ESTIMATION OF CONTRIBUTIONS FROM GEOGRAPHICAL SOURCE REGIONS TO MORTALITY ASSOCIATED WITH $PM_{2.5}$ IN INDIAN CITIES

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DOCTOR OF PHILOSOPHY

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CERTIFICATE

This is to certify that **Shovan Kumar Sahu** has been working under my supervision since January 2015 as a regular registered Ph.D. student. His thesis entitled **"Estimation of contributions from geographical source regions to mortality associated with $PM_{2.5}$ in Indian cities"** is an authentic record of the results obtained from the research work carried out under my supervision in Civil Engineering Department, Indian Institute of Technology Guwahati India. I certify that he has fulfilled all the requirements according to the rules of this institute regarding the investigations embodied in his thesis and this work has not been submitted elsewhere for a degree.

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DECLARATION

I declare that the work embodied in this thesis is the result of investigations carried out by me in the Department of Civil Engineering, Indian Institute of Technology Guwahati India. The sources referred in the creation of this work have been appropriately acknowledged by explicit references or footnotes. Other assistance received has been acknowledged. I have not knowingly copied or used the words or ideas of others without acknowledgement.

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Dedicated

to

my dear

Parents

&

my mentor

Dr. Sri Harsha Kota

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by

Shovan Kumar Sahu

ABSTRACT

The size of the particle can be directly linked to it's potential to cause health risk. Particles having small size can stay for longer in air and can easily bypass nose and throat and penetrate deep into respiratory organs thus resulting in diseases. Particles having size less than 10 μm have the most detrimental effect on health. Particles between sizes 5-10 μm can get deposited in the tracheobronchial tree while those with size between 1-5 μm are deposited in the respiratory bronchioles and the alveoli which can even penetrate into lungs. Particles of size less than 1 μm can enter cell tissue or circulatory system thus severely affecting human.

Failure of Odd-Even scheme in Delhi indicated that the sources resulting in high $PM_{2.5}$ pollution in Indian cities is yet to be identified. Emissions and meteorology are two important factors, which effect pollutant concentrations. Emissions directly input pollutants into the atmosphere while meteorology affects their transport, transformation and deposition. Pollutant emissions affecting a city may be emitted from local sources or may have been transported from other states or cities. Identification of region from where high $PM_{2.5}$ concentrations are originating is thus critical to control air pollution. In this study, air mass trajectories were tracked using HYSPLIT model to identify geographical source regions affecting the Indian cities. The proportional residence time of the trajectories in a region was used to estimate contributions of different regions to $PM_{2.5}$ in eight Indian cities Delhi, Lucknow, Patna, Kolkata, Mumbai, Hyderabad, Bangalore and Chennai. Integrated Exposure Response (IER) function was used to link the $PM_{2.5}$ contributions from source regions to health risk and mortality, thus identifying the geographical source region contribution to mortality in the eight Indian cities.

The knowledge of source regions contributing to high $PM_{2.5}$ concentration and associated mortality in Indian cities is critical for regulating agencies in enforcing control policies. Source region contributions can be estimated using HYSPLIT back trajectory analysis while the estimation of mortality associated with $PM_{2.5}$ contributed by geographical source region to cities can only be done using risk functions since epidemiological studies of $PM_{2.5}$ are still scarce in India. Risk function is an effective tool to estimate risk due to pollutant concentration when mortality/hospitalization data is unavailable. Risk estimates provided by IER risk function at high $PM_{2.5}$ concentration are based on assumption that interpolation of risk estimates of (Ambient Air Pollution) AAP, SHS and AS will provide plausible risk estimates at high $PM_{2.5}$ concentration, which is yet to be validated. Due to unavailability of any $PM_{2.5}$ based health study in India, a time series epidemiological study was carried out in Delhi. The study evaluated the potential health risk associated with $PM_{2.5}$ to be 0.57 (95% CI: 0.45, 0.69). The study also found that reduction of $PM_{2.5}$ concentration from current levels to World Health Organization (WHO) and National Ambient Air Quality Standards (NAAQS) of 10 µg/m^3 and 40 µg/m^3 respectively resulted in 51 and 39 fewer deaths per 100000 population respectively. The IER risk estimates were compared to that evaluated in the Delhi and was found to only slightly overestimate mortality by 2.14% per 100000. IER risk function was used to estimate the health risks in 7 other Indian cities, since the mortality data was not available in these cities. Health risk in the eight cities in different seasons indicated higher Chronic Obstructive Pulmonary Disease (COPD), Lung Cancer (LC), Ischemic Heart Disease (IHD) and Stroke risks in the Northern (COPD=1.35, LC=1.50, IHD=1.39, Stroke=2.06) and Eastern cities (COPD=1.27, LC=1.38, IHD=1.35, Stroke=1.93) as compared to in Southern (COPD=1.17, LC=1.22, IHD=1.28, Stroke=1.54) or Western cities(COPD=1.18, LC=1.24,

IHD=1.28, Stroke=1.59). HYSPLIT $PM_{2.5}$ contribution from geographical source regions were clubbed with IER risk function to estimate the source region contribution to $PM_{2.5}$ associated mortality. Punjab, Haryana, Uttar Pradesh and Pakistan contributed 75% of total premature mortality in Delhi. Indian Ocean was a major contributor to premature mortality due to $PM_{2.5}$ concentration in western and southern cities of Mumbai, Hyderabad, Bangalore and Chennai accounting for 18.5%, 32% 54% and 99% of total premature mortality during winter. Reduction of emission from both local and regional sources were found to yield significant reduction in pollution of all cities except Delhi and Lucknow where regional and local control strategies respectively are needed.

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LIST OF SYMBOLS

Symbol	Description
RR	Relative Risk
c_{cf}	Counterfactual concentration (μg/m^3)
c	Average $PM_{2.5}$ concentrations (μg/m^3)
I_{HI}	Higher AQI value
I_{LO}	Lower AQI value
BR_{HI}	Higher Breakpoint Concentration
BR_{LO}	Lower Breakpoint Concentration
ER	Excessive Risk
β	Exposure-response relationship coefficient
μ	Mortality
$f(t)$	Smooth function of time
M	Mortality rate (per 1000)
Pop	Total Population
$\Delta Mort$	Excess premature mortality
y_0	Baseline mortality rate (per 100000)
$X(t)$	Initial position of trajectory
$X_1(t)$	First guess position of trajectory
U	Velocity vector
C_{cs}	$PM_{2.5}$ concentration contribution to city c from state s (μg/m^3)
C_s	$PM_{2.5}$ concentration associated with trajectories passing through state s
T_s	Number of trajectory endpoints passing through state s
T	Total number of back trajectory endpoints

LIST OF ABBREVIATIONS

Syntax	Description
WHO	World Health Organization
CPCB	Central Pollution Control Board
NAAQS	National Ambient Air Quality Standards
AQI	Air Quality Index
SPM	Suspended Particulate Matter
CMB	Chemical Mass Balance
PCA	Principal Component Analysis
PMF	Positive Matrix Factorization Model
HYSPLIT	Hybrid Single Particle Lagrangian Integrated Trajectory Model
IER	Integrated Exposure Response function
AAP	Ambient Air Pollution
SHS	Second Hand Smoke
AS	Active Smoke
IHD	Ischemic Heart Disease
COPD	Chronic Obstructive Pulmonary Disease
LC	Lung Cancer
HAP	Household Air Pollution
CMAQ	Community Multi Scale Air Quality Model
EDGAR	Emissions Database for Global Atmospheric Research
MOVES	Motor Vehicle Emission Simulator
PSCF	Potential Source Contribution Function
CPF	Conditional Probability Function
RH	Relative Humidity

CHAPTER 1

INTRODUCTION

1.1 GENERAL

Over the decades, high population growth has triggered rapid industrialization, urbanization and increase in vehicular traffic that has led to deterioration of air quality. Furthermore, the lack of strict implementation of environmental regulations has only added to the pollution woes in developing countries like India. According to the World Health Organization (WHO) WHO (2016) in 2016, half of the top 20 polluted cities in terms of $PM_{2.5}$ concentration are in India. In order to monitor the air pollution trends, Central Pollution Control Board (CPCB) has set up 591 monitoring stations around the country covering 248 cities/towns in 28 states and 5 union territories (CPCB, 2008). In 2015, for example, averaged concentrations of SO_2 and NO_2 in major cities did not exceed National Ambient Air Quality Standards (NAAQS) in any of the major cities in India. Even the eight hour concentrations of O_3 and CO in major cities in north (47.8 µg/m^3 and 1.26 mg/m^3), east (48.1 µg/m^3 and 1.73 mg/m^3), west (58.6 µg/m^3 and 1.27 mg/m^3) and south India (58.6 µg/m^3 and 0.94 mg/m^3) were less than the NAAQS standards (100 µg/m^3 and 2 mg/m^3). In contrary, the annual average concentration of $PM_{2.5}$ in major cities in north, east, west and south Indian cities were 3.3, 3.7, 2.3 and 1.6 times of NAAQS standard (40 µg/m^3) (Garaga et al., 2018).

Predominant sources of these regulated pollutants differ. For example in New Delhi, residential, transport and industrial sectors are major sources for $PM_{2.5}$ (S. K. Sahu et al., 2011); transport sector is the major source of CO and NOx (Aneja et al., 2001); and industries are major source for SO_2 (Sadavarte & Venkataraman, 2014). A WHO report observed that air pollution resulted in around seven million deaths globally in 2012, of which South East Asian region, dominated by India, accounted for 2.3 million (WHO, 2014b). In all India accounted for 25.7% of global

premature deaths due to $PM_{2.5}$ exposure in the year 2015 (IHME, 2017). Therefore, this calls for knowledge about the most dangerous pollutant to formulate stricter laws and better control strategies since aggravated concentrations of criteria pollutants pose constant threat to human health and environment.

As the absolute concentrations of these pollutants differ, it is necessary to bring these pollutants onto a similar scale for direct comparison. To alleviate this, Ott (1978) suggested a scheme to transform the weighted values of air pollutant concentrations into a single or set of numbers, referred to as Air Quality Index (AQI), wherein bigger the AQI greater the pollution, higher is the health risk, and vice versa. The quality of air is reported as good, satisfactory, moderate, poor, very poor or severe depending on the overall AQI, calculated using individual AQI of pollutants considered. Several developed nations in the world, including the US, UK, Australia, and Canada, have their own AQI. These vary by the range of the index, and the methodology used to estimate species specific and overall AQI. For example, UK (COMEAP, 2011) and Canada (H. Chen & Copes, 2013) classify AQI into four categories, with AQI ranging from 0 to 10. However, the US uses six categories, ranging from 0 to 500. Moreover, while Canada uses a non-linear aggregate of AQI of different species based on their exposure-response relationships, the US and UK use maximum AQI of different species, to estimate the overall AQI. Few studies in India, tried to analyse air quality using AQI in cities. For example, M. Sharma et al. (2003) used Indian NAAQS and health standards by the US, for suspended particulate matter (SPM), CO, NO_2, O_3, SO_2, and PM_{10} to estimate AQI in an Indian city. Bhaskar and Mehta (2010) used the US method to estimate AQI at thirteen different sites in Ahmadabad city in India, and observed strong seasonal effects on AQI. Banerjee and Srivastava (2011) estimated AQI proposed by Rao and Rao (1979), which uses the percentage mean of measured concentrations normalized by their corresponding NAAQS.

Their analysis at an industrialized region, for PM_{10}, SPM, SO_2 and NO_2, indicated moderate to heavy air pollution, for most part of the year. Bishoi et al. (2009) used yearlong concentrations of CO, SO_2, PM_{10}, O_3 and NO_2, to compare AQI estimated using the US methodology and factor analysis. Results show that while both methodologies follow similar trends, the US methodology estimated higher AQI than factor analysis.

However, in order to quantify the health risk posed by a pollutant, it is essential to study the association between pollutant exposure and health outcome. These studies also aid regulatory agencies in fixing the pollution reduction targets to a level that would minimize the health risk of the exposed group. Health risk based studies are quite common in western countries. A. J. Cohen et al. (2005) observed that the relationship between relative risk and cardiopulmonary diseases, lung cancer, and acute respiratory infections in children was linear between $PM_{2.5}$ concentrations of 7.5 µg m^{-3} to 50 µg m^{-3}, and flattened thereafter. C. A. Pope, 3rd et al. (2009) observed that a nonlinear power function expressed the relationship between relative risk of ischemic heart disease, cardiovascular disease and cardiopulmonary disease mortality from cigarette smoking. Results also indicated that initially cardiovascular disease mortality increased steeply with increase in concentration of fine particulate matter and flattened out at higher exposure concentrations. In India, mainly due to lack of mortality data, there are very few studies relating health risk and pollutant concentrations. Dholakia et al. (2014) studied the short term association between PM_{10} concentration and mortality for five Indian cities. Balakrishnan et al. (2011) and Rajarathnam et al. (2011) did a time series study on Chennai and Delhi respectively and obtained the risk co-efficient of PM_{10}, NO_2 and SO_2. Similarly, Sarath K. Guttikunda and Goel (2013) studied the health impacts of PM_{10} and $PM_{2.5}$ in Delhi using exposure response coefficients from R. W. Atkinson et al. (2012). Chowdhury and Dey (2016) used satellite data for calculating nationwide

$PM_{2.5}$ exposure and a risk model to estimate cause specific premature deaths from ambient $PM_{2.5}$ exposure.

Region of origin of high $PM_{2.5}$ and associated risks is a critical knowledge that can assist authorities in taking an informed decision while formulating mitigation policies. Local receptor models such as CMB, PCA, PMF etc. are commonly used to identify sources since such models are based on observation data they are considered to be fairly accurate. However, these models are limited by frequency, spatial coverage, co-linearity between source profiles and secondary pollutant and requirement of prior knowledge of sources in some cases (Burr & Zhang, 2011). Source oriented models can be used to estimate pollutant concentration due to a source in any area which can be associated with risk or mortality using a risk function or a health study. Lelieveld et al. (2015) estimated the global contribution of seven outdoor air pollution sources to mortality. Similarly Hu et al. (2017) estimated premature mortality attributed to $PM_{2.5}$ sources in China and observed that industrial and residential were the major sources. Source oriented models although widely used require emission inventory for estimating pollutant concentration, uncertainty in emission inventories over India often over or underestimates pollutant concentration (Karambelas et al., 2018; S Ramachandran et al., 2015). The air mass trajectories reaching a site is associated with pollutant emissions, which the trajectories pick up as it passes over industrial stacks, vehicles and other sources, backtracking these trajectories can help us identify pollutant sources required to regulate air quality. Hybrid Single Particle Lagrangian Integrated Trajectory Model (HYSPLIT) is an effective tool to track air mass trajectories emanating from an area and hence can provide the required information. The knowledge of region of origin of pollutants resulting in high concentration in the study area comes in handy for regulating agencies in determining the designated areas, which require stricter control policies. For e.g.: Saikat Ghosh et al. (2015)

conducted a study in Delhi using HYSPLIT which indicated Punjab, Haryana, Uttar Pradesh and Pakistan contributed majorly to high $PM_{2.5}$ concentration in Delhi, the regulating agencies can thus use this information and enforce strict regional laws instead of enforcing local control policies to bring down the level of pollution in Delhi.

1.2 RESEARCH OBJECTIVE

The major objective of the research is to estimate the contribution to health risk at major cities in India due to $PM_{2.5}$ emissions from different source regions using HYSPLIT back trajectories. The subtasks undertook to accomplish the above objective is as follows:

1. Estimation of health risk in Delhi due to high $PM_{2.5}$ concentrations using time series epidemiological study with $PM_{2.5}$ concentration data collected by CPCB and mortality data from the Office of Statistics, Delhi.
2. Validation of $PM_{2.5}$ related health risk estimates from Integrated Exposure Response function (IER) in India at high $PM_{2.5}$ concentration using risk estimates in Delhi from a time series study, thus enabling us to use the function elsewhere in India.
3. Estimation of contribution to risk due to $PM_{2.5}$ in Indian cities from geographical source regions by coupling $PM_{2.5}$ linked HYSPLIT back trajectories and IER risk function.

1.3 WORK PLAN

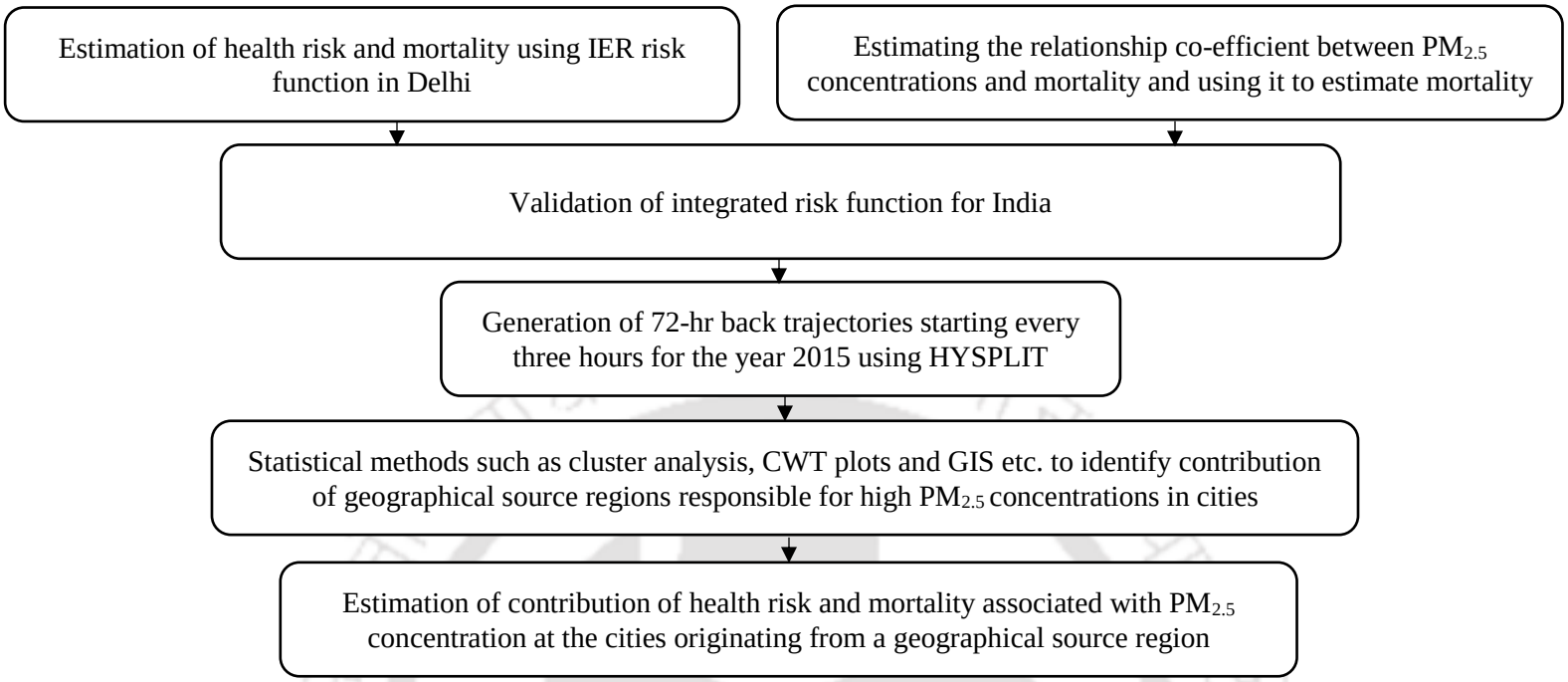

Figure 1. 1 Work flow used to estimate geographical source region contribution to health risk and mortality in cities using HYSPLIT

1.4 NOVELTY OF THE RESEARCH

1. Short-term exposure response study of $PM_{2.5}$, which is the best indicator to the health risk levels due to air pollution (WHO, 2014a), was carried out to for the first time in India using recorded health data.
2. Quantification of health risk due to high $PM_{2.5}$ concentration using recorded health data in a megacity of India.
3. Validation of IER risk estimates at high $PM_{2.5}$ concentration which was absent till date due to limited studies in cities having high $PM_{2.5}$ concentration.
4. Source region and their contributions to high $PM_{2.5}$ concentration and associated health risks and mortality observed in major cities in India is identified using back trajectory analysis.

