

Copyright Undertaking

This thesis is protected by copyright, with all rights reserved.

By reading and using the thesis, the reader understands and agrees to the following terms:

1. The reader will abide by the rules and legal ordinances governing copyright regarding the use of the thesis.
2. The reader will use the thesis for the purpose of research or private study only and not for distribution or further reproduction or any other purpose.
3. The reader agrees to indemnify and hold the University harmless from and against any loss, damage, cost, liability or expenses arising from copyright infringement or unauthorized usage.

IMPORTANT

If you have reasons to believe that any materials in this thesis are deemed not suitable to be distributed in this form, or a copyright owner having difficulty with the material being included in our database, please contact lbsys@polyu.edu.hk providing details. The Library will look into your claim and consider taking remedial action upon receipt of the written requests.

**CRITICAL COMPONENTS SHAPING THE HEALTH IMPACT
OF PM_{2.5} IN HONG KONG: INSIGHTS FROM CHEMICAL
SPECIATION, SOURCES, AND BIOACCESSIBILITY**

YANG QIAN

PhD

The Hong Kong Polytechnic University

**This program is jointly offered by The Hong Kong Polytechnic
University and Southern University of Science and Technology**

2026

The Hong Kong Polytechnic University

Department of Civil and Environmental Engineering

Southern University of Science and Technology

Department of Earth and Space Science

**Critical components shaping the health impact of PM_{2.5} in Hong
Kong: insights from chemical speciation, sources, and bioaccessibility**

YANG Qian

**A thesis submitted in partial fulfillment of the requirements for the
degree of Doctor of Philosophy**

October 2025

Certificate of Originality

I hereby declare that this thesis is my work and that, to the best of my knowledge and belief, it reproduces no material previously published or written, nor material that has been accepted for the award of any other degree or diploma, except where due acknowledgment has been made in the text.

_____ (Signed)

_____ YANG Qian _____ (Name of student)

Abstract

Air pollution remains a critical global health issue, with fine particulate matter (PM_{2.5}) recognized as a major pollutant due to its ability to penetrate deep into the lung and enter the bloodstream, posing systemic health impacts. *In vitro* cytotoxicity analysis is widely adopted approaches for quantifying acute cytotoxicity, while health risk assessment is for accessing chronic inhalation-related health impacts induced by PM_{2.5}. Previous health impact assessments of PM_{2.5} primarily focused on its mass concentration. However, emerging evidence suggests that the health impact of PM_{2.5} is not always directly correlated with its overall mass, but instead with its specific components, sources, and bioaccessibility. To address these research gaps, this study presents an integrated analysis of PM_{2.5}-related health impacts in Hong Kong, a megacity in south China and one of the world's most traffic-intensive regions. This thesis adopts component-specific and source-resolved perspectives, integrating chemical characterization, source apportionment, bioaccessibility analysis, and assessments of both acute biological toxicity and chronic health risks, to enhance the understanding of PM_{2.5}'s health impacts.

Not all PM_{2.5} fractions pose equal risks to human respiratory health. Specific components, despite constituting a small fraction of the total mass, may contribute considerably to inhalation toxicity. Thus, detailed investigations into these highly toxic components are needed. Moreover, the acute and chronic health effects caused by specific emission sources with toxic components are still insufficiently characterized, particularly in densely populated urban environments. Thus, this study firstly detailed the chemical speciation of PM_{2.5} using the samples collected from roadside, general urban, and coastal background areas in Hong Kong over a year. To evaluate component-specific health impacts, acute cytotoxicity was assessed using intracellular reactive oxygen species (ROS) and cell viability with the BEAS-2B cell line, along with chronic

inhalation health risks according to USEPA guidelines. As a result, trace metals were the primary drivers of both acute and chronic toxicity, with EC also contributing significantly, while polycyclic aromatic hydrocarbons (PAHs) had a lesser role.

Moreover, to investigate the key sources contributing most to health impacts, the study identified major sources of PM_{2.5} by Positive matrix factorization (PMF), including secondary aerosols, vehicle emissions, resuspended dust, shipping emissions, sea salt, and a mixed source (*i.e.*, combustion & industrial emissions). Source contributions and regional effects were further analyzed using monthly profiles and back-trajectory airmass clusters via the Hybrid Single-Particle Lagrangian Integrated Trajectory (HYSPLOT) model. To quantify the source-resolved health impacts of PM_{2.5}, we developed a novel source-resolved cytotoxicity model that integrates source profiles with biological toxicity datasets. Our analyses demonstrated two key findings: (1) local on-road traffic emissions dominated the induction of acute ROS (contributing to 84% of acute ROS effects in roadside PM_{2.5} and 63% in ambient urban PM_{2.5}); (2) in contrast, regional combustion and industrial emissions posed more substantial chronic health risks, especially for aerosols in urban and coastal areas.

Building upon our experiments, we observed that even completely insoluble substances could induce cytotoxicity, albeit through mechanisms and toxic effects that may differ from those of bioaccessible substances. This prompted us to consider the impact of bioaccessibility on cytotoxicity. Firstly, an improved extraction method using simulated epithelial lung fluid separated the bioaccessible and lung-fluid-insoluble fractions of PM_{2.5}. The bioaccessibility of key toxic components was then analyzed. Among trace metals, As, Cd, Sb, and V showed high bioaccessibility (50–80%), while Fe, Mn, and Zn exhibited lower bioaccessibility (<20%). Most parent PAHs had bioaccessibility below 10%, indicating that their toxicity primarily stems from insoluble particle phases rather than dissolved forms in lung fluid. A pioneering aspect of this study is the evaluation of *in vitro* toxicity for both bioaccessible and lung-fluid-

insoluble fractions, demonstrating that insoluble components, particularly lung-fluid-insoluble inorganic fractions, significantly induce intracellular ROS. This highlights the need to consider both soluble and insoluble fractions of PM_{2.5} in further health impact assessments.

As a whole, this study enhanced the understanding of PM_{2.5} health impacts in a densely populated coastal megacity by integrating chemical speciation, toxicological, and bioaccessibility analyses. The findings emphasized the importance of targeted, toxicity-oriented air pollution control strategies and highlighted the need for continued refinement of methodologies and comprehensive risk assessments in future research.

Acknowledgement

This PhD thesis is the culmination of an intellectual odyssey—one made richer by the immense kindness of those who shared their wisdom. I begin with profound appreciation for their guidance, without which my PhD study may not have been finished.

First and foremost, I owe an immeasurable debt to my chief supervisor, Prof. Xiangdong LI, for his continued guidance and support throughout my PhD studies. His immense passion and high standards towards research have always inspired and motivated me during my studies. Moreover, I am equally grateful to my co-supervisor, Prof. Bin CHEN at SUSTech, for his kind support and understanding.

I must acknowledge the Southern University of Science and Technology and the National Natural Science Foundation of China for funding my research project.

I would like to express my sincere appreciation to Dr. Jiawen XIE, Prof. Jianzhen YU, and her group members, Dr. Wingsze CHOW and Dr. Shan WANG, for sharing their knowledge without reservation on PM_{2.5} chemical components and PMF analyses. I should also give deep thanks to my senior group mate, Ms. Yihua WANG, for her patient teaching, continuous collaboration, and discussion during the cytotoxicity experiments. Special thanks to CEE technicians, Mr. W.S. Lam, Mr. Jazz CHAN, Mrs. Carman IP, Mr. Bosco TAN, Mr. Jonathan CHAN, and Ms. Celine CHE, for their professional skills, rigorous spirit, and willingness to troubleshoot experimental challenges with me. My thanks also go to other members and alumni of Prof. Li's research group, including Dr Jinli Cui, Dr Tangtian He, Dr. Jue ZHAO, Dr. Xiaohua ZHANG, Ms. Xin WU, Ms. Siqing WANG, Dr. Xiaohao SUN, Mr. Zehua YAN, Dr. Yao LU, Dr. Lu ZHANG, Dr. Haotian ZHENG, Dr. Ke CHEN, Dr. Shangchao XIONG,

Dr. Yangyang WU, Dr. Fang GUO, Dr. Danyang TAO, Ms. Yichen XU, and Mr. Hongyu SHI for their help and support during my PhD study.

I would sincerely appreciate the support of my beloved family, my lovely cat, and my boyfriend, who share in my struggles and triumphs. Their support enabled me to move forward without any hindrances throughout the entire journey.

As I put the final touches on my student stage, I would like to express my gratitude and pride for persevering all along.

Content

Certificate of Originality.....	III
Abstract.....	I
Acknowledgement.....	IV
List of Tables.....	IX
List of Figures	X
List of Abbreviations	XIV
1 Introduction.....	17
1.1 Introduction	17
1.2 Research objectives	20
1.3 Thesis organization.....	21
2 Literature Review.....	23
2.1 Major chemical components of urban PM _{2.5} and their sources	23
2.1.1 Inorganic chemical composition	23
2.1.2 Organic carbonaceous aerosol.....	26
2.2 Bioaccessibility of PM _{2.5}	27
2.3 Sources apportionment and air mass back-trajectory analysis	33
2.4 Acute in vitro cytotoxicity induced by PM _{2.5}	35
2.5 Chronic inhalation health risks.....	37
2.6 Summary	39
3 Methodology	42
3.1 Sample collection	42
3.2 Chemical composition analysis	43
3.2.1 Water-soluble ions	44
3.2.2 Trace metals	44
3.2.3 X-ray absorption near-edge structures (XANES) analysis	45

3.2.4 Carbonaceous species.....	45
3.2.5 Nonpolar organic compounds	45
3.2.6 pH of aerosol.....	46
3.2.7 QA/QC	47
3.3 Inhalation bioaccessibility extraction and assessment procedure.....	49
3.3.1 Bioaccessible extraction by SELF.....	49
3.3.2 Bioaccessibility analysis of PM _{2.5} -bound key toxic components	50
3.3.3 Phase separation and reconstitution for toxicological exposure.....	52
3.4 Source apportionment.....	53
3.5 Back trajectory analysis.....	55
3.6 Chronic health risk assessment.....	56
3.6.1 Chronic health risk assessment of critical toxic components	56
3.6.2 Source-resolved health risk	61
3.7 Cytotoxicity	62
3.7.1. Component-specific cytotoxicity	62
3.7.2 Mixture cytotoxicity model.....	64
3.7.3. Source-resolved cytotoxicity.....	66
3.8 Statistical method	67
4 Component-specific Health Impact of PM_{2.5} in Hong Kong.....	68
4.1 Current research and data summary in the typical megacity in the Greater Bay Area	68
4.2 Chemical composition of PM _{2.5} in Hong Kong	77
4.1.1 Inorganic chemical species.....	79
4.1.2 Organic chemical species	81
4.2 Correlation analysis of vehicular characteristics with key toxic components in PM _{2.5}	84
4.3 Acute cytotoxicity assessment.....	86
4.3.1 Cell ROS generation induced by total PM _{2.5}	86
4.3.2 Component-specific contribution to PM _{2.5} -induced ROS generation.....	89
4.3.3 Combined toxicity assessment of PM _{2.5} components.....	96

4.4 Chronic inhalation health risk assessment.....	98
4.5 Summary	102
5 Source-resolved Health Impact of PM_{2.5}	105
5.1 Source apportionment.....	105
5.1.1 Source profiles	106
5.1.2 Source contribution to key toxic components in PM _{2.5}	110
5.2 Regional effect assessment.....	111
5.3 Source-resolved acute and chronic health impact	116
5.4 Summary	120
6 Bioaccessibility-dependent Health Impact of Major Toxic Components in PM_{2.5}.....	123
6.1 Inhalation bioaccessibility of key toxic components.....	123
6.1.1 Bioaccessibility of PM _{2.5} -bound trace metals.....	123
6.1.2 Bioaccessibility of PM _{2.5} -bound PAHs.....	127
6.1.3 Spatial comparison for bioaccessibility of key toxic components in PM _{2.5}	128
6.1.4 The effect of aerosol pH.....	130
6.2 Bioaccessibility-adjusted inhalation health risks.....	133
6.3 Cytotoxicity comparison of PM _{2.5} fractions via bioaccessibility.....	136
6.4 Summary	140
7 Conclusions, Limitations, and Recommendations.....	144
Appendix 1	148
Appendix 2	152
Appendix 3	158
References	162

List of Tables

Table 2-1 The major emission source of individual trace metals in traffic-related PM _{2.5}	25
Table 3-1 Information and description of sampling sites in this study.....	43
Table 3-2 Program setting of the heating block	44
Table 3-3 Limitations of detection in this study.....	45
Table 3-4 The compositions and concentrations of modified simulated epithelial lung fluid (pH=7.4)	50
Table 3-5 Detailed Settings information for GC-MS	51
Table 3-6 Parameters are used in the calculation of carcinogenic risks and hazard quotients in this study.....	60
Table 4-1 Major chemical composition of PM _{2.5} (µg/m ³) in MK, TW, and WB in the past decade (original data provided by HKEPD).....	70
Table 4-2 The reference list for PAHs composition data summary in the past decade in HK PM _{2.5}	74
Table 4-3 Fractional composition of energy of major vehicular types at the Hung Hom Cross Harbor in 2021.	86
Table 4-4 Fractional contribution of intracellular ROS induced by major analyzed toxic components	93
Table 4-5 Exposure concentrations of individual components in the mixture and the result of DR and IPQ.....	97
Table 5-1 PMF boots map and error estimation result of the Hong Kong PM _{2.5} output profile.....	106
Table 5-2 PMF output source profile of PM _{2.5} in Hong Kong, 2021.	109
Table 6-1 Seasonal average bioaccessibility (mean ± S.D.) (%) of major metals of PM _{2.5} in Hong Kong 2021.....	126

List of Figures

Figure 1-1 Research framework of this study	21
Figure 2-1 Schematic highlighting the inhalation pathways of A) fibers inside the alveolar space interacting with macrophages; B) poorly soluble metals remaining in the alveolar space, alongside transfer through cellular mechanisms; and C) & D) soluble metals in intra- and extra-cellular regions	29
Figure 4-1 Sampling sites and the measurement result of PM _{2.5} in the past decades in Hong Kong.	69
Figure 4-2 The decade average major chemical composition of PM _{2.5} in roadside, urban, and suburban sites in Hong Kong (the original data provided by HKEPD).	69
Figure 4-3 The carbonaceous composition of PM _{2.5} in MK, TW, and WB in the past decade (the original data provided by HKEPD).....	71
Figure 4-4 The PM _{2.5} -loading trace metal composition at MK, TW, and WB in the past decade	73
Figure 4-5 The PM _{2.5} -loading PAHs composition and characteristic ratios in the past decade.	76
Figure 4-6 PM _{2.5} concentration calculated from the net weight of filters collected in three sites in this study.	77
Figure 4-7 Seasonal average concentration of analyzed components of samples at three sites in Hong Kong, 2021.....	79
Figure 4-8 The annual variation area diagram of major harmful trace metal concentrations in PM _{2.5}	80
Figure 4-9 XANES spectra illustration for arsenic speciation of PM _{2.5} in Hong Kong 2021.....	81
Figure 4-10 Seasonal average concentration of major PAHs in PM _{2.5}	83

Figure 4-11 Vehicular characteristic composition at Hom Hung cross-harbor and correlation analysis with major hazardous components in roadside PM _{2.5}	85
Figure 4-12 ROS generation induced by total extracted PM _{2.5} in Hong Kong 2021.	88
Figure 4-13 Effective concentration for individual analyzed toxin at ROS induction ratio equals to 1.5 (EC _{IR1.5}) related to control based on the BEAS-2B cell line.	89
Figure 4-14 Seasonal variation of major analyzed toxic components' contribution to total extraction-induced ROS generation in PM _{2.5} collected in HK 2021.	90
Figure 4-15 Comparison between fractional mass contribution versus intracellular ROS contribution of analyzed components in PM _{2.5}	91
Figure 4-16 The combined effect of EC-metals with the same molar ratio as real PM _{2.5} samples based on BEAS-2B cell line.	95
Figure 4-17 DR results and the fractional concentrations of EC, Fe, and Zn in the HK PM _{2.5} samples.....	96
Figure 4-18 Health risks induced by trace metals, EC (surrogated by diesel particulate), and PAHs.	99
Figure 4-19 Decade variation of inhalation health risks of PM _{2.5} in Hong Kong	100
Figure 5-1 Source profiles for PM _{2.5} samples in Hong Kong 2021.....	108
Figure 5-2 Source contribution to key chemical components of PM _{2.5} in Hong Kong 2021.	111
Figure 5-3 Monthly variation of source apportionment results in Roadside and Ambient PM _{2.5}	114
Figure 5-4 24h-back trajectory clusters-associated air mass affected the typical nonlocal sources of PM _{2.5} in Hong Kong during warm and cold seasons in	

2021.....	116
Figure 5-5 Source-resolved contributions to overall mass concentration, acute oxidative stress induction, and chronic health risks induced by key toxic components (metals+EC+PAHs) of PM _{2.5} in Hong Kong.	120
Figure 6-1 Parallel comparison and cluster analysis of trace metal bioaccessibility in Hong Kong PM _{2.5}	124
Figure 6-2 Parallel comparison of EPA PAHs' bioaccessibility in ambient and roadside PM _{2.5}	128
Figure 6-3 PLS-DA analysis result of key components' bioaccessibility in PM _{2.5} collected from roadside, urban, and background sites.	129
Figure 6-4 The in-situ pH value of aerosol with non-linear regression analysis	130
Figure 6-5 Multiple linear regression analysis of major sources and pH values in Hong Kong 2021	131
Figure 6-6 Correlation coefficient heatmap of bioaccessibility with toxic unit of ROS and in-situ pH of aerosol	132
Figure 6-7 The comparison of composition and inhalation carcinogenic risk induced by bioaccessible and total PAHs in PM _{2.5}	134
Figure 6-8 The comparison of source-resolved carcinogenic risk induced by key components based on mass and bioaccessible concentration	135
Figure 6-9 Linear regression analysis for bioaccessibility versus A) cell viability of metals; B) intracellular ROS potency of metals; C) intracellular ROS potency of PAHs. D) The Spearman correlation coefficient analysis of intracellular ROS potency of total PM _{2.5} with bioaccessibility of each toxic component.....	137
Figure 6-10 A) Cell viability comparison among four fractions with 4-time dilution separated from the overall traffic-dominant PM _{2.5} . B) Intracellular ROS comparison between four fractions with 2-time dilution separated from	

the overall traffic-dominant PM_{2.5}. C) Cell viability comparison between four fractions with a 4-time dilution separated from the overall general urban PM_{2.5}. D) Intracellular ROS comparison between four fractions with 4-time dilution separated from the overall general urban PM_{2.5}. 138

List of Abbreviations

Abbreviations	Full name
6F	6th Floor of Z block, The Hong Kong Polytechnic University
Ace	Acenaphthene
ANOVA	Analysis of variance
Ant	Anthracene
ATCC	American Type Culture Collection
BaA	Benzo [a] anthracene
BaP	Benzo [a] pyrene
BbF	Benzo [b] fluoranthene
BEAS-2B	Immortalized human bronchial epithelial cell model
BghiP	Benzo [g, h, i] perylene
BIO	Bioaccessible inorganic fraction
BkF	Benzo [k] fluoranthene
BO	Bioaccessible organic fraction
BrC	Brown carbon
BSA	Bovine serum albumin
Chr	Chrysene
CcdP	Cyclopenta [c, d] pyrene
CR	Carcinogenic risk
DacA	Dibenz [a, c] anthracene
DaeP	Dibenzo [a, e] pyrene
DahA	Dibenz [a, h] anthracene
DCFH-DA	2',7'-Dichlorofluorescein diacetate
DCM	Dichloromethane
DNA	Deoxyribonucleic acid
DMEM	Dulbecco's modification of Eagle's medium

DMSO	Dimethyl Sulfoxide
DPPC	Dipalmitoyl phosphatidylcholine
EC	Elemental carbon
EC_{IR1.5}	Effect concentration with an induction ratio of 1.5
FBS	Fetal Bovine Serum
Flu	Fluoranthene
GBA	Greater Bay Area
HK	Hong Kong
HKEPD	Hong Kong Environmental Protection Department
HQ	Hazard quotient
HT	Hok Tsui (Hong Kong)
HPLC	High-performance liquid chromatography
HULIS	Humic-like substances
HYSPLIT	Hybrid Single-Particle Lagrangian Integrated Trajectory
IC₅₀	The concentration inhibits 50.0% of cell viability
IcdP	Indeno [1,2,3-cd] pyrene
ICP-MS	Inductively coupled plasma mass spectrometry
IRIS	Integrated Risk Information System
ISIO	Insoluble inorganic fraction
ISO	Insoluble organic fraction
IUR	Inhalation unit risk
KC	Kwai Chung
MDL	Method detection limit
MK	Mong Kok
MTS	3-(4,5-dimethylthiazol-2-yl)-5-(3-carboxymethoxyphenyl)-
NAAQS	National Ambient Air Quality Standard
Nap	Naphthalene
NCR	Non-carcinogenic risk
NOAA	National Oceanic and Atmospheric Administration

NPOCs	Non-polar organic compounds
OC	Organic carbon
OEHHA	Office of Environmental Health Hazard Assessment
OM	Organic aerosol mass
PAH	Polycyclic aromatic hydrocarbon
PBS	Phosphate-buffered saline
PES	Polyethersulfone
Phe	Phenanthrene
PM_{2.5}	Particulate matter with a diameter of 2.5 µm or less
PMF	Positive matrix factorization
PRD	Pearl River Delta
PTFE	Polytetrafluoroethylene
Per	Perylene
Pyr	Pyrene
RD	Roadside
RfC	Reference concentration
ROS	Reactive oxygen species
RSD	Relative standard deviation
SELF	Simulated epithelial lung fluid
t-bhp	Tert-butyl hydroperoxide
TC	Total carbon
TD-GCMS	Thermal desorption-gas chromatography mass spectrometry
USEPA	United States Environmental Protection Agency
WB	Clearwater Bay
WHO	World Health Organization
WSI	Water-soluble ions
XANES	X-ray absorption near-edge structures
YL	Yuen Long (YL)

1 Introduction

1.1 Introduction

According to the World Health Organization (WHO) guidelines (2021), the majority of the global population is exposed to air quality that exceeds guideline levels. PM_{2.5}, with an aerodynamic diameter of 2.5 micrometers or less, ranks among the top ten risk factors for disease, contributing to over 4 million deaths in 2019 (Sang et al., 2022). Its small size and large surface area enable it to carry toxicants such as trace metals and polycyclic aromatic hydrocarbons (PAHs), which are linked to respiratory, cardiovascular, and carcinogenic diseases (Cheng et al., 2022; Fuller et al., 2022). Given its severe health impacts, more importance should be attached to PM_{2.5} pollution, especially in rapidly urbanizing regions, such as China's Guangdong-Hong Kong-Macao Greater Bay Area (GBA), where this study is focused.

Assessing the health impacts of PM_{2.5}'s toxic components requires integrating acute and chronic evaluation methods. The intracellular reactive oxygen species (ROS), as a widely used indicator of acute cytotoxicity (Gurgueira et al., 2002; Kelly, 2003), are implicated in inflammation, apoptosis, and other immediate cellular responses (Juan et al., 2021; Leni et al., 2020; Veerappan et al., 2019). Prolonged ROS generation can lead to cumulative damage, particularly in DNA, triggering mutations that drive respiratory diseases and chronic carcinogenesis (Yu et al., 2022). Therefore, health impact analyses that integrate acute cytotoxicity and chronic health risk assessment help to provide a clearer understanding of the pathways linking acute exposure to chronic diseases.

The health impacts of PM_{2.5} are driven not only by its mass concentration but also by its specific toxic components (Jin et al., 2017; D. Wu et al., 2022), including trace metals, polycyclic aromatic hydrocarbons (PAHs), and elemental carbon (EC) (Briggs & Long, 2016; Rahmatinia et al., 2021; Thangavel et al., 2022). Trace metals are known

to be biologically toxic and raise adverse health effects, including respiratory and cardiovascular disorders (Meng et al., 2013; Tchounwou et al., 2012). PAHs formed during incomplete combustion processes, particularly from gasoline and diesel engines, are recognized as potent carcinogens and known to exert toxic effects through several mechanisms, including ROS induction, genotoxicity, carcinogenicity, and endocrine disruption (Borm et al., 2005; Kim et al., 2013; Kramer et al., 2021; Wang et al., 2022). EC is considered comparable to and strongly correlated with black carbon (BC), which are sometimes used interchangeably depending on the specific analytical methods (Aurell et al., 2015; Boongla et al., 2021; Luo et al., 2021; Phairuang et al., 2023). Herein, BC is a subcategory of fine particles, associated with long-term mortality burden and various diseases such as high blood pressure and neurodegenerative diseases (Baumgartner et al., 2014; Shang et al., 2022; Wei et al., 2023). As a significant insoluble fraction of PM_{2.5}, EC has recently been investigated for its contribution to acute cytotoxicity, induced by combustion-emitted PM_{2.5}, although direct evidence of its contribution to the biological toxicity of ambient PM_{2.5} remains insufficient (Chen et al., 2024; The World Health Organization European Center for Environment and Health, 2012). In short, the detailed quantification of component-specific contributions of these key components to overall toxicity in ambient PM_{2.5} in such a coastal megacity with rapid urbanization remains significant.

Effective mitigation of the health impacts of PM_{2.5} requires targeting not only the key components but also the key emission sources. For instance, PM_{2.5} from household biomass combustion is significantly more toxic than coal-fired power plant emissions due to its elevated carbonaceous content (Wu et al., 2022). In China's GBA, a dynamic economic hub spanning 56,000 km², PM_{2.5} pollution poses a persistent challenge despite early air quality control efforts (Chow et al., 2022a). Compared with regions like Beijing-Tianjin-Hebei, the GBA benefits from relatively better air quality; however, rapid urbanization, increasing vehicle ownership, and regional influences continue to

drive PM_{2.5} levels. Megacities like Hong Kong, Guangzhou, and Shenzhen are affected by local traffic and regional continental emissions, necessitating a detailed understanding of PM_{2.5}'s most toxic sources to underpin targeted control strategies (Fu et al., 2023; Xing et al., 2023). In addition, previous studies have focused on source apportionment based on chemical profiles or the *in vitro* toxicity of specific sources separately (Chow et al., 2022a; Yan et al., 2022b; Wu et al., 2022), highlighting a research gap in connecting source profiles with *in vitro* toxicity analysis. For the source apportionment of PM_{2.5}, existing studies primarily focus on detailed chemical source profiles, while few efforts evaluate key toxic components and the relative contributions of local versus nonlocal sources. For the *in vitro* toxicity of PM_{2.5}, several recent studies have targeted the *in vitro* toxic effects generated by PM_{2.5} from specific sources, such as cooking, shipping, and vehicle emissions (Chen et al., 2024; Guo et al., 2023; Wu et al., 2018). These studies emphasized the higher cytotoxicity of PM_{2.5} from specific sources compared with ambient ones, as well as the toxicity mechanism of the critical toxic components. However, for the general urban PM_{2.5} emitted by various sources, the challenge remains in quantifying the source-specific contributions to the overall biological effect induced by PM_{2.5}.

In addition to specific components and sources, the bioaccessibility of PM_{2.5} fractions also influences inhalation health impacts, especially in *in vitro* toxicity analysis. Firstly, current studies on PM_{2.5} toxicology often focus on total PM_{2.5} or specific components extracted using non-physiological solvents, such as water or organic solvents, which poorly mimic the interactions with lung fluid (Chen et al., 2019; Jin et al., 2019; Leclercq et al., 2017a; Palleschi et al., 2018). These methods fail to reflect real human exposure, as not all PM_{2.5} components are fully soluble in lung fluid or capable of crossing the gas-blood barrier (Innes et al., 2021; Soukup & Becker, 2001). To address this limitation, bioaccessibility was incorporated into *in vitro* toxicity analyses using simulated lung fluid to simulate the environmental real inhalation conditions. Secondly,

the toxicity mechanisms of PM_{2.5} may vary depending on its bioaccessibility. Soluble components, such as certain metals and ions, readily cross the blood barrier, causing systemic toxicity. In contrast, insoluble fractions, such as lung-fluid-insoluble organics, primarily induce localized effects, such as membrane disruption (Meng et al., 2013). Recent studies have highlighted that bioaccessible components can trigger oxidative stress and cellular damage, whereas insoluble particles can also induce reactive oxygen species (ROS) and pro-inflammatory responses by interacting with cell membranes or molecular targets (Leclercq et al., 2017a; Øvrevik et al., 2015). Thus, investigating the toxicological effects of bioaccessible and lung-fluid-insoluble PM_{2.5} fractions is critical for understanding component-specific health impacts under environmentally realistic exposure scenarios.

Overall, systematically investigating these research gaps would add depth to our understanding of the acute and chronic health concerns induced by urban PM_{2.5}, providing actionable insights for targeted air pollution mitigation strategies in rapidly urbanizing coastal regions, such as Hong Kong, through component-specific, source-resolved, and bioaccessibility-dependent perspectives.

1.2 Research objectives

This study aimed to address three main scientific questions related to the inhalation health impacts of PM_{2.5} in Hong Kong through six-tiered objectives organized into three main chapters. As shown in **Fig. 1-1**, this study developed a holistic framework to examine how key toxic components, specific sources, and bioaccessibility drive the inhalation health impacts of PM_{2.5}. The detailed objectives of this study are listed below:

- (1) to elucidate the chemical composition of PM_{2.5} in Hong Kong, with a focus on identifying key toxic constituents (*e.g.*, EC, trace metals, PAHs);
- (2) to assess the relative contributions of individual PM_{2.5} components to

- acute cytotoxicity and chronic health risks;
- (3) to resolve the source profiles and spatial distributions of PM_{2.5} and assess the regional effects;
- (4) to quantify the contributions of specific sources to acute cytotoxicity and chronic health risks induced by PM_{2.5};
- (5) to compare the bioaccessibility of major toxic PM_{2.5} components (trace metals and PAHs) across different spatial and seasonal contexts;
- (6) to evaluate how bioaccessibility affects PM_{2.5}-induced health impacts and compare the acute cytotoxicity of PM_{2.5} fractions differentiated by bioaccessibility (bioaccessible vs. lung-fluid-insoluble, organic vs. inorganic).

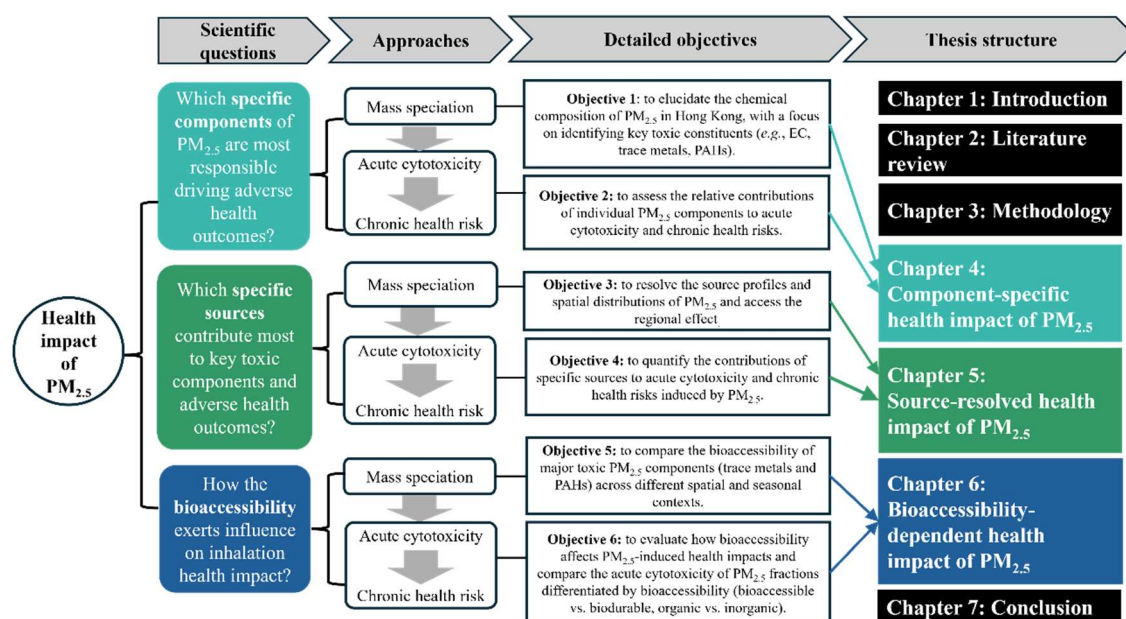


Figure 1-1 Research framework of this study

1.3 Thesis organization

This thesis consisted of seven chapters. The present part provides some background

information on PM_{2.5} pollution, clarifying gaps in identifying health-related PM_{2.5} compositions for both non-organic and organic fractions, and states the purpose of this PhD project. **Chapter 1** gave an overall introduction, including research background, current gaps, and major objectives of this study. **Chapter 2** provided a comprehensive review of current research on dissecting PM_{2.5} compositions, their related acute and chronic inhalation health impacts, and protocols for key components of *in vitro* toxicity detection techniques over the past decades. In addition, this chapter was accompanied by elucidations of critical research gaps in investigating the component-specific biological effects in complex mixtures and the bioaccessible chemical substances related to health risks in PM_{2.5}. **Chapter 3** presented the details of the sampling campaign and the experimental routine for achieving the research objectives. **Chapter 4** detailed the chemical characterization, the cytotoxicity induced by PM_{2.5}, and the contribution of each critical component (*i.e.*, EC, trace metals, and PAHs). **Chapter 5** described the results of source apportionment, air mass back-trajectory analysis, and evaluates the regional effect. In addition, a source-resolved health impacts model was established to quantify the contributions of each source to acute cytotoxicity and chronic health risks associated with overall PM_{2.5} in Hong Kong. **Chapter 6** focused on how bioaccessibility affects acute and chronic health impacts. The comparison of cytotoxicity among different fractions, based on bioaccessibility and polarity, was expounded. Finally, the significant findings, implications for future research, and limitations of this study were generally summarized in **Chapter 7**.

2 Literature Review

2.1 Major chemical components of urban PM_{2.5} and their sources

2.1.1 Inorganic chemical composition

The inorganic chemical composition of PM_{2.5} is mainly composed of WSI, metals, and EC. PM_{2.5}-bound WSI are mainly composed of SO₄²⁻ and NO₃⁻, Cl⁻, NH₄⁺, Na⁺, Ca²⁺, Mg²⁺, etc., which are produced mainly by oxidation and reduction reactions and then emitted to the atmosphere, forming the secondary particles (Hong et al., 2022). WSI are key constituents of PM_{2.5}, typically accounting for 20% to 70% of the particulate mass, with some studies reporting averages around 68% (Guo et al., 2023; Hong et al., 2022). These ions significantly influence the hygroscopicity and acidity of PM_{2.5}, affecting particle growth and atmospheric chemistry (Guo et al., 2023). The dominant ions within this group are sulfate (SO₄²⁻), nitrate (NO₃⁻), and ammonium (NH₄⁺), collectively known as secondary inorganic aerosols, which often make up over 80% of the total WSI in PM_{2.5} (Hong et al., 2022; Huy et al., 2020). Sulfate and nitrate primarily form through both direct emissions and the atmospheric oxidation of precursor gases, such as SO₂ and NO_x, in both the gas and aqueous phases. Meanwhile, ammonium acts as a neutralizing cation, stabilizing these acidic components (Q. Guo et al., 2023; Hong et al., 2022). In addition to these, chloride (Cl⁻), sodium (Na⁺), calcium (Ca²⁺), magnesium (Mg²⁺), potassium (K⁺), and other crustal or sea salt-derived ions are also present, with their proportions varying depending on local sources and meteorological conditions (Hong et al., 2022; Z. Wu et al., 2021). For example, Ca²⁺ and Mg²⁺ often originate from crustal dust. They can be more abundant during certain seasons or under specific weather patterns, such as intense winter monsoons that increase dust transport in inland areas (Hong et al., 2022), while at coastal areas, the characteristics of high Ca²⁺ and Mg²⁺ usually indicate sea salt (Chow et al., 2022a; Wong et al., 2022). In addition, a current particle-size distribution study shows that NH₄⁺ is more concentrated

in fine particles, whereas Ca^{2+} tends to dominate in coarser fractions, reflecting their distinct sources and atmospheric behavior (Wu et al., 2021). The concentrations and relative proportions of these ions can fluctuate markedly between pollution episodes and clean periods, with secondary inorganic ions typically increasing during pollution events due to enhanced oxidation and atmospheric processing (Hong et al., 2022).

Another major inorganic component of $\text{PM}_{2.5}$ is trace metals, including toxic elements such as lead (Pb), cadmium (Cd), arsenic (As), and zinc (Zn). These metals originate from anthropogenic sources, including industrial emissions, vehicular exhaust, and coal combustion, with concentrations varying by geography and season. **Table 2-1** lists the primary sources of some trace metal elements. Those trace metals —Zn, Ni, Pb, Cu, Cr, and V—are typical trace metal ions in traffic-related $\text{PM}_{2.5}$ (Pulles et al., 2012; Xie et al., 2020). Among them, Fe is the main element of rich iron particles in traffic source $\text{PM}_{2.5}$ particles. Ni, V, and Pb are also closely related to automobile fuel combustion. Besides, non-exhaust traffic emissions from brake friction, tire wear, and road dust were recognized as a primary source of urban PM, significantly increasing the concentrations of Cu, Fe, and Mn (Demir et al., 2022; Fussell et al., 2022a; Zhang et al., 2020). Cr is related to automobile exhaust. Thus, characterizing the sources and chemical profiles of $\text{PM}_{2.5}$ -bound trace metals is essential for informing air quality policies and mitigation strategies, underscoring its importance in environmental and public health research.

Table 2-1 The major emission source of individual trace metals in traffic-related PM_{2.5}

Trace metal	Primary Forms	Emission source	References
Cu	CuO, CuSO ₄	Road dust, diesel vehicle emissions, and mechanical friction	Demir et al., 2022; Fussell et al., 2022b
Fe	Fe ₂ O ₃ , Fe ₃ O ₄ , Fe ₂ (SO ₄) ₃	Tire wear, brake pad friction, crustal material	Zhang et al., 2020; Chow et al., 2022a
Zn, Cd	ZnO, ZnSO ₄ , ZnCl ₂ , CdSO ₄ , CdO, CdCl ₂	Tire wear, brake pad friction	Xie et al., 2020; Wang et al., 2021
Cr, As	Cr ₂ O ₃ , CrO ₃ , As ₂ O ₃	Fossil fuel combustion	Xue et al., 2020; Jin et al., 2019
Mg, Al	MgCO ₃ , MgSO ₄ , Al	Dust caused by road surface friction, crustal material	Hong et al., 2022; Wu et al., 2021
Ni, Pb, V	NiO, NiSO ₄ , PbO, PbSO ₄ , V ₂ O ₅ , VOSO ₄	Fossil fuel combustion, heavy oil residual	Tao et al., 2017; Wu et al., 2018

EC, a major insoluble fraction of PM_{2.5}, also comes from traffic-related combustion and coal burning. EC is considered comparable and strongly correlated with black carbon (BC), which is sometimes used interchangeably depending on the specific analysis methods (Aurell et al., 2015; Boongla et al., 2021; Luo et al., 2021). EC refers to the thermally stable carbon fraction of total carbon (TC), typically measured using the thermal-optical reflectance protocol (*i.e.*, IMPROVE, IMPROVE_A, and NIOSH), which is distinct from organic carbon (OC) (Chow et al., 1993, 2001, 2007). Studies have reported that motor vehicle emissions and EC-PM_{2.5} concentrations show a strong positive correlation (Peters et al., 2004). EC is an important component of optical absorption in traffic source PM_{2.5}, which can reduce atmospheric visibility. Its structure is relatively complex, comprising chain-like structures (such as carbon black, which is abundant in urban aerosols), round ball-like structures (such as torballs), and irregular carbon particles. Current studies have linked BC (or EC) with a long-term mortality burden and various diseases, including high blood pressure and neurodegenerative

diseases (Baumgartner et al., 2014; Wei et al., 2023; Shang et al., 2022). In addition to potential chronic hazards, EC has been shown to induce acute cytotoxicity, including mitochondrial oxidative stress, mitophagy, and mitochondrial dysfunction (Li et al., 2013; Morimoto et al., 2013; Niranjana & Thakur, 2017). Due to their large surface area, EC serves as a universal carrier for toxic semi-volatile organics and metals, such as PAHs, Pb, and Cr, leading to severe biological toxic effects (Chen et al., 2024; Jiang et al., 2018; Lindner et al., 2017; Wang et al., 2021). The first detection of ambient black carbon in the human brain, indicating the possibility of BC particles penetrating the brain, has brought renewed attention to the investigation of the related health risks of BC and its primary component, EC (Min et al., 2023).

2.1.2 Organic carbonaceous aerosol

Carbon aerosols in PM_{2.5} mainly include elemental carbon (EC) and organic carbon (OC). Analytical techniques such as thermal-optical analysis, chromatography, and spectroscopy have been used to characterize OC, revealing that specific OC fractions are distinguishable from EC (Bautista et al., 2015; Chow et al., 2007). Organic carbon (OC) is a significant and complex component of PM_{2.5}, typically accounting for 10% to 70% of the total particulate matter mass, depending on the location and pollution sources (Pan et al., 2022; Wang et al., 2021). OC in PM_{2.5} originates from both primary emissions, such as vehicle exhaust, biomass burning, industrial activities, and secondary formation processes in the atmosphere, where volatile organic compounds (VOCs) undergo oxidation to form secondary organic carbon (SOC) (Cheng et al., 2010; Hu et al., 2022; Turpin & Huntzicker, 1995). This dual origin makes OC composition highly variable and influenced by environmental conditions and emission sources. Chemically, OC encompasses a wide variety of organic compounds, including alkanes, PAHs, oxygenated organics, and humic-like substances, which can be either water-soluble or insoluble (Acuña Askar et al., 2022; Lyu et al., 2019). The water-soluble organic carbon (WSOC) fraction often contains brown carbon (BrC) chromophores that

absorb ultraviolet and visible light, thereby impacting atmospheric radiative forcing and climate by contributing to solar radiation absorption and reducing snow albedo (Acuña Askar et al., 2022; L. Zhang et al., 2021).

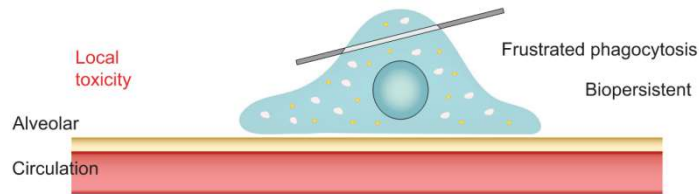
PAHs bound to PM_{2.5} are a class of hazardous organic pollutants primarily formed from the incomplete combustion of fossil fuels, biomass burning, and other combustion processes (Kim et al., 2013; T. Wang et al., 2022). They consist mainly of multi-ring structures, with three to seven rings. The main PAHs in PM_{2.5} include Naphthalene (Nap), Acenaphthene (Ace), Fluorene (Flu), Anthracene (Ant), Fluoranthene (Fla), Pyrene (Pyr), Benzo [e] pyrene (BeP), Benzo [a] anthracene (BaA), Benzo [k] fluoranthene (BkF), Benzene [b] fluoranthene (BbF), Benzo [a] pyrene (BaP), Dibenzo [a, h] anthracene (DahA), Indene [1, 2, 3-cd] pyrene (IcdP), Benzo [g, h, i] phenanthrene (BghiP), etc. (Ho et al., 2008; Yu et al., 2011). PAHs are carcinogens and highly toxic, including BaP, BaA, BbF, BkF, DahA, and IcdP, which are classified as probable carcinogens (Chen et al., 2022; Kim et al., 2013; Sun et al., 2022). Fla, Pyr, and BeP and BghiP are regarded as common carcinogens, and BaP has the strongest carcinogenicity (Kim et al., 2013; Wu et al., 2010). Studies have found that PAH concentrations in traffic intersections are significantly higher than indoor PM_{2.5} levels, indicating that PAHs in environmental particles primarily originate from traffic pollution. Studies have also confirmed that BbF, BkF, BaP, BeP, and DahA can be regarded as the representatives of traffic source PAHs; among them, BghiP and IcdP are regarded as the representatives of gasoline combustion, and Flu, Pyr, and BkF are regarded as the representatives of diesel combustion (Kim et al., 2013; Rahmatinia et al., 2021; Zhang et al., 2015).

2.2 Bioaccessibility of PM_{2.5}

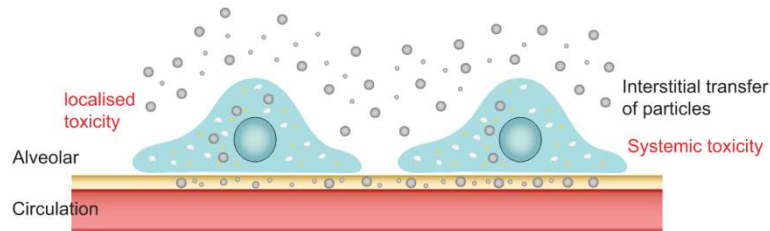
Bioaccessibility, defined as the fraction of a substance released in lung fluid and available for body absorption (Hillwalker & Anderson, 2014; Wei et al., 2018; Zhou et

al., 2022), is a critical factor in assessing the health risks associated with inhaled particulate matter. This concept of bioaccessibility is distinct from biodurability, which refers to the extent to which particles and fibers resist chemical, physical, and other physiological clearance mechanisms within the body (Jurinski & Rimstidt, 2001; Utembe et al., 2015). While biodurability focuses on the persistence of substances within the body, bioaccessibility emphasizes their availability for absorption, which directly influences potential health outcomes. The behavior of particulate matter components after entering deep lungs, described by bioaccessibility and biodurability, may produce different oriented health effects: (1) On the one hand, in particle and fiber toxicology, the onset of disease can be determined by the residence time and/or behavior of foreign substances in the biological environment; (2) On the other hand, the importance of solubility, a factor closely related to bioaccessibility, recognized by International Agency for Research on Cancer (IARC), in contributing to both local effects in the respiratory system and systemic effects at secondary sites (Heim et al., 2020). Therefore, considering solely the total concentration of pollutants or only the bioaccessible fraction can lead to biased health risk assessment results for PM_{2.5}-bound compounds. A consensus from previous studies is that considering bioaccessibility can help avoid overestimating inhalation human health risks (Li et al., 2019; Luo et al., 2019; Sun et al., 2022).

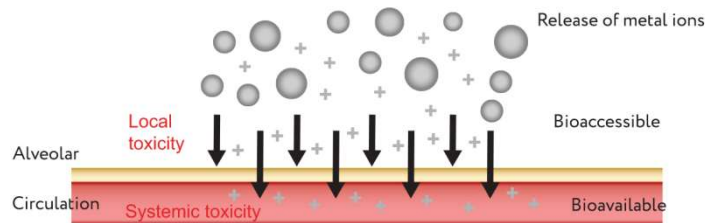
(A) **Poorly soluble fibres:** inside alveolar space, interacting with macrophage



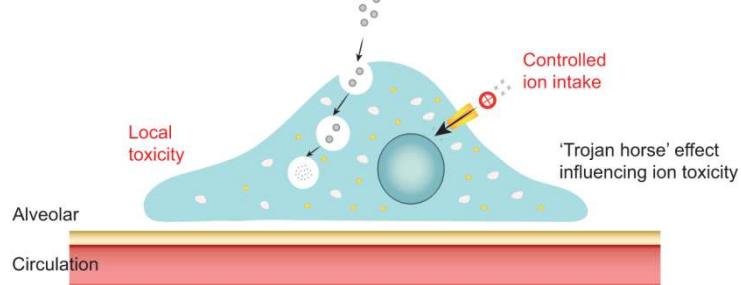
(B) **Poorly soluble particles:** inside alveolar space



(C) **Metal ions:** soluble in alveolar space



(D) **Metal ions:** soluble in macrophage



1092

1093 **Figure 2-1** Schematic highlighting the inhalation pathways of A) fibers inside the
 1094 alveolar space interacting with macrophages; B) poorly soluble metals remaining in
 1095 the alveolar space, alongside transfer through cellular mechanisms; and C) & D)
 1096 soluble metals in intra- and extra-cellular regions (Innes et al., 2021).

1097 Utilizing simulated body fluids for bioaccessibility and biodurability assessments of
 1098 materials is a promising approach that can enhance our understanding of how materials
 1099 behave in biological environments (Innes et al., 2021). Although there are no

standardized recipes, the SBFs used in inhalation bioaccessibility analysis mainly aim to simulate the internal lung environment, including (1) the most popular one in previous studies, Gamble's solution restoring the interstitial lung environment; (2) simulated epithelial lung fluid (SELF) mimicking the lung lining fluid and (3) artificial lysosomal fluid (ALF) simulating the intracellular environment of phagocytic cells (Gamble, 1945; Innes et al., 2021; Kademoglou et al., 2018; Li et al., 2019). Among these, SELF was recognized as an optimization based on Gamble's solution, simulating the epithelial lung fluid conditions of healthy, non-smoking humans (Boisa et al., 2014; Zhou et al., 2022). Besides inorganic salts whose types and concentrations are close to the published data of *in vivo* body fluids, antioxidants, high molecular mass proteins, and dipalmitoyl phosphatidylcholine (DPPC) were added in SELF to approximate the real respiratory tract fluids (Caldwell et al., 2001; Davies & Feddah, 2003; Gregory et al., 1991; Kendall, 2007). DPPC is a surfactant generated by type II alveolar epithelial cells and released into the alveolar space, which can decrease the surface tension at the air-liquid interface (Bernhard, 2016; Hassoun et al., 2018; Kademoglou et al., 2018). Previous studies have shown that DPPC concentration can significantly affect the adsorption or release of specific compounds in SLFs (Pelfrène et al., 2017; Q. Zhao et al., 2019). In addition, concentrations of DPPC and bovine serum albumin (BSA) are the adjustable parameters for approximating the lung fluid condition suffering inflammation (Zhou et al., 2022).

The bioaccessibility of PM_{2.5}-bound major chemical components varies significantly with both seasonal and spatial factors. These results may primarily be related to differences in the sources of major components in PM_{2.5} and to the influence of other components in the complex mixture (Luo et al., 2019). As the identified major toxic components in PM_{2.5}, trace metals and PAHs have become the primary components considered in inhalation bioaccessibility research (Huang et al., 2016; Li et al., 2019; Liu et al., 2023). In winter, higher concentrations and more acidic conditions can

enhance the bioaccessibility of metals and other toxic components. In summer, photochemical reactions can increase the bioaccessibility of certain organic compounds. Spatially, urban and industrial areas tend to have higher bioaccessibility of harmful components due to the presence of more acidic and reactive pollutants. Understanding these variations of bioaccessibility is crucial for assessing the health risks associated with PM_{2.5} exposure and for developing effective air quality management strategies.

For the analyzed trace metal elements, bioaccessibility varies with distance from the dominant emission sources, airborne abundance, chemical speciation, and matrix effects (Leclercq et al., 2017; Luo et al., 2011, 2019). A previous study reported that the bioaccessibility of trace metals in airborne particles, affected by different emission sources, depends on the composition of the sources in the area (Hernández et al., 2018; Huang et al., 2016). Hernández et al. (2018) reported that the bioaccessibility of trace metals collected in the industrial area were in order of Cd (39.3%) > Mn (34.1%) > Pb (14.8%), and Mn (23.0%) > Cd (22.0%) > Pb (11.4%) at the urban site (Hernández et al., 2018). Wang et al. (2021) reported the bioaccessible portion of PM_{2.5} collected in the coastal urban area around by heavy traffic flow, as which V, Pb, Cu, and Zn exhibited comparatively high bioaccessibility rates (V: 78.5%–93.7%, Pb: 79.2%–83.8%, Cu: 69.6%–78.4%, Zn: 65.8%–77.8%), whereas Mn, Ni, and Co demonstrated relatively low bioavailability rates (Mn: 55.8%–66.0%; Ni: 28.7%–63.0%; Co: 33.1%–48.7%), which also emphasized the higher inhalation health concern induced by the traffic-related metals (Wang et al., 2021).

The bioaccessibility of PAHs depends on several factors, including their volatility, solubility, particle size, and the presence of other substances that can affect their absorption. For inhalation exposure, physicochemical properties such as particle size and composition can influence the bioaccessibility of PAHs. Some studies have examined the transformation products that result from the aging of PAHs and their potential impact on bioaccessibility. For example, the oxidation of PAHs can lead to the

formation of oxygenated PAHs (oxy-PAHs), which may have different properties and bioaccessibility compared with the parent compounds. Additionally, particle size and composition can affect the bioaccessibility of PAHs during inhalation. Studies have shown that finer particles have higher deposition rates and may exhibit greater bioaccessibility than larger particles. Additionally, the presence of other substances, such as organic matter or coatings on particles, can influence the release and bioaccessibility of PAHs.

The bioaccessibility of a compound is not set in stone; rather, it depends on various chemical and physical conditions in the body fluids. For instance, studies have shown that the solubility of PM-bound compounds is higher in acidic environments, which represent an inflammatory condition, such as artificial lysosomal fluid (ALF), compared with neutral conditions, such as Gamble's solution (Calas et al., 2017; Expósito et al., 2024). Additionally, the temperature at which extraction or inhalation occurs can influence solubility and reaction kinetics (Innes et al., 2021). The agitation method (*e.g.*, shaking, sonication) can influence the dissolution of a compound in SLFs, leading to variability in measured bioaccessibility (Liu et al., 2023; Pelfrène et al., 2017). In addition to the leaching fluid condition, the ratio of solid phase to fluid volume (*i.e.*, S/L ratio) and the physical characteristics of the particles may also significantly influence bioaccessibility (Besis et al., 2022; Leclercq et al., 2017a; Pelfrène et al., 2017). Besides, the dissolution rate and bioaccessibility could be enhanced by higher S/L ratios and higher surface area-to-volume ratios compared with larger particles (Leclercq et al., 2017a; Midander et al., 2010).

Current studies have investigated the cytotoxicity induced by total PM_{2.5} or specific components extracted from PM_{2.5} (Chen et al., 2019; Jin et al., 2019; Kermani et al., 2021; Rahmatinia et al., 2021). However, the leaching agents they have most often used are pure water or organic solvents, which are seldom physiologically relevant to internal body fluids (Leclercq et al., 2017a; Palleschi et al., 2018). Meanwhile, it is worth noting

that not all components can fully diffuse into the lung fluid or even cross the gas-blood barrier (Innes et al., 2021; Soukup & Becker, 2001; Zhou et al., 2022). Roper and his colleagues have also underscored that the ultimate goal of extracting PM_{2.5} for toxicological analysis is to obtain substances representative of real human exposure (Roper et al., 2019). Furthermore, for some insoluble organics, the cosolvent used to extract them may expose them to cells in a state completely inconsistent with their actual state in lung fluid. Additionally, extraction methods used for PM_{2.5}, such as ultrasonic or vortex, can alter the structure of sensitive compounds within the particulate matter before toxicity assessments, potentially leading to unexpected biases in the results (Palleschi et al., 2018; Roper et al., 2019). To explore the toxic effects of particulate matter under *in vitro* simulated inhalation conditions, it is necessary to embed a bioaccessible state into the extraction process, thereby better mimicking realistic conditions.

2.3 Sources apportionment and air mass back-trajectory analysis

The EPA Positive Matrix Factorization (PMF) 5.0 model is an advanced multivariate receptor model developed by the USEPA to facilitate sophisticated environmental data analysis, with a primary focus on source apportionment of pollutants across diverse matrices, including air, water, sediments, and other environmental media (USEPA, 2015). This robust tool is widely used to identify and quantify the contributions of distinct pollution sources to environmental samples, thereby supporting the formulation and evaluation of air and water quality standards, advancing exposure assessment research, and enhancing environmental forensics investigations. By using this model, several studies have reported the detailed source profiles of urban PM in the GBA (Chow et al., 2022a; Li et al., 2020; Tao et al., 2017; Wong et al., 2022). Incorporating trace-metal tracers, a significant increase in the contribution of vehicular emissions to urban PM_{2.5} has been observed in Shenzhen, a megacity in the GBA adjacent to Hong Kong (Yan et al., 2022). Similarly, nine sources of PM_{2.5}, including primary biogenic

emissions first reported, have been identified across six sites in Hong Kong by using multiple organic tracers (Chow et al., 2022a).

Another model, the HYSPLIT, is widely used to generate air mass back trajectories into clusters (Draxler & Hess, 1998; Stein et al., 2015; Park et al., 2022). The HYSPLIT model, developed by the Air Resources Laboratory of the National Oceanic and Atmospheric Administration (NOAA), is an atmospheric transport and dispersion model applied in air pollution research to simulate pollutant movement, dispersion, chemical transformation, and deposition across local to global scales (Draxler & Hess, 1998; Stein et al., 2015). Recent research has demonstrated the growing use of the HYSPLIT model to identify potential source areas and the long-range transport of pollutants, thereby assisting in understanding regional pollution patterns and the impact of distant sources on local air quality, by utilizing meteorological data (Draxler & Hess, 1998; Wong et al., 2022). For example, integrated approaches coupling HYSPLIT with meteorological models such as WRF and satellite data have been employed to trace PM_{2.5} emissions from biomass burning in regions such as Central Kalimantan, revealing spatial distribution patterns and the meteorological influences on pollutant dispersion (Nurlatifah et al., 2025). In addition, Wong et al. (2022) utilized the HYSPLIT model to group air mass back trajectories into different clusters and combined them with the source profile of PM₁₀ obtained by the PMF model, which resolves the input data matrix into sources. The air mass trajectories from different clusters supported the interpretability of the PMF results, thereby elucidating the source contributions to coarse particulate matter in Hong Kong. Methodologically, HYSPLIT supports both particle and puff dispersion modeling, using meteorological inputs to simulate pollutant transport in three dimensions, thereby enhancing understanding of pollutant source-receptor relationships and informing air quality management strategies (Yerramilli et al., 2012). However, existing studies primarily focus on detailed chemical source profiles. Few efforts are directed at evaluating the key toxic components and the relative

contributions of local versus non-local sources.

