>5	C	Qinghuayuan Community
DL	Background	Suburban	R>5	D	Qinghuayuan Community

Notes:

(1) R<3: Stations located within Third-Ring Road; R3-5: Stations located between third-ring to fifth-ring; R>5: Stations located outside fifth-ring.

(b) Research stations

Research stations	Urban-suburban groups	Ring groups	Nearest distance
Chegongzhuang	Urban	R<3	WS, GY, TT, GC
Beijing Normal University	Urban	R<3	DS
China University of Geosciences	Urban	R<3	-
North Forth-Ring Road	Urban	R3-5	ATZX, SY
Tsinghua University	Urban	R3-5	-
Peking University	Urban	R3-5	HD
Qinghuayuan Community	Urban	R3-5	CP, DL
North Fifth-Ring Road	Urban	R3-5	SY
Shangdianzi	Suburban	R>5	HR

Government groups by MEP

Government sampling sites were originally divided into three groups by MEP as introduced in the Table 3-13. Spatial analysis could not be conducted for government groups because there is only one research station--Shangdianzi locates in suburban area while two MEP groups, suburban and background groups locate in the suburban area of Beijing. Thus, average percentages of elements in one research station could not be applied to two groups.

Urban -suburban groups by Cluster

Cluster analysis in Minitab® Release 17 was used to analyze the correlation of $PM_{2.5}$ mass concentrations between 12 governmental sampling sites. The dendrogram (Figure 3-3) showed that eight urban stations divided by MEP have high similarity in $PM_{2.5}$ mass concentration, and the remaining four stations CP, DL, HR, and SY have high similarity in $PM_{2.5}$ mass concentration as well. According to the results of cluster analysis and

geographical locations, 12 government stations were divided into two groups—urban group and suburban group.

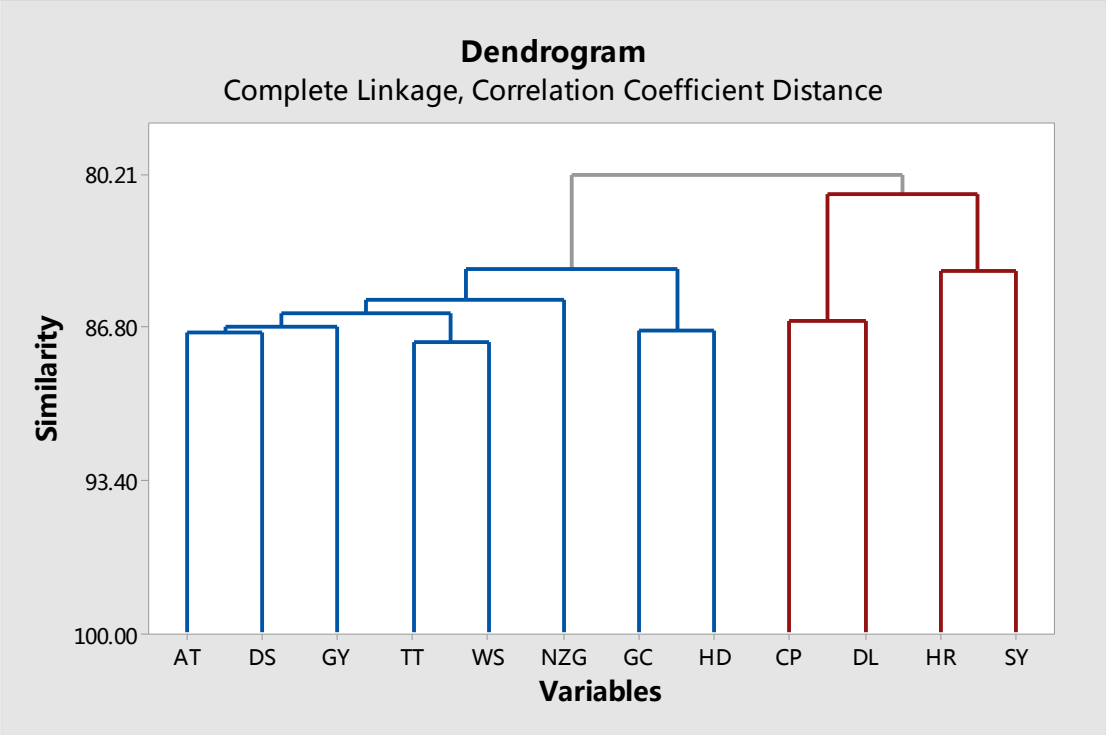


Figure 3-3: Dendrogram of cluster analysis for 12 sampling sites in Beijing.

According to the locations of research stations, nine research stations were distributed to two groups--eight for urban group and one for suburban group. Shangdianzi is the only research station that locates in the suburban area of Beijing. By using the average percentages of each element in all research stations and these two groups, CFs were calculated and shown in Figure 3-4.

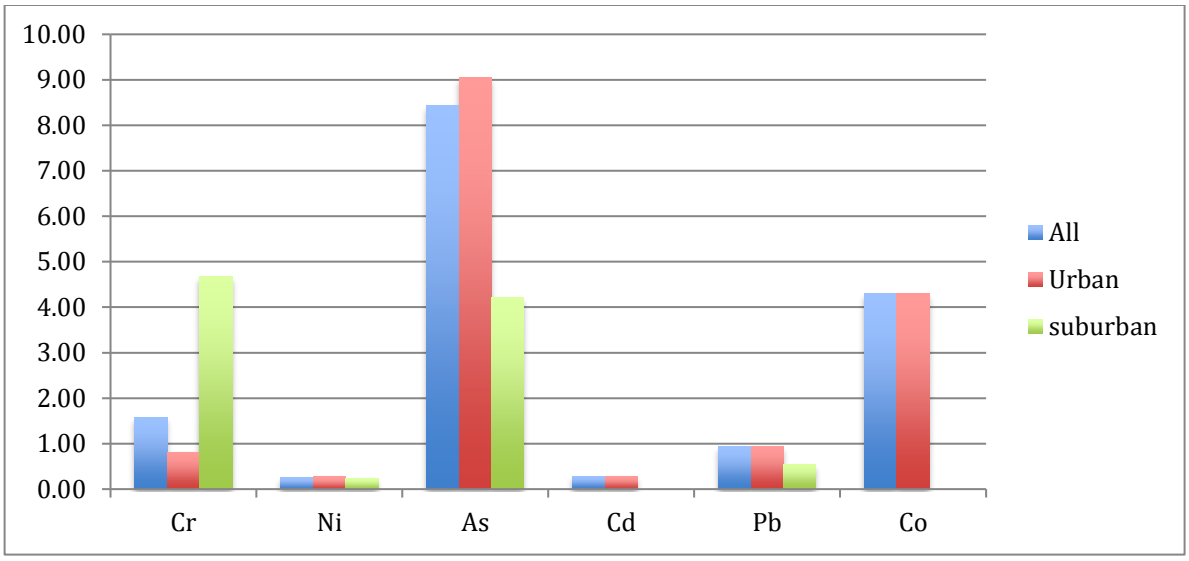


Figure 3-4: Contribution factors for cancer risks and in all stations, urban group, and suburban group.

Notes:

(1) Cr is shown by times E+04, others are shown by times E+05 in cancer risks.

(2) All interpreted the CF in all research stations.

Take cancer risks for example, compare to all research stations group, Ni is the only element that observed similar CF values in three different groups. However, Ni only accounts for around 1% of the overall contribution of the cancer risks (shown in Figure 3-2). Although Cd and Co have the same CF value in all research stations group and urban group, Cd and Co were not detected in the suburban research station. Cd, and Co together account for approximate 15%, 20%, and 0%, respectively in three different groups.

From Figure 3-4 (a), Cr, As, and Pb have remarkable differences in three different groups, Cr, As, and Pb together accounts for a large percentage of overall contribution. Cr contributes the most to overall cancer contribution factor in all research stations group and suburban group with approximate 52.6% and 90.4%, respectively (Table 3-14). As account for 39.3% of the overall cancer contribution factor, which is the largest

percentage in urban group. Considerable differences of the CF values were observed for Cr and Al in different groups.

It was concluded that the differences of CF values exist between all research stations and urban-suburban groups, and between urban group and suburban group. The cancer risks are expected to have different distributions in all research station, urban, and suburban groups.

Table 3-14: Distributions of contribution factors for cancer risks in all, urban, and suburban groups.

	All	Urban	Suburban
Cr	52.63%	35.49%	90.35%
Ni	0.85%	1.16%	0.45%
As	28.09%	39.32%	8.12%
Cd	0.96%	1.25%	NA
Pb	3.14%	4.04%	1.05%
Co	14.33%	18.69%	NA

For non-cancer hazard quotients, Pb is the only element has similar CF values in three different groups as shown in Figure 3-4 (b). However, Pb only contributes less than 3% of the overall non-cancer contribution factors as interpreted in Table 3-15. The same CF values were observed for Cd, Co, and V in all research stations group and in urban group, however, these three elements together account for only less than 3%. The clear differences could be seen for the rest 9 kinds of elements (Al, Ba, Cr, Mn, Ni, As, P, Cl, and Se) in three different groups. The main contributor in all research stations group and urban group is Cl, slightly difference could be observed in two different groups. For suburban group, Cl was not detected in the research station. Cr and P are the major contributors in suburban group with approximate 29.40% for both elements.

Overall, the non-cancer hazard quotients CF values and the distributions of the CF percentage vary in different groups. The way to divide governmental sampling sites into urban and suburban groups is feasible and can interpret the risk distribution in urban and suburban locations. According to classification of groups, average percentages of elemental components were calculated for both groups, respectively.

Ring groups by geographical locations

Beijing city has special layout. The city spreads out with four loops--Second-Ring Road, Third-Ring Road, Forth-Ring Road, and Fifth-Ring Road, circle from the urban core to the outer area of Beijing. Most historical sites, parks, and shopping centers locate within the Third-Ring Road, some residences buildings and universities locate within Third-Ring Road to Fifth-Ring Road, and most residences and industries are distributed outside the Fifth-Ring Road. The different compositions of buildings and population density may cause different traffic, heating, and biomass emissions. Thus, an analysis of cancer risks R<3, R3-5, and R>5 could be conducted.

Research stations were divided into three corresponding groups as well according to their geographical locations as shown in Table 3-13 (b). CF values were also analyzed for three ring groups (Figure 3-5), here takes cancer risks for an example.

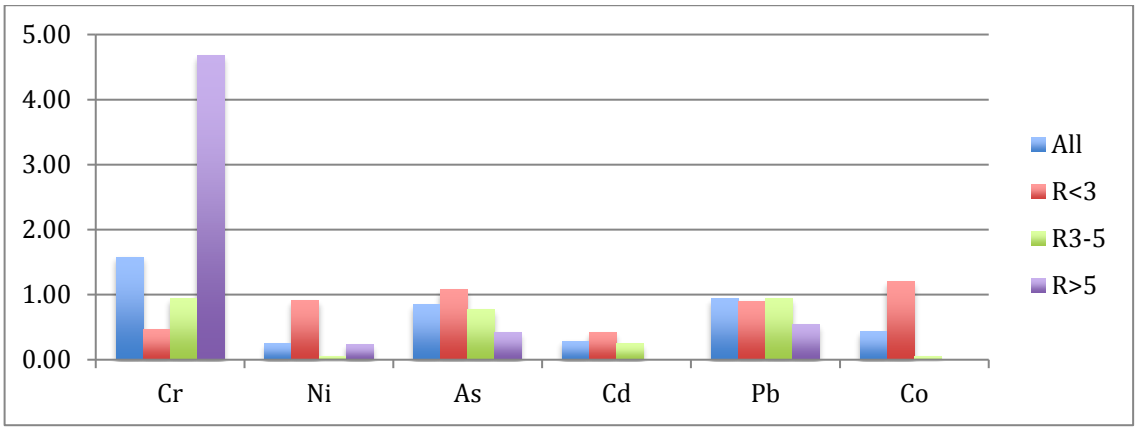


Figure 3-5: Contribution factors for cancer risks in R<3 group, R3-5 group, and R>5 group.

Notes:

(1) Cr, As, and Co are shown by times E+04, others are shown by times E+05 in cancer risks.

For cancer risks, Cr, As, and Co are three major elements that account for a large percentage of the overall CF in four groups. Cr, As, and Co account for 96.1%, 92.5%,

93.2%, and 98.5% in all, R<3, R3-5, and R>5 groups, respectively (Table 3-15). Variances could be seen for all the elements between all research stations and ring groups and within ring groups. Hence, it is reasonable to expect the distributions of the cancer risks vary in different groups.

Table 3-15: Distributions of contribution factors for cancer risks in all, R<3, R3-5, and R>5 groups.

	All	R<3	R3-5	R>5
Cr	52.63%	15.45%	49.73%	90.35%
Ni	0.85%	3.09%	0.28%	0.45%
As	28.09%	36.48%	41.10%	8.12%
Cd	0.96%	1.43%	1.29%	
Pb	3.14%	3.05%	5.02%	1.05%
Co	14.33%	40.56%	2.37%	

For non-cancer hazard quotients, clear differences of CF values could be observed for all elements except Pb between all research stations group and ring groups and within ring groups (Figure 3-5 (b)). P and Ba were only detected in R>5 research station, while Cd, Cl, and Se were not detected in R>5 research station. However, those differences could not have big influences on the non-cancer hazard quotients because Cl account for more than 85% of the overall CF in all groups except for R>5 group which did not detect Cl in the research station as shown in Table 3-15. Cl has the largest CF values in R3-5, followed by all and R<3 group. Thus, non-cancer hazard quotients are expected to have different risk values and different distributions in different groups.

According to the groups divided, average $PM_{2.5}$ mass concentrations and average percentages of elemental components were calculated for each group. Cancer and non-cancer hazard quotients were estimated as well.

ANOVA grouping analysis

Minitab® 17 was used to analyze the concentration variance between and within different government stations by using analysis of variance (ANOVA) function. According to Figure 3-6, governmental stations were divided into three groups as shown in Table 3-13 (a).

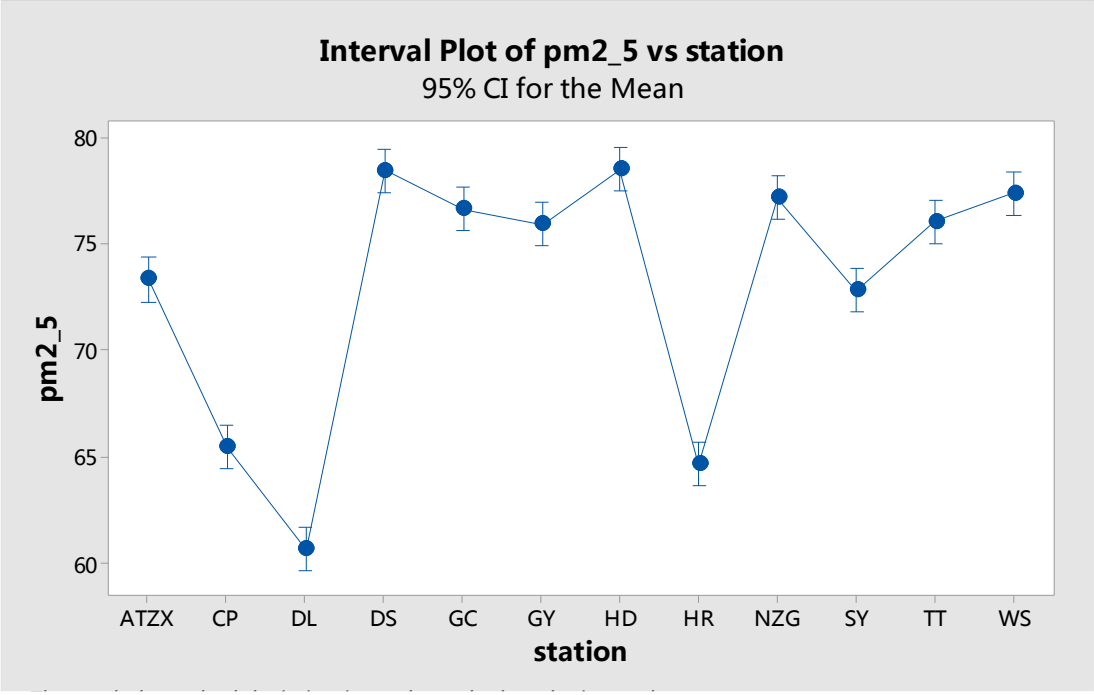


Figure 3-6: ANOVA analysis of 12 sampling sites in Beijing.

Different from any other grouping method, Figure 3-6 showed that ATZX and SY stations have similar variance in $PM_{2.5}$ mass concentrations from 2013 to 2015 (group A2). ATZX is an urban station while SY is a suburban station. SY has the smallest distance to ATZX than any other suburban stations and ATZX has the smallest distance to suburban station SY than any other urban stations. There is a possibility that these two stations have the most similar variance.

Unfortunately, there is no research station could be distributed to group A2. There is only one research station located between ATZX and SY—North Fifth-Ring Road. Compare to distance from North Fifth-Ring Road to SY, distances from North Fifth-Ring Road to any other urban stations are even shorter. For this reason, it is not reasonable to use North Fifth-Ring road to stand for the element percentages of group A2. Thus, ANOVA grouping analysis could not be conducted.

Nearest distance analysis

Research stations in previous studies and governmental stations were found in the map. According to the distance between research stations and government stations stated above, the research stations and governmental stations could be set together in pairs. The results were illustrated in the Table 3-13.

Two research stations –China University of Geosciences and Tsinghua University were not used in this analysis because no government station has the closest distance with these two research stations. Besides, compare to other research stations, China University of Geosciences and Tsinghua University have less representation because these two research stations only provide concentrations for 3 and 4 elements, respectively. In this way, percentages of elements in research stations were used to represent the components in closest government stations. Cancer and non-cancer hazard quotients were estimated using the percentages from close research stations.

3.5.5 Uncertainty analysis

Uncertainty analysis of health risks was conducted. For cancer risks, the uncertainty includes three parts: three-year average $PM_{2.5}$ mass concentration, percentages used to estimate the components concentrations, and the parameters used to estimate LADI. The uncertainty of three-year average $PM_{2.5}$ mass concentration was presented by using the 95% confidence level. The uncertainty of the percentages used to estimate the components concentrations was assumed to be 20% because the elemental components concentrations were estimated from previous study. The uncertainty of parameters used to estimate LADI was assumed to be 30% because the parameters such as body weight, inhalation rate, exposure duration varies with gender, age, personal habits etc. The overall uncertainty was summed by each category and the range of cancer risks was calculated.

For non-cancer hazard quotient, the uncertainty was only divided into two parts: three-year average $PM_{2.5}$ mass concentration and the percentages used to estimate the components concentrations. The overall uncertainty was summed by these two categories and the range of non-cancer hazard quotient was calculated

3.5.6 Comparison between Beijing and Windsor, Canada

Windsor, Canada (42˚17'N, 83˚00'W) locates in the southernmost of Canada (Statistics Canada, 2012). The latitude of Windsor is close with Beijing (39°54′N, 116°25′E). The mass concentration in Windsor from 2013 to 2015 was 9.9 μg/m3, within the NAAQS. Beijing's $PM_{2.5}$ mass concentrations were around 7 times than Windsor's from 2013 to 2015.

Health risks of Windsor assessed before were summarized to compare with Beijing. Because two elements Cl and P were not provided, the risks estimated from the same elements in Beijing were taken out to compare with the risks calculated from Windsor. The distributions of elemental contributors of health risks of three-year average cancer risks, annual averages, and seasonal averages were analyzed.

3.6 Consistency of analytical methods

EPA provides UR, RfC for elements not ions, for example, when they provide information file on the IRIS, Chlorine was used not Chloride. The toxic factor file for chlorine is available from: https://cfpub.epa.gov/ncea/iris2/chemicalLanding.cfm?substance_nmbr=405. From literature review, previous studies used elements to estimate the health risks not by irons, for example: Hu et al., 2012 and Yang et al., 2013 (Table 3-16). There are two kinds of preparation methods found from previous papers: acid digestion and water extraction for PM2.5 samples. Samples prepared with acid digestion when analyzed may yield higher concentrations for elements. Therefore, concentrations from acid digestion were employed to assess the health risks in Beijing and Windsor.

Analytical method for Beijing is consistent as shown in Table 3-16, however it was not consistent in Windsor. In the papers that data was taken from, acid digestion was used as preparation methods for elements except for Yu et al., 2013 and Du et al., 2010, shown in Table 3-16. The preparation methods were not mentioned in these two papers. In Windsor, ICP-MS with acid digestion was used. Percentages for source apportionment in Beijing were employed from elements not ions. The analytical method is consistent for

both Beijing and Windsor now. The results in Windsor have been corrected and recalculated with data from acid digestion. All the results in Windsor have been corrected.

Table 3-16: Analytical method and preparation method for previous studies.

Author	Analytical method for elements	Preparation method
He et al., 2001	XRF	Acid digestion
Sun et al., 2006	ICP-AES	Acid digestion
Duan et al., 2006	XRF	Acid digestion
Song et al., 2006	XRF	Acid digestion
Cui et al., 2008	ICP-MS and ICP-AES	Acid digestion
Du et al., 2010	NAA and ICP-MS.	Acid digestion
Yu et al., 2012	ICP-MS	Not mentioned
Zhao et al., 2013	ICP-AES	Acid digestion
Yu et al., 2013	PIXE	Not mentioned
Yang et al., 2014	ICP-MS	Acid digestion
Tao et al., 2014	ICP-MS	Acid digestion

CHAPTER 4
RESULTS AND DISCUSSION

4.1 Temporal results in Beijing

4.1.1 Three-year average

Cancer risks

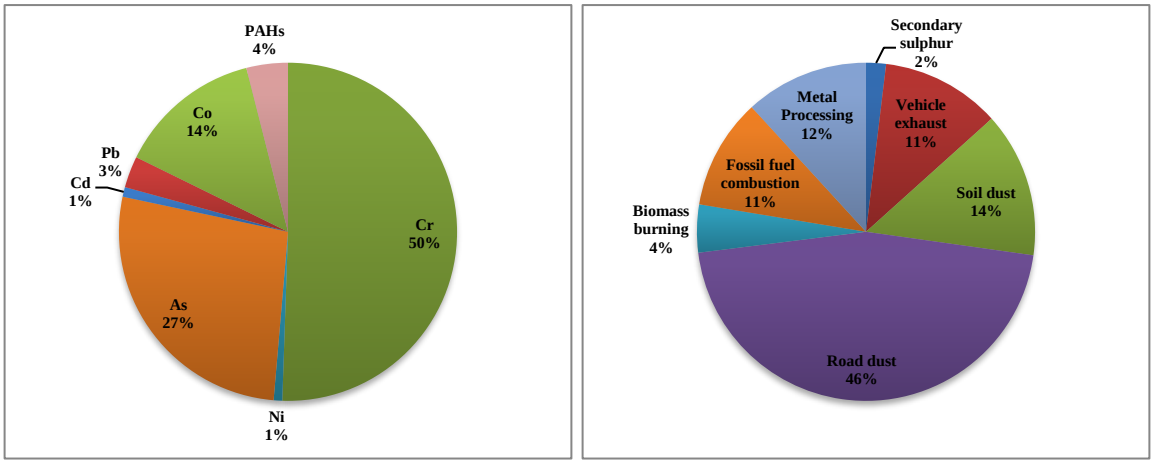
The average $PM_{2.5}$ mass concentration from 2013 to 2015 was 73.7 μg/m^3 in Beijing, over twice the CNAAQS grade two standards (35 μg/m^3). Some general statistics of three-year $PM_{2.5}$ mass concentrations were calculated as shown in Table 4-1. The corresponding lifetime cancer risk calculated from three-year average is 2.30E-02. This cancer risk is more than two orders of magnitude higher than the USEPA standard for cancer risk (1.00E-04), a clear indication that concentration reduction initiatives should take high priority in order to reduce risk in Beijing.