1.5 ORGANIZATION OF THE THESIS

The thesis is divided into 5 chapters. Chapter 1 provides a brief description of the status of air quality in India and the primary pollutant responsible for it, importance of health risk studies and need for identification of geographical source region responsible for elevated health risk levels and various models used for the same. The chapter further outlines the objective of the study and its novelty. Chapter 2 deals with the literatures reviewed to understand the methods required to accomplish the objective of the present study. In short, literatures using time series study to find association between pollutant concentration and mortality, HYSPLIT studies for identification of trajectory origin using Concentration weighted trajectories (CWT) plots and cluster analysis and studies using Risk functions for estimating health risks due to $PM_{2.5}$ concentration were reviewed. Chapter 3 discusses the research methodology of each subtask required for achieving the final objective. It includes discussion regarding the assumptions made, study area and data sources required for undertaking each subtask. A detailed account on conduction of time series epidemiological studies using Poisson's regression equation and calculation of health risk has been included. The various risk functions available to estimate the health risk in case of absence of health based data has also been listed and the various exposure types and the epidemiological studies taken into consideration while developing those functions is discussed. Finally, it also discusses the identification of $PM_{2.5}$ hotspots using HYSPLIT back trajectory data, Cluster analysis and eventually combining it with health risk function to estimate the contribution of $PM_{2.5}$ associated mortality to the cities from geographical source regions. Chapter 4 discusses the results from each subtask; it lists out the health risk due to $PM_{2.5}$, NO_2 and CO in Delhi and benefits of $PM_{2.5}$ reduction in the city. The chapter also discusses the identification of function best suited for risk estimation in case of absence of health data and its validation at high $PM_{2.5}$ concentration using

the results of Delhi study, in addition it also details out the variation of health risks in other Indian cities. The chapter further discusses the sensitivity study on HYSPLIT back trajectories, the results of back trajectory analysis as well as the results from $PM_{2.5}$ associated mortality contribution to the cities. Chapter 5 provides a brief summary of the findings, the conclusions drawn, possible application, research limitation and future scope.

CHAPTER 2

LITERATURE REVIEW

According to WHO (WHO, 2012), 7 million deaths in 2012 were associated with air pollution. Deaths due to non-communicable diseases such as cardiovascular diseases, stroke, chronic obstructive pulmonary disease and lung cancer make up around 94% of those deaths. The exposure to pollutants can lead to any of the four disease depending on the relevant biological pathways of the exposed individual that are activated due to exposure to $PM_{2.5}$ concentration.

Indian cities are consistently placed among the world's most polluted cities in terms of air pollution. For example, in a recently released report by WHO in 2016 (WHO, 2016) of top ten cities with high $PM_{2.5}$ concentrations, four were in India. Previous studies concluded that China and India accounts for nearly half of total premature deaths due to air pollution (IEA, 2016). Due to unprecedented crackdown on pollution in China, deaths due to outdoor air pollution in India overtook China (Greenpeace, 2016).

As discussed in introduction section of the thesis, as the current study focuses on estimating contributions of $PM_{2.5}$ related mortalities from geographical source regions. Thus, the current review is divided into three parts, i.e. epidemiological studies used to estimate risk of outcome, risk function studies used to estimate risk in absence of outcome data and studies estimating contribution of source region to $PM_{2.5}$ concentration in study area.

2.1 EPIDEMIOLOGICAL STUDIES

Epidemiology is the study of the distribution and determinants of disease frequency in human populations (LaMorte). It attempts to link human health effects (e.g. Lung Cancer) with cause (e.g. Pollutant/chemical exposure). The studies are usually carried out by comparing exposed with

unexposed group. Risk greater than one indicates positive association while less than or equal to one indicates negative or no association between cause and effect. Pollutant concentrations when exceed the safe limits set by the CPCB (2009) results in health risks and may causes several diseases to people. These health risks can be quantified through epidemiological studies. The epidemiological studies are usually classified into three as discussed in figure 2.1 below (LaMorte; Mann, 2003):

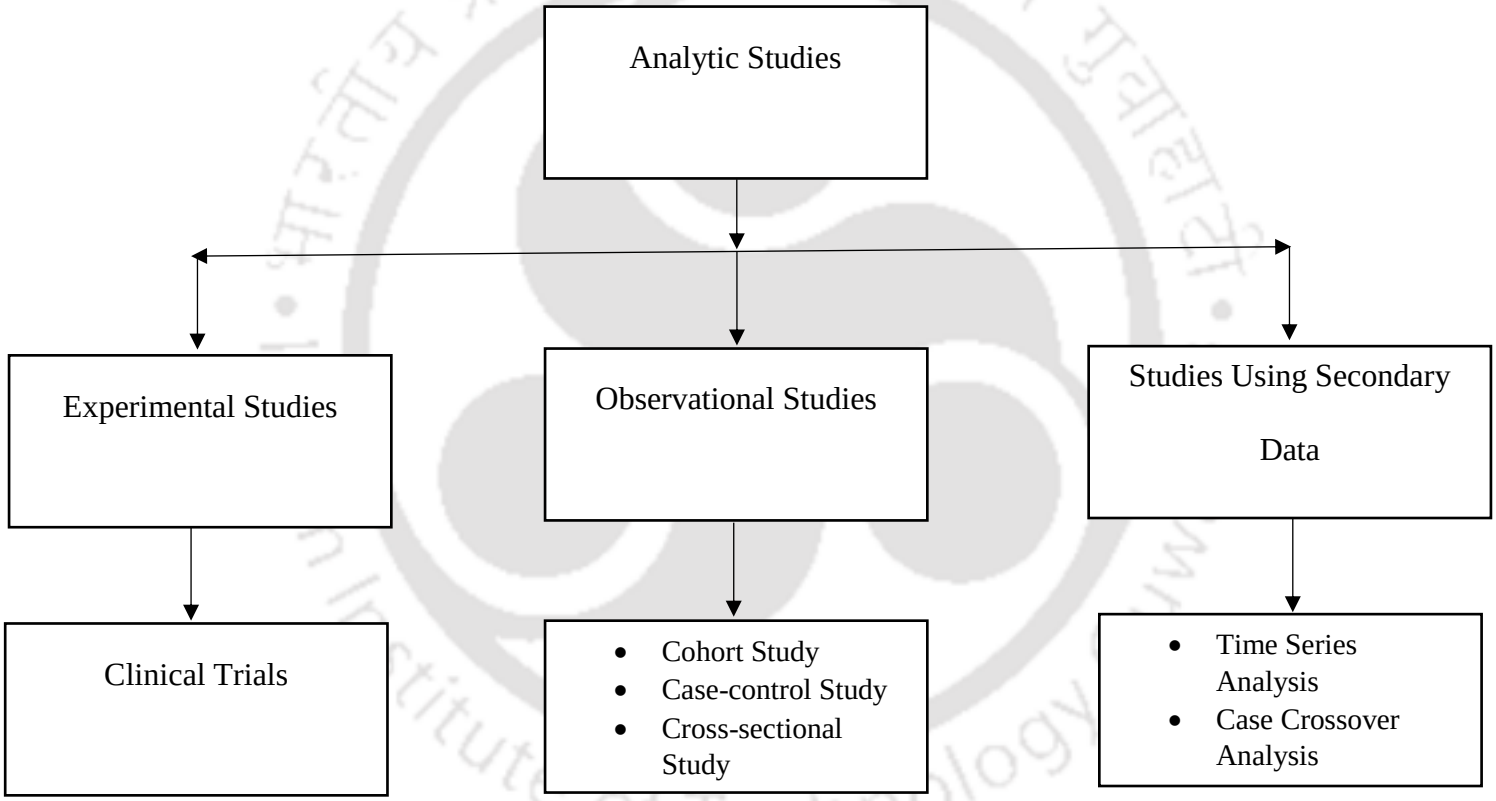

Figure 2. 1 Types of epidemiological studies

In general, both observational studies such as cohort, case-control and cross-sectional and studies using secondary data are usually carried out to identify the effect of air pollutant concentrations on human health while clinical trials are specifically used in the medical field usually to identify

the effectiveness of a drug. Crouse et al. (2015) conducted a 16 years cohort study examining the effect of ambient $PM_{2.5}$, NO_2 and O_3 exposures in Canada and observed that the pollutants had positive associations with non-accidental mortality ($PM_{2.5}$ =1.035, O_3=1.018 and NO_2=1.052) and several other causes of death. Similarly, Beelen et al. (2008) conducted a nine year cohort study to assess effect of mortality on exposure to traffic related air pollution and observed positive association of the pollutants with natural ($PM_{2.5}$ =1.06, Black smoke=1.05, SO_2= 0.97 and NO_2=1.08) and other causes of death. Carey et al. (2013) conducted a four year cohort study to investigate the effect of long term exposure to pollutants and found that all pollutants except O_3 were positively associated ($PM_{2.5}$=1.02, NO_2=1.03, and SO_2=1.04, respectively) with all cause mortality. A case control study by Madrigano et al. (2013) examined the effects of particulate matter in the area with acute myocardial infarction and concluded that an increase in $PM_{2.5}$ concentration resulted in 16% increase in the odds of acute myocardial infraction (AMI). Likewise, Perez-Padilla et al. (1996) conducted a case control study to evaluate the risk of chronic bronchitis and chronic airway obstruction among women above 40 years of age due to cooking with traditional stoves and observed odds ratio to be 3.9 for chronic bronchitis and 1.8 for chronic airway obstruction. A cross sectional study by J. J. Kim et al. (2004) was undertaken to evaluate the effect of traffic related air pollution near busy road, the odds ratio for asthma due to NO_x was observed to be 1.07.

However, observational studies often require investment of considerable amount of time and money, which might not plausible in developing countries like India. In such countries, analysis of archived data stacked at regular intervals using time series analysis or case crossover analysis is the only way forward. Touloumi et al. (1994) conducted a time series study in Athens and found that a 10% reduction in SO_2, smoke and CO result in decrease in mortality by 0.65%, 0.75% and

0.55% respectively, with steeper exposure response slopes. Similarly, Richard T. Burnett et al. (1997) conducted a time series study in sixteen Canadian cities and found that previous day ozone concentration was positively associated with respiratory admissions and the relative risks varied among cities ranging from 1 to 1.09. In order to quantify the respiratory effects in Paris, a time series epidemiological study was conducted by Dab et al. (1996). Results indicated that a 100 $\mu g/m^3$ increase in PM_{13} and black smoke concentrations increased the risk of respiratory death by 17% and 4.1%, respectively. Similarly, Verhoeff et al. (1996) associated mortality counts and air pollution levels in Amsterdam and found that black smoke and PM_{10} were positively associated with mortality and relative risk was 1.19 and 1.06 per 100 $\mu g/m^3$ of the respective pollutants. This study also noted that particulate air pollution even at low levels is associated with a variety of health effects. Z. Xu et al. (2000) observed that the all-cause mortality increases by 1.7% and 2.4% per 100 $\mu g/m^3$ increase in total suspended particulate matter and SO_2, respectively in Shenyang, China. C. A. Pope, 3rd et al. (1991) conducted a study on school children of fifth and fourth grade in Utah valley and found that increased PM_{10} concentrations were associated with approximately 3 to 6% decline in lung functions. Elevated PM_{10} concentration was also associated with increase in respiratory diseases and use of asthma medication. A study conducted in ten cities of Europe found that pooled relative risks of deaths from cardiovascular conditions and respiratory diseases were 1.02 and 1.04 per 50 $\mu g/m^3$ increase in the concentration of black smoke (Zmirou et al., 1998).

Very few such epidemiological studies have been conducted in India compared to western countries which experience lower $PM_{2.5}$ concentrations when compared to India. Dholakia et al. (2014) studied the association of mortality and PM_{10} concentrations in five cities including Shimla, Ahmedabad, Bangalore, Hyderabad and Mumbai and observed that excessive risk per 10 $\mu g/m^3$ of

increase in PM_{10} concentration was highest in Shimla (1.36%) and lowest for Ahmedabad (0.16%). The study also found that the health benefits of reducing pollution are higher in cleaner cities as opposed to dirtier cities. Balakrishnan et al. (2011) did a time series analysis in Chennai and found that a 10 µg/m^3 increase in daily PM_{10} concentration will result in a 0.44% increase in all-cause mortality. Similarly, Rajarathnam et al. (2011) conducted a time series study in Delhi and found that a 10 µg/m^3 increase in PM_{10} and NO_2 concentrations will result in an increase in 0.15% and 0.84%, respectively increase in all-cause mortality. Behera and Balamugesh (2005) studied the effect of indoor air pollution on lung cancer in women and observed that biomass fuels had the risk of developing lung cancer with odds ratio of 5.33 while use of mixed fuel had a risk of 3.04. Unfortunately, none of the studies in India had focused on the health risk due to high ambient $PM_{2.5}$ concentrations which is more dangerous than PM_{10} owing to its ability to penetrate deep into lungs (Xing et al., 2016).

2.2 RISK FUNCTION TO ESTIMATE HEALTH RISK

In order to conduct an epidemiological study, $PM_{2.5}$ concentration and mortality data is required as an input. While $PM_{2.5}$ data is available from nearby monitoring stations it is difficult to obtain the disease specific mortality data, due to poor record keeping. In case of unavailability of mortality data, risk functions which can estimate health risks for a given pollutant concentration can be used. These risk functions are usually obtained by fitting estimated health risk and corresponding $PM_{2.5}$ concentrations from previous epidemiological studies. A.J. Cohen et al. (2004) postulated a linearly increasing risk function for cardiopulmonary mortality due to $PM_{2.5}$ concentrations from 7.5 to 50 µg/m^3, with no further increase in risk at higher concentration. The concentrations were capped at 50 µg/m^3 to avoid unrealistically large estimates of mortality. The counterfactual concentrations were capped at 7.5 µg/m^3 since this was very close to the lowest

$PM_{2.5}$ concentration observed by C. A. Pope, 3rd et al. (2002). In order to evaluate the uncertainty in base case risk estimates, sensitivity analyses were carried out which included: an assumption that excess mortality does not increase with increase in $PM_{2.5}$ concentration above 30 μg/m^3, an assumption that mortality increases linearly above 30 μg/m^3 and an assumption that excess mortality increases as logarithm of $PM_{2.5}$ concentration. Effect of variable $PM_{2.5}$: PM_{10} ratio across the regions and variable counterfactual concentrations to the base case estimates were also quantified using a sensitivity analysis to evaluate the uncertainty with the base case estimates vs plausible alternatives. The WHO recommended the use of log-linear model to estimate the burden of air pollution (B. Ostro, 2004), as linear extrapolation would result in unrealistic large estimates of RR (A.J. Cohen et al., 2004; B. Ostro, 2004). (C. A. Pope et al., 2011) suggested that to understand the shape of exposure-response curve over a wide range, integration of lung cancer and cardiovascular mortality from $PM_{2.5}$ exposures such as Ambient Air Pollution (AAP), Second Hand Smoke (SHS) and Active Smoke (AS) may provide us valuable input. R. T. Burnett et al. (2014) developed an integrated risk function (IER) as shown in equation (2.1) to estimate the health risks due to ischemic heart disease (IHD), stroke, Chronic obstructive pulmonary disease (COPD) and lung cancer (LC) using relative risk information from epidemiological studies of long term exposure to $PM_{2.5}$ from AAP, SHS, AS and household air pollution (HAP).

$$RR = 1 \; for \; c < c_{cf} \tag{2.1}$$

$$RR = 1 + \alpha\left\{1 - \exp\left[-\beta\left(c - c_{cf}\right)^{\delta}\right]\right\} \; for \; c \geq c_{cf}$$

where α, β and δ are related to disease specific coefficients obtained by fitting Relative Risk (RR) information from available studies of ambient air pollution, second hand smoke, active smoke and

household solid cooking fuel (http://ghdx.healthdata.org/sites/default/files/record-attached-files/IHME_CRCurve_ parameters.csv). c is the average $PM_{2.5}$ concentration and c_{cf} (5.8 $\mu g/m^3 \leq c_{cf} \leq 8.8\ \mu g/m^3$) is the counter-factual concentration below which it is assumed that there is no additional risk.

IER model was compared with several other models, many of which were suggested previously. Two Models in which risk increased as a linear function of $PM_{2.5}$ concentration up to 30 $\mu g/m^3$ in one while 50 $\mu g/m^3$ in another were included in the sensitivity analysis. Similarly, a model where risk increases as a logarithm of $PM_{2.5}$ concentration was also included. These models are similar to the ones as already discussed by A.J. Cohen et al. (2004). Further, a modified logarithm model with an unknown parameter to concentration to allow for more flexibility in fitting type specific RR's as postulated by R. T. Burnett et al. (2014) was also considered, a model wherein the exposure was raised to a power as suggested by C. A. Pope, 3rd et al. (2009) and a model similar to IER but with $\delta=1$ was used for comparison with IER.

2.3 REGION OF ORIGIN OF HIGH HEALTH RISK

Recently three dimensional air quality models have been modified as source oriented air quality models for source apportionment purposes. Many such studies have been performed till date to identify the sources: H. Zhang et al. (2012) studied the major sources of sulfate and nitrate which are considered to form a significant fraction of $PM_{2.5}$ using a source oriented version of Community Multiscale Air Quality model (CMAQ). Qiao et al. (2015) studied the dry and wet deposition of sulfate, nitrate and ammonium anions in Jiuzhaigou National Nature Reserve, China using source oriented CMAQ model to identify different emission sectors or source regions responsible for deposition of flux. Similarly, D. X. Wang et al. (2014) studied the contribution of

energy production, industries, transportation, residential activities, windblown dust and others to primary and secondary inorganic $PM_{2.5}$ using CMAQ model and Emissions Database for Global Atmospheric Research (EDGAR) emission inventory as an input. A source apportionment study of sulfate and nitrate particular matter in seven eastern cities of US was done by H. L. Zhang et al. (2014)and on-road gasoline powered vehicles, diesel vehicles, natural gas and coal combustion were found to be the major sources. Kota et al. (2014) used source oriented CMAQ model to evaluate the difference in NO_x and CO emission inventories for southeast Texas generated by MOVES and MOBILE6.2. A study was carried out by H. L. Zhang and Ying (2011) regarding source apportionment of Secondary organic aerosol in Southeast Texas using source oriented CMAQ model. A source apportionment study of formaldehyde using a source oriented CMAQ model was undertaken by H. L. Zhang et al. (2013) to quantify the contribution of vehicle, industry, natural gas combustion, wildfires and biogenic sources to regional primary and secondary formaldehyde concentrations.