2.4 Acute *in vitro* cytotoxicity induced by PM_{2.5}

Cell viability, a critical toxicological endpoint, is widely used to assess the cytotoxic effects of PM_{2.5} exposure in human cells, reflecting the potential acute health impacts of fine particulate matter (Riss et al., 2004). The MTS (3-(4,5-dimethylthiazol-2-yl)-5-(3-carboxymethoxyphenyl)-2-(4-sulfophenyl)-2H-tetrazolium) assay is a colorimetric method that measures cellular metabolic activity as an indicator of cell viability (Cho et al., 2018; Jin et al., 2019). In this assay, viable cells reduce the MTS tetrazolium compound to a formazan product, which is quantifiable by absorbance, providing a reliable measure of cytotoxicity induced by PM_{2.5} components, such as trace metals, EC, and PAHs (Chen et al., 2024; Chen et al., 2024; Wu et al., 2018). The assay's sensitivity and reproducibility make it valuable for evaluating PM_{2.5} toxicity across cell types, such as bronchial epithelial cells (*e.g.*, BEAS-2B), and its results correlate with oxidative stress and inflammatory responses linked to respiratory and cardiovascular diseases.

ROS induced by PM_{2.5} play a crucial role in understanding the health impacts of air pollution, making their investigation of utmost importance (Gurgueira et al., 2002; Kelly, 2003). ROS, such as superoxide anion (O₂⁻), hydroxyl radical (-OH), and hydrogen peroxide (H₂O₂), are highly reactive molecules that can initiate oxidative reactions, leading to the oxidation of lipids, proteins, and DNA (Juan et al., 2021). As mentioned above, the key toxic components in PM_{2.5}, such as trace metals, PAHs, and BC, can lead to ROS generation at the different toxic units. Two commonly employed approaches for ROS analysis are the dithiothreitol (DTT) assay and the intracellular approach. The DTT assay measures PM_{2.5}-induced ROS generation by quantifying the depletion of DTT, a thiol compound, in the presence of PM_{2.5} (Kramer et al., 2021; Lin & Yu, 2019a). It estimates the ROS-generating potential of PM_{2.5} but lacks information

on the specific ROS species and their intracellular effects. In contrast, intracellular approaches use fluorescent probes, such as dichlorodihydrofluorescein diacetate (DCFH-DA), which can enter cells and be oxidized by ROS, generating a fluorescent signal proportional to intracellular ROS levels (Bates et al., 2019; Murphy et al., 2022; Schieber & Chandel, 2014). This approach enables the direct measurement of ROS within cells after exposure to active species in PM_{2.5}, providing insights into their potential downstream effects and a relatively feasible platform for investigating the source-resolved mixed cytotoxicity model.

Existing studies have investigated the acute biological toxicity effects of several specific emissions. For example, Guo et al. (2023) reported that cooking-emitted PM_{2.5} was approximately twice that of general urban PM_{2.5}. Wu et al. (2018) are convinced that shipping-emitted PM_{2.5} can generate a 5.3- to 8.7-fold biological effect compared with those induced by light-polluted ambient PM_{2.5}. Another study found that vehicle-emitted PM_{2.5} in Hong Kong can induce cell membrane damage, oxidative stress, and an inflammatory response in the A549 cell line (Niu et al., 2020). These research efforts highlighted that PM_{2.5} originating from specific sources exhibits greater cytotoxicity than ambient PM_{2.5}, and also explored the toxicity mechanisms of key harmful components.

It is worth noting that several studies have conducted quantitative assessments of source-specific contributions to PM_{2.5}-induced oxidative potential (OP) across different regions. For instance, Y. Cheng et al. (2021) quantified the contributions of traffic emissions and secondary aerosols to PM_{2.5} OP in Hong Kong by combining the positive matrix factorization (PMF) model with the dithiothreitol (DTT) assay. Fang et al. (2016) Fang et al. (2016, Atmospheric Chemistry and Physics, 16, 3865–3879) reported that biomass burning accounted for 35% of the OP in the southeastern region of North America. X.-C. Chen et al. (2020) further confirmed a strong correlation ($r = 0.78$, $p < 0.01$) between traffic emissions (particularly from diesel vehicles) and OP in roadside

PM_{2.5} in Hong Kong. In a study of Brazilian cities, Serafeim et al. (2023) indicated that trace metals (*e.g.*, Fe, Cu) from industrial emissions were the primary drivers of OP. These studies provide methodological references for the source apportionment and toxicity correlation analysis of PM_{2.5} in Hong Kong conducted in this paper.

2.5 Chronic inhalation health risks

PM_{2.5} particles are recognized for their ability to penetrate deep into the respiratory system, leading to adverse health outcomes (Sangkham et al., 2024; Xing et al., 2016). Chronic inhalation of PM_{2.5} poses significant health risks, both carcinogenic and non-carcinogenic, primarily affecting the respiratory and cardiovascular systems (Brook et al., 2010; Yang et al., 2020). Long-term exposure to PM_{2.5} has been robustly linked to premature death, particularly among individuals with pre-existing heart or lung diseases, as well as to reduced lung function growth in children (Yang et al., 2022). Epidemiological evidence indicates that PM_{2.5} exposure contributes to increased incidence and mortality of lung cancer, chronic obstructive pulmonary disease (COPD), ischemic heart disease, stroke, and other chronic conditions (Colonna et al., 2022). The USEPA health risk assessment model is a systematic process for evaluating potential health risks associated with exposure to environmental hazards. This model facilitates an understanding of the impact of pollutants and chemicals on human health and is essential for informed regulatory decision-making and policy development. The inhalation health risks associated with PM_{2.5} exposure have been extensively studied, with a particular focus on non-carcinogenic and carcinogenic hazards, which are quantified through hazard quotient (HQ) and carcinogenic risk (CR) metrics (USEPA, 2009).

The carcinogenic risk of PM_{2.5} is attributed mainly to combustion-derived particles containing PAHs and other organic compounds. These substances induce oxidative stress, DNA damage, inflammation, and tumor-promoting processes (Dilger et al., 2016;

Kim et al., 2013). Notably, ambient PM_{2.5} exposure is a leading risk factor for lung cancer in non-smokers, primarily causing adenocarcinoma in the peripheral lung through tumor-promoting mechanisms rather than direct mutagenesis. Studies suggest that inflammation and immune responses play critical roles in this carcinogenesis pathway. Higher PM_{2.5} doses, such as those encountered in occupational settings or smoking, increase genotoxic effects and the risk of squamous cell carcinoma in the central airways (Ihantola et al., 2022; Platel et al., 2020).

Beyond carcinogenicity, PM_{2.5} also induces non-carcinogenic adverse effects through oxidative stress and systemic inflammation, which exacerbate respiratory diseases like asthma, COPD, and pneumonia. Cardiovascular impacts include arrhythmias, elevated blood pressure, and an increased risk of heart attacks. Vulnerable populations such as the elderly, children, pregnant women, and individuals with pre-existing conditions are more susceptible to these effects. Emerging research also links PM_{2.5} exposure to neurological disorders, kidney disease, and adverse birth outcomes, highlighting its systemic toxicity.

The health risks are compounded by the complex chemical composition of PM_{2.5}, which includes various toxic components, including heavy metals, PAHs, and EC. Heavy metals bound to PM_{2.5} have been identified as significant contributors to health risks. Current research has shown that PM_{2.5}-bound trace metals induce oxidative stress and inflammation, thereby elevating the risks of cardiovascular and respiratory diseases (Brook et al., 2010; Pope et al., 2016). Additionally, their deposition harms ecosystems by contaminating soil and water (Tchounwou et al., 2012). For instance, studies in southwestern China have evaluated the bioavailable ratios of PM_{2.5}-bound heavy metals, calculating both hazard quotients and carcinogenic risks to assess non-carcinogenic and carcinogenic hazards (Han et al., 2021; Xie et al., 2020). Similarly, assessments of trace metals, such as Sb, have demonstrated their potential to cause health issues through inhalation, with HQ calculations indicating notable non-carcinogenic risks (Jiang et al.,

2021). The noncarcinogenic risks from metals such as As have been quantified, with HQ values suggesting varying degrees of health concern depending on the metal and exposure level (Li et al., 2023). Organic components, particularly PAHs, are also critical in determining PM_{2.5} toxicity. PAHs are associated with carcinogenic risks due to their mutagenic and carcinogenic properties. Studies have highlighted the presence of PAHs in PM_{2.5} from combustion sources, such as coal combustion, which significantly contribute to the overall health risk profile (Chen et al., 2022; Groot et al., 2017; Li et al., 2023). The inhalation of PAHs has been linked to increased carcinogenic risk, emphasizing their role as key toxic components. EC, a major component of black carbon, is another toxic element within PM_{2.5} that influences health risks. EC is often co-emitted with organic carbon during combustion processes and has been associated with oxidative stress and inflammatory responses in the respiratory system (Chen et al., 2024; Grahame et al., 2014). The presence of EC enhances the overall toxicity of PM_{2.5}, contributing to both non-carcinogenic and carcinogenic health risks. In summary, the health risks posed by PM_{2.5} depend closely on its chemical composition, with metals, PAHs, and EC playing pivotal roles. Metals contribute primarily to non-carcinogenic risks, as evidenced by HQ assessments, while PAHs are significant contributors to carcinogenic risks. EC acts as a facilitator of oxidative stress, further exacerbating health effects. These findings underscore the importance of characterizing the key toxic components of PM_{2.5} to assess and mitigate inhalation health risks accurately (Chen et al., 2022; Li et al., 2023; Xie et al., 2020).

2.6 Summary

This literature review synthesizes current knowledge on the chemical composition, sources, bioaccessibility, and health impacts of urban PM_{2.5}, with a focus on traffic-related emissions in megacities such as Hong Kong in southern China. PM_{2.5} comprises inorganic components (*e.g.*, WSI like SO₄²⁻, NO₃⁻, NH₄⁺), trace metals like Fe, Zn, Cu from vehicles, EC, and organic aerosols (*e.g.*, OC, HULIS, PAHs). Data from Hong

Kong sites (MK roadside, TW urban, and WB suburban) show declining PM_{2.5} levels over two decades due to emission controls, though traffic-heavy sites have elevated concentrations of EC, metals, and PAHs. PMF and HYSPLIT models identify vehicle emissions, secondary aerosols, biomass/coal combustion, and regional transport as key sources, with winter peaks driven by enhanced secondary formation. Bioaccessibility studies using SBFs (*e.g.*, Gamble's, SELF, ALF) demonstrate component-specific release, influenced by pH, DPPC, and particle properties, underscoring the need for soluble fraction analysis to prevent risk overestimation. Acute *in vitro* cytotoxicity via MTS and ROS assays (*e.g.*, DTT, DCFH-DA) links PM_{2.5} to oxidative stress, inflammation, and mitochondrial dysfunction, with traffic-derived PM_{2.5} showing higher toxicity. Chronic inhalation risks, assessed via EPA models (HQ for non-carcinogenic risk, CR for carcinogenic effects), tie PM_{2.5} to respiratory, cardiovascular, and neurological issues, with metals driving both non-carcinogenic and carcinogenic risks; meanwhile, PAHs and EC mainly contribute to carcinogenic risk.

Extending the work of previous studies, several gaps remain in the following aspects: (1) The extent to which individual key components, particularly EC, contribute to the overall toxicity of ambient and traffic-related PM_{2.5} remains unquantified. (2) Prior research has predominantly focused on source apportionment using chemical profiles or the cytotoxicity of specific sources in isolation, lacks detail on local vs. nonlocal toxic fraction contributions, and few studies use physiologically relevant extraction for toxicity tests. (3) Existing studies on PM_{2.5} cytotoxicity often center on total PM_{2.5} or specific components extracted via non-physiological solvents (*e.g.*, MilliQ water or organic solvents). These approaches poorly simulate interactions with lung fluid and thus fail to reflect real-world human exposure, as not all PM_{2.5} components are fully soluble in lung fluid or able to cross the gas-blood barrier. (4) The toxic mechanisms of PM_{2.5} particles may differ based on their bioaccessibility or biodurability once inhaled into the deep lung. Overall, future work should leverage multi-omics, advanced

1395 modeling, and bioaccessibility-adjusted assessments to address these gaps and guide
1396 targeted air quality strategies in megacities.

3 Methodology

3.1 Sample collection

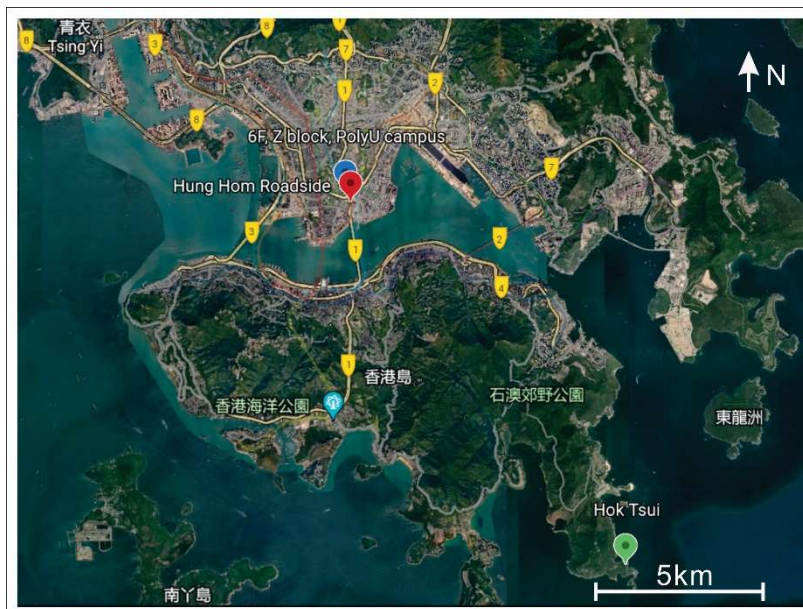


Figure 3-1 Three sample sites of Hong Kong 2021 in this study

Twenty-four-hour $PM_{2.5}$ samples were collected every six days, except on special weather days, from February 2021 to January 2022 at three sampling sites in Hong Kong. **Fig. 3-1** shows the locations of the sampling sites. **Table 3-1** provides the description and characteristics of each site. RD is the roadside sampling site next to the Hung Hom tunnel toll station entrance, which is significantly impacted by fresh vehicular emissions. 6F represents the general urban sampling site located on the sixth floor, Z block, in the Polytechnic University of Hong Kong campus. The coastal background samples were collected at Hok Tsui (HT), located at almost the southernmost edge of Hong Kong Island, facing the clean and pollution-free sea breeze. All the sampling sites were equipped with a high-volume sampler (TH-1000C II, Wuhan Tianhong Instruments Co., Ltd., China). The high-volume sampler collected $PM_{2.5}$ on $8' \times 10'$ quartz fiber filters every six days from noon to noon each sampling period at a rate of $1 \text{ m}^3/\text{min}$, and a total of 140 filter samples were collected during the

campaign. All quartz filters were baked at 500 °C for 5 hours before sample collection. After equilibrating in a desiccator at 25 °C and 40% relative humidity for at least 24 hours, the filters were weighed using a sensitive balance (± 0.0001 g) for the calculation of their net weight.

Table 3-1 Information and description of sampling sites in this study.

City	Site	Location	Altitude (m)	Description	Sampling period	Sampling frequency	Number of samples
Hong Kong	Hung Hom Roadside	22°18'13"N, 114°10'45"E	5	Fresh traffic emission source	Feb 2021- Jan 2022	Weekly	45
Hong Kong	6F, Z block, PolyU campus	22°18'24"N, 114°10'57"E	33	General urban site			43
Hong Kong	Hok Tsui	22°12'34"N, 114°15'29"E	136	Coastal background site			49

3.2 Chemical composition analysis

To access and quantify the major chemical composition of PM_{2.5} in roadside, general urban, and coastal background sites in Hong Kong in 2021, several chemical analyses, including OCEC, water-soluble ions, and trace metals, were conducted on all the samples in this study. PM_{2.5} was calculated by subtracting the net weight of the filter before sampling from the net weight after sampling. However, the PM_{2.5} concentration calculated directly based on the weight of the filters is not very accurate due to the different humidity and unstable flow rate of the instrument. Therefore, the concentration of each principal component of PM_{2.5} was analyzed and added up to approximately re-evaluate the PM_{2.5} concentration as precisely as possible.

3.2.1 Water-soluble ions

For WSI analysis, a 13.0 cm² filter sample was extracted ultrasonically with 15 mL MilliQ water (18 Ωm) for 20 mins. The extraction process was repeated twice, and the extracts were combined. The extracts were then filtered by a 0.22 μm polyethersulfone (PES) filter. Subsequently, the soluble anions (F⁻, Cl⁻, Br⁻, NO₂⁻, NO₃⁻, PO₄³⁻, SO₄²⁻) and cations (NH₄⁺, Na⁺, K⁺, Mg²⁺, Ca²⁺) were analyzed using Ion Chromatography (Thermo Scientific Dionex Integrion HPIC) with Ion Pac AS14 and CS12 (*Dionex*, United States) columns.

3.2.2 Trace metals

The analyzed trace metals include Ba, Ag, Al, As, Ca, Cr, Cd, Co, Cu, Fe, K, Mg, Mn, Ni, Pb, V, and Zn. The ones with considerable inaccuracy were removed. One-thirtieth of each sample was cut and submerged in a mixture of 8 mL 70% high-purity HNO₃ and 2 mL 60% high-purity HClO₄ in test tubes, followed by gentle vortexing. The mixture was digested in a heating block with a progressive temperature rising from 50°C to 190°C, as detailed in **Table 3-2**. Remove the tubes from the heating block when the residual samples are dry. Then, a 10-mL aliquot of 5% (v/v) HNO₃ was added to the test tubes, which were subsequently heated at 70°C for an hour. The final solution was filtered by a PES filter and then analyzed by inductively coupled plasma-mass spectrometry (ICP-MS) (Agilent model 7700, United States) to detect the total concentrations of various elements.

Table 3-2 Program setting of the heating block

Step	Temperature (°C)	Duration (hr)
1	50	0.5
2	75	0.5
3	100	1.5
4	125	2.5

5	150	3
6	190	Until dry

1450

1451 **3.2.3 X-ray absorption near-edge structures (XANES) analysis**

1452 The arsenic speciation of selected PM_{2.5} filter samplers (*i.e.*, one roadside and three
1453 general urban samples) was analyzed by X-ray absorption near-edge structures
1454 (XANES) without pretreatment at the National Synchrotron Radiation Research Center
1455 (NSRRC), Taiwan.

1456 **3.2.4 Carbonaceous species**

1457 OC and EC concentrations were analyzed using the DRI Thermal Optical Carbon
1458 Analyzer (Model 2001A, Atmoslytic, Calabasas, CA, USA) according to the thermal
1459 optical reflectance (TOR) protocol and the IMPROVE_A temperature protocol
1460 (Schauer et al., 2003). The total organic aerosol mass (OM) was calculated by
1461 multiplying the organic carbon (OC) concentration by a coefficient of 1.80 (Malm et
1462 al., 2017; J. Xie et al., 2020).

1463 **3.2.5 Nonpolar organic compounds**

1464 The NPOCs in the roadside and general urban samples were analyzed using the
1465 injection port thermal desorption-gas chromatography-mass spectrometry (TD-GCMS)
1466 method (Agilent 6890 GC/5973, Santa Clara, CA, USA) (Ho et al., 2008; J. Z. Yu et al.,
1467 2011). NPOCs targeted for quantification include C₁₈–C₃₅ n-alkanes, hopanes, and C₁₆–
1468 C₂₂ PAHs. The analyzed NPOCs, along with their explanations of abbreviations and
1469 detection limitations, are listed in **Table 3-3**.

1470 **Table 3-3** Limitations of detection in this study

PM _{2.5} ($\mu\text{g}/\text{m}^3$)	0.694 ^a	Carbon ^c ($\mu\text{g}/\text{m}^3$)	Hopanes & Steranes (ng/m^3)		
Trace metals ($\mu\text{g}/\text{m}^3$)		OC	0.205	22,29,30-trisnorneophopane	0.000153
Al	0.00832	EC	0.0475	22,29,30-trisnorhopane	0.000153
As	0.00174	PAHs ^b (ng/m^3)		ab-norhopane	0.000104
Ba	0.000011	Phenanthrene (Phe)	0.000751	ab-+ba-norhopane	0.000174
Be	0.000290	Anthracene (Ant)	0.000737	ab-hopane	0.000229
Cd	0.000121	Fluoranthene (Flu)	0.000563	ba-hopane	0.000382
Co	0.000680	Pyrene (Pyr)	0.000674	ab-22S-homohopane	0.000174
Cr	0.000360	Cyclopenta[cd]pyrene	0.000195	ab-22R-homohopane	0.000473
Cu	0.00146	Benzo[c]phenanthrene	0.002363	abS-bishomohopane(C32abS-hopane)	0.000174
Fe	0.000760	Benzo[a]anthracene (BaA)	0.000375	abR-bishomohopane(C32abR-hopane)	0.000473
Mn	0.000020	Chrysene (Chr)	0.000167	22S-trishomohopane (C33)	0.000174
Mo	0.000290	Triphenylene	0.002085	22R-trishomohopane (C33)	0.000473
Ni	0.000480	Benzo[b]fluoranthene	0.000396	22S-tetrahomohopane (C34)	0.000174
Pb	0.000460	Benzo[k]fluoranthene	0.000250	22R-tetrahomohopane (C34)	0.000473
Sb	0.00122	Benzo[a]fluoranthene	0.000153	22S-pentashomohopane (C35)	0.000174
Se	0.00329	Benzo[e]pyrene	0.000459	22R-pentashomohopane (C35)	0.000473
Sn	0.00249	Benzo[a]pyrene (BaP)	0.000466	aaa 20S- + baa 20R-cholestane	0.000160
V	0.000370	Perylene (Pyl)	7.64E-05	aa 20R-cholestane	0.000104
Zn	0.00137	Indeno[1,2,3-cd]	0.000313	aaa 20S 24S-methylcholestane	0.000222
WSI ^d ($\mu\text{g}/\text{m}^3$)		Benzo[ghi]perylene	0.000341	aaa 20R 24R-methylcholestane	0.000160
Na ⁺	0.00534	Dibenz[a,h]anthracene	0.000403	aaa 20S 24R/S-ethyl cholestane	0.000222
NH ₄ ⁺	0.235	Dibena[a,c]anthracene	0.00243	aaa 20R-ethylcholestane	6.25E-05
K ⁺	0.0867	Picene (Pic)	0.000313	abb 20R-cholestane	0.000104
Mg ²⁺	0.0748	Benzo[b]chrysene	0.003405	abb 20S-cholestane	0.000153
Ca ²⁺	0.0260	Coronene (Cor)	0.000306	abb 20R-methylcholestane	0.000160
Cl ⁻	0.0690	Dibenzo[a,e]pyrene	0.000341	abb 20S 24S-methylcholestane	0.000160
SO ₄ ²⁻	0.241	1,3,5-triphenylbenzene	0.0177	abb-20R-ethylcholestane	0.000104
NO ₃ ⁻	0.241			abb 20S 24R-ethylcholestane	0.000104

1471 a. Xie et al., 2019

1472 b. Ho et al., 2008; Yu et al., 2011.

1473 c. Calculated from parallel blank samples in this study.

1474 d. Calculated based on the standard with the lowest concentration.

1475 3.2.6 pH of aerosol

1476 The acidity characteristics of aerosols were characterized by $[\text{H}^+]_{\text{total}}$, $[\text{H}^+]_{\text{ins}}$, and in-

situ pH. Among them, the in-situ acidity $[H^+]_{ins}$ directly reflects the concentration of free H^+ in atmospheric aerosols, but it is difficult to measure directly. In this study, the Extended Aerosol Model (E-AIM) was used for estimation (Frieese & Ebel, 2010). Due to limitations in climate conditions and variations in ion concentrations in the sampling area, the AIM-IV model was selected to simulate $PM_{2.5}$ acidity. AIM-IV is mainly used for thermodynamic simulations of the H^+ - NH_4^+ - Na^+ - SO_4^{2-} - NO_3^- - Cl^- - H_2O aerosol system at temperatures ranging from 180 to 330K. This model does not account for the effects of Ca^{2+} , Mg^{2+} , and K^+ ions and is a simplified model under ideal conditions, but it is relatively accurate for estimating the acidity of atmospheric particulate matter. The operation of the AIM-IV model requires the input of average temperature, relative humidity, molar concentrations of water-soluble ions, and total aerosol acidity (*i.e.*, $[H^+]_{total}$) to obtain in-situ acidity (*i.e.*, $[H^+]_{ins}$) and the molar numbers of chemical substances in the aqueous phase. Among them, $[H^+]_{total}$ is the main parameter for the model operation and is calculated only based on the molar concentrations of the involved water-soluble ions (*i.e.*, SO_4^{2-} , NO_3^- , NH_4^+ , Na^+ , and Cl^- five types of ions), and the calculation formula is as follows **Eq. (1)**. The in-situ pH value can be calculated based on the model output with **Eq. (2)**.

$$H_{total}^+ = 2 \times [SO_4^{2-}] + [NO_3^-] + [Cl^-] - [Na^+] - [NH_4^+] \quad (1)$$

$$pH = -\log(f[H_{aq}^+] \times x[H_{aq}^+]) \quad (2)$$

Where $f[H_{aq}^+]$ is the activity coefficient of H^+ in aqueous phase, $x[H_{aq}^+]$ is the molar fraction of H^+ in the aqueous phase.

3.2.7 QA/QC

Quality control (QC) and quality auditing establish the precision, accuracy, and validity of measured values. Quality assurance (QA) integrates quality control, quality auditing, measurement method validation, and sample validation into the measurement process.

1502 The results of QA are data values with specific precisions, accuracy, and validities. For
1503 QAQC, a blank filter cut to 0.522 cm² was analyzed for every 10 samples to ensure the
1504 stability of the DRI thermal carbon analyzer. The standard deviation was calculated to
1505 evaluate accuracy using a triple-replicate parallel analysis every 20 samples. For quality
1506 control of ICP-OES in trace metal analysis, standard reference material (NIST 1648a),
1507 filter blanks, and reagent blanks were treated with the same procedure and detection
1508 method as the samples. Replicates were applied to test the precision of the digestion
1509 and analysis. Subsequently, the TD-GC/MS for NPOCs analysis, the internal standards,
1510 and the organic standard mixture containing PAHs and n-alkanes were listed. No
1511 recovery correction was performed for concentrations determined using TD-GC/MS,
1512 as the samples were analyzed in the same manner as the calibration standards. A
1513 standard sample was added to the sequence every ten samples, and the blank filter cut
1514 to 2 cm² was analyzed per experimental day (five samples). For quality accuracy
1515 evaluation, each blank filter with an eight-μL standard mixture was analyzed three times
1516 to calculate the standard deviation.

3.3 Inhalation bioaccessibility extraction and assessment procedure

3.3.1 Bioaccessible extraction by SELF

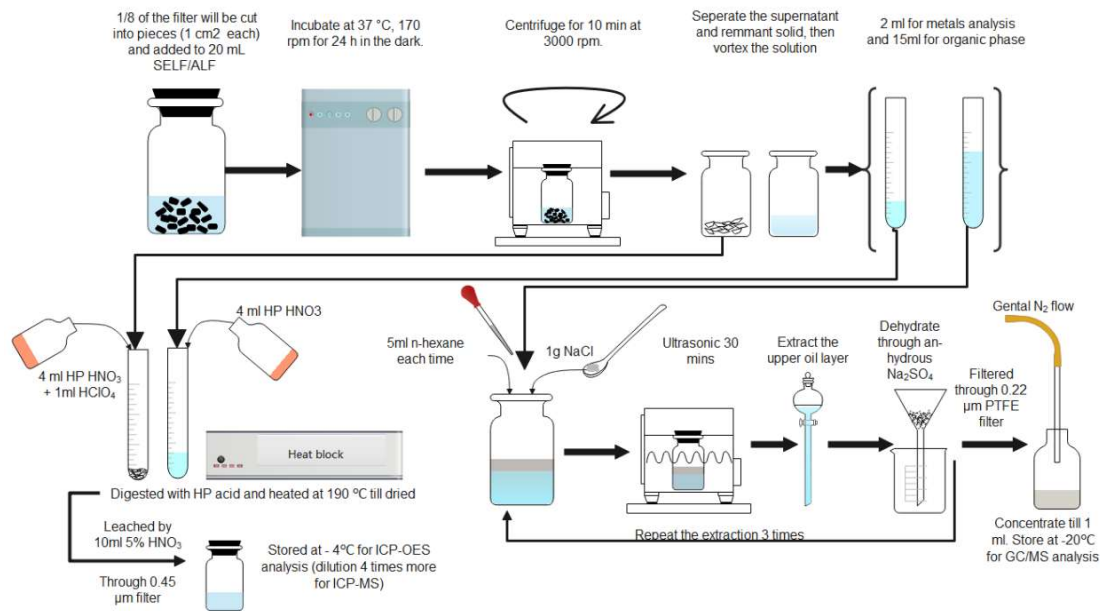


Figure 3-2 Bioaccessibility analysis procedure for key toxic components in PM_{2.5}

The average *in vitro* bioaccessibility was investigated by extracting the soluble portion of PM_{2.5} particles using SELF. The composition listed in **Table 3-4** and the preparation procedure followed are based on previous research (Dean et al., 2017; Y. Li et al., 2019; Zhou et al., 2022). L-Glutathione was purchased from J&K, and the surfactant 1,2-dipalmitoyl-glycerol-3-phosphocholine (DPPC) was obtained from *Oriental Chemicals and Lab Supplies* in Hong Kong. For each selected sample, 1/8 of the filter was cut into pieces (about 0.25 cm² for each piece) and added to 20 mL SELF (S/L ratio was comparable to a range of 1:500 to 1:5000 Julien et al., 2011), and then the mixture was shaken at 37°C and 100 rpm for 24 h in darkness. After that, the mixture was centrifuged for 10 min at 3000 rpm. When a clear boundary between precipitation and supernatant is observed, carefully remove the supernatant and filter the residual solids with a 0.45 µm syringe filter. The supernatant was considered the solution representing the total bioaccessible fraction extracted from the ambient PM_{2.5}. The residual solids together

with those remaining at the spring filter were collected as remnant lung-fluid-insoluble fractions.

Table 3-4 The compositions and concentrations of modified simulated epithelial lung fluid (pH=7.4)

No	Component	Concentration (mg/ L)
1	NaCl	6020
2	CaCl ₂	256
3	Na ₂ HPO ₄	150
4	NaHCO ₃	2700
5	KCl	298
6	MgCl ₂	200
7	Na ₂ SO ₄	72
8	Ascorbic acid	18
9	Uric acid	16
10	Glutathione (GSH)	30
11	Bovine serum albumin (BSA)	260
12	Cysteine	122
13	Dipalmitoyl phosphatidylcholine (DPPC)	100
14	Glycine	376
15	Mucin	500
16	Hyaluronic acid (HA)	200 or 400
17	NaOH or HCl for pH adjustment.	

3.3.2 Bioaccessibility analysis of PM_{2.5}-bound key toxic components

For the bioaccessibility analysis of metal elements, 2 mL of the supernate and the residual solid were digested with the addition of 1 mL of 60% high-purity HClO₄ and 4 mL of 70% HNO₃. Then, they were heated on a heating block according to the

program described above. After being heated until dry, the residual was leached with 5% high-purity HNO₃, following the method. The leachate was analyzed by ICP-MS. For quality control, standard reference matter (NIST 1648a), filter blank, and reagent blank were treated with samples using the same procedure in both the digestion of total/bioaccessible elements and the detection of their concentrations. Replicates are applied to test the precision of the digestion and analysis (RSD < 8%).

For the bioaccessibility analysis of PAHs, the supernatant was extracted three times with 5 mL of dichloromethane (DCM) for 30 minutes using ultrasonic agitation. Then, the combined extracts were dried over anhydrous sodium sulfate and filtered through 0.22 µm PTFE filters. After being concentrated by a high-purity N₂ gas flow, the residue was re-suspended in 1 mL of HPLC-grade n-hexane and stored at –20 °C before GC/MS analysis. The analysis was conducted by GC/MS equipped with an Agilent 6890 GC (Santa Clara, CA, USA) split injector line, and an HP-5ms column(Agilent 19091s-433, 30 m x 250 µm x 0.25 µm) (Ho et al., 2008). Detailed GC/MS setting information is listed in **Table 3-5**.

Table 3-5 Detailed Settings information for GC-MS

Setting	Step	Heating rate	Temperature	Retention
Temperature program	Column oven temperature		120 °C	2.0 min
	Heating process	15°C/min	310 °C	8.0 min
Ion-source			230 °C	

Injection port	280 °C
temperature	
Injection	4 µL

1558

1559 **3.3.3 Phase separation and reconstitution for toxicological exposure**

1560 To provide insights into the potential health impacts of different PM_{2.5} components, the
1561 cytotoxicity induced by the bioaccessible and insoluble organic and inorganic extracts
1562 was compared. As illustrated in **Fig. 3-3**, the extraction procedure was almost identical
1563 to the aforementioned SELF extraction process. 1/8 of the PM_{2.5} filter was cut into
1564 pieces, each measuring 1 cm², and added to 30 mL of SELF. This mixture is incubated
1565 at 37 °C with shaking at 170 rpm for 24 hours in the dark to simulate biological
1566 conditions. After incubation, the mixture is centrifuged for 10 minutes at 3000 rpm to
1567 separate the bioaccessible fraction from the remnant lung-fluid-insoluble fraction. The
1568 remnant lung-fluid-insoluble fraction undergoes further processing to extract different
1569 phases. It is treated with 1 g of NaCl and 5 mL of dichloromethane (DCM), then
1570 subjected to ultrasonic treatment for 30 minutes. This extraction process is repeated
1571 three times to ensure thorough separation. The resulting mixture is then separated into
1572 water and oil layers, yielding two fractions: the insoluble inorganic phase (Fraction 1)
1573 and the insoluble organic phase (Fraction 2). Simultaneously, the bioaccessible fraction
1574 is processed similarly with NaCl and DCM, followed by ultrasonic treatment and
1575 repeated extraction. This results in the separation of the bioaccessible organic phase
1576 (Fraction 3) and the bioaccessible inorganic phase (Fraction 4). All fractions are then
1577 freeze-dried or concentrated under gentle nitrogen flow until dry. The dried extracts are
1578 diluted with Dulbecco's Modified Eagle Medium (DMEM) and stored at -20 °C for

subsequent cell exposure.

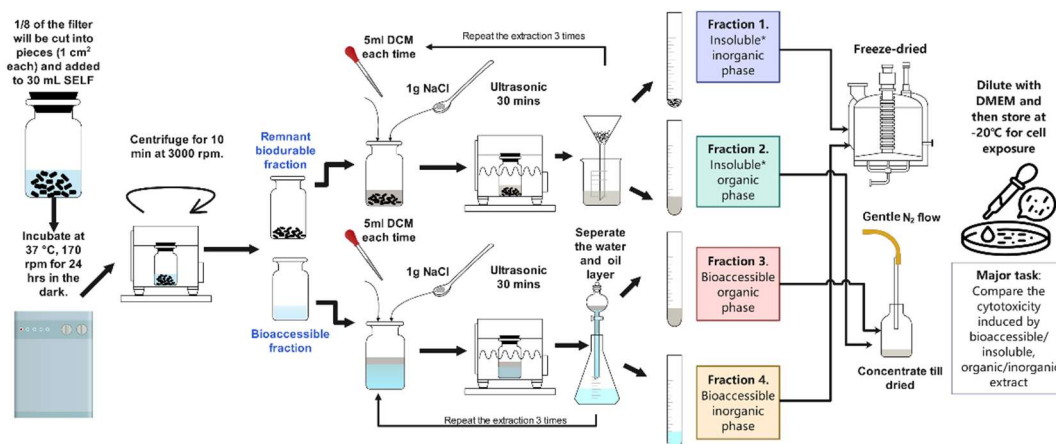


Figure 3-3 Cytotoxicity comparison between bioaccessible and lung-fluid-insoluble fractions of PM_{2.5}

Solvent selection was tailored to the analytical goal of each assay. n-hexane was used for GC-MS analysis to specifically target non-polar organic compounds (*e.g.*, PAHs, alkanes), which are poorly soluble in polar solvents—this ensures accurate quantification of these toxicants in the bioaccessible phase. In contrast, DCM was selected for cytotoxicity assays due to its broader solubility range (encompassing both polar and non-polar organics) and compatibility with cell culture media. This choice ensures that all bioaccessible organic species contributing to oxidative stress—including polar carbonyls and non-polar PAHs—are retained in the exposure matrix, enabling a holistic assessment of cytotoxicity. While the measured species differ slightly between solvents, both were optimized to address their respective analytical objectives without compromising methodological consistency.

3.4 Source apportionment

The EPA PMF receptor model 5.0.14 was employed for the source apportionment analysis of PM_{2.5} samples from three sites in Hong Kong in 2021 to identify possible emission sources of total PM_{2.5} and the key toxic components. The PMF model is

established based on the chemical mass balance equation **Eq. (3)** between composition species concentration (Brown et al., 2015; USEPA, 2015).

$$x_{ij} = \sum_{k=1}^p c_{ik} f_{kj} + r_{ij} \quad (3)$$

In which x_{ij} represents the concentration of species, c_{ik} is the source contribution of factor k to the sample i , f_{kj} is the emission source profile of species j in source factor k , while r_{ij} is the residual of each species. The solutions of c_{ik} and f_{kj} are computed under non-negative constraints and selected by obtaining the converged result with minimum Q value from the user-defined uncertainty of each sample u_{ij} .

$$Q = \sum_{i=1}^n \sum_{j=1}^m (e_{ij}/u_{ij}) \quad (4)$$

The input chemical concentration profile includes OC, EC, major water-soluble ions (Na^+ , NH_4^+ , K^+ , Mg^{2+} , Ca^{2+} , Cl^- , NO_3^- , SO_4^{2-}), trace metals (Al, As, Ba, Cd, Co, Cr, Cu, Fe, Mn, Mo, Ni, Pb, Sb, Se, Sn, V, Ni, Pb, Zn), hopanes (sum of C_{29} -ab-norhopane to C_{35} -22R-pentashomohopane), major toxic PAHs (Flu and Pyr summed as C_{16} -PAHs; CcdP, BaA, and Chr summed as C_{18} -PAHs; BbF, BkF, and BaP summed as C_{20} -PAHs; IcdP and BghiP summed as C_{20} -PAHs, DahA summed as C_{22} -PAHs), and total variable $\text{PM}_{2.5}$ concentrations (USEPA, 2015). The input concentration should be higher than the limit of detection (LOD) (**Table 3-3**) in this study; otherwise, it would be replaced by 1/2 LOD. If the concentration is higher than the LOD, the input uncertainties were calculated using **Eq. (5)**; otherwise, **Eq. (6)** was used for the uncertainty calculation. For OC, EC, water-soluble ions, and trace metals, the error fraction e was 0.100, while for hopanes and PAHs in roadside and general urban sites, the error fraction was estimated between 0.100 and 0.35 (Chow et al., 2022a; Xie et al., 2020).

$$Unc = \sqrt{(0.5 \times LOD)^2 + (e \times conc)^2} \quad (5)$$

$$Unc = \frac{5}{6} \times LOD \quad (6)$$

After removing the data that resulted in out-of-range points, the scaled residuals ranged from -3 to +3 (the percentage of the valid data was >90%). The ratio of Q(robust) to Q(true) was close to 1.00 (<1.10). For most species, the slope and correlation coefficient (R^2) between the predicted and input data were 0.82–1.16 and 0.80–0.99, respectively, indicating a good fit for the model.

The criteria included the interpretability of output profiles from the base model and error estimation profiles, including Bootstrap (BS) summary and Bootstrap-Displacement (BS-DISP) results.

3.5 Back trajectory analysis

Current studies have shown that (1) local emission sources usually contribute to air pollution with negligible seasonal variation (Bodor et al., 2020; Shivani et al., 2019; X. Zhang et al., 2022), while (2) regional sources subject to monsoonal transport and meteorological conditions can vary seasonally in contributions to air pollutants (Im et al., 2018). This study employed two indicators to distinguish between local-dominant and regional-dominant sources, examined monthly variations in source profiles, and analyzed source-resolved back-trajectory air mass clusters.

To evaluate the regional effects on the composition of $PM_{2.5}$ sources, an air mass back-trajectory analysis was conducted using the HYSPLIT model with meteorological data from the Global Data Assimilation System (GDAS) (Draxler & Hess, 1998; Stein et al., 2015). According to the back-trajectory analysis, the monsoon transitioned from a southwestern oceanic flow to a northeastern mixed continental wind pattern in mid-October 2021 in Hong Kong. Therefore, the back trajectory clusters were computed from July 1st to September 30th for the warm season and from November 1st to January

30th for the cold season, based on 24-hour back trajectories at 100m above ground level (AGL), 22°18'N, 114°10'E in Kowloon, Hong Kong. The 24-hour duration for the back-trajectory was based on the sampling period of the PM_{2.5} measurements, which were collected over 24-hour intervals. The 100m AGL was selected to represent the atmospheric conditions near the surface, where PM_{2.5} is primarily influenced by local and regional sources, widely used in current studies (Cui et al., 2021; Kassomenos et al., 2010; Midander et al., 2010). The mass contribution of each regional source to each air mass cluster was calculated and matched to the source apportionment result for the corresponding 24-hour PM_{2.5} sample, which was output by the PMF model. The regional effect on PM_{2.5} sources was also investigated using back-trajectory cluster analysis, with profiles outputted from HYSPLIT (Stein et al., 2015).

3.6 Chronic health risk assessment

3.6.1 Chronic health risk assessment of critical toxic components

To explore the health risk associated with trace metals, a health risk assessment method following USEPA guidelines can be conducted to calculate both cancer risk and non-cancer risk. There are three primary pathways for human exposure to airborne pollutants: inhalation, ingestion, and absorption through the skin (USEPA, 2007). Based on the Integrated Risk Information System (IRIS) database, several trace metals, including As, Cr (VI), Cd, Ni, and Pb, have been identified as carcinogens or allergens to the human body, and were also selected for evaluation of the health risk associated with trace metal exposure in this study. Besides, several other major elements, such as Mn, Co, Fe, Cu, V, and Zn, are also involved in trace metal-induced non-carcinogenic risk. The present study focused on assessing the CR posed by As, Cd, Co, Cr, Ni, and Pb. In contrast, the potential cancer risk and non-carcinogenic potency of the remaining elements detected were not investigated. Therefore, the CR posed by airborne trace metals via inhalation (CR_i) was calculated as in **Eq. (7)**. The non-carcinogenic risks

indicated by Hazard Quotients (HQ_i) were calculated via **Eq. (8)** (USEPA, 1994, 2009):

$$CR_i = \frac{C_i \times ET \times EF \times ED \times IUR}{AT} \quad (7)$$

$$HQ_i = \frac{C_i \times ET \times EF \times ED}{AT \times RfC_i \times 1000 \mu g/mg} \quad (8)$$

Where: C_i ($\mu g/m^3$) is the concentration of trace metal; ET, EF, and ED represent exposure time (hours/day), frequency (days/year), and duration (years), respectively; AT is the averaging time which means the total number of hours in a lifetime when calculating CR but is only considered as the hour numbers in the exposure duration while calculating HQ. IUR means Inhalation Unit Risk ($\mu g/m^3$)⁻¹, and RfC_i refers to reference concentration (mg/m^3) via inhalation (USEPA, 2009). This study used diesel exhaust particulate as the surrogate to investigate the health risk of EC (Hua et al., 2024; Lin et al., 2019). The exposure health risk assessment for EC was estimated based on chronic daily intake ($mg\ kg^{-1}\ day^{-1}$) (USEPA, 2009). The cancer and non-cancer risks were estimated using **Eqs. (7) and (8)**, where RfC_{BC} and IUR_{BC} were $5.0E-3\ mg/m^3$ and $3.0E-4\ (\mu g/m^3)^{-1}$, respectively (Lin et al., 2019; OEHHA, 1998).

The modified factor (MF) for arsenic risk assessment based on its speciation distribution was applied as shown in **Eq.(9)** following (Xie et al., 2020). The total exposure duration is set as 30 years for adults. The values of other parameters are shown in **Table 3-8**.

$$MF_{As} = \frac{R_{cur,As(V)} * TR_{As(V)/As(III)} + R_{cur,As(III)}}{R_{ori,As(V)} * TR_{As(V)/As(III)} + R_{ori,As(III)}} \quad (9)$$

For another key toxic component, PAHs, the BaP-equivalent potential developed by the Office of Environmental Health Hazard Assessment (OEHHA) of the California Environmental Protection Agency (CalEPA) was used to integrate the carcinogenic risk of PAH species in $PM_{2.5}$ (Khairy & Lohmann, 2013; OEHHA, 2005). The BaP-

equivalent (BaP_{eq}) concentration of the individual PAHs was calculated by multiplying their concentration by their relative potency factor (RPF_i).

$$BaP_{eq} conc = \sum_{i=1}^n C_k \times RPF_k \quad (10)$$

Where C_k is the concentration of each PAH species, and RPF_k is each PAH's corresponding toxic equivalent factor. Seventeen carcinogenic PAHs were included in the estimate of inhalation cancer risk, including Phe, Ant, Flu, Pyr, CcdP, BaA, Chr, BbF, BkF, BaF, BaP, IcdP, BghiP, DahA, and DacA. Other measured PAHs were excluded from the estimate of inhalation cancer risk because no RPF values were available for these PAHs. RPF values based on tumor bioassay data from the U.S. EPA's Integrated Risk Information System Program were used. The individual PAHs and their respective RPF values are listed in **Table 3-8**. The incremental lifetime cancer risk (ILCR) via inhalation was used to estimate the exposure risk of airborne PAHs by **Eqs. (11) - (13)**:

$$CDI_k = \frac{BaP_{eq} conc_k \times ET \times EF \times ED \times InhR}{BW \times AT} \quad (11)$$

$$CR_k = CDI_k \times CSF_{inh} \quad (12)$$

$$(k = Flu, CcdP, BaA, Chr, BbF, BkF, BaF, BaP, IcdP, BghiP, DahA, DacA)$$

$$HQ_k = \frac{CDI_k}{RfD_k} \quad (k = Ant, Flu, Pyr, BaP) \quad (13)$$

Where ET means the average exposure time (8h/day), EF is the exposure frequency (350 days/year), AT is the average lifetime (70 years), ED is the exposure duration which is 30 years for adults, InhR is inhalation rate (20 m³ per day), BW is the body weight, SA is the skin area including feet, hands, lower legs and forearms, CF is the correction factor, AF_d is the particle-to-skin adherence factor (Iakovides et al., 2021). All parameters and the RPF_i for each PAH used in the health risk calculation are

1716 listed in **Table 3-6**. According to the latest USEPA guidelines, the acceptable chronic
1717 non-cancer risk threshold is an HQ of 1.0, and the upper limit of CR is 1E-6 (USEPA,
1718 2005, 2009, 2024).

1719

Table 3-6 Parameters are used in the calculation of carcinogenic risks and hazard quotients in this study.

Parameter	Adult	Element	RfCi ^a (mg/m ³)	IUR ^a (µg/m ³) ⁻¹	PAH	RPF _i	RfD (mg/kg/day)
ED (years)	20	V	1.00E-04	n.a.	Phe	0	n.a.
ET (hours day ⁻¹)	8	Mn	5.00E-05	n.a.	Ant	0	3.00E-01
EF (days year ⁻¹)	350	Co	6.00E-06	9.00E-03	Flu	0.08	4.00E-02
AT-non-carcinogen s	175200 hrs	Ni	9.00E-05	2.60E-04 ^b	Pyr	0	3.00E-02
	20 years	As	1.50E-05	4.30E-03	CcdP	0.4	n.a.
AT-carcinogen s	613200 hrs	Cd	1.00E-05	1.80E-03 ^c	BaA	0.2	n.a.
	70 years	Pb	5.00E-04 ^c	1.20E-05 ^d	Chr	0.1	n.a.
AT-carcinogen s (days)	25500	Cr (VI)	1.00E-04	8.40E-02	BbF	0.8	n.a.
BW (kg)	70	Fe	5.00E+00 ^e _f	n.a.	BkF	0.03	n.a.
CF (mg-days/pg-h)	0.000001	Cu	1.00E+00 ^e	n.a.	BaF	0.2	n.a.
InhR (m ³ /day)	20	Zn	5.00E-01 ^e	n.a.	BaP	1	3.00E-04
CSF _{inh} (per mg/kg-day)	3.9	Diesel exhaust particulate	5.00E-03 ^h	0.0003 ^h	IcdP	0.07	n.a.
					BghiP	0.009	n.a.
					DahA	10	n.a.
					DacA	4	n.a.

a. IUR and RfCi obtained from the USEPA website (USEPA, 2018)

b. for nickel soluble salts

c. for cadmium (Diet/Water)

d. for lead phosphate/acetate/subacetate

e. benchmark value obtained from other standards (Oosthuizen et al., 2015)

f. Knafla et al., 2006; OEHHA, 2011

g. Khairy and Lohmann, 2013; Nguyen et al., 2020; Iakovides et al., 2021

3.6.2 Source-resolved health risk

Based on the source apportionment results for each site, the contribution of each source to the main toxic component-induced health risk can be quantified by linking mass concentration contributions to trace metal- and PAH-related carcinogenic and noncarcinogenic health risks (Xie et al., 2020). The relative contribution (RC) of a certain source to individual metals and PAHs at each site can be obtained from the output source profile of the PMF model. As the noncarcinogenic risk of PAHs could not be quantified directly, only trace metals were involved in the source-resolved noncarcinogenic risk calculation based on Eq. (14). And for the source-resolved carcinogenic risk, the cancer risk via inhalation of airborne PAHs and especially toxic trace metals were summated to compare the total contribution of each certain source to the major toxic compounds (trace metals and PAHs) related carcinogenic risk according to Eq. (15).

$$HQ_{s,l} = \sum_i (HQ_{i,l} \times RC_{i,s,l}) \quad (i = \text{As, Cd, Co, Cr, Cu, Fe, Mn, Ni, Pb, V, Zn}) \quad (14)$$

$$CR_{s,l} = \sum_i (CR_{i,l} \times RC_{i,s,l}) + \sum_j (CR_{j,l} \times RC_{j,s,l})$$

$$(i = \text{Co, Ni, As, Cd, and Pb}; j = \text{FLA, CcdP, BaA, Chr, BbF, BaF, BaP, DahA}) \quad (15)$$

Where s is emission sources and l represent sampling locations (RD, 6F and Hok Tsui in Hong Kong). $HQ_{i,l}$ is the non-carcinogenic risk induced by each metal (loid) in a certain sampling location, and $CR_{i,l}$ the inhalation carcinogenic risk caused by selected trace metals and toxic PAHs.

3.7 Cytotoxicity

3.7.1. Component-specific cytotoxicity

To compare the *in vitro* cytotoxicity induced by roadside PM_{2.5} with that of general urban and coastal background samples, and to investigate the component-specific contribution to total PM_{2.5}, cell viability and intracellular ROS were analyzed using the BEAS-2B cell line. The BEAS-2B cell line is a widely used human bronchial epithelial cell model, immortalized using the SV40 large T-antigen hybrid virus (Reddel et al., 1988). This immortalization enables the cells to be useful for *in vitro* studies, although a limitation remains that the biological effects observed with immortalized cells do not fully mimic those of normal human bronchial cells (Courcot et al., 2012). The BEAS-2B cell line was acquired from the American Type Culture Collection (ATCC) and cultured in a humidified atmosphere with 5% CO₂ at 37 °C using DMEM supplemented with 10% heat-inactivated fetal bovine serum and 1% penicillin-streptomycin antibiotics.

To extract PM_{2.5}, a 52 cm² filter sample was ultrasonically extracted with 15 mL of MilliQ water and then with 15 mL of methanol for 30 minutes, separately. After the water-extracted solution was freeze-dried and the methanol-extracted solution was dried using gentle nitrogen gas, the remaining solids were reconstituted in DMEM at a concentration of 1000 µg/mL and then mixed in a 1:1 volume ratio. The volume of sterile DMEM (supplemented with 10% fetal bovine serum and 1% penicillin-streptomycin) was then calculated to reconstitute the sample into a 1000 mg/L stock solution.

Firstly, cell viability analysis was conducted on the samples after 24-hour exposure to the treatment solution using the MTS (ab197010) assay, in which the concentrations of treatment components required to inhibit 50.0% of cell viability (IC₅₀) were determined.

Then, the exposure concentration gradient was designed for the ROS analysis under the IC_{50} , ensuring that the cell apoptosis did not generate further ROS, leading to bias in the obtained results. The cells were seeded at a density of 2×10^5 cells mL^{-1} in black 96-well plates and allowed to grow to confluence for 24 hours. Following the removal of the medium, the cells were washed with PBS and then exposed to 100 μL of $PM_{2.5}$ -extracted solution or serially diluted test chemical standards in the medium.

To investigate ROS induced by individual key toxic compounds, the trace metal chloride standards, selected PAHs, and Printex U carbon black were exposed to the cell line using the same procedure described above. Ultrafine carbon blacks, such as Printex U carbon black, which represent artificial, pure-elemental-carbon particles, are widely used as surrogates for EC (Chen et al., 2024; Li et al., 2013; Niranjana & Thakur, 2017; Shang et al., 2022). Dimethyl sulfoxide (DMSO) was used as a hydrotrope for the stock solution before cell exposure. Tert-butyl hydroperoxide (t-bhp), a well-known inducer of intracellular ROS, was included as a positive reference in each plate (Brown et al., 2007; Kucera et al., 2014; Zhao et al., 2017). After 24 hours of exposure, the treatment medium was replaced with 100 μL of phenol red-free DMEM containing 100 μM DCFH-DA. The cells were incubated for 40 minutes at 37 $^{\circ}C$, followed by two washes with PBS. Fluorescence intensity was measured at 0, 2, and 4 hours using an automated microplate reader with excitation/emission wavelengths of 485/535 nm, respectively. ROS production was quantified as the percentage increase in fluorescence intensity from 0 to 2 hours and from 0 to 4 hours. The ROS induction ratio (IR) of the sample compared with the control was determined using **Eq. (16)**, and the effect concentration at a ROS induction ratio of 1.5 was calculated using linear concentration–effect curves with an intercept of 1 and fitted slope **Eqs. (16) and (18)**.

$$IR = \frac{\%increase\ sample\ t=2}{\%increase\ control\ t=2} \quad (16)$$

$$IR = 1 + slope \times concentration \quad (17)$$

$$EC_{IR1.5} = \frac{0.5}{slope} \quad (18)$$

The chemicals standards used in cell treatment include individual chloride of Cd(II), Cu(II), Mn(II), Ni(II), Pb(II), and Zn(II) from *Shenzhen Xinnake DYF*, As(V), Cr(III), Fe(III), and V(V) from *Wabcan*, Printex U (25 nm) carbon black produced by *Orion*, DMSO from *Sigma-Aldrich*. The pure solid individual PAH standards analyzed in this study consisted of Fla and BbF from *AccuStandard*, BaF and BkF from *Bepure*, Chr, BaP and DahA from *Aladdin*, and DalP from *Supelco*.