Table 4-1: General statistics of $PM_{2.5}$ mass concentration for three-year average and annual average (unit: μg/m^3).

	Three-year (2013-2015)	2013	2014	2015
Mean	73.7	84.4	69.1	68.5
Standard deviation	78.8	79.6	75.3	80.5
Coefficient of variation	107.8	96.7	108.3	118.4
Trimmed mean	63.8	74	60.8	57.6
Minimum	0	1	1	0
Maximum	762	761	737	762
First quartile	16.0	22	15	15
Median	46	58	44	38
Third quartile	100	118	96	89
SE of Mean	0.15	0.27	0.25	0.26

Compare to PAHs, six carcinogenic elements are the major contributors to lifetime cancer risk, which accounts for 96% of total lifetime cancer risk. Among six carcinogenic elements, the top three elements Cr (50%), As (27%), and Co (14%) together contributes

over 90% of lifetime cancer risk as shown in Figure 4-1 (a). PAHs contribute less than 5% of total lifetime cancer risk.



(a) Elements and PAHs (b) Sources

Figure 4-1: Distributions of total lifetime cancer risk by (a) components (b) sources.

Cr is the major contributor with approximate half of the total lifetime cancer risk. The cancer risk of Cr is higher than EPA upper limit (1.00E-04). According to the source apportionment conducted in this study, Cr is mainly from road dust and metal processing. These two major sources contribute 7.68E-03 and 2.68E-03 cancer risks, respectively. Road dust and metal processing accounts for approximate 90% of the Cr cancer risk.

Lifetime cancer risk of As is 6.21E-03, it accounts for 27% of total lifetime cancer risk and has the second-largest contribution to lifetime cancer risk. Major sources of As are road dust, fossil fuel combustion, and vehicle exhaust which contribute corresponding cancer risks of 2.42E-03, 2.17E-03, and 1.18E-03. Secondary sulphur is the smallest contributor to lifetime cancer risk, it only contributes 7% of the As total cancer risk. However, cancer risk of As from secondary sulphur still exceeds EPA upper level threshold (1.00E-04). Co follows As, and is the third-largest contributor to life time cancer risk. However, percentage of Co is 13%, approximate half of the percentage of As. Co has only one potential source--soil dust, which contributes 3.17E-03 cancer risk.

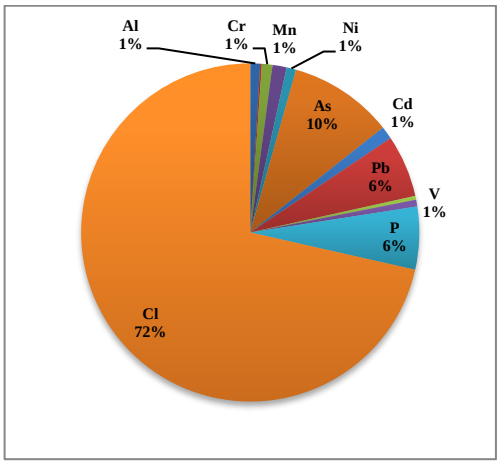
Cancer risk of sixteen EPA-priority PAHs is 9.37E-04. Among which, DBA contributes 47% of total cancer risk, and becomes the top contributor among all the PAHs. PAH emissions are mainly from vehicle emissions (7.86E-04), coal combustion (1.44E-04), and biomass burning (5.75E-06). There are potential cancer risks caused by PAHs from vehicle emissions and coal combustion, because cancer risks from these two sources are higher than EPA upper limit (1.00E-04).

Cancer risks from each of the carcinogenic elements are higher than EPA upper limit (1.00E-04), let alone the total lifetime cancer risk. Ni, which is the smallest contributor of total lifetime cancer risk, was 0.89E-04 higher than EPA upper limit (1.00E-04).

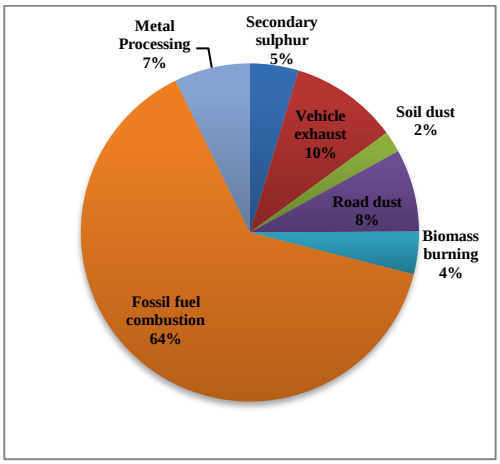
As shown in Figure 4-1 (b), road dust is the primary source of total lifetime cancer risk. It contributes 1.04E-02 cancer risk. Soils dust is the second-largest source of total lifetime cancer risk followed by metal processing, vehicle exhaust, and secondary sulphur with cancer risks ranging from 4.35E-04 to 3.17E-03. Secondary sulphur is the smallest contributor of lifetime cancer risk among all the sources. However, when comparing the cancer risk of secondary sulphur with EPA upper limit (1.00E-04), which should have been compared with total lifetime cancer risks from all sources, the risk of secondary sulphur is 3.35E-04 higher. This indicates that there are potential cancer risks posed by each of seven sources.

Non-cancer hazard quotients

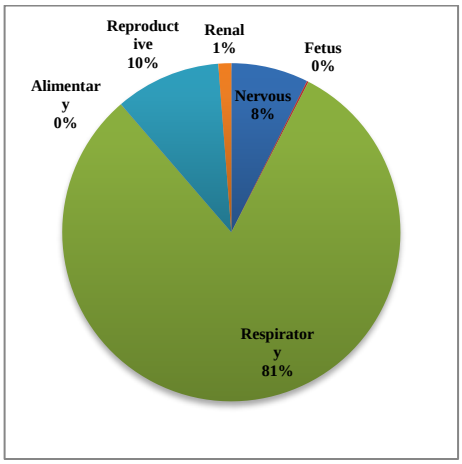
The overall lifetime non-cancer hazard quotient in Beijing is 13.7, higher than EPA upper level of non-cancer hazard quotient (1.0). Among 13 non-carcinogenic elements (Table 2-4), top four contributors Cl, P, Pb, and As accounts for approximate 94% of the total lifetime non-cancer hazard quotient. Only Cl and As have non-cancer hazard quotients higher than EPA upper level (1.0) as illustrated in Figure 4-2 (a). Thus, Cl and As with non-cancer hazard quotients higher than 1 should be considered under given air quality.



(a) Elements



(b) Sources



(c) Target organs

Figure 4-2: Distributions of lifetime non-cancer hazard quotient by (a) components (b) sources and (c) target organs.

It can be concluded from Figure 4-2 (a) that Cl is the primary contributor of non-cancer hazard quotient. Non-cancer hazard quotient from Cl is 9.8, one order of magnitude higher than upper limit of non-cancer hazard quotient (1.0). Among all the sources of Cl, fossil fuel combustion contributes 7.8 non-cancer hazard quotient, accounting for 79% of

total non-cancer hazard quotient of Cl. When talking about non-cancer hazard of Cl, fossil fuel combustion is the primary source to which it is attributed.

As is the second-largest contributor of non-cancer hazard quotient, it accounts for approximate 10% of total non-cancer hazard quotient. Non-cancer hazard quotient from As is only 1/7 of the primary contributor Cl. Non-cancer hazard quotient of As mainly comes from road dust and fossil fuel combustion, however hazard quotient from all As sources are lower than 1.0.

Among all non-carcinogenic elements, Pb is the third-largest contributor of non-cancer hazard quotient. Road dust, vehicle exhaust, biomass burning, and fossil fuel combustion are the major sources of Pb, although the corresponding non-cancer hazard quotients are lower than 1.0. Pb was used as additives in gasoline and it was banned in 1997. Given this, the concentration of Pb should have a decrease trend after 1997. Nowadays, Pb emission is mainly from tire wear not fossil fuel combustion (Smichowski et al., 2008) and this may explained why road dust contributes more Pb than fossil fuel combustion.

Lifetime non-cancer hazard quotients from fossil fuel combustion (8.6), vehicle exhaust (1.4), and road dust (1.1) are all higher than EPA upper limit of non-cancer hazard quotient (1.0). As depicted in Figure 4-2 (b), fossil fuel combustion is the predominant source for non-cancer hazard quotient. Non-cancer hazard quotient estimated for fossil fuel combustion accounts for approximate 64% of total hazard quotient. Cl contributes 90% of non-cancer hazard quotient of fossil fuel combustion. Cl is the first element to attribute to when talking about non-cancer hazard quotient from fossil fuel combustion.

Vehicle exhaust is the second-biggest contributor of lifetime total non-cancer hazard quotient. The non-cancer hazard quotient from vehicle exhaust is 1.4, higher than EPA upper limit (1.0). Road dust followed after vehicle exhaust with 1.1 non-cancer hazard quotients, respectively. Similar to fossil fuel combustion, Cl is the major contributor for vehicle exhaust and metal processing, contributed more than half of corresponding total non-cancer hazard quotients. For road dust, As is the major source with 0.5 non-cancer hazard quotient.

Lifetime total non-cancer hazard quotient was divided into different groups according to their corresponding primary target organ, results are shown in Figure 4-2(c). Respiratory system suffers the most from non-cancer hazard quotient. Lifetime non-cancer hazard quotient of respiratory system is 11.0, higher than EPA upper limit (1.0).

Reproductive system is only influenced by As. Reproductive system impairment is the second-largest among 6 influenced systems with 1.4 hazard quotient. As is mainly from road dust, fossil fuel combustion, and vehicle exhaust. That means, road dust, fossil fuel combustion, and vehicle exhaust are the main sources that cause reproductive system impairment.

Nervous system suffered slightly less than reproductive system, with a hazard quotient of 1.1. Non-cancer hazard quotient of nervous system impairment is contributed by Al, Mn, and Pb. Pb is the main contributor of nervous system impairment. It accounts for 73% of nervous system impairment. Approximate 94% of the Pb comes from road dust, vehicle exhaust, and biomass burning. Thus, it can be concluded that road dust, vehicle exhaust, and biomass burning are the major sources with potential to cause detrimental effects on the nervous system impairment. Besides respiratory system, reproductive system, and nervous system, the hazard quotients from other three systems are all lower than EPA upper threshold of non-cancer risk (1.0).

4.1.2 Annual average

Cancer risks

$PM_{2.5}$ mass concentrations are 84.4 $\mu g/m^3$, 69.1 $\mu g/m^3$, and 68.5 $\mu g/m^3$ for 2013, 2014, and 2015 with corresponding lifetime cancer risks 2.63E-02, 2.16E-02, and 2.14E-02. Cancer risks for 2013, 2014, and 2015 are all two orders of magnitude higher than EPA upper level of cancer risk (1.00E-04). Lifetime cancer risks were decreasing from 2013 to 2015 because $PM_{2.5}$ mass concentrations are decreasing. Percentages and contributions for different components from different sources in 2013, 2014, and 2015 are similar to

the results from three-year lifetime cancer risks. Some items of general statistics of annual $PM_{2.5}$ were calculated as shown in Appendix E.

The top contributor for cancer risks is Cr, which contributed 1.33E-02, 1.11E-02, and 1.09E-02 in 2013, 2014, and 2015, respectively. There is 1.20E-02 (66%) cancer risk of Cr from road dust and 3.09E-03 (23%) from metal processing in 2013. The sum of these two sources contributes around 89% of overall lifetime cancer risk of Cr.

PAHs contribute approximate 4% for overall lifetime cancer risks in 2013, 2014, and 2015. The cancer risks from PAHs are 1.05E-03, 8.55E-04, and 8.52E-04 in these three years. Vehicle emission is the main source of PAHs, contributing 7.16E-04 cancer risk in 2013. The smallest contributor is Ni with 2.24E-04, 1.81E-04, and 1.76E-04 cancer risks from for 2013, 2014, and 2015, respectively. The smallest elemental contribution among three years is 1.76E-04 from Ni in 2013. The remaining cancer risks in 2013 are from As, Co, Pb, and Cd with 7.36E-03, 3.76E-03, 8.23E-04, and 2.51E-04 cancer risks, respectively. Compare to 2013, cancer risks for the same elements are slightly lower in 2014 and become the lowest in 2015.

Road dust is the main source of cancer risk in 2013 as shown in Figure 4-3. Cancer risk from road dust is 3.3 times of the second-largest source soil dust and 24 times of the smallest source secondary sulphur. Cr contributes approximate 73% of road dust cancer risk. When conducting emission control, Cr from road dust should be considered in the first place.

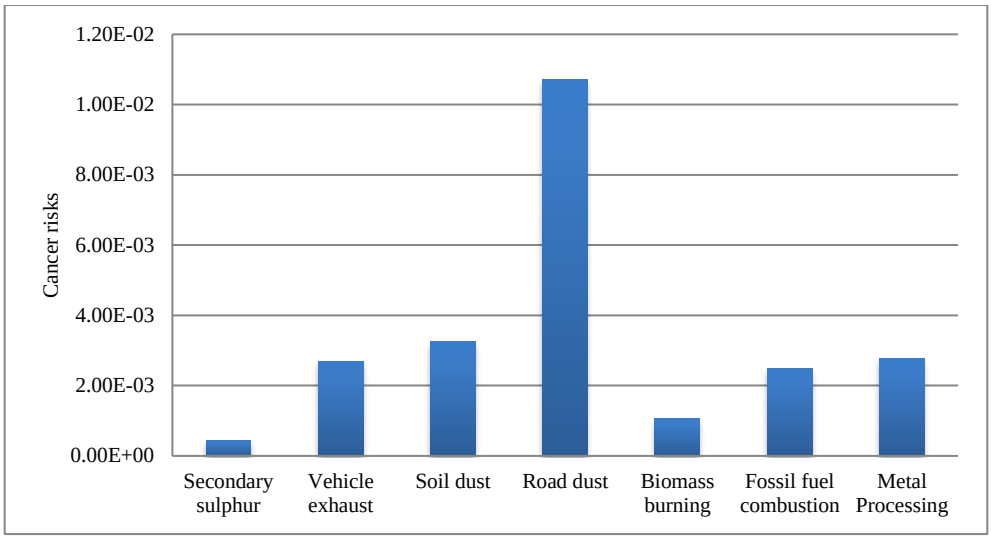


Figure 4-3: The contributions of cancer risks by source in 2013.

Cancer risks from all sources exceed EPA upper limit of cancer risk (1.00E-04). Even the smallest source, secondary sulphur, is over 5 times higher than EPA upper limit (1.00E-04). Besides road dust, the major contributor of different sources are Co for soil dust, Cr for metal processing and biomass burning, and As for vehicle exhaust, fossil fuel combustion, and secondary sulphur. The major contributors should be considered when emission control was conducted in different sources.

Non-cancer hazard quotients

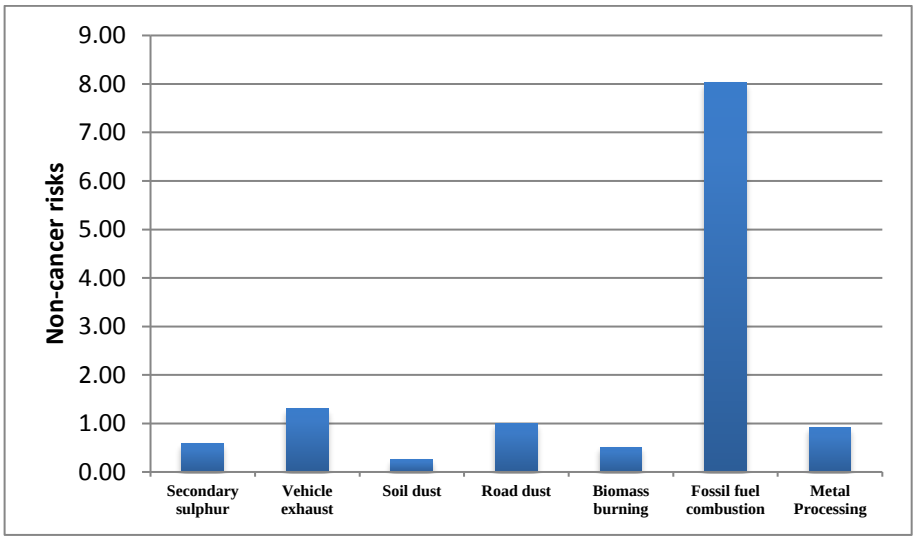
Lifetime total non-cancer hazard quotients are 15.7, 12.9, and 12.8 in 2013, 2014, and 2015, respectively. Compare to EPA non-cancer upper limit (1.0), these hazard quotients are larger. The non-cancer hazard quotients have the same decreasing trend with $PM_{2.5}$ mass concentration. Non-cancer hazard quotient decreased approximate 27% from 2013 to 2015. Among 13 non-carcinogenic elements, non-cancer hazard quotients caused by Cl and As in 2013, 2015, and 2015 are higher than EPA upper limit (1.0). Besides Cl and As, non-cancer hazard quotients caused by remaining 11 non-carcinogenic elements are all less than one, showing no adverse risk.

The top contributor for non-cancer hazard quotients is Cl, non-cancer hazard quotients brought by Cl were 11.2, 9.2, and 9.1 in 2013, 2014, and 2015, respectively. Primary

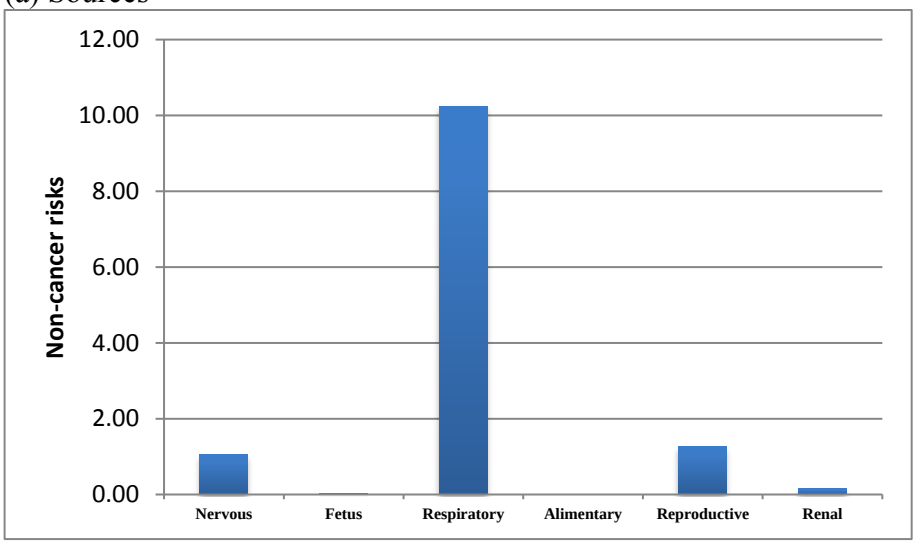
source of Cl is fossil fuel combustion with 8.9, 7.3 and 7.2 non-cancer hazard quotients in 2013, 2014, and 2015 respectively. The remaining sources of Cl with hazard quotient higher than one are vehicle exhaust and road dust. The corresponding non-cancer hazard quotients are 1.6 and 1.2 in 2013.

As is the second-largest contributor of non-cancer hazard quotient, although it only accounts for approximate 14% of total non-cancer hazard quotient. Different from Cl, major source of As is road dust, which offered 0.6 (39%) non-cancer hazard quotient in 2013. Worth to note that fossil fuel combustion is the second-largest contributor of As with 0.6 (35%) non-cancer hazard quotient. Among 13 non-carcinogenic elements, only Cl and As have non-cancer hazard quotients higher than one in 2013. This indicates that the exposure concentrations for these two elements are all higher than corresponding EPA RfC (Table 2-4).

Contributions of different sources and the influences to different target organs in 2013 are shown in Figure 4-4. Similar distributions and influences of non-cancer hazard quotients could be found for 2014, 2015, and three year average as well. Compare to 2013, non-cancer hazard quotients for corresponding sources in 2014 and 2015 are lower. For example, non-cancer hazard quotients of primary source fossil fuel combustion are 9.9, , 8.1, and 8.0 in 2013, 2014, and 2015, respectively. Similarly, non-cancer hazard quotients for different target organs are decreasing from 2013 to 2015.



(a) Sources



(b) Target organs

Figure 4-4: Contributions of non-cancer hazard quotient by (a) sources (b) target organs in 2013.

Due to the large non-cancer hazard quotient brought by Cl, the most vulnerable system is respiratory system in 2013 with 12.6 non-cancer hazard quotients. The non-cancer hazard quotient for respiratory system is mainly from Cl, P contributes only 1/11 of Cl as shown in Figure 4-5. The smallest contributor is Co, which was counted as 0% in the figure.

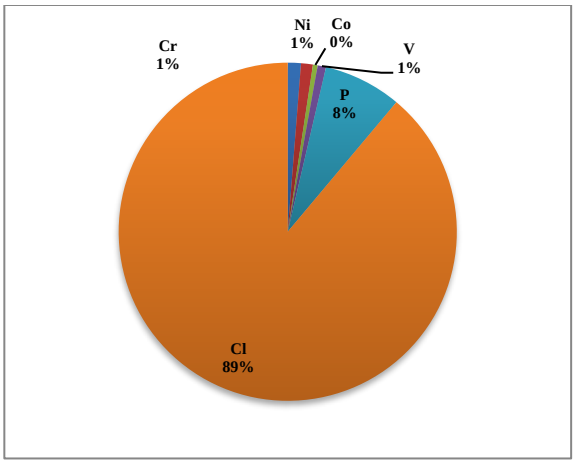


Figure 4-5: Composition of non-cancer hazard quotient for respiratory system impairment in 2013.

Of the total non-cancer hazard quotient, 7.4% is nervous system impairment. Approximate 82% of non-cancer hazard quotients of nervous system impairment come from Pb. According to the major sources of Pb, it can be concluded that the major sources that jeopardies nervous system are road dust, vehicle emission, and biomass burning. Al contributes the least, only 0.2 of neurological non-cancer hazard quotient in 2013. According to these analyses, reduction initiatives should target road dust, vehicle emission, and biomass burning in order to lower the risk of adverse neurological effects.