Unfortunately, such studies in India are very scarce: Marrapu et al. (2014) used WRF-Chem and analysed effectiveness of control measures taken during Delhi Commonwealth games. S. Sharma et al. (2016) analysed the sensitivity of ozone precursors to ground level ozone concentration using WRF-Chem brute force method. Similarly Surendran et al. (2016) used Hemispheric Transport of Air Pollution (HTAP-2) brute force method to analyse the effect on global distribution of ozone due to 20% reduction in anthropogenic emissions from the energy, industry and transport sectors in South Asia. Jena et al. (2015) used WRF-Chem and Fire Inventory from NCAR (FINNv1) brute force method to obtain the influence of biomass burning on ozone concentration. R. Kumar et al. (2013) found that wintertime CO in the boundary layer and free troposphere over India is mostly due to anthropogenic emissions using WRF-Chem. None of the studies had earlier used source

tagged method to identify the sources in India until recently, when Guo et al. (2017) conducted a study in North India using CMAQ with source tagged method and observed that industrial or residential activities were the dominant sources of primary particulate matter while energy and agricultural sectors were observed to be the prime contributors to secondary inorganic aerosols.

However, using region typed Eulerian air quality models in India could yield erroneous conclusions. This is due to uncertainty in emission inventories in India (Garaga et al., 2018; Karambelas et al., 2018; S Ramachandran et al., 2015) which are an important input for the source oriented air quality models might result in severe over or under estimation of sources which defeat the very purpose of conducting the same. Another prominent way is to use Lagrangian models. Hybrid Single Particle Lagrangian Integrated Trajectory Model (HYSPLIT) is one such effective model used to track air mass trajectories emanating from an area to locate potential region of trajectory origin. The trajectories scoop in the emissions from various sources such as vehicles, industries, biomass burning etc. and thus determination of location of the trajectories associated with high pollutant concentration can lead us to possible geographical source region of the pollutants.

(Pekney et al., 2006) conducted potential source contribution function (PSCF) and conditional probability function (CPF) analysis of HYSPLIT trajectories with PMF-modeled source contributions to locate sources. Jaffe et al. (2005) studied the export of mercury from Asia using back trajectory analysis. Similarly, M. Escudero et al. (2006) used HYSPLIT model to determine the contribution from north African dust source areas to PM_{10} concentration over central Iberian Peninsula. Borge et al. (2007) used two-stage atmospheric clusters to analyse influence of long range transport of PM_{10} from North Africa and continental Europe on Madrid and Birmingham.

Likewise, HYSPLIT back trajectory model was used to identify origin of air mass associated with high PM_{10} concentration in Athens and Birmingham due to long-range transport of particles from continental Europe (Vardoulakis & Kassomenos, 2008). Song et al. (2006) used backward trajectory and elemental analyses for source apportionment of $PM_{2.5}$ in Beijing. HYSPLIT back trajectory model was used to identify long-range PM_{10} source contribution to Istanbul (Karaca et al., 2009). Similarly, Y. Liu et al. (2009) analysed the impact of forest fires in Athens using HYSPLIT back trajectories and satellite data. A study in Istanbul evaluated source contributions to PM_{10} profile using HYSPLIT model (Karaca & Camci, 2010). Likewise, a back trajectory analysis was used to identify the origin of daily exceedance of European PM Limit (Miguel Escudero et al., 2007).

A number of such studies have been conducted in India till date: Datta et al. (2010) used HYSPLIT for studying the temporal and spatial variation of SO_2 in Delhi. Tiwari et al. (2012) studied variation of $PM_{2.5}$, PM_{10} and PM_1 using back trajectory analysis. Similarly, Srivastava et al. (2011) studied a dust event in Delhi using HYSPLIT starting heights as 1733m, 740m, 3500m, 3000m and 2500m. Further Kumar and Kumari (2015) used HYSPLIT while studying characterization of aerosol and trace gases over Indo-Gangetic plain. A dust storm in Patna was studied by Giri et al. (2013) using HYSPLIT. Tyagi et al. (2016) conducted an evaluation of gaseous pollutants over NCR region of Delhi and used HYSPLIT to evaluate the origin of pollutants. In Mumbai Sandeep et al. (2013) studied the seasonal variation of Black carbon and its origin source using 3 day HYSPLIT back trajectories. Pavuluri et al. (2010) in Chennai studied the origin of aerosol nitrogen using HYSPLIT. S. Ramachandran and Jayaraman (2003) used HYSPLIT to study the characteristics of aerosol optical depths over Bay of Bengal. V. Kumar et al. (2013) investigated sources and trends of particulate matter during summertime over India using HYSPLIT. Similarly,

Saikat Ghosh et al. (2015) used HYSPLIT to investigate the potential regional and local source regions contributing to $PM_{2.5}$ concentrations in Delhi.

2.4 SUMMARY

The previous epidemiological studies conducted in India have focussed on exposure to PM_{10} and NO_2 with no studies yet on health risk due to $PM_{2.5}$ exposure. Time series epidemiological study can be conducted in Indian cities to estimate health risk of $PM_{2.5}$ concentration. It requires low time and capital investment and hence have found to be the most plausible method to estimate health risk in countries like India. However, such studies require mortality/hospitalization data as well as $PM_{2.5}$ concentration data to estimate the risk of mortality on increase in $PM_{2.5}$ concentration. The mortality/hospitalization data of cities might however not be available. In such cases risk functions can be used to estimate the health risks using $PM_{2.5}$ concentration. These functions provide the relationship between health risk and $PM_{2.5}$ developed using previous epidemiological studies. However, since very few epidemiological studies have been conducted in developing countries like India & China, the risk estimates at high $PM_{2.5}$ are provided by assuming that interpolation of risk estimates from disparate types of exposures such as AAP, SHS and AS will provide plausible risk estimates at high $PM_{2.5}$ concentration. The validation of risk function at high $PM_{2.5}$ concentration can be done using the Time series epidemiological study conducted in Delhi. In order to control the premature mortality due to $PM_{2.5}$ concentration, control agencies required the knowledge regarding the contribution of source region to $PM_{2.5}$ concentration in the city. Such analysis can be done only using modelling techniques such as region-typed Eulerian models like CMAQ & WRF-CHEM or Lagrangian models like HYSPLIT. Eulerian models require proper emission inventory as input which is still lacking for India, leaving HYSPLIT as the only viable option for the study. HYSPLIT back trajectory analysis can provide the source

region contribution of $PM_{2.5}$ which can then be used as input to risk function to also estimate the risk and mortality associated with $PM_{2.5}$ contributed by the source regions.

2.5 KNOWLEDGE GAPS

The following important observations were made after the literature review:

1. Epidemiological studies, which are required for estimating health risks due to air pollution, are very scarce in India.
2. Studies using archived data analyzed using time series are most suitable for countries like India.
3. Present epidemiological studies in India do not estimate health risk due to $PM_{2.5}$ concentrations.
4. Health risk function is the only option currently available in India to estimate health risk as mortality data in different cities is not available.
5. Integrated risk function (IER) is the model used to estimate health risk due to $PM_{2.5}$ concentrations in both developed and developing countries, since it is defined over a wide range of concentrations, however it is still required to be validated at high $PM_{2.5}$ concentration.
6. Incomplete emission inventory for India results in under/over estimation by Eulerian models leaving HYSPLIT as the only option to estimate geographical source region contribution of $PM_{2.5}$ concentrations in Indian cities.
7. No multicity analysis to identify geographical source regions and their contributions to high $PM_{2.5}$ concentrations and associated premature mortality affecting Indian cities is carried out till date.

CHAPTER 3

METHODOLOGY & APPLICATION

The chapter describes in details the various steps taken to achieve the objectives of the current research. The details regarding health risk study undertaken in the city of Delhi to quantify the effect of $PM_{2.5}$ has been outlined in the current chapter. It also discusses the risk functions used for estimating health risks in cities with no mortality data and its validation for Indian cities, which experiences high $PM_{2.5}$ concentration. Finally, the contribution of geographical source region to $PM_{2.5}$ concentration in cities is evaluated using HYSPLIT back trajectories analysis which when linked to risk function will also evaluate the mortality contributed from the geographical source regions.

3.1 TIME SERIES EPIDEMIOLOGICAL STUDY IN DELHI

Ambient $PM_{2.5}$ concentration was observed to be the fifth largest mortality risk factor in 2015 resulting in 4.2 million deaths globally (Aaron J Cohen et al., 2017). High $PM_{2.5}$ exposure has been associated with chronic obstructive pulmonary disease, ischaemic heart, lung cancer, stroke and other severe health hazards (Franklin et al., 2006; Krewski et al., 2009; Tecer et al., 2008). In order to construe the status of air quality in India, National Ambient Air Quality Standards (INAAQS) were adopted in 1982 by the Central Pollution Control Board (CPCB), with further revisions in 1994 and 2009. Pollutant concentrations in Delhi frequently exceed the INAAQS limits, annual average $PM_{2.5}$ (119.63 µg/m^3) and NO_2 (60.36 µg/m^3) concentrations in RK Puram during 2017 were observed to exceed the CPCB limits of 40 µg/m^3 while 8hrs average of CO (2.11 mg/m^3) was also observed to exceeded limits of 2 mg/m^3. However, SO_2 and O_3 concentrations were observed to be within the INAAQS limits of 50 µg/m^3 and 100 µg/m^3 respectively.

Since the INAAQS limits of the pollutants differ, they are required to be converted to the same scale in order to be compared. The concentrations of pollutants are thus converted to a scale between 0-500 depending on the pollutant concentration referred to as Air Quality Index (AQI), wherein higher AQI indicated higher dominance of the pollutant. The pollutant having the highest AQI is considered as the dominant pollutant and its corresponding AQI is considered as overall AQI. The AQI was developed only as a means to communicate to people regarding the quality of air. The AQI, as can be visualized from previous statements is biased towards the dominant pollutant and may well underestimate the health effects due to the combined effect of the pollutants.

Thus, in order to quantify the health risk posed by a pollutant, it is essential to study the association between pollutant exposure and health outcome. These studies are usually referred to as epidemiological studies, among which time series epidemiological study was found to be more plausible for countries like India and the same have been discussed in details in Chapter 2.

3.1.1 Assumptions

- $PM_{2.5}$ concentration in ITO is assumed to be representative of entire Delhi. Similar assumption of considering pollutant measured at a location being representative of the entire city has also been made in several other studies (Garrett & Casimiro, 2011; S.-Y. Kim et al., 2015).
- Risk due to $PM_{2.5}$ concentration is only due to total $PM_{2.5}$ inhaled mass and does not depend on its composition (R. T. Burnett et al., 2014).

- The relative risk is assumed to vary exponentially with $PM_{2.5}$ concentration, in case of all-cause mortality calculations, as suggested by WHO. *(http://www.who.int/quantifying_ehimpacts/publications/ebd5.pdf)*

3.1.2 Study area and data sources

New Delhi, in addition to being the capital of India, is also one of the most densely populated cities in the world. It has a population of 16.7 million with an annual average growth rate of 1.92%. The overall population density is 11,297 per sq. km. It faces extreme temperatures of as low as 7ºC in winter to as high as 48ºC in summer (https://www.wunderground.com/). It received an average rainfall of 889.2 mm from 2011-2014. Such extreme temperatures can severely affect the already deteriorating air pollution in New Delhi. Four years' data ranging from January 1st, 2011 to December 31st of 2014 was used for the analysis. The data was collected at a busiest traffic intersection, Bahadur Shah Zafar Marg(ITO) located at commercial down town of the city, as shown in Fig. 3.1. Hourly concentrations of CO, O_3, NO_2, SO_2, and $PM_{2.5}$ were collected by Central Pollution Control Board (CPCB). Pollutant concentrations vary across a city. However, this is the longest period for which hourly data of these species is available in any location in Delhi. In such situations studies resort to analysis of data collected at a single location. For example, S.-Y. Kim et al. (2015) studied the association of selected components of $PM_{2.5}$ on mortality, using $PM_{2.5}$ data obtained from a centrally located residential site in Denver. Similarly, Garrett and Casimiro (2011) studied the short term effect of $PM_{2.5}$ and O_3 in Lisbon, using data obtained from a monitoring station in Olivas since this was the only station with data for both pollutants. Thus, in this study it is assumed that the concentrations observed in this location are representative of the entire city.

CO was measured using non-dispersive infrared spectroscopy, O_3 and NO_2 using Chemiluminescence, SO_2 using ultra violet fluorescence, and $PM_{2.5}$ using tapered element oscillating microbalance. As hourly PM_{10} concentrations were not available in this location, $PM_{2.5}$ is assumed to be the sole representative of particulate matter in this study. Due to unavailability of data, the percentage of days on which the IND-AQI was not calculated, was 19, 42, 41 and 60 in 2011, 2012, 2013 and 2014, respectively. The mortality data was obtained from the office of births and deaths registration in New Delhi. Due to unavailability of cause specific mortality, non-accidental mortality has been used in this study.

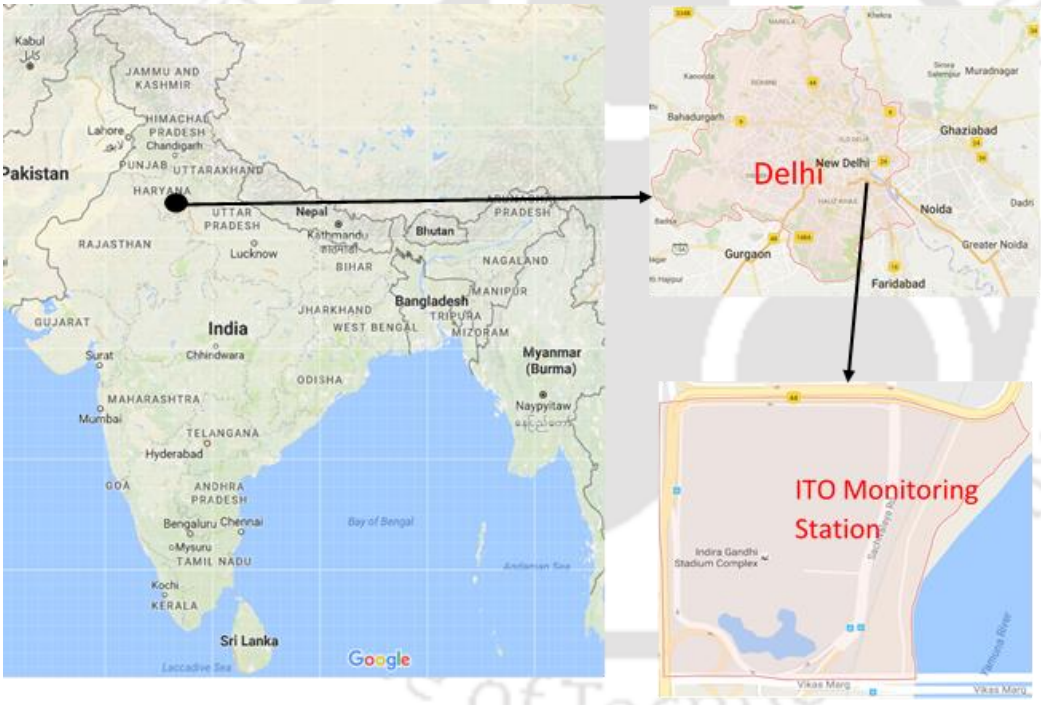

Figure 3. 1 An interactive map showing the study region (Delhi) and the location of the monitoring station (ITO) in India.

3.1.3 Indian AQI

To inform people about the quality of air quickly so that they can take appropriate measures to protect themselves, IND-AQI was released in 2014. The details of IND-AQI are available

elsewhere (CPCB, 2014), and only briefly summarized here. IND-AQI considers concentrations of PM_{10}, $PM_{2.5}$, NO_2, O_3, CO, SO_2, NH_3 and Pb. In a day, while maximum 8 hour running average concentrations of O_3 and CO are used, 24 hour averaged concentrations of other six pollutants are used in calculation. The concentration of each pollutant is converted to a number on a scale of 0-500. The sub AQI (AQI_i) for each pollutant(i) is calculated using equation (3.1)

$$AQI_i = \frac{I_{HI} - I_{LO}}{BR_{HI} - BR_{LO}} \times (C_i - BR_{LO}) + I_{LO} \quad (3.1)$$

where, C_i is the concentration of pollutant 'i'; BR_{HI} and BR_{LO} are breakpoint concentrations greater and smaller to C_i and I_{HI} and I_{LO} are corresponding AQI ranges.

The overall AQI, can be estimated only if the concentrations of minimum three pollutants are available, with at least one of them being either $PM_{2.5}$ or PM_{10}. The IND-AQI is then taken as the maximum AQI_i of the constituent pollutants, denoted as dominating pollutant. The IND-AQI is divided into five categories: good, satisfactory, moderate, poor, very poor and severe depending on whether the AQI falls between 0-50, 51-100, 101-200, 201-300, 301-400 or 401-500, respectively.

3.1.4 Health risk estimation

In order to estimate health risk, relationship between the health effect (mortality in this case) and the pollutant concentration is required to be established. Poisson regression models are widely used to analyze the relation between pollutant concentrations and mortality (Dholakia et al., 2014). The Poisson model used in this study, represented using equation (3.3), is described below (Bhaskaran et al., 2013; Imai et al., 2015).

$$\log(\mu_t) = \gamma + \beta C_t + \alpha K C_t + f(t) \quad (3.3)$$

Where t denotes time; α, γ and β are the regression coefficients; μ is related to mortality; K denotes temperature; and f(t) is a smooth function of time used to remove seasonal and long term trends. Since initial trends are dominated by seasonal and long term patterns, a spline function of time is used to remove them, adding required exposure then allows us to study the short term variation between concentration and mortality.

This study uses relative risk of the pollutants(RR_i) given by Cairncross et al. (2007), as shown in equation (3.2) to estimate health risk:

$$RR_i = \exp\left(\beta_i\left(c_i - c_{cf}\right)\right), c_i > c_{cf} \quad (3.2)$$

where β_i is the exposure-response relationship coefficient, represents the increase in mortality per unit increase in concentrations.

c_i refers to the measured concentration and c_{cf} refers to the concentration of a pollutant below which no adverse health effects can be expected. According to WHO (2005), adverse health effects can be expected for any concentration of $PM_{2.5}$, O_3, NO_2 and SO_2. Thus, in this study c_{cf} of all pollutants except CO, is considered as 0. c_{cf} for CO is considered as 2 mg m^{-3}, as suggested by (CPCB, 2014), and shown in Table 3.1.

To better cogitate its associated health benefits, the possible number of non-accidental deaths averted by reducing PM2.5 concentrations, to suggested levels by WHO and INAAQS was estimated following Shang et al. (2013).