3.7.2 Mixture cytotoxicity model

The additive ROS induction potencies of metals, EC, and major toxic parent PAHs were mixed at the molar concentration ratio corresponding to the actual samples. Following the combined toxicity model (Escher et al., 2013), the deviation between experimental $EC_{IR1.5}$ of the artificial mixture following their corresponding molar ratios in the real sample ($EC_{IR1.5_EP}$) and the CA-model predicted result ($EC_{IR1.5_CA}$) was assessed (Escher et al., 2013; Jin et al., 2019). In which, the toxic equivalent factors of individual compounds (TEQ_i) and extracted $PM_{2.5}$ samples ($TEQ_{PM_{2.5}}$) against that of t-bhp as the reference were calculated based on **Eqs. (19) and (20)**. Then, the fractional contributions of the analyzed components to the effect induced by overall $PM_{2.5}$ were quantified in relation to those of t-bhp using **Eq. (21)**.

$$TEQ_i = \frac{EC_{IR1.5_t-bhp}}{EC_{IR_{1.5_i}}} \quad (19)$$

$$TEQ_{PM_{2.5}} = \frac{EC_{IR_{1.5_t-bhp}}}{EC_{IR_{1.5_PM_{2.5}}}} \quad (20)$$

$$Fractional\ contribution_i = \frac{C_i \times TEQ_i}{TEQ_{PM_{2.5}}} \quad (21)$$

The index of prediction quality (IPQ) is regarded as a quality assurance indicator for the CA-model, calculated from the comparison between prediction toxic effect ($EC_{IR.5_CA}$) and the experimental result of the artificial mixture with known molar concentrations ($EC_{IR1.5_EP}$), where mf_i is molar fraction of compound i in the mixture (Eqs. 22-24). The IPQ within -1.00 to 1.00 indicates a satisfactory agreement between experiments and predictions (Escher et al., 2020). For combined biological effect, the challenges have been reported in many studies that after mixing individual toxic compounds such as trace metals, PAHs, or carbon black, the synergistic or antagonistic interactions lead to a more complicated mixture toxicity mechanism (Guo et al., 2009; Lin & Yu, 2019b; Yu et al., 2018). The non-linear dose-effect curve for the organic-metal mixture using the acellular DTT assay was also reported (Lin & Yu, 2019b). However, such combined interactions (*i.e.*, synergistic or antagonistic) may decrease with lower exposure concentrations and more individual components in the mixture, summarized as the “Funnel Hypothesis” (Jin et al., 2019; Warne & Hawker, 1995). This study considered the negligible effect of synergism or antagonism between compounds on overall toxicity.

$$EC_{IR1.5_CA} = 1 / \sum_{i=1}^n \frac{mf_i}{EC_{IR1.5_i}} \quad (22)$$

$$\text{If } EC_{IR1.5_CA} > EC_{IR1.5_EP} \text{ then } IPQ = \frac{EC_{IR.5_CA}}{EC_{IR.15_EP}} - 1 \quad (23)$$

$$\text{If } EC_{IR1.5_CA} < EC_{IR.5_EP} \text{ then } IPQ = 1 - \frac{EC_{IR.5_CA}}{EC_{IR.15_EP}} \quad (24)$$

We conducted a Deviation Ratio (DR) analysis of our experimental data to complement the CA model. This DR assessment evaluates the suitability of both approaches for capturing complex interactions. Following a comprehensive literature review, we adopted the DR method, first proposed by Belden et al. (2007), to our knowledge. Five approaches exist for calculating DR values: (1) using the interactivity or synergistic

ratio directly as DR; (2) deriving DR from experimental and predicted effective concentrations; (3) calculating DR from toxic unit (TU) values, where the reciprocal of TU equals DR; (4) employing the additive index (AI) method for paired compounds (Marking & Mauck, 1975); and (5) computing DR using the isobologram approach or other methods providing effective concentration values for individual toxicants and mixtures. A DR value greater than 1 suggests synergism, while a value less than 1 indicates antagonism (Macacu, 2020; Pape-Lindstrom & Lydy, 1997). However, there are different definitions without clear, uniform guidelines for the additive threshold of DR. For example, Belden et al. (2007) and Cedergreen (2014) defined the same reasonable range [0.5, 2] of DR. While a recent meta-analysis of mixture toxicity research used [0.8, 1.2] (Carnesecchi et al., 2019). Based on our experimental data, we applied approach 2 to calculate DR values, alongside IPQ as a quality assurance metric for the CA model (Eqs. 23 & 24).

3.7.3. Source-resolved cytotoxicity

After quantifying the contribution of each identified critical component to the extracted PM_{2.5}-induced intracellular ROS generation (Escher et al., 2020; Escher et al., 2013, 2020; Jin et al., n.d.), Eq. (25) was used to calculate the fractional source-resolved cytotoxicity (*FSC*) connecting the source apportionment profiles to cytotoxicity to serve as specific cell-based evidence for a more complete health assessment model, namely, the contribution of individual sources to the critical components related to ROS induction.

$$FSC_{s,l} = \frac{\sum_i (ER_{i,l} \times RC_{i,s,l})}{\sum_s \sum_i (ER_{i,l} \times RC_{i,s,l})} \quad (25)$$

Where *i* represents the identified toxic compound, *l* and *s* are the mean sampling location and source category, respectively, *RC*_{*i,s,l*} is the relative contribution of a specific source, and *ER*_{*i,l*} means the contribution of individual elements to the total

1870 toxic component-induced ROS.

1871 **3.8 Statistical method**

1872 The linear regression, mean value, and standard deviation were calculated via GraphPad
1873 Prism (Version 9.0). One-way ANOVA with Tukey's multiple comparisons test was
1874 performed to assess statistical significance between groups: (*) $P < 0.05$, (**) $P < 0.01$,
1875 and (***) $P < 0.001$. The Spearman correlation coefficient was calculated in R using
1876 the Hmisc package. Partial Least Squares Discriminant Analysis (PLS-DA), a
1877 supervised multivariate technique that combines partial least squares regression with
1878 discriminant analysis to classify samples and identify variables driving separation, was
1879 conducted by Metabo Analyst 6.0.

1880

4 Component-specific Health Impact of PM_{2.5} in Hong Kong

This chapter aims to identify which specific components of PM_{2.5} are most responsible for acute cytotoxicity and chronic health risks, and to compare their toxic potencies in driving adverse health outcomes.

4.1 Current research and data summary in the typical megacity in the Greater Bay Area

The Hong Kong Environmental Protection Department (HKEPD) began examining the chemical components of PM_{2.5} in 2000, as noted in a 2005 study by Louie et al. They currently operate five monitoring sites, including comparisons among general urban, suburban, and roadside sites. While PM_{2.5} mass is used to assess compliance with air quality objectives, analyzing its chemical composition helps identify the sources of elevated levels. By maintaining a long-term record of these compositions, it becomes possible to track trends in response to strategies implemented for reducing emissions. This report focuses on the measurement and validation of PM_{2.5} data from January to December 2021. Information from previous years can be found in the HKEPD's 2022 reports. According to the Hong Kong PM_{2.5} Chemical Composition reports from the HKEPD, the annual PM_{2.5} concentration has decreased continuously over the past decade. The sampling site of HKEPD is shown in **Fig. 4-1**. Compared with other sites, the Mong Kok (MK) site in Kowloon is the one most affected by traffic emissions due to its heavy traffic load.

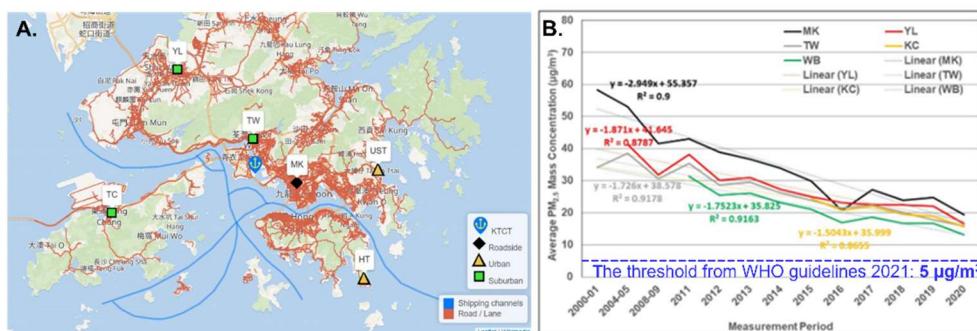


Figure 4-1 Sampling sites and the measurement result of PM_{2.5} in the past decades in Hong Kong. A) Monitoring sites in the Hong Kong PM_{2.5} speciation network (W. S. Chow, Huang, et al., 2022). B) Trends in PM_{2.5} annual averages at the five speciation sites (HKEPD, 2022).

From 2010 to 2020, the annual average PM_{2.5} concentration of all six sites decreased, as shown in **Fig. 4-1B**. However, it is worth noting that the PM_{2.5} concentration in MK, the typical urban roadside site, is always the highest, which decreased from 59.0 µg/m³ in 2000 to 19.5 µg/m³ in 2020, followed by the urban sites Tsuen Wan (TW) and Yuen Long (YL) and the harborside urban site Kwai Chung (KC). Clearwater Bay (WB), located on the HKUST campus, was considered the suburban site with the lowest PM_{2.5} levels. Despite the general decreasing trend over the past two decades, the average PM_{2.5} level declined in 2008 and 2016, which may be attributed to the launch of environmental policies in 2008, including the limitation on low-sulfur diesel, and the significant rainfall brought by the powerful El Niño in 2016 (Chow et al., 2022b).

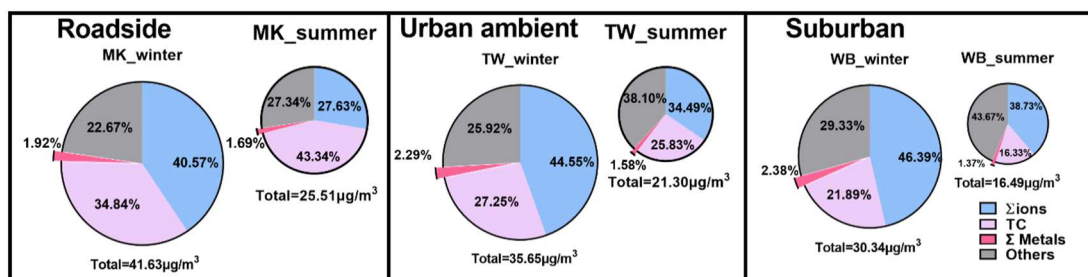


Figure 4-2 The decade average major chemical composition of PM_{2.5} in roadside,

1917 urban, and suburban sites in Hong Kong (the original data provided by HKEPD).

1918

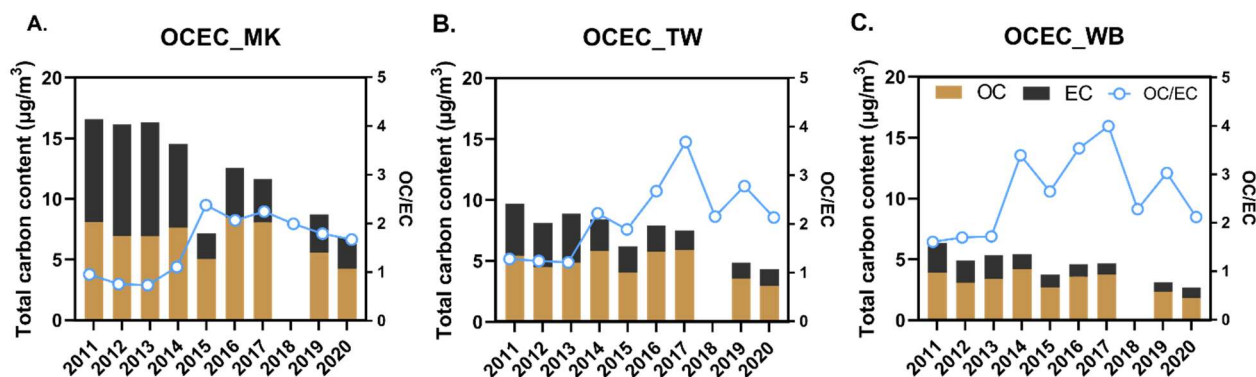
1919 **Table 4-1** Major chemical composition of PM_{2.5} (µg/m³) in MK, TW, and WB in the
1920 past decade (original data provided by HKEPD)

Year	Site	PM _{2.5}	Na ⁺	NH ₄	K ⁺	Cl ⁻	NO ₃	SO ₄ ²⁻	OC	EC	Elements
2011	MK	47.92	0.43	4.37	0.47	0.21	2.45	10.91	8.09	8.48	5.00
	TW	40.56	0.40	4.38	0.49	0.12	1.80	10.91	5.44	4.24	4.96
	WB	35.89	0.51	4.09	0.48	0.11	0.93	11.13	3.91	2.43	5.02
2012	MK	57.38	0.37	3.62	0.34	0.11	1.32	9.87	6.94	9.21	4.64
	TW	48.12	0.48	3.34	0.31	0.10	1.06	9.45	4.50	3.62	4.32
	WB	45.00	0.45	3.09	0.28	0.07	0.52	9.27	3.07	1.81	4.25
2013	MK	41.38	0.33	3.42	0.32	0.12	1.31	9.14	6.92	9.42	4.33
	TW	33.97	0.29	3.36	0.31	0.10	1.17	8.88	4.86	4.01	4.17
	WB	30.38	0.40	3.16	0.32	0.09	0.58	9.25	3.37	1.96	4.22
2014	MK	35.99	0.42	3.28	0.29	0.20	1.53	8.60	7.64	6.91	4.08
	TW	28.38	0.39	3.01	0.29	0.14	0.90	8.57	5.80	2.61	4.28
	WB	22.82	0.47	2.71	0.25	0.12	0.41	8.13	4.19	1.24	3.94
2015	MK	30.17	n.a	1.06	n.a	n.a	n.a	5.65	5.03	2.12	3.53
	TW	26.34	0.38	2.63	0.19	0.13	1.01	7.45	4.05	2.15	3.41
	WB	22.37	0.45	2.40	0.18	0.08	0.53	7.45	2.71	1.02	3.39
2016	MK	27.95	0.33	2.51	0.22	0.11	1.08	6.53	8.45	4.10	3.35
	TW	22.79	0.31	2.43	0.22	0.08	0.65	6.71	5.75	2.15	3.24
	WB	17.34	0.21	2.19	0.20	0.03	0.34	6.09	3.59	1.01	2.98
2017	MK	29.79	0.44	2.45	0.24	0.19	1.36	6.36	8.07	3.59	3.51
	TW	24.75	0.37	2.41	0.23	0.11	0.99	6.60	5.90	1.60	3.36
	WB	19.32	0.38	2.12	0.21	0.07	0.47	6.12	3.73	0.93	3.11
2018	MK	25.42	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.
	TW	22.11	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.
	WB	16.79	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.
2019	MK	27.88	0.36	2.25	0.22	0.14	1.43	5.52	5.59	3.12	3.14
	TW	20.73	0.33	1.99	0.17	0.07	0.86	5.07	3.57	1.28	2.62
	WB	19.02	0.32	1.94	0.19	0.07	0.63	5.20	2.35	0.78	2.74
	MK	18.95	0.34	1.40	0.11	0.16	1.18	4.05	4.24	2.54	2.04
	TW	18.01	0.32	1.22	0.09	0.09	0.77	3.82	2.94	1.38	1.90

2020	WB	13.20	0.31	1.19	0.09	0.09	0.61	3.74	1.83	0.86	1.77
------	----	-------	------	------	------	------	------	------	------	------	------

1921

1922 Regarding variation in chemical composition throughout the year, the total PM_{2.5}
1923 concentration was lowest in summer and peaked in winter. We gathered PM_{2.5}-loading
1924 chemical composition data for the MK, TW, and WB sites—representing roadside,
1925 urban, and suburban sites—from HKEPD for further analysis and comparison. At all
1926 three sites, the water-soluble ions were the primary contributors to total PM_{2.5} in winter.
1927 In contrast, during the summer, the higher fractional content of TC indicated less
1928 secondary aerosol generation, with local traffic emissions taking precedence. Compared
1929 with winter PM_{2.5} speciation, water-soluble ions and metals contributed less to summer
1930 PM_{2.5} at all three sites, especially in urban and suburban regions; the seasonal variation
1931 was significantly greater than at the roadside site. In detail, there were similar seasonal
1932 trends in sulfate aerosol and organic compounds with total PM_{2.5} concentration (**Fig. 4-**
1933 **2**).



1934 **Figure 4-3** The carbonaceous composition of PM_{2.5} in MK, TW, and WB in the past
1935 decade (the original data provided by HKEPD)

1936 Comparing the chemical composition analyses in **Fig. 4-1C** and **Fig. 4-3**, the
1937 component with the most significant difference is TC, especially the element carbon
1938 (also known as black carbon). Moreover, black carbon remained at a high level at MK
1939 throughout the year, indicating that it may originate from tailpipe emissions associated

with stable, high-density traffic. The OC/EC ratio in MK PM_{2.5} ranged from 0.7 to 2.2 over the past decade, suggesting that carbonaceous species were mainly generated by primary sources. Therefore, investigating the black carbon-induced health risks and the combined interactions with other toxic compounds may become increasingly important for enhancing understanding of megacity urban air pollution. Subsequent to the TW and WB sites, the variations of TC, OC, EC, and OC/EC ratio were quite similar, with the lower EC and higher OC/EC ratio in the past decade, which were also reasonable since the regional effect and second organic aerosol generation contributed more than local primary sources in the ambient sites (Wang et al., 2021). Moreover, the peaks in the OC/EC ratio observed in 2014 and 2017 at TW and WB indicated a higher contribution of secondary organic aerosol to ambient PM_{2.5} loading (Turpin & Huntzicker, 1995).

Focusing on key toxic components in HK PM_{2.5}—trace metals and PAHs, the major identified harmful groups of compounds—comparisons were made over the past decade based on analyzed data from HKEPD and literature data from previous studies. As illustrated in **Fig. 4-4**, the concentration of total metal per milligram of PM_{2.5} varied similarly across the three roadside sites, including urban and suburban areas, with a slump in 2012 and 2015, and peaks in 2014 and 2017. Considering local emissions, the dominant influencing factor may be policy adjustments to vehicle and vessel emission controls, such as tightening sulfur content in locally supplied diesel to 0.05% since 2014 and strengthening emission controls for petrol use since September 2014 (Chow et al., 2022b). While the total metal content per milligram of PM_{2.5} decreased slightly in 2019 and 2020, this decrease may be attributed not only to air pollution control policies but also to reduced regional transportation during the COVID-19 lockdown in mainland China, which began at the end of 2019 (Wang et al., 2022; Wen et al., 2022). When comparing the three sites, the fractional concentration of metals, ranging from 15 to 21 micrograms per milligram, was higher in roadside PM_{2.5} compared with other ambient samples in most years, except in 2014. Additionally, there were significant differences

in the composition of individual metals between roadside PM_{2.5} and ambient samples. In roadside samples, Fe, Zn, and Al were the top three contributors to the total metal content in PM_{2.5}. The presence of Fe and Zn can be attributed to non-exhaust emissions related to heavy traffic, specifically from road tire and brake friction. The similarity in the fractional composition of individual metals in urban and suburban PM_{2.5} suggests that they may be dominated by the same sources or regional transportation patterns. Furthermore, the presence of Ba, Mn, and Cu indicates the input of materials from the northern continental crust and industrial emissions. Regarding inhalation health concerns, attention should be given to V and As in roadside PM_{2.5}, as well as to Cr in urban and suburban PM_{2.5} due to their carcinogenic properties. It is worth noting that the fractional content of Pb has consistently decreased over the past decade, which can be attributed to the prohibition of leaded petrol and increased emission standards for vehicles and vessels in the GBA. However, there remains a lack of effective control measures for Ni and V, which are non-carcinogenic but still harmful metals, primarily generated by shipping emissions and the heavy oil industry. Particularly in the post-pandemic era, the recovery of port economies highlights the contribution of shipping emissions to inhalation health risks for the people of Hong Kong.

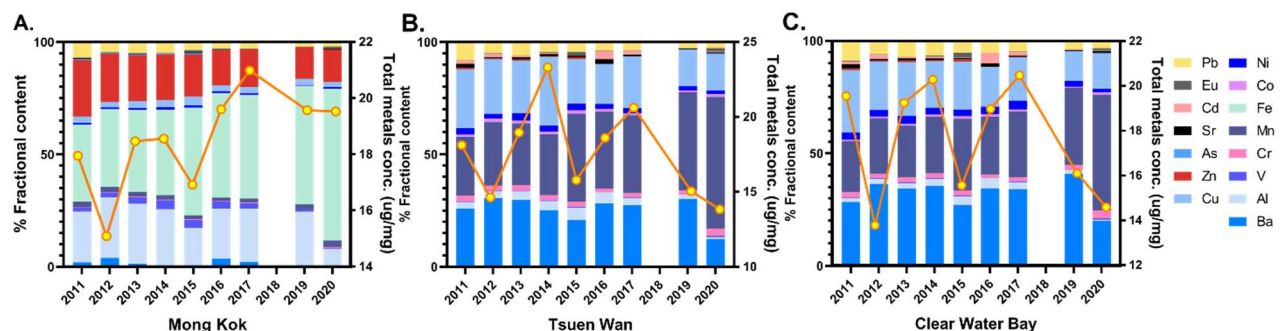


Figure 4-4 The PM_{2.5}-loading trace metal composition at MK, TW, and WB in the past decade

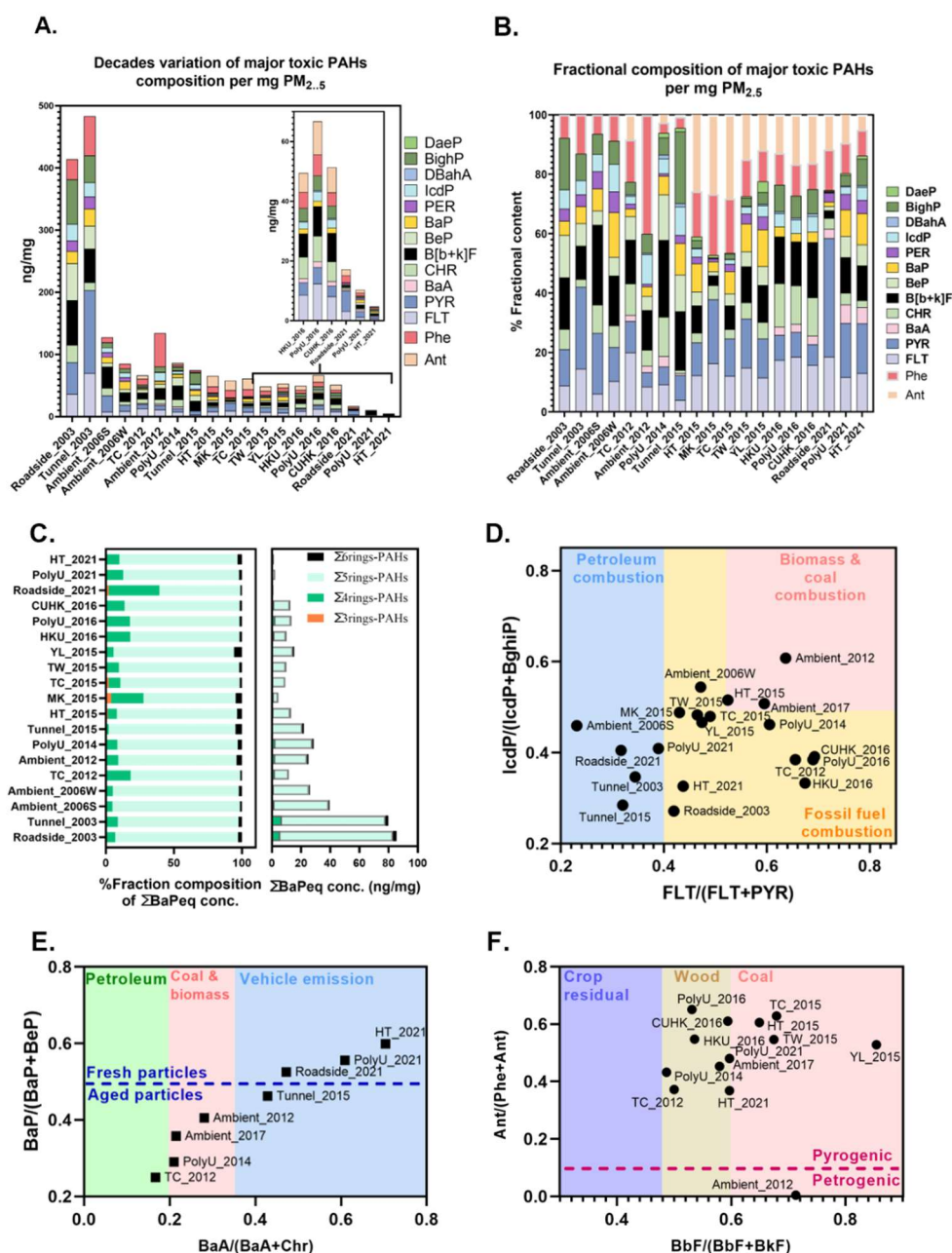
1987 **Table 4-2** The reference list for PAHs composition data summary in the past decade
1988 in HK PM_{2.5}

Title	Sample period	Sample sites	Σ PAHs
A quantitative assessment of source contributions to fine particulate matter (PM _{2.5})-bound polycyclic aromatic hydrocarbons (PAHs) and their nitrated and hydroxylated derivatives in Hong Kong. EP, 2016	2011 - 2012	Hong Kong urban	21
Cytotoxicity of PM _{2.5} vehicular emissions in the Shing Mun Tunnel, Hong Kong. EP, 2016	2015	Shing Mun Tunnel, roadside	21
Nonpolar organic compounds in fine particles: quantification by thermal desorption–GC/MS and evidence for their significant oxidation in ambient aerosols in Hong Kong. Analytical and Bioanalytical Chemistry, 2011	2003	Tunnel and roadside	12
	2006	TC, TW, UST, YL	12
	2010	MK (roadside), TW, UST, YL	12
Relationships between Outdoor and Personal Exposure of Carbonaceous Species and Polycyclic Aromatic Hydrocarbons (PAHs) in Fine Particulate Matter (PM _{2.5}) in Hong Kong. Aerosol and Air Quality Research, 2017	2014	PolyU, rooftop	20
Characteristics of Fine Particulate Matter (PM _{2.5})-Bound n-Alkanes and Polycyclic Aromatic Hydrocarbons (PAHs) in a Hong Kong Suburban Area. Atmosphere 2022	2011-2012	TC, suburban	19
Exposure and health risk assessment of PM _{2.5} -bound polycyclic aromatic hydrocarbons during winter at residential homes: A case study in four Chinese cities. Science of the Total Environment, 2023	2016 - 2017	Urban	18
Characteristics and health risks of personal exposure to particle-bound PAHs for Hong Kong adult residents: From ambient pollution to indoor exposure. Indoor Air, 2021	2014 and 2016	Urban	15
Molecular and elemental marker-based source apportionment of fine particulate matter at six sites in Hong Kong, China. Science of the Total Environment, 2022	2015	TC, MK, TW, YL, HT	22
Attributing risk burden of PM _{2.5} -bound polycyclic aromatic hydrocarbons to major emission sources: A case study in Guangzhou, South China. Atmospheric Environment, 2016	2013 - 2014	Urban and Rural	26
Polycyclic aromatic hydrocarbons in PM _{2.5} in Guangzhou, southern China: Spatiotemporal patterns and emission sources. Journal of Hazardous Materials, 2012	2009	Urban and suburban	18
The cellular effects of PM _{2.5} were collected in Chinese Taiyuan and Guangzhou, and their associations with polycyclic aromatic hydrocarbons (PAHs), nitro-PAHs, and hydroxy-PAHs were examined. Ecotoxicology and Environmental Safety, 2020	2016	Rural	19
Roadside and rooftop measurements of polycyclic aromatic hydrocarbons in PM _{2.5} in urban Guangzhou: Evaluation of vehicular and regional combustion source contributions. Atmospheric Environment, 2011	2006	Urban	18
Characterization of PM _{2.5} in Guangzhou, China: uses of organic markers for supporting source apportionment. Science of the Total Environment, 2016	2012 - 2013	Urban	17
Atmospheric PM _{2.5} -bound polycyclic aromatic hydrocarbons in China's four cities: Characterization, risk assessment, and epithelial-to-mesenchymal transition induced by PM _{2.5} . Atmospheric Pollution Research, 2021	2017- 2018	Urban	18
Temporal and spatial distributions, source identification, and health risk assessment of polycyclic aromatic hydrocarbons in PM _{2.5} from 2016 to 2021 in Shenzhen, China. Environmental Science and Pollution Research, 2023	2016 - 2021	Urban and coastal suburban	16

1989

Subsequently, for the PM_{2.5}-loading PAHs in Hong Kong, analysis data were collected from relevant studies conducted over the past decade. The details of the reference literature are listed in **Table 4-2**. Since 2003, the total PM_{2.5}-bound PAHs composition relative to per mg PM_{2.5} decreased 30~50 times in HK. Between 2016 and 2021 (this study), the total PAHs per unit PM_{2.5} even reduced significantly, which may be due to the COVID-19 lockdown. Within the same year, traffic-dependent sites, including Tunnel and Roadside, exhibited higher PAHs content per unit of PM_{2.5}. For the fractional composition variation, high-molecular-weight PAHs contributed decreasingly, while the light groups were becoming dominant. Especially for traffic-related PM_{2.5}, the reduced emissions of high-molecular-weight PAHs may be attributed to more explicit petroleum policies in Hong Kong over the past decades. Based on BaP-equivalent concentration analysis, the 5-ring group remains the primary contributor to health concerns from PM_{2.5}-bound PAHs; however, its fractional contribution has decreased over the past few years in HK. As illustrated in **Fig. 4-5**, over the past few decades, the sources of PM_{2.5}-bound PAHs have become increasingly complex, involving a mix of combustion sources from petroleum and coal rather than biomass. As the BaA/(BaA + Chr) trend indicates, the dominant sources of PM_{2.5}-bound PAHs in HK have converted from mixed combustion to vehicle emissions. According to BbF/(BbF + BkF), the biomass-related PAHs in HK were mainly composed of wood and coal combustion rather than crop residuals. In general, PAH levels in PM_{2.5} have decreased by approximately 100 times over the past two decades in HK. Therefore, the fractional content of the 5-ring group of PAHs declined while the 4-ring group became more primary, leading to increased inhalation health concerns. The composition variation depended on the source apportionment, which ranged from biomass combustion-dominant to mixed fossil fuel combustion-dominant, and, subsequently, traffic emissions have been emphasized in recent years. Under the influence of the growing use of clean energy sources, improvements in coal combustion quality, and energy conservation and emission-reduction policies, the traditional harmful pollutant,

2018 PAHs, has been effectively controlled in non-heating megacities, such as Hong Kong.
 2019 Meanwhile, traffic emissions have become a significant contributor to PAH levels.



2020 **Figure 4-5** The PM_{2.5}-loading PAHs composition and characteristic ratios in the past
 2021 decade. A) Decade variation, B) fractional composition, and C) BaP-equal
 2022 concentration of major toxic parent PAHs composition per milligram PM_{2.5}; The ratio-
 2023 ratio plots of D) FLT/(FLT + PYR) and IcdP/(IcdP + BghiP); E) BaA/(BaA + Chr) and

BaP/(BaP + BeP); F) BbF/(BbF + BkF) and Ant/(Phe + Ant).

In a previous study, Chen et al. (2020) compared the chemical composition differences of exposure particles between the two major commuter approaches, the MTR and bus. It was determined that commuter exposures in the MTR system were characterized by higher levels of PM_{2.5} and elemental components (*e.g.*, Ca, Cr, Fe, Zn, Ba) compared with bus riding, along with increased cellular and acellular ROS production. However, total traffic-related PM_{2.5} emissions from vehicles are still a source of inaccurate real-time analysis in Hong Kong.

4.2 Chemical composition of PM_{2.5} in Hong Kong

Overall, individuals who spend extended periods commuting by road are exposed to poorer air quality compared with those residing in general urban or coastal background areas. As shown in **Fig. 4-6**, the PM_{2.5} concentrations of samples collected on the roadside (46.7±18.1 µg/m³) and at a general urban site (40.8±19.5 µg/m³) are significantly higher than those sampled at the background site, Hok Tsui (29.9±14.5 µg/m³). The Hung Hom roadside and the Z block of PolyU are relatively close and have similar physical and weather conditions. However, roadside samples are more directly affected by heavy-traffic emissions, and PM_{2.5} concentrations at roadside sites are slightly higher than those at general urban sites throughout the year.

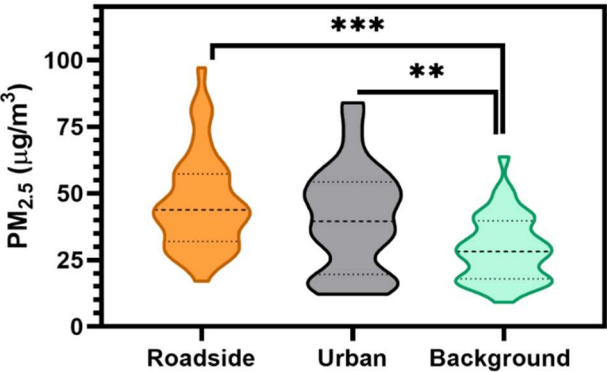


Figure 4-6 PM_{2.5} concentration calculated from the net weight of filters collected in

three sites in this study (* for $p < 0.05$, ** for $p < 0.01$, and *** for $p < 0.001$).

The seasonal averages of the analyzed components in $PM_{2.5}$ samples from these three sites are shown in **Fig. 4-7**, along with the monthly average concentrations listed in Appendix 1, accompanied by the standard deviation (STD) for each month. Conformance with the $PM_{2.5}$ concentration calculated by filter net weight, the annual average concentration of analyzed components was in descending order of RD ($25.4 \pm 10.3 \mu g/m^3$) > 6F ($23.3 \pm 15.0 \mu g/m^3$) > HT ($11.9 \pm 4.90 \mu g/m^3$). Among all analyzed components, organic matter was the primary contributor to total $PM_{2.5}$, accounting for 33.2% in general urban areas, 32.2% in roadside areas, and 28.0% in coastal background $PM_{2.5}$. Sulfate was the second most abundant component, comprising more than 20.0% in general urban and coastal background $PM_{2.5}$ and 17.1% in roadside samples. Then followed by EC, nitrate, and metals, depending on sampling regions. In line with the seasonal trend, the total analyzed composition of all three site samples throughout the year showed a synchronous tendency, decreasing in summer and increasing to a peak in winter, as well as nitrate and OC, which was attributed to the regional effect of winter heating. In contrast, components with slight seasonal variation, such as Metals, EC, and sulfate, were more affected by local emission sources. Regarding spatial discrepancies, EC and nitrate contribute more to the $PM_{2.5}$ mixture in urban samples compared with urban and coastal background samples. Especially EC, which accounted for about 10% of the roadside annual average analyzed composition, while 6% in ambient samples and 4% in background samples reflected the heavier traffic-emission impact on roadside $PM_{2.5}$. By contrast, being far away from pollution sources and facing the wet, clean breeze, the $PM_{2.5}$ composition was more influenced

by natural sources, such as fresh sea salt, which is characterized by a higher percentage concentration of Na^+ and Cl^- .

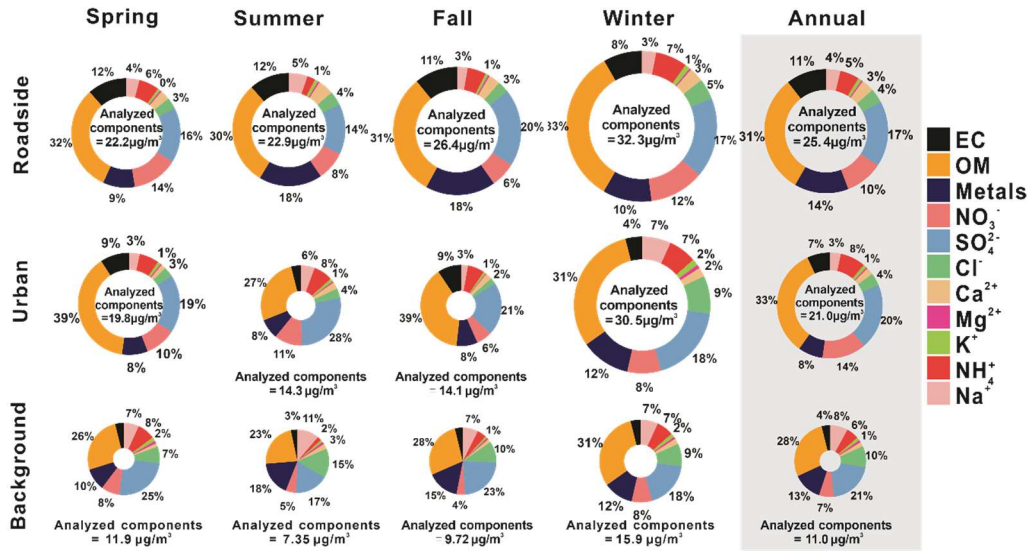


Figure 4-7 Seasonal average concentration of analyzed components of samples at three sites in Hong Kong, 2021.

4.1.1 Inorganic chemical species

The concentrations and components of trace metals and PAHs, regarded as two key hazard groups, varied spatially and seasonally in $\text{PM}_{2.5}$ across different sites. Roadside $\text{PM}_{2.5}$ consistently exhibited the highest average concentrations of the analyzed trace metals, with relatively stationary seasonal variation. For the general urban and coastal background, concentrations of $\text{PM}_{2.5}$ and trace metals declined in spring and summer, peaking in autumn and winter (**Appendix 1**). The trace metal profiles varied significantly across locations (**Fig. 4-8**). For example, the coastal background and general urban atmosphere had a significantly higher presence of As, Cd, Se, and Pb, primarily attributed to the fossil fuel combustions (Wang et al., 2022; Zhu et al., 2021), accounting for 1.80% and 1.54% of the total trace metal concentrations, compared with the 1.17 % of roadside $\text{PM}_{2.5}$ ($p < 0.0001$). In contrast, roadside $\text{PM}_{2.5}$ exhibits higher concentrations of Fe, Al, and Mn associated with traffic-related dust compared with general urban samples, maintaining a stable level throughout the year (Demir et al.,

2022; Fussell et al., 2022b; Zhang et al., 2020). In the coastal background PM_{2.5}, the concentrations of V and Ni (accounting for 0.889% of total metals) were significantly higher than those in urban samples (0.449% of total metals), which may be attributed to the emissions from residual heavy oil combustion and maritime shipping activities in the coastal region (Tao et al., 2017; Wu et al., 2018; Zhai et al., 2023).

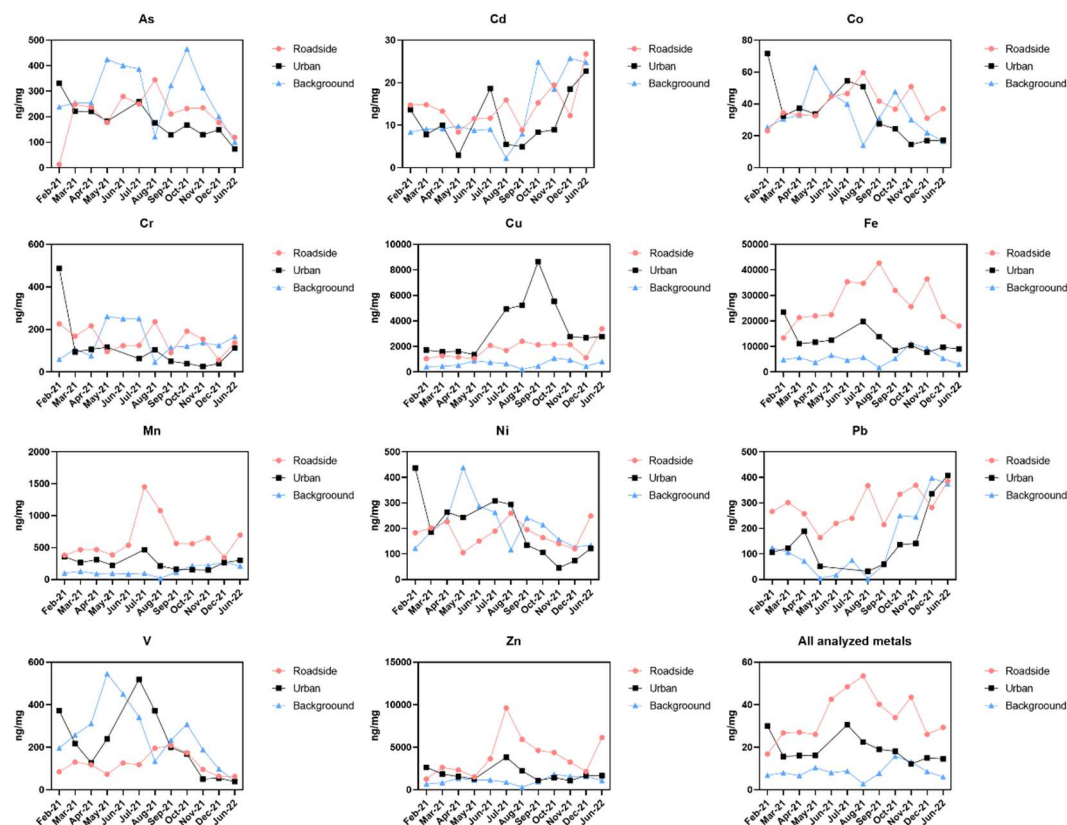


Figure 4-8 The annual variation area diagram of major harmful trace metal concentrations in PM_{2.5}.

For the speciation distribution of As, the XANES spectra were illustrated in **Fig. 4-9**, and the PM_{2.5}-bound arsenic was almost pentavalent in the selected four ambient and roadside samples. Only one general urban sample contained 3.4% trivalent arsenic, leading to severe inhalation health concerns.

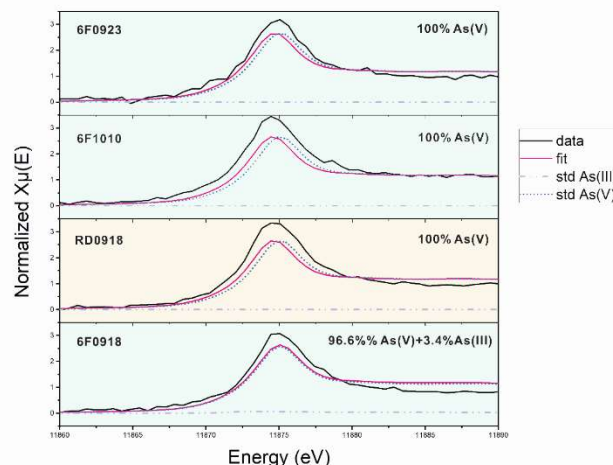


Figure 4-9 XANES spectra illustration for arsenic speciation of PM_{2.5} in Hong Kong 2021.

4.1.2 Organic chemical species

Based on TD-GC/MS analysis of non-polar organic compounds (NPOCs), the concentrations of 24 airborne PAHs and 28 hopanes and steranes were determined for all samples from three sites in Hong Kong in 2021. As a result of proactive air pollution control and environmentally friendly emission policies, PAH concentrations have been decreasing in recent years (Chow et al., 2022a; Chow et al., 2022b), thereby reducing the health risk from PAHs in Hong Kong. The annual average concentration of total PAHs was in a declining order of RD ($0.83 \pm 0.37 \text{ ng/m}^3$) > 6F ($0.69 \pm 0.40 \text{ ng/m}^3$) > HT ($0.21 \pm 0.20 \text{ ng/m}^3$). As well as the sequence of hopanes and steranes annually, average contents decreased from RD ($0.96 \pm 0.36 \text{ ng/m}^3$), 6F ($0.47 \pm 0.27 \text{ ng/m}^3$) to HT ($0.18 \pm 0.14 \text{ ng/m}^3$).

For PAHs (including the 16 EPA parent PAHs), the concentrations have decreased in

2109 Hong Kong due to proactive air pollution control measures and environmentally
2110 friendly emission regulations in recent years (Chow et al., 2022a; Chow et al., 2022b;
2111 Liao & Yu, 2020). The annual average concentration of analyzed PAHs followed a
2112 descending order: roadside (0.830 ± 0.370 ng/m³) > general urban (0.690 ± 0.400 ng/m³) >
2113 coastal background (0.210 ± 0.200 ng/m³) (**Fig. 4-10** and **Appendix 1**). The mass
2114 composition of major PM_{2.5}-bound individual PAHs decreased in the order of: Pyr, Flu,
2115 Phe, BaP, Ant, BbF, BeP. A pronounced seasonal increase was observed in winter,
2116 especially at the general urban and coastal background sites, particularly for high-
2117 molecular-weight PAHs (C₂₀- and C₂₂-PAHs). This common seasonal trend suggested
2118 the same regional sources for high-molecular-weight PAHs at the general urban and
2119 coastal background sites, mainly due to regional transport of heating combustion from
2120 the mainland (Liao & Yu, 2020; Wang et al., 2022). As evidenced in this study, light-
2121 molecular-weight PAHs (C₁₆- and C₁₈-PAHs) in roadside samples were associated with
2122 local vehicle emissions. Specifically, the roadside PM_{2.5}-bound PAHs were
2123 predominantly composed of light-molecular-weight PAHs, with concentrations
2124 significantly higher than those at general urban and coastal background sites, and
2125 without significant seasonal trends.

Interestingly, concentrations of high-molecular-weight PAHs in the general urban and even the coastal background PM_{2.5} were higher than those in the roadside samples. It is possibly due to the “sampling height effect,” in which tall buildings hinder the dispersion of PAHs from regional sources and prevent their deposition at ground level (Liao et al., 2020). To verify this speculation, system experiments will be planned to explore the vertical distribution of PM_{2.5} chemical composition in future research.

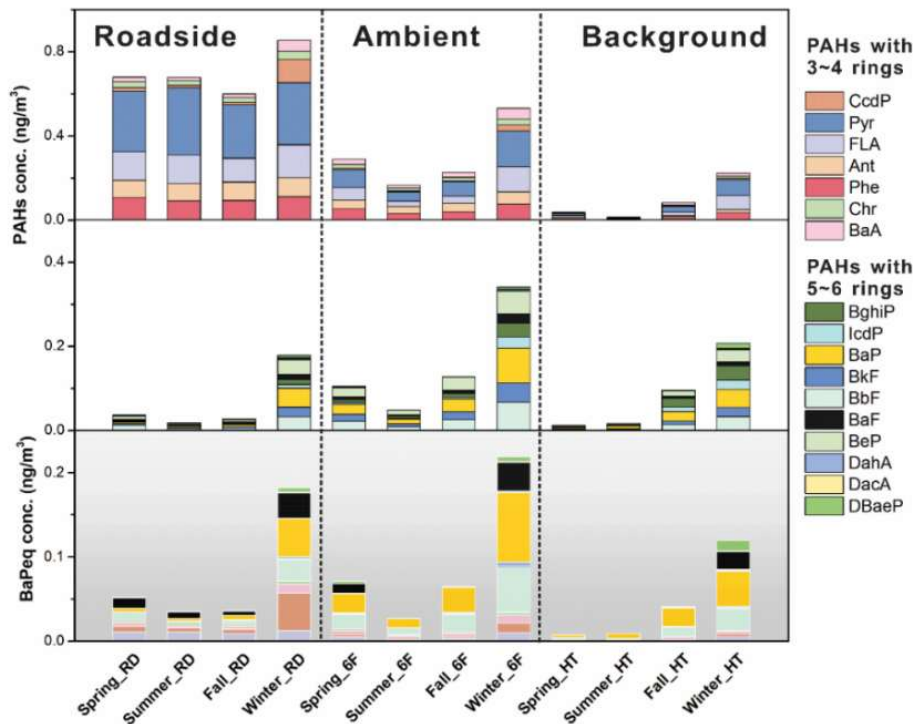


Figure 4-10 Seasonal average concentration of major PAHs in PM_{2.5}.

As one of the essential tracers of traffic emissions, hopanes are significantly enriched in roadside samples, followed by general urban samples, whereas they are seldom detected in coastal background samples (**Appendix 1**). Focus on the seasonal trend of hopanes across all three sites; there is almost no distinct fluctuation in the other two sites, but roadside samples show an increase in spring and a decrease in autumn. This also suggests that, unlike the seasonal variation in total PM_{2.5} and PAHs, the seasonal trend in hopanes in roadside samples is primarily associated with local traffic rather

than any other regional effect. For monomer hopanes and steranes concentration, the trisnorhopane with m/z 66, aa-20R-cholestane with m/z 217, and abb-20R (S)-cholestane with m/z 218 contribute most to the total hopanes and steranes contents. To illustrate the impact of different fossil fuel combustion on transportation emissions of $PM_{2.5}$, the ratio of EC with hopanes and norhopanes was used to apportion the impact of diesel and gasoline. The previous study by Yu et al. (2011) summarized some of the results of the comparison of the hopane (and norhopane)/EC ratio in Hong Kong from both roadside and ambient sites. The study showed that urban samples, primarily affected by diesel combustion, could be clearly distinguished from those from the tunnel or roadside, which are more heavily impacted by gasoline combustion.

4.2 Correlation analysis of vehicular characteristics with key toxic components in $PM_{2.5}$

The daily vehicular traffic data for Hom Hung Cross Harbor were provided by the Transport Department of the Government of the HKSAR. The vehicular flow rates were recorded as motorcycle, private car, taxi, private/public light bus, light/medium/heavy goods, and private/public double-decker bus in both directions at the Hom Hung entrance of the harbor (**Fig. 4-11B**). To investigate the correlation between vehicular energy types and major toxic compounds in traffic-related $PM_{2.5}$, the vehicular characteristic data were sorted by the exact sampling dates and summarized into four energy types: gas, gasoline, diesel, and electricity. The statistical results for vehicular characteristics and energy composition were visualized in **Fig. 4-11A**. The average daily vehicle flow rate through the Cross Harbor on all the sampling days between February 2021 and January 2022 was 107481 per day, composed of 51% gasoline, 29% diesel, 19% gas, and only 1% electric vehicles. Subsequently, for the correlation coefficient analysis, the analyzed major toxins and chemical tracers include OC/EC, sulfate, nitrate, trace metals, and selected high-content PAHs. Illustrated as a clustered heatmap in **Fig. 4-11C**, there is almost no significant correlation between total vehicle

flow and the major toxins, and gas vehicles are seldom affected by the analyzed toxic components. For the dominant energy type, gasoline vehicle emissions significantly correlated with PAHs, including BaP, BaA, and BbF, which are typically associated with incomplete combustion processes (Chow et al., 2022a). Meanwhile, diesel combustion was to some extent associated with metals such as Fe, V, Al, and As. Gas vehicles seldom affect the analyzed toxic components; for the dominant energy type, gasoline vehicle emissions significantly correlated with PAHs, including BaP, BaA, and BbF, and diesel combustion, which, to some extent, was related to metals such as Fe, V, Al, and As. The composition of PAHs in PM_{2.5} emitted from gasoline vehicles showed a higher presence of 5- and 6-ring PAHs. This conclusion has also been emphasized in the current study, which shows that the composition of PAHs in PM_{2.5} emitted from gasoline vehicles was characterized by a higher abundance of 5 and 6-ring PAHs, which are typically associated with incomplete combustion processes (Wang et al., 2022).

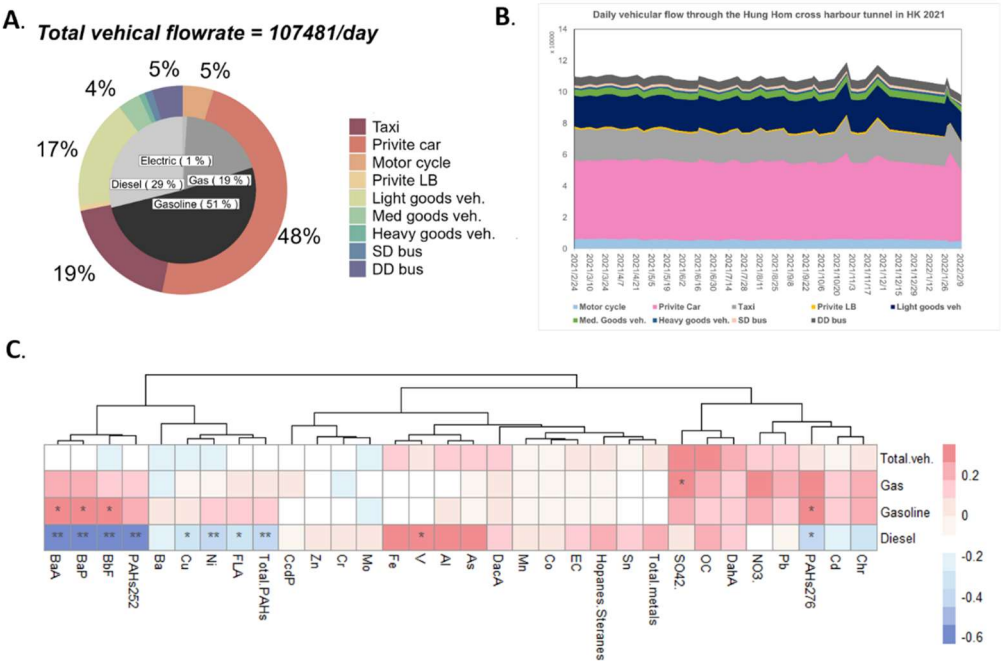


Figure 4-11 Vehicular characteristic composition at Hom Hung cross-harbor and correlation analysis with major hazardous components in roadside PM_{2.5}. A) Annual

vehicular characteristics and energy composition at Hom Hung Cross Harbor. B) Daily statistics of vehicular flow at Hung Hom station. C) Correlation coefficient analysis of vehicular characteristics with the analyzed key toxic components.

Table 4-3 Fractional composition of energy of major vehicular types at the Hung Hom Cross Harbor in 2021.

	Motorcycle	Private car	Taxi	Private LB	Light good	Med. good	Heavy good	SD bus	DD bus
Diesel	0	0	0	66.5%	99.2%	99.9%	100%	92.0 %	100%
Gasoline	99.9%	93.4%	0	33.4%	0.53%	0	0	0	0
Gas	0	1.92%	100 %	0	0	0	0	0	0
Electric	0.14%	4.71%	0	0.09%	0.27%	0.01%	0	8%	0

4.3 Acute cytotoxicity assessment

4.3.1 Cell ROS generation induced by total PM_{2.5}

Before examining the specific contributions of major toxic compounds, the total PM_{2.5}-induced ROS were analyzed as a total variable for roadside, general urban, and coastal background samples, with comparisons between sites and seasons. Illustrated in **Fig. 4-12A**, the average ROS induction potencies of the roadside PM_{2.5} were slightly higher than the general urban and regional background samples, though they were still comparable among these three sites. In detail, the EC_{IR1.5} of nine roadside samples ranged from 63.9 to 236 with a mean of 149 µg/mL; the thirteen general urban samples lay at 52.6 to 389 with a mean value of 183 µg/mL; the EC_{IR1.5} range of regional background PM_{2.5} spread to 82.5 to 459 µg/mL, with the mean at 186 µg/mL, close to the general urban samples. The higher ROS potencies of roadside PM_{2.5} directly affected by traffic-related emissions demonstrated the non-negligible contributions of

2202 these dominant local emissions to overall PM_{2.5}-induced cytotoxicity in Hong Kong. In
2203 the regional background PM_{2.5}, the ROS potencies spiked in winter may result from
2204 regional transport and increased shipping activities (Hong Kong Marine Department,
2205 2021). Surprisingly, the ROS potencies of the regional background samples were
2206 comparable to those of the general urban samples. Two possible explanations might be:
2207 (1) the lower-quality heavy oil combustion from the open sea close to HT, forbidden in
2208 Hong Kong waters, such as Victoria Bay near the 6F and RD sampling site (Air
2209 Pollution Control (Fuel for Vessels) Regulation, 2019; Wu et al., 2018); (2) the
2210 enhanced microbial toxicity carried by northwestern monsoon from the nearby Stanley
2211 Sewage Treatment Works (Drainage Services Department, 2021).

2212 However, there was minimal seasonal variation in the average EC_{IR1.5} values for
2213 roadside and urban samples, indicating that the ROS potency of PM_{2.5} was primarily
2214 influenced by local emissions in urban areas or that non-local emissions were
2215 seasonally divergent from local sources. To some degree, the limited sample size and
2216 varied sampling dates introduced potential biases, suggesting the need for more
2217 comprehensive data to assess seasonal variations in toxicity accurately. To some degree,
2218 the limited sample quantity and different sampling dates led to an inherent limitation in
2219 the results, which may require more relevant, effective toxicity data for an accurate
2220 assessment of seasonal variation.

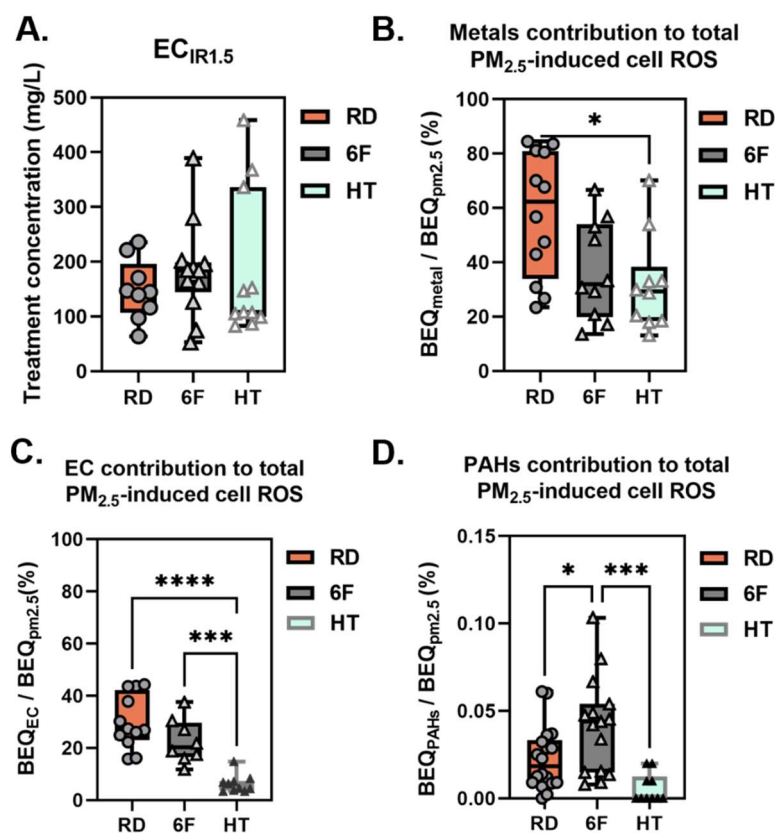


Figure 4-12 ROS generation induced by total extracted $PM_{2.5}$ in Hong Kong 2021. A) Comparison of effective concentration at ROS induction ratio of 1.5 ($EC_{IR1.5}$) between roadside, ambient, and background samples. B) Trace metals contribution; C) EC contribution; D) Major toxic PAHs contribution to total $PM_{2.5}$ -induced ROS generation.