Compare to the respiratory system introduced above, renal system impairment is negligible with 0.2 non-cancer hazard quotient. Cd is the only source for renal system impairment. The non-cancer hazard quotients for the remaining two systems—fetus and alimentary system impairments are lower than one, thus, the non-cancer hazard quotients are acceptable for this two systems.

4.1.3 Seasonal average

Cancer risks

$PM_{2.5}$ mass concentrations in four seasons are in an order of winter (99.3 μg/m^3) > autumn (71.3 μg/m^3) > spring (70.5 μg/m^3) > summer (61.4 μg/m^3) in Beijing. $PM_{2.5}$ mass concentrations in each season are all above grade-II standard of CNAAQS (35 μg/m^3).

Cancer risks are in the order of summer (7.47E-03) > winter (6.27E-03) > spring (5.37E-03) > autumn (4.78E-03). Cancer risks in each season are all one magnitude higher than EPA upper limit of cancer risk (1.00E-04).

Clear variance could be observed between different seasons. Each season should have contributed ¼ (25%) of total cancer risk. However, winter and summer have higher contribution of 34% and 28% respectively, while contributions were lower in spring (20%) and autumn (18%). Besides, the highest cancer risk in summer and the lowest cancer risk in autumn varied 56% of cancer risk.

Among four seasons, winter has the highest $PM_{2.5}$ mass concentration while summer has the lowest $PM_{2.5}$ mass concentration. However, when estimating cancer risks in four seasons, summer has highest cancer risk. Cancer risks were calculated by using SF to times LADI. Cr has the largest SF among six carcinogenic elements, which means it may have the largest influence on cancer risk. Among all the factors that influence LADI, $PM_{2.5}$ mass concentration and average percentage of element vary in different seasons. $PM_{2.5}$ mass concentration in winter is 62% higher than summer while the average percentage of Cr in winter is only 26% of average percentage in summer. The differences of $PM_{2.5}$ mass concentration and average percentage cause LADI of Cr in summer 7.83E-02 higher than in winter and cancer risk of Cr in summer is 3.28E-03 higher than in winter.

Beside Cr, the cancer risks of Ni range from 5.00E-04 to 4.31E-05, with 12% higher in summer than in winter. The remaining four carcinogenic elements As, Cd, Pb, and Co are all higher in winter than in summer. However the differences are too small to explain the difference made by Cr between summer and winter. The distinct differences of $PM_{2.5}$ mass concentration between winter and summer only helped winter have the second-largest cancer risk among four seasons.

Autumn has the second-highest $PM_{2.5}$ mass concentration, however the lowest corresponding cancer risk. Spring followed after autumn in $PM_{2.5}$ mass concentration, however has higher cancer risk than autumn. The differences of cancer risks between autumn and spring mainly come from Cr and As, two major contributors of cancer risk.

Cancer risk of Cr is 2.10E-03 in autumn and 2.67E-03 in spring, with 5.70E-04 higher in spring. Cancer risk of As is 1.86E-03 in autumn and 2.21E-03 in spring, with 3.90E-04 higher in spring. Besides Cr and As, cancer risk of Co is also lower in autumn than spring with only 6.80E-06 differences. The remaining three carcinogenic elements Pb, Cd, and Ni are higher in autumn than spring with corresponding 1.00E-04, 1.50E-05, and 2.40E-06 differences, which are not big enough to make autumn have higher cancer risk than spring.

The orders of seasonal cancer risks for elemental components and PAHs are different as shown in Figure 4-6. The biggest variance could be seen for Ni with 91% coefficient of variance (CV). Ni peaks in summer and have lowest concentration in spring, but it only has limited influence on the seasonal cancer risk. The seasonal variance of As is the smallest among all components with 30% CV. Total cancer risks in four seasons are relatively constant with 20% CV.

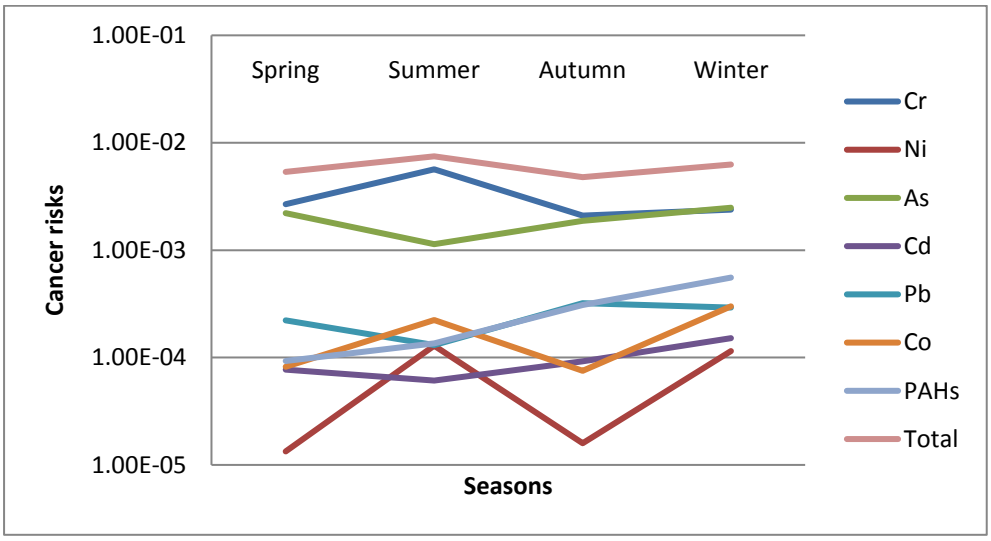
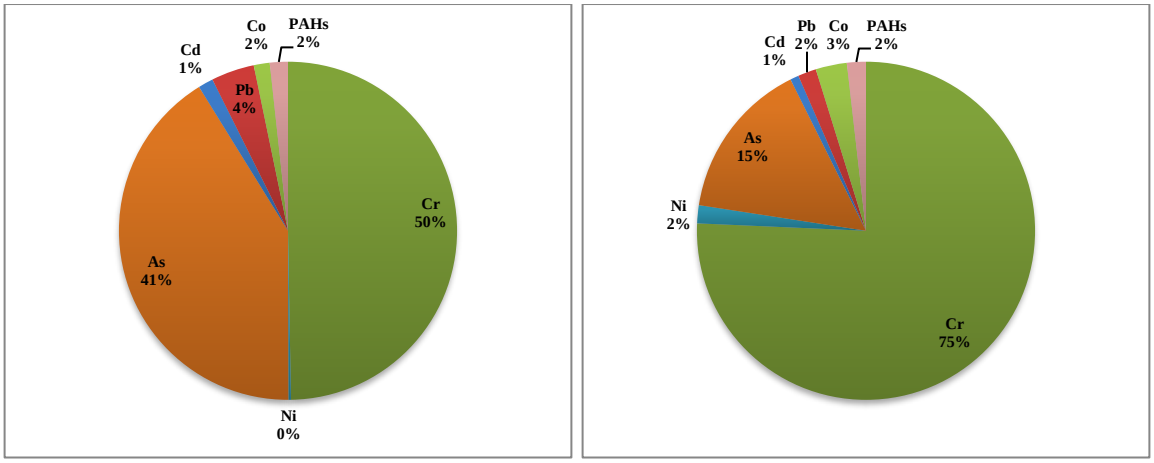


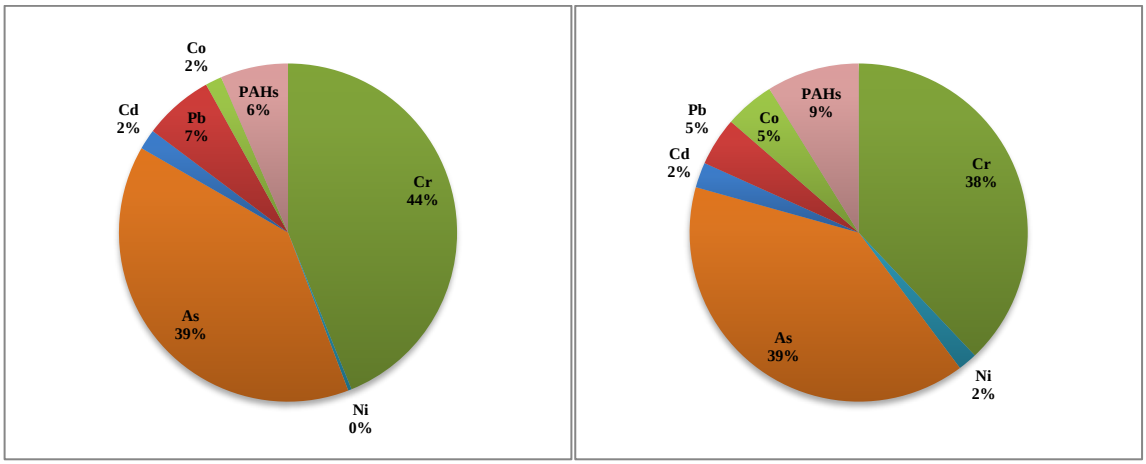
Figure 4-6: Seasonal variance of total cancer risks, elemental cancer risks, and PAHs cancer risks.

Cr contributed 5.66E-03 cancer risk in summer, followed by spring, winter, and autumn with 2.67E-03, 2.38E-03, and 2.10E-03, respectively. Cr contributes up to 75% of total

cancer risk in summer, followed with 50%, 44%, and 38% in spring, autumn, and winter, respectively (Figure 4-7). Compare to summer, the percentage of Cr decreased around 50% in winter. Winter has higher Cr cancer risk than autumn, however the percentage of Cr is higher in autumn than in winter. The reason of this is because $PM_{2.5}$ mass concentration in winter is approximate 40% higher than in autumn. Besides, road dust is the primary source of Cr. In order to control Cr emission, road dust should be the first one to attribute to.



(a) Spring (b) Summer



(c) Autumn (d) Winter

Figure 4-7: Distributions of the cancer risks in four seasons (a) Spring, (b) Summer, (c) Autumn, and (d) Winter by components.

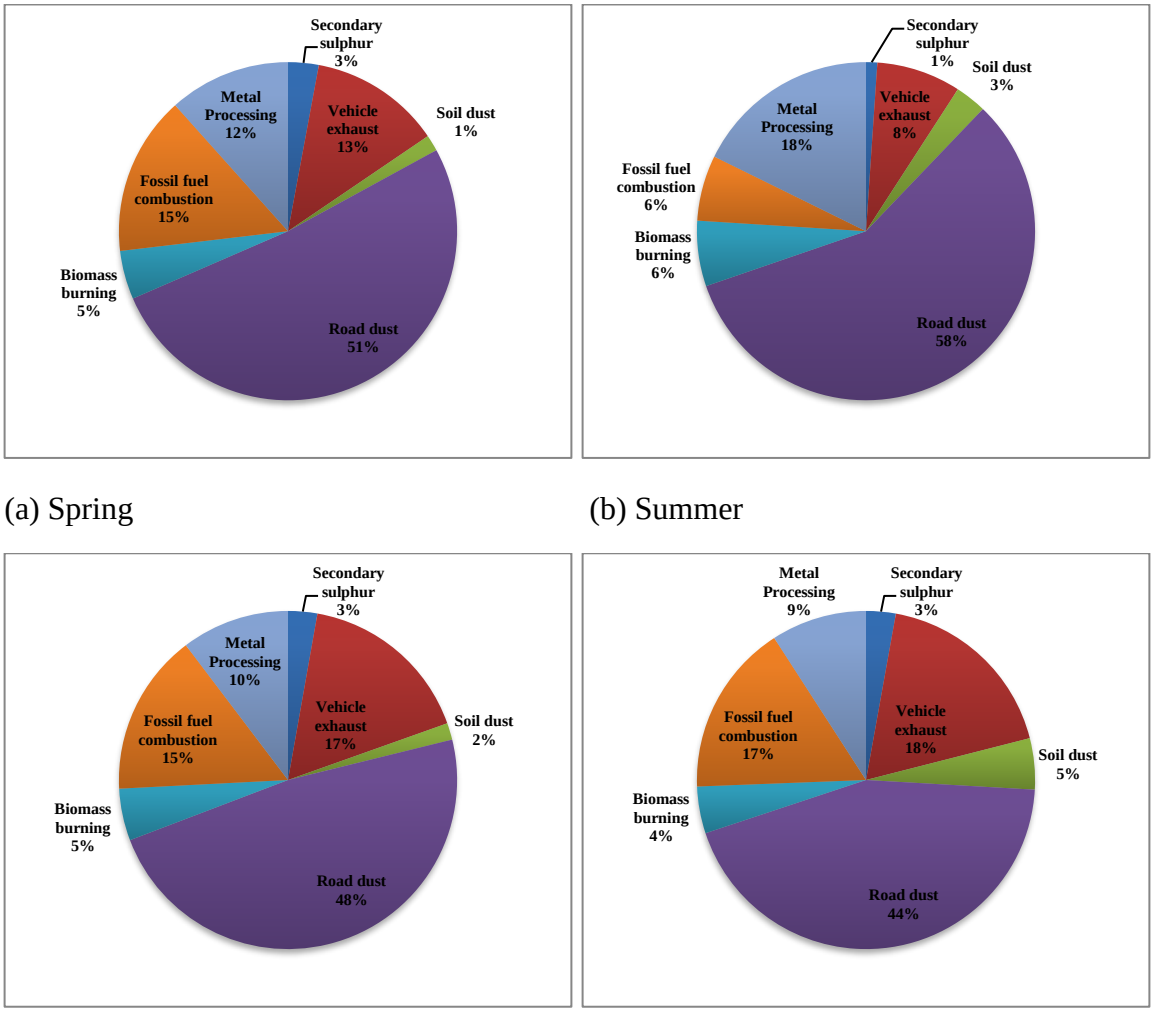
Among six carcinogenic elements, As is the second-largest contributor with percentages ranging from 41% in spring to 15% in summer. The order of As percentages is spring > autumn = winter > summer. The cancer risk of As in winter is 2.48E-03, which is the largest among all the seasons. Followed by spring, autumn, and summer with 2.21E-03, 1.87E-03, and 1.14E-03 cancer risks. Cancer risk of As in winter is higher than in spring while the percentage of As is bigger in spring than in winter, because the $PM_{2.5}$ mass concentration in winter is higher than in spring.

The percentages of Pb vary from 7% in autumn to 2% in summer. Cancer risk of Pb is higher in autumn with 3.21E-04, while lower in winter, spring, and summer with 2.91E-04, 2.21E-04, and 1.31E-04, respectively. Different from Cr and As, cancer risks of Pb have the same order in four seasons with Pb percentages. Major sources of Pb are road dust, vehicle exhaust, biomass burning, and fossil fuel combustion.

Compare to Cr, As, and Pb, which contribute large percentages of cancer risk, Ni, Cd, and Co contribute relatively small fractions in four seasons ranging from 0% to 5%. The highest percentage is 5% of Co in winter.

Percentages of PAHs are 2%, 2%, 6%, and 9% in spring, summer, autumn, and winter, respectively, increased from 2% in spring to 9% in winter. The cancer risks of PAHs increased from 9.28E-05 in spring to 5.55E-04 in winter as well. The PAHs cancer risk in summer accounts for 2% of the total cancer risk, the same percentage as in spring. However, the cancer risk in summer is 4.32E-04 higher than in spring. Vehicle exhaust is the primary source of PAHs, accounts for approximate 84% of the total PAHs cancer risk. BaP is the primary PAH of the cancer risk in four seasons, contributing from 41% to 54% of total PAHs cancer risk.

Road dust is the primary source of cancer risks in four seasons as shown in Figure 4-8. Percentages of road dust cancer risks are in the order of summer (58%) > spring (51%) > autumn (48%) > winter (44%). The cancer risks from road dust in four seasons ranged from 4.27E-03 in summer to 2.25E-03 in autumn. Cr is the predominant contributor of cancer risk from road dust. The percentages of Cr in road dust cancer risk were 58% in winter, 61% in autumn, 65% in spring, and 87% in summer.



(a) Spring (b) Summer

(c) Autumn (d) Winter

Figure 4-8: Distributions of seasonal cancer risks in (a) spring, (b) summer, (c) autumn, and (d) winter by sources.

Besides road dust, the orders of remaining six sources are different in four seasons. Fossil fuel combustion is the second impactful source in spring, it fell into the forth impactful source in summer but back to the third impactful source in autumn and winter. Winter has the highest fossil fuel combustion cancer risk among four seasons.

Vehicle exhaust is also significant in total cancer risks. It was the second-largest source in spring and summer while the third impactful source in autumn and winter. Cancer risks of vehicle exhaust are in order of winter (3.18E-03) > autumn (7.86E-04) > spring (6.65E-

04) > summer (5.98E-04). Either soil dust or secondary sulphur is the smallest contributor in four seasons.

Non-cancer hazard quotients

Lifetime overall non-cancer hazard quotient estimated by the average of four seasons is 12.6, higher than EPA upper limit of non-cancer hazard quotients (1.0). Non-cancer hazard quotient in winter is 26.2, followed by spring, autumn, and summer with 10.4, 9.2, and 4.5 respectively. Non-cancer hazard quotient in winter is even higher than the sum of non-cancer hazard quotients in the remaining three seasons. If non-cancer hazard quotients were contributed equally from four seasons, each season shares 25% of the total non-cancer hazard quotient. As depicted in Figure 4-9, winter accounts for 52% of the lifetime non-cancer hazard quotient, 27% higher than its share. Spring, autumn, and summer contributes 21%, 18%, and 9% of total non-cancer hazard quotient, relatively lower than corresponding share of 25%.

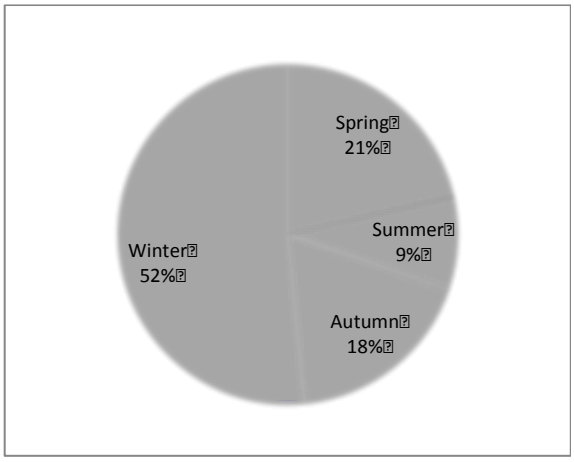
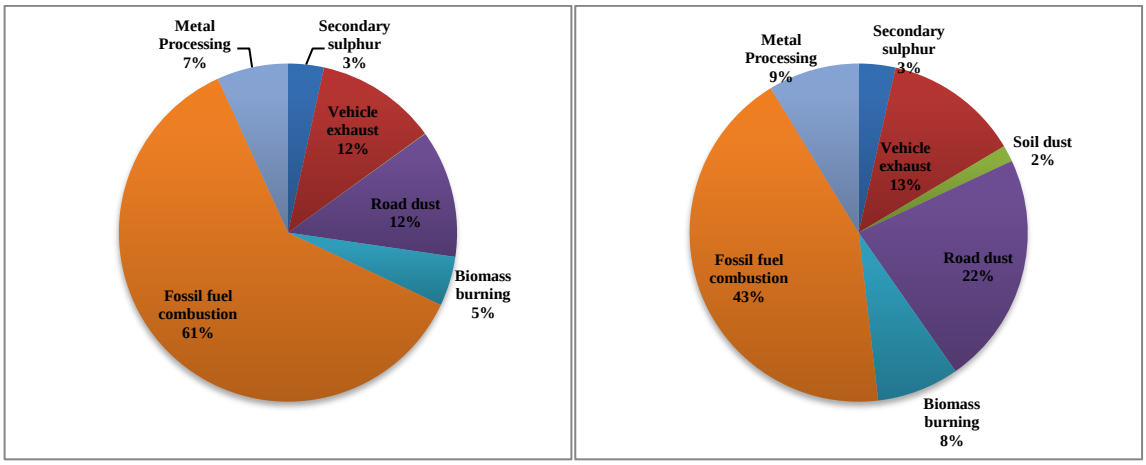


Figure 4-9: Distributions of lifetime non-cancer hazard quotients estimated by seasons.

The primary sources of non-cancer hazard quotients in four seasons are fossil fuel combustion as shown in Figure 4-10. Non-cancer hazard quotient of fossil fuel combustion in winter is 26.2, followed by spring, autumn, and summer with 10.4, 9.2, and 4.5 non-cancer hazard quotients, respectively. Winter heating leads to the increase of

non-cancer hazard quotient in winter. Percentages of fossil fuel combustion and non-cancer hazard quotients have the same seasonal trend.

Road dust is the second-largest contributor in spring, summer, and autumn and the third-largest contributor in winter. Non-cancer hazard quotients of road dust are in the order of winter (1.6) > autumn (1.4) > spring (1.2) >summer (0.95). As and Pb are the major contributors of road dust. In winter, As from road dust is the largest among all elements for road dust with 0.85 and the lowest one is Pb in summer.



(a) Spring (b) Summer

(c) Autumn (d) Winter

Figure 4-10: Distributions of non-cancer hazard quotients in (a) spring, (b) summer, (c) autumn, and (d) winter.

Vehicle exhaust is the second-largest source in spring and winter, while the third-largest contributor in summer and autumn. Non-cancer hazard quotients of vehicle exhaust peaks in winter with 2.4, followed by spring, autumn, and summer with 1.2, 1.2, and 0.55 non-cancer hazard quotients. The lowest non-cancer hazard quotient in summer consists of non-cancer hazard quotients from Cr, Mn, Ni, As, Pb, V, and Cl and the highest one is As with 1.0.

Percentages of metal processing are relatively constant by seasons compare to other sources, ranged from 7% to 9%. Non-cancer hazard quotients of metal processing in four seasons have the same order with vehicle exhaust. The highest value is 1.9 in winter, while the lowest value is 0.4 in summer. Secondary sulphur accounted for 3% of the total non-cancer hazard quotients in all seasons. However, non-cancer hazard quotients are changing in four seasons with the order of winter (0.8) > spring (0.4) > autumn (0.3) > (0.2) in summer. Soil dust is the smallest contributor of non-cancer hazard quotients. Percentages of soil dust were small in different seasons ranging from 0.03% to 0.4%.

Overall, non-cancer hazard quotients by sources in spring, summer, and autumn are in the same order of fossil fuel combustion > road dust > vehicle exhaust > metal processing > biomass burning > secondary sulphur > soil dust, different from in Winter. Non-cancer hazard quotients of sources are in order of fossil fuel combustion > vehicle exhaust > metal processing > road dust > biomass burning > secondary sulphur > soil dust. Road dust falls from the second-largest source to the forth-largest source in winter. Non-cancer hazard quotient in winter is the most condensed among four seasons. Fossil fuel combustion contributes 69% of total non-cancer hazard quotient in winter. Non-cancer hazard quotient in summer is relatively dispersed. The largest contributor, fossil fuel combustion, only contributed approximate 43% of total non-cancer hazard quotient.