Table 3. 1 Breakpoints of different pollutants in IND-AQI (CPCB, 2014)

AQI Category (Range)	$PM_{2.5}$ 24-hr	NO_2 24-hr	O_3 8-hr	CO 8-hr	SO_2 24-hr
Good (0-50)	0-30	0-40	0-50	0-1.0	0-40
Satisfactory (51-100)	31-60	41-80	51-100	1.1-2.0	41-80
Moderate (101-200)	61-90	81-180	101-168	2.1-10	81-380
Poor (201-300)	91-120	181-280	169-208	10.1-17	381-800
Very Poor (301-400)	121-250	281-400	209-748	17.1-34	801-1600
Severe (401-500)	250+	400+	748+	34+	1600+

Note: While CO concentrations are expressed in mg/m-3; the other pollutants are expressed in µg m^{-3}.

The expected number of premature deaths (PD_i) due to a pollutant exposure can be calculated by equation 3.4, using mortality rate (M) and total population (Pop).

$$PD_i = M \times \frac{RR_i - 1}{RR_i} \times Pop \tag{3.4}$$

The relative risk (RR_i), in equation 3.4, was calculated by equation 3.2, using c_i as the averaged $PM_{2.5}$ concentration during the analysis period, and the c_{cf} as the targeted $PM_{2.5}$ limit.

3.2 RISK FUNCTION TO ESTIMATE HEALTH RISK

As evident from previous sections, the health studies give us an insight on the health outcome due to an exposure. Such studies are extremely handy in quantifying the effects of air pollution. Unfortunately, such studies are not very common in developing countries like India where the pollutant concentrations are observed to be the highest. This is due to health studies such as cohort studies are expensive and time taking. Similarly, case control studies and cross sectional studies

requires health data, which might not be available to public in many countries. In such cases to study the health risks in countries like India where health data is not available we can make use of risk functions. These functions are based upon previous health studies, which are compiled to obtain the relationship between pollutant concentration and health risk. These functions are specific to a pollutant so risk functions of $PM_{2.5}$ cannot be used to estimate health risk due to exposure to PM_{10} concentration or vice versa. The Risk functions (IER) are based on assumption that risk will depend on mass of particulate matter, mass of heavier particulate matter such as PM_{10} are more prone to deposition (wet or dry) as compared to lighter particulate matter particles such as $PM_{2.5}$ and hence will have lesser effect on health as compared to $PM_{2.5}$ and thus will follow different curve.

3.2.1 Assumptions

- The RR's calculated are a function of inhaled $PM_{2.5}$ mass from AAP, AS, SHS and HAP and the toxicity depends on the mass of $PM_{2.5}$ inhaled since studies regarding health effects of components of particulate matter haven't been conducted done yet and hence cannot be evaluated (R. T. Burnett et al., 2014).
- The RR of mortality is a function of long term cumulative exposure and does not depend on temporal pattern of exposure (R. T. Burnett et al., 2014) .
- The RR due to each type of exposure is independent of other types of exposure (R. T. Burnett et al., 2014).
- Single value baseline mortality rate for India from WHO was assumed to be representative of all states.

3.2.1 Linearly increasing function

A. J. Cohen et al. (2005) developed a risk function to estimate global burden of disease in 14 WHO regions based on mortality from cardiopulmonary causes, lung cancer and acute respiratory infections in children from 0 to 5 years. However, the studies used for developing the function were based on risk coefficients from (Pope et al., 2002) for adults and a meta-analysis summary of five time-series studies of mortality in case of children. As no studies from developing countries were included, it resulted in large variations in burden estimates and hence could not be extended to smaller geographical regions. Burden estimates were evaluated based on assumptions that health risk varies linearly between concentrations of 7.5 and 50 $\mu g/m^3$ and thereafter remains constant.

3.2.2 Power function

C. A. Pope, 3rd et al. (2009) used the relative risks for ambient air pollution and cigarette smoking from American Cancer Society (ACS) cohorts, and Second hand smoke (SHS) estimates from other cohort studies to postulate a risk function for cardiovascular mortality. However, different measures of exposure were used in different studies. i.e. exposure in ambient air pollution studies and cigarette smoking are measured in $\mu g/m^3$ and number of cigarettes smoked per day, respectively. For calculation purposes, average daily dose was used as the common metric. The average daily dose inhaled is measured by multiplying $PM_{2.5}$ concentration with average inhalation rate. The average inhalation rate for the study was taken as 18 m^3/day, and average $PM_{2.5}$ dose inhaled per cigarette was taken as 12 mg. In case of SHS exposure, for low to moderate and moderate to high exposure, the average $PM_{2.5}$ was estimated to be 20 $\mu g/m^3$ and 50 $\mu g/m^3$, respectively. Curve fitting revealed non-linear power function to be the best fit for all the estimates (equation 3.5).

$$RR = 1 + 0.2968(dose)^{0.2107} \quad (3.5)$$

C. A. Pope et al. (2011) fitted a curve for estimating lung cancer risk due to $PM_{2.5}$ concentration to understand whether the risks of lung cancer follows similar trend as cardiovascular diseases. As discussed above, previous estimates considered cardiovascular and lung cancer mortality to flatten out at 50 µg/m^3 (A. J. Cohen et al., 2005). If the lung cancer risk estimates do not flatten at high $PM_{2.5}$ concentrations similar to cardiovascular diseases, the risk due to lung cancer will be severely underestimated in areas with high $PM_{2.5}$ concentrations. American Cancer Society (ACS)-$PM_{2.5}$ cohort was the source of risk estimates from active cigarette smoking. Risk estimates of ambient $PM_{2.5}$ concentration were obtained from ACS CPS-II cohort (C. A. Pope, 3rd et al., 2002; C. A. Pope, 3rd et al., 2004; C. A. Pope, 3rd et al., 1995), Harvard six cities analyses (Dockery et al., 1993; Laden et al., 2006) and Women's health initiative study (Miller et al., 2007) while SHS estimates were obtained from 2006 Surgeon General's Report (Health & Services, 2006) and INTERHEART study (Teo et al., 2006). Lung cancer exposure-response plot indicated a monotonic increasing risk estimate with increasing $PM_{2.5}$ concentrations as given in equation (3.6).

$$RR = 1 + 0.3195(dose)^{0.7433} \quad (3.6)$$

The equations 3.5 and 3.6 can be expressed commonly as equation (3.7)

$$RR = 1 + \alpha(dose)^{\beta} \quad (3.7)$$

Recently, Chowdhury and Dey (2016) fitted separate curves for COPD, IHD, Lung Cancer, Stroke and acute lower respiratory infection (ALRI) using equation (3.7) with risk estimates from 141

different cohort studies across different regions and exposures from ambient $PM_{2.5}$ concentration, SHS, active smoking (AS) and household air pollution (HAP). The α and β values are computed for each of the four disease based on the risk estimates as obtained from previous studies.

3.2.3 Integrated exposure response function (IER)

R. T. Burnett et al. (2014) developed an integrated risk function using Relative Risk (RR) from epidemiological studies due to exposure from ambient air pollution (AAP), SHS, AS and HAP. The risk estimates for AAP was obtained from cohort studies conducted across the globe, SHS and AS risk estimates were obtained as discussed by C. A. Pope et al. (2011) and risk estimates for HAP were provided by (Smith et al., 2014). Since the estimates from different types of exposure are different, they were converted to a common metric (C. A. Pope, 3rd et al., 2009; C. A. Pope et al., 2011) as explained in section above. Single cigarette was observed to be equivalent to exposure to a daily ambient $PM_{2.5}$ concentration of 667 $\mu g/m^3$ and the same information was used to convert no of cigarettes smoked into daily ambient $PM_{2.5}$ concentration in this study.

The function as provided in equation (2.1) was based on information from previous studies, which indicated that risk estimates due to cardiovascular diseases initially increases swiftly and flattens out at higher $PM_{2.5}$ concentration (C. A. Pope, 3rd et al., 2009), while in case of lung cancer the risk estimates increases monotonically with increase in $PM_{2.5}$ concentration (C. A. Pope et al., 2011).

For $c<c_{cf}$

$$RR_{IER}(c) = 1$$

For c>=c_{cf}

$$RR_{IER}(z) = 1 + \alpha\{1 - \exp\left[-\gamma\left(c - c_{cf}\right)^{\delta}\right]$$

c=exposure to $PM_{2.5}$ in $\mu g/m^3$

c_{cf}=concentration below which the risk is assumed to be zero

The unknown parameters (α, γ, δ) are obtained by nonlinear regression methods.

3.3 GEOGRAPHICAL SOURCE REGION CONTRIBUTION TO MORTALITY IN CITIES

Identification of region from where high $PM_{2.5}$ is originating is critical to reduction of $PM_{2.5}$ concentrations. Receptor models do a decent job in identifying sources but they cannot differentiate between local or regional sources (Burr & Zhang, 2011). Eulerian models such as WRF-CHEM and CMAQ have been used to identify regions of high $PM_{2.5}$ concentration previously (Hu et al., 2017; Lelieveld et al., 2015; Mead et al., 2018; Y. Zhang et al., 2017). However, lack of proper emission inventories in India required as input in these models might result in some regions being overlooked (Karambelas et al., 2018; S Ramachandran et al., 2015). Tracking of air mass trajectories, which usually capture and thus transports $PM_{2.5}$ concentrations while blowing over industrial stacks, household chimneys and other sources of $PM_{2.5}$ concentration is another way of determining the geographical source regions of high $PM_{2.5}$ concentration. Hybrid single particle Lagrangian integrated trajectory (HYSPLIT) model has been used in many studies for forecasting or back tracking air mass trajectories using meteorological models as input (Stein et al., 2015; Uria-Tellaetxe & Carslaw, 2014). The contribution from each region to high $PM_{2.5}$

concentration in the city is determined by proportional residence time of air mass trajectory in the region. The corresponding $PM_{2.5}$ concentration contributed from a region can be further linked to health risks and mortality using risk functions, thus providing us the geographical source region contribution to mortality in cities.

3.3.1 Assumptions

- $PM_{2.5}$ data collected at a single location in the 8 cities are representative of the $PM_{2.5}$ concentration of the respective cities (Garrett & Casimiro, 2011; S.-Y. Kim et al., 2015).
- Only the advection component is considered when running trajectories in HYSPLIT. (Stein et al., 2015).
- The contribution of a geographical source region to $PM_{2.5}$ concentration in a city depends on the proportional residence time of the trajectories passing through the source region (Fleming et al., 2012; Stohl et al., 2002).
- The conditions within the mixing layer height is assumed to be well mixed (Stein et al., 2015).

3.3.2 Study area and data sources

Eight Indian cities, which are in top 15 most populated cities in the country, were selected based on availability of $PM_{2.5}$ data as shown in Figure 3.2. The Indian cities chosen for the study includes Patna and Kolkata in the east, Bangalore, Chennai and Hyderabad in the south, Mumbai in the west, and New Delhi and Lucknow in the north. $PM_{2.5}$ data was obtained from the Central Pollution Control Board (CPCB) (http://www.cpcb.gov.in/CAAQM/frmUserAvgReportCriteria.aspx) for all locations except Kolkata, for which $PM_{2.5}$ data was downloaded from the US consulate (http://photos.state.gov/libraries/india), details of monitoring stations in each city are provided in

Appendix II. Archived global reanalysis data at 2.5º resolution from National Oceanic and Atmospheric Administration (ftp://arlftp.arlhq.noaa.gov/archives/reanalysis/RP) was used for meteorological input, provided at 17 pressure levels. The analysis period, 2015-2016 was divided into four seasons, viz. December-February (Winter), March-May (Pre-monsoon), June-August (Monsoon) and September-November (Post-monsoon). $PM_{2.5}$ was measured using tapered element oscillating microbalance (TEOM) and beta attenuation methods (CPCB, 2011). The monitoring sites were chosen such that air is free flowing and inlet is away from source & other interferences. The height of inlet was greater than 3 m and samplers were at least 2 m apart (CPCB, 2011). Figure 3.2 indicated high annual average $PM_{2.5}$ concentrations in the cities located in the Indo-Gangetic plains: Delhi, Lucknow, Patna and Kolkata as compared to in other parts of the country.

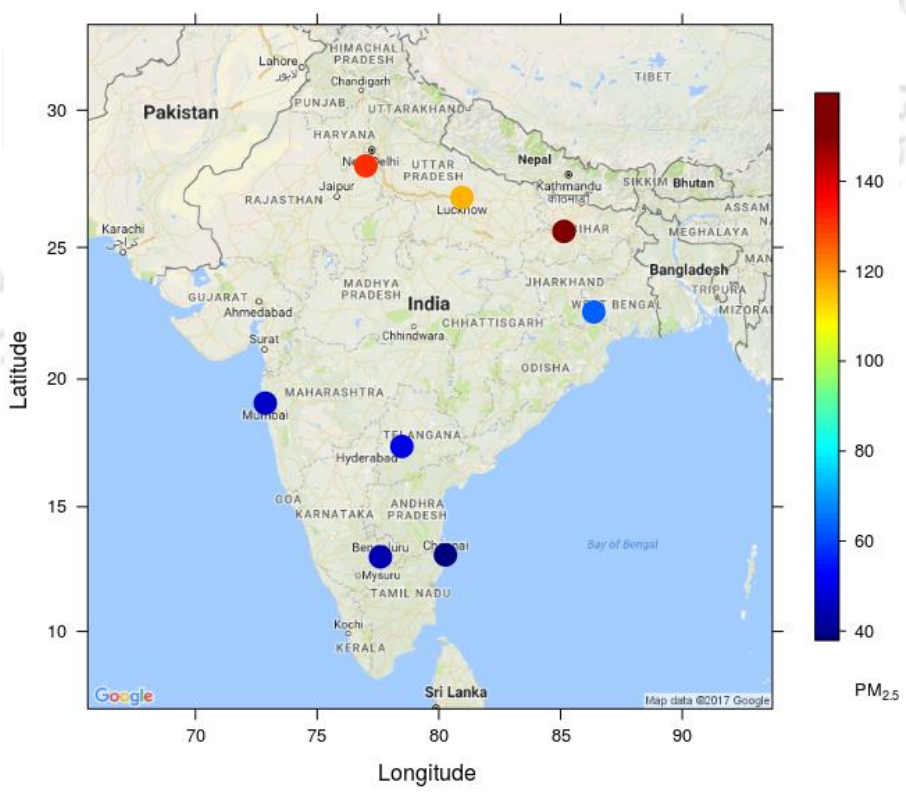

Figure 3. 2 Indian map showing the cities studied. Colour intensity shows the annual average $PM_{2.5}$ ($\mu g/m^3$) concentrations.

3.3.3 HYSPLIT back trajectory

The HYSPLIT model developed by National Oceanic and Atmospheric Administration (NOAA) was used to calculate air mass trajectories. The details of the model were explained in Draxier and Hess (1998) and only briefly mentioned here. This Lagrangian model uses a puff and particle approach to estimate the horizontal and vertical dispersion, respectively as explained in equations (3.8) and (3.9)

$$X_1(t + \Delta t) = X(t) + U(X, t)\,\Delta t \quad (3.8)$$

$$X(t + \Delta t) = X(t) + 0.5\,[\,U(X, t) + U(X_1, t + \Delta t)\,]\,\Delta t \quad (3.9)$$

where, X, X_1 and U denote initial, first guest positions and velocity vector, respectively.

The HYSPLIT model was run in R using Openair package (Carslaw & Ropkins, 2012; Team, 2016).

3.3.4 Cluster analysis

The idea of clustering involves reducing distance between a cluster centre and its points while maximizing the distance between two cluster centres to segregate or group objects with common features. Clustering reduces errors due to inaccurate meteorological data, interpolation and truncation by averaging out errors associated with individual trajectories (Stohl et al., 2002). This also enables location of origin and concentrations associated with trajectories aiding identification of high pollution regions. Cluster analysis in this study was carried out using openair package in R, which uses K-medoids clustering algorithm based on Euclidean distance between the trajectories to identify clusters. A medoid is defined as an object of a set or a cluster whose average dissimilarity to other objects in the set or cluster is same. K-medoids are more robust to noise and outliers as compared to K-means cause K-means select the cluster centre while K-medoids selects

the most centred member (Velmurugan & Santhanam, 2010). Partition Around Medoids (PAM), the most common realization of K-medoids (https://www.cs.umb.edu/cs738/pam1.pdf), was used in this study. The algorithm has two phases:

1. BUILD: In this k "centrally located" clusters are initially selected
2. SWAP: Improvement in quality of clustering is attempted in this step by swapping selected objects with unselected objects.

Optimum no of clusters was identified by conducting several non-parametric tests as suggested by Saikat Ghosh et al. (2015). Firstly, Kolmogorov-Smirnov test was conducted to identify if the cluster distribution was normal, in which a rejection of null hypothesis confirms non-normal distribution. Secondly, Kruskal-Wallis test was conducted to identify whether the clusters belonged to the same population, in which rejection of confirms that not all clusters were same. Finally, to identify which clusters were similar a pairwise Mann-Whitney test was conducted wherein rejection of the null hypothesis indicate that clusters differ.

3.3.5 IER model for risk estimation in Indian cities

As indicated in previous sections, IER model performed better as compared to the other existing models at high $PM_{2.5}$ concentration. However, it is imperative to validate the IER model in Indian cities. The health risk study in Delhi was used to compare with risk estimates of IER by studying the variation of mortality per 100000 population across different $PM_{2.5}$ exposure evaluated as per equation (3.10)

$$\Delta Mort = y_o[(RR-1)/RR]Pop \tag{3.10}$$

where: y_o refers to baseline mortality rate for a particular disease provided by WHO, RR is the relative risk and Pop refers to Total population.

3.3.6 Health risk associated with trajectories

Health risk of trajectories reaching the study area was calculated corresponding to the median $PM_{2.5}$ concentration of the trajectory. The health risk corresponding to a $PM_{2.5}$ concentration is usually calculated using equation (3.2) and (3.3) as provided in earlier section.

$$RR_i = \exp\left(\beta_i(c_i - c_{cf})\right), c_i > c_{cf}$$

$$\log(\mu_t) = \gamma + \beta C_t + \alpha K C_t + f(t)$$

However, in order to estimate β required to calculate risk using equation (3.3), we require mortality or health data which isn't readily available at all locations. Integrated risk function provided by (R. T. Burnett et al., 2014) and obtained by integrating available RR information from various studies can be used to calculate risk of Chronic Obstructive Pulmonary Disease (COPD), Lung Cancer (LC), Ischemic Heart Disease (IHD) and Stroke due to $PM_{2.5}$ concentration, as shown in equation (2.1) can be used in such cases.

For $c<c_{cf}$

$$RR_{IER}(c) = 1$$

For $c>=c_{cf}$

$$RR_{IER}(z) = 1 + \alpha\{1 - \exp\left[-\gamma\left(c - c_{cf}\right)^{\delta}\right]$$

A comparison of mortality obtained using IER with time series study in Delhi which indicated IER to provide good risk estimates have already been discussed in details in previous chapter and the same risk function has been used to estimate the risk and mortality corresponding $PM_{2.5}$ concentration in other cities.