Individual toxic compounds induced 1.5-fold intracellular ROS relative to control

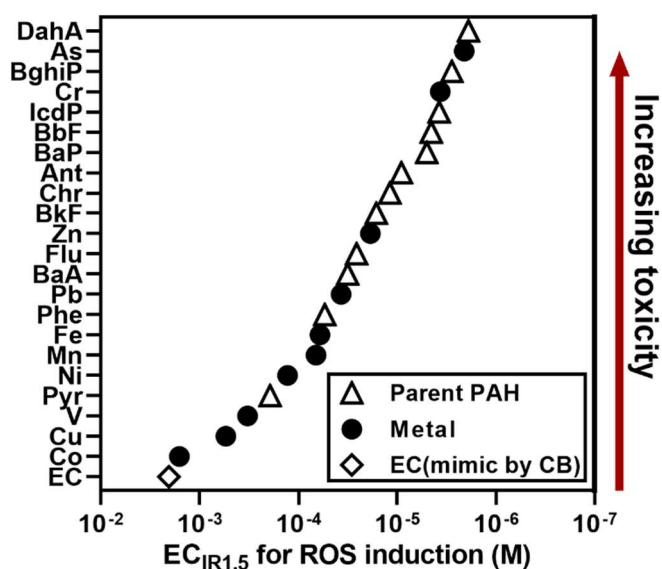


Figure 4-13 Effective concentration for individual analyzed toxin at ROS induction ratio equals to 1.5 ($EC_{IR1.5}$) related to control based on the BEAS-2B cell line.

Following the additive ROS potencies induced by the mixture of identified toxins in PM_{2.5}, we first tested cell viability and ROS generation in cells exposed to individual metals, PAHs, and EC. The average $EC_{IR1.5}$ for each component was listed in **Appendix 2** and **Fig. 4-13**, in which the ROS potencies of selected PAHs were in the order of DahA > BbF > BaP > CHR > FLA > BaA, from 1.89×10^{-6} (M) to 3.17×10^{-5} (M). Although the parent PAHs were reported as inactive compounds in the acellular ROS measurements, Jin et al. (2019) emphasized the possibility that the inactive chemicals may be modified within the cell-based assay in ROS detection (Jin et al., 2019). In comparison, for the analyzed metals, As (V) and Cr (VI) were the most toxic, with $EC_{IR1.5}$ values of 2.08×10^{-6} (M) and 3.64×10^{-6} (M), followed by Zn (II) > Pb (II) > Fe (III) > Mn (II) > Ni (II) > V (V) > Cu (II) > Co (II). The Printex U carbon black, a surrogate for EC, showed the lowest molar ROS generation potency among all the tested

chemicals in this study, with the $EC_{IR1.5}$ equaling $2.03E-3$ (M).

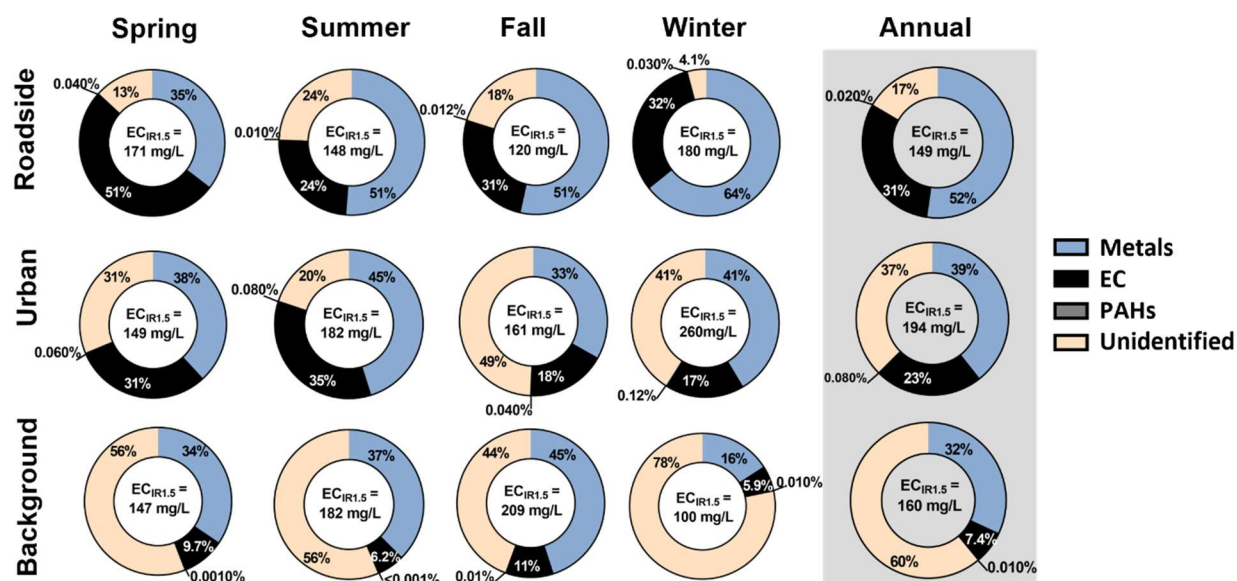


Figure 4-14 Seasonal variation of major analyzed toxic components' contribution to total extraction-induced ROS generation in $PM_{2.5}$ collected in HK 2021.

Among the fractional contributions of ROS potencies generated by all analyzed components, the trace metal group emerged as the primary contributor in Hong Kong $PM_{2.5}$, especially in the roadside $PM_{2.5}$ (accounting for 52.2% annually of total ROS generation, with seasonal fluctuations ranging from 35.4% in spring to 64.2% in winter) (Fig. 4-14 and Table 4-4). Without the direct effect of traffic sources, the contribution of trace metals declined in the corresponding seasons in both the general urban (39.3%) and coastal background $PM_{2.5}$ (32.2%) areas. Nevertheless, there was no doubt that trace metals still played the leading role in $PM_{2.5}$ -related intracellular ROS in Hong Kong. Furthermore, Fe, Zn, and As are associated with traffic and fossil fuel combustion, contributing the most among all the samples. Fe, mainly from re-suspension dust and mechanical friction, accounted for over half of the metal-related intracellular ROS in roadside $PM_{2.5}$. For the general urban and coastal background $PM_{2.5}$, As, and Cr related to combustion & industrial emissions became more prominent. It is worth noting that As and Cr made up only a tiny fraction of the total $PM_{2.5}$ mass

(As: 0.00300% ~ 0.00600%, Cr: 0.00346% ~ 0.00483%) but contributed to more than 10.0% of the overall PM_{2.5}-induced intracellular ROS (Fig. 4-15).

In addition to trace metals, EC, one of the major inorganic insoluble components in PM_{2.5}, also played a significant role in the PM_{2.5}-induced intracellular ROS, especially in roadside and general urban PM_{2.5} (31.2% and 23.2%, respectively) (Fig. 4-14). Moreover, there was almost no seasonal variation in the contribution of EC, indicating that it was dominated by local sources, particularly vehicle emissions, rather than regional combustion.

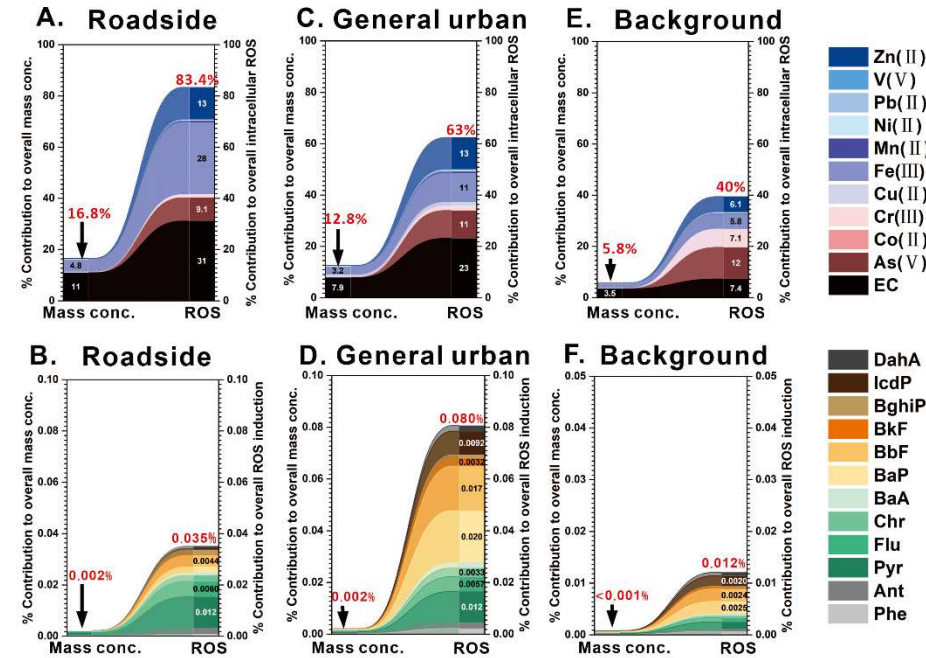


Figure 4-15 Comparison between fractional mass contribution versus intracellular ROS contribution of analyzed components in PM_{2.5}. A) EC, metals, and B) parent PAHs in roadside PM_{2.5}. C) EC, metals, and D) parent PAHs in general urban PM_{2.5}. E) EC, metals, and F) parent PAHs in coastal background PM_{2.5}.

The tested twelve PAHs, known for their high monomer cytotoxicity, contributed negligibly on an annual basis (around 0.0200% to 0.0700% in the roadside PM_{2.5},

0.0100% ~ 0.0300% in the coastal background PM_{2.5}, and increased to 0.0400%~0.120% in the general urban samples) (Fig. 4-15). Detailed in Table 4-4, light-molecular-weight PAHs (*e.g.*, Phe, Ant, Pyr, Flu, Chr, BaA) related to local traffic emissions constituted approximately 70.4% of the ROS induced by total PAHs in roadside PM_{2.5}. The increasing contribution of PAHs-induced ROS in general urban and coastal background sites, with a significant seasonal trend, emphasized the regional transport of combustion & industrial emissions in winter (less than 0.0100% of total PM_{2.5}-induced ROS in spring and summer, but increasing to 0.0300% after the monsoon diversion) (Fig. 4-15). Moreover, the contribution of high-molecular-weight PAHs increased to about 65.2% in the general urban and coastal background PM_{2.5}, which is predominantly influenced by combustion & industrial emissions. Among them, BaP, BbF, and IcdP warrant particular attention for future toxicity-focused control of PM_{2.5}-bound PAHs under regional transport.

Notably, the unexplained fraction shown in Fig. 4-14 ranged from 44.3% to 78.2% across seasons in the coastal background PM_{2.5}, indicating a significant unknown component in the total PM_{2.5}-induced ROS generation. The elevated cytotoxicity observed in winter at the coastal background site, along with a substantial proportion of unexplained components, was likely attributable to the influence of the nearby Stanley Sewage Treatment Works, which was exacerbated by the northern monsoon, potentially introducing additional microbial toxicity (Drainage Services Department, 2021).

2296 **Table 4-4** Fractional contribution of intracellular ROS induced by major analyzed toxic
2297 components

	EC _{IR1.5}	Contribution to total mass of			Contribution to total PM _{2.5} -induced ROS (%)			
		Metals	EC	PAHs	Metals	EC	PAHs	Unknown
RD210428	170.6	5.5%	15.6%	0.0014%	35.4%	51.5%	0.0747%	13.1%
RD210505	144.6	3.9%	11.5%	0.0007%	50.2%	35.5%	0.0330%	14.3%
RD210520	236.2	8.8%	10.5%	0.0000%	32.4%	18.6%	0.0000%	49.0%
RD210526	63.8	8.3%	12.6%	0.0009%	71.1%	19.0%	0.0160%	9.9%
RD210826	97.5	11.3%	14.6%	0.0011%	33.5%	31.7%	0.0404%	34.8%
RD210906	116.4	12.2%	14.0%	0.0009%	69.7%	29.4%	0.0265%	0.9%
RD210928	147.5	7.4%	9.0%	0.0003%	49.3%	32.2%	0.0211%	18.5%
RD211115	221.6	5.5%	8.9%	0.0009%	57.2%	37.0%	0.0541%	5.7%
RD211207	139.3	5.3%	9.3%	0.0010%	71.2%	26.3%	0.0503%	2.4%
6F210310	176.7	2.6%	8.3%	0.0008%	43.8%	42.2%	0.0721%	13.9%
6F210324	74.8	2.6%	6.8%	0.0004%	21.2%	17.9%	0.0149%	60.9%
6F210408	196.1	3.5%	7.0%	0.0009%	49.2%	31.8%	0.0784%	18.9%
6F210505	161.9	4.5%	9.4%	0.0007%	69.5%	25.7%	0.0308%	4.7%
6F210826	201.9	7.2%	10.1%	0.0016%	20.7%	44.2%	0.1331%	35.0%
6F210906	127.0	8.1%	9.5%	0.0012%	24.1%	13.3%	0.0256%	62.6%
6F210927	189.6	4.3%	10.8%	0.0018%	42.2%	25.6%	0.0721%	32.1%
6F211004	165.1	3.8%	3.8%	0.0004%	33.1%	13.9%	0.0236%	53.0%
6F211108	280.3	4.7%	4.3%	0.0030%	55.5%	20.6%	0.3024%	23.6%
6F211115	388.8	6.3%	6.4%	0.0014%	33.3%	18.9%	0.0889%	47.7%
6F211126	183.9	4.0%	6.3%	0.0018%	54.4%	19.9%	0.1071%	25.6%
6F211207	185.3	6.3%	5.4%	0.0011%	52.6%	22.1%	0.0687%	25.2%
6F210109	52.6	2.7%	3.4%	0.0005%	11.4%	6.0%	0.0162%	82.6%
HT210310	146.8	1.2%	2.8%	0.0001%	34.4%	9.7%	0.0054%	55.9%
HT210526	152.5	4.1%	4.3%	0.0003%	56.9%	7.9%	0.0144%	35.2%
HT210616	98.8	2.1%	4.6%	0.0002%	30.4%	7.4%	0.0031%	62.2%
HT210713	108.1	3.0%	3.3%	0.0002%	29.7%	4.4%	0.0078%	65.9%
HT210812	367.6	3.1%	3.0%	0.0003%	32.8%	5.1%	0.0142%	62.0%
HT210906	336.0	2.3%	4.6%	0.0003%	70.5%	17.4%	0.0101%	12.1%
HT210927	82.5	2.4%	3.0%	0.0002%	19.5%	4.0%	0.0069%	76.6%
HT211207	105.3	3.1%	4.0%	0.0015%	17.8%	4.0%	0.0336%	78.2%
HT211230	86.3	1.7%	3.4%	0.0000%	17.1%	8.4%	0.0000%	74.5%
HT220109	107.2	3.4%	6.7%	0.0014%	12.7%	5.4%	0.0253%	81.9%

2298

Many previous studies have reported the synergistic or antagonistic interaction after mixing individual toxins, such as metals, PAHs, or carbon blacks, which lead to a more complicated mixture toxicity mechanism (Feng et al., 2017; Guo et al., 2009; Lin & Yu, 2019b; Yu et al., 2018). However, such a mixture effect depends on the exposure concentration and the number of components in the mixture. The “Funnel Hypothesis” raised by Warne & Hawker (1995) demonstrated that the combined interaction may decrease with the increasing number of individual components in the mixture (Warne & Hawker, 1995). A Previous study reported a non-linear dose-effect curve for the organic-metal mixture, however, using the acellular DTT assay (Lin & Yu, 2019b). Our cell-based ROS analysis experiment, under combined EC-metal exposure, illustrated in Fig. 5-5, also suggested that the ROS potencies can be regarded as additive at a relatively low exposure concentration range of metal components, with a linear dose-effect curve. The additive ROS induction potencies of metals, EC, and major toxic parent PAHs were mixed at the corresponding molar concentration ratio of real samples and evaluated following the combined toxicity model proposed by Jin et al. (2019). The deviation was assessed in comparison to the CA-model predicted result (Jin et al., 2019). BC in real-world PM_{2.5} is a complicated mixture mainly consisting of elemental carbon and various tiny organic compounds, such as PAHs, absorbed on the particle surface, which makes the accurate quantification of BC’s contribution to total PM_{2.5} difficult. To the greatest extent, we alleviate this issue by testing pure elemental carbon, also known as artificial carbon black, and the major toxic PAHs separately, and calculating their toxicity contributions within the model mentioned above. The obtained EC contribution only represented the minimum estimation bound for the real BC’s contribution.

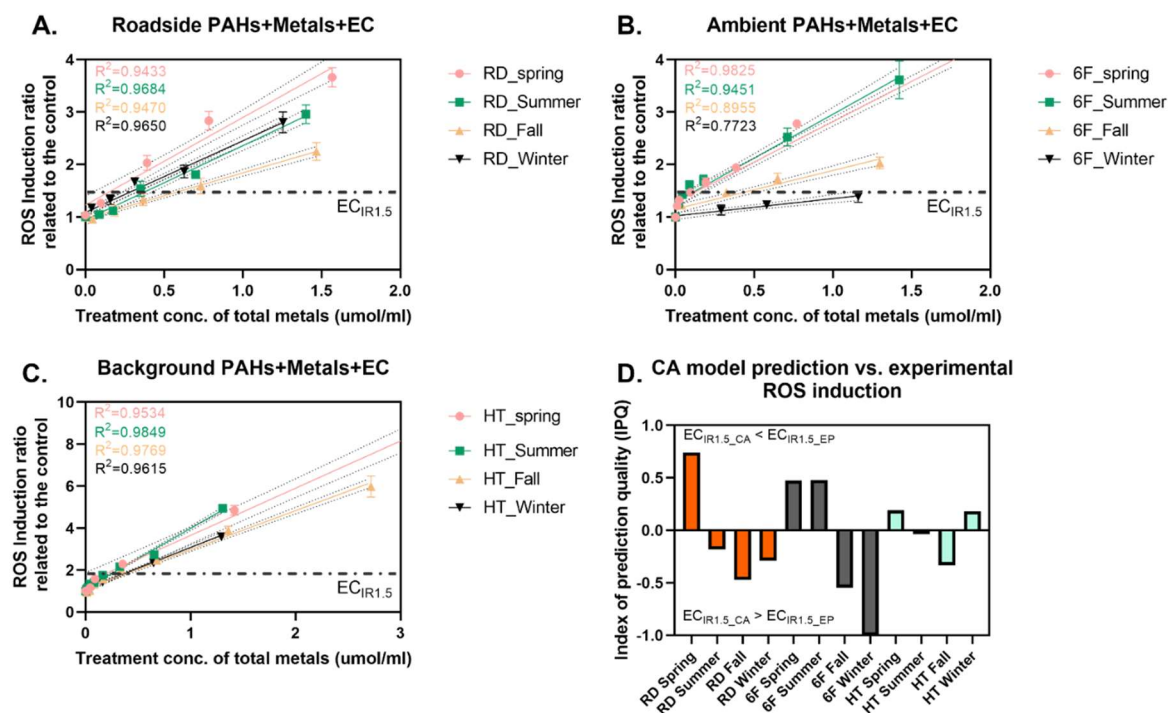


Figure 4-16 The combined effect of EC-metals with the same molar ratio as real $PM_{2.5}$ samples based on BEAS-2B cell line.

Overall, this study aimed to demystify the complexity and delineate the contributions of key components to toxicity, focusing on the primary toxic endpoint, intracellular ROS. Illustrated in [Fig. 4-15](#), trace metals and EC composed 16.8% $PM_{2.5}$ mass while contributing 83.4% intracellular ROS in roadside samples, which indicated they were still the critical toxic components of great concern in traffic-related $PM_{2.5}$. For PAHs, although their toxicity contributions were tens of times those of the mass contributions, their total toxic contribution was still negligible. However, it is crucial to acknowledge the limitations of assessing toxicity based solely on ROS generation, as this does not fully capture the multifaceted mechanisms of mixture toxicity, including genotoxicity and inflammation. Concretely, relying strictly on a single approach may (1) overlook the significant contributors like Fe and Zn in general urban $PM_{2.5}$ of Hong Kong, despite their minor roles in traditional models; (2) underestimate the contributions of the components that exert harmful effects primarily through genotoxicity or inflammation, such as As, Cr (Kouokam et al., 2022; Bustaffa et al., 2014; Kaur et al. 2024), and PAHs

(Kim et al., 2013). Besides, limitations also existed in accurately quantifying BC's contribution to total PM_{2.5} using a mixture of surrogate EC with PAHs to represent the real-world BC, which consists mostly of EC and tiny organic compounds such as PAHs absorbed on the particle surface (Briggs & Long, 2016; Niranjana & Thakur, 2017). The derived contribution of EC mixed with PAHs only represents a conservative estimation of the actual contribution of real-world BC.

4.3.3 Combined toxicity assessment of PM_{2.5} components

To assess potential synergistic or antagonistic interactions, the DR model was conducted based on our intracellular ROS result (Table 4-5). Our DR values ranged from 0.50 to 1.52, falling within the [0.5, 2] range, suggesting predominantly additive effects with some synergistic and antagonistic interactions. Specifically, samples from RD-spring, 6F spring, 6F summer, HT spring, and HT winter exhibited DR values of 1.18–1.52, indicative of synergistic effects. Conversely, samples from RD summer, RD fall, RD winter, 6F fall, 6F winter, HT summer, and HT fall showed DR values of 0.50–0.96, suggesting antagonistic effects to varying degrees. These findings align with the IPQ values from the CA model, reinforcing the predominantly additive nature of the samples analyzed.

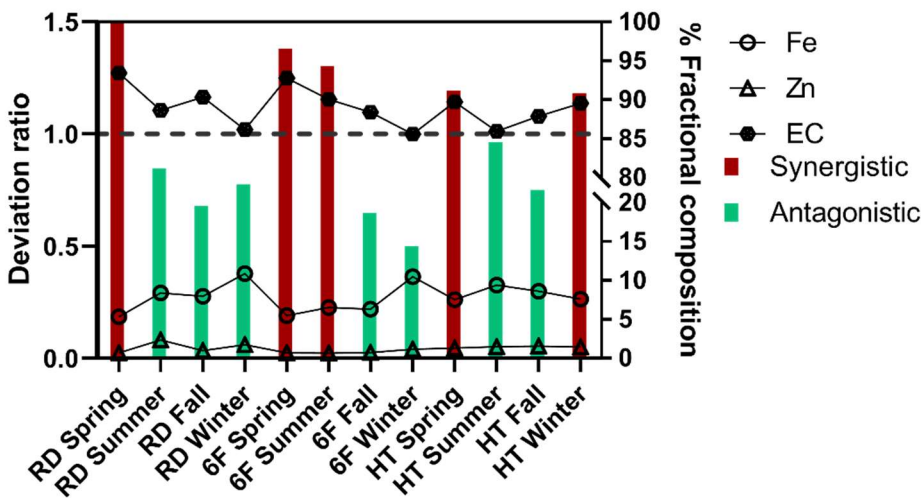


Figure 4-17 DR results and the fractional concentrations of EC, Fe, and Zn in the HK PM_{2.5} samples.

In addition, correlation analysis revealed a positive correlation between DR values and EC ($r = 0.77$, $p = 0.0037$) and a negative correlation with Fe ($r = -0.70$, $p = 0.012$), suggesting these components may drive the combined effects in Hong Kong PM_{2.5} samples (**Fig. 4-17**). For PAHs, their extremely low concentrations ($\sum \text{PAHs} < 1.0$ ng/mg) in our samples likely contributed to negligible combined biological effects, showing no significant correlation or contribution.

Table 4-5 Exposure concentrations of individual components in the mixture and the result of DR and IPQ

Exposure conc. ($\mu\text{mol/mL}$)	RD Spring	RD Summer	RD Fall	RD Winter	6F Spring	6F Summer	6F Fall	6F Winter	HT Spring	HT Summer	HT Fall	HT Winter
As	1.8E-03	3.9E-03	2.6E-03	5.4E-03	1.8E-03	1.8E-03	2.0E-03	2.2E-03	1.2E-02	2.3E-02	2.0E-02	4.5E-03
Co	3.9E-04	1.0E-03	6.1E-04	1.6E-03	4.0E-04	4.7E-04	4.2E-04	3.4E-04	1.5E-03	3.6E-03	2.3E-03	1.0E-03
Cr	5.0E-04	2.0E-03	7.2E-04	1.3E-03	2.1E-04	2.2E-04	7.7E-04	5.1E-04	6.2E-03	1.8E-02	7.6E-03	6.7E-03
Cu	1.5E-02	3.9E-02	2.5E-02	7.8E-02	2.1E-02	6.7E-02	1.1E-01	5.2E-02	2.6E-02	5.2E-02	3.2E-02	2.7E-02
Fe	3.3E-01	9.4E-01	4.7E-01	1.1E+00	1.7E-01	1.8E-01	1.6E-01	2.4E-01	4.3E-01	4.9E-01	4.7E-01	3.9E-01
Mn	8.8E-03	1.8E-02	9.2E-03	2.3E-02	4.5E-03	3.5E-03	2.6E-03	5.2E-03	1.1E-02	6.9E-03	9.0E-03	1.1E-02
Ni	2.9E-03	4.2E-03	2.6E-03	6.1E-03	2.8E-03	3.0E-03	2.0E-03	1.2E-03	7.8E-03	2.5E-02	1.4E-02	8.1E-03
Pb	2.7E-03	3.6E-03	2.2E-03	7.2E-03	1.4E-03	5.8E-04	9.8E-04	2.6E-03	5.4E-03	2.7E-04	4.2E-03	8.2E-03
V	1.8E-03	3.3E-03	3.3E-03	2.5E-03	2.0E-03	4.1E-03	3.4E-03	1.3E-03	1.4E-02	3.7E-02	2.0E-02	9.7E-03
Zn	4.3E-02	2.6E-01	5.5E-02	1.7E-01	2.1E-02	1.7E-02	1.9E-02	2.6E-02	7.3E-02	7.6E-02	8.3E-02	7.4E-02
FLA	1.8E-05	2.6E-05	1.2E-05	3.7E-05	4.7E-06	3.7E-06	6.0E-06	8.2E-06	1.1E-05	2.6E-06	4.3E-06	1.8E-05
CHR	3.3E-06	3.7E-06	2.3E-06	5.7E-06	1.3E-06	1.8E-06	1.7E-06	1.9E-06	1.5E-06	7.3E-07	1.4E-06	2.7E-06
BaP	7.7E-07	0.0E+00	7.7E-07	8.2E-06	1.5E-06	1.8E-06	4.0E-06	6.4E-06	2.4E-06	4.0E-06	3.1E-06	1.3E-05
BaA	3.1E-06	1.7E-06	2.0E-06	1.0E-05	1.9E-06	2.0E-06	2.9E-06	3.5E-06	2.8E-06	4.0E-06	3.6E-06	6.1E-06
BbF	2.2E-06	4.9E-07	6.3E-07	6.1E-06	1.4E-06	1.4E-06	3.0E-06	4.9E-06	2.9E-06	3.0E-06	3.4E-06	8.5E-06
BkF	3.5E-07	1.1E-07	3.8E-07	4.1E-06	1.1E-06	1.2E-06	2.1E-06	3.1E-06	2.1E-06	1.6E-06	1.8E-06	5.6E-06
DahA	2.4E-07	0.0E+00	8.6E-08	3.7E-07	7.0E-08	0.0E+00	0.0E+00	3.4E-07	0.0E+00	0.0E+00	6.0E-07	1.2E-06
EC	5.9E+00	9.9E+00	5.3E+00	8.6E+00	2.9E+00	2.6E+00	2.3E+00	2.0E+00	5.1E+00	4.5E+00	4.8E+00	4.6E+00
Experimental ECIR1.5 ($\mu\text{mol/mL}$)	0.34	0.35	0.57	0.37	0.35	0.34	0.64	0.59	0.22	0.17	0.27	0.25
Predicted ECIR1.5 ($\mu\text{mol/mL}$)	0.52	0.30	0.39	0.28	0.48	0.44	0.41	0.29	0.27	0.17	0.20	0.30
DR	1.52	0.85	0.68	0.77	1.38	1.30	0.65	0.50	1.19	0.96	0.75	1.18
IPQ	0.52	-0.18	-0.47	-0.29	0.38	0.30	-0.55	-1.00	0.19	-0.04	-0.33	0.18

4.4 Chronic inhalation health risk assessment

The chronic inhalational health risks posed by metals, PAHs, and EC were calculated at all sampling sites based on the annual average concentrations of each analyzed component, with a focus on both the CR and non-carcinogenic risks (*i.e.*, hazard quotient, HQ) for adults, as shown in **Fig. 4-18A**. The component-specific contributions of individual components to total CR and HQ were illustrated in **Fig. 4-18B and C**, respectively. Given the relatively low overall contribution of PAHs to CR, the carcinogenic risk of airborne PAHs with non-zero RPF coefficients was enlarged in the right subfigure of **Fig. 4-18C**.

The major contributing elements differed between acute intracellular ROS and chronic health risks. Specifically, the top three contributors to CR associated with PM_{2.5} in 2021 were hexavalent Cr (56.4% ~ 66.5%), pentavalent As (15.9% ~ 21.3%), and Co (11.3% ~ 17.3%), all commonly associated with the combustion of fossil fuels, across all samples. For HQ induced by all analyzed components, a significant presence of Mn was observed, especially in roadside samples (36.3%), linked to the resuspension of road dust. In addition, V and Ni were also highlighted as important contributors to HQ, especially at the coastal background site, where V accounted for 10.9% and Ni contributed 10.3%, attributed to the heavy maritime oil emissions in the coastal area.

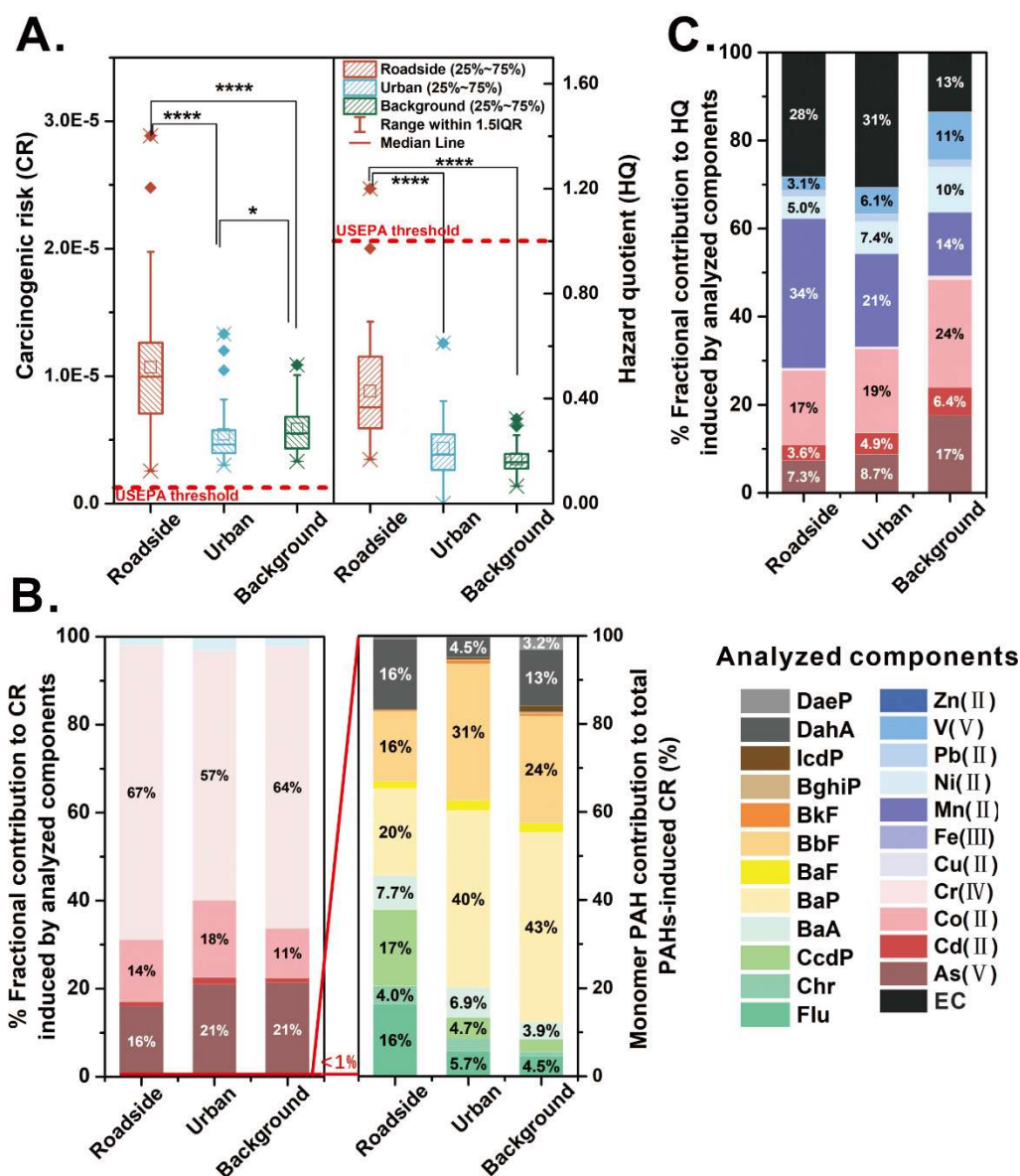


Figure 4-18 Health risks induced by trace metals, EC (surrogated by diesel particulate), and PAHs. A) The total carcinogenic and hazard quotient induced by analyzed components; B) Fraction contribution of analyzed components to hazard quotient; C) Fraction contribution of analyzed components to carcinogenic risks.

Similar to the acute intracellular ROS results, PAHs contributed negligibly to overall chronic health risks induced by all analyzed components in Hong Kong PM_{2.5}, accounting for 0.600% of the overall CR in general urban PM_{2.5}, and even less than

0.0600% in the samples from the other two sites. The analysis of PAH-related CR highlighted a broad distribution and notable seasonal fluctuations in PAH concentrations, driven by regional prevailing wind patterns. Exhibiting close correspondence with the intracellular ROS results, the composition of inhalation cancer risks attributable to each PAH compound varied across sites. In detail, the contribution of light-molecular-weight PAHs (*e.g.*, Flu, CcdP, and Chr) significantly increased in roadside PM_{2.5} ($p < 0.005$). Despite relatively low unit toxicity, they may pose a considerable contribution due to the high concentration associated with local vehicle emissions. Besides, the high-molecular-weight PAHs with higher unit toxicity were brought to the front in the general urban and coastal background samples, predominantly linked to combustion & industrial emissions (Chow et al., 2022a; Liu et al., 2017) and dominated by regional transport dynamics. In addition, for the general urban and coastal background samples, BaP and BbF are primary contributors to not only PAH-induced intracellular ROS, but also chronic health risks related to PAHs (Fig. 4-18C).

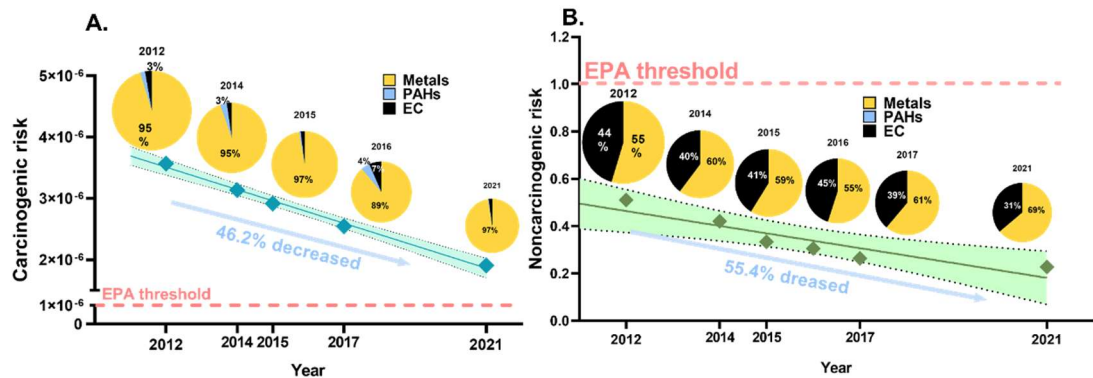


Figure 4-19 Decade variation of inhalation health risks of PM_{2.5} in Hong Kong

To further evaluate the decade-long variations of chronic inhalation health risks associated with PM_{2.5}, historical data from HKEPD were harmonized with our 2021 measurements in both CR and HQ, based on annual average concentrations of metals,

PAHs, and EC across representative general urban sites in Hong Kong. This analysis drew on historical data alongside our 2021 measurements, providing a detailed picture of risk changes over time (**Fig. 4-19**).

As shown in **Fig. 4-19** (left panel), the total CR decreased significantly, falling from approximately $4.5\text{E-}6$ in 2012 to $1.5\text{E-}6$ in 2021, a 46.2% reduction. This decline reflects the effectiveness of air quality management strategies, including stricter controls on vehicular emissions (*e.g.*, Euro VI standards phased in from 2014) and industrial upgrades (*e.g.*, technical memorandums for power plants released in 2015 and 2017) (Chow, Liao, et al., 2022a). In addition, external factors like reduced activity during 2020-2021 COVID-19 lockdowns may have amplified recent drops (Wang et al., 2024). Throughout the decade, metals remained the primary contributors to CR, making up 95–98% of the total risk each year. PAHs and EC had minor roles, with PAHs contributing 3–4% in earlier years (*e.g.*, 3% in 2012 and 2014) and dropping to 1% by 2021, while EC stayed negligible at 1–2%. Despite the overall decrease, CR levels stayed above the USEPA's acceptable threshold of $1\text{ E-}6$, highlighting the ongoing public health concern from metal-laden $\text{PM}_{2.5}$, particularly from combustion-related sources such as Cr(VI), As(V), and Co, as noted in our 2021 analysis.

Similarly, the noncarcinogenic risk (*i.e.*, HQ) showed a notable decline, decreasing from approximately 0.5 in 2012 to 0.2 in 2021 (within the EPA threshold of 1.0), a 55.4% reduction (**Fig. 4-19**, right panel). This improvement suggests safer inhalation exposure levels for noncarcinogenic effects, likely due to reductions in $\text{PM}_{2.5}$ mass and key toxic components. The proportional contribution of metals to HQ increased over the decade, rising from 55% in 2012 to 69% in 2021 (with intermediate values of 44% in 2014, 40% in 2015, 41% in 2017). Meanwhile, EC's share decreased from 44% in 2012 to 31% in 2021, indicating reduced impact from diesel-related emissions due to fleet upgrades and improved fuel quality. PAHs continued to contribute minimally to HQ (<1% throughout), consistent with their small role in overall chronic risks.

These trends over the decade demonstrate the success of pollution control measures in lowering PM_{2.5}-related health risks, while highlighting the persistent dominance of metals in both CR and HQ. The growing relative importance of metals in non-carcinogenic risks points to the need for targeted actions on sources such as road dust resuspension (*e.g.*, Mn) and maritime emissions (*e.g.*, V and Ni), as supported by our site-specific observations. Future monitoring should include emerging contaminants and real-time exposure data to enhance these risk assessments.

4.5 Summary

This chapter systematically examined the component-specific health impacts of PM_{2.5} in Hong Kong by analyzing chemical composition, correlations with vehicular emissions, acute cytotoxicity, and chronic inhalation health risks. As a whole, frequent commuters and transportation-related workers face higher acute inhalation toxicity and long-term inhalation health risks than residents in the general urban and background areas. The roadside PM_{2.5} exhibited significantly higher potencies in both 20-year-based hazard quotient and lifetime carcinogenic risk induced by metals, EC, and PAHs ($p < 0.005$). For intracellular ROS, one of the indicators for acute biological effect, the roadside PM_{2.5} exhibited slightly higher potencies compared with the other sites. Moreover, different components contributed variably to acute and chronic effects. The key findings are summarized as follows:

- (1) PM_{2.5} concentrations and components differed significantly across roadside, urban, and coastal background sites. Roadside sites ($46.7 \pm 18.1 \mu\text{g}/\text{m}^3$) exhibited higher concentrations than urban ($40.8 \pm 19.5 \mu\text{g}/\text{m}^3$) and background sites ($29.9 \pm 14.5 \mu\text{g}/\text{m}^3$), primarily driven by traffic emissions. Organic matter was the dominant component (28.0–33.2%), followed by sulfate (17.1–20.0%), EC, nitrate, and metals. Seasonally, total PM_{2.5} and components like nitrate showed winter peaks due to regional heating emissions, while metals, EC, and sulfate (influenced by

2466 local sources) exhibited minimal seasonal fluctuations. Spatial discrepancies
2467 highlighted EC (10% at roadside vs. 4% at background) and traffic-related metals
2468 (*i.e.*, Fe, Al, Mn) as markers of roadside pollution. In contrast, background sites
2469 were enriched with sea salt (*i.e.*, Na⁺, Cl⁻) and maritime emission-related metals
2470 (*i.e.*, V, Ni).

2471 (2) For inorganic species, roadside PM_{2.5} was dominated by traffic-related metals (*i.e.*,
2472 Fe, Al, Mn), while urban and background sites had higher levels of fossil fuel
2473 combustion-related metals (*i.e.*, As, Cd, Se, Pb). Coastal background sites showed
2474 elevated V and Ni, attributable to maritime shipping and residual oil combustion.

2475 (3) For organic species, PAH concentrations followed the order roadside (0.83±0.37
2476 ng/m³) > urban (0.69±0.40 ng/m³) > background (0.21±0.20 ng/m³). Light-
2477 molecular-weight PAHs (C₁₆–C₁₈) dominated roadside samples (linked to local
2478 vehicles), while high-molecular-weight PAHs (C₂₀–C₂₂) in urban/background sites
2479 reflect regional transport. Hopanes, as traffic tracers, were enriched in roadside
2480 samples with spring peaks tied to local traffic load.

2481 (4) According to the correlations analysis between key toxic components with
2482 vehicular characteristics, gasoline vehicles (51% of total flow) strongly correlated
2483 with 5–6 ring PAHs (*i.e.*, BaP, BaA, BbF) from incomplete combustion. In
2484 comparison, diesel vehicles (29%) were associated with metals (*i.e.*, Fe, V, Al, As).
2485 Gas and electric vehicles (19% and 1%, respectively) showed negligible
2486 correlations with the analyzed toxic components.

2487 (5) Standing from a component-specific perspective, trace metals were the primary
2488 contributors to acute intracellular ROS (52.2% annually at roadside, 39.3% in
2489 urban, 32.2% in background), with Fe (traffic-related), As, and Cr (combustion-
2490 related) being key drivers. EC contributed 31.2% (roadside) and 23.2% (urban) to

2491 ROS, with stable seasonal contributions indicating local (vehicular) sources. PAHs,
2492 despite high individual toxicity, contributed minimally (0.02–0.12%).

2493 (6) Regarding the chronic health risks, CR was dominated by Cr(VI) (56.4–66.5%),
2494 As(V) (15.9–21.3%), and Co (11.3–17.3%), linked to fossil fuel combustion. Non-
2495 carcinogenic risks (HQ) were driven by Mn (36.3% at roadside, from road dust
2496 resuspension), V, and Ni (coastal background, from maritime emissions). PAHs
2497 contributed negligibly to overall CR (<0.6%).

2498 (7) Both carcinogenic and noncarcinogenic risks associated with PM_{2.5} inhalation
2499 showed a significantly decreasing trend in the past decade, indicating practical
2500 improvements in air quality and related health risks over this period, in which
2501 metals are the consistent main contributors to chronic health risks.

2502 This chapter has clarified the differences in the health impacts of various components
2503 of Hong Kong's PM_{2.5} and identified key contributing substances, laying a foundation
2504 for further exploration of source-resolved health impacts. The next chapter focused on
2505 source apportionment of PM_{2.5}, tracing the major emission sources of the
2506 aforementioned key components to reveal the specific contributions of different sources
2507 to mass concentration, acute cytotoxicity, and chronic inhalation health risks.

2508

5 Source-resolved Health Impact of PM_{2.5}

In addition to specific components, clearly profiling the sources responsible for these key toxic components is also significant. Chapter 5 aimed at answering two-tiered questions: (1) what the major sources and regional transport pathways are contributing to PM_{2.5} pollution; (2) how different emission sources of PM_{2.5} quantitatively contribute to cellular toxicity and health risks.

5.1 Source apportionment

Based on the examination, the most reasonable and optimal solution for all PM_{2.5} samples from roadside, general urban, and regional background sites was seven factors, with all 100 runs converging and Q(Robust)/Q(True) values ranging from 0.950 to 1.00. The criteria included the interpretability of output profiles from the base model and error estimation profiles, including Bootstrap (BS) summaries and Bootstrap-Displacement (BS-DISP) results, which are attached in **Table 5-1**. All factors were mapped with more than 95.0% accuracy between the bootstrapped and original results in the BS test. For the BS-DISP analysis, 100-run solutions were acceptable, with decreases in Q values of -1.38% and -1.29% before and after constrained processing, respectively, without any factor swaps. All factors were identified based on the input species with the highest loadings for each factor.

2528 **Table 5-1** PMF boots map and error estimation result of the Hong Kong PM_{2.5} output
 2529 profile.

**** Bootstrap Summary of Base Run #96 ****								
Number of Bootstrap	100							
Bootstrap random seed:	61							
Min. Correlation R-Value:	0.6							
DISP Diagnostics:								
Error Code:	0							
Largest decrease in Q:	-							
%dQ:	117.16							
	-1.29							
Swaps by Factor:	0	0	0	0	0	0		
BS Mapping:								
	Factor 1	Factor 2	Factor 3	Factor 4	Factor 5	Factor 6	Factor 7	Unmapped
Boot Factor 1	99	1	0	0	0	0	0	0
Boot Factor 2	0	99	0	0	0	0	0	0
Boot Factor 3	0	0	99	0	0	0	0	0
Boot Factor 4	0	0	0	100	0	0	0	0
Boot Factor 5	0	1	0	0	98	0	0	1
Boot Factor 6	0	0	0	0	0	97	2	0
Boot Factor 7	0	0	3	0	0	0	96	0

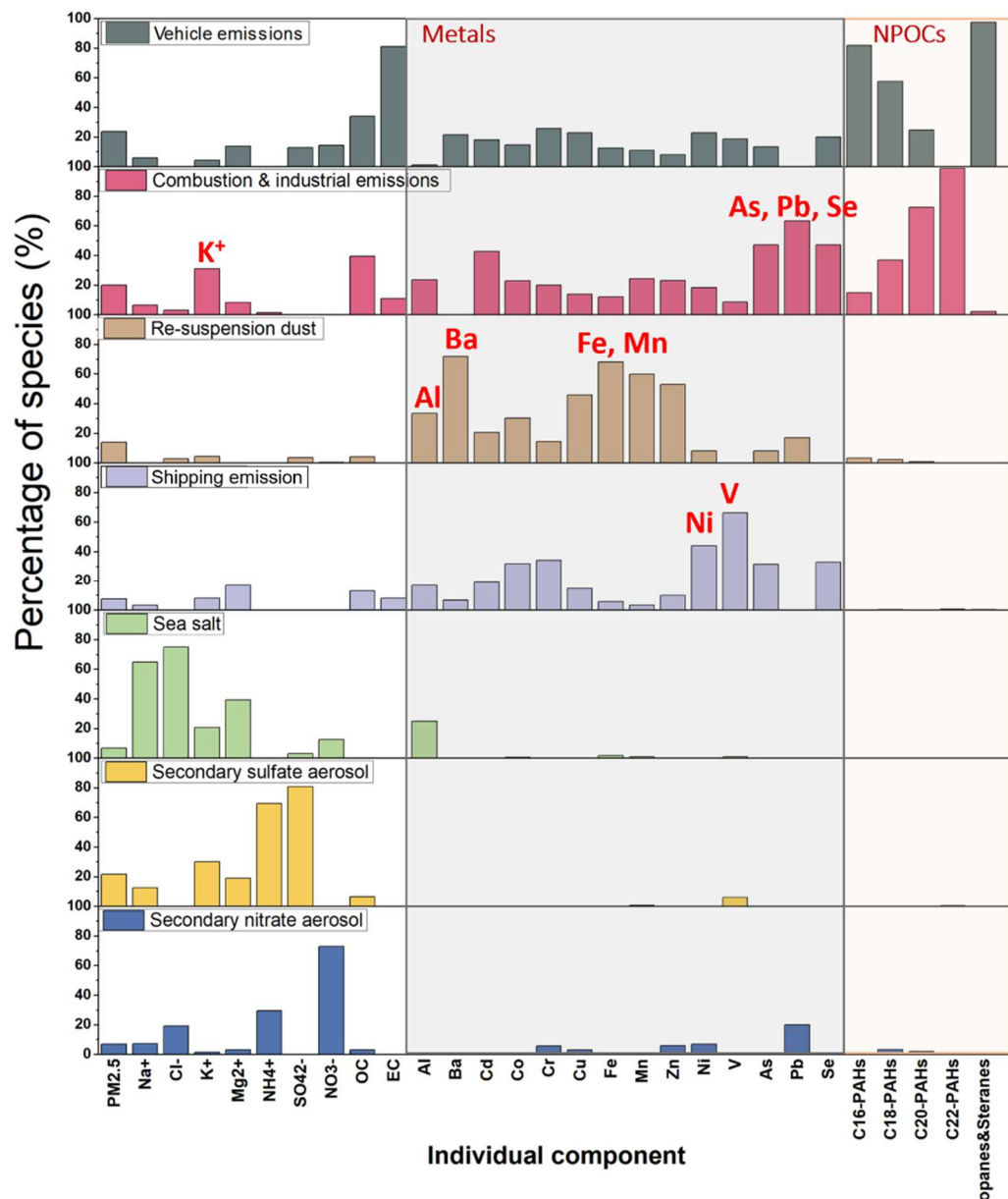
2530

2531 5.1.1 Source profiles

2532 Based on the examination, the most reasonable and optimal solution for all PM_{2.5}
 2533 samples from the roadside, general urban, and coastal background sites was the 7-factor
 2534 model, with all 100 runs converging and Q(Robust)/Q(True) within 0.950~1.00. Source
 2535 profiles calculated from all PM_{2.5} samples in this study are shown in **Fig. 5-1**. Factor 1

was associated with vehicle emissions, as indicated by high hopane and EC concentrations. The OC/EC mass ratio of 0.880 closely aligns with the reported value characteristic of vehicle emissions in Hong Kong (Cheng et al., 2010, 2015; Chow et al., 2022b), which is explicitly associated with liquid fuel combustion. This factor is also considered a primary local contributor to light-molecular-weight PAHs. Subsequently, high loadings of K^+ , As, Pb, Se, and OC suggested Factor 2 is a mixed source primarily controlled by regional transport, comprising biomass and coal combustion as well as industrial emissions (collectively termed combustions & industrial emissions) (Cheng et al., 2015; Chow et al., 2022a; Gao et al., 2015). Moreover, the richness in high-molecular-weight PAHs further supported the association of this factor with combustion (Gao et al., 2015). Compared with previous studies reporting $PM_{2.5}$ in 2015 in the GBA, our findings indicate that industrial emissions contribute less than the 12-20% observed at six sites in Hong Kong and are relatively similar to the nearby city of Macao, which reported a 10% contribution (Chow et al., 2022a; Wang et al., 2019). Subsequently, Factor 3 is identified as re-suspension dust containing crustal materials and non-exhaust road dust, characterized by high contents of crustal elements such as Al, Ba, Fe, and Mn, with medium loadings of traffic-indicating metals (*e.g.*, Cu, Fe, and Zn) attributed to the friction of brake and tire (Demir et al., 2022; Fussell et al., 2022b; Zhang et al., 2020). Factor 4 is characterized by the abundance of trace metals, V and Ni, indicating the residual heavy oil associated with shipping emission (Tao et al., 2017; Wu et al., 2018; Zhai et al., 2023). Factor 5 is defined as sea salt with a significant presence of Na^+ , Cl^- , and Mg^{2+} , with the $[Na^+]/[Cl^-]$ molar ratio of 1.05 (Chow et al., 2022a; Wong et al., 2022). Factor 6 is attributed to the secondary sulfate aerosol formation process, with a high contribution from ammonium and sulfate, predominantly combined as $(NH_4)_2SO_4$, as evidenced by a $[SO_4^{2-}]/[NH_4^+]$ molar ratio of 0.680. The high loading of NO_3^- and NH_4^+ in Factor 7, with a ratio of 1.21, indicates the secondary nitrate aerosol formation. More details about the output source profile are summarized in **Table 5-2**.

2564



2565

Figure 5-1 Source profiles for PM_{2.5} samples in Hong Kong 2021.

2566

2567 **Table 5-2** PMF output source profile of PM_{2.5} in Hong Kong, 2021.

Species conc. in µg/m ³	Factor 1 Vehicle emissions	Factor 2 Combusti on & industrial emissions	Factor 3 Re- suspensio n dust	Factor 4 Shipping emission	Factor 5 Sea salt	Factor 6 Secondar y sulfate aerosol	Factor 7 Secondar y nitrate aerosol
PM _{2.5}	6.8E+00	5.7E+00	3.9E+00	2.1E+00	1.9E+00	6.2E+00	2.0E+00
Na ⁺	4.6E-02	4.9E-02	0.0E+00	2.5E-02	5.0E-01	9.6E-02	5.6E-02
NH ₄ ⁺	0.0E+00	1.5E-02	0.0E+00	0.0E+00	0.0E+00	8.1E-01	3.4E-01
K ⁺	8.2E-03	6.0E-02	8.3E-03	1.5E-02	4.0E-02	5.8E-02	2.9E-03
Mg ²⁺	1.5E-02	9.0E-03	0.0E+00	1.8E-02	4.3E-02	2.0E-02	3.2E-03
Cl ⁻	0.0E+00	2.9E-02	2.6E-02	0.0E+00	7.1E-01	0.0E+00	1.8E-01
SO ₄ ²⁻	4.6E-01	0.0E+00	1.2E-01	5.0E-03	1.1E-01	2.9E+00	0.0E+00
NO ₃ ⁻	2.8E-01	0.0E+00	5.0E-03	0.0E+00	2.5E-01	0.0E+00	1.4E+00
OC	9.9E-01	1.2E+00	1.2E-01	3.8E-01	0.0E+00	1.9E-01	8.9E-02
EC	1.1E+00	1.5E-01	0.0E+00	1.1E-01	0.0E+00	0.0E+00	0.0E+00
Al	8.5E-03	1.7E-01	2.4E-01	1.2E-01	1.8E-01	0.0E+00	0.0E+00
As	1.1E-03	3.8E-03	6.3E-04	2.5E-03	0.0E+00	0.0E+00	0.0E+00
Ba	3.8E-03	0.0E+00	1.3E-02	1.2E-03	0.0E+00	0.0E+00	0.0E+00
Cd	6.6E-05	1.6E-04	7.5E-05	7.0E-05	0.0E+00	0.0E+00	0.0E+00
Co	1.8E-04	2.9E-04	3.8E-04	4.1E-04	8.6E-06	0.0E+00	9.7E-07
Cr	7.1E-04	5.4E-04	3.9E-04	9.4E-04	0.0E+00	0.0E+00	1.6E-04
Cu	1.0E-02	6.3E-03	2.1E-02	6.6E-03	0.0E+00	0.0E+00	1.3E-03
Fe	7.9E-02	7.5E-02	4.3E-01	3.6E-02	1.0E-02	0.0E+00	0.0E+00
Mn	1.4E-03	3.1E-03	7.6E-03	4.1E-04	1.1E-04	7.5E-05	0.0E+00
Ni	1.4E-03	1.1E-03	4.9E-04	2.7E-03	0.0E+00	0.0E+00	4.2E-04
Pb	0.0E+00	5.1E-03	1.4E-03	0.0E+00	0.0E+00	0.0E+00	1.6E-03
Se	2.1E-03	4.9E-03	0.0E+00	3.4E-03	0.0E+00	0.0E+00	0.0E+00
V	1.1E-03	5.3E-04	0.0E+00	4.1E-03	4.6E-05	3.7E-04	0.0E+00
Zn	6.4E-03	1.8E-02	4.2E-02	7.7E-03	0.0E+00	0.0E+00	4.6E-03
Phe	3.9E-05	7.2E-06	2.1E-06	0.0E+00	1.2E-08	9.3E-07	2.5E-07
Ant	3.3E-05	4.2E-06	2.7E-06	0.0E+00	0.0E+00	0.0E+00	0.0E+00
Flu	5.1E-05	1.2E-05	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00
Pyr	8.6E-05	1.5E-05	6.9E-06	0.0E+00	0.0E+00	0.0E+00	0.0E+00
CcdP	5.4E-06	2.6E-06	0.0E+00	0.0E+00	0.0E+00	1.5E-08	0.0E+00
BaA	4.6E-06	1.2E-05	0.0E+00	1.8E-07	0.0E+00	0.0E+00	1.7E-06
Chr	9.7E-06	3.8E-06	2.6E-07	0.0E+00	1.9E-08	0.0E+00	0.0E+00
BbF	6.6E-07	1.6E-05	0.0E+00	0.0E+00	0.0E+00	0.0E+00	9.4E-07
BkF	0.0E+00	1.2E-05	6.5E-08	0.0E+00	2.3E-08	0.0E+00	2.7E-08
BaP	0.0E+00	2.0E-05	0.0E+00	1.2E-07	1.7E-08	7.6E-08	1.6E-08
IcdP	5.3E-08	5.9E-06	0.0E+00	0.0E+00	0.0E+00	0.0E+00	7.7E-09
BghiP	0.0E+00	5.2E-06	0.0E+00	0.0E+00	1.4E-07	0.0E+00	0.0E+00
DahA	4.3E-08	3.0E-07	0.0E+00	5.2E-08	0.0E+00	0.0E+00	0.0E+00
Hopanes	5.9E-04	1.3E-05	0.0E+00	1.3E-06	5.0E-07	0.0E+00	0.0E+00

5.1.2 Source contribution to key toxic components in PM_{2.5}

Illustrated in **Fig. 5-2A**, vehicle emissions (43%) contributed most to the roadside PM_{2.5} annually, followed by the declining sequence: combustions & industrial emissions (18.9%), secondary sulfate aerosols (17.5%), re-suspension dust (16.0%), secondary nitrate aerosols (2.2%), sea salt (1.55%), and shipping emission (1.21%). Among all, traffic-related emissions, including vehicular engines and non-exhaust emissions during on-road transport, were the primary contributors to PM_{2.5}-bound trace metals and PAHs. For PAHs, the low-molecular-weight PAHs, such as Pyr, Flu, and Chr, were dominated by the local on-road vehicle emissions, especially from diesel vehicles. For trace metals, Cu, Fe, Mn, and Zn were significantly affected by re-suspended dust. Besides local traffic, combustion & industrial emissions, which dominated regional transport, also significantly contributed to hazardous, high-molecular-weight PAHs and metals, including As (almost pure pentavalent arsenic, illustrated in **Fig. 4-4**), Cd, Co, and Pb, which lead to severe individual toxicity in roadside PM_{2.5}.