Respiratory system is most vulnerable among all the target systems (Table 4-1). The highest non-cancer hazard quotient appeared in winter, followed by spring, autumn, and summer. Respiratory system impairment accounts for approximate 83% of total non-cancer hazard quotient in winter, which is due to high Cl emission in winter. The lowest percentage of respiratory system impairment is in summer, respiratory system impairment accounts for approximate 55% of total non-cancer hazard quotient. Non-cancer hazard quotient of respiratory system in winter is approximate 8.9 times than in summer.

Table 4-2: Non-cancer hazard quotients of different target organs in four seasons.

	Nervous	Fetus	Respiratory	Alimentary	Reproductive	Renal
Spring	1.2	0.00	7.1	0.0	1.9	0.2
Summer	0.8	0.00	2.5	0.0	1	0.2
Autumn	1.9	0.00	5.4	0.0	1.7	0.3
Winter	1.7	0.00	21.8	0.0	2.2	0.5

Reproductive system impairment is the second-largest non-cancer hazard quotient in spring, summer, and winter. It becomes the third-largest in autumn. Non-cancer hazard quotient of reproductive system impairment in summer is the smallest among all seasons. However, percentage of reproductive system impairment in summer is highest among four seasons with approximate 22%. Percentages of reproductive system impairment in summer and spring are similar with 18%. Lowest percentage of reproductive system impairment is observed in winter with 8%. On the contrary, non-cancer hazard quotient of reproductive system impairment in winter is the highest among four seasons.

Compare to the system impairments introduced above, non-cancer hazard quotients of renal system are low. The highest non-cancer hazard quotient of renal system impairment is 0.5 in winter, which is only 2% of the total non-cancer hazard quotient. Alimentary system impairment and fetus impairment are the two systems that have the non-cancer hazard quotients equal to 0. Ba is the only element that cause impairment to alimentary system, however Ba was only detected in summer with a small concentration. Thus, the non-cancer hazard quotients of alimentary system impairment were zero in all seasons.

4.1.4 Hours of day

Cancer risks

Overall lifetime cancer risk calculated by hours is 2.36E-02, two magnitudes higher than EPA upper limit of cancer risk (1.00E-04). Hourly $PM_{2.5}$ mass concentrations ranged from 84.6 to 70.0 $\mu g/m^3$, 21% varied over 24 hours as shown in Figure 4-11. A decreasing trend is observed for $PM_{2.5}$ mass concentrations from midnight to 7:00. $PM_{2.5}$ mass concentration decreased from 81.6 to 70.4 $\mu g/m^3$. $PM_{2.5}$ mass concentration

increased slightly after 7:00, peaks at 11:00 with 74.9 μg/m^3. It starts to decrease after 11:00, however the decreasing trend didn't last long. The daily low ends up the decreasing in 14:00 with 70.02 μg/m^3. The second increase trend starts from 14:00 and peaks in 22:00, which has the highest $PM_{2.5}$ mass concentration 84.6 μg/m^3. The peak time slot of day is observed between 21:00 and 24:00 with 84.4, 84.6, 84.2, and 84.4 μg/m^3 $PM_{2.5}$ mass concentrations, respectively.

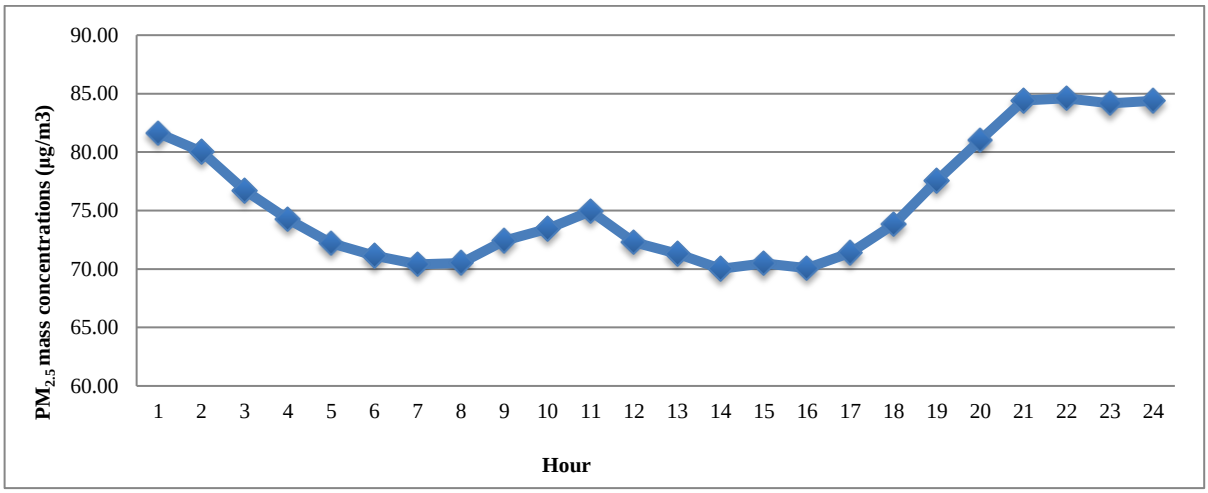


Figure 4-11: Hours of day $PM_{2.5}$ mass concentrations.

Cancer risks in nighttime (from 19:00 to 3:00) are relatively higher than daytime (from 4:00 to 18:00). If cancer risks were contributed equally from each of 24 hours, each hour should have accounted for approximate 4.17% of total cancer risk. The actual percentage of each hour were calculated and listed in Table 4-2. From 4 to 18, the percentages of cancer risks ranged from 4.10% to 3.87%, lower than corresponding share of 4.17%. On the contrary, the percentages from 19 to 3 are higher than corresponding share of 4.17%. The higher cancer risks in nighttime could due to the busy freight transportation during the night, the exhaust of some freight trucks did not meet the standards.

Table 4-3: Percentages of cancer risk for hours of day.

Hour	Percentage	Hour	Percentage
0	4.66%	12	3.98%
1	4.49%	13	3.93%
2	4.45%	14	3.86%
3	4.23%	15	3.89%
4	4.10%	16	3.86%
5	3.98%	17	3.94%
6	3.92%	18	4.07%
7	3.88%	19	4.28%
8	3.89%	20	4.45%
9	4.00%	21	4.66%
10	4.05%	22	4.66%
11	4.13%	23	4.66%

The overall trend of cancer risks in 24 hours agrees with $PM_{2.5}$ mass concentration as illustrated in Figure 4-12. $PM_{2.5}$ mass concentrations for the peak time from 21:00 to 24:00 are slightly different but cancer risks for these four different hours are the same -- 1.10E-03.

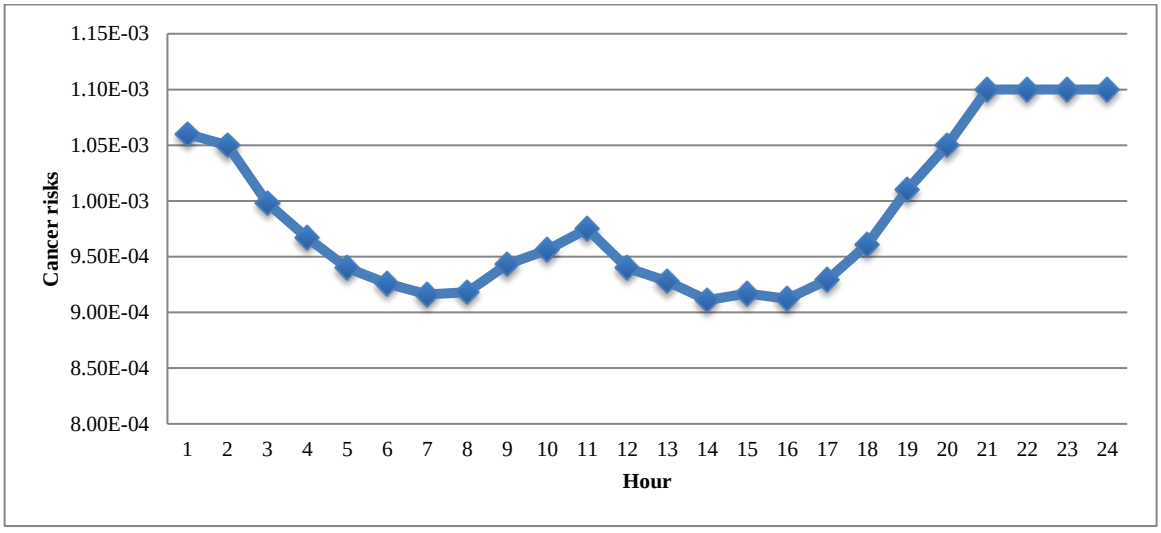


Figure 4-12: Cancer risks estimated from different hours.

Percentages and orders of cancer risks by components and sources are the same in different hours. Besides, percentages and orders of cancer risks in different hours are the same as in three-year average cancer risks. Details were discussed in previous sections.

Non-cancer hazard quotients

The overall lifetime non-cancer hazard quotient calculated by hours of day is 14.0. Non-cancer hazard quotients for each hour vary from 13.0 to 15.7. The highest non-cancer hazard quotient is at 22:00 while the daily low is at 14:00. The daily trend of non-cancer hazard quotients as shown in Figure 4-13 agrees with the trend of $PM_{2.5}$ mass concentrations as shown in Figure 4-11.

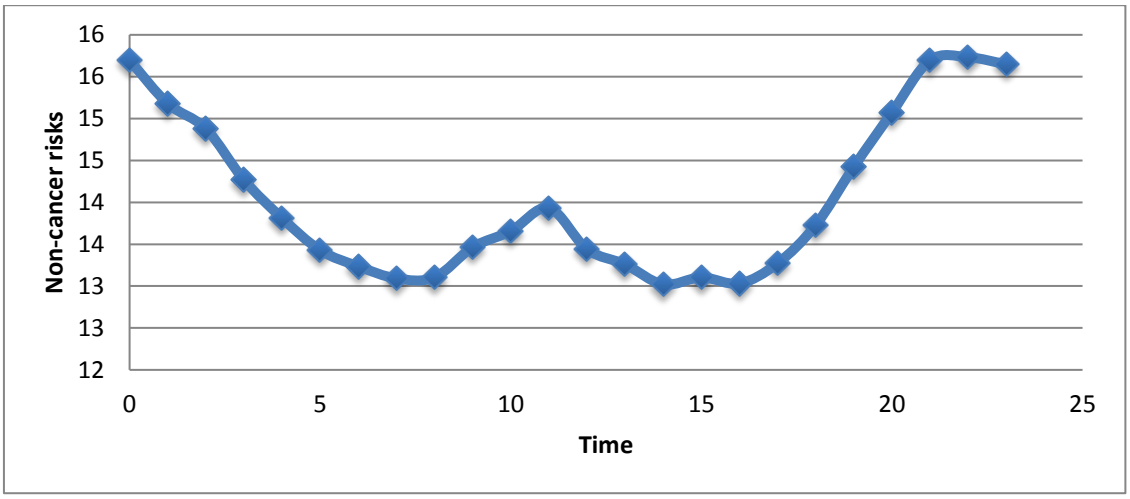


Figure 4-13: Non-cancer hazard quotients estimated from hours of day.

Non-cancer hazard quotients in nighttime (from 19:00 to 3:00) are relatively higher than daytime (from 4:00 to 18:00). Non-cancer hazard quotients in nighttime are higher than the average non-cancer hazard quotient of hours of day (14.0), while non-cancer hazard quotients in daytime are lower. Percentages of non-carcinogenic elements for hours of day are similar to the percentages of elements in three-year average. The same situation could be observed for source apportionment as well. Percentages of different target organs in different hours are similar to three-year average, shown in Figure 4-3(c). Details were introduced again in this section.

4.2 Spatial results in Beijing

4.2.1 Urban-suburban groups

Cancer risks

Due to absent of PAHs concentration, cancer risks discussed in this subsection only include risks from six carcinogenic elements. The overall trend of cancer risks in different groups is the same as what we predicted by CF values in Figure 3-4 (a). Overall lifetime cancer risks in both urban area and suburban area are higher than EPA upper limit of cancer risks (1.00E-04).

$PM_{2.5}$ mass concentration is 79.0 μg/m^3 in urban area with corresponding lifetime cancer risk 1.82E-02. Compare to all stations of Beijing (All), $PM_{2.5}$ mass concentration in urban area is 3.4 μg/m^3 higher while total cancer risk is 4.5E-03 lower. The higher cancer risk in All is due to Cr, the only element that has higher cancer risk in All than urban area among six carcinogenic elements as shown in Figure 4-14. Besides Cr, the cancer risks from the rest five elements in urban area are all higher than All.

For suburban area, $PM_{2.5}$ mass concentration is 68.3 μg/m^3 with 3.53E-02 cancer risk. Compare to All, $PM_{2.5}$ mass concentration in suburban is 7.4 μg/m^3 lower while the cancer risk is 1.26E-02 higher. The distinct difference between All and suburban area is mainly due to Cr. Cancer risks of Cr is 1.19E-02 in All while 3.19E-02 in suburban area. Except for Cr, the other five carcinogenic elements all have higher cancer risks in All than suburban area. Co and Cd were not detected in suburban area, cancer risk of Co is 3.25E-03 in All and Cd is 3.34E-04. Among six carcinogenic elements, Pb is the only carcinogenic element that has higher cancer risk in suburban area than in All.

Distinct difference exists between urban area and suburban area. $PM_{2.5}$ concentration in urban area is 10.8 μg/m^3 higher than suburban area while the cancer risk is 1.71E-02 lower. Cancer risk in suburban area is approximate 2 times of cancer risk in urban area. The larger Cr cancer risk observed in suburban area explains why cancer risk is higher in suburban area than in urban area. Cancer risk of Cr in suburban area is 5.0 times higher

than in urban area. Except for Cd and Co, which were not detected in suburban research station, cancer risks of remaining carcinogenic elements Ni, As, and Pb are all higher in urban area.

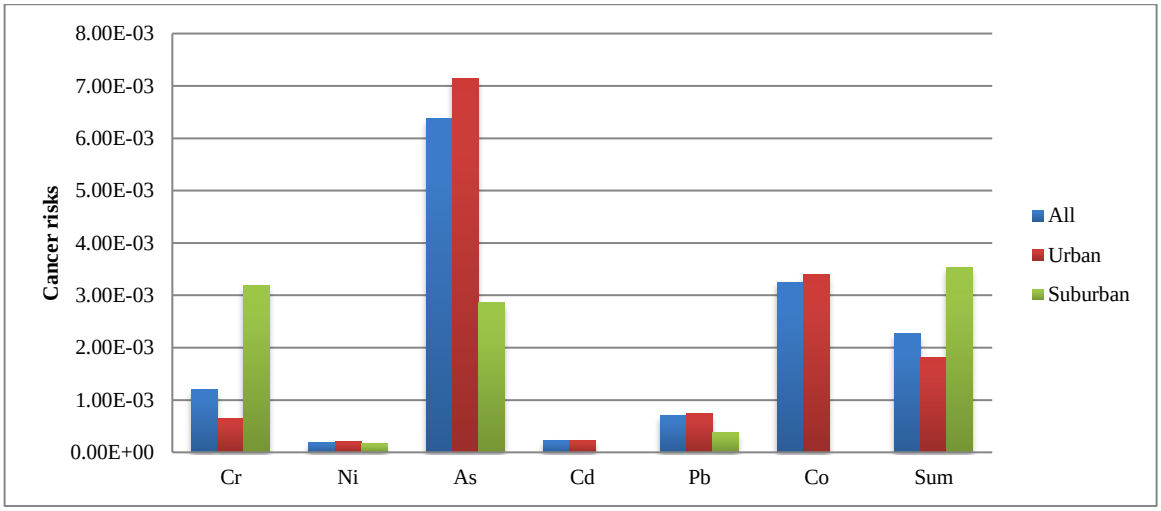
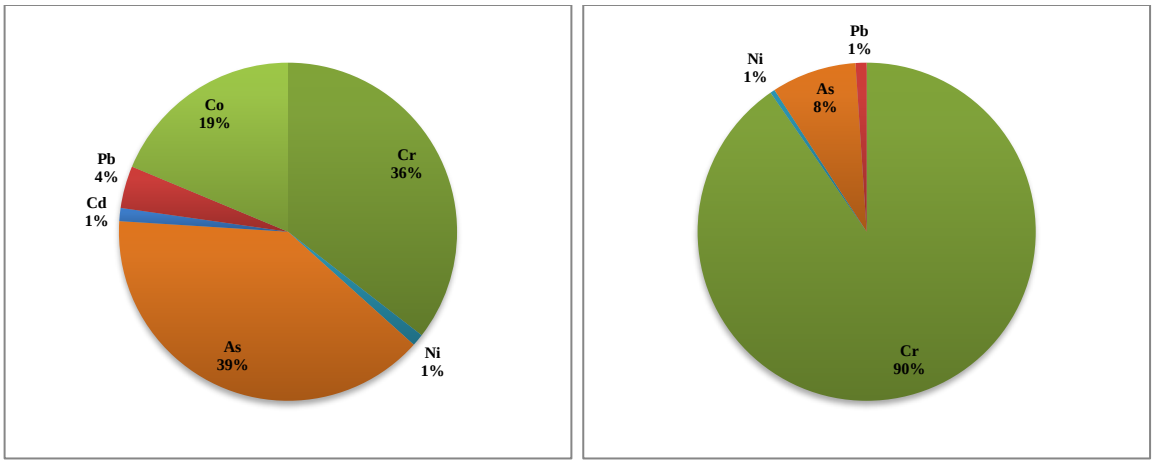


Figure 4-14: Cancer risks in all stations, urban area, and suburban area of Beijing (Cr and sum are shown in 1/10).

Distributions of cancer risks are quite different between urban area and suburban area. The contribution order of cancer risks is As > Cr > Co > Pb > Cd > Ni in urban area and Cr > As > Pb > Ni in suburban area (Figure 4-15). As is the largest contributor of total cancer risk in urban area, it accounts for 39% of cancer risk. However, As is the second-largest contributor in suburban area, cancer risk of As in suburban area is 40% of cancer risk of As in urban area. Cr accounts for approximate 36% of the urban area cancer risk, while this percentage increases to 90% in suburban area. Co and Cd account for approximate 19% and 1% of total cancer risk in urban area, while these two elements were not detected in suburban area. Compare to other elements, percentages of Pb and Co are small with 4% and 1% in urban area and 1% and 1% in suburban area.

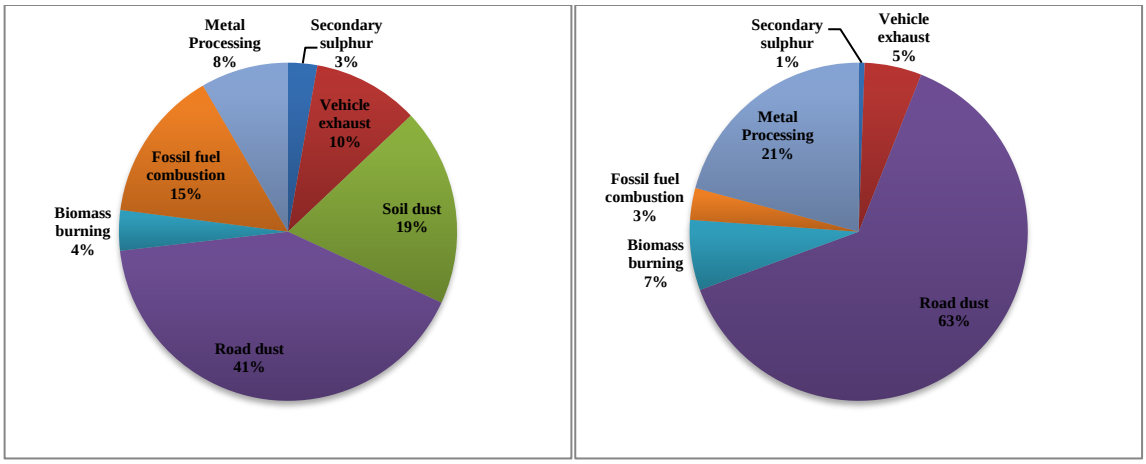


(a) urban area (b) suburban area

Figure 4-15: Distributions of elemental cancer risks in (a) urban area and (b) suburban area by elements.

Cancer risk in urban area has seven sources and are in order of road dust > soil dust > fossil fuel combustion > vehicle exhaust > metal processing > biomass burning > secondary sulphur as illustrated in Figure 4-16. Compare to urban area, cancer risk in suburban area only has six sources. The order of the contribution sources in suburban area is road dust > metal processing > biomass burning > vehicle exhaust > fossil fuel combustion > secondary sulphur. There is no cancer risk distributed to soil dust, because Co, which is the only contributor of soil dust among six carcinogenic elements, was not detected in suburban area. Cancer risks of soil dust, fossil fuel combustion, and secondary sulphur are higher in urban area than in suburban area, while vehicle exhaust, road dust, biomass burning, and metal processing are higher in suburban area than in urban area.

Road dust is the primary source of cancer risk in both urban area and suburban area. Road dust contributes approximate 41% and 63% of total cancer risk in urban area and suburban area, respectively. Soil dust is the second-largest source in urban area, accounting for approximate 19% of total cancer risk in urban area. As mentioned above, Co was not detected in suburban area, thus there is no cancer risk from soil dust in suburban area.



(a) urban area (b) suburban area

Figure 4-16: Distributions of elemental cancer risks in (a) urban area and (b) suburban area by sources.

Fossil fuel combustion is the third-largest contributor in urban area and the fifth-largest contributor in suburban area. Cancer risk of fossil fuel combustion accounts for approxiamte 15% of total cancer risk in urban area and 3% in suburban area. Cancer risk of fossil fuel combustion in urban area is 2.4 times of corresponding cancer risk in suburban area. Cancer risks of vehicle exhaust are similar in both urban and suburban areas with 1.82E-03 and 1.93E-03, respectively. Percentage of vehicle exhaust in suburban area is 6% higher than urban area.

Metal processing is the sixth-biggest contributor in urban area with approximate 8% of total cancer risk. However, metal processing is the second-largest source in suburban area with 21% of total cancer risk. Cancer risk of metal processing in urban area is 4.9 times of corresponding cancer risk in suburban area. Biomass burning follows after metal processing in both urban and suburban area, accounting for 4% and 7%, respectively. Secondary sulphur is the smallest contributor in both urban and suburban area, with 3% and 1% of total cancer risks, respectively.

Non-cancer hazard quotients

Among thirteen non-carcinogenic elements, Ba and P were not detected in urban area and Cd, Co, V, Cl, and Se were not detected in suburban area. Generally, trend of non-cancer

hazard quotients is similar to what we predicted from CF values as shown in Figure 3-5 (b). Overall lifetime non-cancer hazard quotient is 14.3 in urban area and 2.6 in suburban area. Non-cancer hazard quotient in urban area is 5.4 times higher than in urban area (Figure 4-17).