3.3.7 Contribution to premature mortality in cities from states and neighbouring countries

Estimation of premature mortality in cities due to $PM_{2.5}$ concentration originating from states and neighbouring countries can help us understand the severity of pollution contributed from geographical source regions. Health risk estimation using risk function (explained above) requires $PM_{2.5}$ concentration as input, merging of observed $PM_{2.5}$ concentration data with HYSPLIT back trajectory run outputs provides the $PM_{2.5}$ concentration associated with location of trajectory endpoints. The $PM_{2.5}$ contribution to cities from states and neighbouring countries was then estimated using the equation (3.11) as given by (Cheng et al., 2013):

$$C_{cs} = C_s \times \frac{T_s}{T} \tag{3.11}$$

where C_{cs} indicates $PM_{2.5}$ concentration contribution to city c from state s; C_s indicates average $PM_{2.5}$ concentration associated with trajectories passing through state s; T_s indicates number of trajectory endpoints passing through or originating from state s and T indicates total number of back trajectory endpoints (Both T_s and T do not include trajectory endpoint at hour=0). The health risk corresponding to the location based $PM_{2.5}$ concentration is estimated using equation (2.1) provided above while the premature mortality is estimated using equation (3.10).

CHAPTER 4

RESULTS AND DISCUSSION

The chapter discusses the results obtained at various steps of the study. The results of the health risk study undertaken in Delhi and validation of the IER risk function at high $PM_{2.5}$ concentration using the results in Delhi have been discussed in details in the current chapter. Finally, geographical source region contribution to mortality due to $PM_{2.5}$ in Indian cities is discussed.

4.1 TIME SERIES EPIDEMIOLOGICAL STUDY IN DELHI

The current subsection discusses the results of epidemiological study in Delhi. A quick look at the variation of pollutant concentration and AQI at the study area reveals the status of air quality and the most dangerous pollutant at the site. The quantification of health risk due to the most dangerous pollutant and the benefit of its reduction to INAAQS and WHO standards are then discussed.

4.1.1 Seasonal, long term and effects of temperature on mortality

Fig. 4.1 indicates the monthly variation of $PM_{2.5}$ concentrations, mortality and temperature for the period 2011-2014 in Delhi. The trend clearly shows lower $PM_{2.5}$ concentration during monsoon while high $PM_{2.5}$ concentration during winter and post-monsoon. As can be observed from the plot that reduction in temperature results in increase of death and vice-versa, these are usually due to the deaths in extreme temperature such as deaths due to extreme cold. Likewise, there might be certain deaths due to diseases, which occurs in every season or in one or two year interval indicating seasonal and long-term effects. Since we want to estimate mortality only due to $PM_{2.5}$ concentration, we need to remove the deaths due to temperature, seasonal and long-term effects. The equation 3.3 used to estimate the relationship between $PM_{2.5}$ concentration and mortality uses a spline function of time f(t) to model and remove the seasonal and long term effects. The effect

of temperature is then modelled using categorical variables for deciles. The residual deaths were exposed to $PM_{2.5}$ concentration to evaluate the health effects.

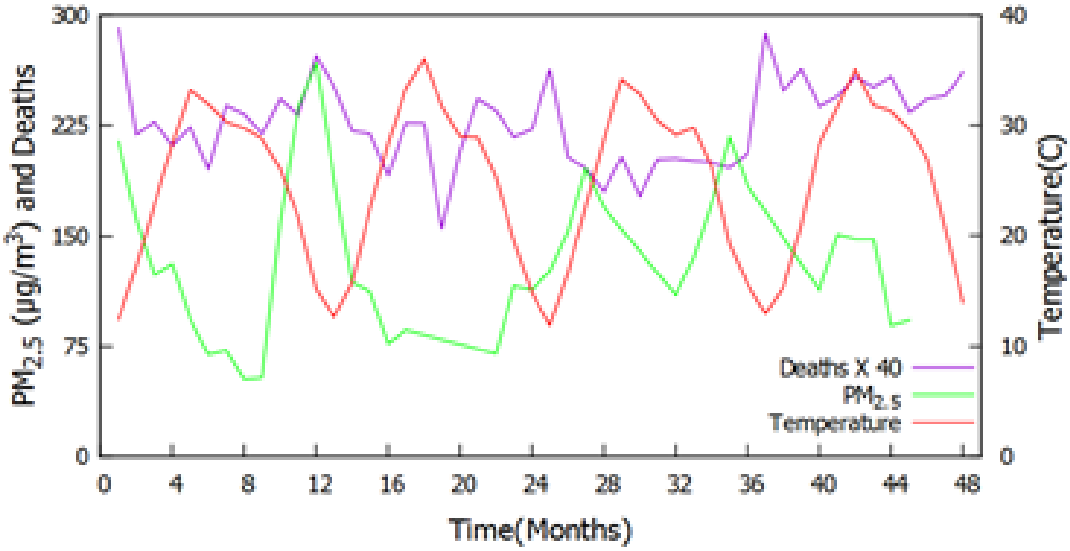

Figure 4. 1 Time series of monthly average $PM_{2.5}$ (µg m^{-3}) and deaths in left y-axis and temperature (℃) in right y-axis.

4.1.2 Variation of pollutant concentration at the study site

Since both AQI and health risk estimation are based on pollutant concentrations exceeding the safe limits, a study of yearly variation of pollutant concentration and its comparison with INAAQS gives us an overview of the current state of air pollution at the site. For comparison with INAAQS, the measured hourly data of $PM_{2.5}$, SO_2 and NO_2 were converted into daily averaged concentrations, and the hourly concentrations of O_3 and CO were used to estimate daily maximum 8 hour running average. Fig. 4.2 shows the change in concentrations of $PM_{2.5}$, SO_2, O_3, NO_2 and CO, from 2011 to 2014. The percentage of days for each pollutant exceeded INAAQS is also shown. Results indicate that in contrary to other pollutants, $PM_{2.5}$ had maximum median concentrations in 2013.

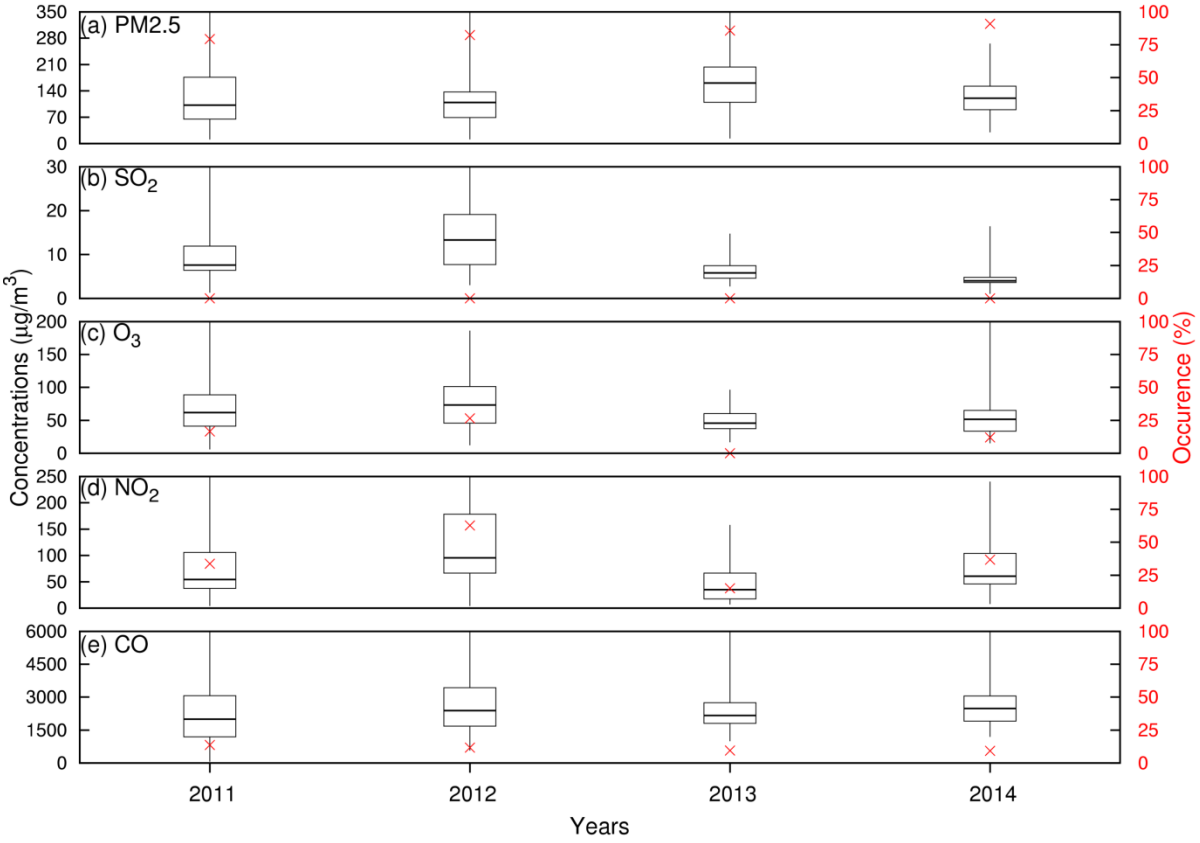

Figure 4. 2 Change in concentrations of CO, NO_2, O_3, SO_2 and $PM_{2.5}$ from 2011 to 2014 at New Delhi. Left Y axis indicates concentration; right Y-axis using red crosses represents percentage of data, which exceed the INAAQS. The horizontal lines of box indicate 25, 50 and 75 percentile while the whiskers indicate min and max values.

Panel (a) in Fig. 4.2 shows that median $PM_{2.5}$ concentrations in all the four years exceeded the corresponding INAAQS of 100 µg m^{-3}. $PM_{2.5}$ concentrations exceeded INAAQS during 80, 82, 86 and 90% of days in 2011, 2012, 2013 and 2014, respectively.

SO_2 concentrations never exceeded INAAQS in the four years, as observed from panel (b) of the figure. Similar conclusion was derived by Goyal and Sidhartha (2003) from their air quality analysis during 1996 to 2001 at New Delhi. This indicates that SO_2 may not be a major air pollutant in New Delhi. However, the contribution of SO_2 in the formation of secondary sulfate particles might be significant, and should be explored further. Panel (c) shows that O_3 concentrations varied

from 5 to 323 µg m^{-3} during the analysis period. O_3 concentrations exceeded the corresponding INAAQS of 100 µg m^{-3} for 16% in 2011, 26% in 2012, 0% in 2013, and 12% in 2014. Panel (d) shows that NO_2 concentrations exceeded the INAAQS of 80 µg m^{-3} limit 33.67% of the days in 2011, 62% of days in 2012, 15% of days in 2013, and 36% of days in 2014. CO concentrations reached a maximum of 10 mg m^{-3} during the period. Panel (e) shows that CO exceeded corresponding INAAQS value of 2 mg m^{-3}; 14%, 11%, 9.4% and 9.2% of days in 2011, 2012, 2013 and 2014, respectively. This implies that $PM_{2.5}$ is the major pollutant, followed by NO_2 and CO in New Delhi. However, more studies in Delhi where both PM_{10} and $PM_{2.5}$ concentrations are available are necessary in future to support this conclusion.

4.1.3 Variation of AQI over the years

A study of AQI variation over the years provides us an idea of air quality in the city. Fig. 4.3 indicates the yearly variation of frequency of days falling in the six IND-AQI categories, good, satisfactory, moderate, poor, very poor and severe, during 2011-2014. Due to the lack of availability of data of other pollutants only concentrations of $PM_{2.5}$, CO, NO_2, SO_2 and O_3 were used to estimate the IND-AQI in this study.

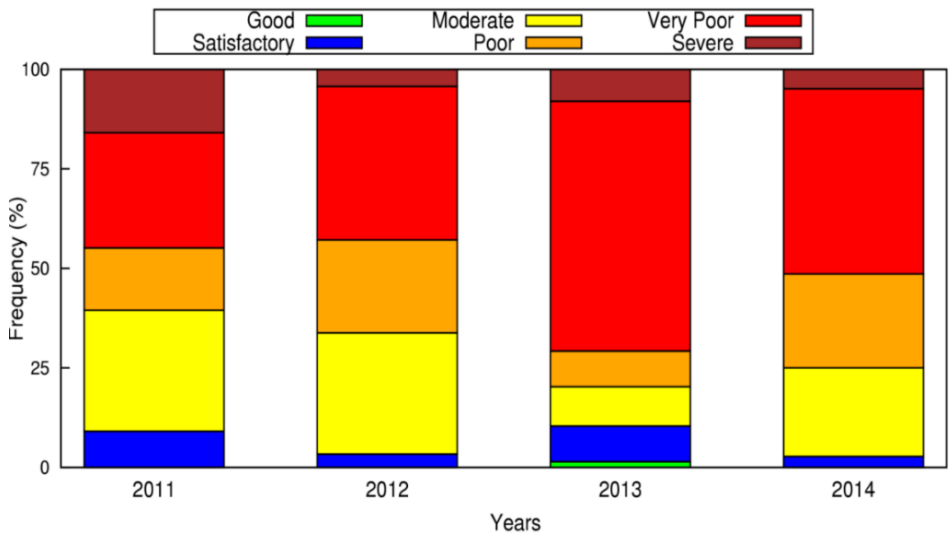

Figure 4. 3 Change in frequency of days (%) in various IND-AQI categories from 2011-2014.

The corresponding frequency of dominating species in each of those years is shown in Table 4.1. $PM_{2.5}$ was the dominant species in 75 to 90% of the days in those four years. Maximum dominance of NO_2 was observed during 2012 (17%), and O_3 during 2011 (8%).

Table 4. 1 Variation of domination of pollutants during 2011 to 2014

Year	SO_2	NO_2	$PM_{2.5}$	CO	O_3
2011	0	2	81	9	8
2012	0	17	75	4	4
2013	0	2	88	10	0
2014	0	0	90	9	1

While none of these years had days dominated by SO_2, CO dominance was around 9% in most of the years, except 2012. Zero dominant days of SO_2 is expected as it never violates INAAQS, as observed from Fig. 4.2. Fig. 4.3 indicates that at least 60% of the days in all the years were poor. Moreover, 70% of days in 2013 were either very poor or severe. In comparison, 44, 53 and 52% of such days exist in 2011, 2012 and 2014, respectively. This could be due to higher $PM_{2.5}$ concentrations in 2013 as observed in Figure 4.2. Only 6% of the days in all the four years were either good or satisfactory.

Previous studies have shown that meteorological parameters have significant effect on pollutant concentrations, For e.g higher concentrations of poly aromatic hydrocarbons, sulfates and nitrates in $PM_{2.5}$ in New Delhi (Pant et al., 2015), have a strong co-relation with temperature and RH (Y.

Wang et al., 2005). Additionally, studies in this region showed a strong co-relation of reactive pollutants like O_3, with temperature and RH (Gaur et al., 2014). To explore this, seasonal differences in IND-AQI and dominant species as a function of relative humidity (RH) and temperature were studied as shown in Figure 4.4. The seasons were categorized as winter (December-February), pre-monsoon (March-May), monsoon (June-August) and post-monsoon (September-November). Analysis indicates that concentrations of all the pollutants decreased during monsoon. This is due to wet scavenging of pollutants due to higher precipitation during that season.

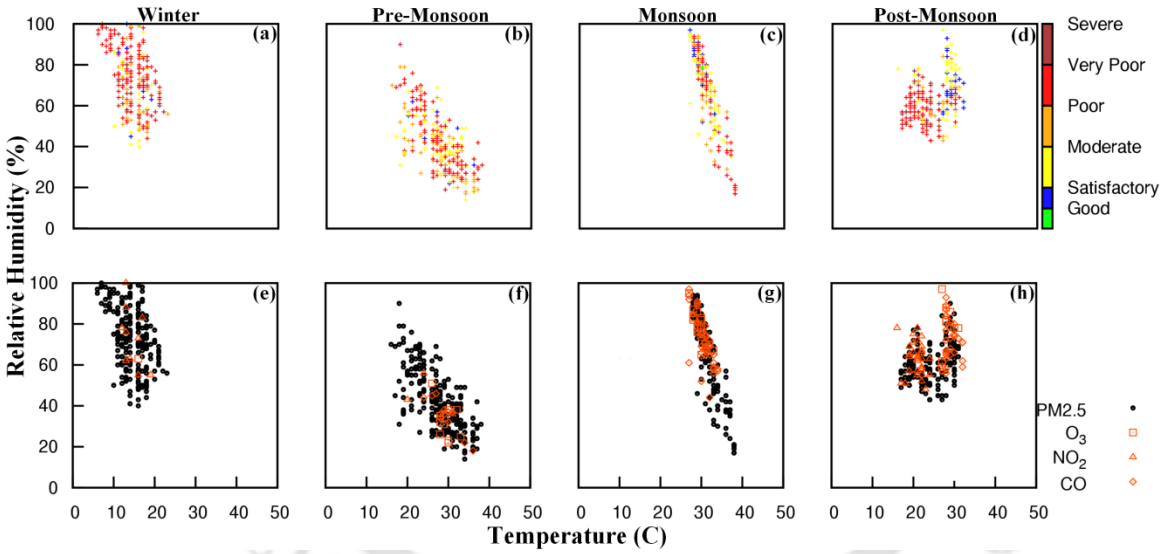

Figure 4. 4 Scatter plot of relative humidity versus temperature as a function of IND-AQI (panels a-d), and dominant species (panels e-h), for different seasons; winter, pre-monsoon, monsoon and post-monsoon.

Panels (a) to (d), indicate that winter had highest, around 72%, and monsoon had least, 32%, very poor and severe days. Panels (e) to (h) show a clear decreasing trend of $PM_{2.5}$ dominance from winter to post-monsoon. While, $PM_{2.5}$ was dominant in 95% of days in winter, it dominated IND-AQI during 68 and 70% of days of monsoon and post-monsoon seasons, respectively. Moreover, CO dominated during 18% of days in monsoon, and both CO and NO_2 dominated post-monsoon

by around 13-14% of days each. Previous studies have indicated that in addition to wet deposition, variation in atmospheric mixing layer heights, air flow pattern can also be a main reason for seasonal differences of pollutant concentrations in this region (Bisht et al., 2015; L. K. Sahu et al., 2009). To examine further, 24-hr back trajectories, originating from the sampling location, were generated using National Oceanic and Atmospheric Administration's Hybrid Single Particle Lagrangian Integrated Trajectory (HYSPLIT) model and the results of the same have been provided in Appendix A.

4.1.4 Health risk estimation

To estimate the potential risk associated with the concentrations of a pollutant, relative risk associated with a pollutant was calculated using equation 3.2. Estimated increase in all-cause mortality, was 0.69%(95% CI: 0.17%, 1.21%), 0.88%(95% CI: 0.14%, 1.63%), and 3.77%(95% CI: -0.6%, 8.34%) per 10 μg m^{-3} increase of $PM_{2.5}$, NO_2, and 1 mg m^{-3} CO, respectively. Non-significant risk factors were obtained for SO_2 and Ozone. This clearly indicates again that $PM_{2.5}$ is associated with major health risk in Delhi.