For the general urban PM_{2.5}, the secondary sulfate aerosol became the primary contributor (34%) to PM_{2.5} mass, followed by vehicle emissions (28%), combustions & industrial emissions (23%) (**Fig. 5-2B**). Due to the site being on the rooftop, on-road re-suspension dust contributed only a limited amount (6.68%) to the general urban PM_{2.5}. Then followed by secondary nitrate aerosol (4.5%), shipping emission (2.7%), and sea salt (1.59%). In contrast to the roadside samples, combustions & industrial emissions became more crucial for PM_{2.5}-bound trace metals and PAHs than vehicle emissions.

Regarding the regional background PM_{2.5} shown in **Fig. 5-2C**, the top two contributors were secondary sulfate aerosol and combustion & industrial emissions (41% and 31%). Their relatively higher contributions than in the other site samples were attributed to (1) the monsoon-dominated pollutant transport and (2) fewer non-shipping anthropogenic

activities nearby. Moreover, under regional effects from the northern continents, combustion & industrial emissions consisted of the most considerable portion of the total mass loading of PAHs (83%), especially the highly toxic groups (C₂₀–C₂₂), which remained relatively stable during regional transport (Choi et al., 2010; Tsiodra et al., 2021). Marine sources were also emphasized in this coastal regional background site, including sea salt (7.6%) and shipping emissions (9.4%), regarded as major local anthropogenic sources.

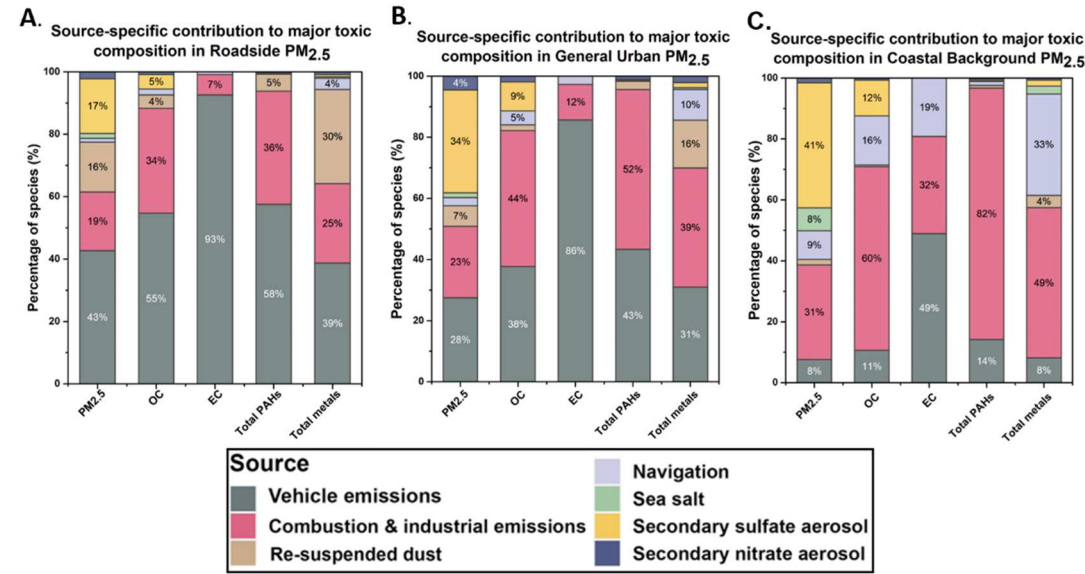


Figure 5-2 Source contribution to key chemical components of PM_{2.5} in Hong Kong 2021. A) Roadside; D) General urban; C) Coastal background PM_{2.5}.

5.2 Regional effect assessment

To investigate the monthly variation of the source apportionment and assess the regional effect on the mass loading of PM_{2.5} in Hong Kong, the factor contributions of each sample output from the PMF model were sorted by sampling month. The average mass loadings of sources were calculated month by month, site by site, as illustrated in **Fig. 5-3**. Owing to instrument failure and abnormal data unsatisfactory with the PMF calculation, monthly source apportionment results were lacking for Jun and July 2021 on the 6F site, February 2021, and January 2022 on the HT site. The monsoon

transitioned from a southwestern oceanic flow to a northeastern mixed continental wind pattern in mid-September 2021 at HK, as indicated by the weather records of the HK Observatory.

Current studies have shown that (1) local emission sources, such as vegetation, traffic emissions, and industrial activities, contribute to air pollution with negligible seasonal variation (Bodor et al., 2020; Shivani et al., 2019; Zhang et al., 2022), while (2) regional sources subject to monsoonal transport and meteorological conditions can vary seasonally in contributions to air pollutants (Im et al., 2018). This study employed two indicators to distinguish between local-dominant and regional-dominant sources, examined monthly variations in source profiles, and analyzed source-resolved back-trajectory airmass clusters.

Firstly, as shown in **Fig. 5-3**, sources exhibiting minimal seasonal variation were identified as primary local anthropogenic contributors to PM_{2.5} in Hong Kong, including on-road vehicle emissions and resuspension dust. In contrast, combustion & industrial emissions, as well as secondary aerosol formation processes, whose contributions varied seasonally, were characterized as being dominated by regional influences. Especially for the general urban and coastal background PM_{2.5}, the enhanced regional transport from the northern continent during winter heating was supported by a significantly increased proportion of combustion & industrial emissions (ranging from 6.76% to 16.7% in warm seasons and increasing to 36.6% to 41.2% in cold seasons). However, these combustion emissions may be confounded with local waste burning and cremation emissions near the general urban site. In addition, secondary aerosols also exhibited remarkable monthly fluctuation in their contributions to PM_{2.5}, echoing the previous source apportionment studies in Hong Kong (Cheng et al., 2015; Chow et al., 2022a; Wong et al., 2022). After the monsoon reversed, the decreased temperature altered rainfall, synoptic wind patterns, atmospheric boundary layer height, and aerosol source intensity (Sun et al., 2022), potentially facilitating the

formation of secondary aerosols. Compared with the coastal background samples, secondary nitrate aerosol contributed more to the urban PM_{2.5}, indicating the impact of anthropogenic activities. The increased contribution of secondary sulfate aerosol during the winter heating period in these three sites underscored the strong correlation with combustion emissions from regional transport. Beginning in mid-September, the monsoon reversed, and the decrease in temperature led to changes in rainfall, synoptic wind patterns, atmospheric boundary layer height, and aerosol source intensity (Sun et al., 2022). These factors can influence the patterns of aerosol and trace gaseous pollution, potentially leading to an increase in the contribution of secondary nitrate aerosol to total PM_{2.5}. However, the mechanism and primary artificial or natural contribution to the secondary nitrate aerosol generation are still hard to clarify. Concerning the second sulfate aerosol which is mainly traced by (NH₄)₂SO₄ in this study, the higher contributions to the total PM_{2.5} mass were observed in roadside and general urban samples than coastal background ones, indicating the pivotal role of artificial input. Moreover, the upsurges of the second sulfate aerosol contribution in all three sites during the winter heating period suggested the indispensable correlation with the coal combustion emission under the regional transport effect.

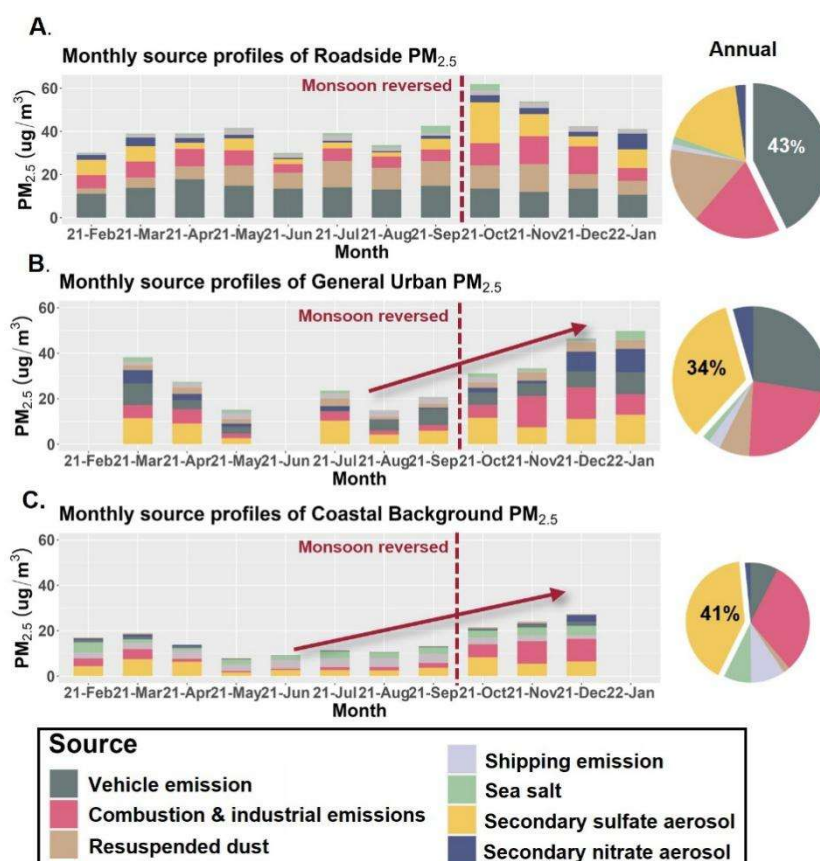


Figure 5-3 Monthly variation of source apportionment results in Roadside and Ambient PM_{2.5}. A) Roadside; D) General urban; C) Coastal background PM_{2.5}.

Subsequently, the differences in source-resolved clusters obtained by HYSPLIT and METEOTRACK between the warm and cold seasons can serve as another helpful indicator for determining whether the emission source is mainly influenced by local or regional diffusion. The regional effect was more detailedly investigated with back trajectory cluster analysis with the profiles outputted from Hysplit and Meteotrack software using the meteorological data from the 1° horizontal resolution Global Data Assimilation System (Stein et al., 2015). The 24-hour back trajectories were calculated with a 6-hour interval for each month from July to September and November to January, then clustered as the average 24-hour air mass trajectories for warm and cold seasons. Three clusters of air mass were summarized in both warm and cold seasons. Illustrated in the upper subfigures in **Fig. 5-4**, the average 24-hour air mass in warm seasons was

dominated by the relatively clean southern and southeastern oceanic clusters 1 and 2 (40.9% and 43.0%) and the western continental cluster 3 (16.2%), representing the short-distance transport. After the monsoon reversed, the significant contributors to the air mass in Hong Kong changed to long-term northeastern coastwise cluster 1 (53.5%) and northern continental clusters 2 and 3 (21.6% and 24.9%). Without local winter heating or seasonal industrial emissions in Hong Kong, combustions & industrial emissions with significant differences in contribution to air mass clusters were almost attributed to regional transport. Besides, our results also showed an increased association of secondary sulfate aerosol with coastwise northeastern clusters in cold seasons, which was identified as a regional-dominant source, consistent with the abovementioned evidence.

However, the back-trajectory cluster analysis can only determine the direction of air mass but cannot accurately estimate the transport distance, which may lead to some interpretation bias. For example, shipping emissions showed a strong association with clusters 1 and 2, which may not be attributed to long-term transport but rather to resuspension by the prevailing southern wind in the coastal area. The effect of mixing various local sources with complicated regional inputs makes it even harder to separate the local and regional air mass loadings accurately.

The average 24-hour air mass in the warm season is composed of 40.9% southern oceanic cluster 1, 43.0% eastern oceanic cluster 2, and 16.2% western continental cluster 3, representing the short-distance transport. Connecting with the source apportionment result, the contribution of cluster-associated air mass to the major non-local emission sources could be evaluated, which was shown in the bottom part of **Fig. 5-4**. Generally, the dominated clean oceanic air mass brought relatively few air mass loadings in the warm season, except for the shipping emission and heavy oil combustion contributed by the southern cluster 1 and eastern cluster 2 from the open sea. After the monsoon reversed, the major contributors to the air mass in HK changed to 53.5% long-

term northeastern coastwise cluster 1, 21.6%, and 24.9% northern continental cluster 2 & 3. As **Fig. 5-4B** showed, the loadings emitted from combustion were significantly accumulated by the northern continental transport in the cold season. It is worth noting that the second nitrate aerosol also increased sensitively, contributed mainly by clusters 1 and 2 mixed with the combustion emissions at the coastal background site. However, for general urban sites affected by mixed local sources and complicated regional input, the evaluation was still limited to separating the local and non-local air mass loadings.

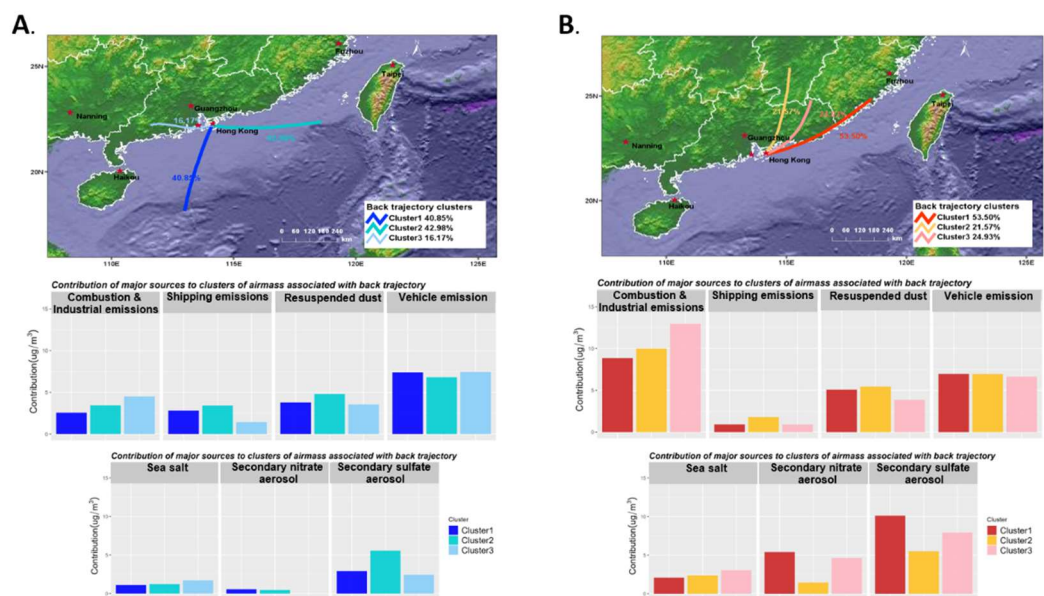


Figure 5-4 24h-back trajectory clusters-associated air mass affected the typical nonlocal sources of $\text{PM}_{2.5}$ in Hong Kong during warm and cold seasons in 2021. A) Warm season (July to September). B) Cold season (November to January).

5.3 Source-resolved acute and chronic health impact

Previous studies have tended to focus on source apportionment based on chemical profiles or cytotoxicity separately. While these studies have highlighted the prevalence of clarifying the sources and toxicity mechanism of the key toxic components in $\text{PM}_{2.5}$, there is still a significant research gap in our understanding of addressing the source-

resolved cytotoxicity, which may enhance the current model-based health risk assessment in the *in vitro* cell assay way.

Combining all analyzed components (*i.e.*, ten trace metals, twelve PAHs, and EC), traffic-related emissions, including vehicle emissions, resuspension road dust, and shipping emissions, were identified as the major local contributors threatening both acute and chronic inhalation health impacts in Hong Kong (**Fig. 5-5**). Firstly, vehicle emissions emerged as the predominant source of intracellular ROS induction (47.5%) and chronic health risks (44.6% HQ and 45.1% CR) in roadside PM_{2.5}, which were attributed to the high contents of EC and carcinogenic metals, Cr and Co. In the general urban samples, vehicle emissions contributed 44.0% of intracellular ROS, with a 27.5% mass contribution, while decreasingly contributing to chronic carcinogenic risk (32.0%). Besides vehicular emissions, non-exhaust emissions should be given importance in further acute-toxic-oriented pollution control, evidenced by the resuspended dust contributing about 34.7% to intracellular ROS in the roadside PM_{2.5}. However, the risks associated with re-suspension dust were reduced with long-term exposure and distance from on-road traffic. This was because Fe, Mn, and Zn, which are abundant in resuspension dust, while posing minimal long-term carcinogenic risks, can induce acute cytotoxicity through ROS induction and increase the non-cancer health hazards. In addition to on-road traffic, the importance of marine transport emissions was underscored in coastal background aerosol, with shipping emissions responsible for 29.6% of intracellular ROS potencies and even contributing increasingly to chronic health risks (37.1% HQ and 46.4% CR) induced by all analyzed components. This is attributed to the carcinogenic metals Cr, Co, and Ni present considerably in shipping emissions (**Fig. 5-1**).

Besides the local traffic-related sources, a mixed source containing combustion (from coal and biomass burning) and industrial emissions, which dominated the regional effect, was also identified as a primary contributor to inhalation health impacts in Hong

2740 Kong. Specifically, combustion & industrial emissions with high contents of As, Cr,
2741 Pb, and HMW-PAHs contributed between 15.7% and 29.9% to total ROS generation
2742 in roadside and general urban sites, while increasing to 49.0% in the coastal background
2743 PM_{2.5}. Moreover, combustion & industrial emissions were significant latent hazards
2744 associated with prolonged inhalation risk for Hong Kong aerosol. This mixed source
2745 contributed to approximately one-third of the carcinogenic and non-cancer health
2746 hazards in general urban PM_{2.5}, and nearly half in coastal background samples. The
2747 greater impact observed in coastal background samples may be attributed to their
2748 relatively high sampling positions and open surroundings, which make them more
2749 susceptible to seasonal regional air mass distribution, particularly during winter, when
2750 northern continental transport occurs. (Liao et al., 2020, 2021).

2751 The contribution of sources to health impacts often hardly aligns with their share of the
2752 total PM_{2.5} mass. For example, combustion & industrial emissions, despite constituting
2753 only 31% of the coastal background PM_{2.5} mass, primarily contributed to acute
2754 intracellular ROS (49.0%) and chronic health risks (45.8% HQ and 42.5% CR) induced
2755 by all analyzed components (**Fig. 5-5C**). This is attributed to their relatively high
2756 contents of compounds with severe individual carcinogenic risk. Another example is
2757 shipping emissions in the coastal background PM_{2.5}, despite accounting for only 9.35%
2758 of the total PM_{2.5} mass, which emerged as a significant source for both intracellular
2759 ROS potencies (29.6%) and chronic health risks (*i.e.*, 46.4% CR and 37.1% HQ) (**Fig.**
2760 **5-5C**). Conversely, secondary sulfate aerosol, which contributed a significant portion
2761 of PM_{2.5} mass (17.5%, 33.7%, and 41.1% of roadside, general urban, and coastal
2762 background PM_{2.5}, respectively), primarily consisted of water-soluble ions, resulting in
2763 negligible health impacts at all three sites in this study. However, a recent study also
2764 indicated that secondary sulfate aerosol barely made a direct contribution to
2765 intracellular ROS but may affect the toxic potencies of PM_{2.5} by indirect mechanisms
2766 such as increasing acidity and solubility of metals (Song et al., 2024). A key limitation

that warranted acknowledgment here was that the denominator of the pie chart reflects only the additive effects induced by metals, EC, and PAHs, rather than the **overall effects** of PM_{2.5} (**Fig. 5-5**). This distinction was necessary because the chronic health risks associated with the entire PM_{2.5} mixture remained unquantifiable at present. Thus, we also need to clarify that other unanalyzed substances also contributed to cytotoxicity and inhalation-related health risks, though their proportion remains unclear, particularly for coastal background samples. Nevertheless, we maintained that the discussion based on this pie chart remained meaningful, as it offered a critical framework for systematically extending source apportionment from mass concentration to acute cytotoxicity, and ultimately to long-term inhalation risks.

Overall, to effectively control PM_{2.5}-induced hazards, targeting local and regional anthropogenic sources that contain specific toxic components is crucial rather than solely considering their contributions to general mass concentrations.

Despite our mixed toxicity model, which the CA-model has evidenced following the previous study, that the effects of the individual toxins are additive at low exposure concentrations corresponding to the real concentration of HK PM_{2.5}, there is still an inevitable defect in investigating the indistinct combined effect induced by the addition or antagonism between real pollutants. However, our source-resolved cytotoxicity provided an intersectional view, helping better understand how the emission sources dominated the observed cytotoxicity in the human epithelial cell assay.

As a whole, our source-resolved cytotoxicity research demonstrated (1) the inconsistency between mass loadings, the *in vitro* toxicity effect, and chronic health risks contributed by the same source; (2) future toxicity-oriented air pollution control should attach importance to local traffic-related sources, including non-exhaust emissions; (3) the cytotoxicity and inhalation health risks were commensurately influenced by extraneous airmass distribution mixed with local emissions (*e.g.*,

combustions & industrial emissions and secondary sulfate aerosol); (4) the local fresh on-road traffic emissions were more dominant in the acute intracellular ROS induction, while the regional sources under long-term transport such as combustion & industrial emissions still a leading danger to chronic inhalation health risks in Hong Kong.

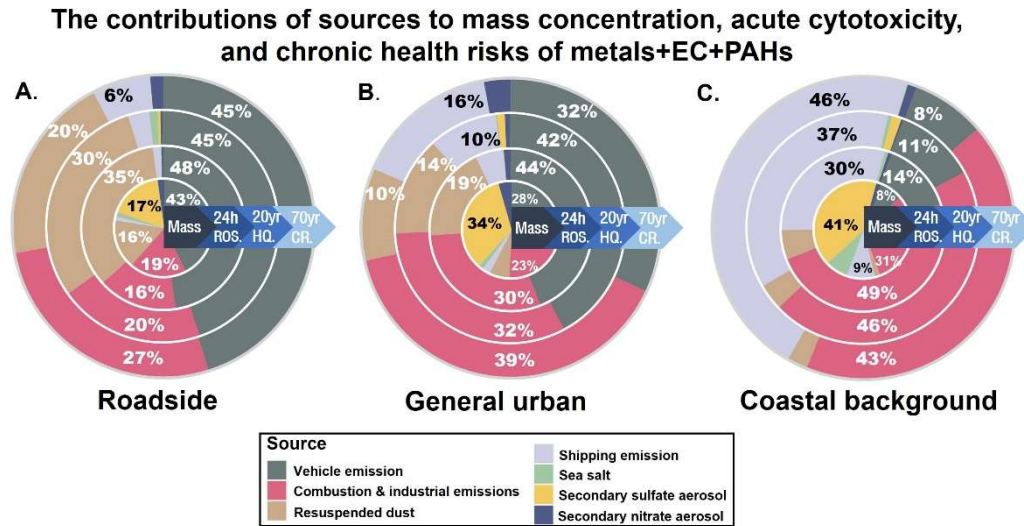


Figure 5-5 Source-resolved contributions to overall mass concentration, acute oxidative stress induction, and chronic health risks induced by key toxic components (metals+EC+PAHs) of PM_{2.5} in Hong Kong. A) Roadside; B) General urban; C) Coastal background. A limitation that needs to be acknowledged here is that the denominator of the pie chart represents the additive effects induced by metals, EC, and PAHs instead of overall effects, as the chronic health risks induced by the overall PM_{2.5} mixture are still unquantifiable

5.4 Summary

This chapter systematically investigated the source-resolved health impacts of PM_{2.5} in Hong Kong, integrating source apportionment, regional effect assessment, and analyses of acute and chronic health risks associated with specific emission sources. By applying a 7-factor PMF model and combining back-trajectory cluster analysis, this chapter identified key sources of PM_{2.5} and their contributions to toxic components and health

outcomes, providing insights into the linkages between emission sources, pollutant composition, and health impacts. The main findings were summarized below:

(1) Seven primary sources of Hong Kong PM_{2.5} were identified using the PMF model: vehicle emissions, combustion & industrial emissions (regional transport-dominated), re-suspended dust, shipping emissions, sea salt, secondary sulfate aerosol, and secondary nitrate aerosol. Vehicle emissions were the dominant contributor to roadside PM_{2.5} (43% of mass), driven by high levels of EC, hopanes, and light-molecular-weight PAHs. In general urban area, secondary sulfate aerosol (34%) and vehicle emissions (28%) were primary sources, while coastal background PM_{2.5} was dominated by secondary sulfate aerosol (41%) and combustion & industrial emissions (31%), reflecting strong regional transport influences.

(2) A significant regional effect driven by the monsoon was observed based on source-resolved air mass clusters. Warm seasons (*i.e.*, July–September) were dominated by clean oceanic air masses, reducing regional pollutant inputs. Cold seasons (*i.e.*, November–January) saw increased northern continental air masses, enhancing contributions from combustion & industrial emissions (36.6–41.2% in cold seasons vs. 6.76–16.7% in warm seasons) and secondary aerosols, particularly in urban and background areas. This confirmed the significant role of regional transport in shaping PM_{2.5} composition and toxicity in Hong Kong.

(3) For source-resolved acute cytotoxicity, traffic-related sources (*i.e.*, vehicle emissions and re-suspended dust) were the primary contributors to roadside ROS (47.5% and 34.7%, respectively), driven by EC, Fe, and Zn. In coastal background areas, shipping emissions (29.6%) and combustion & industrial emissions (49.0%) dominated ROS induction, linked to Cr, Ni, and As.

(4) Regarding the chronic inhalation health risks, vehicle emissions contributed the most to roadside carcinogenic (CR: 45.1%) and non-carcinogenic (HQ: 44.6%) risks, attributable to Cr and Co. Combustion & industrial emissions dominated background areas (CR: 42.5%; HQ: 45.8%), while shipping emissions were significant in coastal regions (CR: 46.4%; HQ: 37.1%) due to Cr and Ni.

(5) There is a non-negligible discrepancy between mass and health impact. For example, sources like secondary sulfate aerosol, despite high mass contributions (17.5–41.1%), had negligible health impacts, whereas low-mass sources (*e.g.*, shipping emissions, 9.35% in background) exerted substantial toxic effects, emphasizing the need for toxicity-oriented pollution control.

In a word, this chapter clarified the key sources driving PM_{2.5}-related health impacts in Hong Kong, highlighting the critical role of local traffic emissions in acute biological toxicity and the growing concern about regional sources for chronic health risks. The next chapter shifted the perspective to bioaccessibility-dependent health impacts, exploring how the bioaccessibility of these analyzed components modulates their actual biological effects and health risks.

6 Bioaccessibility-dependent Health Impact of Major Toxic Components in PM_{2.5}

Based on the experiments in previous chapters, we realized that bioaccessibility also exerted influence on the inhalation health risks and cytotoxicity. As an enhancing investigation, this chapter tried to initially address the unclear questions: (1) what implications bioaccessibility have for human exposure and health risks; (2) how the cytotoxic effects of PM_{2.5} differ among its bioaccessible and lung-fluid-insoluble fractions.

6.1 Inhalation bioaccessibility of key toxic components

Bioaccessibility, on the other hand, refers to the fraction of a compound that is released and available for absorption in the body after ingestion or inhalation. It is influenced by factors such as the physical and chemical properties of the compound, the matrix in which it is present, and the physiological conditions of the exposure route (*e.g.*, gastrointestinal tract, respiratory system). Consequently, the bioaccessibility of key constituents in PM_{2.5}, including metals and PAHs, may result in mechanism-based biases when evaluating inhalation-induced health effects.

6.1.1 Bioaccessibility of PM_{2.5}-bound trace metals

The pulmonary bioaccessibility of trace metals was quantified with the SELF-extraction mentioned above. The bioaccessible ratio related to the total mass concentration of metals in this study was clustered and illustrated in **Fig. 6-1**. For the analyzed trace metal elements, bioaccessibility varied with distance from the dominant emission sources, airborne abundance, chemical speciation, and matrix effects.

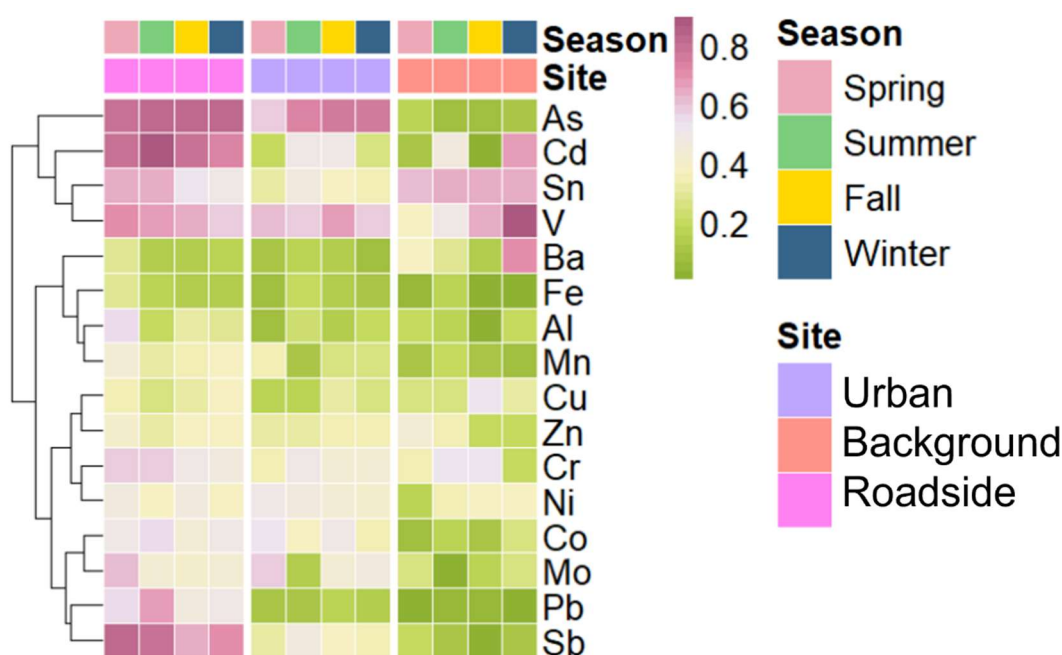


Figure 6-1 Parallel comparison and cluster analysis of trace metal bioaccessibility in Hong Kong PM_{2.5}.

For roadside PM_{2.5}, the overall means of bioaccessible concentration for all analyzed elements were in order of As (81.3%) > Cd (81.3%) > V (65.9%) > Pb (56.8%) > Cr (53.7%) > Co (50.2%) > Ni (40.9%) > Mn (36.9%) > Zn (35.8%) > Cu (31.9%) > Fe (17.7%); While for general urban samples, the order were listed as As (69.3%) > V (60.6%) > Ni (45.4%) > Co (42.9%) > Cr (41.8%) > Cd (34.4%) > Zn (32.4%) > Mn (26.0%) > Cu (26.9%) > Pb (12.8%) > Fe (12.7%), which is almost in similar order to some extent with roadside ones, except Pb and Cd. For Pb in general urban samples, the higher content of sulfate and organic compounds in the PM_{2.5} prompted the formation of stable forms, including PbSO₄, PbO, and Pb-organic complexes (Luo et al., 2019; Xie et al., 2019). The significant deviation in bioaccessibility of Cd between roadside and general urban PM_{2.5} is attributed to an extremely low content, even less than the detection limitation of Cd in general urban samples. Regarding the cluster analysis, the dust-composed elements Al, Ba, and Fe are consistently the most insoluble in SELF, which indicates relatively limited concern for pulmonary health. On the contrary, the trace metals, V, and Cr, which are typically generated by fossil fuel

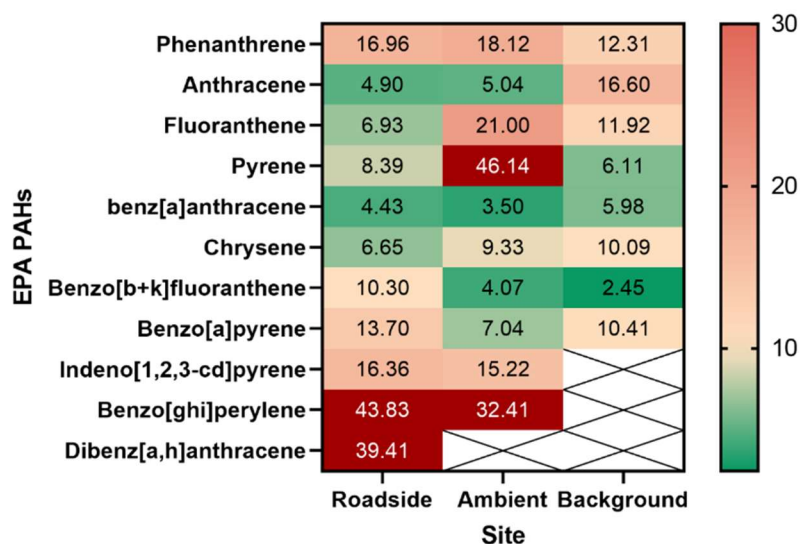
combustion and industrial emissions, showed not only greater unit toxicity but also higher bioaccessibility and exchangeability, representing a non-negligible contribution to inhalation health risk assessment. Comparing with roadside samples with general urban ones, the bioaccessibility of major harmful metals, including As, Cr, Mn, Pb, V are higher in the PM_{2.5} affected significantly by traffic-related emission than those more depended on coal combustion emission which as reported by previous studies (Julien et al., 2011; Luo et al., 2019; Ren et al., 2021). The difference could be interpreted as follows: metals from vehicle emissions are often absorbed on particle surfaces or in a free state, with little restriction, whereas those in coal combustion-related particles are typically associated with complex mineral matrices (Xie et al., 2019).

Table 6-1 Seasonal average bioaccessibility (mean $\pm$ S.D.) (%) of major metals of PM_{2.5} in Hong Kong 2021.

	Roadside				Ambient				Coastal background			
	spring	summer	fall	winter	spring	summer	fall	winter	spring	summer	fall	winter
As	77.65% $\pm$ 12.58	83.20% $\pm$ 3.43	81.26% $\pm$ 10.27	82.88% $\pm$ 6.53	56.56% $\pm$ 29.79	72.02% $\pm$ 16.32	75.94% $\pm$ 5.70	74.71% $\pm$ 3.49	16.10% $\pm$ 8.23	6.51% $\pm$ 2.77	8.98% $\pm$ 0.74	9.57% $\pm$ 0
Ba	27.94% $\pm$ 14.26	14.86% $\pm$ 4.70	13.99% $\pm$ 4.03	15.63% $\pm$ 2.55	10.59% $\pm$ 8.64	16.61% $\pm$ 8.03	12.74% $\pm$ 4.31	8.56% $\pm$ 2.97	38.12% $\pm$ 29.97	45.69% $\pm$ 20.18	12.4% $\pm$ 4.95	68.83% $\pm$ 39.01
Cd	77.93% $\pm$ 9.04	89.13% $\pm$ 4.55	80.09% $\pm$ 8.40	72.60% $\pm$ 8.05	20.71% $\pm$ 6.86	50.37% $\pm$ 11.33	50.55% $\pm$ 22.54	25.70% $\pm$ 7.87	9.59% $\pm$ 2.77	45.69% $\pm$ 24.77	0.46% $\pm$ 0	23.98% $\pm$ 7.04
Co	47.87% $\pm$ 22.55	56.52% $\pm$ 12.79	42.09% $\pm$ 23.48	48.14% $\pm$ 15.82	52.53% $\pm$ 17.87	36.48% $\pm$ 13.31	48.27% $\pm$ 14.82	34.88% $\pm$ 8.69	8.90% $\pm$ 4.12	17.96% $\pm$ 13.62	10.54% $\pm$ 1.24	23.98% $\pm$ 10.48
Cr	57.77% $\pm$ 9.23	56.73% $\pm$ 6.68	48.92% $\pm$ 6.43	46.80% $\pm$ 5.96	33.19% $\pm$ 14.85	48.67% $\pm$ 6.31	42.33% $\pm$ 16.98	44.46% $\pm$ 6.44	35.42% $\pm$ 16.14	53.35% $\pm$ 33.92	52.23% $\pm$ 23.38	20.67% $\pm$ 1.87
Cu	33.86% $\pm$ 13.13	26.65% $\pm$ 3.06	31.80% $\pm$ 8.01	38.55% $\pm$ 5.09	22.91% $\pm$ 6.17	28.20% $\pm$ 4.41	31.93% $\pm$ 5.41	25.18% $\pm$ 4.01	26.90% $\pm$ 5.76	26.89% $\pm$ 8.38	51.9% $\pm$ 6.66	9.04% $\pm$ 0
Fe	28.09% $\pm$ 12.55	15.18% $\pm$ 4.80	12.37% $\pm$ 5.39	12.97% $\pm$ 5.94	7.67% $\pm$ 2.22	20.41% $\pm$ 6.85	13.41% $\pm$ 2.21	10.94% $\pm$ 2.97	5.21% $\pm$ 2.79	16.77% $\pm$ 14.63	3.21% $\pm$ 1.26	1.67% $\pm$ 0.11
Mn	44.58% $\pm$ 7.86	32.74% $\pm$ 7.84	34.50% $\pm$ 6.25	36.32% $\pm$ 8.15	33.24% $\pm$ 6.47	14.09% $\pm$ 8.01	26.29% $\pm$ 6.01	25.78% $\pm$ 7.16	11.84% $\pm$ 9.06	19.15% $\pm$ 1.96	9.77% $\pm$ 2.26	9.04% $\pm$ 7.0
Mo	59.74% $\pm$ 5.48	42.16% $\pm$ 13.10	41.43% $\pm$ 12.79	44.29% $\pm$ 7.28	58.79% $\pm$ 14.54	12.72% $\pm$ 9.23	43.97% $\pm$ 7.50	46.20% $\pm$ 10.46	25.73% $\pm$ 2.28	1.54% $\pm$ 1.41	16.31% $\pm$ 0	24.33% $\pm$ 23.66
Ni	45.42% $\pm$ 5.11	37.22% $\pm$ 12.06	47.35% $\pm$ 7.50	36.71% $\pm$ 11.90	49.68% $\pm$ 5.13	45.61% $\pm$ 5.57	44.58% $\pm$ 5.41	41.43% $\pm$ 7.73	15.77% $\pm$ 8.93	34.78% $\pm$ 13.95	5.45% $\pm$ 5.65	37.01% $\pm$ 2.87
Pb	55.65% $\pm$ 21.27	67.72% $\pm$ 19.39	45.03% $\pm$ 10.32	48.14% $\pm$ 11.79	10.85% $\pm$ 2.88	12.04% $\pm$ 4.42	16.70% $\pm$ 4.51	12.25% $\pm$ 4.29	2.87% $\pm$ 3.03	3.3% $\pm$ 0	5.45% $\pm$ 5.41	1.59% $\pm$ 1.13
Sn	64.85% $\pm$ 12.79	63.06% $\pm$ 6.16	52.75% $\pm$ 6.74	50.52% $\pm$ 6.16	30.97% $\pm$ 13.55	47.14% $\pm$ 7.76	36.90% $\pm$ 11.89	35.54% $\pm$ 8.81	61.05% $\pm$ 0	65.04% $\pm$ 28.20	64.01% $\pm$ 0	62.85% $\pm$ 0
V	71.28% $\pm$ 6.64	67.55% $\pm$ 5.44	63.20% $\pm$ 12.48	58.44% $\pm$ 16.17	60.50% $\pm$ 5.21	59.08% $\pm$ 10.04	66.44% $\pm$ 3.70	57.15% $\pm$ 16.89	36.284% $\pm$ 9.84	49.8% $\pm$ 16.70	64.94% $\pm$ 5.75	88.86% $\pm$ 0
Zn	41.30% $\pm$ 8.86	30.38% $\pm$ 8.03	36.70% $\pm$ 5.92	37.50% $\pm$ 7.80	30.12% $\pm$ 6.09	31.69% $\pm$ 6.46	34.73% $\pm$ 1.41	33.33% $\pm$ 5.53	42.33% $\pm$ 16.98	35.44% $\pm$ 25.79	19.75% $\pm$ 12.25	18.59% $\pm$ 3.68
Phe	17.64% $\pm$ 10.41	17.00% $\pm$ 5.09	22.11% $\pm$ 9.22	11.08% $\pm$ 7.71	24.18% $\pm$ 13.49	20.01% $\pm$ 17.06	8.14% $\pm$ 1.74	20.14% $\pm$ 15.82	7.12% $\pm$ 0.58	13.55% $\pm$ 7.15	6.10% $\pm$ 2.15	11.93% $\pm$ 13.90
Ant	5.39% $\pm$ 2.16	4.95% $\pm$ 1.45	4.7% $\pm$ 0.67	4.54% $\pm$ 1.29	5.80% $\pm$ 4.133	3.678% $\pm$ 2.18	2.77% $\pm$ 0.89	7.91% $\pm$ 2.05	19.15% $\pm$ 11.78	23.07% $\pm$ 0	11.48% $\pm$ 0.82	12.68% $\pm$ 10.02
Flu	9.3% $\pm$ 11.51	5.34% $\pm$ 5.43	9.18% $\pm$ 10.33	3.91% $\pm$ 5.22	24.27% $\pm$ 15.91	17.23% $\pm$ 11.97	21.16% $\pm$ 12.73	21.33% $\pm$ 8.56	3.79% $\pm$ 2.04	27.1% $\pm$ 0	13.12% $\pm$ 4.69	3.67% $\pm$ 2.42
Pyr	2.39% $\pm$ 13.18	6.75% $\pm$ 7.73	9.91% $\pm$ 15.94	6.39% $\pm$ 10.36	43.2% $\pm$ 37.15	26.17% $\pm$ 18.82	50.20% $\pm$ 28.87	65.00% $\pm$ 0.34	3.89% $\pm$ 3.84	10.52% $\pm$ 7.62	4.44% $\pm$ 0.22	5.57% $\pm$ 3.97
BaA	2.39% $\pm$ 1.55	5.66% $\pm$ 3.52	8.75% $\pm$ 10.52	0.91% $\pm$ 0.55	2.5% $\pm$ 2.17	5.97% $\pm$ 3.32	3.74% $\pm$ 3.15	1.78% $\pm$ 0	3.46% $\pm$ 0.20	14.53% $\pm$ 8.29	3.76% $\pm$ 0.23	2.18% $\pm$ 0.31
Chr	6.17% $\pm$ 1.18	7.06% $\pm$ 4.16	9.00% $\pm$ 6.87	4.37% $\pm$ 0.94	7.92% $\pm$ 7.06	9.25% $\pm$ 3.64	8.30% $\pm$ 4.84	11.86% $\pm$ 3.69	7.65% $\pm$ 2.34	n.a.	8.23% $\pm$ 0	14.4% $\pm$ 1.60
B[b+k]F	7.49% $\pm$ 8.57	24.73% $\pm$ 17.30	7.62% $\pm$ 6.70	1.35% $\pm$ 0.35	1.73% $\pm$ 1.78	5.52% $\pm$ 6.37	1.24% $\pm$ 0.80	7.77% $\pm$ 3.81	2.12% $\pm$ 1.55	6.64% $\pm$ 3.25	0.69% $\pm$ 0	0.34% $\pm$ 0.27
BaP	15.07% $\pm$ 10.75	28.12% $\pm$ 0	8.77% $\pm$ 2.34	2.84% $\pm$ 0.24	6.14% $\pm$ 3.68	13.21% $\pm$ 9.52	4.52% $\pm$ 3.20	4.29% $\pm$ 3.93	14.22% $\pm$ 8.66	11.66% $\pm$ 4.87	9.41% $\pm$ 1.74	6.36% $\pm$ 0.99
IcdP	n.a.	n.a.	n.a.	16.36% $\pm$ 0	n.a.	33.71% $\pm$ 0	4.61% $\pm$ 0	7.33% $\pm$ 5.24	n.a.	n.a.	n.a.	n.a.
BghiP	37.68% $\pm$ 25.67	n.a.	82.97% $\pm$ 0	10.83% $\pm$ 1.54	11.5% $\pm$ 5.90	61.92% $\pm$ 28.16	20.03% $\pm$ 17.16	36.18% $\pm$ 34.39	n.a.	n.a.	n.a.	n.a.

6.1.2 Bioaccessibility of PM_{2.5}-bound PAHs

Separated by liquid-liquid extraction with DCM, the organic phases were concentrated and purified for bioaccessible PAHs analysis using GC/MS. The bioaccessibility of EPA PAHs, as indicated by the most bioaccessible ratios falling within the range of 5-16%, varied depending on the specific compounds. Among the highly bioaccessible compounds were Phe, Flu, IcdP, BghiP, and DahA, while BaA, Chr, and B [b+k] F (the signal peaks of BbF and BkF were overlapped) were classified as low-bioaccessible compounds, as shown in **Fig. 6-2**. The bioaccessibility of these PAHs was influenced by both their mass concentration in real samples and their saturated water solubility, with bioaccessibility increasing as the mass concentration approaches solubility thresholds. Furthermore, when considering the same extraction conditions, PAHs with 5-6 rings from roadside samples exhibited higher bioaccessibility compared with those from ambient sources. However, there were a couple of high-molecular-weight PAHs that leaked valid bioaccessible ratios in coastal background PM_{2.5}, which was attributed to the extremely low contents that fell under the equipment detection limitation. Overall, except for BghiP and DahA, the bioaccessibility of most analyzed PAHs was negatively



correlated with molecular weight.

Figure 6-2 Parallel comparison of EPA PAHs' bioaccessibility in ambient and roadside PM_{2.5}.

6.1.3 Spatial comparison for bioaccessibility of key toxic components in PM_{2.5}

The PLS-DA of bioaccessibility data for analyzed metals and PAHs in PM_{2.5} samples revealed clear environmental gradients among roadside, urban, and background sites. The scores plot, accounting for 40.6% variance on Component 1 and 16.6% on Component 2, demonstrated robust separation, with background samples clustering tightly on the negative end of Component 1, indicative of lower overall bioaccessibility due to minimal anthropogenic influences (**Fig. 6-3A**). Urban and roadside samples shifted positively, reflecting heightened bioaccessibility from pollution sources, though with partial overlap suggesting shared vehicular and combustion impacts; roadside samples extended further along Component 1, highlighting traffic dominance, while urban samples showed greater spread on Component 2, likely from diverse regional industrial and combustion contributions.

Illustrated in **Fig. 6-3B**, Variable Importance in Projection (VIP) scores identified key discriminators (>1 threshold), including As, Sb, Mo, Pyr, Ba, Co, which exhibited marked inter-group variances in bioaccessibility percentages. These high-VIP variables underscored source-specific and speciation-driven differences between the bioaccessibility of key components. For example, As, Co, and Pyr, which mainly depend on diesel combustion and are rich in traffic emissions, exhibited higher bioaccessibility in roadside PM_{2.5}. It was possibly attributed to the close direct emission, finer size, and relatively low-level oxidative aging of the particles. In the coastal background environment, the salt and other components present in marine aerosols may also have an impact on the bioaccessibility of metals and PAHs. In addition, background sites featured uniquely higher Ba bioaccessibility, possibly from natural mineral sources,

contrasting with anthropogenic lows elsewhere. These findings implied elevated inhalation risks in urban and roadside areas, particularly for carcinogenic PAHs and metals, aligning with studies linking long-term exposure to lung cancer and emphasizing the need for site-specific mitigation.

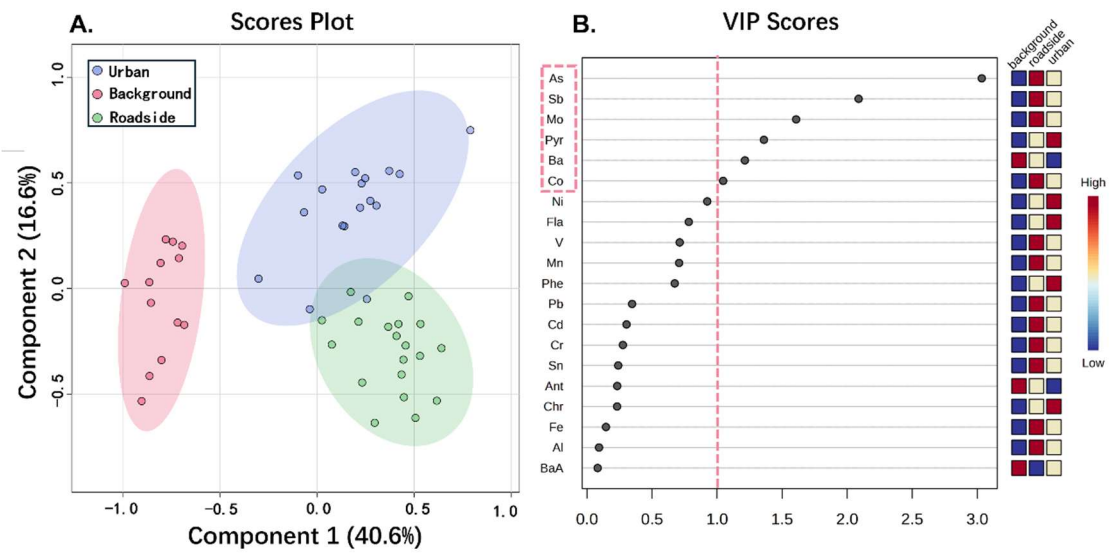


Figure 6-3 PLS-DA analysis result of key components’ bioaccessibility in PM_{2.5} collected from roadside, urban, and background sites. A) Score plot of major components in PLS-DA analysis; B) Variable Importance in Projection (VIP) scores identified key discriminators.

6.1.4 The effect of aerosol pH

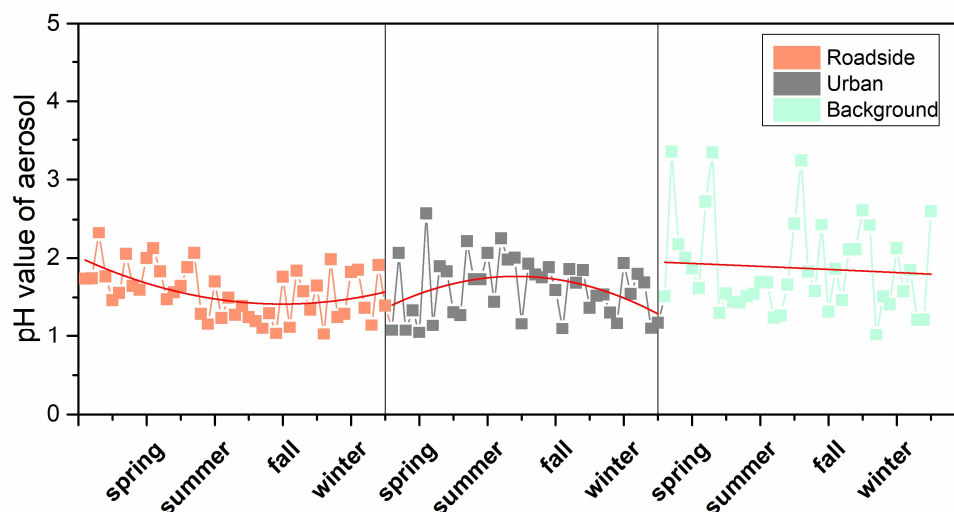


Figure 6-4 The in-situ pH value of aerosol with non-linear regression analysis

The investigation revealed that aerosol acidity in Hong Kong was governed by a complex interplay between site-specific emission profiles and seasonal meteorological shifts. Spatially, the roadside environment maintained the highest levels of acidity (mean pH ~ 1.55), primarily driven by local tailpipe exhaust and the acidic dissolution of resuspension dust. In contrast, the coastal background site exhibited significantly higher and more variable pH levels (mean pH ~ 1.87), where the influx of coarse sea salt and marine air masses provided a critical, albeit inconsistent, neutralizing capacity. Seasonal trends further exacerbated these spatial differences; during the winter monsoon, the transport of continental pollutants rich in secondary sulfates and nitrates consistently depresses pH across all monitoring sites. Conversely, the summer months, dominated by southerly marine air, show a marked increase in pH, suggesting that marine-derived cations played a vital role in mitigating the intense acidity typical of Hong Kong urban area.

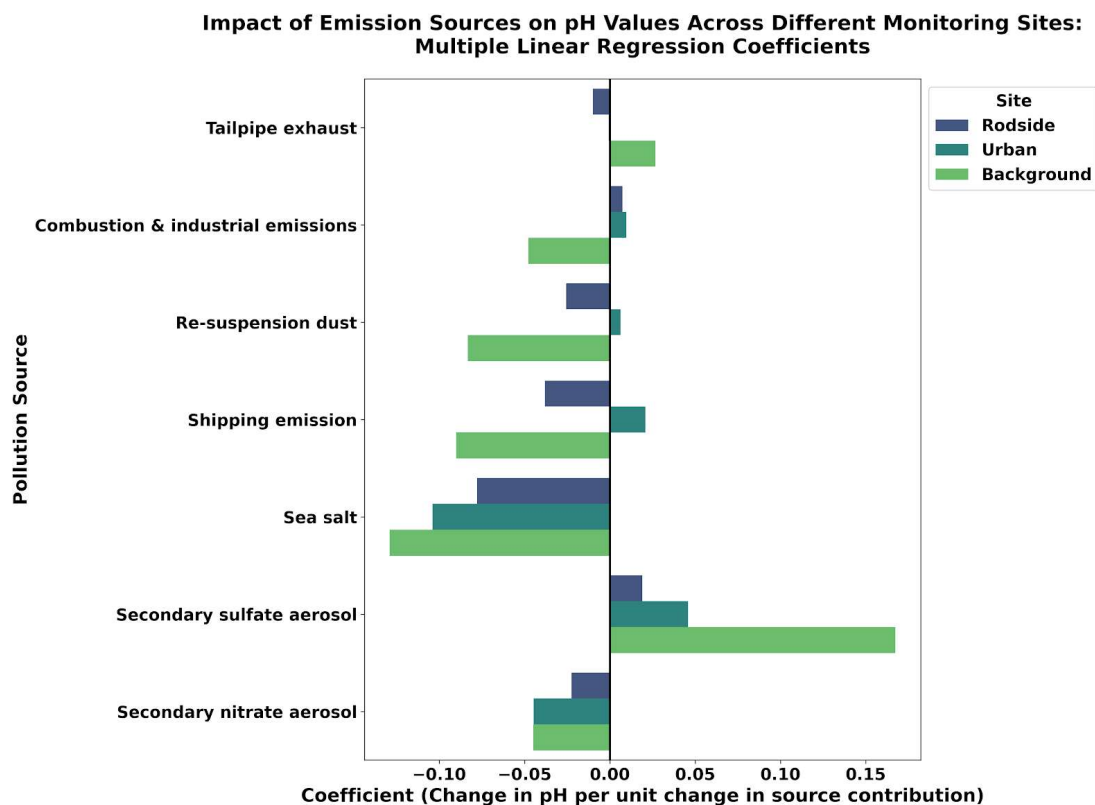


Figure 6-5 Multiple linear regression analysis of major sources and pH values in Hong Kong 2021

The multiple linear regression (MLR) analysis revealed that aerosol acidity (pH) is highly localized and governed by distinct source-receptor relationships across the three monitoring sites. At background and urban sites, secondary aerosol formation, particularly secondary sulfate, exhibited a significant positive correlation with pH, suggesting that sulfate-rich environments may be associated with partially neutralized aerosol populations. Conversely, the roadside site was uniquely dominated by primary mechanical and natural sources, where resuspended dust and sea salt significantly predicted increased acidity, likely due to insufficient neutralizing species to offset the acidic precursors inherent to high-traffic corridors. These findings imply that a "one-size-fits-all" atmospheric model cannot accurately predict pH; rather, local emission profiles, ranging from tailpipe exhaust to shipping emissions, interact with regional

secondary pollutants to create site-specific chemical profiles. For policymakers, this suggests that mitigating aerosol acidity requires targeted strategies: urban and background areas may benefit from managing secondary precursor gases, while roadside environments require focused control of non-exhausted road dust and tailpipe emissions.