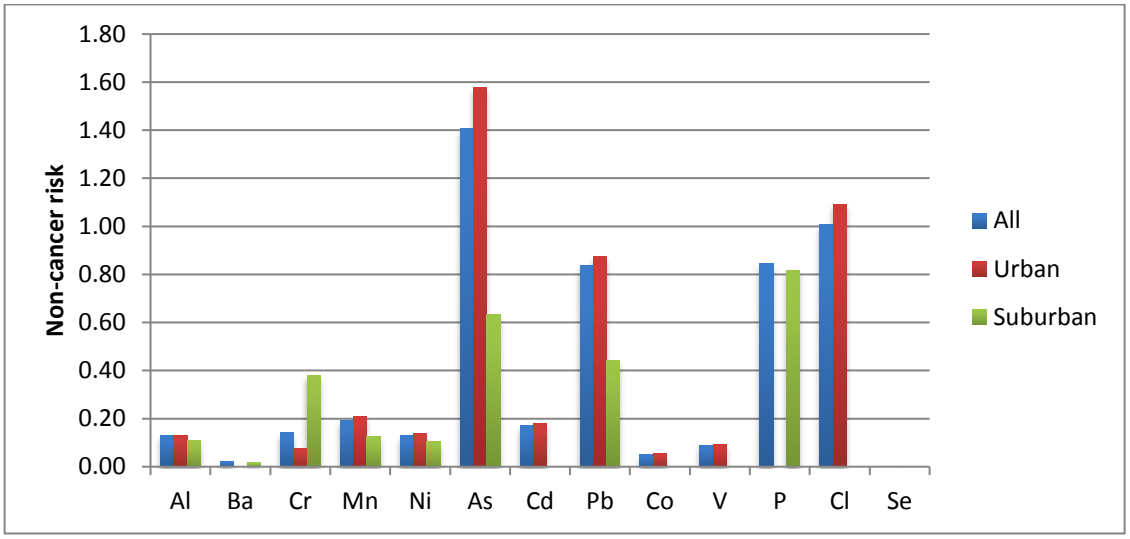
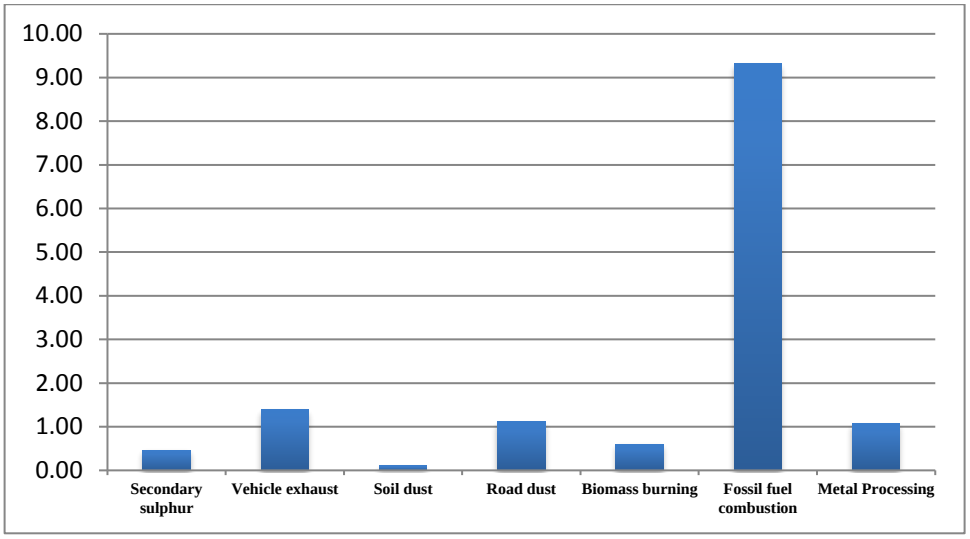


Figure 4-17: Non-cancer hazard quotients in all stations, urban area, and suburban area of Beijing (Cl is shown in 1/10).

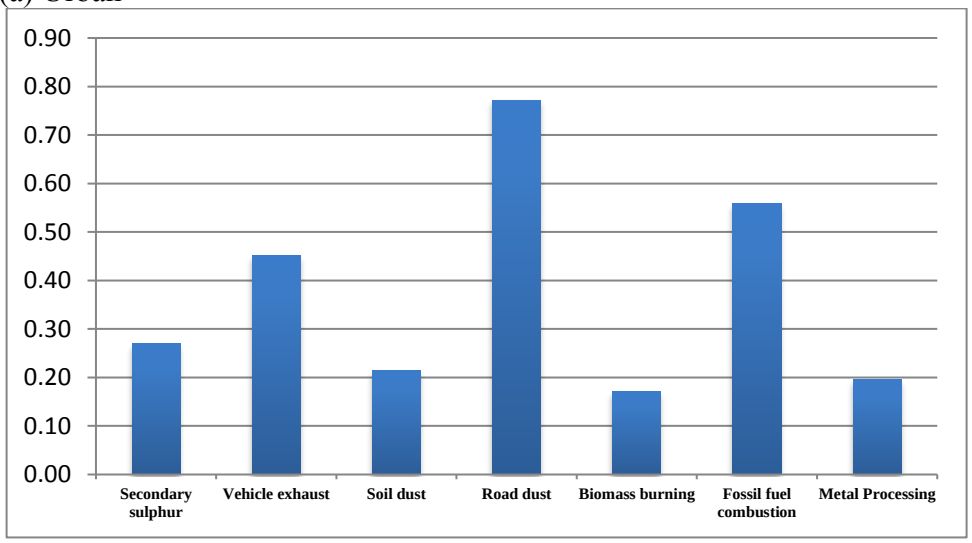
The difference of non-cancer hazard quotient between urban and suburban area is 11.6. Non-cancer hazard quotient in urban area is 5.4 times of suburban area. The non-cancer hazard quotient for Cl is 10.9 in urban area while 0 in suburban area because Cl was not detected in suburban. Difference caused by Cl accounts for approximate 94% of total difference. Besides Cl, Cd, Co, V and Se were not detected in suburban either. P, Cr, and Ba are higher in suburban area with differences of 0.8, 0.3, and 0.02, the remaining nine non-carcinogenic elements are lower in suburban area with the differences ranging from 0.02 to 0.94.

Distributions of elemental non-cancer hazard quotient in urban and suburban areas are different. Non-cancer hazard quotient of fossil fuel combustion is 9.3 in urban area and 0.6 in suburban area. Non-cancer hazard quotient of fossil fuel combustion in urban area is 16.7 times of the corresponding non-cancer hazard quotient in suburban area. Fossil fuel combustion contributes approximate 9.3 of total non-cancer hazard quotient in urban

area, followed by vehicle exhaust, road dust, metal processing, biomass burning, secondary sulphur, and soil dust, with corresponding non-cancer hazard quotient of 1.4, 1.1, 1.1, 0.6, 0.5, and 0.1 as shown in Figure 4-18.



(a) Urban



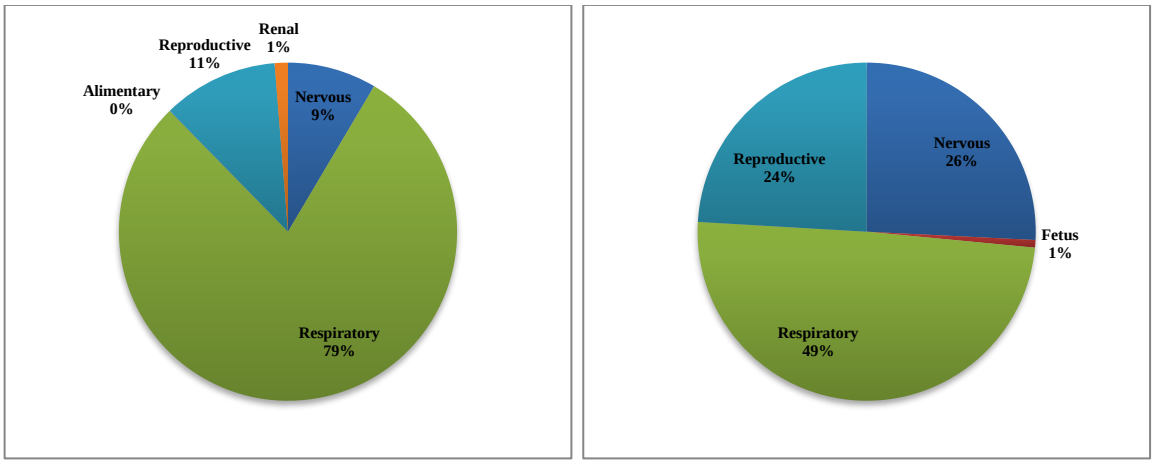
(b) Suburban

Figure 4-18: Distributions of elemental non-cancer hazard quotients in (a) urban area and (b) suburban area by sources.

Non-cancer hazard quotients in suburban area are dispersed. The non-cancer hazard quotients in different sources are all lower than EPA upper threshold of hazard quotient (1.0). Road dust is the major contributor in suburban area, however it only accounts for

approximate 29% of total amount. Fossil fuel combustion, which is the major source in urban area, is the second-largest contribution in suburban area.

The numbers of influenced target organs are different in urban and suburban area. There are five target organs in urban area and four in suburban area as illustrated in Figure 4-19. Among five influenced system impairments in urban area, the most vulnerable system impairment is respiratory system impairment accounting for approximate 79% of total non-cancer hazard quotient. Followed by reproductive system impairment, nervous system impairment, renal system impairment, and alimentary system impairment ranging from 11% to 1% of total non-cancer hazard quotient.



(a) Urban (b) Suburban

Figure 4-19: Distributions of elemental non-cancer hazard quotients in (a) urban area and (b) suburban area by sources.

Respiratory system impairment is also the most vulnerable system impairment in suburban area with non-cancer hazard quotient, which accounts for approximate 49% of total non-cancer hazard quotient. Non-cancer hazard quotient of respiratory system impairment in urban area is 8.7 times higher than in suburban area. Non-cancer hazard quotients of reproductive system impairment, nervous system impairment in suburban area are similar, accounting for 25% of total non-cancer hazard quotient. Percentages of reproductive system impairment and nervous system impairment in suburban area are

higher than corresponding percentages in urban area, however the non-cancer hazard quotients of reproductive system impairment and nervous system impairment in suburban area are approximate half of corresponding non-cancer hazard quotients in urban area.

The least-influenced system in urban area is alimentary system, with only 0.01 non-cancer hazard quotient. Non-cancer hazard quotient of fetus is zero in urban area, because Ba was not detected in urban area. Fetus suffers the smallest influence in suburban area. Non-cancer hazard quotients of alimentary system and renal system in suburban area are zero, because Se and Cd, the elements influence alimentary system and renal system, were not detected in suburban area.

4.2.2 Ring groups

Cancer risks

Overall trend of cancer risks in ring groups is similar to what we predicted by CF values as shown in Figure 3-5 (a). Overall lifetime cancer risks in ring groups are all higher than EPA upper limit of cancer risks (1.00E-04) with 2.34E-02, 1.48E-02, and 3.53E-02, respectively. Cd and Co were not detected in R>5 group, thus the cancer risks of these two elements are. Besides Ni in R3-5, cancer risks of single carcinogenic elements in different ring groups are all higher than EPA upper limit (1.00E-04), let alone the total cancer risks.

$PM_{2.5}$ mass concentration is 79.1 $\mu g/m^3$ in R<3 group, 5.4 $\mu g/m^3$ higher than three-year average. However, overall lifetime cancer risk is 2.00E-04 lower than All group as shown in Figure 4-20. Among six carcinogenic elements, Cr is the only one that has higher cancer risks in All than in R<3 group. Besides Cr, cancer risks from the remaining five carcinogenic elements in R>3 group are higher than corresponding cancer risks in All.

For R3-5 group, $PM_{2.5}$ mass concentration is 78.8 $\mu g/m^3$. Compare to All, $PM_{2.5}$ mass concentration in R3-5 is 5.1 $\mu g/m^3$ higher while cancer risk is 7.90E-03 lower. Cancer risks of Cr is 6.28E-02 higher in R3-5 than in All, approximate 6 times of overall lifetime cancer risk than in All. Besides Cr, Pb is also higher in R3-5 than in All with only

approximate 6% higher. The remaining four carcinogenic elements are higher in All than in R3-5 group ranging from 9.3 times for Co to 1.05 times for As.

Total cancer risks and the distribution of elemental cancer risks in R>5 group is the same as in suburban area. Overall lifetime cancer risks are in order of (R>5) > (All) > (R<3) > (R3-5). Cancer risk of Cr in R>5 group is way much higher than in other groups with 4.3, 8.8 times of R3-5 and R<3, respectively.

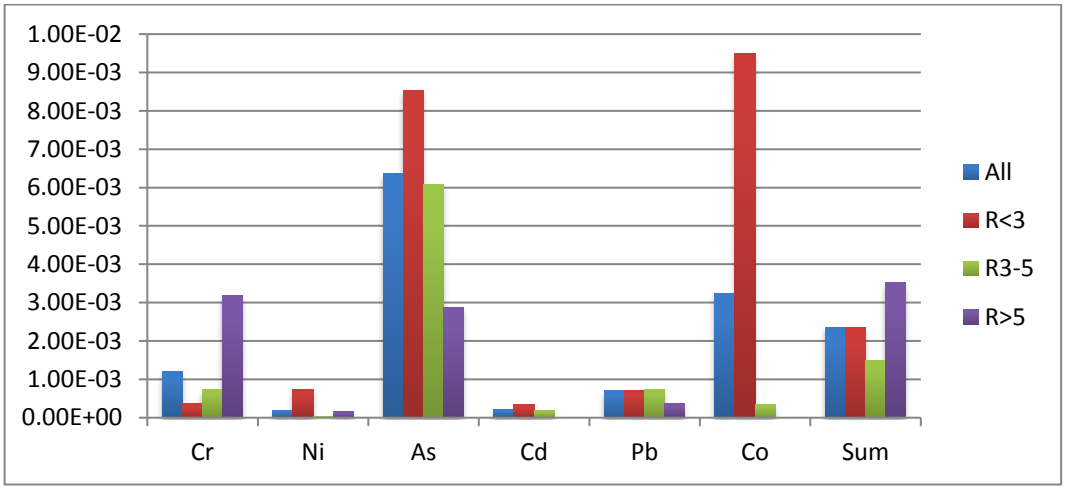
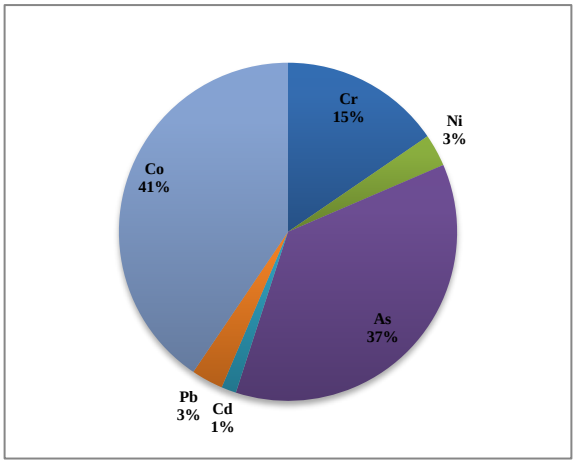


Figure 4-20: Cancer risks in all stations, R<3, R3-5, and R>5 of Beijing (Cr and sum are shown in 1/10).

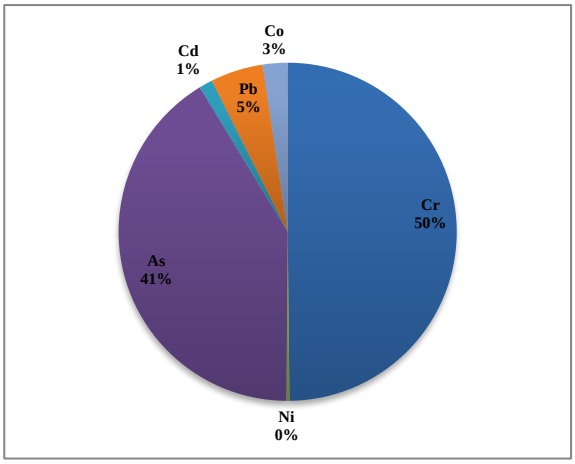
Co is the major contributor of cancer risk within third ring area, contributing approximate 41% of total cancer risk as shown in Figure 4-21(a). Percentages of Co decrease with increase of numbers of ring. It is 3% in R3-5 and 0% in R>5. Cr is the primary contributor in both R3-5 and R>5 and the third-largest contributor in R<3. Percentages of Cr increase with the numbers of ring. Cr contributed 15%, 50%, and 90% in R<3, R3-5, and R>5, respectively.

Cancer risks of As are decreasing with the increase of numbers of ring, while percentages of As in total cancer risk peaks in R3-5 followed By R<3 and R>5 with 41%, 37%, and 8%, respectively. Pb has the same order with As with corresponding percentages of 5%, 3%, and 1%. Ni only accounts for small fractions of total cancer risks in all groups,

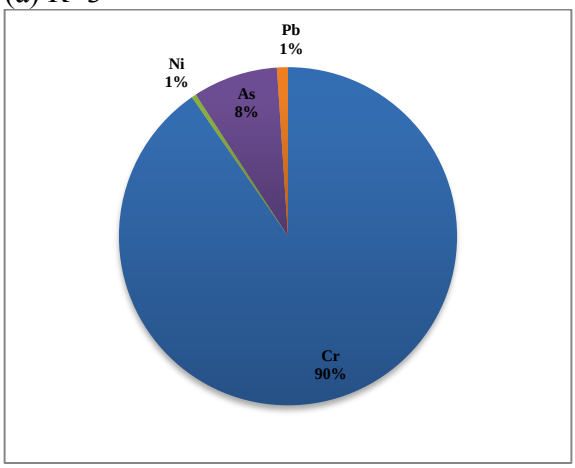
ranging from 1% to 3%. Cd was not detected in R>5 and Cd accounts for 1% in both R3-5 and R<3.



(a) R<3



(b) R3-5



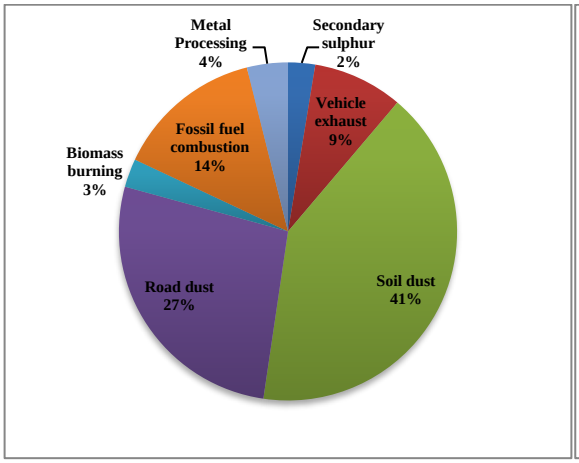
(c) R>5

Figure 4-21: Distributions of elemental cancer risks in (a) R<3, (b) R3-5, and (c) R>5 by elements.

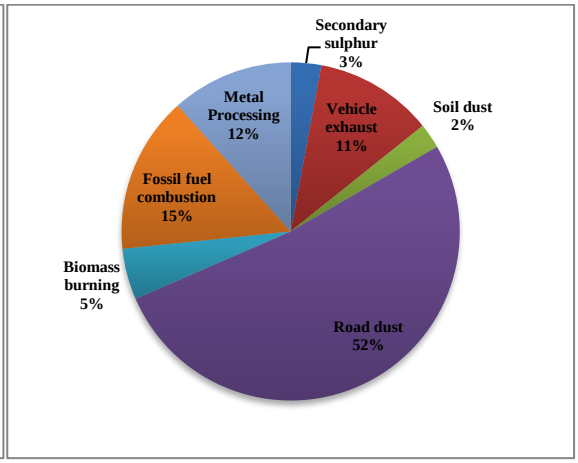
Soil dust is the main source of lifetime overall cancer risk in R<3 as shown in Figure 4-22(a). Among six carcinogenic elements, Co is the only one that 100% comes from soil dust. Thus, the order of soil dust is the same as the order of Co, accounting for 41%, 2%, and 0% in R<3, R3-5, and R>5, respectively. Road dust is the second-largest contributor in R<3, while the primary contributor in both R3-5 and R>5. Road dust accounts for approximate 27%, 52%, and 63% of total cancer risks in R<3, R3-5, and R>5,

respectively. The same order could be seen for metal processing and biomass burning ranging from 3% to 7%, and from 4% to 21%, respectively.

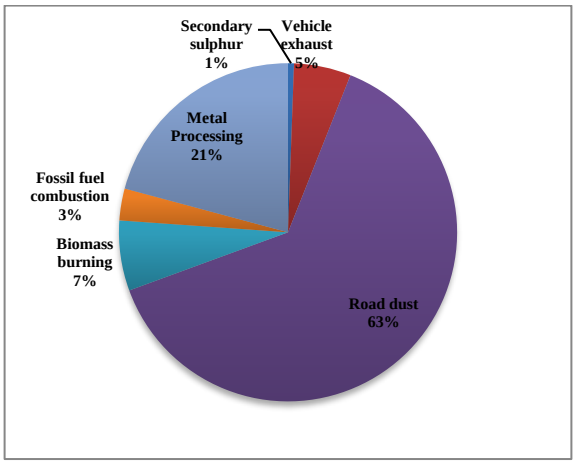
Fossil fuel combustion, vehicle exhaust, and secondary sulphur have the same order in ring groups. The peak percentage is shown in R3-5 group, followed by R<3 and R>5 groups. For example, fossil fuel combustion accounts for approximate 15% in R3-5, followed by 14% in R<3 and 3% in R>5. Secondary sulphur contributes the smallest cancer risk among these three sources, ranging from 1% to 3%.



(a) R<3



(b) R3-5



(c) R>5

Figure 4-22: Distributions of elemental cancer risks in (a) R<3, (b) R3-5, and (c) R>5 by sources.

Non-cancer hazard quotients

The overall lifetime non-cancer hazard quotients are 13.3, 14.7, and 2.6 in R<3, R3-5, and R>5, respectively, they are all higher than EPA upper limit (1.0). The highest non-cancer hazard quotient was calculated in R3-5, followed by R<3 and R>5. Compare to All, the non-cancer hazard quotient is 0.4 lower in R<3, 1.0 higher in R3-5, and 11.1 higher in R>5. Trend of elemental non-cancer hazard quotients in different ring groups are similar to what we expected in Figure 3-5 (b), as shown in Figure 4-23.

Among thirteen non-carcinogenic elements, Ba and P were not detected in R<3 and R3-5 and Cd, Co, V, Cl, and Se were not detected in R>5 area. Most of non-cancer hazard quotients from single element are lower than one except for As and Cl in R<3 and R3-5.