Table 4.2 shows the % increase in all-cause mortality per 10 μg m^{-3} increase in $PM_{2.5}$ concentration estimated by different studies around the world. In comparison with other major countries, no such studies were done in India. The exposure-response relationship co-efficient for $PM_{2.5}$ is similar in major cities in the world. This value lies in the range of global estimate by R W Atkinson et al. (2014), and closer to several studies in China (R. Chen et al., 2011; Dai et al., 2004) and the US (Bart Ostro et al., 2006). The $PM_{2.5}$ concentration alongside the mortality-$PM_{2.5}$ relationship co-efficient indicates lower $PM_{2.5}$ concentration is associated with higher co-efficient value. This is

in accordance to the findings by (Dholakia et al., 2014), who observed that relative health benefits of reducing pollution are higher for cleaner cities as opposed to in dirtier cities.

Table 4. 2 Increase in all-cause mortality (%) per 10 µg m^{-3}increase of $PM_{2.5}$ at different cities around the world

Place (Study Year)	**Reference**	**% increase in mortality per 10 µg/m^3 increase in $PM_{2.5}$ concentration**	**$PM_{2.5}$ concentration**
Global Review	(R W Atkinson et al., 2014)	1.04 (0.52-1.56)	-
USA (1979-1983)	(Klemm, 2003)	1.2 (0.8-1.6)	18.2
USA (1999-2005)	(Zanobetti & Schwartz, 2009)	0.98 (0.75-1.22)	5.6-24.9
USA (2000-2008)	(Levy et al., 2012)	1.2 (0.5-1.9)	-
California (1999-2002)	(Bart Ostro et al., 2006)	0.6 (0.2-1.0)	14-29
Australia (1996-1999)	(Simpson et al., 2005)	0.9 (-0.7-2.5)	7.5-9.0
Beijing (2007-2008)	(R. Chen et al., 2011)	0.53 (0.37-0.69)	82±52
Shenyang (2006-2008)	(R. Chen et al., 2011)	0.35 (0.17-0.53)	94±52
Shanghai (2004-2005)	(Huang et al., 2009)	0.30 (0.06-0.54)	56.4±1.34
Shanghai (2002-2003)	(Dai et al., 2004)	0.85 (0.32-1.89)	-
Xian (2004–2008)	(Huang et al., 2012)	0.20 (0.07-0.33)	176.7±103.8
New Delhi (2011-2014)	Current Study	0.69 (0.17-1.21)	133

The benefit of reducing $PM_{2.5}$ concentration to INAAQS and WHO standards were estimated using equation 3.4. Decrease of $PM_{2.5}$ concentrations from current level to 10 µg m^{-3} will result in a reduction of 8826 deaths in 2011 as provided in Table 4.3. This is similar to the estimated deaths by Sarath K. Guttikunda and Goel (2013) due to $PM_{2.5}$ in New Delhi as 7350-16200 in the year 2010. Table 4.3 also gives the projected reduction in all-cause mortality and number of reduction in non-accidental deaths due to decrease in current $PM_{2.5}$ concentration to the levels suggested by INAAQS and WHO. The averaged all-cause mortality rate data for the year 2011 to 2014 was obtained from statistical handbooks of Delhi during those years (DES, 2014) as 0.625%.

Results indicate that, when the current levels are reduced to meet INAAQS annual limits of $PM_{2.5}$ i.e. 40 µg m^{-3}, the all-cause mortality in New Delhi will be reduced by 6.20%, with 39 premature deaths avoided per 100000 people. Similarly, if the WHO levels of 35, 25, 15 and 10 µg m^{-3} of $PM_{2.5}$ are met, the number of premature deaths per 100000 people will be reduced by 41, 45, 49 and 51, respectively.

Table 4. 3 Projected reduction in all-cause mortality and corresponding reduction in deaths, due to proposed reduction in $PM_{2.5}$ concentrations for Delhi.

Proposed $PM_{2.5}$ limit (µg m^{-3})	**Reduction in all-cause mortality (%)**	**Reduction in deaths (per 100000 people)**	**Total reduction in deaths**
40	6.20	39	6749
35	6.52	41	7095
25	7.17	45	7788
15	7.80	49	8480
10	8.12	51	8826

4.2 RISK FUNCTION TO ESTIMATE HEALTH RISK

The health risk estimation discussed in the previous chapter was possible only due to the availability of health data records. Unlike in Delhi, other cities in India do not report health data hence similar health studies cannot be replicated in other cities. Risk functions are a viable option in such cases, however these risk functions are yet to be validated at high $PM_{2.5}$ concentrations which majority of the Indian cities record. The current subsection discusses the validation of IER function at high $PM_{2.5}$ concentration using Delhi study and also the variation of the health risks of four diseases IHD, Stroke, COPD and LC at 8 cities across India.

4.2.1 Validation of IER model for Indian cities

IER model estimates at high $PM_{2.5}$ concentration was based on risk estimates of SHS and AS studies and it was assumed that interpolation of risk estimates of AAP, SHS and AS will provide plausible risk estimates at high $PM_{2.5}$ concentration, which however is yet to be established. The health risk study in Delhi was used to compare with risk estimates of IER by studying the variation of mortality per 100000 population across different $PM_{2.5}$ exposure evaluated as per equation (3.10).

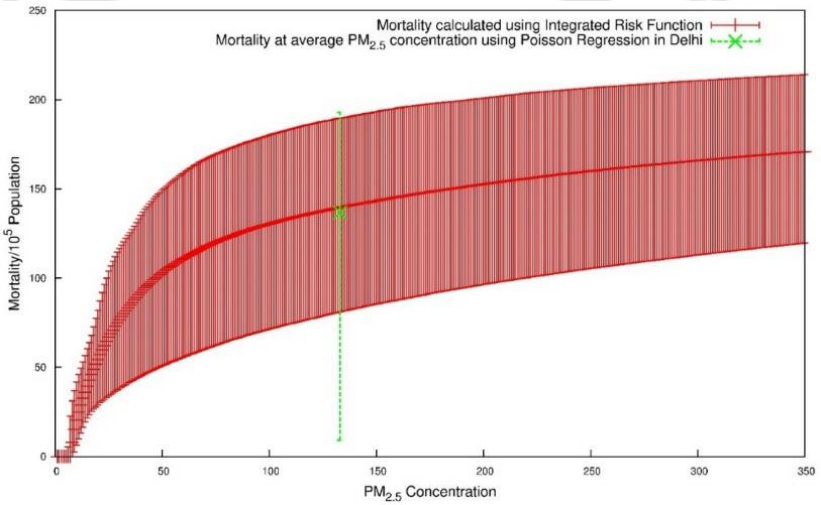

Figure 4. 5 Mortality estimated using IER and Poisson regression at Delhi

Fig 4.5 indicates a comparative study of mortality per 100000 population estimates using IER and Poisson regression in Delhi. The mid bars reflect the median estimate while the top and bottom ones reflect 97.5% and 2.5% confidence intervals (CI) in case of IER estimates indicated in red while the green ones are the estimates obtained using time series study in Delhi and the top and bottom bars in this indicate estimates obtained using 95% CI of risk co-efficient. IER was found to predict 2.14% more mortality per 100000 population than time series study in Delhi.

4.2.2 Variations of $PM_{2.5}$ and meteorological parameters in Indian cities

A study of variation of $PM_{2.5}$ and meteorological parameters in a city provides a brief overview of air quality in the city and its dependence on meteorology. The average concentrations in north, east, south and west were 125, 62, 46 and 44 $\mu g/m^3$ respectively. The ratio of winter to monsoon $PM_{2.5}$ concentrations in Delhi, Lucknow, Patna, Kolkata, Hyderabad, Bangalore, Chennai and Mumbai were 3.2, 6.9, 4.62, 3.64, 1.6, 1.8, 0.7 and 3.1, respectively. In addition to emissions, $PM_{2.5}$ concentrations in a city depend on meteorology. Fig 4.6 shows the seasonal variations of temperature and RH in the selected cities in India. The northern and eastern India experience extreme climatic conditions, i.e. cold winters and very hot summers, while western and southern parts are usually hot and humid with cool winters and hot summers. For example, in Delhi and Chennai, the differences between median temperatures in winter and pre-monsoon were 130℃ and 50℃, respectively. Such differences in meteorological parameters could also be a reason for the wide variations of $PM_{2.5}$ concentrations across the country.

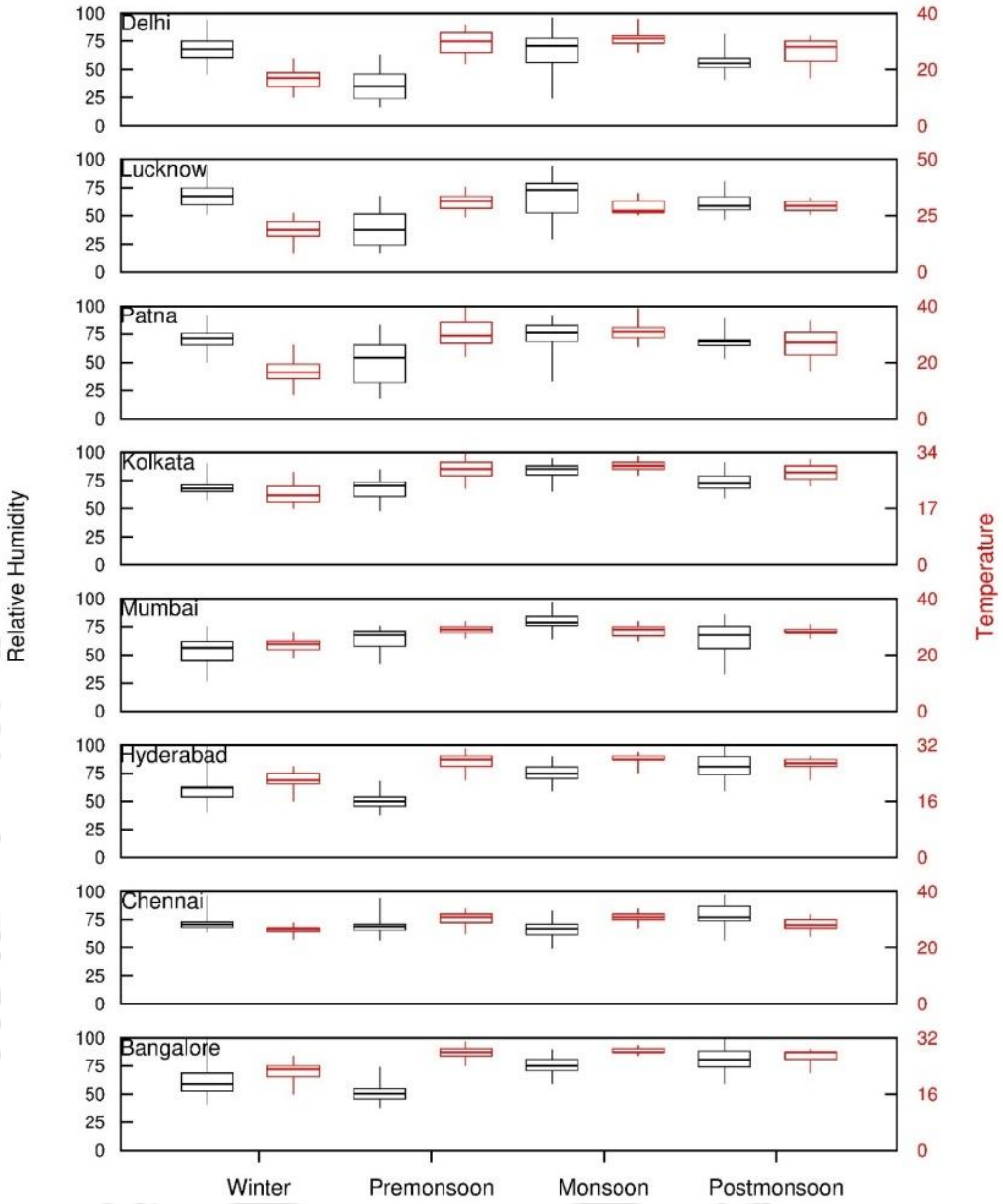

Figure 4. 6 Change in RH and Temperature in each season for each city. The horizontal lines of box indicate 25, 50 and 75 percentile while the whiskers indicate min and max values with the left Y-axis representing RH values and right Y-axis representing temperature.

Table 4.4 indicates the mean, median, minimum, maximum and number of days of $PM_{2.5}$ concentration data available at the selected cities. $PM_{2.5}$ concentrations were greater in cities in north (208 µg/m^3) and east (184 µg/m^3) as compared to in other cities. Winter was observed to be the season with highest $PM_{2.5}$ (126.61 µg/m^3) concentration followed by post-monsoon (86 µg/m^3). Wet deposition in monsoon resulted in low $PM_{2.5}$ concentration in all cities except Chennai.

Table 4. 4 Seasonal statistics of $PM_{2.5}$ concentrations ($\mu g/m^3$) for each city.

City	Statistics	Winter	Pre-Monsoon	Monsoon	Post-Monsoon
Delhi	Average	209.49	112	65.88	148.57
	Median	197.21	94.33	55.59	117.17
	Min	23.17	12.33	10.50	11
	Max	547.73	513.67	368	547.83
	Count	80	56	90	88
Lucknow	Average	206.19	100	29.70	127.66
	Median	187.58	93.17	29.53	81.21
	Min	3.34	17.23	1.15	1.30
	Max	560.61	500.66	88.91	584.63
	Count	89	62	76	76
Patna	Average	233.13	81.12	50.4	172.2
	Median	202	68	49	151
	Min	47.67	8	4	30
	Max	705	546	117	603
	Count	79	90	87	80
Kolkata	Average	135.4	49.44	17.23	68.7
	Median	120	40	15	56.5
	Min	7	5	1	2
	Max	427	205	72	383
	Count	68	66	54	42
Mumbai	Average	61.68	30.93	37.80	53
	Median	58.44	25.57	36.51	47.85
	Min	2.25	0.01	3.75	4.55
	Max	144.13	144.11	117.18	140.55
	Count	62	90	89	88
Hyderabad	Average	58.44	45.78	31.93	59.94
	Median	54.72	43	27	50
	Min	9	11	8	2
	Max	176	163	176.67	175.50
	Count	79	81	89	78
Chennai	Average	39.46	32.80	51.64	31.93
	Median	36.44	28.94	48.49	28.02
	Min	4	2.1	1.1	1.40
	Max	145.77	139.41	146.62	149.30
	Count	70	88	90	89
Bangalore	Average	69.15	53.07	21.75	32
	Median	66.88	44.79	20.02	25.23
	Min	3.9	4.5	1.96	1.7
	Max	194.17	188.68	145.06	170.16
	Count	89	79	70	80

Seasonal variation of highly polluted, i.e. top 10 percentile hours, at selected cities is shown in Fig 4.7 Moving from north to south, the percentage of highly polluted hours falling in winter and post-monsoon decreases. Highly polluted hours in winter and post-monsoon at north, east, west and south were 96, 90, 94 and 64 %, respectively. In Chennai, the percentage of highly polluted hours in monsoon was higher than winter.

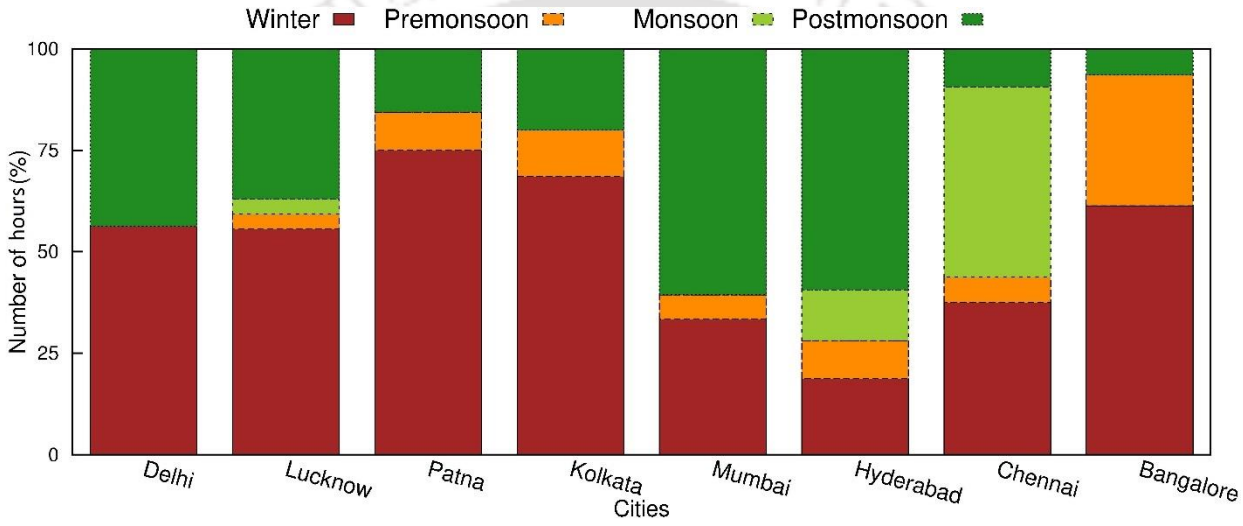

Figure 4. 7 Number of hours (%) associated with top 10% $PM_{2.5}$ concentrations in each city in each season.

4.2.3 Risk estimation in cities across India

In order to estimate the risks in cities across India, IER function was used. Risk was estimated in eight cities: New Delhi and Lucknow (North), Patna and Kolkata (East), Mumbai (West) and Bangalore, Chennai and Hyderabad (South). Figures of health risk in four different seasons viz. Winter (Dec-Feb), Pre-monsoon (Mar-May), Monsoon (Jun-Aug) and Post-Monsoon (Sep-Nov) was evaluated and is depicted in Fig 4.8.

Health risk in eight cities in different seasons indicated higher COPD, LC, IHD and Stroke risks in the Northern (COPD=1.35, LC=1.50, IHD=1.39, Stroke=2.06) and Eastern cities (COPD=1.27, LC=1.38, IHD=1.35, Stroke=1.93) as compared to in Southern (COPD=1.17, LC=1.22, IHD=1.28, Stroke=1.54) or Western cities (COPD=1.18, LC=1.24, IHD=1.28, Stroke=1.59). Risk due to Stroke in northern and eastern cities during winter and post-monsoon were observed to be 1.17 and 1.41 times to that of in southern cities. Risk of Stroke was observed to be the highest: North= 1.37-1.52, South =1.20-1.31, East=1.40-1.52 and West=1.24-1.35 times to that of other diseases. COPD (1.24) and IHD (1.32) risks were observed to be similar in all cities. The median risks in Delhi, Lucknow and Patna were observed to be similar during winter (COPD=1.60, LC=1.90, IHD=1.49, STROKE=2.15) across all diseases with Patna being the city experiencing the highest health risk during the same period. Patna is observed to have higher risk during winter (1.01 times) and post-monsoon (1.011 times) as compared to Delhi while it is vice versa during monsoon and pre-monsoon.