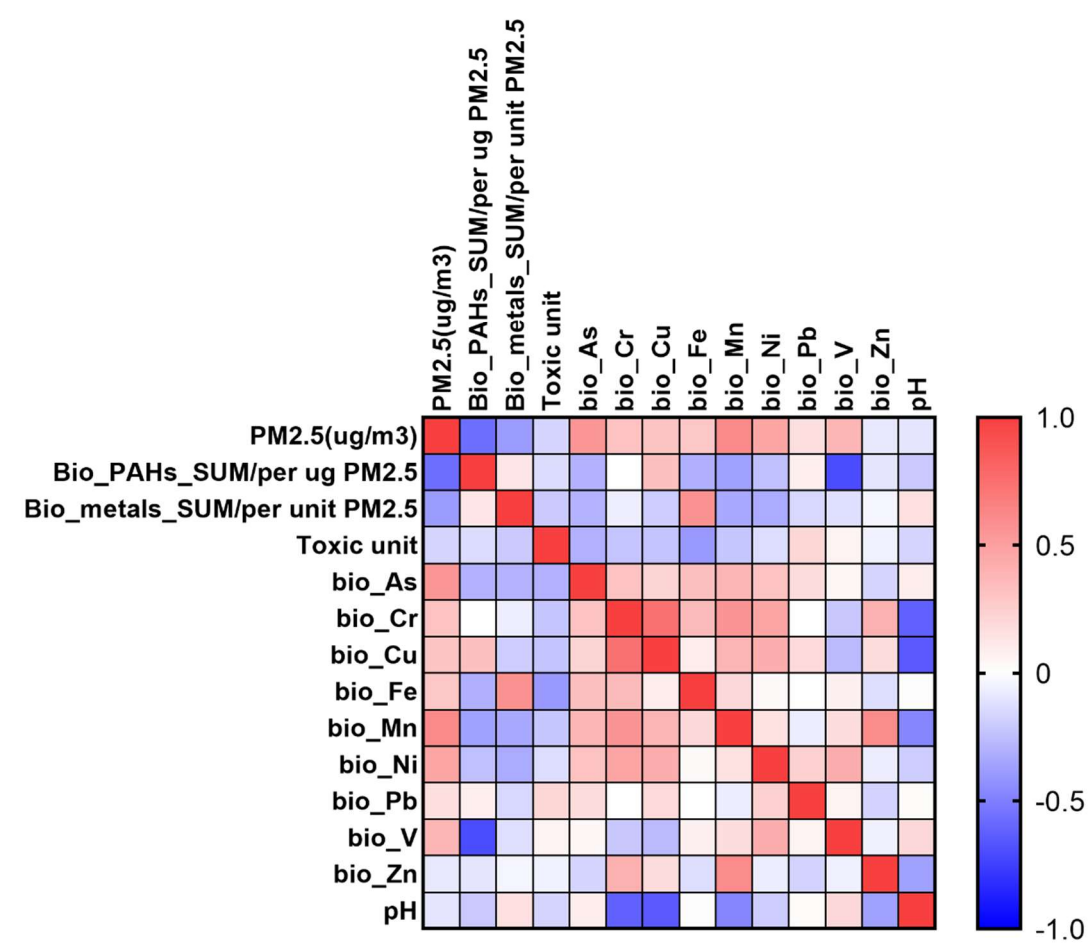


Figure 6-6 Correlation coefficient heatmap of bioaccessibility with toxic unit of ROS and in-situ pH of aerosol

These fluctuations in aerosol pH carried significant implications for the bioaccessibility of inhaled pollutants and their subsequent impact on public health. The MLR and correlation data indicated that the low-pH conditions prevalent at roadside and urban

sites facilitated the dissolution of transition metals, such as Cr, Cu, and Mn. Once solubilized, these metals became highly bioaccessible, potentially triggering ROS generation in the lung lining fluid and inducing systemic oxidative stress (Fang et al., 2017). Furthermore, the acidic environment may alter the chemical partitioning and degradation pathways of organic pollutants, such as PAHs, potentially leading to the formation of more toxic nitrated derivatives (Liu et al., 2023; Xie et al., 2018).

6.2 Bioaccessibility-adjusted inhalation health risks

The investigation into the impact of bioaccessibility on the total concentrations and inhalation CR of PAHs revealed a significant decrease of one magnitude in both parameters. Shown in **Fig. 6-7**, after SELF extraction, there is a 77.8%, 78.1% and 92.6% reduction of total PAHs concentration in roadside, urban, and coastal background samples, which is below the European Commission Standard, 1 ng/m³ (*EU Air Quality Standards - European Commission*, 2010). Moreover, it is notable that the increased content of 4-ring PAHs is in the bioaccessible fraction of general urban PM_{2.5}. Compared with the total PAHs loading, the fractional composition of 5~6 rings bioaccessible PAHs decreased in the ambient and background PM_{2.5}, while increased in the fresher PM_{2.5} collected at the roadside. In contrast to the PAH-induced health risk, for the lower molecular weight group, notably, the bioaccessibility factor played a pivotal role in the inhalation CR induced by 5-ring PAHs in roadside PM_{2.5}, as it was elevated in this location, whereas it decreased at the other two sites. Conversely, upon considering bioaccessibility, the fractional CR for 4-ring PAHs exhibited an increase, possibly attributable to varying degrees of particle aging emitted from diverse sources and undergoing transformations. These findings highlight the intricate relationship among bioaccessibility, PAH concentrations, and inhalation CR, shedding light on the complex dynamics of PAH exposure and the potential health risks associated with different PAHs in various environmental contexts.

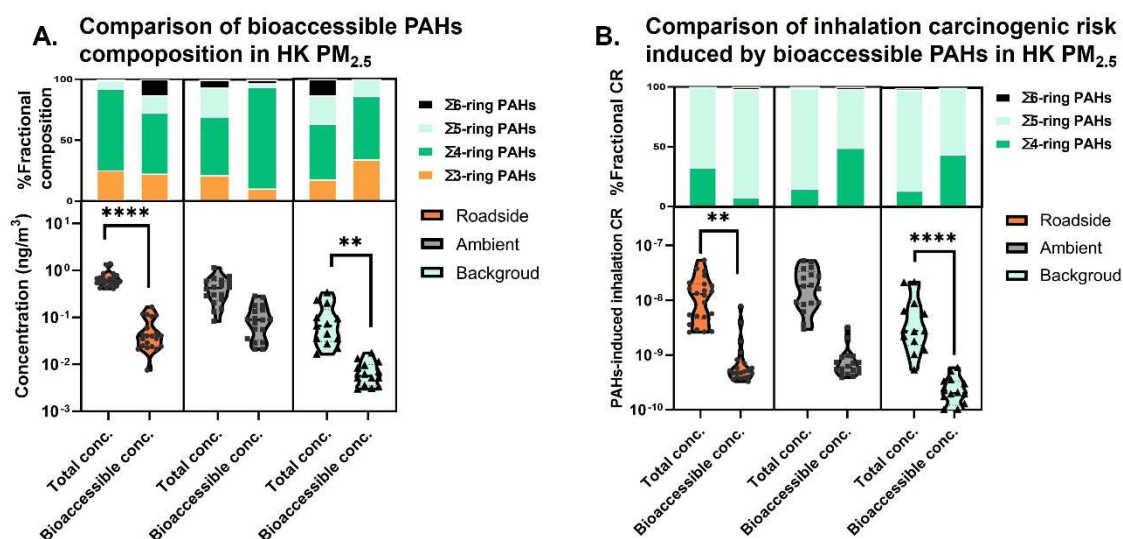


Figure 6-7 The comparison of composition and inhalation carcinogenic risk induced by bioaccessible and total PAHs in PM_{2.5}. A) Comparison of bioaccessible PAHs composition and concentration; B) Comparison of inhalation carcinogenic risk induced by bioaccessible PAHs in PM_{2.5}.

The source-resolved health risks induced by bioaccessible key components differed slightly from those based solely on mass concentration (**Fig. 6-8**). For metal-induced carcinogenic risks, as shown in the left two columns of each figure, the contributions from on-road traffic emissions (*i.e.*, vehicle emissions and resuspended dust) decreased to varying degrees after bioaccessibility was taken into account. This reduction primarily resulted from the fact that heavy metals prevalent in on-road traffic emissions typically existed in stable valence states and exhibited limited solubility, thereby reducing their bioaccessible fractions. In contrast, after considering bioaccessibility, the mixed regional source, which included combustion and industrial emissions, played a more significant role compared with the mass-based assessment. This shift was mainly attributed to the higher bioaccessibility of anthropogenic metals such as As and Cd, which were relatively abundant in combustion and industrial sources. Notably, the fractional contribution of shipping emissions also increased, particularly in coastal

background PM_{2.5}. This trend could be explained by hallmark element V, which exhibited relatively high bioaccessibility. These findings suggested that the realistic health risks associated with shipping emissions might have been underestimated in previous mass-based assessments.

For PAH-induced carcinogenic risks, there was little variation in the fractional contributions of sources in both urban and coastal background PM_{2.5}, with combustion and industrial emissions remaining the dominant sources. However, the PAH-induced carcinogenic risk associated with vehicle emissions decreased after accounting for bioaccessibility. This decrease was due to the predominance of low-molecular-weight PAHs in vehicle emissions, which generally exhibited lower solubility compared with high-molecular-weight PAHs. Overall, these results highlighted the importance of considering bioaccessibility in health risk assessments, as it provided a more realistic estimation of the potential health impacts from different emission sources. Ignoring bioaccessibility could lead to the underestimation or overestimation of health risks, particularly for sources with metals or PAHs that differed significantly in their solubility and bioaccessibility.

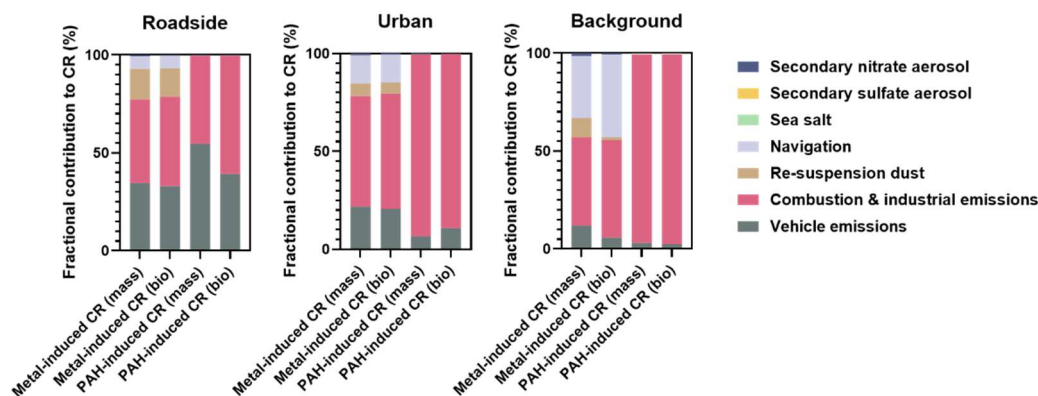


Figure 6-8 The comparison of source-resolved carcinogenic risk induced by key components based on mass and bioaccessible concentration

6.3 Cytotoxicity comparison of PM_{2.5} fractions via bioaccessibility

The relationship between bioaccessibility and cytotoxicity endpoints, such as cell viability and intracellular ROS, varied depending on the pollutant type, with metals (Fig. 6-9A and B) and PAHs (Fig. 6-9C) serving as key examples. For metals like Cd and As, bioaccessibility assessed through *in vitro* digestion models showed a moderate negative correlation with IC₅₀ ($R^2 = 0.2092$) and EC_{IR1.5} ($R^2 = 0.0679$), indicating that higher bioaccessibility enhances toxicity by increasing cellular uptake and inducing ROS generation or cell death. In contrast, PAHs such as BaP exhibited a weaker correlation ($R^2 = 0.0333$) between bioaccessibility and EC_{IR1.5}, suggesting that metabolic activation by enzymes like cytochrome P450 plays a more dominant role in toxicity, including ROS production (Xue & Warshawsky, 2005), than bioaccessibility alone. Correlation analyses further revealed that bioaccessibility of high-molecular-weight PAHs and certain metals (*e.g.*, Cd, BaP) positively influences ROS levels in PM_{2.5}-exposed cells. In contrast, low-bioaccessibility metals (*e.g.*, Fe, Mn) showed weaker or negative associations. These findings highlighted that bioaccessibility was a critical determinant of exposure dose and toxicity. Its impact was modulated by chemical speciation, metabolic processes, and cellular responses, necessitating a comprehensive evaluation in specific environmental and biological contexts.

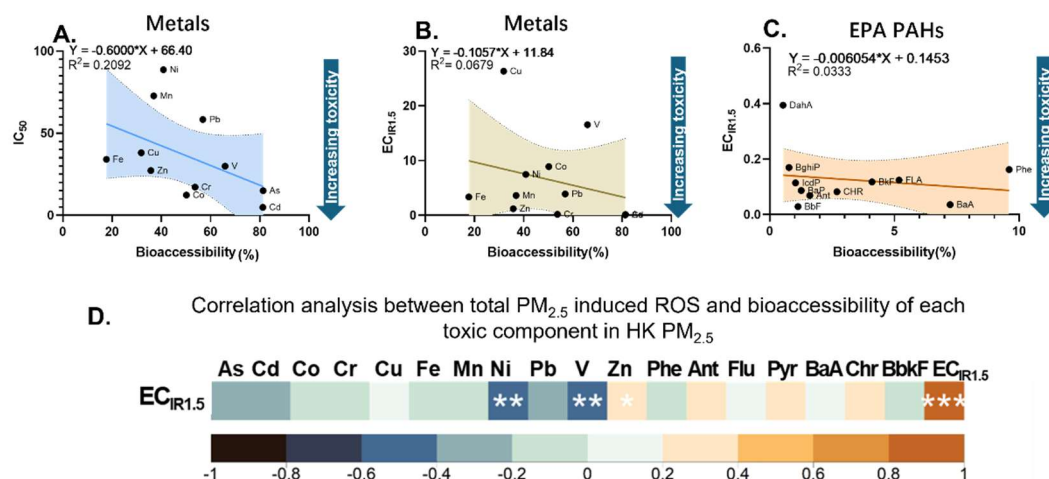


Figure 6-9 Linear regression analysis for bioaccessibility versus A) cell viability of metals; B) intracellular ROS potency of metals; C) intracellular ROS potency of PAHs. D) The Spearman correlation coefficient analysis of intracellular ROS potency of total PM_{2.5} with bioaccessibility of each toxic component (* for $p < 0.05$, ** for $p < 0.01$, and *** for $p < 0.001$).

The Spearman correlation coefficient was calculated for the tested trace metals and parent PAHs in PM_{2.5}. As shown in **Fig. 6-9D**, most metals exhibited a weakly negative correlation with total PM_{2.5}-induced EC_{IR1.5}, which decreased with increasing cytotoxicity. Meanwhile, the total PM_{2.5}-induced EC_{IR1.5} was positively related to the bioaccessibility of most tested PAHs. However, this was not enough to declare that the higher the biological absorption rate of PAHs, the smaller the toxic effect on cells. As the content of PAHs in PM_{2.5} in Hong Kong was relatively low, the lower the content of PAHs in the lung fluid, the higher the bioaccessibility. However, the lower the content of PAHs, the smaller the effect on cytotoxicity. It was worth noting that the bioaccessibility of V and Ni was significantly and negatively correlated with the EC_{IR1.5}(PM_{2.5}), indicating that the higher bioaccessibility of shipping emission-related components will enhance the cytotoxicity of total PM_{2.5}.

Generally, this investigation suggests that particulate-phase components of PM_{2.5}, which are insoluble in lung fluid, can also cause oxidative stress and cell death. Further study is still necessary to determine the distinct roles and contributions of PM_{2.5} fractions with varying bioaccessibility (or solubility in lung fluid) to the total PM_{2.5}-induced cytotoxicity.

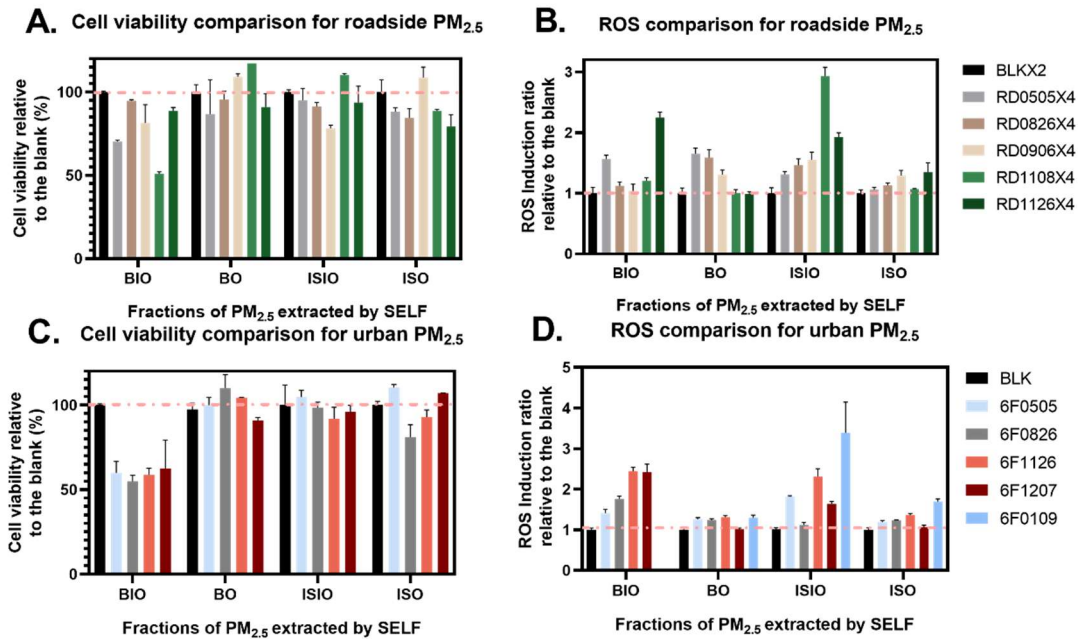


Figure 6-10 A) Cell viability comparison among four fractions with 4-time dilution separated from the overall traffic-dominant PM_{2.5}. B) Intracellular ROS comparison between four fractions with 2-time dilution separated from the overall traffic-dominant PM_{2.5}. C) Cell viability comparison between four fractions with a 4-time dilution separated from the overall general urban PM_{2.5}. D) Intracellular ROS comparison between four fractions with 4-time dilution separated from the overall general urban PM_{2.5}. **Abbr:** BIO = bioaccessible inorganic fraction, BO = bioaccessible organic fraction, ISIO = SELF-insoluble inorganic fraction, ISO = SELF-insoluble organic fraction, and SELF = simulated epithelial lung fluid.

The *in vitro* toxicity of PM_{2.5} fractions, categorized by bioaccessible and lung-fluid-insoluble properties as well as organic and inorganic compositions, showed significant

variation. The BIO fraction, likely enriched with traffic-related metals such as As (81.3%) and Cd (81.3%), reduced viability to 50%, reflecting increased bioaccessibility during colder months due to heightened traffic and heating emissions (Hernández et al., 2018). The ISIO fraction, which contained EC and dust-derived elements such as Fe (17.7%), exhibited negligible cell viability but induced marketable ROS. Based on our experimental observation and previous studies, this may be attributed to the physical properties, including: (1) a larger surface area increasing ROS generation; (2) induction of cell membrane disruption with ROS generation (G. Liu et al., 2021; Qi et al., 2019; D. Wang et al., 2013). In addition, the morphology and delicate size of particles may enhance the abovementioned effects (Zou et al., 2016). The BO fraction, potentially containing PAHs such as Phe and Flu, with a bioaccessibility of 5–16% (**Fig. 6-2**), induced ROS levels 1.5–2 times higher than those of the blank in November samples, likely due to metabolic activation enhanced by winter heating sources (Luo et al., 2021). It is worth noting that, under the same dilution ratio, the ISO fraction from both sites exhibited virtually no toxicity, with cell viability above 80% and ROS induction at near-baseline levels. This low toxicity likely resulted from organic compounds existing as insoluble particles in lung fluid, limiting epithelial cell uptake (Pelfrêne et al., 2012). Many existing studies have extracted PM_{2.5} organic matter using solvents like dichloromethane and employed co-solvents to enhance solubility in exposure media, potentially overestimating toxicity. These organics predominantly remain particulate in lung fluid, thereby reducing absorption and associated toxic effects (Innes et al., 2021; Oomen et al., 2003; Xue & Warshawsky, 2005). Investigating the key toxic components with higher bioaccessibility could provide critical insights, offering a more accurate representation of inhalation risks and challenging conventional exposure models.

Roadside and general urban samples differed markedly in toxicity profiles, driven by distinct emission sources. As displayed in **Fig. 6-10**, roadside samples demonstrated

significant toxicity, with variations influenced by seasonal changes. Samples collected in the cold season (*e.g.*, RD1108X4, RD1126X4) exhibited greater reductions in cell viability and higher ROS induction compared with those from the warm season (*e.g.*, RD0505X4). The fractions from general urban samples showed lower toxicity than those from roadside samples at the same dilution ratio, and seasonal variations influenced the observed effects. Samples collected during the cold season (*e.g.*, 6F1126, 6F1207) showed moderate reductions in cell viability and ROS induction, with the BIO fraction maintaining viability above 80% and ROS levels below 1.2, possibly due to coal combustion-derived metals such as As (Wang et al., 2021). Summer samples (*e.g.*, 6F0505) displayed the least toxicity, likely due to lower PM_{2.5} concentrations and decreased metal bioaccessibility during warm months. Seasonal differences may have arisen from the stabilizing effect of mineral matrices in coal combustion emissions, which limited metal availability compared with traffic-related sources.

However, it should be acknowledged that the accurate exposure concentrations of all fractions extracted by SELF from overall PM_{2.5} were unavailable due to the inherent solute in SELF, which limits cytotoxicity based solely on the same dilution ratio. In the future, more accurate methods for SELF-extraction and concentration quantification of the extract need to be considered. Subsequently, combined with the mixture-cytotoxicity model, the fractional contributions of different bioaccessible and lung-fluid-insoluble fractions to the overall cytotoxicity could be determined.

6.4 Summary

This chapter systematically investigated the bioaccessibility-dependent health impact of major toxic components in PM_{2.5}. The study quantified the pulmonary bioaccessibility of trace metals and PAHs in PM_{2.5} collected from roadside, urban, and coastal background sites in HK, using SELF extraction and GC/MS analysis. Spatial

and seasonal variations in bioaccessibility were compared, and PLS-DA with VIP analysis was applied to identify key discriminators among sites and sources. The chapter further evaluated the influence of bioaccessibility on inhalation carcinogenic risk for both metals and PAHs, and explored the relationship between bioaccessibility and cytotoxicity endpoints, including cell viability and intracellular ROS, through *in vitro* assays. Toxicity profiles of PM_{2.5} fractions separated by bioaccessibility and chemical nature (BIO, BO, ISIO, ISO) were compared across different sites and seasons. The main contents of the findings were listed as follows:

(1) This chapter first defined bioaccessibility and its relevance for inhalation exposure of PM_{2.5}-bound metals and PAHs. Then, we quantified pulmonary bioaccessibility of trace metals in PM_{2.5} from roadside, urban, and coastal background sites in HK using the SELF extraction. As a result, bioaccessibility of trace metals and PAHs varied by site, with roadside samples showing higher values for key toxic metals (*i.e.*, As, Cd, V, Cr) and selected PAHs, indicating site-specific mitigation strategies are needed.

(2) Significant spatial and seasonal differences in bioaccessibility were observed, mainly driven by anthropogenic local emission sources and chemical speciation.

(3) The aerosol pH in Hong Kong is driven by a complex interplay of site-specific emissions and seasonal monsoons, with the most intense acidity found at roadside locations due to heavy tailpipe exhaust and dust re-suspension. These fluctuations in acidity significantly enhance the bioaccessibility of toxic transition metals and the harmful transformation of organic pollutants, thereby increasing the oxidative stress and respiratory risks posed to the local population.

(4) This study evaluated bioaccessibility-adjusted CR for metals and PAHs, then

compared with mass-based estimates, in which bioaccessibility-adjusted CR for metals and PAHs was much lower than mass-based estimates, with PAHs showing a reduction of one order of magnitude.

(5) In addition to inhalation health risks, this study investigated cytotoxicity of PM_{2.5} fractions with different bioaccessibility and chemical properties. As a conclusion to this exploratory study, cytotoxicity was closely linked to bioaccessibility, with the BIO and ISIO fractions showing the highest toxicity, especially during the cold season. Organic fractions (*i.e.*, BO and ISO) showed minimal toxicity after equal-volume extraction. However, water-soluble organic carbon (WSOC) and humic-like substances (HULIS) are widely recognized as key contributors to PM_{2.5} oxidative potential (Chen & Yu, 2025; Ma et al., 2018; Wang et al., 2022), and their relevance to toxicity is fully acknowledged. Importantly, the bioaccessible organic fraction (BO) analyzed in this chapter overlaps substantially with WSOC, as both represent water-soluble organic species in simulated lung fluid. This study focused on well-characterized toxic components (trace metals, PAHs, EC) with established toxicity mechanisms and risk coefficients to ensure robust quantitative risk assessment. To address this gap, future research will integrate targeted chemical characterization (*e.g.*, liquid chromatography-mass spectrometry) and toxicological assays to quantitatively assess the specific contributions of WSOC and HULIS to PM_{2.5} toxicity, building on the findings herein.

In general, bioaccessibility is crucial for health risk assessment. Traditional mass-based assessments may either underestimate or overestimate risks, particularly for sources with varying solubility and bioaccessibility of metals or PAHs. The SELF extraction and quantification methods need to be improved to more accurately obtain the exposure concentrations of each PM_{2.5} fraction. Future studies should integrate mixture cytotoxicity models to clarify the contributions of fractions with different

3209 bioaccessibility and biodurability to overall toxicity, thereby characterizing the health
3210 impacts of PM_{2.5} more precisely.

3211

7 Conclusions, Limitations, and Recommendations

This study enhances understanding of the inhalation health impacts of PM_{2.5}-bound toxic components in Hong Kong by integrating insights from acute and chronic toxicity, chemical-specific, source-resolved, and bioaccessibility perspectives.

Firstly, the major chemical compositions, including trace metals, OC, EC, water-soluble ions, and NPOCs, were analyzed. Based on multiple tracers, source apportionment of PM_{2.5} in Hong Kong was performed using the EPA PMF model. Then, the air mass back-trajectory analysis was conducted using the HYSPLIT model with meteorological data from the GDAS to evaluate the regional effects on the source profiles.

Secondly, to compare the acute cytotoxicity induced by roadside PM_{2.5} with that of general urban and coastal background samples and to investigate the component-specific contribution to PM_{2.5}-induced toxicity, intracellular ROS were chosen as the sole toxicity endpoint in the BEAS-2B cell line. The tested key toxic components include ten trace metals, twelve PAHs, and EC. The fractional contributions of each component to the total PM_{2.5}-induced acute cytotoxicity were evaluated. Combining with the above source profiles, we investigated the source-resolved cytotoxicity induced by each analyzed compound. To the best of our knowledge, our study represents the first attempt to develop a source-resolved cytotoxicity model that combines source profiles with biological toxicity induction. Shifting the focus to long-term health risks, the source-resolved health risk associated with the analyzed key toxic compounds (*i.e.*, trace metals, EC, and PAHs) was determined using the USEPA chronic health risk assessment model.

Thirdly, to deepen our understanding of the inhaled pathways of PM_{2.5}-bound toxic components in the human respiratory system, the bioaccessibility of these components

was analyzed after extracting total PM_{2.5} with artificial lung fluid, which simulated the pulmonary alveolar environment. The bioaccessible fractions of major identified toxic groups, metals, and parent PAHs were analyzed. Previous studies have primarily focused on the major chemical composition and source profiles based on the mass concentration of PM_{2.5} through spatial and temporal comparisons. Our study enhances the traditional source-resolved health risk assessment by incorporating the bioaccessible fractions of total PM_{2.5}. Moreover, further cytotoxicity experiments were conducted to compare the intracellular reaction of different portions separated via bioaccessibility and polarity. The main findings are summarized as follows:

- (1) Component-specific analyses reveal that trace metals are the primary drivers of PM_{2.5}-induced acute intracellular ROS and chronic health risks (*i.e.*, CR and HQ) in Hong Kong, followed by EC, an understudied insoluble fraction, while PAHs play a minor role.
- (2) This study innovatively quantifies EC's contribution to the biological toxic effects induced by overall ambient PM_{2.5}, highlighting the need to address the biological toxic mechanism of insoluble fractions in PM_{2.5}.
- (3) Traffic-related emissions were the dominant contributors to acute cytotoxicity in urban PM_{2.5}. Notably, non-exhaust emissions accounted for a substantial proportion of this toxicity, emphasizing the need for toxicity-oriented air pollution control targeting non-exhaust emissions in Hong Kong.
- (4) In addition to local traffic emissions, regional combustion & industrial emissions, transported over long distances, significantly influenced chronic inhalation health risks, despite minimal local coal combustion.
- (5) The bioaccessibility of chemical components in PM_{2.5} varied spatially and seasonally, depending on the dominant emission sources. The bioaccessibility

of most trace metals was higher in roadside PM_{2.5} than in ambient samples, attributed to differences in valence, forms, and degree of aging.

(6) Echoing with previous analysis of EC, the *in vitro* toxicity comparison between bioaccessible and lung-fluid-insoluble phases indicated that the lung-fluid-insoluble fraction in PM_{2.5} can also lead to acute cytotoxicity, especially the inorganic fraction, inducing significant intracellular ROS.

(7) In addition, this investigation confirmed that some current toxicological studies may stretch the actual toxic effect induced by lung-fluid-insoluble organic components in PM_{2.5}, by non-polar solvent extraction or adding a solubilizer during the exposure experiment. Although it is not meant to imply that their toxicity is negligible, when these lung-fluid-insoluble organic compounds are inhaled into the deep lungs, they still exist as a poorly soluble particulate phase. Their actual biological effect may differ significantly from that of their dissolved form.

These insights have important implications for the development of targeted, toxicity-oriented air pollution control strategies, particularly in densely populated coastal megacities. However, it is crucial to acknowledge the limitations present throughout the study, as they warrant further investigation:

- (1) Our current cytotoxicity assessment is primarily based on cell viability and intracellular ROS; it is important to note that certain groups of components, such as PAHs, may primarily affect genetic toxicity as an alternative endpoint. Therefore, it is necessary to compare multiple cytotoxicity endpoints to gain a deeper understanding of the contribution of different components to the overall cytotoxicity in real PM_{2.5}.
- (2) The linear addition supported by the CA-model was only feasible in relatively

light-polluted areas. As the concentration and complexity of PM_{2.5} increase, the toxic effects of its components become nonlinear and more complex. Therefore, unraveling the mechanisms of synergism and antagonism in the actual composition of PM_{2.5} and quantifying their contributions using the deviation ratio model or response surface modeling poses significant implications that warrant further investigation.

- (3) This targeted analysis failed to comprehensively identify all major contributing components, especially in coastal background PM_{2.5}, emphasizing the necessity for complementary non-targeted screening methods to detect other emerging contributors, particularly biological compounds that may play significant yet overlooked roles.
- (4) Furthermore, it should be recognized as a limitation that using only the BEAS-2B cell line does not entirely reflect the diverse responses of various lung cell types (*e.g.*, alveolar cells and fibroblasts) or the combined effects at the organ or whole-organism level (Li et al., 2024; Zhou et al., 2024).

In light of these limitations, future research should employ multiple cytotoxicity endpoints to capture the full spectrum of PM_{2.5}-induced biological effects, explore the non-linear and interactive toxicities among PM_{2.5} components using advanced modeling approaches, and incorporate non-targeted screening to identify overlooked contributors, especially biological compounds. Additionally, expanding *in vitro* assessments to include diverse lung cell types and *in vivo* models will be essential for a more comprehensive evaluation of health risks associated with ambient PM_{2.5} exposure.

Appendix 1

This section provides the experimental results of PM_{2.5} chemical analyses.

1. Monthly average concentration (mean ± S.D.) (ng/m³) of major metals of PM_{2.5} in Hong Kong 2021

Site	Month	Ba	Al	As	Cd	Co	Cr	Cu	Fe	Mn	Mo	Ni	Pb	Sb	Se	Sn	V	Zn
Roadside	Feb-21	17.97±3.70	436.57±61.25	7.13±0.56	0.55±0.09	0.86±0.14	8.40±2.67	38.96±2.76	196.34±100.67	14.09±2.56	2.08±0.08	14.09±1.17	2.08±0.83	6.80±0.69	9.89±0.90	3.15±1.46	47.32±0.44	13.64±12.45
	Mar-21	22.09±10.76	912.85±495.21	9.24±4.41	0.59±0.38	1.31±0.66	6.51±3.81	45.86±15.89	330.66±484.83	30.68±19.98	2.53±0.69	7.55±3.56	12.21±8.39	5.55±1.43	12.48±3.49	16.31±6.80	4.81±2.37	113.11±98.36
	Apr-21	29.47±15.97	1409.83±870.21	12.24±4.40	0.68±0.17	1.74±0.83	9.96±2.30	57.64±34.28	148.50±598.78	8.71±12.73	2.75±0.55	10.52±2.86	14.32±5.11	6.48±1.20	15.16±3.57	19.39±5.92	6.12±2.40	135.31±46.49
	May-21	46.22±28.24	1464.84±774.07	11.66±3.64	0.58±0.11	2.18±1.17	6.33±0.72	68.85±33.39	539.17±888.43	25.18±7.65	2.94±0.90	6.90±1.10	10.81±2.62	7.31±1.89	14.28±1.82	23.38±8.92	4.65±0.89	108.34±44.96
	Jun-21	35.17±12.54	1057.03±483.57	9.20±2.40	0.38±0.10	1.48±0.31	4.10±1.06	68.43±16.90	164.84±247.21	17.49±2.68	2.36±0.59	4.94±0.86	7.22±1.47	6.43±1.17	12.32±1.97	18.83±3.77	4.15±0.82	224.27±168.00
	Jul-21	93.50±42.58	1303.98±755.18	11.05±3.50	0.54±0.25	2.10±0.78	5.76±2.76	74.04±20.94	574.25±582.28	9.80±57.07	2.63±0.50	8.46±3.30	10.91±4.88	7.04±1.07	15.47±3.00	23.05±5.36	5.16±1.81	2139.41±1246.69
	Aug-21	35.52±23.65	966.36±144.21	8.86±0.32	0.41±0.08	1.63±0.37	6.22±1.60	65.11±13.85	192.12±399.60	29.35±6.28	3.12±0.80	6.86±0.88	9.62±3.62	6.48±0.31	13.15±1.30	20.05±3.53	5.18±2.56	786.06±778.38
	Sep-21	52.75±12.80	1681.08±865.42	12.07±4.86	0.49±0.32	2.31±0.63	5.40±3.37	103.23±16.17	727.96±477.87	9.93±10.35	4.39±0.91	10.45±3.53	11.00±8.25	7.74±1.66	12.10±4.26	26.52±7.15	11.42±3.06	242.08±26.11
	Oct-21	40.56±6.70	1558.36±701.13	13.19±3.90	0.88±0.21	2.15±0.47	10.87±1.87	120.26±11.82	468.04±165.72	32.38±9.03	4.41±0.77	9.90±4.33	19.55±4.88	7.81±0.68	15.19±3.94	27.40±6.79	10.25±3.08	241.65±23.23
	Nov-21	54.70±23.59	1513.85±598.48	12.88±3.25	1.08±0.25	2.73±1.01	8.17±7.57	114.75±25.45	950.02±803.66	4.59±13.89	3.91±0.98	7.89±2.86	20.85±6.76	8.30±1.43	15.08±2.30	30.60±6.62	5.30±3.51	182.39±64.15
	Dec-20	31.84±16.51	1089.29±676.29	10.12±4.11	0.70±0.39	1.78±0.93	3.26±0.50	64.04±27.66	244.42±684.23	9.92±10.48	2.76±0.65	6.88±2.41	16.15±7.66	7.01±1.60	12.35±2.09	24.55±6.69	3.66±0.36	123.46±83.33
	Jun-22	46.25±41.50	545.27±228.25	5.83±1.87	1.24±0.13	1.75±0.51	6.75±2.40	157.71±30.68	357.75±379.22	3.18±13.93	4.26±1.58	11.95±5.67	17.92±2.76	3.17±0.60	5.25±1.19	19.66±6.75	3.09±1.18	486.38±454.83
Ambient	Feb-21	8.18±2.75	530.50±145.18	4.70±0.48	0.75±0.12	1.10±0.22	7.44±3.10	31.94±21.53	353.67±122.49	5.96±3.41	70.63±14.77	6.85±2.28	3.31±3.28	3.61±0.27	9.41±0.32	10.48±2.33	1.13±1.14	42.87±27.81
	Mar-21	10.85±9.60	987.93±732.25	7.11±5.22	1.05±0.51	1.19±0.91	3.41±2.40	72.44±67.38	331.76±491.59	7.24±13.82	91.26±68.52	8.19±7.81	9.27±3.60	4.07±1.73	9.74±3.97	12.64±7.99	2.87±2.70	79.88±76.80
	Apr-21	11.15±5.66	976.50±441.28	7.73±3.31	0.64±0.30	1.42±0.76	3.51±2.17	66.42±38.41	189.18±302.27	13.19±7.55	53.79±44.22	10.25±4.65	8.60±4.99	3.86±1.27	9.45±3.63	10.88±3.61	3.24±2.35	62.27±27.38
	May-21	10.05±3.87	759.28±333.90	4.92±1.87	0.68±0.11	1.02±0.27	2.74±1.36	58.41±50.83	141.02±232.18	9.73±6.50	75.70±24.50	7.87±2.15	5.10±3.88	2.91±0.16	7.11±0.83	9.74±2.22	2.32±1.89	47.59±22.85
	Jul-21	6.51±0.80	503.28±331.07	3.65±1.53	0.59±0.03	0.73±0.11	2.86±0.55	13.47±10.57	223.62±18.32	3.86±0.20	54.62±3.79	4.98±2.50	1.77±0.46	2.81±0.27	6.51±0.81	8.47±0.80	0.75±0.94	27.40±14.71
	Aug-21	8.85±1.08	212.80±48.28	2.79±0.38	0.66±0.03	0.94±0.12	1.70±0.43	300.80±68.53	254.55±48.22	4.84±1.21	63.66±6.39	5.24±1.24	2.56±0.96	3.54±0.27	7.67±0.51	10.72±1.54	2.62±0.77	43.91±14.94
	Sep-21	12.17±2.36	516.77±269.09	5.22±1.74	0.81±0.14	1.14±0.19	1.55±0.40	337.66±59.86	375.55±93.06	6.82±1.70	83.61±11.38	6.40±2.89	4.97±2.83	4.39±0.29	9.64±0.95	14.32±3.29	3.48±1.32	48.50±12.93
	Oct-21	12.97±1.14	1193.24±689.45	9.00±3.81	0.97±0.22	1.16±0.08	2.40±0.99	257.96±38.75	488.46±51.82	8.56±3.39	101.05±5.88	3.99±0.18	8.91±3.16	5.04±0.70	12.30±2.26	31.13±7.05	2.44±0.35	80.23±23.27
	Nov-21	19.21±4.57	1125.88±327.85	10.81±2.27	0.97±0.30	1.42±0.40	2.14±1.47	236.79±43.47	333.23±233.23	17.16±6.05	2.26±0.52	4.16 ±0.31	17.10±8.49	5.66±0.54	12.73±1.89	18.26±3.91	2.70±0.04	99.98±34.48
	Dec-20	16.48±4.28	758.88±559.94	7.77±2.53	1.45±0.55	1.46±0.56	2.26±1.04	131.91±40.14	329.88±127.68	15.68±4.72	50.39±45.44	6.93±1.89	9.59±11.38	3.93±0.82	8.64±2.60	18.11±2.18	3.69±1.47	98.78±30.31
	Jun-22	31.57±26.15	1031.80±379.70	6.79±2.09	1.24±0.18	1.08±0.11	5.66±4.15	139.22±22.42	540.44±49.26	14.61±5.46	2.64±0.32	6.35±1.71	21.72±4.13	1.02±0.38	1.97±0.65	14.27±11.42	2.32±1.45	85.62±32.25
Background	Feb-21	13.94±2.25	1091.31±361.80	8.92±2.15	0.34±0.09	0.88±0.12	2.12±0.70	8.34±4.47	167.41±40.26	3.82±0.23	1.19±0.11	3.90±0.11	7.80±3.00	5.53±0.40	12.01±1.16	11.08±0.47	7.01±0.21	21.05±2.57
	Mar-21	3.92±2.72	1048.62±783.54	8.28±4.43	0.31±0.20	1.03±0.36	2.72±1.08	8.09±5.66	219.85±175.56	5.59±5.02	1.24±0.31	7.01±5.81	5.86±4.91	5.45±0.63	11.95±2.26	11.32±1.44	7.39±1.01	32.51±30.75
	Apr-21	2.44±0.19	548.74±528.03	5.76±2.40	0.23±0.04	0.81±0.09	1.85±1.53	6.08±0.67	89.13±30.89	2.85±1.27	1.26±0.24	5.81±1.78	3.36±0.31	5.04±0.32	10.19±1.41	10.62±0.40	7.66±1.34	32.69±2.55
	May-21	2.87±0.66	368.61±1164.22	8.30±7.14	0.16±0.07	0.85±0.11	3.86±0.96	4.69±1.39	105.35±42.56	2.51±1.50	1.19±0.23	6.09±2.25	2.57±0.94	5.27±1.11	11.86±3.70	10.29±0.36	7.37±0.71	14.72±2.24
	Jun-21	2.51±0.29	670.57±111.98	5.88±0.62	0.14±0.02	0.74±0.04	4.00±3.63	4.90±1.37	70.80±11.92	1.84±0.34	1.22±0.07	4.48±0.29	2.06±0.51	4.95±0.09	10.64±0.33	10.10±0.12	7.18±0.61	18.37±3.71
	Jul-21	2.64±0.57	1262.35±909.47	8.97±4.03	0.20±0.06	0.89±0.06	3.89±0.31	7.87±1.72	124.46±20.60	2.38±0.54	1.23±0.10	5.42±1.15	2.99±1.58	5.48±0.43	12.56±1.75	10.89±0.58	7.35±0.69	20.32±7.57
	Aug-21	2.42±0.18	923.19±192.57	7.63±0.53	0.18±0.06	0.96±0.06	2.64±1.57	6.84±1.07	221.07±156.60	2.55±0.93	1.24±0.11	5.98±1.62	2.83±1.98	5.19±0.16	11.43±0.49	10.71±0.27	7.92±0.91	33.74±19.64
	Sep-21	2.86±1.06	1343.29±524.95	9.81±3.02	0.26±0.15	0.99±0.15	3.18±1.69	9.34±2.62	156.60±74.41	3.65±1.31	1.41±0.39	7.21±2.65	3.45±2.57	5.90±0.72	13.05±1.68	11.76±0.82	7.69±0.62	28.42±16.26
	Oct-21	3.96±1.02	1132.88±466.97	9.37±2.78	0.51±0.19	1.03±0.11	2.75±1.69	16.95±10.11	237.77±80.18	5.08±1.90	1.54±0.29	6.43±0.91	6.79±2.47	5.79±0.48	13.05±1.50	12.45±0.57	6.61±0.39	38.83±12.95
	Nov-21	5.06±1.89	1453.20±792.29	11.37±4.37	0.70±0.27	1.13±0.16	5.14±3.06	31.24±19.17	332.92±148.02	8.57±3.22	1.73±0.24	5.82±1.39	10.77±3.95	6.17±0.64	14.77±2.41	13.01±0.69	7.08±0.29	60.38±18.88
	Dec-20	4.65±1.44	1034.76±715.04	9.64±3.73	0.89±0.45	1.04±0.27	4.45±1.33	14.59±4.71	241.06±74.27	10.44±3.25	1.61±0.32	6.56±3.26	13.86±7.00	5.03±2.26	11.77±1.04	11.13±4.35	6.46±3.59	67.42±12.53
	Jun-22	4.38±0.73	727.32±650.16	4.15±4.02	1.08±0.19	0.73±0.16	9.95±6.23	24.61±6.16	109.11±51.43	8.77±2.55	0.98±0.43	5.75±2.78	15.79±3.61	2.22±0.75	7.64±3.87	5.38±2.25	1.58±0.76	38.96±12.36

2. Monthly average concentration (mean $\pm$ S.D.) ($\mu\text{g}/\text{m}^3$) of major components of PM_{2.5} in Hong Kong 2021

Site	Month	Na ⁺	NH ₄ ⁺	K ⁺	Mg ²⁺	Ca ²⁺	Cl ⁻	SO ₄ ²⁻	NO ₃ ⁻	Metals	OC	EC	SUM
Roadside	Feb-21	0.55	1.44	0.20	0.08	0.41	0.43	4.08	2.04	1.12	5.18	1.90	17.44
	Mar-21	1.00 $\pm$ 0.28	1.76 $\pm$ 1.35	0.23 $\pm$ 0.16	0.13 $\pm$ 0.05	0.65 $\pm$ 0.40	0.90 $\pm$ 0.35	4.39 $\pm$ 2.05	4.42 $\pm$ 3.94	2.03 $\pm$ 1.15	7.41 $\pm$ 2.51	2.41 $\pm$ 1.14	24.84
	Apr-21	0.91 $\pm$ 0.75	0.90 $\pm$ 0.37	0.14 $\pm$ 0.09	0.09 $\pm$ 0.07	0.72 $\pm$ 0.66	0.71 $\pm$ 0.49	2.86 $\pm$ 1.83	2.40 $\pm$ 1.60	2.63 $\pm$ 1.58	7.65 $\pm$ 1.75	3.16 $\pm$ 1.51	22.17
	May-21	1.62 $\pm$ 0.56	0.84 $\pm$ 0.59	0.18 $\pm$ 0.06	0.18 $\pm$ 0.06	1.44 $\pm$ 0.62	1.11 $\pm$ 0.58	4.42 $\pm$ 2.05	2.70 $\pm$ 0.99	3.76 $\pm$ 1.76	8.56 $\pm$ 1.28	2.88 $\pm$ 0.84	27.68
	Jun-21	1.08 $\pm$ 0.10	0.36 $\pm$ 0.05	0.13 $\pm$ 0.02	0.09 $\pm$ 0.03	0.59 $\pm$ 0.08	0.61 $\pm$ 0.06	2.42 $\pm$ 0.53	1.28 $\pm$ 0.21	2.64 $\pm$ 0.63	5.28 $\pm$ 0.81	2.59 $\pm$ 0.53	17.09
	Jul-21	1.00 $\pm$ 0.17	0.38 $\pm$ 0.42	0.33 $\pm$ 0.18	0.07 $\pm$ 0.02	0.95 $\pm$ 0.32	0.99 $\pm$ 0.46	2.93 $\pm$ 1.91	1.69 $\pm$ 0.70	5.35 $\pm$ 2.28	6.44 $\pm$ 1.33	2.48 $\pm$ 1.14	22.62
	Aug-21	0.62 $\pm$ 0.31	0.22 $\pm$ 0.13	0.15 $\pm$ 0.08	0.07 $\pm$ 0.04	0.79 $\pm$ 0.49	0.62 $\pm$ 0.28	2.38 $\pm$ 0.62	0.97 $\pm$ 0.35	3.17 $\pm$ 1.12	6.07 $\pm$ 1.04	2.85 $\pm$ 0.77	17.93
	Sep-21	0.58 $\pm$ 0.32	1.09 $\pm$ 0.86	0.16 $\pm$ 0.14	0.12 $\pm$ 0.00	0.70 $\pm$ 0.55	0.63 $\pm$ 0.32	4.15 $\pm$ 3.33	1.51 $\pm$ 0.81	6.53 $\pm$ 5.88	7.72 $\pm$ 1.62	2.94 $\pm$ 0.61	25.90
	Oct-21	1.00 $\pm$ 0.97	3.22 $\pm$ 1.63	0.35 $\pm$ 0.23	0.17 $\pm$ 0.07	0.85 $\pm$ 1.05	1.18 $\pm$ 1.18	10.5 $\pm$ 6.33	2.96 $\pm$ 2.33	3.59 $\pm$ 0.78	11.92 $\pm$ 0.52	2.77 $\pm$ 0.61	38.58
	Nov-21	1.25 $\pm$ 0.73	1.95 $\pm$ 1.55	0.32 $\pm$ 0.12	0.14 $\pm$ 0.09	1.71 $\pm$ 0.58	1.34 $\pm$ 0.85	6.42 $\pm$ 4.84	2.73 $\pm$ 1.27	3.97 $\pm$ 1.50	11.98 $\pm$ 1.29	2.54 $\pm$ 0.45	34.36
	Dec-21	1.33	0.87	0.27	0.15	1.29	1.07	3.66	2.29	2.66	12.11	2.64	28.33
	Jun-22	0.76 $\pm$ 0.52	2.63 $\pm$ 1.26	0.43 $\pm$ 0.23	0.11 $\pm$ 0.06	0.56 $\pm$ 0.50	1.61 $\pm$ 0.58	5.45 $\pm$ 3.25	4.78 $\pm$ 2.02	2.21 $\pm$ 2.62	6.90 $\pm$ 1.76	2.44 $\pm$ 0.83	27.87
Urban	Feb-21	0.07	0.15	0.04	0.06	0.13	0.21	0.11	0.93	1.03	3.43	0.53	6.69
	Mar-21	0.73 $\pm$ 0.37	1.06 $\pm$ 1.00	0.20 $\pm$ 0.13	0.08 $\pm$ 0.06	0.22 $\pm$ 0.14	0.96 $\pm$ 0.17	4.21 $\pm$ 3.30	1.40 $\pm$ 0.63	1.70 $\pm$ 1.45	9.79 $\pm$ 4.76	2.19 $\pm$ 1.91	21.63
	Apr-21	0.80 $\pm$ 0.51	1.58 $\pm$ 1.16	0.24 $\pm$ 0.16	0.11 $\pm$ 0.05	0.36 $\pm$ 0.30	0.53 $\pm$ 0.27	4.75 $\pm$ 3.59	2.45 $\pm$ 0.63	1.67 $\pm$ 1.45	6.51 $\pm$ 4.76	1.75 $\pm$ 1.08	20.76
	May-21	0.80 $\pm$ 0.32	1.11 $\pm$ 0.86	0.19 $\pm$ 0.12	0.08 $\pm$ 0.06	0.40 $\pm$ 0.14	0.56 $\pm$ 0.11	3.97 $\pm$ 3.02	1.61 $\pm$ 0.27	1.15 $\pm$ 0.53	3.88 $\pm$ 2.18	0.56 $\pm$ 0.86	14.33
	Jul-21	0.89	1.58	0.23	0.09	0.46	0.68	5.05	1.64	1.07	3.12	0.00	14.83
	Aug-21	0.25 $\pm$ 0.07	0.56 $\pm$ 0.31	0.07 $\pm$ 0.05	0.04 $\pm$ 0.03	0.20 $\pm$ 0.06	0.12 $\pm$ 0.03	2.33 $\pm$ 0.71	0.40 $\pm$ 0.11	0.93 $\pm$ 0.24	5.05 $\pm$ 1.65	1.33 $\pm$ 0.77	11.28
	Sep-21	0.30 $\pm$ 0.06	0.91 $\pm$ 0.39	0.12 $\pm$ 0.03	0.05 $\pm$ 0.01	0.26 $\pm$ 0.13	0.26 $\pm$ 0.21	3.23 $\pm$ 1.22	0.72 $\pm$ 0.23	1.16 $\pm$ 0.12	5.22 $\pm$ 0.90	1.39 $\pm$ 0.60	13.59
	Oct-21	0.85 $\pm$ 0.23	1.77 $\pm$ 1.21	0.26 $\pm$ 0.06	0.12 $\pm$ 0.06	0.85 $\pm$ 0.66	0.88 $\pm$ 0.30	6.19 $\pm$ 2.88	2.09 $\pm$ 0.27	1.73 $\pm$ 0.16	6.72 $\pm$ 0.83	1.19 $\pm$ 0.23	21.02
	Nov-21	0.41 $\pm$ 0.34	1.03 $\pm$ 0.71	0.19 $\pm$ 0.13	0.04 $\pm$ 0.03	0.58 $\pm$ 0.58	0.74 $\pm$ 0.45	3.89 $\pm$ 0.87	1.21 $\pm$ 0.13	2.47 $\pm$ 0.66	8.79 $\pm$ 1.20	1.29 $\pm$ 0.25	20.64
	Dec-21	0.88 $\pm$ 0.71	2.46 $\pm$ 2.47	0.27 $\pm$ 0.21	0.08 $\pm$ 0.06	0.54 $\pm$ 0.39	1.38 $\pm$ 1.03	5.90 $\pm$ 2.95	6.78 $\pm$ 5.91	2.05 $\pm$ 0.72	10.11 $\pm$ 1.15	1.75 $\pm$ 0.56	32.19
	Jun-22	1.18 $\pm$ 0.99	2.87 $\pm$ 1.15	0.43 $\pm$ 0.42	0.16 $\pm$ 0.11	0.58 $\pm$ 0.87	1.85 $\pm$ 0.96	6.25 $\pm$ 2.62	6.68 $\pm$ 3.33	1.91 $\pm$ 1.96	8.71 $\pm$ 1.06	1.92 $\pm$ 0.47	32.54
Background	Feb-21	1.11 $\pm$ 0.28	0.78 $\pm$ 0.26	0.31 $\pm$ 0.22	0.19 $\pm$ 0.04	0.23 $\pm$ 0.07	1.51 $\pm$ 0.74	2.67 $\pm$ 0.72	1.21 $\pm$ 0.44	1.39 $\pm$ 0.42	3.98 $\pm$ 0.93	0.55 $\pm$ 0.15	13.92
	Mar-21	0.80 $\pm$ 0.29	1.06 $\pm$ 0.55	0.18 $\pm$ 0.06	0.14 $\pm$ 0.03	0.21 $\pm$ 0.08	0.88 $\pm$ 0.58	3.20 $\pm$ 1.37	1.10 $\pm$ 0.88	1.40 $\pm$ 1.02	3.27 $\pm$ 1.19	0.48 $\pm$ 0.28	12.71
	Apr-21	0.63 $\pm$ 0.21	0.94 $\pm$ 0.72	0.19 $\pm$ 0.07	0.10 $\pm$ 0.04	0.15 $\pm$ 0.11	0.43 $\pm$ 0.27	2.96 $\pm$ 1.32	0.73 $\pm$ 0.32	0.75 $\pm$ 0.57	2.45 $\pm$ 0.37	0.38 $\pm$ 0.12	9.72
	May-21	0.65 $\pm$ 0.47	0.03 $\pm$ 0.04	0.08 $\pm$ 0.06	0.09 $\pm$ 0.05	0.18 $\pm$ 0.04	0.91 $\pm$ 0.65	0.97 $\pm$ 0.31	0.36 $\pm$ 0.20	1.42 $\pm$ 1.37	1.24 $\pm$ 0.05	0.20 $\pm$ 0.03	6.09
	Jun-21	0.86 $\pm$ 0.50	0.05 $\pm$ 0.04	0.10 $\pm$ 0.07	0.10 $\pm$ 0.04	0.13 $\pm$ 0.01	0.76 $\pm$ 0.65	1.42 $\pm$ 0.41	0.34 $\pm$ 0.12	0.84 $\pm$ 0.12	1.86 $\pm$ 0.24	0.23 $\pm$ 0.09	6.69
	Jul-21	0.97 $\pm$ 0.62	0.40 $\pm$ 0.35	0.09 $\pm$ 0.03	0.09 $\pm$ 0.02	0.24 $\pm$ 0.09	1.54 $\pm$ 1.51	1.47 $\pm$ 1.05	0.45 $\pm$ 0.12	1.50 $\pm$ 0.90	1.94 $\pm$ 0.36	0.30 $\pm$ 0.14	8.92
	Aug-21	0.81 $\pm$ 0.36	0.22 $\pm$ 0.18	0.09 $\pm$ 0.02	0.08 $\pm$ 0.03	0.13 $\pm$ 0.01	0.90 $\pm$ 0.59	1.43 $\pm$ 0.34	0.32 $\pm$ 0.09	1.27 $\pm$ 0.36	1.48 $\pm$ 0.10	0.25 $\pm$ 0.05	6.97
	Sep-21	0.87 $\pm$ 0.27	0.43 $\pm$ 0.56	0.11 $\pm$ 0.06	0.10 $\pm$ 0.04	0.22 $\pm$ 0.08	1.06 $\pm$ 0.35	1.98 $\pm$ 1.72	0.41 $\pm$ 0.14	1.63 $\pm$ 0.63	2.05 $\pm$ 0.50	0.33 $\pm$ 0.13	9.18
	Oct-21	0.67 $\pm$ 0.52	0.75 $\pm$ 0.60	0.10 $\pm$ 0.12	0.06 $\pm$ 0.07	0.12 $\pm$ 0.14	0.99 $\pm$ 0.72	4.17 $\pm$ 0.81	0.47 $\pm$ 0.01	1.52 $\pm$ 0.54	4.26 $\pm$ 0.63	0.49 $\pm$ 0.15	13.31
	Nov-21	1.06 $\pm$ 0.36	0.78 $\pm$ 0.30	0.24 $\pm$ 0.09	0.15 $\pm$ 0.04	0.39 $\pm$ 0.17	1.30 $\pm$ 0.53	2.95 $\pm$ 0.81	0.71 $\pm$ 0.19	2.00 $\pm$ 0.98	4.98 $\pm$ 0.47	0.57 $\pm$ 0.08	15.14
	Dec-21	1.11 $\pm$ 0.41	1.51 $\pm$ 1.04	0.25 $\pm$ 0.14	0.12 $\pm$ 0.05	0.28 $\pm$ 0.08	1.61 $\pm$ 1.09	2.89 $\pm$ 1.17	2.22 $\pm$ 1.43	1.47 $\pm$ 0.82	5.06 $\pm$ 0.60	0.78 $\pm$ 0.25	17.30