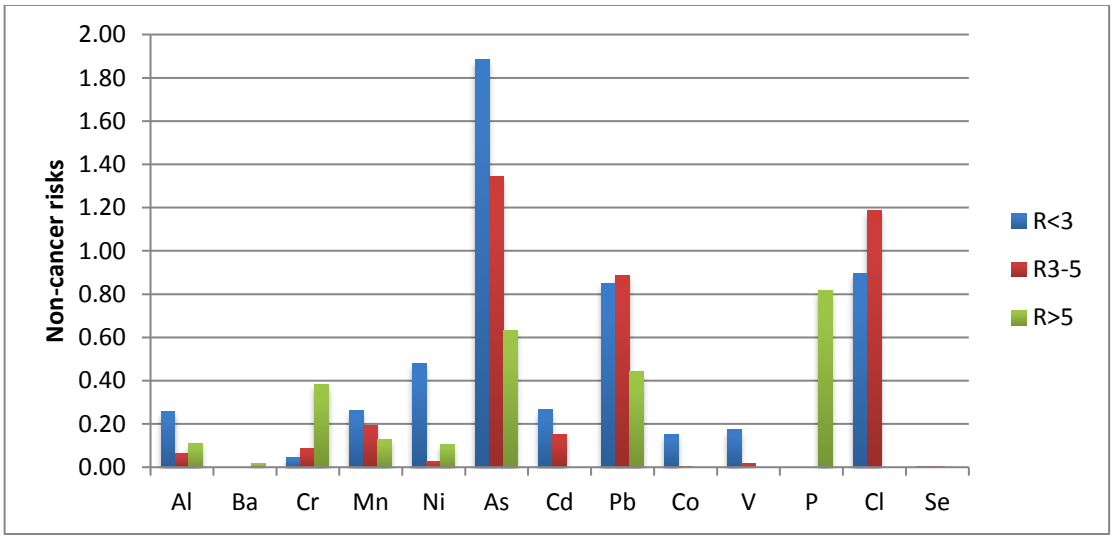


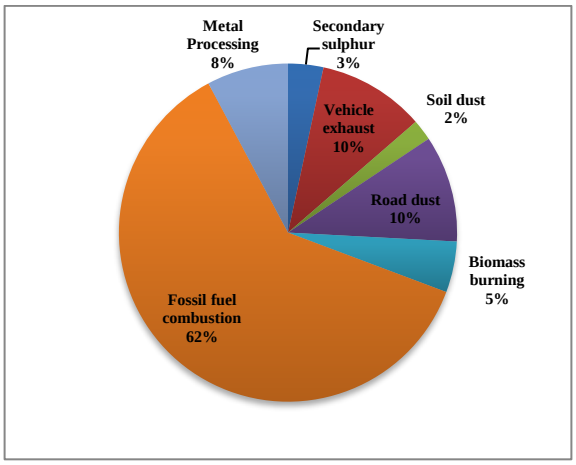
Figure 4-23: Non-cancer hazard quotients in all stations, R<3, R3-5, and R>5 of Beijing (Cl is shown in 1/10).

Among all non-carcinogenic elements, the biggest difference between All and R<3 is from Cl, 1.1 lower in R<3 than in All. P has the second-largest difference, 0.9 lower in R<3 than in All. Besides Cl and P, Ba and Cr are slightly lower in R<3 with the differences of 0.02 and 0.1, respectively. The remaining elements are all higher in R<3 than in All.

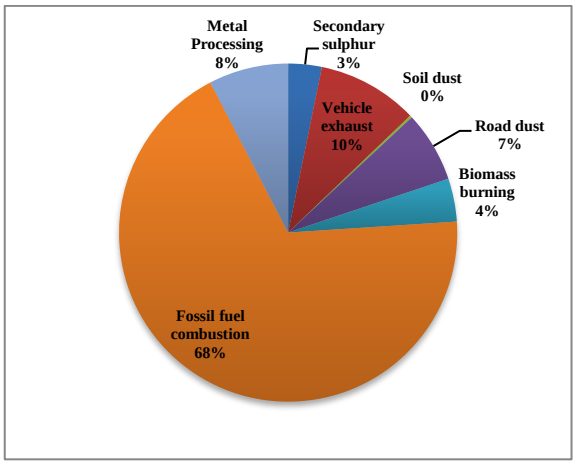
R3-5 is the only group that has higher non-cancer hazard quotient than All among all ring groups. The largest difference comes from Cl as well, which is 1.8 higher in R3-5 than in All. Except for Se, non-cancer hazard quotients of which are zero in R3-5 and in All, the remaining non-carcinogenic elements are all lower in R3-5 than in All.

R>5 has the same area as suburban area, non-cancer hazard quotients of each non-carcinogenic element are the same in these two groups. Thus, order of sources and differences of non-carcinogenic elements are the same as introduced in non-cancer hazard quotients in suburban area.

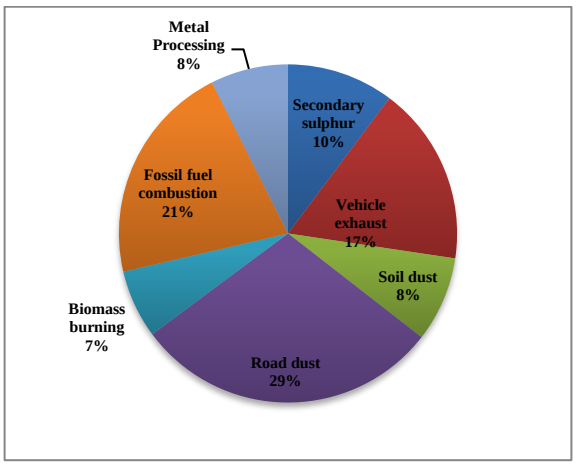
Overall, the distributions of non-cancer hazard quotients are similar in R<3 and R3-5 as shown in Figure 4-24. Fossil fuel combustion is the major contributor in R<3 and R3-5 with corresponding non-cancer hazard quotient of 8.0 and 9.9. Besides, non-cancer hazard quotients for vehicle exhaust, road dust, and metal processing in R<3 and R3-5 are all higher than EPA upper limit (1.0). The distribution of non-cancer hazard quotient in R>5 group is more dispersed. Road dust is the major source in R>5 with corresponding non-cancer hazard quotient of 0.77. Thus, the non-cancer hazard quotients from all the sources in R>5 groups are all lower than EPA upper limit (1.0).



(a) R<3



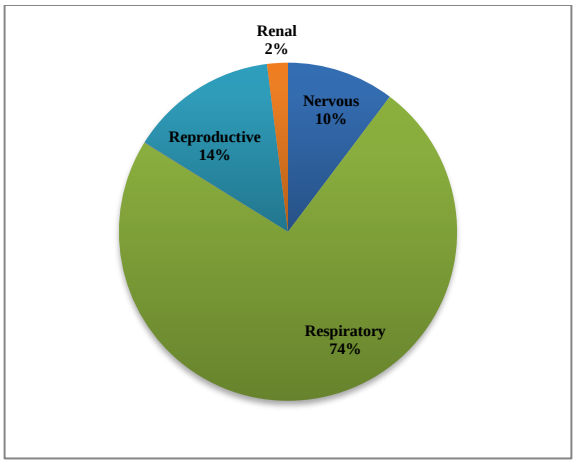
(b) R3-5



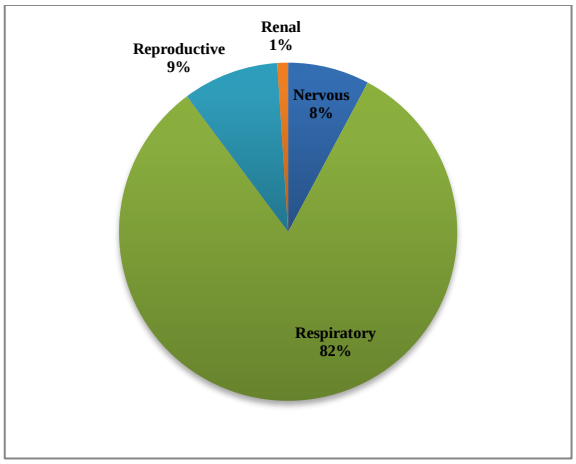
(c) R>5

Figure 4-24: Distributions of non-cancer hazard quotients in (a) R<3, (b) R3-5, and (c) R>5 by sources.

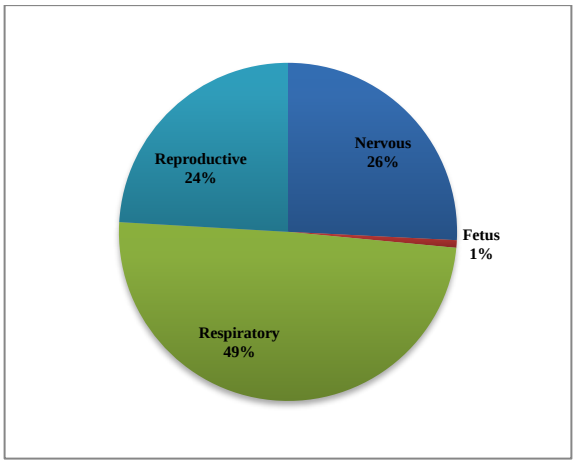
Distributions of non-cancer hazard quotients in R<3 and R3-5 are similar as shown in Figure 4-25. In R<3 and R3-5, respiratory system accounts for approximate ¾ of total non-cancer hazard quotient with 9.8 and 12.0 respectively. Besides respiratory system, the non-cancer hazard quotients of reproductive system, nervous system are all higher than one in R<3 and R3-5.



(a) R<3



(b) R3-5



(c) R>5

Figure 4-25: Distributions of non-cancer hazard quotients in (a) R<3, (b) R3-5, and (c) R>5 by target organs.

In R>5 group, non-cancer hazard quotient of respiratory system accounts for approximate 49% of total amount, followed by nervous system (26%) and reproductive system (24%). The non-cancer hazard quotient in R>5 group is the only one that higher than EPA upper limit of non-cancer hazard quotient (1.0). The non-cancer hazard quotients of reproductive system, nervous system, and fetus are all lower than one, ranging from 0.02 to 0.7.

4.2.3 Nearest distance groups

Cancer risks

Cancer risks of governmental stations are all higher than EPA upper limit (1.00E-04). The highest cancer risk is in HR and the lowest cancer risk is in DL. Cancer risk in HR is 62 times of cancer risk in DL. As we mentioned before, urban area has higher $PM_{2.5}$ mass concentrations than suburban area. For the suburban area, east part (HR and SY) has higher $PM_{2.5}$ mass concentration than west part (DL and CP). Similarly, east part of suburban area has higher cancer risks than west part. For urban area, cancer risks in south part (WS, DS, GY, GC, and TT) are higher than north part (HD, NZG, and ATZX) (Table 4-4).

Table 4-4: Cancer risks in twelve governmental sampling sites.

Station	WS	DS	GY	TT	GC	HD
Cr		3.69E-03				5.73E-03
Ni		7.39E-04				4.06E-05
As	1.49E-02	6.64E-03	1.46E-02	1.47E-02	1.48E-02	2.12E-03
Cd						1.98E-04
Pb	7.84E-04	6.42E-04	7.69E-04	7.70E-04	7.80E-04	4.84E-04
Co		9.70E-03				3.81E-04
Sum	1.57E-02	2.14E-02	1.54E-02	1.54E-02	1.56E-02	8.95E-03
Station	NZG	ATZX	SY	HR	CP	DL
Cr	1.15E-03	1.08E-03	1.47E-02	3.73E-02		
Ni	3.67E-05	3.45E-05	4.60E-05	1.86E-04		
As	4.14E-03	3.90E-03	4.63E-03	3.35E-03		
Cd	9.53E-05	8.97E-05	2.76E-04			
Pb	4.83E-04	4.54E-04	1.09E-03	4.35E-04	6.64E-04	6.19E-04
Co	3.38E-04	3.18E-04				
Sum	6.24E-03	5.87E-03	2.08E-02	4.12E-02	6.64E-04	6.19E-04

Four (WS, GY, TT, and GC) out of five stations in south part were linked with CGZ station and only two carcinogenic elements were detected in CGZ—As and Pb. As is the primary contributor accounting for approximate 95% of overall lifetime cancer risk. DS also locates in the south part of urban area, the closest research station is Beijing Normal University. Among five carcinogenic elements, cancer risk of Co is the highest

accounting for 45% of total cancer risk, followed by As, Cr, Ni, and Pb with 31%, 17%, 4%, and 3%, respectively.

In the north part of urban area, the highest $PM_{2.5}$ mass concentration and cancer risk are both in HD, cancer risks in NZG and ATZX are 2.71E-03 and 3.08E-03 lower. Primary contributor is Cr in HD, AS in NZG, and ATZX with approximate 63%, 66%, and 66% of overall lifetime cancer risks. The sum of cancer risks of As and Cr accounts for over 80% of total cancer risk in all stations in north part of urban area.

Although HR and SY have different research stations, orders of carcinogenic elements in SY and HR are similar. Five carcinogenic elements were detected in SY, they are in order of Cr > As > Pb > Cd > Ni. Four carcinogenic elements in HR, order of cancer risks is Cr > As > Pb > Ni. Cr is the primary contributor in both HR and SY, while Ni is the smallest contributor.

The highest and lowest cancer risks are both observed in suburban area. Compare to HR and SY, cancer risks of CP and DL, both of which locate in the west part of suburban area, are much lower. Pb is the only carcinogenic element detected in CP and DL. Although the cancer risks are low in CP and DL, cancer risks of them are still higher than EPA upper limit (1.00E-04).

The main source is road dust in all governmental stations except for DS, the main source of DS is soil dust as shown in Table 4-5. In the south part of urban area, road dust accounts for 39% of total cancer risk in WS, GY, TT, and GC stations. Cancer risks of fossil fuel combustion are the second-largest source among all the sources in WS, GY, TT, and GC, with approximate 34% of total cancer risk. Besides, vehicle exhaust, secondary sulphur, and biomass burning accounts for approximate 19%, 7%, and 1% of total cancer risks, respectively.

DS has different distributions of cancer risks from any other stations in south part of urban area. Soil dust is the primary contributor, followed by road dust, fossil fuel combustion, vehicle exhaust, metal processing, biomass burning, and secondary sulphur with corresponding percentages of 45%, 26%, 12%, 8%, 4%, 3%, and 2%.

Table 4-5: Distributions of cancer risks in twelve governmental stations by sources.

	Secondary sulphur	Vehicle exhaust	Soil dust	Road dust	Biomass burning	Fossil fuel combustion	Metal Processing
WS	1.04E-03	3.05E-03	0.00E+00	6.15E-03	1.96E-04	5.27E-03	0.00E+00
DS	4.65E-04	1.61E-03	9.70E-03	5.51E-03	6.19E-04	2.59E-03	9.24E-04
GY	1.03E-03	2.99E-03	0.00E+00	6.03E-03	1.92E-04	5.17E-03	0.00E+00
TT	1.03E-03	2.99E-03	0.00E+00	6.04E-03	1.93E-04	5.18E-03	0.00E+00
GC	1.04E-03	3.03E-03	0.00E+00	6.12E-03	1.95E-04	5.24E-03	0.00E+00
HD	1.48E-04	7.64E-04	3.81E-04	4.82E-03	5.33E-04	7.82E-04	1.32E-03
NZG	2.90E-04	9.64E-04	3.38E-04	2.59E-03	2.11E-04	1.49E-03	2.68E-04
ATZX	2.73E-04	9.07E-04	3.18E-04	2.43E-03	1.99E-04	1.40E-03	2.52E-04
SY	3.24E-04	1.76E-03	0.00E+00	1.20E-02	1.31E-03	1.70E-03	3.39E-03
HR	2.34E-04	2.25E-03	0.00E+00	2.61E-02	2.77E-03	1.26E-03	8.59E-03
CP	0.00E+00	1.79E-04	0.00E+00	2.79E-04	1.66E-04	3.98E-05	0.00E+00
DL	0.00E+00	1.67E-04	0.00E+00	2.60E-04	1.55E-04	3.72E-05	0.00E+00

For the north part of urban area, NZG and ATZX have the same distribution of cancer risks, different from HD. Of the total cancer risk in HD, 55% is road dust besed, followed by metal processing, fossil fuel combustion, vehicle exhaust, biomass burning, soil dust, and secondary sulphur with corresponding percentages of 15%, 9%, 9%, 6%, 4%, and 2%. Cancer risks in NZG and ATZX are in order of road dust > fossil fuel combustion > vehicle exhaust > soil dust > secondary sulphur > metal processing > biomass burning with 42%, 24%, 16%, 5%, 5%, 4%, and 3%, respectively.

Cancer risk of road dust in HR is more than two times of corresponding cancer risk in SY. Road dust contributes 59% of total cancer risk in SY and percentage of road dust is 4% higher in HR. Metal processing contributes 17% of total cancer in SY, percentage of metal processing is also 4% higher in HR. Besides road dust and metal processing, Percentages of vehicle exhaust, biomass burning, fossil fuel combustion, and secondary sulphur are all lower than 10%. Cancer risks of soil dust are zero in all suburban stations.

There are only four sources in the west part of suburban area, road dust, vehicle exhaust, biomass burning, and fossil fuel combustion. The corresponding percentages are 42%, 27%, 25%, and 6% in both CP and DL stations. Cancer risks of secondary sulphur, soil dust, and metal processing are zero in CP and DL.

Non-cancer hazard quotients

Generally, non-cancer hazard quotients in all stations are all higher than EPA upper limit (1.0), ranging from 1.0 to 15.5. The highest cancer risk is in NZG station, while the lowest one is in DL as shown in Table 4-6. Cancer risk in NZG is 15.5 times in DL. Non-cancer hazard quotients in suburban area are higher than in urban area. In suburban area, cancer risks in east part (HR and SY) are 3 times higher than in west part (DL and CP).

Table 4-6: Distributions of non-cancer hazard quotients in twelve government stations by elements.

Elements	WS	DS	GY	TT	GC	HD	NZG	ATZX	SY	HR	CP	DL
Al		0.26					0.04	0.04		0.13	0.07	0.07
Ba										0.02		
Cr		0.04				0.07	0.01	0.01	0.18	0.44		
Mn		0.27					0.19	0.18	0.14	0.15	0.20	0.19
Ni		0.49				0.03	0.02	0.02	0.03	0.12		
As	3.29	1.47	3.23	3.24	3.28	0.47	0.91	0.86	1.02	0.74		
Cd						0.16	0.08	0.07	0.22			
Pb	0.93	0.76	0.92	0.92	0.93	0.58	0.57	0.54	1.29	0.52	0.79	0.74
Co		0.15				0.01	0.01	0.01				
V		0.18					0.02	0.02				
P										0.95		
Cl	9.00		8.84	8.85	8.96		13.63	12.82				
Se												
Sum	13.23	3.63	12.99	13.00	13.17	1.30	15.49	14.57	2.88	3.07	1.07	1.00

In WS, GY, TT, and GC, only four non-carcinogenic elements (As, Pb, Cl, and Se) were detected. Of total non-cancer hazard quotients, 68% is from Cl. Cl is followed by As, Pb, and Se with corresponding percentages of 25%, 7%, and 1%. There are eight non-carcinogenic elements in DS station, of which As is the main contributor accounting for 40% of total non-cancer hazard quotient. Cr is thcontributor, it only accounts for 1% of total non-cancer hazard quotient. Among eight non-carcinogenic elements in DS, Cr is the only element that has non-cancer hazard quotient lower than one.

The non-cancer hazard quotient in HD is the lowest among 8 urban area stations. Cl, which is usually the major contributor, was not detected in HD. Among six non-carcinogenic elements in HD, non-cancer hazard quotient of Pb is the highest while Co is the lowest one. Although Pb is the highest, the non-cancer hazard quotient of it is only 0.6. Compare to the non-cancer hazard quotients of Cl, Pb is less than 1/15 of Cl.

Non-cancer hazard quotient of NZG and ATZX are the top two among all the stations, NZG is 0.9 higher than ATZX. NZG and ATZX have nine non-carcinogenic elements, more than any other stations. Non-cancer hazard quotients of Cl are extremely high in these two stations, 13.6 in NZG and 12.8 in ATZX. Compare to WS, which has the highest Cl non-cancer hazard quotient among the remaining 10 stations, non-cancer hazard quotients of Cl in NZG and ATZX are 4.6 and 3.8 higher than in WS.

SY and HR locate in the east part of suburban area. Compare to DL and CP in the west part of suburban area, more non-carcinogenic elements were detected in SY and HR. Six non-carcinogenic elements in SY and eight in HR, while only three in DL and CP. Pb is the main source in SY contributing approximate 45% of total non-cancer hazard quotient and P is the main source in HR contributing approximate 31% of total non-cancer hazard quotient. Similar to SY, Pb is the primary contributor in both CP and DL. However, percentages of Pb in CP and DL are higher than Pb in SY with approximate 74% of total non-cancer hazard quotient.

Among eight urban area stations, fossil fuel combustion is the primary source in WS, GY, TT, GC, NZG and ATZX, while road dust is the primary source in DS and HD as shown

in Table 4-7. Road dust is also the primary source in four suburban stations. The non-cancer hazard quotient of fossil fuel combustion in DS, HD, SY, HR, CP, and CL are lower than one. Besides the stations mentioned above, the non-cancer hazard quotients of fossil fuel combustion in the remaining stations are all higher than one.

Table 4-7: Distributions of non-cancer hazard quotients in twelve governmental sampling stations by sources.

	Secondary sulphur	Vehicle exhaust	Soil dust	Road dust	Biomass burning	Fossil fuel combustion	Metal Processing
WS	0.5	1.5	0.0	1.7	0.5	8.3	0.7
DS	0.1	0.6	0.3	1.1	0.4	0.8	0.3
GY	0.5	1.5	0.0	1.6	0.5	8.2	0.7
TT	0.5	1.5	0.0	1.6	0.5	8.2	0.7
GC	0.5	1.5	0.0	1.7	0.5	8.3	0.7
HD	0.0	0.2	0.0	0.5	0.2	0.2	0.0
NZG	0.5	1.4	0.0	0.6	0.6	11.1	1.2
ATZX	0.5	1.3	0.0	0.6	0.5	10.5	1.1
SY	0.1	0.6	0.0	1.1	0.3	0.4	0.1
HR	0.3	0.5	0.3	0.9	0.2	0.7	0.2
CP	0.0	0.3	0.0	0.4	0.2	0.0	0.1
DL	0.0	0.3	0.0	0.3	0.2	0.0	0.1

The non-cancer hazard quotients distributed by different systems are listed in Table 4-8. The most vulnerable systems in WS, GY, TT, GC, NZG, and ATZX are respiratory system with non-cancer hazard quotients ranging from 1.5 to 13.7. In HD, SY, CP, and DL, the most vulnerable system is nervous system with non-cancer hazard quotient ranging from 0.6 to 1.4. The hazard quotients of alimentary system are zero and the hazard quotients of fetus are zero except for HR, the hazard quotient of fetus in HR is 0.02. Either alimentary system or fetus is the least affected systems among six systems.

Table 4-8: Distributions of non-cancer hazard quotients in twelve governmental sampling stations by target organs.