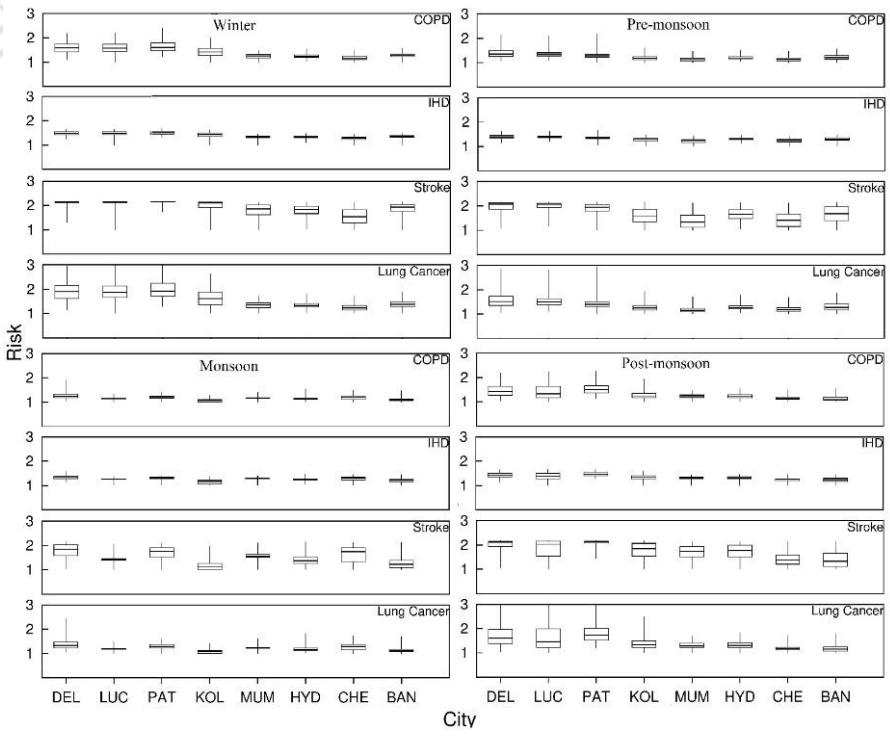

Figure 4. 8 Health risk variation associated with clusters reaching the 8 cities during four seasons. The horizontal lines of box indicate 25, 50 and 75 percentile while the whiskers indicate min and max values

4.3 GEOGRAPHICAL SOURCE REGION CONTRIBUTION TO MORTALITY IN CITIES

The health risk estimated in the cities due to high $PM_{2.5}$ concentration in the previous section can be tackled only if the geographical source region of these high risk areas are identified and appropriate control strategies are implemented. The current subsection discusses identification of geographical source regions using HYSPLIT back trajectory clusters and proportional residence time of the trajectories in a region. It also discusses the sensitivity of HYSPLIT back trajectories on model input. The source region contribution to mortality in Indian cities due to $PM_{2.5}$ is also discussed in the current subsection.

4.3.1 Sensitivity of HYSPLIT back trajectories on model inputs

The two main inputs required to run HYSPLIT are:

1. Starting height of trajectory 2. Meteorological data

Starting height affects possible end points of the trajectory. The starting height changes throughout the trajectory depending on meteorological parameters. This starting height is an assumed height of the station in the model. The degree of similarity between the model and actual height depends on the resolution of topographical features. For e.g.: a model having outputs at 1 degree resolution would mean that all the topographical features in that 1 degree grid will have exact same height. Many a times in case of a hill being present it tends to get flattened out, hence a suitable starting height must be chosen to include in the height factor of the hill. Trajectories with very low starting height may hit the ground early and thus would result in information loss. On contrary, very high starting height could result in trajectories not entering the mixing layer. Optimum height for

starting a trajectory would be the middle of planetary boundary layer, which keeps varying. Since, starting height of trajectories is only provided at the beginning of the analysis, selection of starting heights are usually done based on previous studies. Many previous studies have pointed out that 500 m is the optimum height within which the mixing layer height is usually found (Dimitriou & Kassomenos, 2016). Since the conditions within the mixing layer height is assumed to be well mixed hence the output provided by HYSPLIT at 500m should be similar to that at ground level (Stein et al., 2015). Table 4.5 lists out the studies along with the starting height of the trajectories. Most of the studies either used 100, 500 or 1000 m as starting height during their simulation. Thus, the current study also performed analysis at starting heights of 100, 500 and 1000 m.

Table 4. 5 HYSPLIT studies and the starting height used to obtain the trajectories.

Region	Study	Trajectory height (meters)	Type of study
		India	
	(Kulshrestha et al., 2009)	500	Regional
	(Cherian et al., 2010)	10, 100, 500 and 1000	Local and regional
	(Datta et al., 2010)	500	Local and regional
	(Giri et al., 2013)	500, 1000 and 1500	Local and regional
	(Kumar & Kumari, 2015)	300, 500 and 700	Local and regional
	(Sandeep et al., 2013)	500	Regional
	(Pavuluri et al., 2010)	500	Regional
	(V. Kumar et al., 2013)	100	Local and regional
		World	
	(Saliba et al., 2007)	500	Local and regional
	(Vardoulakis & Kassomenos, 2008)	100, 500 and 1000	Regional
	(Herbert et al., 2005)	100, 500 and 1000	Local and regional
	(Pekney et al., 2006)	500	Local and regional
	(N. Liu et al., 2011)	500	Local and regional
	(Gildemeister et al., 2007)	500	Local and regional
	(Dimitriou & Kassomenos, 2016)	500	Local and regional
	(X. Xu & Akhtar, 2009)	500	Local and regional
	(S. Yu et al., 2014)	100	Local and regional

Fig 4.9 shows that CWT plots for the northern and eastern cities using 100, 500 and 1000 m as trajectory starting height while plots for western and southern cities are provided in Appendix-II.

The CWT plots at 100, 500 and 1000 m are observed to be similar barring the spatial extent of the trajectories. This could be the reason for opting 500 m as starting height by most of the studies in past as observed from Table 4.5. Thus, even though analysis was carried out for all three trajectory heights, only results from 72 hours back trajectory simulation with 500 m starting height is shown.

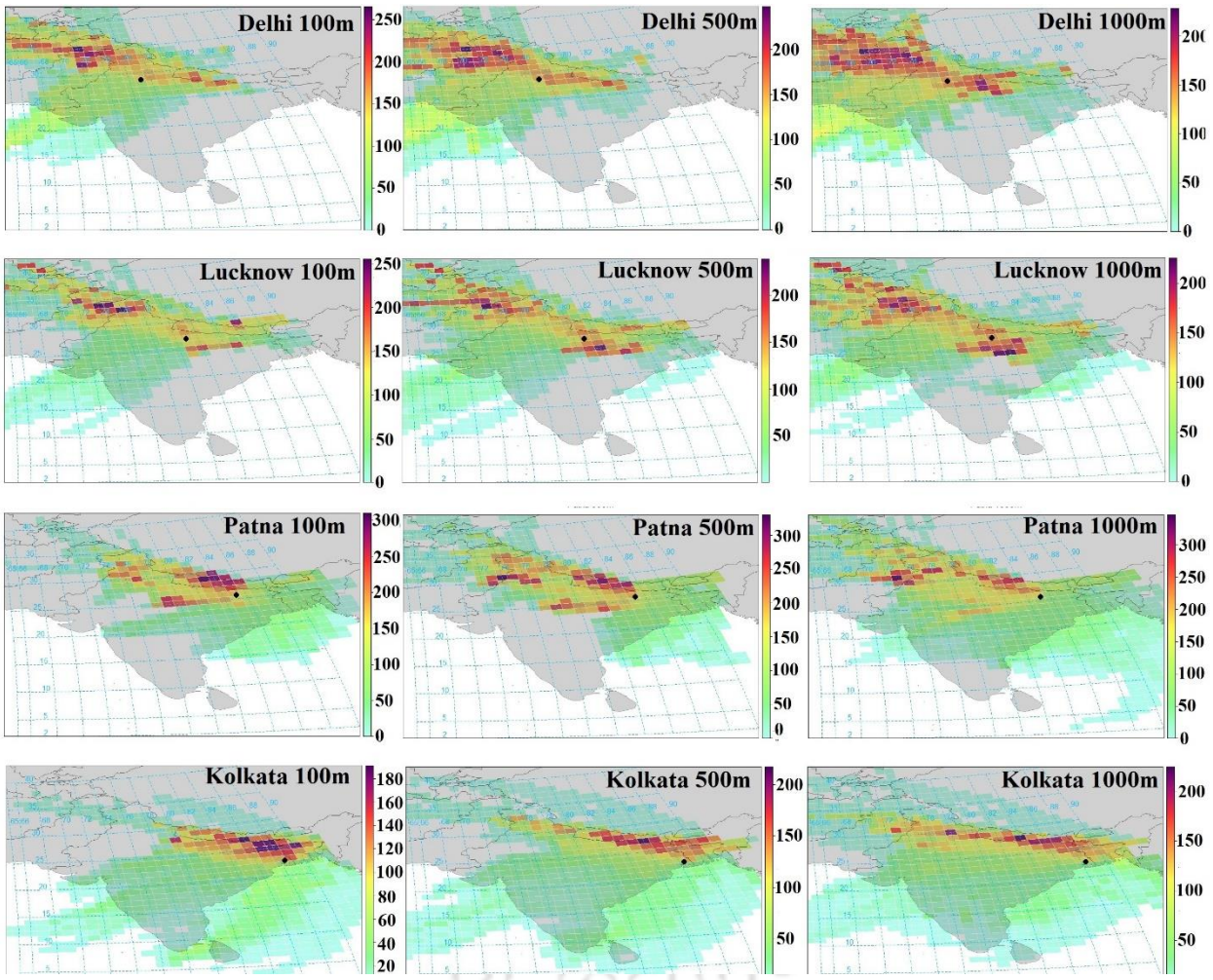

Figure 4. 9 CWT plots of northern and eastern cities at different heights

Meteorological data is an important input required for HYSPLIT as it provides wind speed and direction, which are critical in determining the direction and extent of trajectories. The archived meteorological data usually used in HYSPLIT are either analysis or reanalysis data. Atmospheric

data usually consists of observation data recorded at weather station as well as predictions provided by the forecasting models. These predicted and observed data is assimilated using a data assimilation scheme. Reanalysis data provide a multivariate, spatially complete, and coherent record of the global atmospheric circulation which unlike analysis data, is produced with a single version of a data assimilation system including the forecasting method and hence is not affected by changes in method (Dee et al., 2011). The different meteorological data currently available in ARL format that can be used as an input to HYSPLIT are provided in Table 4.6.

Table 4. 6 Meteorological data available in ARL format to be used as input in HYSPLIT.

Meteorological data	**Resolution**	**Extent**	**Data Availability**	**Vertical layers**
EDAS	40 KM	US	JAN 2004-present	26
NAMS	12km, 12km & 2km	US, Alaska & Hawaii	MAR 2010-present	40
GDAS1	1°	Global	DEC 2004-present	23
GDAS0.5	0.5°	Global	SEP 2007-present	23
NCEP/NCAR Reanalysis	2.5°	Global	1948-present	17
HRRR	3 km	US	JUN 2015-present	36
NAM	12 km	US	MAY 2007-present	26
NAR Reanalysis	32 km	US	1979-present	23

From the above data sets, it is clear that only GDAS1°, GDAS0.5° and NCEP/NCAR Reanalysis can be used as inputs to our HYSPLIT analysis in India for the year 2015. HYSPLIT outputs with GDAS0.5° (uses Global Forecast System (GFS) meteorological model and Global Data Assimilation System (GDAS)) and NCEP/NCAR Reanalysis (uses Climate Data Assimilation

System, CDAS) as meteorological inputs were compared in different cities for different seasons, a few of which are shown in Fig 4.10 while the rest are included in Appendix-II. It was observed that barring few cases, both meteorological data sources provided similar outputs. Since, reanalysis data are quality checked and homogenous throughout, it was selected for meteorological inputs in the current study.

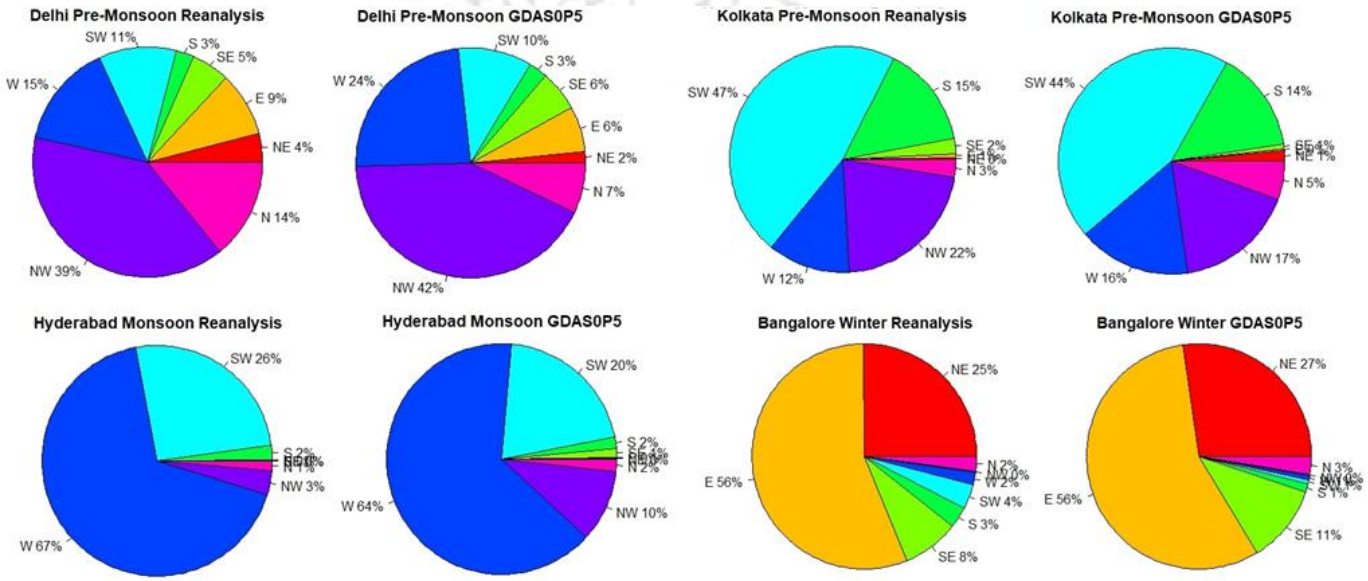

Figure 4. 10 % Wind direction in each sector of HYSPLIT outputs using NCEP/NCAR Reanalysis and GDAS0.5° meteorological data

4.3.2 Trajectory cluster to identify its origin

Clustering of back trajectories is an effective method to track its origin. Fig 4.11 indicates the origin of trajectory clusters while Figure 4.12 indicates the concentration associated with the clusters in winter. Table 4.7 indicates the $PM_{2.5}$ contribution (>10%) from Indian states and neighbouring countries to selected Indian cities during winter. Table and plots for other seasons are provided in Appendix-II. Analysis using polar plots were also done to corroborate our results from cluster analysis, the polar plots and it's subsequent results have been provided in Appendix-III to avoid repetition of the results (similar to cluster analysis). A correlation analysis was also

carried out to identify the co-relation of $PM_{2.5}$ with other pollutants and meteorology in order to determine the possible sources responsible for high $PM_{2.5}$ concentration in the cities. The results of the analysis have been provided in Appendix-III since extraction of any meaningful results from the analysis required further study thus causing deviation from the main objective of estimating source region contributions to $PM_{2.5}$ concentration in Indian cities.