3. Seasonal average concentration (pg/m³) of major NPOCs of PM_{2.5} in Hong Kong 2021

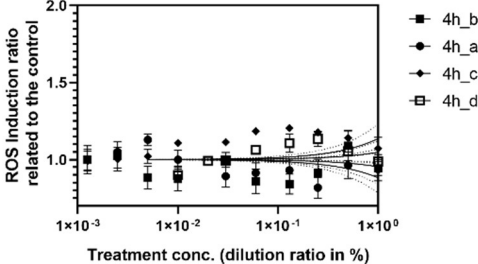
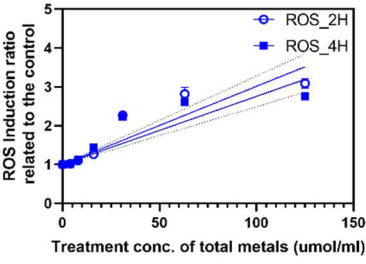
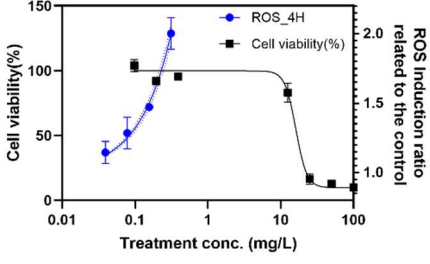
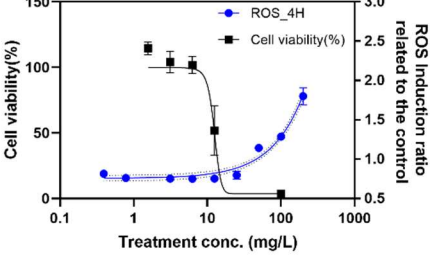
	m/z	Roadside								Urban								Background							
		Spring_RD		Summer_RD		Fall_RD		Winter_RD		Spring_6F		Summer_6F		Fall_6F		Winter_6F		Spring_HT		Summer_HT		Fall_HT		Winter_HT	
		Conc	STD	Conc	STD	Conc	STD	Conc	STD	Conc	STD	Conc	STD	Conc	STD	Conc	STD	Conc	STD	Conc	STD	Conc	STD	Conc	STD
Phenanthrene	178	104.91	37.74	90.77	32.15	93.02	29.91	105.21	39.03	46.13	39.10	21.81	24.57	42.02	9.27	75.63	25.97	8.27	0.02	3.39	0.61	10.96	6.94	35.47	12.91
Anthracene	178	82.89	31.82	81.62	33.19	86.47	27.04	0.09	31.89	37.16	28.34	25.24	19.75	40.46	10.21	57.65	14.49	6.12	2.62	0.00	0.00	0.01	5.49	13.41	0.06
Fluoranthene	202	136.35	39.43	135.66	49.52	113.15	26.26	0.15	97.88	52.26	43.24	17.50	21.97	45.41	18.53	119.71	47.04	7.11	1.65	1.85	0.33	0.07	16.05	68.33	28.87
Pyrene	202	288.41	85.07	319.68	132.92	253.91	71.58	0.28	129.46	75.41	61.41	30.22	38.46	80.20	26.68	170.53	70.50	8.80	1.66	2.82	0.54	0.07	23.15	73.51	22.20
Cyclopenta[cd]pyrene	226	15.97	3.54	12.08	5.40	12.66	4.00	0.10	10.35	6.13	5.40	3.22	3.20	7.56	4.23	27.82	35.91	0.87	0.29	0.30	0.03	0.01	2.65	7.21	2.77
benzo[c]phenanthrene	228	11.99	2.11	8.06	3.74	8.84	2.75	0.02	7.71	193.07	529.23	1.77	2.07	5.61	2.87	13.28	5.74	0.72	0.13	0.09	0.13	0.01	1.78	5.46	1.25
benz[a]anthracene	228	22.06	8.26	13.42	11.44	17.65	10.22	0.05	28.00	22.63	15.17	9.44	9.94	26.83	11.08	50.82	20.21	3.56	0.09	3.54	0.41	0.02	7.44	15.59	0.17
Chrysene	228	27.52	7.27	23.17	9.48	22.58	6.72	0.03	10.62	15.63	11.15	9.20	13.31	18.52	4.88	29.15	13.50	1.57	0.31	1.61	1.62	0.01	2.63	10.07	5.33
Triphenylene	228	25.48	10.49	16.83	12.07	18.85	8.93	0.05	26.38	25.51	17.84	10.24	12.27	28.24	7.89	55.90	17.04	4.65	0.50	0.94	0.77	0.03	8.03	25.02	13.67
Benzo[b]fluoranthene	252	12.57	14.42	3.93	5.08	5.68	4.16	0.03	27.84	19.11	15.28	6.50	8.26	27.40	21.11	67.48	32.79	2.59	1.28	1.95	0.13	0.03	10.63	32.86	10.78
Benzo[k]fluoranthene	252	3.94	4.57	3.39	5.27	3.93	5.20	0.02	13.65	14.80	10.59	5.02	5.00	19.18	12.16	45.56	25.61	1.54	2.18	2.05	0.40	0.02	8.09	22.06	8.86
Benzo[a]fluoranthene	252	1.62	3.23	2.27	3.09	4.72	3.47	0.01	12.40	5.69	4.07	1.77	2.01	10.78	7.37	22.06	13.32	1.65	1.55	1.63	0.10	0.01	5.48	8.88	6.71
Benzo[e]pyrene	252	8.08	4.08	3.74	6.22	5.34	5.49	0.03	23.13	18.64	12.90	7.93	9.98	30.24	20.82	54.02	26.61	3.05	1.69	2.56	1.22	0.03	9.48	29.06	13.09
Benzo[a]pyrene	252	4.22	6.03	2.75	4.79	5.14	7.31	0.04	28.04	20.18	16.13	9.13	3.53	36.00	29.72	82.70	56.06	3.00	0.15	5.64	4.91	0.04	25.53	41.88	8.31
Perylene	252	10.32	15.05	15.34	13.57	16.83	12.36	0.06	63.31	23.28	21.52	8.69	9.65	18.43	6.24	48.50	21.06	6.74	8.40	9.58	1.79	0.02	1.09	21.26	24.28
Indeno[1,2,3-cd]pyrene	276	1.59	1.76	0.59	1.90	0.27	0.65	0.01	5.50	4.37	6.14	1.30	1.12	7.66	10.86	26.83	16.10	0.00	0.00	0.00	0.00	0.02	11.08	22.87	3.31
Benzo[ghi]perylene	276	2.10	2.57	0.83	2.23	1.07	1.42	0.01	7.74	6.52	5.88	5.23	1.10	13.38	17.14	32.12	18.95	0.00	0.00	3.26	4.60	0.03	12.88	34.08	9.61
Dibenz[a,h]anthracene	278	1.18	1.91	0.75	1.69	0.47	1.63	0.00	2.00	0.98	2.78	0.00	0.00	0.54	1.89	3.38	3.29	0.00	0.00	0.00	0.00	0.00	0.00	2.14	3.03
Dibena[a,c]anthracene	278	0.51	0.88	0.24	0.81	0.00	0.00	0.00	0.98	0.27	0.77	0.00	0.00	0.15	0.51	1.09	1.14	0.00	0.00	0.00	0.00	0.00	0.00	0.78	1.10
Picene	278	0.64	1.37	1.09	3.63	0.61	1.39	0.00	1.59	1.36	2.38	0.00	0.00	0.33	0.83	1.59	1.88	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Benzo[b]chrysene	278	0.46	1.44	0.00	0.00	0.00	0.00	0.00	2.10	0.80	1.50	0.00	0.00	0.11	0.37	0.90	1.47	0.00	0.00	0.00	0.00	0.00	0.00	1.08	1.53
Coronene	300	0.34	0.87	0.00	0.00	0.00	0.00	0.00	2.00	0.96	2.67	0.00	0.00	0.47	1.63	1.89	2.92	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Dibenzo[a,e]pyrene	302	0.88	1.45	0.20	0.54	0.59	1.30	0.00	2.53	2.40	4.43	0.63	0.55	1.96	4.69	5.43	4.85	0.00	0.00	0.00	0.00	0.01	1.08	12.49	14.54
1,3,5-triphenylbenzene	306	19.43	17.78	17.85	26.91	11.01	6.18	0.03	36.28	35.66	38.11	4.76	3.43	15.48	11.50	57.53	21.94	2.51	0.19	1.36	0.15	0.01	1.02	11.92	5.35
22,29,30-trisnorneophopane	191	54.03	9.92	31.37	9.01	21.82	8.25	0.03	7.24	10.86	5.27	6.63	1.26	16.20	3.08	14.02	6.78	15.30	20.18	3.31	0.82	0.00	0.68	495.42	175.38
22,29,30-trisnorhopane	191	45.79	8.71	24.84	7.47	16.35	5.82	0.03	15.08	8.20	5.12	4.62	2.26	14.32	2.48	11.50	4.70	12.28	16.06	0.83	0.16	0.00	0.29	3.82	2.24
ab-norhopane	191	315.56	91.08	256.66	69.46	178.03	65.75	0.21	86.75	80.68	76.08	34.03	29.91	102.90	22.93	85.61	22.47	88.40	112.60	4.58	0.34	0.03	0.31	2.65	1.41
ab-+ba-norhopane	191	31.55	12.35	27.39	6.96	20.79	6.49	0.02	8.95	9.56	9.43	5.12	4.64	13.76	3.55	11.80	2.16	9.71	10.97	0.99	0.07	0.01	0.25	26.49	16.63
ab-hopane	191	251.43	95.76	240.31	63.34	174.11	60.85	0.18	82.39	87.19	90.26	44.29	47.59	104.58	30.01	86.67	20.25	73.65	87.12	5.58	0.51	0.02	0.02	6.30	5.42

3. Seasonal average concentration (pg/m³) of major NPOCs of PM_{2.5} in Hong Kong 2021 (continued)

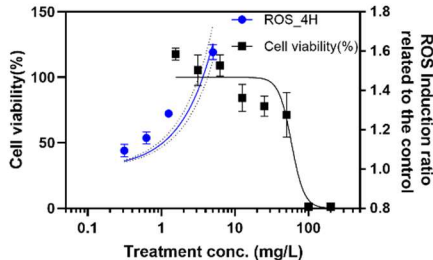
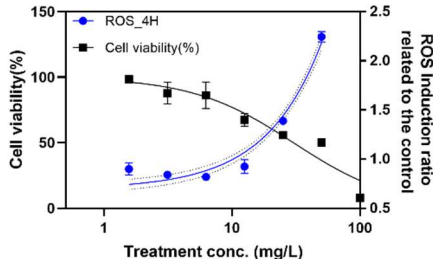
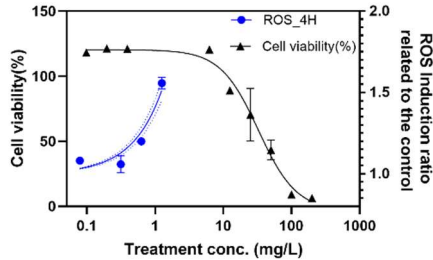
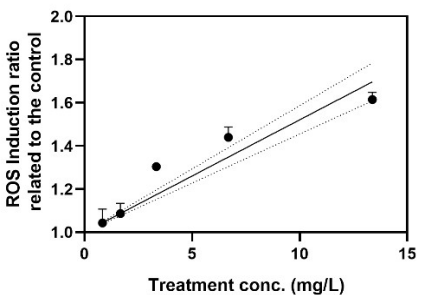
	m/z	Roadside								Urban								Background							
		Spring_RD		Summer_RD		Fall_RD		Winter_RD		Spring_6F		Summer_6F		Fall_6F		Winter_6F		Spring_HT		Summer_HT		Fall_HT		Winter_HT	
		Conc	STD	Conc	STD	Conc	STD	Conc	STD	Conc	STD	Conc	STD	Conc	STD	Conc	STD	Conc	STD	Conc	STD	Conc	STD	Conc	STD
ba-hopane	191	28.14	13.53	25.58	6.48	19.23	5.91	0.02	10.69	11.13	11.41	5.60	6.08	12.75	3.68	11.46	2.55	8.86	9.54	1.14	0.36	0.00	0.40	20.74	8.43
ab-22S-homohopane	191	103.75	47.56	105.04	33.13	91.29	30.82	0.09	41.90	47.48	55.20	30.13	39.44	41.17	11.14	39.27	11.33	29.68	31.17	4.26	0.63	0.01	0.52	3.39	0.80
ab-22R-homohopane	191	94.62	41.31	90.78	31.24	82.31	27.70	0.08	34.23	44.95	53.83	29.06	37.85	37.28	10.06	36.81	11.74	26.98	27.07	4.30	0.04	0.01	0.89	8.25	1.97
abS-bishomohopane	191	61.38	32.26	58.42	21.11	53.26	17.95	0.06	25.02	31.63	36.74	22.41	30.59	25.11	7.51	24.11	8.69	15.73	12.20	3.65	0.28	0.01	0.10	9.47	3.77
abR-bishomohopane	191	50.04	26.84	45.77	18.03	41.82	14.99	0.04	17.52	25.37	30.12	19.49	26.73	20.27	6.26	20.03	7.02	11.97	8.28	3.47	0.01	0.00	0.48	5.80	2.83
22S-trishomohopane (C33)	191	35.90	21.15	33.54	15.17	30.84	11.20	0.03	14.16	21.67	25.84	15.45	21.53	15.24	5.00	15.03	5.58	8.39	4.29	3.33	0.11	0.00	0.54	3.70	0.58
22R-trishomohopane (C33)	191	27.22	14.78	24.48	11.11	23.50	9.96	0.03	11.20	16.63	19.05	11.34	15.50	12.28	4.86	12.31	4.36	6.33	2.73	2.76	0.36	0.00	0.17	4.40	2.14
22S-tetrahomohopane (C34)	191	23.50	12.88	22.21	11.76	20.73	6.98	0.03	13.31	16.89	17.43	13.10	15.25	12.21	3.77	13.82	4.06	6.29	0.95	4.69	0.06	0.01	0.57	3.07	0.84
22R-tetrahomohopane (C34)	191	19.53	13.19	16.18	7.80	15.39	5.58	0.02	8.81	18.21	24.40	11.33	12.17	9.69	3.13	13.96	6.76	4.90	1.69	2.44	0.41	0.00	1.38	5.86	1.20
2S-pentashomohopane (C35)	191	18.31	12.48	16.82	8.80	14.63	6.16	0.02	7.67	11.19	14.15	8.67	12.87	8.26	3.39	8.25	2.63	3.67	0.58	2.04	0.26	0.00	1.58	3.90	0.62
2R-pentashomohopane (C35)	191	12.90	7.69	12.71	6.95	10.16	4.10	0.02	7.15	8.98	12.20	6.21	10.75	6.53	2.55	6.74	2.21	3.82	1.95	1.46	0.04	0.00	1.00	2.53	0.53
iaa 20S- +baa 20R-cholestane	217	25.10	5.77	16.07	2.93	9.26	3.72	0.01	5.97	4.03	2.07	2.54	0.98	9.48	1.47	8.28	3.63	0.90	0.52	1.10	0.34	0.00	0.26	2.00	0.09
aa 20R-cholestane	217	36.96	11.36	30.67	7.87	17.24	7.27	0.02	5.84	9.34	7.47	2.43	0.73	13.24	4.58	8.19	4.24	1.05	0.13	1.04	0.56	0.00	0.55	1.58	0.98
aa 20S 24S-methylcholestane	217	15.13	5.95	13.73	2.53	10.34	4.16	0.01	6.57	4.57	5.21	2.23	1.06	6.84	1.12	6.07	1.37	0.71	0.02	1.08	0.34	0.00	0.22	3.53	3.60
aa 20R 24R-methylcholestane	217	22.77	10.50	23.95	6.02	16.58	6.27	0.02	5.74	6.72	6.23	3.61	2.73	11.79	2.72	8.41	1.93	1.66	0.34	0.98	0.61	0.00	0.01	1.58	1.22
aa 20S 24R/S-ethylcholestane	217	26.02	15.73	30.27	9.43	22.03	8.71	0.02	9.62	9.75	9.21	5.23	4.46	13.32	2.88	9.20	3.28	2.15	0.34	1.47	0.14	0.00	0.27	2.02	0.79
aaa 20R-ethylcholestane	217	29.96	14.84	31.46	8.65	25.20	9.11	0.02	8.88	12.27	10.38	6.53	7.35	16.19	5.90	13.62	4.24	3.16	0.91	2.02	1.62	0.00	0.69	2.53	1.48
abb 20R-cholestane	218	33.06	8.89	20.51	4.84	12.99	4.83	0.02	9.87	7.54	4.86	4.32	2.59	12.47	2.20	10.08	3.21	1.08	0.44	0.71	0.03	0.00	0.40	3.79	1.80
abb 20S-cholestane	218	28.75	11.76	18.64	3.91	12.10	4.51	0.02	7.00	5.78	4.19	3.21	1.93	10.75	1.96	8.48	2.61	0.83	0.39	0.46	0.04	0.00	0.10	1.74	0.55
abb 20R-methylcholestane	218	20.60	8.47	18.33	4.07	13.00	3.92	0.02	5.88	7.03	6.20	3.25	2.22	10.11	1.75	7.73	2.05	1.14	0.28	0.48	0.07	0.00	0.04	1.55	0.59
bb 20S 24S-methylcholestane	218	17.78	8.20	16.11	3.41	11.96	3.69	0.01	2.91	5.94	5.65	3.01	2.85	9.27	2.28	8.28	2.30	0.96	0.06	0.65	0.03	0.00	0.09	1.43	0.83
abb-20R-ethylcholestane	218	22.18	11.11	21.60	5.32	16.58	5.76	0.02	6.47	8.24	7.63	4.19	4.44	9.38	1.47	7.87	1.75	1.50	0.64	1.72	1.62	0.01	0.25	1.76	1.18
abb 20S 24R-ethylcholestane	218	19.83	10.43	19.18	4.74	15.21	5.25	0.02	7.07	7.36	6.89	4.42	4.74	8.21	1.48	7.29	1.73	1.41	0.60	1.26	1.00	0.01	0.34	11.26	14.49

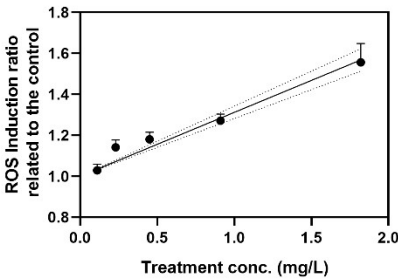
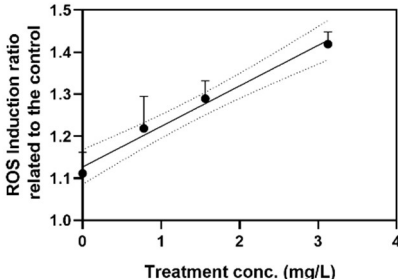
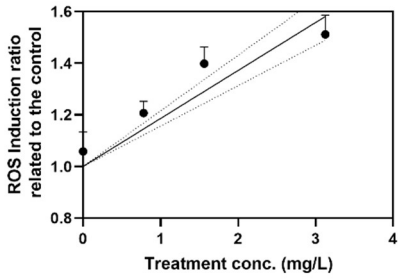
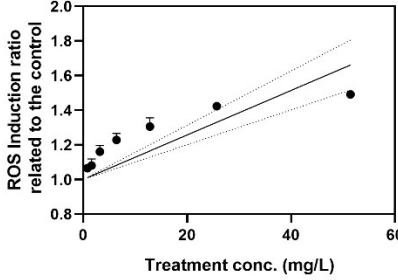
Appendix 2

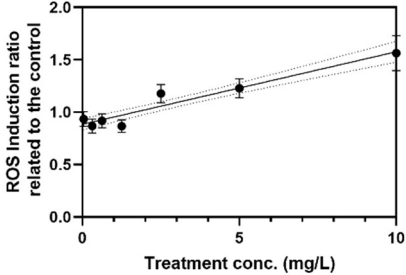
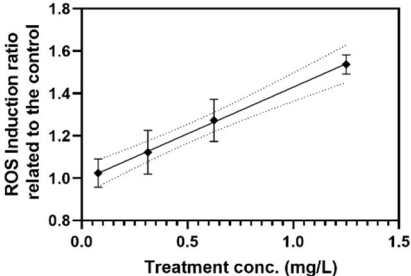
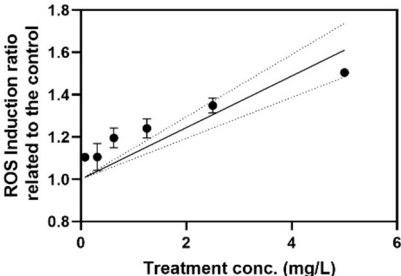
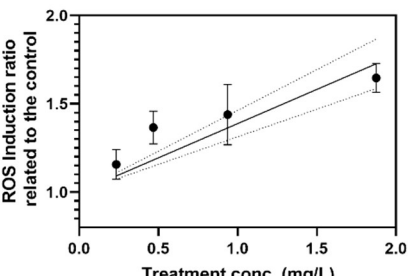
This section profiles individual chemicals for their potencies in inducing intracellular ROS in PM_{2.5}.

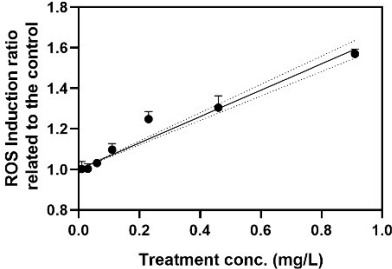
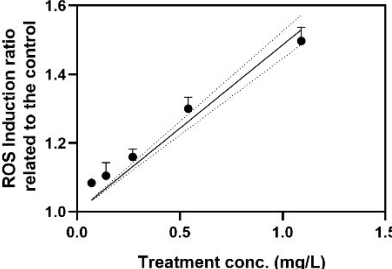
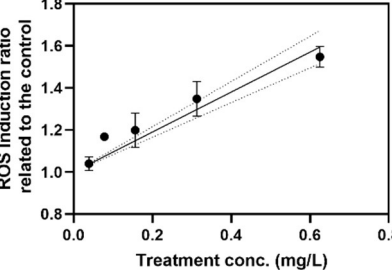
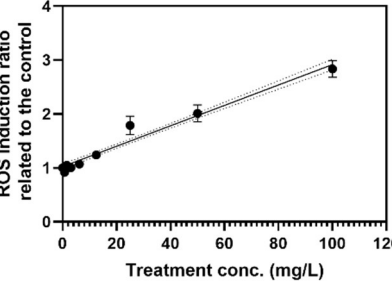
Chemical	Concentration-effect curve	EC _{IR1.5} (M)	REP _i
DMSO		n.a	n.a
t-BHP		2.84E-05 (±1.52E-05)	1
As		2.08E-06 (±8.22E-07)	13.7
Co		1.60E-03 (±1.11E-03)	1.78E-02

Chemical	Concentration-effect curve	EC _{IR1.5} (M)	REP _i
Cr		$3.64\text{E-}06$ $(\pm 2.35\text{E-}06)$	7.81
Cu		$5.40\text{E-}04$ $(\pm 4.80\text{E-}04)$	5.27E-02
Fe		$6.03\text{E-}05$ $(\pm 1.16\text{E-}05)$	0.47
Mn		$6.60\text{E-}05$ $(\pm 3.78\text{E-}05)$	0.43
Ni		$1.28\text{E-}04$ $(\pm 7.56\text{E-}03)$	0.22

Chemical	Concentration-effect curve	EC _{IR1.5} (M)	REP _i
Pb	 The graph for Pb shows two data series: ROS_4H (blue circles) and Cell viability (%) (black squares). The x-axis is Treatment conc. (mg/L) on a log scale from 0.1 to 100. The left y-axis is Cell viability (%) from 0 to 150. The right y-axis is ROS Induction ratio related to the control from 0.8 to 1.8. ROS_4H increases from ~1.0 at 0.1 mg/L to ~1.6 at 10 mg/L, then decreases. Cell viability remains near 100% until ~10 mg/L, then drops sharply to ~0% at 100 mg/L.	$3.68\text{E-}05$ $(\pm 2.71\text{E-}05)$	0.77
V	 The graph for V shows two data series: ROS_4H (blue circles) and Cell viability (%) (black squares). The x-axis is Treatment conc. (mg/L) on a log scale from 1 to 100. The left y-axis is Cell viability (%) from 0 to 150. The right y-axis is ROS Induction ratio related to the control from 0.5 to 2.5. ROS_4H increases from ~1.0 at 1 mg/L to ~2.3 at 100 mg/L. Cell viability decreases from ~100% at 1 mg/L to ~0% at 100 mg/L.	$3.25\text{E-}04$ $(\pm 1.91\text{E-}04)$	$8.76\text{E-}02$
Zn	 The graph for Zn shows two data series: ROS_4H (blue circles) and Cell viability (%) (black triangles). The x-axis is Treatment conc. (mg/L) on a log scale from 0.1 to 1000. The left y-axis is Cell viability (%) from 0 to 150. The right y-axis is ROS Induction ratio related to the control from 1.0 to 2.0. ROS_4H increases from ~1.0 at 0.1 mg/L to ~1.5 at 10 mg/L, then decreases. Cell viability remains near 100% until ~10 mg/L, then drops sharply to ~0% at 1000 mg/L.	$1.86\text{E-}05$ $(\pm 1.38\text{E-}05)$	1.53
Phenanthrene (Phe)	 The graph for Phenanthrene shows ROS Induction ratio related to the control (black circles). The x-axis is Treatment conc. (mg/L) from 0 to 15. The y-axis is ROS Induction ratio related to the control from 1.0 to 2.0. The data points show a linear increase from ~1.0 at 0 mg/L to ~1.6 at 15 mg/L.	$5.40\text{E-}05$ $(\pm 4.78\text{E-}05)$	0.53

Chemical	Concentration-effect curve	EC _{IR1.5} (M)	REP _i
Anthracene (Ant)		9.01E-06 (±5.79E-06)	3.16
Fluoranthene (Fla)		2.56E-05 (±1.08E-05)	1.11
Chrysene (Chr)		1.18E-05 (±8.15E-06)	2.41
Pyrene (Pyr)		1.93E-04 (±1.88E-04)	0.15

Chemical	Concentration-effect curve	EC _{IR1.5} (M)	REP _i
Benz [a] anthracene (BaA)		3.17E-05 (±1.63E-05)	0.90
Benzo [b] fluoranthene (BbF)		4.50E-06 (±2.01E-06)	6.33
Benzo [k] fluoranthene (BkF)		1.62E-05 (±1.24E-05)	1.75
Benzo [a] pyrene (BaP)		4.99E-06 (±4.11E-06)	5.69

Chemical	Concentration-effect curve	EC _{IR1.5} (M)	REP _i
Benzo [ghi] perylene (BghiP)		2.78E- 06 (±1.88E- 06)	10.2
Indeno[1,2,3- cd] pyrene (IcdP)		3.73E- 06 (±1.21E- 06)	7.62
Dibenzo [a,h] anthracene (DahA)		1.89E- 06 (±8.76E- 07)	15.1
Carbon black (CB)	Printex U 	2.03E- 03 (±1.78E- 03)	1.40E-02

Appendix 3

This section includes the results of QAQC for chemical analyses.

Sample Name	As	Cd	Co	Cr	Cu	Fe	Mn
	193.759 ng/m ³	226.502 ng/m ³	238.892 ng/m ³	267.716 ng/m ³	327.396 ng/m ³	259.941 ng/m ³	259.373 ng/m ³
Control1-1	2.99	0.13	0.41	0.97	1.65	4.79	0.08
Control1-2	3.33	0.16	0.50	0.60	2.15	7.69	0.13
Control1-3	3.63	0.16	0.43	0.72	2.10	11.37	0.24
Control2-1	3.11	0.09	0.64	1.29	9.04	17.22	n.a.
Control2-2	7.95	0.13	0.65	4.29	9.38	43.94	4.75
Control3-1	3.29	0.13	0.85	1.90	9.18	7.85	n.a.
Control3-2	3.33	0.13	0.85	1.90	9.18	4.48	n.a.
Control3-3	1.31	0.71	0.74	1.02	3.26	23.13	4.63
Blank1-1	2.72	0.13	0.41	0.72	1.46	2.65	n.a.
Blank1-2	2.97	0.13	0.43	0.60	1.48	1.47	0.03
Blank1-3	3.01	0.14	0.45	0.58	1.60	1.21	0.07
Blank2-1	3.72	0.13	0.84	2.72	11.23	130.94	0.81
Blank2-2	5.60	0.15	0.83	20.10	10.91	155.31	2.78
Blank3-1	3.68	0.14	0.85	1.34	9.84	45.72	0.02
Blank3-2	3.91	0.14	0.76	2.63	9.78	44.30	0.13
Blank3-3	1.74	0.77	0.68	3.81	4.22	89.13	5.32
Ref1-1	45.54	17.22	10.10	30.78	118.84	7032.97	152.28
Ref1-2	48.92	17.97	11.05	38.43	131.82	7860.75	168.83
Ref1-3	45.19	17.15	9.94	24.55	119.91	7155.37	146.06
Ref2-1	24.37	9.20	5.55	13.11	64.22	3581.69	71.69
Ref2-2	24.19	8.97	5.66	4.16	65.11	3688.78	51.77
Ref3-1	22.95	8.40	5.30	11.10	59.77	3379.06	55.86
Ref3-2	22.24	8.66	5.55	5.37	61.91	3496.65	44.65
Sample Name	Mo	Ni	Pb	Sb	Se	V	Zn
	202.095 ng/m ³	231.604 ng/m ³	168.215 ng/m ³	217.581 ng/m ³	204.050 ng/m ³	311.071 ng/m ³	213.856 ng/m ³
Control1-1	0.52	0.51	1.96	3.20	6.88	0.72	2.69
Control1-2	0.54	0.67	2.53	3.66	7.88	0.78	5.07
Control1-3	0.51	0.64	2.86	3.58	7.58	0.69	14.43
Control2-1	0.73	1.29	n.a.	4.38	8.56	5.51	0.00
Control2-2	0.99	2.10	0.95	5.05	11.44	5.60	16.90
Control3-1	0.78	1.19	n.a.	4.77	9.80	5.64	1.71
Control3-2	0.77	1.19	n.a.	4.89	9.94	5.66	0.00

Control3-3	0.30	1.32	8.47	0.47	0.55	0.36	19.21
Blank1-1	0.55	0.48	1.87	3.20	7.10	0.69	2.28
Blank1-2	0.47	0.53	2.06	3.29	6.96	0.67	2.40
Blank1-3	0.46	0.55	2.06	3.56	7.60	0.76	2.19
Blank2-1	1.97	6.56	1.31	4.82	9.86	5.59	32.02
Blank2-2	2.90	7.21	2.37	5.27	13.06	5.68	25.26
Blank3-1	0.98	1.87	n.a.	4.98	10.03	5.71	12.45
Blank3-2	1.06	1.96	n.a.	5.09	9.96	5.75	12.72
Blank3-3	0.85	4.75	9.57	1.22	3.29	0.37	36.47
Ref1-1	4.29	16.26	1424.28	11.58	29.00	26.86	993.93
Ref1-2	4.71	17.79	1581.36	12.63	33.62	29.89	1115.80
Ref1-3	4.36	16.15	1401.51	12.06	29.18	26.86	990.55
Ref2-1	3.17	8.93	688.66	9.00	17.74	17.52	515.02
Ref2-2	3.06	8.98	681.72	9.91	17.52	17.17	521.25
Ref3-1	2.94	8.31	626.57	10.07	19.04	16.28	478.02
Ref3-2	2.94	8.75	649.16	10.02	17.79	15.96	489.76

Element	Low-Level Recovery (%)	High-Level Recovery (%)	RSD (%)
Aluminum (Al)	42.2±3.5	57.3±1.8	2.6
Arsenic (As)	88.5±4.2	95.1±1.8	2.5
Barium (Ba)	87.6±3.9	96.8±1.5	3.0
Cadmium (Cd)	94.2±2.8	103.6±1.4	2.2
Chromium (Cr)	85.7±3.8	93.6±1.5	3.1
Cobalt (Co)	84.3±4.5	96.5±2.1	3.8
Copper (Cu)	88.9±3.2	95.8±1.7	2.9
Iron (Fe)	91.2±2.7	96.8±1.2	2.2
Magnesium (Mg)	76.4±4.1	85.9±1.9	3.3

Element	Low-Level Recovery (%)	High-Level Recovery (%)	RSD (%)
Manganese (Mn)	87.8±4.3	105.5±2.4	3.5
Nickel (Ni)	82.4±4.7	92.8±2.5	4.1
Lead (Pb)	93.5±3.1	103.9±1.6	2.7
Antimony (Sb)	85.6±4.4	97.2±2.0	3.6
Vanadium (V)	89.7±3.6	104.8±1.8	3.0
Zinc (Zn)	90.8±3.3	105.8±1.5	2.8

EPA PAHs	Low-Level Recovery (%)	High-Level Recovery (%)	RSD (%)
Naphthalene (Nap)	83.5±5.7	93.2±2.6	4.0
Acenaphthylene (Acy)	81.2±6.1	91.5±3.0	4.4
Acenaphthene (Ace)	84.1±5.4	94.0±2.5	3.9
Fluorene (Flu)	82.8±5.6	92.7±2.8	4.1
Phenanthrene (Phe)	82.6±5.5	92.3±2.8	3.9
Anthracene (Ant)	80.3±6.0	90.9±3.2	4.3
Fluoranthene (Fla)	83.2±5.3	93.5±2.7	3.8
Pyrene (Pyr)	84.5±5.2	93.1±2.6	3.7
Benzo[a]anthracene (BaA)	79.8±5.9	89.6±3.0	4.2

EPA PAHs	Low-Level Recovery (%)	High-Level Recovery (%)	RSD (%)
Chrysene (Chr)	81.7±5.6	91.8±2.9	4.0
Benzo[b]fluoranthene (BbF)	78.5±6.2	89.8±2.9	4.5
Benzo[k]fluoranthene (BkF)	77.9±6.3	89.2±3.1	4.6
Benzo[a]pyrene (BaP)	78.2±6.1	89.5±2.8	4.4
Indeno[1,2,3-cd]pyrene (IcdP)	79.2±5.8	88.7±3.1	4.3
Dibenz[a,h]anthracene (DahA)	78.8±6.0	88.3±3.2	4.4
Benzo[ghi]perylene (BghiP)	79.5±5.7	89.4±3.0	4.2

References

- Acuña Askar, K., González, L. T., Mendoza, A., Kharissova, O. V., Rodríguez-Garza, A., Lara, E. M., Campos, A., López-Serna, D., Bautista-Carrillo, L. M., Alfaro-Barbosa, J. M., & Longoria-Rodríguez, F. E. (2022). Chemical composition, optical properties and sources of PM_{2.5} from a highly urbanized region in northeastern Mexico. *Frontiers in Environmental Science*, 10. <https://doi.org/10.3389/fenvs.2022.929449>
- Air Pollution Control (Fuel for Vessels) Regulation (2019). <https://www.elegislation.gov.hk/hk/cap311AB>
- Aurell, J., Gullett, B. K., & Tabor, D. (2015). Emissions from southeastern U.S. grasslands and pine savannas: Comparison of aerial and ground field measurements with laboratory burns. *Atmospheric Environment*, 111, 170–178. <https://doi.org/10.1016/j.atmosenv.2015.03.001>
- Bates, J. T., Fang, T., Verma, V., Zeng, L., Weber, R. J., Tolbert, P. E., Abrams, J. Y., Sarnat, S. E., Klein, M., Mulholland, J. A., & Russell, A. G. (2019). Review of Acellular Assays of Ambient Particulate Matter Oxidative Potential: Methods and Relationships with Composition, Sources, and Health Effects. *Environmental Science & Technology*, 53(8), 4003–4019. <https://doi.org/10.1021/acs.est.8b03430>
- Baumgartner, J., Zhang, Y., Schauer, J. J., Huang, W., Wang, Y., & Ezzati, M. (2014). Highway proximity and black carbon from cookstoves as a risk factor for higher blood pressure in rural China. *Proceedings of the National Academy of Sciences*, 111(36), 13229–13234. <https://doi.org/10.1073/pnas.1317176111>
- Bautista, A. T., Pabroa, P. C. B., Santos, F. L., Quirit, L. L., Asis, J. L. B., Dy, M. A. K., & Martinez, J. P. G. (2015). Intercomparison between NIOSH, IMPROVE_A, and EUSAAR_2 protocols: Finding an optimal thermal–optical protocol for philippines OC/EC samples. *Atmospheric Pollution Research*, 6(2), 334–342. <https://doi.org/10.5094/APR.2015.037>
- Belden, J. B., Gilliom, R. J., & Lydy, M. J. (2007). How well can we predict the toxicity of pesticide mixtures to aquatic life? *Integrated Environmental Assessment and Management*, 3(3), 364–372. <https://doi.org/10.1002/ieam.5630030307>
- Bernhard, W. (2016). Lung surfactant: Function and composition in the context of development and respiratory physiology. *Annals of Anatomy - Anatomischer Anzeiger*, 208, 146–150. <https://doi.org/10.1016/j.aanat.2016.08.003>
- Besis, A., Gallou, D., Avgenikou, A., Serafeim, E., & Samara, C. (2022). Size-dependent in vitro inhalation bioaccessibility of PAHs and O/N PAHs—Implications to inhalation risk assessment. *Environmental Pollution (Barking, Essex: 1987)*, 301, 119045. <https://doi.org/10.1016/j.envpol.2022.119045>
- Bodor, Z., Bodor, K., Keresztesi, Á., & Szép, R. (2020). Major air pollutants seasonal variation analysis and long-range transport of PM₁₀ in an urban environment with specific climate condition in Transylvania (Romania). *Environmental Science and Pollution Research*, 27(30), 38181–38199. <https://doi.org/10.1007/s11356-020-09838-2>
- Boisa, N., Elom, N., Dean, J. R., Deary, M. E., Bird, G., & Entwistle, J. A. (2014). Development and

- application of an inhalation bioaccessibility method (IBM) for lead in the PM₁₀ size fraction of soil. *Environment International*, 70, 132–142. <https://doi.org/10.1016/j.envint.2014.05.021>
- Boongla, Y., Chanonmuang, P., Hata, M., Furuuchi, M., & Phairuang, W. (2021). The characteristics of carbonaceous particles down to the nanoparticle range in rangsit city in the bangkok metropolitan region, thailand. *Environmental Pollution*, 272, 115940. <https://doi.org/10.1016/j.envpol.2020.115940>
- Borm, P., Cakmak, G., Jermann, E., Weishaupt, C., Kempers, P., Vanschooten, F., Oberdorster, G., & Schins, R. (2005). Formation of PAH–DNA adducts after in vivo and vitro exposure of rats and lung cells to different commercial carbon blacks. *Toxicology and Applied Pharmacology*, 205(2), 157–167. <https://doi.org/10.1016/j.taap.2004.10.020>
- Briggs, N. L., & Long, C. M. (2016). Critical review of black carbon and elemental carbon source apportionment in Europe and the United States. *Atmospheric Environment*, 144, 409–427. <https://doi.org/10.1016/j.atmosenv.2016.09.002>
- Brook, R. D., Rajagopalan, S., Pope, C. A., Brook, J. R., Bhatnagar, A., Diez-Roux, A. V., Holguin, F., Hong, Y., Luepker, R. V., Mittleman, M. A., Peters, A., Siscovick, D., Smith, S. C., Whitsel, L., & Kaufman, J. D. (2010). Particulate matter air pollution and cardiovascular disease. *Circulation*, 121(21), 2331–2378. <https://doi.org/10.1161/CIR.0b013e3181d8e1>
- Brown, D. M., Hutchison, L., Donaldson, K., MacKenzie, S. J., Dick, C. A. J., & Stone, V. (2007). The effect of oxidative stress on macrophages and lung epithelial cells: The role of phosphodiesterases 1 and 4. *Toxicology Letters*, 168(1), 1–6. <https://doi.org/10.1016/j.toxlet.2006.10.016>
- Brown, S. G., Eberly, S., Paatero, P., & Norris, G. A. (2015). Methods for estimating uncertainty in PMF solutions: Examples with ambient air and water quality data and guidance on reporting PMF results. *Science of The Total Environment*, 518–519, 626–635. <https://doi.org/10.1016/j.scitotenv.2015.01.022>
- Bustaffa, E., Stoccoro, A., Bianchi, F., & Migliore, L. (2014). Genotoxic and epigenetic mechanisms in arsenic carcinogenicity. *Archives of Toxicology*, 88(5), 1043–1067. <https://doi.org/10.1007/s00204-014-1233-7>
- Calas, A., Uzu, G., Martins, J. M. F., Voisin, D., Spadini, L., Lacroix, T., & Jaffrezou, J.-L. (2017). The importance of simulated lung fluid (SLF) extractions for a more relevant evaluation of the oxidative potential of particulate matter. *Scientific Reports*, 7(1), 11617. <https://doi.org/10.1038/s41598-017-11979-3>
- Caldwell, R. A., Grubb, B. R., Tarran, R., Boucher, R. C., Knowles, M. R., & Barker, P. M. (2001). In Vivo Airway Surface Liquid Cl⁻ Analysis with Solid-State Electrodes. *Journal of General Physiology*, 119(1), 3–14. <https://doi.org/10.1085/jgp.119.1.3>
- Carneseccchi, E., Svendsen, C., Lasagni, S., Grech, A., Quignot, N., Amzal, B., Toma, C., Tosi, S., Rortais, A., Cortinas-Abrahantes, J., Capri, E., Kramer, N., Benfenati, E., Spurgeon, D., Guillot, G., & Dorne, J. L. C. M. (2019). Investigating combined toxicity of binary mixtures in bees: Meta-analysis of laboratory tests, modelling, mechanistic basis and implications for risk assessment. *Environment International*, 133, 105256. <https://doi.org/10.1016/j.envint.2019.105256>

Cedergreen, N. (2014). Quantifying synergy: A systematic review of mixture toxicity studies within environmental toxicology. *PLOS ONE*, 9(5), e96580. <https://doi.org/10.1371/journal.pone.0096580>

Chen, H., & Yu, J. Z. (2025). An online instrument for assessing oxidative potential of ambient particulate matter via dithiothreitol assay, using particle-into-liquid sampler (PILS) coupled with improved light absorption measurement. *Atmospheric Environment*, 343, 120980. <https://doi.org/10.1016/j.atmosenv.2024.120980>

Chen, Q., Luo, X.-S., Chen, Y., Zhao, Z., Hong, Y., Pang, Y., Huang, W., Wang, Y., & Jin, L. (2019). Seasonally varied cytotoxicity of organic components in PM_{2.5} from urban and industrial areas of a Chinese megacity. *Chemosphere*, 230, 424–431. <https://doi.org/10.1016/j.chemosphere.2019.04.226>

Chen, X., Wu, D., Tan, Y., Song, X., Chen, J., & Li, Q. (2024). Absence of a Causal Link between Elemental Carbon Exposure and Short-Term Respiratory Toxicity in Human-Derived Organoids and Cellular Models. *Environmental Science & Technology*. <https://doi.org/10.1021/acs.est.4c11256>

Chen, X.-C., Cao, J.-J., Ward, T. J., Tian, L.-W., Ning, Z., Gali, N. K., Aquilina, N. J., Yim, S. H.-L., Qu, L., & Ho, K.-F. (2020). Characteristics and toxicological effects of commuter exposure to black carbon and metal components of fine particles (PM_{2.5}) in Hong Kong. *Science of The Total Environment*, 742, 140501. <https://doi.org/10.1016/j.scitotenv.2020.140501>

Chen, X.-C., Ward, T. J., Ho, K.-F., Sarkar, C., & Webster, C. (2022). Characteristics and health risks of personal exposure to particle-bound PAHs for Hong Kong adult residents: From ambient pollution to indoor exposure. *Indoor Air*, 32(1), e12956. <https://doi.org/10.1111/ina.12956>

Chen, Y., Zhang, L., Wu, Y., Huang, W., Luo, Z., Li, Y., Qi, Y., Liu, X., Shen, G., Liu, S., & Tao, S. (2024). Cytotoxicity and Epithelial Barrier Toxicity of Fine Particles from Residential Biomass Pellet Burning. *Environ. Sci. Technol.*

Cheng, H., Narzo, A. D., Howell, D., Yevdokimova, K., Zhang, J., Zhang, X., Pan, Q., Zhang, Z., Rogers, L., & Hao, K. (2022). Ambient Air Pollutants and Traffic Factors Were Associated with Blood and Urine Biomarkers and Asthma Risk. *Environmental Science & Technology*, 56(11), 7298–7307. <https://doi.org/10.1021/acs.est.1c06916>

Cheng, Y., Lee, S. C., Ho, K. F., Chow, J. C., Watson, J. G., Louie, P. K. K., Cao, J. J., & Hai, X. (2010). Chemically-specified on-road PM_{2.5} motor vehicle emission factors in Hong Kong. *Science of The Total Environment*, 408(7), 1621–1627. <https://doi.org/10.1016/j.scitotenv.2009.11.061>

Cheng, Y., Lee, S., Gu, Z., Ho, K., Zhang, Y., Huang, Y., Chow, J. C., Watson, J. G., Cao, J., & Zhang, R. (2015). PM_{2.5} and PM_{10-2.5} chemical composition and source apportionment near a Hong Kong roadway. *Particuology*, 18, 96–104. <https://doi.org/10.1016/j.partic.2013.10.003>

Cheng, Y., Ma, Y., Dong, B., Qiu, X., & Hu, D. (2021). Pollutants from primary sources dominate the oxidative potential of water-soluble PM_{2.5} in hong kong in terms of dithiothreitol (DTT) consumption and hydroxyl radical production. *Journal of Hazardous Materials*, 405, 124218. <https://doi.org/10.1016/j.jhazmat.2020.124218>

Cho, C.-C., Hsieh, W.-Y., Tsai, C.-H., Chen, C.-Y., Chang, H.-F., & Lin, C.-S. (2018). In Vitro and In

Vivo Experimental Studies of PM_{2.5} on Disease Progression. *International Journal of Environmental Research and Public Health*, 15(7), Article 7. <https://doi.org/10.3390/ijerph15071380>

Choi, H., Harrison, R., Komulainen, H., & Saborit, J. M. D. (2010). Polycyclic aromatic hydrocarbons. In *WHO Guidelines for Indoor Air Quality: Selected Pollutants*. World Health Organization. <https://www.ncbi.nlm.nih.gov/books/NBK138709/>

Chow, J. C., Watson, J. G., Chen, L.-W. A., Chang, M. C. O., Robinson, N. F., Trimble, D., & Kohl, S. (2007). The IMPROVE_A temperature protocol for thermal/optical carbon analysis: Maintaining consistency with a long-term database. *Journal of the Air & Waste Management Association*, 57(9), 1014–1023. <https://doi.org/10.3155/1047-3289.57.9.1014>

Chow, J. C., Watson, J. G., Pritchett, L. C., Pierson, W. R., Frazier, C. A., & Purcell, R. G. (1993). The thermal/optical reflectance carbon analysis system: Description, evaluation and applications in U.S. air quality studies. *Atmospheric Environment. Part A. General Topics*, 27(8), 1185–1201. [https://doi.org/10.1016/0960-1686\(93\)90245-T](https://doi.org/10.1016/0960-1686(93)90245-T)

Chow, J. C., Watson, John G., Crow, Dale, Lowenthal, Douglas H., & Merrifield, T. (2001). Comparison of IMPROVE and NIOSH carbon measurements. *Aerosol Science and Technology*, 34(1), 23–34. <https://doi.org/10.1080/02786820119073>

Chow, W. S., Huang, X. H. H., Leung, K. F., Huang, L., Wu, X., & Yu, J. Z. (2022). Molecular and elemental marker-based source apportionment of fine particulate matter at six sites in Hong Kong, China. *Science of The Total Environment*, 813, 152652. <https://doi.org/10.1016/j.scitotenv.2021.152652>

Chow, W. S., Liao, K., Huang, X. H. H., Leung, K. F., Lau, A. K. H., & Yu, J. Z. (2022a). *Measurement Report: Ten-year trend of PM_{2.5}: major components and source tracers from 2008 to 2017 in an urban site of Hong Kong, China* [Preprint]. Aerosols/Field Measurements/Troposphere/Chemistry (chemical composition and reactions). <https://doi.org/10.5194/acp-2022-100>

Colonna, K. J., Koutrakis, P., Kinney, P. L., Cooke, R. M., & Evans, J. S. (2022). Mortality Attributable to Long-Term Exposure to Ambient Fine Particulate Matter: Insights from the Epidemiologic Evidence for Understudied Locations. *Environmental Science & Technology*, 56(11), 6799–6812. <https://doi.org/10.1021/acs.est.1c08343>

Courcot, E., Leclerc, J., Lafitte, J.-J., Mensier, E., Jaillard, S., Gosset, P., Shirali, P., Pottier, N., Broly, F., & Lo-Guidice, J.-M. (2012). Xenobiotic metabolism and disposition in human lung cell models: Comparison with in vivo expression profiles. *Drug Metabolism and Disposition*, 40(10), 1953–1965. <https://doi.org/10.1124/dmd.112.046896>

Cui, L., Song, X., & Zhong, G. (2021). Comparative analysis of three methods for HYSPLIT atmospheric trajectories clustering. *Atmosphere*, 12(6), Article 6. <https://doi.org/10.3390/atmos12060698>

Davies, N. M., & Feddah, M. R. (2003). A novel method for assessing dissolution of aerosol inhaler products. *International Journal of Pharmaceutics*, 255(1), 175–187. [https://doi.org/10.1016/S0378-5173\(03\)00091-7](https://doi.org/10.1016/S0378-5173(03)00091-7)

De Groot, G. M., Oomen, A. G., & Mennen, M. G. (2017). *Evaluation of health risks of playing sports*

on synthetic turf pitches with rubber granulate. RIVM. <https://doi.org/10.21945/RIVM-2017-0017>

Dean, J. R., Elom, N. I., & Entwistle, J. A. (2017). Use of simulated epithelial lung fluid in assessing the human health risk of Pb in urban street dust. *Science of The Total Environment*, 579, 387–395. <https://doi.org/10.1016/j.scitotenv.2016.11.085>

Demir, T., Karakaş, D., & Yenisoý-Karakaş, S. (2022). Source identification of exhaust and non-exhaust traffic emissions through the elemental carbon fractions and Positive Matrix Factorization method. *Environmental Research*, 204, 112399. <https://doi.org/10.1016/j.envres.2021.112399>

Dilger, M., Orasche, J., Zimmermann, R., Paur, H.-R., Diabaté, S., & Weiss, C. (2016). Toxicity of wood smoke particles in human A549 lung epithelial cells: The role of PAHs, soot and zinc. *Archives of Toxicology*, 90(12), 3029–3044. <https://doi.org/10.1007/s00204-016-1659-1>

Drainage Services Department. (2021, January 21). *Stanley Sewage Treatment Works – Stanley Sewage Treatment Works*. Drainage Services Department. <https://www.dsd.gov.hk/EN/HTML/20520.html>

Escher, B.I., Braun, G., & Zarfl, C. (2020). Exploring the Concepts of Concentration Addition and Independent Action Using a Linear Low-Effect Mixture Model. *Environmental Toxicology and Chemistry*, 39(12), 2552–2559. <https://doi.org/10.1002/etc.4868>

Escher, B. I., Stapleton, H. M., & Schymanski, E. L. (2020). Tracking complex mixtures of chemicals in our changing environment. *Science*, 367(6476), 388–392. <https://doi.org/10.1126/science.aay6636>

Escher, B. I., van Daele, C., Dutt, M., Tang, J. Y. M., & Altenburger, R. (2013). Most Oxidative Stress Response In Water Samples Comes From Unknown Chemicals: The Need For Effect-Based Water Quality Trigger Values. *Environmental Science & Technology*, 47(13), 7002–7011. <https://doi.org/10.1021/es304793h>

EU air quality standards—European commission. (n.d.). Retrieved June 11, 2025, from https://environment.ec.europa.eu/topics/air/air-quality/eu-air-quality-standards_en

Expósito, A., Markiv, B., Santibáñez, M., Fadel, M., Ledoux, F., Courcot, D., & Fernández-Olmo, I. (2024). Ascorbate oxidation driven by PM_{2.5}-bound metal(loid)s extracted in an acidic simulated lung fluid in relation to their bioaccessibility. *Air Quality, Atmosphere & Health*, 17(1), 177–189. <https://doi.org/10.1007/s11869-023-01436-8>

Fang, T., Guo, H., Zeng, L., Verma, V., Nenes, A., & Weber, R. J. (2017). Highly Acidic Ambient Particles, Soluble Metals, and Oxidative Potential: A Link between Sulfate and Aerosol Toxicity. *Environmental Science & Technology*, 51(5), 2611–2620. <https://doi.org/10.1021/acs.est.6b06151>

Fang, T., Verma, V., Bates, J. T., Abrams, J., Klein, M., Strickland, M. J., Sarnat, S. E., Chang, H. H., Mulholland, J. A., Tolbert, P. E., Russell, A. G., & Weber, R. J. (2016). Oxidative potential of ambient water-soluble PM_{2.5} in the southeastern united states: Contrasts in sources and health associations between ascorbic acid (AA) and dithiothreitol (DTT) assays. *Atmospheric Chemistry and Physics*, 16(6), 3865–3879. <https://doi.org/10.5194/acp-16-3865-2016>

Feng, L., Yang, X., Asweto, C. O., Wu, J., Zhang, Y., Hu, H., Shi, Y., Duan, J., & Sun, Z. (2017). Low-

dose combined exposure of nanoparticles and heavy metal compared with PM_{2.5} in human myocardial AC16 cells. *Environmental Science and Pollution Research*, 24(36), 27767–27777. <https://doi.org/10.1007/s11356-017-0228-3>

Friese, E., & Ebel, A. (2010). Temperature dependent thermodynamic model of the system H⁽⁺⁾-NH₄⁽⁺⁾-Na⁽⁺⁾-SO₄²⁻-NO₃⁻-Cl⁻-H₂O. *The Journal of Physical Chemistry. A*, 114(43), 11595–11631. <https://doi.org/10.1021/jp101041j>

Fu, J., Ji, J., Luo, L., Li, X., Zhuang, X., Ma, Y., Wen, Q., Zhu, Y., Ma, J., Huang, J., Zhang, D., & Lu, S. (2023). Temporal and spatial distributions, source identification, and health risk assessment of polycyclic aromatic hydrocarbons in PM_{2.5} from 2016 to 2021 in Shenzhen, China. *Environmental Science and Pollution Research*, 30(47), 103788–103800. <https://doi.org/10.1007/s11356-023-29686-0>

Fuller, R., Landrigan, P. J., Balakrishnan, K., Bathan, G., Bose-O'Reilly, S., Brauer, M., Caravanos, J., Chiles, T., Cohen, A., Corra, L., Cropper, M., Ferraro, G., Hanna, J., Hanrahan, D., Hu, H., Hunter, D., Janata, G., Kupka, R., Lanphear, B., ... Yan, C. (2022). Pollution and health: A progress update. *The Lancet Planetary Health*, 6(6), e535–e547. [https://doi.org/10.1016/S2542-5196\(22\)00090-0](https://doi.org/10.1016/S2542-5196(22)00090-0)

Fussell, J. C., Franklin, M., Green, D. C., Gustafsson, M., Harrison, R. M., Hicks, W., Kelly, F. J., Kishta, F., Miller, M. R., Mudway, I. S., Oroumijeh, F., Selley, L., Wang, M., & Zhu, Y. (2022a). A Review of Road Traffic-Derived Non-Exhaust Particles: Emissions, Physicochemical Characteristics, Health Risks, and Mitigation Measures. *Environmental Science & Technology*, 56(11), 6813–6835. <https://doi.org/10.1021/acs.est.2c01072>

Gamble, J. L. (1945). Chemical Anatomy, Physiology and Pathology of Extracellular Fluid. *The Quarterly Review of Biology*, 20(2). <https://doi.org/doi.org/10.1086/394820>

Gao, B., Wang, X.-M., Zhao, X.-Y., Ding, X., Fu, X.-X., Zhang, Y.-L., He, Q.-F., Zhang, Z., Liu, T.-Y., Huang, Z.-Z., Chen, L.-G., Peng, Y., & Guo, H. (2015). Source apportionment of atmospheric PAHs and their toxicity using PMF: Impact of gas/particle partitioning. *Atmospheric Environment*, 103, 114–120. <https://doi.org/10.1016/j.atmosenv.2014.12.006>

Grahame, T. J., Klemm, R., & Schlesinger, R. B. (2014). Public health and components of particulate matter: The changing assessment of black carbon. *Journal of the Air & Waste Management Association* (1995), 64(6), 620–660. <https://doi.org/10.1080/10962247.2014.912692>

Gregory, T. J., Longmore, W. J., Moxley, M. A., Whitsett, J. A., Reed, C. R., Fowler III, A. A., Hudson, L. D., Maunder, R. J., Crim, C., & Hyers, T. M. (1991). Surfactant chemical composition and biophysical activity in acute respiratory distress syndrome. *Journal of Clinical Investigation*, 88(6), 1976–1981. Scopus. <https://doi.org/10.1172/JCI115523>

Guo, B., Zebda, R., Drake, S. J., & Sayes, C. M. (2009). Synergistic effect of co-exposure to carbon black and Fe₂O₃ nanoparticles on oxidative stress in cultured lung epithelial cells. *Particle and Fibre Toxicology*, 6(1), 4. <https://doi.org/10.1186/1743-8977-6-4>

Guo, Q., Chen, K., & Xu, G. (2023). Characteristics and sources of water-soluble inorganic ions in PM_{2.5} in urban nanjing, China. *Atmosphere*, 14(1), Article 1. <https://doi.org/10.3390/atmos14010135>

- Guo, Z., Chen, X., Wu, D., Huo, Y., Cheng, A., Liu, Y., Li, Q., & Chen, J. (2023). Higher Toxicity of Gaseous Organics Relative to Particulate Matters Emitted from Typical Cooking Processes. *Environmental Science & Technology*, 57(44), 17022–17031. <https://doi.org/10.1021/acs.est.3c05425>
- Gurgueira, S. A., Lawrence, J., Coull, B., Murthy, G. G. K., & González-Flecha, B. (2002). Rapid increases in the steady-state concentration of reactive oxygen species in the lungs and heart after particulate air pollution inhalation. *Environmental Health Perspectives*, 110(8), 749–755. <https://doi.org/10.1289/ehp.02110749>
- Han, Y., Wang, Z., Zhou, J., Che, H., Tian, M., Wang, H., Shi, G., Yang, F., Zhang, S., & Chen, Y. (2021). PM_{2.5}-bound heavy metals in southwestern China: Characterization, sources, and health risks. *Atmosphere*, 12(7), Article 7. <https://doi.org/10.3390/atmos12070929>
- Hassoun, M., Royall, P. G., Parry, M., Harvey, R. D., & Forbes, B. (2018). Design and development of a biorelevant simulated human lung fluid. *Journal of Drug Delivery Science and Technology*, 47, 485–491. <https://doi.org/10.1016/j.jddst.2018.08.006>
- Hernández-Pellón, A., Nischkauer, W., Limbeck, A., & Fernández-Olmo, I. (2018). Metal(loid) bioaccessibility and inhalation risk assessment: A comparison between an urban and an industrial area. *Environmental Research*, 165, 140–149. <https://doi.org/10.1016/j.envres.2018.04.014>
- HKEPD. (2022). *PM 2.5 speciation study in Hong Kong* (Hong Kong Environmental Protection Department, Wan Chai, HongKong.). HKEPD. https://www.epd.gov.hk/epd/english/environmentinhk/air/study/rpts/pm25_study.html
- Ho, S. S. H., Yu, J. Z., Chow, J. C., Zielinska, B., Watson, J. G., Sit, E. H. L., & Schauer, J. J. (2008). Evaluation of an in-injection port thermal desorption-gas chromatography/mass spectrometry method for analysis of non-polar organic compounds in ambient aerosol samples. *Journal of Chromatography A*, 1200(2), 217–227. <https://doi.org/10.1016/j.chroma.2008.05.056>
- Hong Kong Marine Department. (2021). *Port of Hong Kong Statistical Tables 2021*. <https://www.mardep.gov.hk/>
- Hong, X., Yang, K., Liang, H., & Shi, Y. (2022). Characteristics of water-soluble inorganic ions in PM_{2.5} in typical urban areas of Beijing, China. *ACS Omega*, 7(40), 35575–35585. <https://doi.org/10.1021/acsomega.2c02919>
- Hu, W., Zhou, H., Chen, W., Ye, Y., Pan, T., Wang, Y., Song, W., Zhang, H., Deng, W., Zhu, M., Wang, C., Wu, C., Ye, C., Wang, Z., Yuan, B., Huang, S., Shao, M., Peng, Z., Day, Douglas. A., ... Wang, X. (2022). Oxidation Flow Reactor Results in a Chinese Megacity Emphasize the Important Contribution of S/IVOCs to Ambient SOA Formation. *Environmental Science & Technology*, 56(11), 6880–6893. <https://doi.org/10.1021/acs.est.1c03155>
- Hua, C., Ma, W., Zheng, F., Zhang, Y., Xie, J., Ma, L., Song, B., Yan, C., Li, H., Liu, Z., Liu, Q., Kulmala, M., & Liu, Y. (2024). Health risks and sources of trace elements and black carbon in PM_{2.5} from 2019 to 2021 in Beijing. *Journal of Environmental Sciences*, 142, 69–82. <https://doi.org/10.1016/j.jes.2023.05.023>

- Huang, X., Cheng, J., Bo, D., Betha, R., & Balasubramanian, R. (2016). Bioaccessibility of Airborne Particulate-Bound Trace Elements in Shanghai and Health Risk Assessment. *Frontiers in Environmental Science*, 4. <https://www.frontiersin.org/articles/10.3389/fenvs.2016.00076>
- Huy, D. H., Thanh, L. T., Hien, T. T., & Takenaka, N. (2020). Comparative study on water-soluble inorganic ions in PM_{2.5} from two distinct climate regions and air quality. *Journal of Environmental Sciences*, 88, 349–360. <https://doi.org/10.1016/j.jes.2019.09.010>
- Iakovides, M., Iakovides, G., & Stephanou, E. G. (2021). Atmospheric particle-bound polycyclic aromatic hydrocarbons, n-alkanes, hopanes, steranes and trace metals: PM_{2.5} source identification, individual and cumulative multi-pathway lifetime cancer risk assessment in the urban environment. *Science of The Total Environment*, 752, 141834. <https://doi.org/10.1016/j.scitotenv.2020.141834>
- Ihantola, T., Hirvonen, M.-R., Ihalainen, M., Hakkarainen, H., Sippula, O., Tissari, J., Bauer, S., Di Bucchianico, S., Rastak, N., Hartikainen, A., Leskinen, J., Yli-Pirilä, P., Martikainen, M.-V., Miettinen, M., Suhonen, H., Rönkkö, T. J., Kortelainen, M., Lamberg, H., Czech, H., ... Jalava, P. I. (2022). Genotoxic and inflammatory effects of spruce and brown coal briquettes combustion aerosols on lung cells at the air-liquid interface. *Science of The Total Environment*, 806, 150489. <https://doi.org/10.1016/j.scitotenv.2021.150489>
- Im, U., Christensen, J. H., Geels, C., Hansen, K. M., Brandt, J., Solazzo, E., Alyuz, U., Balzarini, A., Baro, R., Bellasio, R., Bianconi, R., Bieser, J., Colette, A., Curci, G., Farrow, A., Flemming, J., Fraser, A., Jimenez-Guerrero, P., Kitwiroon, N., ... Galmarini, S. (2018). Influence of anthropogenic emissions and boundary conditions on multi-model simulations of major air pollutants over Europe and North America in the framework of AQMEII3. *Atmospheric Chemistry and Physics*, 18(12), 8929–8952. <https://doi.org/10.5194/acp-18-8929-2018>
- Innes, E., Yiu, H. H. P., McLean, P., Brown, W., & Boyles, M. (2021). Simulated biological fluids – a systematic review of their biological relevance and use in relation to inhalation toxicology of particles and fibres. *Critical Reviews in Toxicology*, 51(3), 217–248. <https://doi.org/10.1080/10408444.2021.1903386>
- Jiang, J., Wu, Y., Sun, G., Zhang, L., Li, Z., Sommar, J., Yao, H., & Feng, X. (2021). Characteristics, accumulation, and potential health risks of antimony in atmospheric particulate matter. *ACS Omega*, 6(14), 9460–9470. <https://doi.org/10.1021/acsomega.0c06091>
- Jiang, S., Shang, M., Mu, K., Jiang, N., Wen, H., Wang, R., Wu, H., & Li, W. (2018). In vitro and in vivo toxic effects and inflammatory responses induced by carboxylated black carbon-lead complex exposure. *Ecotoxicology and Environmental Safety*, 165, 484–494. <https://doi.org/10.1016/j.ecoenv.2018.09.040>
- Jin, L., Luo, X., Fu, P., & Li, X. (2017). Airborne particulate matter pollution in urban China: A chemical mixture perspective from sources to impacts. *National Science Review*, 4(4), 593–610. <https://doi.org/10.1093/nsr/nww079>
- Jin, L., Xie, J., Wong, C. K. C., Chan, S. K. Y., Abbaszade, G., Schnelle-Kreis, J., Zimmermann, R., Li, J., Zhang, G., Fu, P., & Li, X. (2019). Contributions of City-Specific Fine Particulate Matter (PM_{2.5}) to Differential *In Vitro* Oxidative Stress and Toxicity Implications between Beijing and Guangzhou of China.