	Nervous	Fetus	Respiratory	Alimentary	Reproductive	Renal
WS	0.93	0.00	9.00	0.00	3.29	0.00
DS	1.30	0.00	0.86	0.00	1.47	0.00
GY	0.92	0.00	8.84	0.00	3.23	0.00
TT	0.92	0.00	8.85	0.00	3.24	0.00
GC	0.93	0.00	8.96	0.00	3.28	0.00
HD	0.58	0.00	0.10	0.00	0.47	0.16
NZG	0.81	0.00	13.69	0.00	0.91	0.08
ATZX	0.76	0.00	12.88	0.00	0.86	0.07
SY	1.44	0.00	0.21	0.00	1.02	0.22
HR	0.79	0.02	1.52	0.00	0.74	0.00
CP	1.07	0.00	0.00	0.00	0.00	0.00
DL	1.00	0.00	0.00	0.00	0.00	0.00

4.3 Uncertainty analysis

Beijing

The three-year average $PM_{2.5}$ mass concentration and 95% intervals was 73.7 (±0.298) ug/m^3. The corresponding uncertainty calculated equals (0.298/73.7)*100%=0.4%. For cancer risks, the overall uncertainty summed by the three categories introduced before equals to 20%+30%+0.4%=50.4%. Lifetime cancer risk in Beijing is 2.30E-02, giving the uncertainty range of [3.46E-02, 1.14E-02]. However, the upper range and lower range of lifetime cancer risks are both higher than EPA upper threshold of cancer risks (1.00E-04), locating in the same category that some remediation are desirable. Thus, the uncertainty of cancer risks will not lead to the change of cancer risk category.

For non-cancer hazard quotient, the overall uncertainty equals to 20%+0.4%=20.4%. Hazard quotient in Beijing is 13.7, causing the uncertainty range of [16.5, 10.9]. The upper range and lower range of hazard quotient are higher than EPA upper limit of non-cancer hazard quotient (1.0), indicating that some non-cancer effects may occur. Hence, the uncertainty of non-cancer hazard quotient will not cause the change of non-cancer hazard quotient category.

Windsor

The cancer risk in Windsor is 8.40E-04. The uncertainty range in Windsor can be estimated from [8.40E-04*(1+50.4%), 8.40E-04*(1-50.4%)]=[1.26E-03, 3.99E-04]. The higher and lower ranges of lifetime cancer risks in Windsor are all higher than EPA upper limit of cancer risks (1.00E-04). Thus, the uncertainty of cancer risks will not lead to the change of cancer risk category.

Non-cancer hazard quotient in Windsor is 0.14. The uncertainty range was estimated by [0.14*(1+50.4%), 0.14*(1-50.4%)]=[0.21, 0.07]. The upper and lower range of hazard quotient are still lower than EPA upper limit of non-cancer hazard quotient of 1.0. Therefore, the uncertainty of non-cancer hazard quotient will not cause the change of category for non-cancer hazard quotient in Windsor.

4.4 Windsor, Canada

4.4.1 Three-year average

Cancer risks

Three-year average $PM_{2.5}$ mass concentration is 8.9 μg/m^3 in Windsor, lower than CNAAQS, NAAQS, and WHOAQG standards. There are two overall lifetime cancer risks estimated for Windsor, one used PAH concentrations from 2003 to 2007 (cancer risk 03-07) and another one used PAH concentrations from 2010 to 2012 (cancer risk 10-12). Overall lifetime cancer risks and lifetime cancer risks for elements and components are listed in Table 4-9.

Table 4-9: Distributions of lifetime cancer risks in Windsor.

Components	Cancer risks	Components	Cancer risks
Cr	4.20E-04	Cr	4.20E-04
Co	1.40E-05	Co	1.40E-05
Ni	8.93E-06	Ni	8.93E-06
As	3.25E-04	As	3.25E-04
Cd	1.66E-05	Cd	1.66E-05
Pb	1.78E-05	Pb	1.78E-05
PAHs 03-07	3.65E-05	PAHs 10-12	3.82E-05
Total	8.38E-04	Total	8.40E-04

Total lifetime cancer risks in Windsor are 8.38E-04 and 8.40E-04, all higher than EPA upper limit (1.00E-04). Total lifetime cancer risk 03-07 is 2.00E-06 lower than cancer risk 10-12, because PAHs cancer risk from 2003 to 2008 is 1.70E-06 lower than PAHs cancer risk from 2010 to 2012. Percentages of elements and PAHs are the same in both cancer risk 03-07 and cancer risk 10-12 groups. Thus, in this study, the higher cancer risk will be used to compare with Beijing.

Contributions of elements and PAHs in Windsor are in order of Cr > As > PAHs > Pb > Cd > Co > Ni as shown in Figure 4-26. Cr and As are the major contributors with corresponding percentages of 50% and 39%, followed by PAHs with contribution of 4%. Ni is the least contributor among all contributors, with only 1%.

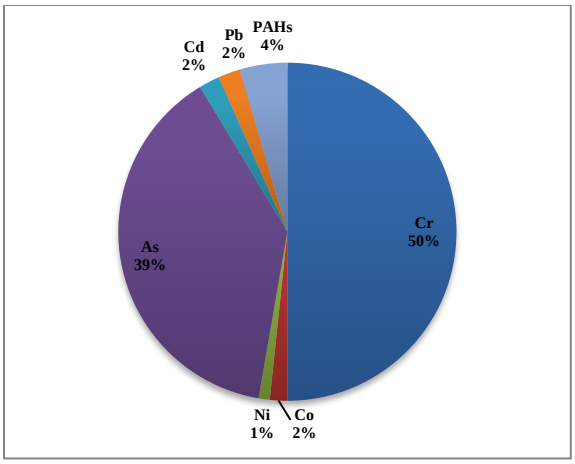


Figure 4-26: Contribution of lifetime cancer risks in Windsor.

Non-cancer hazard quotients

Total lifetime non-cancer hazard quotient in Windsor is 0.14 as shown in Table 4-9. Total lifetime non-cancer hazard quotient in Windsor is lower than EPA upper limit (1.0). Except for P and Cl, eleven non-carcinogenic elements were detected in Windsor as shown in Table 4-9. Although Co and Se were detected in Windsor, the non-cancer hazard quotients were too small, shown as 0.000 in Table 4-9.

Table 4-10: Contribution of lifetime non-cancer hazard quotients in Windsor.

Elements	HQ	Elements	HQ
Al	0.004	Pb	0.014
Ba	0.004	Co	0.000
Cr	0.005	V	0.006
Mn	0.013	P	NA
Ni	0.006	Cl	NA
As	0.072	Se	0.000
Cd	0.013	Total	0.14

Distribution of non-cancer hazard quotients in Windsor is shown in Figure 4-27. In Windsor, non-cancer hazard quotients of non-carcinogenic elements are in order of As > Pb > Mn > Cd > V > Ni> Cr > Ba > Al > Co > Se. As is the largest contributor in Windsor with approximate 50%.

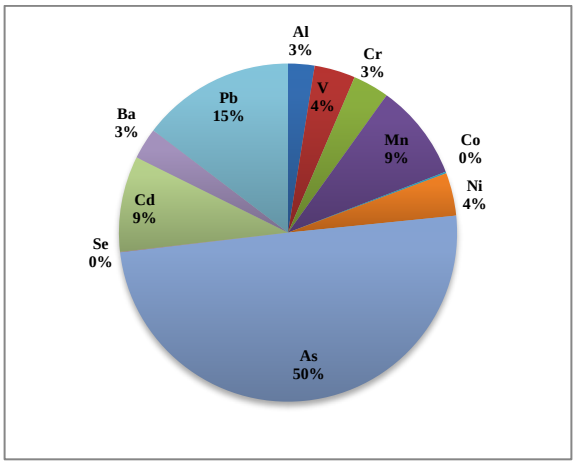


Figure 4-27: Distributions of lifetime non-cancer hazard quotients in Windsor.

4.3.2 Annual average

Cancer risks

In Windsor, $PM_{2.5}$ mass concentrations were 9.3 μg/m^3, 8.5 μg/m^3, and 9.0 μg/m^3 in 2013, 2014, and 2015, respectively. $PM_{2.5}$ mass concentrations were increasing from 2013 to 2015. Cancer risks were 7.76E-04, 9.03E-04, and 8.09E-04 in 2013, 2014, and 2015, respectively. The trend of cancer risks in Windsor is different from $PM_{2.5}$ mass concentration. Cancer risk of Windsor is the highest in 2014, followed by 2015 and 2013. Cancer risks of Windsor are all higher than EPA upper limit (1.00E-04) although the $PM_{2.5}$ mass concentrations are within CNAAQS, NAAQS standards of 10 μg/m^3.

The contribution orders in 2013 and 2014 are the same, Cr > PAHs > As > Pb > Cd > Co >Ni as shown in Table 4-11. The contribution order is Cr > PAHs > As > Pb > Cd > Ni >Co in 2015, different from 2013 and 2014. The contribution orders are overall similar. Cr and As are the markers of road dust and fossil fuel combustion.

Table 4-11: Cancer risks of $PM_{2.5}$ in 2013, 2014, and 2015.

Year	2013	2014	2015
Cr	3.61E-04	4.70E-04	4.14E-04
Co	1.56E-05	1.34E-05	1.29E-05
Ni	8.81E-06	9.98E-06	7.41E-06
As	3.12E-04	3.40E-04	3.16E-04
Cd	1.82E-05	1.53E-05	1.68E-05
Pb	2.11E-05	1.69E-05	1.49E-05
PAHs	3.56E-04	3.79E-04	3.56E-04
Total	7.76E-04	9.03E-04	8.09E-04

Non-cancer hazard quotients

Total non-cancer hazard quotients in Windsor are 0.15, 0.14, and 0.14 in 2013, 2014, and 2015, lower than EPA upper limit (1.0) as depicted in Table 4-12. Non-cancer hazard quotients in these three years are close. In Windsor, the orders of non-cancer hazard quotients from 2013 to 2015 are different from the order of $PM_{2.5}$ mass concentration and the order of cancer risks. Eleven non-carcinogenic elements were detected in Windsor in 2013, 2014, and 2015, P and Cl were not detected.

Table 4-12: Non-cancer hazard quotients of $PM_{2.5}$ in 2013, 2014, and 2015.

	2013	2014	2015
Al	4.03E-03	3.38E-03	3.59E-03
V	6.13E-03	5.52E-03	5.20E-03
Cr	4.30E-03	5.59E-03	4.93E-03
Mn	1.47E-02	1.17E-02	1.40E-02
Co	2.47E-04	2.13E-04	2.05E-04
Ni	5.83E-03	6.60E-03	4.90E-03
As	6.88E-02	7.50E-02	6.97E-02
Se	5.62E-05	4.81E-05	7.63E-05
Cd	1.45E-02	1.21E-02	1.33E-02
Ba	4.46E-03	4.07E-03	4.47E-03
Pb	2.51E-02	2.01E-02	1.77E-02
HQ	0.15	0.14	0.14

As is the primary contributor of non-cancer hazard quotient in both Windsor from 2013 to 2015 as shown in Table 4-12. Percentage of As peaks in 2014 with 54%, followed by

2015 and 2013 with 50% and 46%, respectively. Pb is the second largest contributor among eleven non-carcinogenic elements in Windsor from 2013 to 2015.

4.4.2 Seasonal average

Cancer risks

$PM_{2.5}$ mass concentrations in Windsor are 8.5 μg/m^3, 10.4 μg/m^3, 7.8 μg/m^3, and 9.3 μg/m^3 in spring, summer, autumn, and winter, respectively. $PM_{2.5}$ mass concentration peaks in winter, followed by summer, autumn, and spring. Seasonal $PM_{2.5}$ mass concentrations are all lower than CNAAQS grade-I standard. Cancer risks in four seasons are 1.94E-04, 2.47E-04, 2.35E-04, 1.61E-04 from spring to winter, respectively as shown in Table 4-13. Seasonal cancer risks are in order of summer > autumn > spring > winter, different from order of $PM_{2.5}$ mass concentration. Although $PM_{2.5}$ mass concentration in summer is the highest among four seasons, cancer risk of autumn is the lowest. In Windsor, overall lifetime cancer risk calculated by four seasons is 2.09E-03, higher than EPA upper limit (1.00E-04).

Table 4-13: Seasonal cancer risks in Windsor.

	Spring	Summer	Autumn	Winter
Cr	9.19E-05	1.23E-04	1.10E-04	9.46E-05
Co	3.80E-06	4.22E-06	3.11E-06	3.05E-06
Ni	2.36E-06	2.95E-06	2.14E-06	1.58E-06
As	8.05E-05	1.01E-04	9.78E-05	4.38E-05
Cd	3.62E-06	3.86E-06	5.03E-06	3.86E-06
Pb	4.45E-06	4.46E-06	4.97E-06	3.78E-06
PAHs	7.01E-06	8.26E-06	1.19E-05	1.07E-05
Total	1.94E-04	2.47E-04	2.35E-04	1.61E-04

In Windsor, Cr is the biggest contributor in all four seasons, ranged from 47% to 59%, as shown in Table 4-14. As is the second-largest contributor in all four seasons ranged from 27% to 42%. Clear seasonal variations could be observed for top two contributors in Windsor, Cr and As. Cr has the largest contribution in winter while As has the least contribution in Winter.

Top two contributors of cancer risks are the same in Windsor, Cr and As. The sum of these two contributors accounts for more than 80% of total cancer. Besides the top two contributors, orders of the remaining elements and PAHs are different in four seasons in Windsor. The contribution differences between remaining elements and PAHs are small because percentages of remaining elements are ranging from 1% to 2%.

Overall, the contribution orders of components are similar in spring, summer, autumn, but different in winter. In winter, the Cr contribution raised to 59% and PAHs contribution raised to 7%. As contribution fall down to 27%, approximate 15% less than in spring, summer, and autumn.

Table 4-14: Seasonal distributions of cancer risks in Windsor.

Components	Spring	Summer	Autumn	Winter
Cr	47%	50%	47%	59%
Co	2%	2%	1%	2%
Ni	1%	1%	1%	1%
As	42%	41%	42%	27%
Cd	2%	2%	2%	2%
Pb	2%	2%	2%	2%
PAHs	4%	3%	5%	7%

Non-cancer hazard quotients

Seasonal non-cancer hazard quotients in Windsor are 0.14, 0.17, 0.17, and 0.10 from spring to winter. Total lifetime non-cancer hazard quotient from four different seasons is 0.15 in Windsor, lower than EPA upper limit of non-cancer hazard quotient (1.0). Non-cancer hazard quotients in four seasons are in order of autumn = summer > spring > winter in Windsor. Non-cancer hazard quotients of Co and Se are lower than 0.01 in four seasons in Windsor, thus shown as 0.00 in Table 4-15.

Table 4-15: Seasonal non-cancer hazard quotients of Windsor.

Seasons	Spring	Summer	Autumn	Winter
Al	4.20E-03	4.72E-03	3.30E-03	2.59E-03
V	5.39E-03	7.46E-03	6.15E-03	3.59E-03
Cr	4.38E-03	5.86E-03	5.23E-03	4.50E-03
Mn	1.29E-02	1.30E-02	1.48E-02	1.18E-02
Co	2.41E-04	2.68E-04	1.98E-04	1.94E-04
Ni	6.25E-03	7.81E-03	5.66E-03	4.18E-03
As	7.11E-02	8.88E-02	8.64E-02	3.86E-02
Se	5.37E-05	5.61E-05	6.89E-05	5.10E-05
Cd	1.15E-02	1.23E-02	1.60E-02	1.23E-02
Ba	4.13E-03	5.43E-03	4.84E-03	2.79E-03
Pb	2.12E-02	2.12E-02	2.37E-02	1.80E-02
Sum	0.14	0.17	0.17	0.10

As is the primary contributor in four seasons and as shown in Table 4-16. Percentages of As are ranging from 39% to 52% in four seasons in Windsor, peaks in summer and becomes the lowest in winter. The second-largest contributor in Windsor is Pb in all four seasons.

Table 4-16: Seasonal distributions of non-cancer hazard quotients in Windsor.

Seasons	Spring	Summer	Autumn	Winter
Al	3%	3%	2%	3%
V	4%	4%	4%	4%
Cr	3%	3%	3%	5%
Mn	9%	8%	9%	12%
Co	0%	0%	0%	0%
Ni	4%	5%	3%	4%
As	51%	52%	51%	39%
Se	0%	0%	0%	0%
Cd	8%	7%	9%	12%
Ba	3%	3%	3%	3%
Pb	15%	12%	14%	18%

Major contributors in Windsor are As, Pb, Mn, and Cd in all four seasons. These four contributors together account for more than 70% of the total non-cancer hazard quotient

in all four seasons. The contributions for the remaining elements are relatively small, contributing up to 4% of the total hazard quotient.

4.5 Comparison between Beijing, Windsor, and previous studies

Most of previous studies were taken place in China, only one was found outside China in Mexico as shown in Table 2-3. Seven out of fourteen previous studies estimated $PM_{2.5}$-bound 16 EPA priority PAHs only. PAHs were associated with cancer risks, thus non-cancer hazard quotients were not estimated in these studies. Elemental components estimated were different in the remaining seven studies.

PAHs

Cancer risks caused by $PM_{2.5}$-bound PAHs from previous studies are in the order of Beijing (4.48E-04) > Wuhan (8.06E-05) >Shanghai (4.56E-05) > Huzhou (8.70E-06) > Jinan (6.80E-06) > Hefei (3.86E-06) > Xiangtan (1.76E-06). Beijing is the only city that has cancer risks caused by $PM_{2.5}$-bound PAHs higher than EPA upper limit of cancer risks (1.00E-04). Beijing was observed the highest $PM_{2.5}$-bound PAHs cancer risk (4.48E-04), two orders of magnitude higher than EPA upper threshold (1.00E-04). Besides, the cancer risks caused by $PM_{2.5}$-bound PAHs in Wuhan, Shanghai, Windsor, Huzhou, Jinan, Heifei, and Xiangtan are all within the acceptable threshold [1.00E-06, 1.00E-04] of USEPA.

Lifetime cancer risk caused by $PM_{2.5}$-bound PAHs estimated in this study is 9.37E-04 from 2013 to 2015. Compare to previous study in Beijing (Wang et al., 2008), cancer risk of $PM_{2.5}$-bound PAHs in this study is 9.37E-04, approximate 2.1 times higher than in Beijing (Wang et al., 2008). The $PM_{2.5}$-bound PAHs in Wuhan is the second largest, however the $PM_{2.5}$-bound PAHs cancer risk of Beijing (9.37E-04) in this study higher than in Wuhan. Besides, the $PM_{2.5}$-bound PAHs cancer risk of Beijing (9.37E-04) in this study is two orders of magnitude higher in Huzhou, Wuhan, Jinan, and Xiangtan. Among all the cities mentioned above, Beijing is the only city that provides winter heating. Winter heating produces lots of emission. Meanwhile, the cold and dry weather during

winter in Beijing is not conducive to the flow of atmosphere. Thus, the winter heating partially explains the higher $PM_{2.5}$-bound PAHs cancer risk in Beijing (Wang et al., 2008).

Cancer risks

Cancer risks caused by elemental components in different cities are in the order of The Pearl River Delta Region (the PRDR) (2.24E-04) > Nanjing (8.01E-05) > Guangzhou (1.01E-05) > Changsha (8.44E-07) > Xiamen (9.49E-08). The cancer risks caused by elemental components in the PRDR is the only one that higher than EPA upper threshold of cancer risk (1.00E-04). The cancer risks caused by elemental components in Nanjing and Guangzhou are within acceptable threshold of EPA cancer risk [1.00E-06, 1.00E-04]. Besides, the cancer risks caused by elemental components in Changsha and Xiamen are negligible (<1.00E-06).

Cancer risk caused by elemental components in Beijing is 2.27E-02. Compare to the corresponding cancer risks mentioned above, the cancer risk in Beijing is at least two magnitudes higher. The number of carcinogenic elements detected is also higher in Beijing than in other cities mentioned above, as shown in Table 4-17. Elemental components cancer risk estimated in Windsor was 5.40E-04, also higher than other cities in China except for Beijing. There was six carcinogenic elements estimated in this study, Cr, Ni, As, Cd, Pb, and Co. Except for Nanjing, which has the same carcinogenic elements as in this study, the other cities used 3 to 4 carcinogenic elements only. For example, there were only three carcinogenic elements (Cd, Ni, and Cd) detected in Xiamen.

Table 4-17: Contributions of elemental components' cancer risks in different cities.

	The PRDR	Beijing (same element)	Xiamen	Beijing (same element)	Changsha	Beijing (same element)	Nanjing	Beijing (same element)	Guangzhou	Beijing (same element)	Beijing	Windsor
Study	Zhang et al., 2016	This study	Zhuang et al., 2016	This study	Yang et al., 2014	This study	Hu et al., 2012	This study	Li et al., 2016	This study	This study	This study
Pb	1.00E-06	7.03E-04			2.67E-10	7.03E-04	3.25E-06	7.03E-04			7.03E-04	1.78E-05
Ni	2.70E-07	1.93E-04	1.11E-09	1.93E-04			7.10E-07	1.93E-04	8.70E-08	1.93E-04	1.93E-04	8.93E-06
Cr	2.00E-04	1.19E-02	7.76E-08	1.19E-02			4.48E-05	1.19E-02	3.41E-06	1.19E-02	1.19E-02	4.20E-04
Cd	1.17E-06	2.18E-04	1.61E-08	2.18E-04	9.41E-08	2.18E-04	1.16E-06	2.18E-04	3.51E-08	2.18E-04	2.18E-04	1.66E-05
As	2.15E-05	6.37E-03			7.50E-07	6.37E-03	9.16E-06	6.37E-03	6.94E-06	6.37E-03	6.37E-03	3.25E-04
Co							2.10E-05	3.25E-03			3.25E-03	1.40E-05
Total	2.24E-04	1.94E-02	9.49E-08	1.23E-02	8.44E-07	7.29E-03	8.01E-05	2.26E-02	1.05E-05	1.87E-02	2.26E-02	8.40E-04

Cr is the major contributor for cancer risk in the PRDR, Xiamen, Nanjing, Windsor, and Beijing. The cancer risks caused by Cr ranged 1.19E-02 to 7.76E-08, accounting for from 89.5% to 52.7% of total cancer risks in the PRDR, Xiamen, Nanjing, and Beijing. In Guangzhou and Changsha, As is the major contributor with 6.94E-06 and 7.50E-07 cancer risks. The percentages of As in Guangzhou and Changsha are 66.1% and 88.9%, respectively. In China, $PM_{2.5}$ bound As is mainly from coal, fuel, and industrial activities (Jiang et al., 2015; Tan & Duan, 2013). Higher As concentrations were detected by the sample machines locate in industrial areas of Changsha and Guangzhou (Yang et al., 2014; Li et al., 2016). This is the reason why As is the major contributor in Changsha and Guangzhou.