Environmental Science & Technology, 53(5), 2881–2891. <https://doi.org/10.1021/acs.est.9b00449>

Juan, C. A., Pérez de la Lastra, J. M., Plou, F. J., & Pérez-Lebeña, E. (2021). The Chemistry of Reactive Oxygen Species (ROS) Revisited: Outlining Their Role in Biological Macromolecules (DNA, Lipids and Proteins) and Induced Pathologies. *International Journal of Molecular Sciences*, 22(9), 4642. <https://doi.org/10.3390/ijms22094642>

Julien, C., Esperanza, P., Bruno, M., & Alleman, L. Y. (2011). Development of an in vitro method to estimate lung bioaccessibility of metals from atmospheric particles. *Journal of Environmental Monitoring*, 13(3), 621. <https://doi.org/10.1039/c0em00439a>

Kademoglou, K., Giovanoulis, G., Palm-Cousins, A., Padilla-Sanchez, J. A., Magnér, J., de Wit, C. A., & Collins, C. D. (2018). *In Vitro* Inhalation Bioaccessibility of Phthalate Esters and Alternative Plasticizers Present in Indoor Dust Using Artificial Lung Fluids. *Environmental Science & Technology Letters*, 5(6), 329–334. <https://doi.org/10.1021/acs.estlett.8b00113>

Kassomenos, P., Vardoulakis, S., Borge, R., Lumbreras, J., Papaloukas, C., & Karakitsios, S. (2010). Comparison of statistical clustering techniques for the classification of modelled atmospheric trajectories. *Theoretical and Applied Climatology*, 102(1), 1–12. <https://doi.org/10.1007/s00704-009-0233-7>

kaur, R., Garkal, A., Sarode, L., Bangar, P., Mehta, T., Singh, D. P., & Rawal, R. (2024). Understanding arsenic toxicity: Implications for environmental exposure and human health. *Journal of Hazardous Materials Letters*, 5, 100090. <https://doi.org/10.1016/j.hazl.2023.100090>

Kelly, F. J. (2003). Oxidative stress: Its role in air pollution and adverse health effects. *Occupational and Environmental Medicine*, 60(8), 612–616. <https://doi.org/10.1136/oem.60.8.612>

Kendall, M. (2007). Fine airborne urban particles (PM_{2.5}) sequester lung surfactant and amino acids from human lung lavage. *American Journal of Physiology-Lung Cellular and Molecular Physiology*, 293(4), L1053–L1058. <https://doi.org/10.1152/ajplung.00131.2007>

Kermani, M., Rahmatinia, T., Oskoei, V., Norzaee, S., Shahsavani, A., Farzadkia, M., & Kazemi, M. H. (2021). Potential cytotoxicity of trace elements and polycyclic aromatic hydrocarbons bounded to particulate matter: A review on in vitro studies on human lung epithelial cells. *Environmental Science and Pollution Research*, 28(40), 55888–55904. <https://doi.org/10.1007/s11356-021-16306-y>

Khairy, M. A., & Lohmann, R. (2013). Source apportionment and risk assessment of polycyclic aromatic hydrocarbons in the atmospheric environment of Alexandria, Egypt. *Chemosphere*, 91(7), 895–903. <https://doi.org/10.1016/j.chemosphere.2013.02.018>

Kim, K.-H., Jahan, S. A., Kabir, E., & Brown, R. J. C. (2013). A review of airborne polycyclic aromatic hydrocarbons (PAHs) and their human health effects. *Environment International*, 60, 71–80. <https://doi.org/10.1016/j.envint.2013.07.019>

Kouokam, J. C., Meaza, I., & Wise, J. P. (2022). Inflammatory effects of hexavalent chromium in the lung: A comprehensive review. *Toxicology and Applied Pharmacology*, 455, 116265. <https://doi.org/10.1016/j.taap.2022.116265>

- Kramer, A. L., Dorn, S., Perez, A., Roper, C., Titaley, I. A., Cayton, K., Cook, R. P., Cheong, P. H.-Y., & Massey Simonich, S. L. (2021). Assessing the oxidative potential of PAHs in ambient PM_{2.5} using the DTT consumption assay. *Environmental Pollution*, 285, 117411. <https://doi.org/10.1016/j.envpol.2021.117411>
- Kucera, O., Endlicher, R., Roušar, T., Lotková, H., Garnol, T., Drahota, Z., & ĽCervinková, Z. (2014). The effect of tert-butyl hydroperoxide-induced oxidative stress on lean and steatotic rat hepatocytes in vitro, *Oxid. Med. Cell. Longev.*, 2014, 752506.
- Leclercq, B., Alleman, L. Y., Perdrix, E., Riffault, V., Happillon, M., Strecker, A., Lo-Guidice, J.-M., Garçon, G., & Coddeville, P. (2017a). Particulate metal bioaccessibility in physiological fluids and cell culture media: Toxicological perspectives. *Environmental Research*, 156, 148–157. <https://doi.org/10.1016/j.envres.2017.03.029>
- Leni, Z., Cassagnes, L. E., Daellenbach, K. R., Haddad, I. E., Vlachou, A., Uzu, G., Prévôt, A. S. H., Jaffrezou, J.-L., Baumlin, N., Salathe, M., Baltensperger, U., Dommen, J., & Geiser, M. (2020). Oxidative stress-induced inflammation in susceptible airways by anthropogenic aerosol. *PLOS ONE*, 15(14).
- Li, H., Yin, N., Yang, R., & Faiola, F. (2024). Advancing environmental toxicology in vitro: From immortalized cancer cell lines to 3D models derived from stem cells. *Environment & Health*, 2(6), 332–349. <https://doi.org/10.1021/envhealth.3c00199>
- Li, Q., Shang, J., & Zhu, T. (2013). Physicochemical characteristics and toxic effects of ozone-oxidized black carbon particles. *Atmospheric Environment*, 81, 68–75. <https://doi.org/10.1016/j.atmosenv.2013.08.043>
- Li, R., Gao, Y., Chen, Y., Peng, M., Zhao, W., Wang, G., & Hao, J. (2023). Measurement report: Rapid changes of chemical characteristics and health risks for highly time resolved trace elements in PM_{2.5} in a typical industrial city in response to stringent clean air actions. *Atmospheric Chemistry and Physics*, 23(8), 4709–4726. <https://doi.org/10.5194/acp-23-4709-2023>
- Li, T., Li, J., Jiang, H., Chen, D., Zong, Z., Tian, C., & Zhang, G. (2020). Source Apportionment of PM_{2.5} in Guangzhou Based on an Approach of Combining Positive Matrix Factorization with the Bayesian Mixing Model and Radiocarbon. *Atmosphere*, 11(5), 512. <https://doi.org/10.3390/atmos11050512>
- Li, Y., Juhasz, A. L., Ma, L. Q., & Cui, X. (2019). Inhalation bioaccessibility of PAHs in PM_{2.5}: Implications for risk assessment and toxicity prediction. *Science of The Total Environment*, 650, 56–64. <https://doi.org/10.1016/j.scitotenv.2018.08.246>
- Liao, H.-T., Chang, J.-C., Tsai, T.-T., Tsai, S.-W., Chou, C. C.-K., & Wu, C.-F. (2020). Vertical distribution of source apportioned PM_{2.5} using particulate-bound elements and polycyclic aromatic hydrocarbons in an urban area. *Journal of Exposure Science & Environmental Epidemiology*, 30(4), 659–669. <https://doi.org/10.1038/s41370-019-0153-2>
- Liao, H.-T., Lee, C.-L., Tsai, W.-C., Yu, J. Z., Tsai, S.-W., Chou, C. C. K., & Wu, C.-F. (2021). Source apportionment of urban PM_{2.5} using positive matrix factorization with vertically distributed measurements of trace elements and nonpolar organic compounds. *Atmospheric Pollution Research*,

12(4), 200–207. <https://doi.org/10.1016/j.apr.2021.03.007>

Liao, K., & Yu, J. Z. (2020). Abundance and sources of benzo[a]pyrene and other PAHs in ambient air in Hong Kong: A review of 20-year measurements (1997–2016). *Chemosphere*, 259, 127518. <https://doi.org/10.1016/j.chemosphere.2020.127518>

Lin, M., & Yu, J. Z. (2019a). Dithiothreitol (DTT) concentration effect and its implications on the applicability of DTT assay to evaluate the oxidative potential of atmospheric aerosol samples. *Environmental Pollution*, 251, 938–944. <https://doi.org/10.1016/j.envpol.2019.05.074>

Lin, M., & Yu, J. Z. (2019b). Effect of metal-organic interactions on the oxidative potential of mixtures of atmospheric humic-like substances and copper/manganese as investigated by the dithiothreitol assay. *Science of The Total Environment*, 697, 134012. <https://doi.org/10.1016/j.scitotenv.2019.134012>

Lin, W., Dai, J., Liu, R., Zhai, Y., Yue, D., & Hu, Q. (2019). Integrated assessment of health risk and climate effects of black carbon in the Pearl River Delta region, China. *Environmental Research*, 176, 108522. <https://doi.org/10.1016/j.envres.2019.06.003>

Lindner, K., Ströbele, M., Schlick, S., Webering, S., Jenckel, A., Kopf, J., Danov, O., Sewald, K., Buj, C., Creutzenberg, O., Tillmann, T., Pohlmann, G., Ernst, H., Ziemann, C., Hüttmann, G., Heine, H., Bockhorn, H., Hansen, T., König, P., & Fehrenbach, H. (2017). Biological effects of carbon black nanoparticles are changed by surface coating with polycyclic aromatic hydrocarbons. *Particle and Fibre Toxicology*, 14(1), 8. <https://doi.org/10.1186/s12989-017-0189-1>

Liu, B., Zhang, Y., Talifu, D., Ding, X., Wang, X., Abulizi, A., Zhao, Q., Zhang, X., & Zhang, R. (2023). In Vitro Bioaccessibility and Health Risk of Heavy Metals from PM_{2.5}/PM₁₀ in Arid Areas—Hotan City, China. *Atmosphere*, 14(7), Article 7. <https://doi.org/10.3390/atmos14071066>

Liu, D., Lin, T., Syed, J. H., Cheng, Z., Xu, Y., Li, K., Zhang, G., & Li, J. (2017). Concentration, source identification, and exposure risk assessment of PM_{2.5}-bound parent PAHs and nitro-PAHs in atmosphere from typical Chinese cities. *Scientific Reports*, 7(1), 10398. <https://doi.org/10.1038/s41598-017-10623-4>

Liu, G., Yan, X., Wang, S., Yu, Q., Jia, J., & Yan, B. (2021). Elucidation of the Critical Role of Core Materials in PM_{2.5}-Induced Cytotoxicity by Interrogating Silica- and Carbon-Based Model PM_{2.5} Particle Libraries. *Environmental Science & Technology*, 55(9), 6128–6139. <https://doi.org/10.1021/acs.est.1c00001>

Liu, Y., He, Y., Han, B., Liu, H., Tao, S., & Liu, W. (2023). Sewage discharge and organic matter affect the partitioning behaviors of different polycyclic aromatic hydrocarbons in a river surface sediment-pore water system. *Journal of Hazardous Materials*, 446, 130757. <https://doi.org/10.1016/j.jhazmat.2023.130757>

Luo, K., Zeng, Y., Li, M., Man, Y., Zeng, L., Zhang, Q., Luo, J., & Kang, Y. (2021). Inhalation bioaccessibility and absorption of polycyclic aromatic hydrocarbons (PAHs) in indoor PM_{2.5} and its implication in risk assessment. *Science of The Total Environment*, 774, 145770. <https://doi.org/10.1016/j.scitotenv.2021.145770>

- Luo, X., Yu, S., & Li, X. (2011). Distribution, availability, and sources of trace metals in different particle size fractions of urban soils in Hong Kong: Implications for assessing the risk to human health. *Environmental Pollution*, 159(5), 1317–1326. <https://doi.org/10.1016/j.envpol.2011.01.013>
- Luo, X., Zhao, Z., Xie, J., Luo, J., Chen, Y., Li, H., & Jin, L. (2019). Pulmonary bioaccessibility of trace metals in PM_{2.5} from different megacities simulated by lung fluid extraction and DGT method. *Chemosphere*, 218, 915–921. <https://doi.org/10.1016/j.chemosphere.2018.11.079>
- Luo, Z., Zhang, L., Li, G., Du, W., Chen, Y., Cheng, H., Tao, S., & Shen, G. (2021). Evaluating co-emissions into indoor and outdoor air of EC, OC, and BC from in-home biomass burning. *Atmospheric Research*, 248, 105247. <https://doi.org/10.1016/j.atmosres.2020.105247>
- Lyu, R., Shi, Z., Alam, M. S., Wu, X., Liu, D., Vu, T. V., Stark, C., Fu, P., Feng, Y., & Harrison, R. M. (2019). Insight into the composition of organic compounds ($\geq$ C₆) in PM_{2.5} in wintertime in Beijing, China. *Atmospheric Chemistry and Physics*, 19(16), 10865–10881. <https://doi.org/10.5194/acp-19-10865-2019>
- Ma, Y., Cheng, Y., Qiu, X., Cao, G., Fang, Y., Wang, J., Zhu, T., Yu, J., & Hu, D. (2018). Sources and oxidative potential of water-soluble humic-like substances (HULIS_{WS}) in fine particulate matter (PM_{2.5}) in Beijing. *Atmospheric Chemistry and Physics*, 18(8), 5607–5617. <https://doi.org/10.5194/acp-18-5607-2018>
- Macacu, A. (2020). *Dose–response analysis of toxicological and pharmacological mixtures with the model deviation ratio method: problems and solutions*.
- Malm, W. C., Schichtel, B. A., Hand, J. L., & Collett Jr., J. L. (2017). Concurrent Temporal and Spatial Trends in Sulfate and Organic Mass Concentrations Measured in the IMPROVE Monitoring Program. *Journal of Geophysical Research: Atmospheres*, 122(19), 10,462–10,476. <https://doi.org/10.1002/2017JD026865>
- Marking, L. L., & Mauck, W. L. (1975). Toxicity of paired mixtures of candidate forest insecticides to rainbow trout. *Bulletin of Environmental Contamination and Toxicology*, 13(5), 518–523. <https://doi.org/10.1007/BF01685172>
- Meng, Q., Richmond-Bryant, J., Lu, S.-E., Buckley, B., Welsh, W. J., Whitsel, E. A., Hanna, A., Yeatts, K. B., Warren, J., Herring, A. H., & Xiu, A. (2013). Cardiovascular Outcomes and the Physical and Chemical Properties of Metal Ions Found in Particulate Matter Air Pollution: A QICAR Study. *Environmental Health Perspectives*, 121(5), 558–564. <https://doi.org/10.1289/ehp.1205793>
- Midander, K., de Frutos, A., Hedberg, Y., Darrie, G., & Wallinder, I. O. (2010). Bioaccessibility studies of ferro-chromium alloy particles for a simulated inhalation scenario: A comparative study with the pure metals and stainless steel. *Integrated Environmental Assessment and Management*, 6(3), 441–455. <https://doi.org/10.1002/ieam.32>
- Min, K., Hao, S., Lin, Y., Lu, D., Lv, Y., Zhang, W., Ji, N., Liu, Q., & Jiang, G. (2023). An Attempt to Detect Ambient Black Carbon in the Human Brain Using Mass Spectrometry Imaging. *Environmental Science & Technology Letters*, 10(12), 1165–1172. <https://doi.org/10.1021/acs.estlett.3c00664>

- Morimoto, Y., Horie, M., Kobayashi, N., Shinohara, N., & Shimada, M. (2013). Inhalation Toxicity Assessment of Carbon-Based Nanoparticles. *Accounts of Chemical Research*, 46(3), 770–781. <https://doi.org/10.1021/ar200311b>
- Murphy, M. P., Bayir, H., Belousov, V., Chang, C. J., Davies, K. J. A., Davies, M. J., Dick, T. P., Finkel, T., Forman, H. J., Janssen-Heininger, Y., Gems, D., Kagan, V. E., Kalyanaraman, B., Larsson, N.-G., Milne, G. L., Nyström, T., Poulsen, H. E., Radi, R., Van Remmen, H., ... Halliwell, B. (2022). Guidelines for measuring reactive oxygen species and oxidative damage in cells and in vivo. *Nature Metabolism*, 4(6), 651–662. <https://doi.org/10.1038/s42255-022-00591-z>
- Niranjan, R., & Thakur, A. K. (2017). The Toxicological Mechanisms of Environmental Soot (Black Carbon) and Carbon Black: Focus on Oxidative Stress and Inflammatory Pathways. *Frontiers in Immunology*, 8. <https://www.frontiersin.org/articles/10.3389/fimmu.2017.00763>
- Niu, X., Chuang, H.-C., Wang, X., Ho, S. S. H., Li, L., Qu, L., Chow, J. C., Watson, J. G., Sun, J., Lee, S., Cao, J., & Ho, K. F. (2020). Cytotoxicity of PM_{2.5} vehicular emissions in the Shing Mun Tunnel, Hong Kong. *Environmental Pollution*, 263, 114386. <https://doi.org/10.1016/j.envpol.2020.114386>
- Nurlatifah, A., Kombara, P. Y., Pratama, A., Faristyawan, R., Rakhman, A. A., & Noviausti, N. (2025). Utilisation of WRF-HYSPLIT modelling approach and GEMS to identify PM_{2.5} sources in central kalimantan – study case: 2023 forest fire. *Journal of Southern Hemisphere Earth Systems Science*, 75(1), NULL-NULL. <https://doi.org/10.1071/ES24006>
- OEHHA. (1998). *Diesel Exhaust Particulate*. Office of Environmental Health Hazard Assessment (OEHHA). <https://oehha.ca.gov/chemicals/diesel-exhaust-particulate>
- OEHHA, C. (2005). *Air Toxics Hot Spots Program Risk Assessment Guidelines. Part II. Technical Support Document for Describing Available Cancer Potency Factors*. Sacramento, CA: CalEPA. CalEPA. Office of Environmental Health Hazard Assessment, California
- Oomen, A. G., Rempelberg, C. J. M., Bruil, M. A., Dobbe, C. J. G., Pereboom, D. P. K. H., & Sips, A. J. A. M. (2003). Development of an in vitro digestion model for estimating the bioaccessibility of soil contaminants. *Archives of Environmental Contamination and Toxicology*, 44(3), 0281–0287. <https://doi.org/10.1007/s00244-002-1278-0>
- Øvrevik, J., Refsnes, M., Låg, M., Holme, J. A., & Schwarze, P. E. (2015). Activation of Proinflammatory Responses in Cells of the Airway Mucosa by Particulate Matter: Oxidant- and Non-Oxidant-Mediated Triggering Mechanisms. *Biomolecules*, 5(3), Article 3. <https://doi.org/10.3390/biom5031399>
- Palleschi, S., Rossi, B., Armiento, G., Montereali, M. R., Nardi, E., Mazziotti Tagliani, S., Inglessis, M., Gianfagna, A., & Silvestroni, L. (2018). Toxicity of the readily leachable fraction of urban PM_{2.5} to human lung epithelial cells: Role of soluble metals. *Chemosphere*, 196, 35–44. <https://doi.org/10.1016/j.chemosphere.2017.12.147>
- Pan, S., Qiu, Y., Li, M., Yang, Z., & Liang, D. (2022). Recent developments in the determination of PM_{2.5} chemical composition. *Bulletin of Environmental Contamination and Toxicology*, 108(5), 819–823. <https://doi.org/10.1007/s00128-022-03510-w>

- Pape-Lindstrom, P. A., & Lydy, M. J. (1997). Synergistic toxicity of atrazine and organophosphate insecticides contravenes the response addition mixture model. *Environmental Toxicology and Chemistry*, 16(11), 2415–2420. <https://doi.org/10.1002/etc.5620161130>
- Park, J.-M., Lee, T.-J., & Kim, D.-S. (2022). Improving PMF source reconciliation with cluster analysis for PM_{2.5} hourly data from Seoul, Korea. *Atmospheric Pollution Research*, 13(5), 101398. <https://doi.org/10.1016/j.apr.2022.101398>
- Pelfrène, A., Cave, M., Wragg, J., & Douay, F. (2017). In Vitro Investigations of Human Bioaccessibility from Reference Materials Using Simulated Lung Fluids. *International Journal of Environmental Research and Public Health*, 14(2), 112. <https://doi.org/10.3390/ijerph14020112>
- Pelfrène, A., Waterlot, C., Mazzuca, M., Nisse, C., Cuny, D., Richard, A., Denys, S., Heyman, C., Roussel, H., Bidar, G., & Douay, F. (2012). Bioaccessibility of trace elements as affected by soil parameters in smelter-contaminated agricultural soils: A statistical modeling approach. *Environmental Pollution*, 160, 130–138. <https://doi.org/10.1016/j.envpol.2011.09.008>
- Phairuang, W., Piriyaakarnsakul, S., Inerb, M., Hongtieab, S., Thongyen, T., Chomanee, J., Boongla, Y., Suriyawong, P., Samae, H., Chanonmuang, P., Suwattiga, P., Chetianukornkul, T., Panyametheekul, S., Amin, M., Hata, M., & Furuuchi, M. (2023). Ambient nanoparticles (PM_{0.1}) mapping in thailand. *Atmosphere*, 14(1), Article 1. <https://doi.org/10.3390/atmos14010066>
- Platel, A., Privat, K., Talahari, S., Delobel, A., Dourdin, G., Gateau, E., Simar, S., Saleh, Y., Sotty, J., Antherieu, S., Canivet, L., Alleman, L.-Y., Perdrix, E., Garçon, G., Denayer, F. O., Lo Guidice, J. M., & Nessler, F. (2020). Study of in vitro and in vivo genotoxic effects of air pollution fine (PM_{2.5-0.18}) and quasi-ultrafine (PM_{0.18}) particles on lung models. *Science of The Total Environment*, 711, 134666. <https://doi.org/10.1016/j.scitotenv.2019.134666>
- Pope, C. A., Bhatnagar, A., McCracken, J. P., Abplanalp, W., Conklin, D. J., & O'Toole, T. (2016). Exposure to fine particulate air pollution is associated with endothelial injury and systemic inflammation. *Circulation Research*, 119(11), 1204–1214. <https://doi.org/10.1161/CIRCRESAHA.116.309279>
- Pulles, T., Denier van der Gon, H., Appelman, W., & Verheul, M. (2012). Emission factors for heavy metals from diesel and petrol used in European vehicles. *Atmospheric Environment*, 61, 641–651. <https://doi.org/10.1016/j.atmosenv.2012.07.022>
- Qi, Z., Song, Y., Ding, Q., Liao, X., Li, R., Liu, G., Tsang, S., & Cai, Z. (2019). Water soluble and insoluble components of PM_{2.5} and their functional cardiotoxicities on neonatal rat cardiomyocytes in vitro. *Ecotoxicology and Environmental Safety*, 168, 378–387. <https://doi.org/10.1016/j.ecoenv.2018.10.107>
- R. R. Draxler & G. D. Hess. (1998). An overview of the HYSPLIT_4 modeling system for trajectories, dispersion, and deposition. *Aust. Meteor. Mag.*, 47, 295–308.
- Rahmatinia, T., Kermani, M., Farzadkia, M., Nicknam, M. H., Soleimanifar, N., Mohebbi, B., Jafari, A. J., Shahsavani, A., & Fanaei, F. (2021). Potential cytotoxicity of PM_{2.5}-bound PAHs and toxic metals collected from areas with different traffic densities on human lung epithelial cells (A549). *Journal of*

Environmental Health Science and Engineering, 19(2), 1701–1712. <https://doi.org/10.1007/s40201-021-00724-8>

Reddel, R., Ke, Y., Gerwin, B., McMenamin, M., Lechner, J., Su, R. T., Brash, D., Park, J.-B., Rhim, J., & Harris, C. (1988). Transformation of human bronchial epithelial cells by infection with SV40 or adenovirus-12 SV40 hybrid virus, or transfection via strontium phosphate coprecipitation with a plasmid containing SV40 early region genes. *Cancer Research*. <https://www.semanticscholar.org/paper/Transformation-of-human-bronchial-epithelial-cells-Reddel-Ke/10d2f83c0ad6dc15318947daf4da3f07a897973c>

Ren, Y., Luo, Q., Zhuo, S., Hu, Y., Shen, G., Cheng, H., & Tao, S. (2021). Bioaccessibility and public health risk of heavy Metal(loid)s in the airborne particulate matter of four cities in northern China. *Chemosphere*, 277, 130312. <https://doi.org/10.1016/j.chemosphere.2021.130312>

Riss, T. L., Moravec, R. A., Niles, A. L., Duellman, S., Benink, H. A., Worzella, T. J., & Minor, L. (2004). Cell viability assays. In S. Markossian, A. Grossman, H. Baskir, M. Arkin, D. Auld, C. Austin, J. Baell, K. Brimacombe, T. D. Y. Chung, N. P. Coussens, J. L. Dahlin, V. Devanarayan, T. L. Foley, M. Glicksman, K. Gorshkov, S. Grotegut, M. D. Hall, S. Hoare, J. Inglese, ... X. Xu (Eds.), *Assay Guidance Manual*. Eli Lilly & Company and the National Center for Advancing Translational Sciences. <http://www.ncbi.nlm.nih.gov/books/NBK144065/>

Roper, C., Delgado, L. S., Barrett, D., Massey Simonich, S. L., & Tanguay, R. L. (2019). PM_{2.5} Filter Extraction Methods: Implications for Chemical and Toxicological Analyses. *Environmental Science & Technology*, 53(1), 434–442. <https://doi.org/10.1021/acs.est.8b04308>

Sang, S., Chu, C., Zhang, T., Chen, H., & Yang, X. (2022). The global burden of disease attributable to ambient fine particulate matter in 204 countries and territories, 1990–2019: A systematic analysis of the Global Burden of Disease Study 2019. *Ecotoxicology and Environmental Safety*, 238, 113588. <https://doi.org/10.1016/j.ecoenv.2022.113588>

Sangkham, S., Phairuang, W., Sherchan, S. P., Pansakun, N., Munkong, N., Sarndhong, K., Islam, Md. A., & Sakunkoo, P. (2024). An update on adverse health effects from exposure to PM_{2.5}. *Environmental Advances*, 18, 100603. <https://doi.org/10.1016/j.envadv.2024.100603>

Schauer, J. J., Mader, B. T., DeMinter, J. T., Heidemann, G., Bae, M. S., Seinfeld, J. H., Flagan, R. C., Cary, R. A., Smith, D., Huebert, B. J., Bertram, T., Howell, S., Kline, J. T., Quinn, P., Bates, T., Turpin, B., Lim, H. J., Yu, J. Z., Yang, H., & Keywood, M. D. (2003). ACE-Asia Intercomparison of a Thermal-Optical Method for the Determination of Particle-Phase Organic and Elemental Carbon. *Environmental Science & Technology*, 37(5), 993–1001. <https://doi.org/10.1021/es020622f>

Schieber, M., & Chandel, N. S. (2014). ROS Function in Redox Signaling and Oxidative Stress. *Current Biology : CB*, 24(10), R453–R462. <https://doi.org/10.1016/j.cub.2014.03.034>

Serafeim, E., Basis, A., Kouras, A., Farias, C. N., Yera, A. B., Pereira, G. M., Samara, C., & de Castro Vasconcellos, P. (2023). Oxidative potential of ambient PM_{2.5} from são paulo, brazil: Variations, associations with chemical components and source apportionment. *Atmospheric Environment*, 298, 119593. <https://doi.org/10.1016/j.atmosenv.2023.119593>

- Shang, Y., Xue, W., Kong, J., Chen, Y., Qiu, X., An, X., Li, Y., Wang, H., & An, J. (2022). Ultrafine black carbon caused mitochondrial oxidative stress, mitochondrial dysfunction and mitophagy in SH-SY5Y cells. *Science of The Total Environment*, 813, 151899. <https://doi.org/10.1016/j.scitotenv.2021.151899>
- Shivani, Gadi, R., Sharma, S. K., & Mandal, T. K. (2019). Seasonal variation, source apportionment and source attributed health risk of fine carbonaceous aerosols over National Capital Region, India. *Chemosphere*, 237, 124500. <https://doi.org/10.1016/j.chemosphere.2019.124500>
- Song, X., Wu, D., Chen, X., Ma, Z., Li, Q., & Chen, J. (2024). Toxic Potencies of Particulate Matter from Typical Industrial Plants Mediated with Acidity via Metal Dissolution. *Environmental Science & Technology*. <https://doi.org/10.1021/acs.est.4c00929>
- Soukup, J. M., & Becker, S. (2001). Human Alveolar Macrophage Responses to Air Pollution Particulates Are Associated with Insoluble Components of Coarse Material, Including Particulate Endotoxin. *Toxicology and Applied Pharmacology*, 171(1), 20–26. <https://doi.org/10.1006/taap.2000.9096>
- Stein, A. F., Draxler, R. R., Rolph, G. D., Stunder, B. J. B., Cohen, M. D., & Ngan, F. (2015). NOAA's HYSPLIT Atmospheric Transport and Dispersion Modeling System. *Bulletin of the American Meteorological Society*, 96(12), 2059–2077. <https://doi.org/10.1175/BAMS-D-14-00110.1>
- Sun, J., Shen, Z., Zhang, T., Kong, S., Zhang, H., Zhang, Q., Niu, X., Huang, S., Xu, H., Ho, K.-F., & Cao, J. (2022). A comprehensive evaluation of PM_{2.5}-bound PAHs and their derivative in winter from six megacities in China: Insight the source-dependent health risk and secondary reactions. *Environment International*, 165, 107344. <https://doi.org/10.1016/j.envint.2022.107344>
- Sun, P., Nie, W., Chi, X., Huang, X., Ren, C., Xue, L., Shan, Y., Wen, L., Li, H., Chen, T., Qi, Y., Gao, J., Zhang, Q., & Ding, A. (2022). Aircraft Study of Secondary Aerosols in Long-Range Transported Air Masses from the North China Plain by a Mid-Latitude Cyclone. *Journal of Geophysical Research: Atmospheres*, 127(4), e2021JD036178. <https://doi.org/10.1029/2021JD036178>
- Sun, S., Zheng, N., Wang, S., Li, Y., Hou, S., An, Q., Chen, C., Li, X., Ji, Y., & Li, P. (2022). Inhalation Bioaccessibility and Risk Assessment of Metals in PM_{2.5} Based on a Multiple-Path Particle Dosimetry Model in the Smelting District of Northeast China. *International Journal of Environmental Research and Public Health*, 19(15), 8915. <https://doi.org/10.3390/ijerph19158915>
- Tao, J., Zhang, L., Cao, J., Zhong, L., Chen, D., Yang, Y., Chen, D., Chen, L., Zhang, Z., Wu, Y., Xia, Y., Ye, S., & Zhang, R. (2017). Source apportionment of PM_{2.5} at urban and suburban areas of the Pearl River Delta region, south China—With emphasis on ship emissions. *Science of The Total Environment*, 574, 1559–1570. <https://doi.org/10.1016/j.scitotenv.2016.08.175>
- Tchounwou, P. B., Yedjou, C. G., Patlolla, A. K., & Sutton, D. J. (2012). Heavy metal toxicity and the environment. *Experientia Supplementum* (2012), 101, 133–164. https://doi.org/10.1007/978-3-7643-8340-4_6
- Thangavel, P., Park, D., & Lee, Y.-C. (2022). Recent Insights into Particulate Matter (PM_{2.5})-Mediated Toxicity in Humans: An Overview. *International Journal of Environmental Research and Public Health*, 19(12), 7511. <https://doi.org/10.3390/ijerph19127511>

The World Health Organization European Centre for Environment and Health. (2012). *Health Effects of Black Carbon*. The World Health Organization European Centre for Environment and Health. <https://www.unep.org/resources/report/health-effects-black-carbon>

Tsiotra, I., Grivas, G., Tavernarakis, K., Bougiatioti, A., Apostolaki, M., Paraskevopoulou, D., Gogou, A., Parinos, C., Oikonomou, K., Tsagkarakis, M., Zarmas, P., Nenes, A., & Mihalopoulos, N. (2021). Annual exposure to polycyclic aromatic hydrocarbons in urban environments linked to wintertime wood-burning episodes. *Atmospheric Chemistry and Physics*, 21(23), 17865–17883. <https://doi.org/10.5194/acp-21-17865-2021>

Turpin, B. J., & Huntzicker, J. J. (1995). Identification of secondary organic aerosol episodes and quantitation of primary and secondary organic aerosol concentrations during SCAQS. *Atmospheric Environment*, 29(23), 3527–3544. [https://doi.org/10.1016/1352-2310\(94\)00276-Q](https://doi.org/10.1016/1352-2310(94)00276-Q)

USEPA. (1994). *Health effects notebook for hazardous air pollutants*, US EPA, Office of Air Quality Planning and Standards.

USEPA. (2005). *Guidelines for carcinogen risk assessment (EPA/630/P-03/001F)*. Risk Assessment Forum. <https://www.epa.gov/risk/guidelines-carcinogen-risk-assessment>

USEPA. (2007). *Framework for Metals Risk Assessment*. <https://www.epa.gov/risk/framework-metals-risk-assessment>

USEPA. (2009). *Risk Assessment Guidance for Superfund. Volume I. Human Health Evaluation Manual. Part F(Final)*. <https://www.epa.gov/risk/risk-assessment-guidance-superfund-rags-part-f>.

USEPA. (2015). *EPA Positive Matrix Factorization (PMF) 5.0 Fundamentals and User Guide*. <https://www.epa.gov/air-research/positive-matrix-factorization-model-environmental-data-analyses>

USEPA. (2024). *Risk assessment for other effects*. <https://www.epa.gov/fera/risk-assessment-other-effects>

Veerappan, I., Sankareswaran, S. K., & Palanisamy, R. (2019). Morin Protects Human Respiratory Cells from PM_{2.5} Induced Genotoxicity by Mitigating ROS and Reverting Altered miRNA Expression. *International Journal of Environmental Research and Public Health*, 16(13), Article 13. <https://doi.org/10.3390/ijerph16132389>

Wang, B., Li, Y., Tang, Z., & Cai, N. (2021). The carbon components in indoor and outdoor PM_{2.5} in winter of Tianjin. *Scientific Reports*, 11(1), Article 1. <https://doi.org/10.1038/s41598-021-97530-x>

Wang, D., Pakbin, P., Shafer, M. M., Antkiewicz, D., Schauer, J. J., & Sioutas, C. (2013). Macrophage reactive oxygen species activity of water-soluble and water-insoluble fractions of ambient coarse, PM_{2.5} and ultrafine particulate matter (PM) in los angeles. *Atmospheric Environment*, 77, 301–310. <https://doi.org/10.1016/j.atmosenv.2013.05.031>

Wang, L., Bao, S., Liu, X., Wang, F., Zhang, J., Dang, P., Wang, F., Li, B., & Lin, Y. (2021). Low-dose exposure to black carbon significantly increase lung injury of cadmium by promoting cellular apoptosis. *Ecotoxicology and Environmental Safety*, 224, 112703. <https://doi.org/10.1016/j.ecoenv.2021.112703>

- Wang, Q., Huang, X. H. H., Tam, F. C. V., Zhang, X., Liu, K. M., Yeung, C., Feng, Y., Cheng, Y. Y., Wong, Y. K., Ng, W. M., Wu, C., Zhang, Q., Zhang, T., Lau, N. T., Yuan, Z., Lau, A. K. H., & Yu, J. Z. (2019). Source apportionment of fine particulate matter in Macao, China with and without organic tracers: A comparative study using positive matrix factorization. *Atmospheric Environment*, 198, 183–193. <https://doi.org/10.1016/j.atmosenv.2018.10.057>
- Wang, Q., Wang, S., Cheng, Y. Y., Chen, H., Zhang, Z., Li, J., Gu, D., Wang, Z., & Yu, J. Z. (2022). *Chemical evolution of secondary organic aerosol tracers during high PM_{2.5} episodes at a suburban site in Hong Kong over 4 months of continuous measurement* [Preprint]. Aerosols/Field Measurements/Troposphere/Chemistry (chemical composition and reactions). <https://doi.org/10.5194/acp-2022-302>
- Wang, S., Hu, G., Yu, R., Shen, H., & Yan, Y. (2021). Bioaccessibility and source-specific health risk of heavy metals in PM_{2.5} in a coastal city in China. *Environmental Advances*, 4, 100047. <https://doi.org/10.1016/j.envadv.2021.100047>
- Wang, S., Wang, Q., Zhu, S., Zhou, M., Qiao, L., Huang, D., Ma, Y., Lu, Y., Huang, C., Fu, Q., Duan, Y., & Yu, J. Z. (2022). Hourly organic tracers-based source apportionment of PM_{2.5} before and during the Covid-19 lockdown in suburban Shanghai, China: Insights into regional transport influences and response to urban emission reductions. *Atmospheric Environment*, 289, 119308. <https://doi.org/10.1016/j.atmosenv.2022.119308>
- Wang, S., Zhu, Y., Jang, J.-C., Jiang, M., Yue, D., Zhong, L., Yuan, Y., Zhang, M., & You, Z. (2024). Modeling assessment of air pollution control measures and COVID-19 pandemic on air quality improvements over greater bay area of China. *Science of The Total Environment*, 926, 171951. <https://doi.org/10.1016/j.scitotenv.2024.171951>
- Wang, T., Zhao, J., Liu, Y., Peng, J., Wu, L., & Mao, H. (2022). PM_{2.5}-Bound Polycyclic Aromatic Hydrocarbons (PAHs), Nitrated PAHs (NPAHs) and Oxygenated PAHs (OPAHs) in Typical Traffic-Related Receptor Environments. *Journal of Geophysical Research: Atmospheres*, 127(5), e2021JD035951. <https://doi.org/10.1029/2021JD035951>
- Warne, M. S. J., & Hawker, D. W. (1995). The Number of Components in a Mixture Determines Whether Synergistic and Antagonistic or Additive Toxicity Predominate: The Funnel Hypothesis. *Ecotoxicology and Environmental Safety*, 31(1), 23–28. <https://doi.org/10.1006/eesa.1995.1039>
- Wei, J., Wang, J., Li, Z., Kondragunta, S., Anenberg, S., Wang, Y., Zhang, H., Diner, D., Hand, J., Lyapustin, A., Kahn, R., Colarco, P., Silva, A. da, & Ichoku, C. (2023). Long-term mortality burden trends attributed to black carbon and PM_{2.5} from wildfire emissions across the continental USA from 2000 to 2020: A deep learning modelling study. *The Lancet Planetary Health*, 7(12), e963–e975. [https://doi.org/10.1016/S2542-5196\(23\)00235-8](https://doi.org/10.1016/S2542-5196(23)00235-8)
- Wen, L., Yang, C., Liao, X., Zhang, Y., Chai, X., Gao, W., Guo, S., Bi, Y., Tsang, S.-Y., Chen, Z.-F., Qi, Z., & Cai, Z. (2022). Investigation of PM_{2.5} pollution during COVID-19 pandemic in Guangzhou, China. *Journal of Environmental Sciences*, 115, 443–452. <https://doi.org/10.1016/j.jes.2021.07.009>
- Wong, Y. K., Liu, K. M., Yeung, C., Leung, K. K. M., & Yu, J. Z. (2022). Measurement report:

Characterization and source apportionment of coarse particulate matter in Hong Kong: insights into the constituents of unidentified mass and source origins in a coastal city in southern China. *Atmospheric Chemistry and Physics*, 22(7), 5017–5031. <https://doi.org/10.5194/acp-22-5017-2022>

World Health Organization. (2021). *WHO global air quality guidelines: Particulate matter (PM_{2.5} and PM₁₀), ozone, nitrogen dioxide, sulfur dioxide and carbon monoxide*. World Health Organization. <https://apps.who.int/iris/handle/10665/345329>

Wu, D., Li, Q., Ding, X., Sun, J., Li, D., Fu, H., Teich, M., Ye, X., & Chen, J. (2018). Primary Particulate Matter Emitted from Heavy Fuel and Diesel Oil Combustion in a Typical Container Ship: Characteristics and Toxicity. *Environmental Science & Technology*, 52(21), 12943–12951. <https://doi.org/10.1021/acs.est.8b04471>

Wu, D., Zheng, H., Li, Q., Jin, L., Lyu, R., Ding, X., Huo, Y., Zhao, B., Jiang, J., Chen, J., Li, X., & Wang, S. (2022). Toxic potency-adjusted control of air pollution for solid fuel combustion. *Nature Energy*, 7(2), 194–202. <https://doi.org/10.1038/s41560-021-00951-1>

Wu, Z., Liu, D., Zhao, T., Su, Y., & Zhou, B. (2021). Size distributions of water-soluble inorganic ions in atmospheric aerosols during the meiyu period in the yangtze river delta, China. *Frontiers in Environmental Science*, 9. <https://doi.org/10.3389/fenvs.2021.788115>

Xie, J., Jin, L., Cui, J., Luo, X., Li, J., Zhang, G., & Li, X. (2020). Health risk-oriented source apportionment of PM_{2.5}-associated trace metals. *Environmental Pollution*, 262, 114655. <https://doi.org/10.1016/j.envpol.2020.114655>

Xie, J.-J., Yuan, C.-G., Shen, Y.-W., Xie, J., He, K.-Q., Zhu, H.-T., & Zhang, K.-G. (2019). Bioavailability/speciation of arsenic in atmospheric PM_{2.5} and their seasonal variation: A case study in Baoding city, China. *Ecotoxicology and Environmental Safety*, 169, 487–495. <https://doi.org/10.1016/j.ecoenv.2018.11.026>

Xie, S.-Y., Lao, J.-Y., Wu, C.-C., Bao, L.-J., & Zeng, E. Y. (2018). In vitro inhalation bioaccessibility for particle-bound hydrophobic organic chemicals: Method development, effects of particle size and hydrophobicity, and risk assessment. *Environment International*, 120, 295–303. <https://doi.org/10.1016/j.envint.2018.08.015>

Xing, C., Wang, Y., Yang, X., Zeng, Y., Zhai, J., Cai, B., Zhang, A., Fu, T.-M., Zhu, L., Li, Y., Wang, X., & Zhang, Y. (2023). Seasonal variation of driving factors of ambient PM_{2.5} oxidative potential in Shenzhen, China. *Science of The Total Environment*, 862, 160771. <https://doi.org/10.1016/j.scitotenv.2022.160771>

Xing, Y.-F., Xu, Y.-H., Shi, M.-H., & Lian, Y.-X. (2016). The impact of PM_{2.5} on the human respiratory system. *Journal of Thoracic Disease*, 8(1), E69–E74. <https://doi.org/10.3978/j.issn.2072-1439.2016.01.19>

Xue, W., & Warshawsky, D. (2005). Metabolic activation of polycyclic and heterocyclic aromatic hydrocarbons and DNA damage: A review. *Toxicology and Applied Pharmacology*, 206(1), 73–93. <https://doi.org/10.1016/j.taap.2004.11.006>

- Yan, R.-H., Peng, X., Lin, W., He, L.-Y., Wei, F.-H., Tang, M.-X., & Huang, X.-F. (2022). Trends and Challenges Regarding the Source-Specific Health Risk of PM_{2.5}-Bound Metals in a Chinese Megacity from 2014 to 2020. *Environmental Science & Technology*, 56(11), 6996–7005. <https://doi.org/10.1021/acs.est.1c06948>
- Yang, L., Li, C., & Tang, X. (2020). The impact of PM_{2.5} on the host defense of respiratory system. *Frontiers in Cell and Developmental Biology*, 8. <https://doi.org/10.3389/fcell.2020.00091>
- Yang, X., Wang, Q., Han, F., Dong, B., Wen, B., Li, L., Ruan, H., Zhang, S., Kong, J., Zhi, H., Wang, C., Wang, J., Zhang, M., & Xu, D. (2022). Pulmonary Benefits of Intervention with Air Cleaner among Schoolchildren in Beijing: A Randomized Double-Blind Crossover Study. *Environmental Science & Technology*, 56(11), 7185–7193. <https://doi.org/10.1021/acs.est.1c03146>
- Yerramilli, A., Dodla, V. B. R., Challa, V. S., Myles, L., Pendergrass, W. R., Vogel, C. A., Dasari, H. P., Tuluri, F., Baham, J. M., Hughes, R. L., Patrick, C., Young, J. H., Swanier, S. J., & Hardy, M. G. (2012). An integrated WRF/HYSPLIT modeling approach for the assessment of PM_{2.5} source regions over the mississippi gulf coast region. *Air Quality, Atmosphere, & Health*, 5(4), 401–412. <https://doi.org/10.1007/s11869-010-0132-1>
- Yu, H., Wei, J., Cheng, Y., Subedi, K., & Verma, V. (2018). Synergistic and Antagonistic Interactions among the Particulate Matter Components in Generating Reactive Oxygen Species Based on the Dithiothreitol Assay. *Environmental Science & Technology*, 52(4), 2261–2270. <https://doi.org/10.1021/acs.est.7b04261>
- Yu, J. Z., Huang, X. H. H., Ho, S. S. H., & Bian, Q. (2011). Nonpolar organic compounds in fine particles: Quantification by thermal desorption–GC/MS and evidence for their significant oxidation in ambient aerosols in Hong Kong. *Analytical and Bioanalytical Chemistry*, 401(10), 3125–3139. <https://doi.org/10.1007/s00216-011-5458-5>
- Yu, W., Tu, Y., Long, Z., Liu, J., Kong, D., Peng, J., Wu, H., Zheng, G., Zhao, J., Chen, Y., Liu, R., Li, W., & Hai, C. (2022). Reactive Oxygen Species Bridge the Gap between Chronic Inflammation and Tumor Development. *Oxidative Medicine and Cellular Longevity*, 2022(1), 2606928. <https://doi.org/10.1155/2022/2606928>
- Zhai, J., Yu, G., Zhang, J., Shi, S., Yuan, Y., Jiang, S., Xing, C., Cai, B., Zeng, Y., Wang, Y., Zhang, A., Zhang, Y., Fu, T.-M., Zhu, L., Shen, H., Ye, J., Wang, C., Tao, S., Li, M., ... Yang, X. (2023). Impact of Ship Emissions on Air Quality in the Greater Bay Area in China under the Latest Global Marine Fuel Regulation. *Environmental Science & Technology*, acs.est.3c03950. <https://doi.org/10.1021/acs.est.3c03950>
- Zhang, J., Peng, J., Song, C., Ma, C., Men, Z., Wu, J., Wu, L., Wang, T., Zhang, X., Tao, S., Gao, S., Hopke, P. K., & Mao, H. (2020). Vehicular non-exhaust particulate emissions in Chinese megacities: Source profiles, real-world emission factors, and inventories. *Environmental Pollution*, 266, 115268. <https://doi.org/10.1016/j.envpol.2020.115268>
- Zhang, L., Luo, Z., Li, Y., Chen, Y., Du, W., Li, G., Cheng, H., Shen, G., & Tao, S. (2021). Optically Measured Black and Particulate Brown Carbon Emission Factors from Real-World Residential

Combustion Predominantly Affected by Fuel Differences. *Environmental Science & Technology*, 55(1), 169–178. <https://doi.org/10.1021/acs.est.0c04784>

Zhang, X., Yan, Y., Duan, X., Chai, J., Li, R., Xu, Y., Li, Z., & Peng, L. (2022). Sources and Seasonal Variance of Ambient Volatile Organic Compounds in the Typical Industrial City of Changzhi, Northern China. *Atmosphere*, 13(3), Article 3. <https://doi.org/10.3390/atmos13030393>

Zhang, Y., Yao, Z., Shen, X., Liu, H., & He, K. (2015). Chemical characterization of PM_{2.5} emitted from on-road heavy-duty diesel trucks in China. *Atmospheric Environment*, 122, 885–891. <https://doi.org/10.1016/j.atmosenv.2015.07.014>

Zhao, Q., Li, Y., Chai, X., Zhang, L., Xu, L., Huang, J., Ning, P., & Tian, S. (2019). Interaction of nano carbon particles and anthracene with pulmonary surfactant: The potential hazards of inhaled nanoparticles. *Chemosphere*, 215, 746–752. <https://doi.org/10.1016/j.chemosphere.2018.10.016>

Zhao, W., Feng, H., Sun, W., Liu, K., Lu, J.-J., & Chen, X. (2017). Tert-butyl hydroperoxide (t-BHP) induced apoptosis and necroptosis in endothelial cells: Roles of NOX4 and mitochondrion. *Redox Biology*, 11, 524–534. <https://doi.org/10.1016/j.redox.2016.12.036>

Zhou, P., Kong, Y., & Cui, X. (2022). Inhalation Bioaccessibility of Polycyclic Aromatic Hydrocarbons in PM_{2.5} under Various Lung Environments: Implications for Air Pollution Control during Coronavirus Disease-19 Outbreak. *Environmental Science & Technology*, 56(7), 4272–4281. <https://doi.org/10.1021/acs.est.1c08052>

Zhou, Y., Li, C., Chen, Y., Yu, Y., Diao, X., Chiu, R., Fang, J., Shen, Y., Wang, J., Zhu, L., Zhou, J., & Cai, Z. (2024). Human airway organoids and multimodal imaging-based toxicity evaluation of 1-nitropyrene. *Environmental Science & Technology*, 58(14), 6083–6092. <https://doi.org/10.1021/acs.est.3c07195>

Zhu, S., Wang, Q., Qiao, L., Zhou, M., Wang, S., Lou, S., Huang, D., Wang, Q., Jing, S., Wang, H., Chen, C., Huang, C., & Yu, J. Z. (2021). Tracer-based characterization of source variations of PM_{2.5} and organic carbon in Shanghai influenced by the COVID-19 lockdown. *Faraday Discussions*, 226, 112–137. <https://doi.org/10.1039/D0FD00091D>

Zou, Y., Jin, C., Su, Y., Li, J., & Zhu, B. (2016). Water soluble and insoluble components of urban PM_{2.5} and their cytotoxic effects on epithelial cells (A549) *in vitro*. *Environmental Pollution*, 212, 627–635. <https://doi.org/10.1016/j.envpol.2016.03.022>