To keep the consistency of carcinogenic elements in different study, a same element comparison was conducted. Same element comparison was defined as "using the cancer risk or non-cancer hazard quotient of same elements in both cities to conduct comparison." Cancer risks of the same element used in previous study and Beijing were summed, results are depicted in Table 4-17. The differences between Beijing and other cities did not change much. Beijing is still more than two orders of magnitude higher than all other cities. That is to say, the $PM_{2.5}$ cancer risk in Beijing is really high.

Non-cancer hazard quotient

Non-cancer hazard quotients were estimated in Mexico (0.96), Nanjing (2.96), Jinan (0.86), Xiamen (7.74E-05), Guangzhou (1.97E-08), the PRDR (1.43) (Table 4-18). Non-cancer hazard quotients in Guangzhou, Xiamen, Mexico, and Jinan are lower than EPA upper level of non-cancer hazard quotients (<1.00), there is no appreciable or adverse risks. However, non-cancer hazard quotients in Nanjing, and the PRDR are higher than EPA upper level of non-cancer hazard quotients (>1.00), there is a possibility that some non-cancer effects may occur.

The non-cancer hazard quotient in Beijing is 13.7, higher than all other cities in China and Mexico. Non-cancer hazard quotient in Windsor is 0.15, higher than Xiamen and Guangzhou and lower than the remaining cities in China. Major contributors of non-

cancer hazard quotients in different places are different. For example, Pb is the major contributor in Mexico (70.8%) and in Guangzhou (54.5%), while Co is the major contributor in Nanjing (38.1%).

Table 4-18: Contributions of elemental components' non-cancer hazard quotients in different cities.

	Mexico	Beijing (same element)	Nanjing	Beijing (same element)	Jinan	Beijing (same element)	Xiamen	Beijing (same element)	Guangzhou	Beijing (same element)
Study	R.V. Diaz et al., 2009	This study	Hu et al., 2012	This study	Yang et al., 2013	This study	Zhuang, 2016	This study	Li et al., 2016	This study
Al										
Ba										
Cr	0.02	0.14	0.11	0.14	0.13	0.14	6.46E-05	0.14		
Mn	0.04	0.19	0.95	0.19	0.37	0.19	5.18E-06	0.19	8.95E-09	0.19
Ni	3.66E-05	0.12	0.17	0.12	7.04E-05	0.12	6.43E-08	0.12		
As			0.41	1.37	6.51E-03	1.37				
Cd			0.19	0.17	1.19E-03	0.17	2.52E-06	0.17		
Pb	0.68	0.82			1.06E-02	0.82	5.06E-06	0.82	1.07E-08	0.82
Co			1.13	0.05	0.35	0.05				
V	7.84E-04	0.09			1.04E-04	0.09				
P										
Cl	0.22	9.81								
Se										
Total	0.96	11.2	3.0		0.9	2.95	7.74E-05	1.4	1.97E-08	1.0

Continued from Table 4-18.

	The PRDR	Beijing (same element)	Beijing	Windsor
Study	Zhang et al., 2016	This study	This study	This study
Al			0.13	0.004
Ba			0.02	0.004
Cr	0.08	0.14	0.14	0.004
Mn			0.19	0.013
Ni	0.24	0.12	0.12	0.006
As	0.93	1.37	1.37	0.003
Cd	0.18	0.17	0.17	0.013
Pb			0.82	0.002
Co			0.05	0.000
V			0.09	0.006
P			0.82	
Cl			9.81	
Se			0.00	0.000
Total	1.4	1.8	13.7	0.15

Overall, cancer risks caused by $PM_{2.5}$-bound PAHs and elemental components, and non-cancer hazard quotients in Beijing are all higher than corresponding cancer risks and non-cancer hazard quotients in other cities in China and in Mexico. The higher health risks in Beijing should be noticed and the emission control strategies should be taken.

Same element comparison was also conducted for non-cancer hazard quotients in Beijing and previous studies. As shown in Table 4-18, the differences between Beijing and other cities decreased a lot. The non-cancer hazard quotient in Nanjing and Beijing are 3.0 and 2.0, respectively. Nanjing is the only city has higher non-cancer hazard quotient than Beijing for same element comparison. The non-cancer hazard quotients of Co and Mn in Nanjing are higher than in Beijing.

CHAPTER 5
CONCLUSIONS AND RECOMMENDATIONS

5.1 Conclusions

The lifetime health risks due to exposure to ambient $PM_{2.5}$ bound elements and polycyclic aromatic hydrocarbons (PAHs) from 2013 to 2015 in Beijing were estimated. The conclusions about cancer risks are:

Beijing:

(1) The lifetime cancer risk is 2.30E-02, two orders of magnitude higher than EPA upper threshold (1.00E-04). Elemental components and PAHs contributions of cancer risk are in the order of Cr (50%) > As (27%) > Co (14%) > total PAHs (4%) > Pb (3%) > Cd (1%) and Ni (1%). Road dust is the primary source with 46% contribution among seven sources, followed by soil dust (14%), metal processing (12%), fossil fuel combustion (11%), vehicle exhaust (11%), biomass burning (4%), and secondary sulphur (2%).

(2) In 2013, 2014, and 2015, lifetime cancer risks are 2.63E-02, 2.16E-02, and 2.14E-02. The cancer risk contribution order of elemental components and PAHs in 2013, 2014, and 2015 are the same as in three-year average.

(3) Seasonal cancer risks are 5.37E-03, 7.47E-03, 4.78E-03, and 8.93E-03 in spring, summer, autumn, and winter. Percentages of seasonal cancer risks were higher than its share (25%) in summer (31%) and winter (26%), meanwhile lower than its share in spring (23%) and autumn (20%). The orders of components are different in all four seasons. Cr is the major contributor in spring (50%), summer (75%), and autumn (44%), while As is the major contributor in winter (39%). As is the marker of coal burning, winter heating of coal burning caused high As emission during winter in Beijing (Wang et al., 2008). Road dust is the major contributor (44%-58%) of cancer risk in all four seasons among seven sources.

(4) Hours of day cancer risks ranged from 9.11E-04 to 1.10E-03, higher in night time (from 19:00 to 3:00) than in day time (from 4:00 to 18:00).

(5) The cancer risk is higher in suburban area than in urban area. Across Beijing, the cancer risk in urban area (8 stations) is 1.82E-02, while 3.53E-02 in suburban area

(4 stations). Both cancer risks are higher than EPA upper threshold (1.00E-04). Major contributors are As (39%), Cr (36%), and Co (19%) in urban area and Cr (90%) and As (8%) in suburban area.

(6) The highest cancer risks in ring group of Beijing was observed in R>5 group (3.53E-02), followed by R<3 (2.34E-02) and R3-5 (1.48E-02) by using different elemental components profiles. Co is major contributor in R<3, while Cr is the major contributor in R3-5 and R>5.

(7) The cancer risks in 12 sampling sites ranged from 6.19E-04 to 4.12E-02. The highest cancer risk was observed in Huairou station, while the lowest cancer risk was from Dingling. Contributions of components vary largely for different stations due to different elemental components profiles used in 12 stations. As is the major contributor in 6 out of 12 sampling stations.

(8) Road dust is the major source among seven sources of cancer risks in all temporal studies in Beijing. For spatial studies in Beijing, road dust is the major source of cancer risks in almost all spatial studies except for in R<3 group and in Dongsi station, the major source of which is soil dust.

Windsor:

(1) The lifetime cancer risk in Windsor is 8.40E-04. The cancer risk in Windsor is higher than EPA upper threshold of cancer risk. Component concentrations are in order of Cr (50%) > As (39%) > PAHs (4%) > Pb (2%) > Cd (2%) > Co (2%) > Ni (1%).

(2) Cancer risks are 7.76E-04, 9.03E-04, and 8.09E-04 from 2013 to 2015, higher than EPA upper limit. Major contributors are Cr and As from 2013 to 2015.

(3) Seasonal cancer risks in Windsor ranged from 2.47E-04 to 1.61E-04. Percentages of total cancer risks in summer (30%) and autumn (28%) are higher than its share (25%), while lower in spring (23%) and winter (19%). Major contributor is Cr in all four seasons.

(4) Cancer risks in Beijing and in Windsor are higher than other cities in China and in Mexico (Table 2-3). Cancer risk caused by $PM_{2.5}$-bound PAHs in Beijing from this study is 9.37E-04, higher than all the cities in previous studies. Cancer risk caused by $PM_{2.5}$-bound PAHs in Windsor is 2.79E-05, lower than previous studies

conducted in Beijing and Wuhan, higher than the remaining cities in previous studies.

Conclusions about non-cancer hazard quotients are:

Beijing:

(1) Non-cancer hazard quotient estimated by three-year average $PM_{2.5}$ mass concentration was 13.7, higher than EPA upper limit of non-cancer hazard quotient. Major contributors of non-cancer hazard quotients are Cl (72%), As (10%), Pb (6%), and P (6%). Fossil fuel combustion is the major source among seven sources of non-cancer hazard quotients, accounting for 64% of non-cancer hazard quotient. Respiratory system impairment is the most vulnerable system impairment among six affected system impairments, accounting for 81% of non-cancer hazard quotient.

(2) Non-cancer hazard quotients estimated by annual average concentrations are 15.7, 12.9, 12.8 in 2013, 2014, and 2015 respectively, higher than EPA upper limit of non-cancer hazard quotient. Major contributor, source, and most vulnerable system are the same as in three-year average.

(3) Seasonal non-cancer hazard quotients range from 4.5 to 26.2. Winter accounts for 52% of total non-cancer hazard quotient, higher than its share (25%). Percentages of non-cancer hazard quotients in spring (21%), autumn (18%), and summer (9%) are lower than its share. Major contributor of non-cancer hazard quotient is Cl in all four seasons (33%-87%). Fossil fuel combustion (43%-69%) is the major source among seven sources of non-cancer hazard quotients in all four seasons.

(4) Hours of day non-cancer hazard quotients vary from 13.0 to 15.7. Non-cancer hazard quotients were higher in night time than in day time, due to the corresponding higher $PM_{2.5}$ mass concentrations in night time than in day time. Major contributor and source are the same as three-year average.

(5) Non-cancer hazard quotient is 14.3 in urban area and 2.6 in suburban area, higher than EPA upper level. Major contributors are Cl (77%), As (11%), and Pb (6%) in urban area, while P (31%), As (24%), Pb (17%), Cr (14%) in suburban area. Fossil fuel combustion (65%) is the major source among seven sources of non-

cancer hazard quotient in urban area, while road dust (29%) is the major source in suburban area.

(6) Non-cancer hazard quotients in ring groups are in the order of R3-5 (14.7), R<3 (13.3), R>5 (2.6). Non-cancer hazard quotients are all higher than EPA upper limit of non-cancer hazard quotient. Major contributors are Cl (65%) and As (14%) in R<3, Cl (81%) and As (9%) in R3-5, and P (31%), As (24%), Pb (17%), and Cr (14%) in R>5. Fossil fuel combustion is the major source among seven sources of non-cancer hazard quotients in R<3 (62%) and R3-5 (68%), while road dust is the major source in R>5 (29%).

(7) Non-cancer hazard quotients in nearest distance group ranged from 1.0 to 15.5. The highest non-cancer hazard quotient was observed in Nongzhanguan station, while lowest non-cancer hazard quotient was observed in Changping station. Cl is the major source in six stations out of 12 stations, accounting for 68% of total non-cancer hazard quotients. Fossil fuel combustion (63%) is the major source in 6 out of 12 stations, and road dust (29%-31%) is major sources for the remaining 6 stations.

(8) Respiratory system impairment is the most vulnerable system impairment among all temporal studies. Respiratory system impairment is the most vulnerable system impairment in almost all spatial studies, except for nearest distance group. For nearest distance group, respiratory system impairment is the most vulnerable system impairment in 7 out of 12 stations among six affected system impairments.

Windsor:

(1) Non-cancer hazard quotients in Windsor estimated from three-year average was 0.15, higher than EPA upper limit of non-cancer hazard quotient. Major contributors of non-cancer hazard quotient are As (50%), Cd (15%), Pb (9%), and Mn (9%).

(2) Non-cancer hazard quotients estimated were 0.15, 0.14, and 0.14 in 2013, 2014, and 2015 respectively, higher than EPA upper limit of non-cancer hazard quotients. Major contributors of non-cancer hazard quotients are As (46%-54%), Pb (13%-17%), Cd (9%-10%), and Mn (8%-10%).

(3) Seasonal non-cancer hazard quotients were 0.14, 0.17, 0.17, and 0.10 in spring,

summer, autumn, and winter respectively, higher than EPA upper limit of non-cancer hazard quotients. Percentages of non-cancer hazard quotients are higher than its share in summer (30%) and in autumn (28%), while lower than its share in spring (23%) and in winter (19%). Major contributors of seasonal non-cancer hazard quotients are the same as in three-year average.

(4) Non-cancer hazard quotients in Beijing are higher than non-cancer hazard quotients in other cities in China and in Mexico. Non-cancer hazard quotient in Beijing is higher than in Windsor as well. Except for Xiamen and Guangzhou, non-cancer hazard quotients in other cities in China and in Mexico are higher than in Windsor.

5.2 Recommendations

- The $PM_{2.5}$ mass concentrations used in this study are $PM_{2.5}$ outdoor ambient concentration. Outdoor ambient concentration is different from personal exposure concentration. Hence, future studies may employ personal exposure concentrations to assess health risk. If the sampling costs too much, future study can employ a personal exposure model to calculate personal exposure concentration.
- Personal habits such as wearing a mask may lower the exposure time and personal exposure concentration. Future study can take uncertainties of personal habits into consideration to estimate the health risks.
- The exposure parameters, such as inhalation rate, are different for man, woman, and children. Future studies could estimate the health risks for different genders and age groups.
- The concentrations of $PM_{2.5}$-bound PAHs, elemental components, and source profiles in Beijing were estimated According to previous studies. Future study can employ sampled $PM_{2.5}$-bound PAHs concentrations, elemental components concentrations from MEP or other available sources to estimate health risks and conduct source apportionment.
- The health risk calculated in this study was only for the route of exposure by inhalation. Airborne particles can also result in health risks through other routes,

such as ingestion and dermal, by depositing on food, drinks and fallout on the skin during outdoor and indoor activities (Hu et al., 2012). Future studies can estimate health risks from different routes.

- Future study can employ maps to present the spatial distribution of the health risks because health risks associated with air pollution is related to population density, average population group age, presence of facilities such as schools, sport fields, hospitals etc.
- The concentrations analyzed by ICP-MS with acid digestion in Windsor from 2013-2015 are available now. Future study can recalculate the health risks by using concentrations analyzed by acid digestion to keep the consistency of data in Beijing and Windsor.
- A regression analysis could be employed to extrapolate the decreasing trend of PAHs concentrations in Windsor. This could be used to prove that PAHs concentrations from 2010 to 2012 can better present the PAHs concentrations from 2013 to 2015 in Windsor.

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APPENDICES

APPENDIX A: Results of calculated average concentrations

Groups	Concentration ($\mu g/m^3$)	Groups	Concentration ($\mu g/m^3$)	Groups	Concentration ($\mu g/m^3$)	Groups	Concentration ($\mu g/m^3$)
Three-year average	75.61	0	84.39	10	73.44	20	81.03
2013	87.4	1	81.62	11	74.94	21	84.41
2014	70.63	2	80.04	12	72.30	22	84.60
2015	68.81	3	76.72	13	71.31	23	84.16
Spring	70.46	4	74.27	14	70.02	Urban	79.01
Summer	61.38	5	72.22	15	70.49	Suburban	68.25
Autumn	71.31	6	71.15	16	70.06	R<3	79.08
Winter	99.3	7	70.40	17	71.39	R3-5	78.78

APPENDIX B: Results of health risks in Beijing

(a) Overall, annual, and seasonal groups

	Mass concentration (mg/m^3)	Total Cancer risks	Cancer risks from elements	Cancer risks from PAHs	Total hazard quotients
Three-year average	73.72	2.36E-02	2.27E-02	9.37E-04	13.7
2013 annual	84.39	2.73E-02	2.62E-02	1.05E-03	15.7
2014 annual	69.05	2.21E-02	2.12E-02	8.75E-04	12.9
2015 annual	68.47	2.15E-02	2.06E-02	8.52E-03	12.8
Spring	70.46	5.37E-03	5.28E-03	9.28E-05	10.4
Summer	61.38	7.47E-03	7.34E-03	1.36E-04	4.5
Autumn	71.31	4.78E-03	4.47E-03	3.09E-04	9.2
Winter	99.3	8.93E-03	5.72E-03	3.22E-03	26.2

(b) Hours of day

Time	Mass concentration (mg/m^3)	Total cancer risks	Cancer risks from elements	Cancer risks from PAHs	Total non-cancer hazard quotients
0	84.39	1.10E-03	1.05E-03	4.36E-05	15.7
1	81.62	1.06E-03	1.02E-03	4.21E-05	15.2
2	80.04	1.05E-03	1.00E-03	4.96E-05	14.9
3	76.72	9.98E-04	9.59E-04	3.96E-05	14.3
4	74.27	9.67E-04	9.28E-04	3.83E-05	13.8
5	72.22	9.40E-04	9.03E-04	3.70E-05	13.4
6	71.15	9.26E-04	8.89E-04	3.67E-05	13.2
7	70.40	9.16E-04	8.80E-04	3.63E-05	13.1
8	70.50	9.18E-04	8.81E-04	3.64E-05	13.1
9	72.42	9.43E-04	9.05E-04	3.74E-05	13.5
10	73.44	9.56E-04	9.18E-04	3.79E-05	13.7
11	74.94	9.75E-04	9.37E-04	3.87E-05	13.9
12	72.30	9.40E-04	9.04E-04	3.65E-05	13.4
13	71.31	9.28E-04	8.91E-04	3.68E-05	13.3
14	70.02	9.11E-04	8.75E-04	3.61E-05	13.0
15	70.49	9.17E-04	8.81E-04	3.64E-05	13.1
16	70.06	9.12E-04	8.76E-04	3.62E-05	13.0
17	71.39	9.29E-04	8.92E-04	3.69E-05	13.3
18	73.81	9.61E-04	9.23E-04	3.81E-05	13.7
19	77.57	1.01E-03	9.70E-04	4.00E-05	14.4
20	81.03	1.05E-03	1.01E-03	4.18E-05	15.1
21	84.41	1.10E-03	1.06E-03	4.36E-05	15.7
22	84.60	1.10E-03	1.06E-03	4.37E-05	15.7
23	84.16	1.10E-03	1.05E-03	4.34E-05	15.6

(c) Ring groups and urban and suburban groups

	Mass concentration (mg/m^3)	Elemental cancer risk	Elemental non-cancer hazard quotient
R<3	79.08	2.34E-02	13.3
R3-5	78.78	1.48E-02	14.7
R>5	68.25	3.53E-02	2.6
Urban	79.01	1.82E-02	14.3
Suburban	68.25	7.29E-03	2.6

(d) Nearest distance group

Groups	Mass concentration (mg/m^3)	Element cancer risk	Element non-cancer hazard quotient
WS-CGZ	79.47	1.57E-02	13.2
DS-BNU	80.78	2.14E-02	3.6
GY-CGZ	77.99	1.54E-02	13.0
TT-CGZ	78.08	1.54E-02	13.0
GC-CGZ	79.08	1.56E-02	13.2
HD-PU	81.55	8.95E-03	1.3
NZG-N4R	79.61	6.24E-03	15.5
ATZX-N4R	74.89	5.87E-03	14.6
SY-N5R	75.21	2.08E-02	2.9
HR-SDZ	79.61	4.12E-02	3.1
CP-QHY	67.89	6.64E-04	1.1
DL-QHY	75.21	6.19E-04	1.0

APPENDIX C: $PM_{2.5}$ mass concentrations in Windsor ($\mu g/m^3$)

	2013	2014	2015	Three -year	Spring	Summer	Autumn	Winter
$PM_{2.5}$ mass concentrations	9.3	8.5	9.0	8.8	8.5	10.4	7.8	9.3

APPENDIX D: Health risks in Windsor

(a) Cancer risks

	2013	2014	2015	Three-year
Cr	3.61E-04	4.70E-04	4.14E-04	4.20E-04
Co	1.56E-05	1.34E-05	1.29E-06	1.40E-06
Ni	8.81E-06	9.98E-06	7.41E-06	8.93E-06
As	3.12E-04	3.40E-04	3.16E-04	3.25E-04
Cd	1.82E-05	1.53E-05	1.68E-05	1.66E-05
Pb	2.11E-05	1.69E-05	1.49E-06	1.78E-05
NAP	1.09E-05	1.15E-05	0.00E+00	1.12E-05
PAHs 03-07	2.65E-05	2.43E-05	2.57E-05	2.53E-05
PAHs 10-12	2.82E-05	2.58E-05	2.74E-05	2.70E-05
Total 03-07	7.74E-04	9.01E-04	8.08E-04	8.38E-04
Total 10-12	7.76E-04	9.03E-04	8.09E-04	8.40E-04
	Spring	Summer	Autumn	Winter
Cr	9.19E-05	1.23E-04	1.10E-04	9.46E-05
Co	3.80E-06	4.22E-06	3.11E-06	3.05E-06
Ni	2.36E-06	2.95E-06	2.14E-06	1.58E-07
As	8.05E-05	1.01E-05	9.78E-05	4.38E-05
Cd	3.62E-06	3.86E-06	5.03E-06	3.86E-06
Pb	4.45E-06	4.46E-06	4.97E-06	3.78E-06
NAP	2.55E-06	4.32E-06	2.48E-06	1.92E-06
PAHs 03-07	4.80E-06	5.89E-06	2.81E-06	1.20E-05
PAHs 10-12	4.46E-06	3.94E-06	9.43E-06	8.82E-06
Total 03-07	1.94E-04	2.49E-04	2.28E-04	1.64E-04
Total 10-12	1.94E-04	2.47E-04	2.35E-04	1.61E-04

(b) Non-cancer hazard quotients

	2013	2014	2015	Three-year
Al	0.004	0.003	0.004	0.004
V	0.006	0.005	0.005	0.006
Cr	0.004	0.006	0.005	0.005
Mn	0.015	0.017	0.014	0.013
Co	0.000	0.000	0.000	0.000
Ni	0.006	0.007	0.005	0.006
As	0.069	0.075	0.070	0.072
Se	0.000	0.000	0.000	0.000
Cd	0.015	0.012	0.013	0.013
Ba	0.005	0.004	0.004	0.004
Pb	0.025	0.020	0.018	0.021
Total	0.15	0.14	0.14	0.14
	Spring	Summer	Autumn	Winter
Al	0.004	0.005	0.003	0.003
V	0.005	0.007	0.006	0.004
Cr	0.004	0.006	0.005	0.005
Mn	0.013	0.013	0.015	0.018
Co	0.000	0.000	0.000	0.000
Ni	0.006	0.008	0.006	0.004
As	0.071	0.088	0.086	0.039
Se	0.000	0.000	0.000	0.000
Cd	0.012	0.012	0.016	0.012

Ba	0.004	0.005	0.005	0.003
Pb	0.021	0.021	0.024	0.018
Total	0.14	0.17	0.17	0.10

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