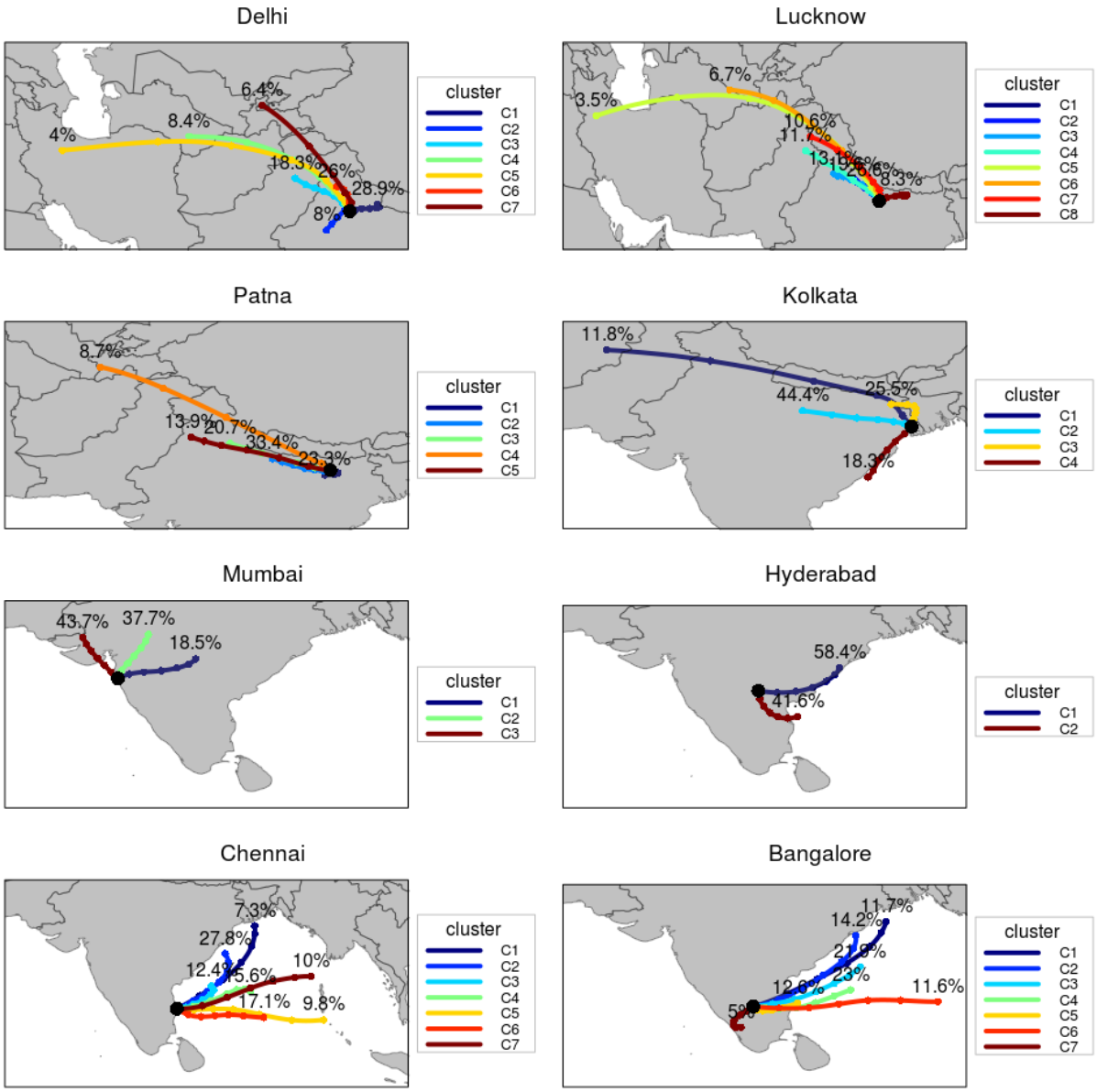

Figure 4. 11 Optimum no. of clusters in each city during winter

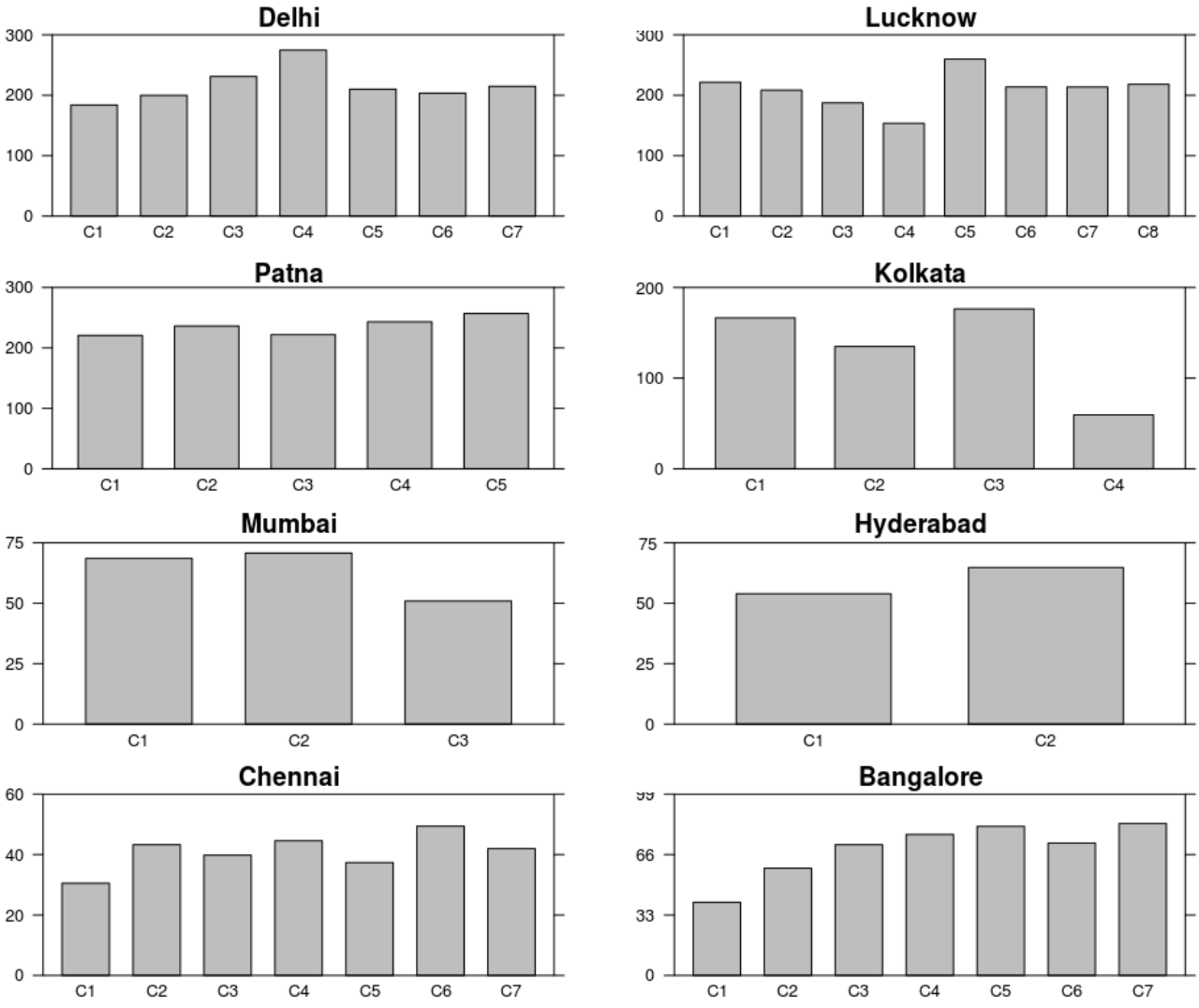

Figure 4. 12 Average $PM_{2.5}$ ($\mu g/m^3$) concentration associated with each cluster for each city in winter

Table 4. 7 % $PM_{2.5}$ concentration contributed to the city from Indian states and neighbouring countries in winter

Delhi	**UP**	**HAR**	**PUN**	**PAK**
% Contribution	31.387	18.671	13.609	10.365
Lucknow	**UP**			
% Contribution	69.681			
Patna	**UP**	**BHR**		
% Contribution	44.273	35.294		
Kolkata	**WB**	**BAN**	**JHR**	**ODS**
% Contribution	31.906	12.850	10.710	9.724
Mumbai	**MAH**	**GUJ**	**IO**	**MP**
% Contribution	32.145	25.833	23.229	10.694
Hyderabad	**IO**	**TEL**	**AP**	
% Contribution	32.417	24.722	22.670	
Chennai	**IO**			
% Contribution	98.860			
Bangalore	**IO**	**TN**	**KAR**	
% Contribution	56.949	16.613	14.958	

4.3.2.1 *North India*

In Delhi during winter 63% of the trajectories originated in the north-west. Irrespective of their region of origin, all trajectory clusters were associated with $PM_{2.5}$ concentrations of above 190 µg/m^3. Table 4.7 indicates Uttar Pradesh (31%), Punjab (18.67%), Haryana (13.61%) and Pakistan (10.36%) being the source locations of high $PM_{2.5}$ concentration affecting Delhi during winter. During pre-monsoon, the majority of the trajectories still originated from north-west but were associated with lower average $PM_{2.5}$ concentrations (above 90 µg/m^3) compared to winter with Punjab (21%), Haryana (24.40%), Pakistan (11.57%) and Rajasthan (10.5%) being the primary hotspots. Monsoon indicated a change in trend of trajectory clusters with majority (64%) of them originating in south-west and the clusters were associated with much lower average $PM_{2.5}$ concentrations of 65 µg/m^3 with Rajasthan (30.44%), Haryana (19%) and Indian Ocean (12.36%)

being the regions of high $PM_{2.5}$ concentrations. Trajectories in post-monsoon were more scattered than in other seasons: North-West (41%), East (31%) and South-West (27.7%). Uttar Pradesh (24.17%), Punjab (15.39%), Haryana (21.17%) and Pakistan (14.03%) were observed to be the $PM_{2.5}$ hotspots.

Trajectory clusters in Lucknow were similar to that in Delhi in all seasons with 93 and 94% of trajectories originating from north-west during winter and pre-monsoon respectively. Short trajectory clusters (55%) with average $PM_{2.5}$ concentration of above 190 $\mu g/m^3$ in winter and 80 $\mu g/m^3$ in pre-monsoon indicated dominance of local sources during the period. Uttar Pradesh was observed to be the major contributor to $PM_{2.5}$ concentration in winter (70%) and pre-monsoon (60.5%). In monsoon, 26% of the trajectories originated from south-west while trajectories in post-monsoon were evenly spread out (46% from northwest followed by 11% from south-west, 17% from east and 26.4% from north). Trajectory clusters originating during monsoon were associated with $PM_{2.5}$ concentration below the NAAQS limits of 60 $\mu g/m^3$ while during post-monsoon, shorter trajectory clusters C3 and C4 passing through U.P. and Nepal were observed to be associated with concentrations above 120 $\mu g/m^3$ during post-monsoon. Uttar Pradesh was observed to be a major hotspot (>43%) in all seasons indicating possibility of dominant local sources.

4.3.2.2 *East India*

Patna was observed to be affected by sources in north-west during winter with all trajectories being associated with average $PM_{2.5}$ concentrations of above 200 $\mu g/m^3$ and possible $PM_{2.5}$ hotspots in Bihar (35.29%) and Uttar Pradesh (44.27%). During pre-monsoon, shorter cluster C1 traversing through Bihar and Uttar Pradesh was the only one associated with $PM_{2.5}$ concentrations of above

100 µg/m^3 thus indicating possible source of high $PM_{2.5}$ concentration affecting Patna to be located in the same. Trajectories during monsoon were observed to be associated with $PM_{2.5}$ concentrations below the NAAQS limits of 60 µg/m^3. During post-monsoon, short clusters covering Uttar Pradesh and Bihar were observed to be associated with $PM_{2.5}$ concentrations above 120 µg/m^3. Bihar and Uttar Pradesh were observed to be the major contributor to $PM_{2.5}$ concentration (>62%) in Patna in all seasons except monsoon.

In Kolkata during winter, shorter clusters C2 and C3 comprising of 70% of the total trajectories were associate with average $PM_{2.5}$ concentrations of above 130 µg/m^3 and West Bengal (32%), Bangladesh (13%) and Jharkhand (10.71%) were the principal sources of high $PM_{2.5}$ concentration observed in the city during the period. During pre-monsoon, clusters C2 and C3 (constituting 36% of all trajectories) passing through West Bengal, Uttar Pradesh and Jharkhand having average $PM_{2.5}$ concentration above 70 µg/m^3 were found to be the probable $PM_{2.5}$ source locations. During monsoon, none of the clusters had $PM_{2.5}$ concentrations above 60 µg/m^3 indicating possible wet scavenging. In post-monsoon shorter trajectories C2 & C3 comprising of 44% of total trajectories were associated with average $PM_{2.5}$ concentrations of above 70 µg/m^3 with West Bengal (38.6%), Bangladesh (17.56%) and Jharkhand (12.78%) being the prime contributors to high $PM_{2.5}$ concentration observed in Patna.

4.3.2.3 *West India*

In Mumbai, winter and post-monsoon were the only seasons where clusters associated with $PM_{2.5}$ concentrations above NAAQS limits of 60 µg/m3 were observed. Maharashtra (32.14%), Madhya Pradesh (10.694%), Gujrat (25.83%) and Indian Ocean (23.23%) during winter while Maharashtra (42%) and Indian Ocean (42%) during post-monsoon were observed to be the prominent hotspots

of $PM_{2.5}$ concentration affecting Mumbai. Pre-monsoon and Monsoon were observed to be associated with clusters primarily originating from Ocean (82.55% and 99.47%) which might be the reason behind low $PM_{2.5}$ concentration being associated with the same.

4.3.2.4 *South India*

In Hyderabad, similar to Mumbai no clusters in pre-monsoon and monsoon were associated with $PM_{2.5}$ concentrations above 60 µg/m^3. Shorter Cluster C2 passing through Andhra Pradesh and Telangana in winter were associated with $PM_{2.5}$ concentration of above 60 µg/m^3, contributing to 24.72% and 22.67% of $PM_{2.5}$ concentration respectively in Hyderabad. During post-monsoon only cluster C1 (40% of trajectories) was associated with average $PM_{2.5}$ concentrations above 60 µg/m^3, Telangana (26.69%), Maharashtra (10.31%) and Andhra Pradesh (14.85%) being the primary contributors to high $PM_{2.5}$ concentration observed in Hyderabad during the same period.

In Bangalore during winter and pre-monsoon, short trajectory clusters passing through Karnataka and Tamil Nadu were observed to be associated with $PM_{2.5}$ concentrations above 70 and 60 µg/m^3 respectively, Tami Nadu and Karnataka contributed to 16.61% and 15% of high $PM_{2.5}$ concentration observed in Bangalore during winter while 22.7% and 21.64% of $PM_{2.5}$ concentration during pre-monsoon. None of the clusters in monsoon and post-monsoon were associated with $PM_{2.5}$ concentrations above 60 µg/m^3 which can be attributed to large percentage of trajectories originating from Indian Ocean (80% and 61% respectively).

In Chennai $PM_{2.5}$ concentrations in monsoon (51.64 µg/m^3) were observed to be higher as compared to that in winter (39.46 µg/m^3). Apart from cluster C2 in monsoon with higher trajectory contribution from landmass (34%) and lowest from Indian Ocean (66.6%) observed in during the same season, none of the clusters in any other season were associated with average $PM_{2.5}$

concentration above 60 µg/m^3. Indian Ocean contributed above 66% of the total trajectories reaching Chennai in all seasons with contribution in winter being above 98%.

4.3.3 Premature mortality contribution in cities from states and neighbouring countries

Contribution to premature mortality in cities from Indian states and neighbouring countries responsible for 10% or greater contribution to $PM_{2.5}$ concentration in the respective cities during winter is shown in Fig 4.13.

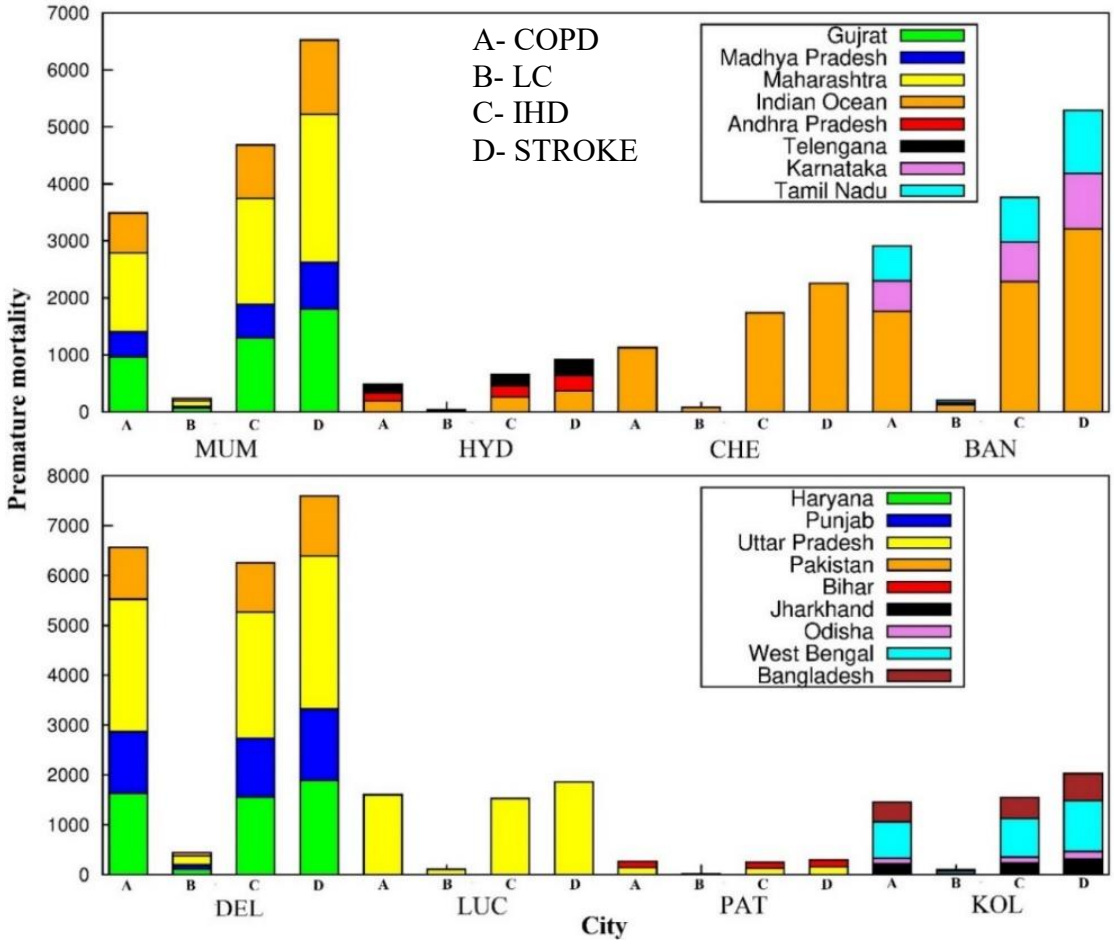

Figure 4. 13 Pre-mature mortality contribution from Indian states and neighboring countries during winter

Uttar Pradesh contributed to 30% of the total premature mortality in Delhi with 2653 premature mortality due to COPD, 2528 due to IHD, 3069 due to stroke and 181 due to lung cancer. Punjab and Haryana accounting for 33% of total premature mortality in Delhi due to high $PM_{2.5}$ concentration and were responsible for 2872 deaths due to COPD, 196 deaths due to lung cancer, 2737 and 3323 deaths due to IHD and stroke respectively. Meanwhile high $PM_{2.5}$ concentration from Pakistan resulted in 1043 premature mortality due to COPD, 994 due to IHD, 1207 due to stroke and 71 due to lung cancer. Premature mortality in Lucknow was primarily contributed by high $PM_{2.5}$ concentration from Uttar Pradesh, which was responsible for 1604 deaths due to COPD, 1530 due to IHD, 1860 due to stroke and 109 due to lung cancer.

Bihar and Uttar Pradesh were the primary contributors to premature mortality in Patna responsible for 79% of total mortality including 267 deaths due to COPD, 248 due to IHD, 296 due to stroke and 18 due to lung cancer. Kolkata was affected by high $PM_{2.5}$ concentration originating from Jharkhand, Orissa, West Bengal and Bangladesh which accounted for 67% of total deaths. West Bengal accounted for majority of the premature deaths (33%) due to $PM_{2.5}$ concentration with 724 deaths due to COPD, 773 due to IHD, 1013 due to stroke and 50 due to lung cancer. Bangladesh' Jharkhand and Odisha accounted for 18%, 10% and 5% of total premature mortality respectively.

Maharashtra and Gujrat accounted for 61% of total premature mortality due to high $PM_{2.5}$ concentration in Mumbai with 2351 deaths due to COPD, 3152 due to IHD, 4395 due to stroke and 161 due to lung cancer. Indian Ocean too accounted for a significant portion of premature deaths (18.5%) due to $PM_{2.5}$ concentration and was responsible for 2991 total deaths.

$PM_{2.5}$ concentration from Andhra Pradesh and Telangana were observed to be responsible for 291 deaths due to COPD, 397 due to IHD, 553 due to stroke and 20 due to lung cancer in Hyderabad,

accounting for 49% of total premature mortality. Large contribution of high $PM_{2.5}$ concentration from Karnataka and Tamil Nadu resulted in 35% of total premature mortality due to air pollution in Bangalore, which included 1142 deaths due to COPD, 1478 due to IHD, 2076 due to stroke and 79 due to lung cancer. Chennai was observed to be highly influenced by marine pollution form Indian Ocean, which contributed to 99% of total deaths due to air pollution resulting in 1129 deaths due to COPD, 1743 due to IHD, 2260 due to stroke and 77 due to lung cancer. Additionally Indian Ocean was observed as a major contributor to premature mortality due to high $PM_{2.5}$ concentration in all the cities in southern Indian resulting in 32% of premature deaths in Hyderabad and 54% in Bangalore.

4.4 SUMMARY

4.4.1 Time series epidemiological study in Delhi

The study analysed data collected at a busy traffic junction during 2011 to 2014 at New Delhi, to determine the most dangerous pollutant in New Delhi. AQI was used to identify the dominant pollutant while a health risk study quantified the deaths due to high concentration of the pollutant. Investigation showed that $PM_{2.5}$ was the dominant pollutant, during all the seasons, with dominant days being 24% higher in winter and pre-monsoon, than monsoon and post-monsoon. Moreover, significant differences in very poor and severe days were not observed on weekdays and weekends, with $PM_{2.5}$ being the dominant pollutant in all days. However, this conclusion might vary if PM_{10} data is included in the analysis, and more studies are needed to explore this further. Additional investigation revealed that, this could be due to long range transport of air pollutants from different regions. This shows that air quality in New Delhi can be ameliorated only due to better policies in neighbouring states.

The potential health risk associated with $PM_{2.5}$ was greater than CO and NO_2. The excessive risk per µg m^{-3} associated with $PM_{2.5}$, NO_2 and CO were obtained as 0.57 (95% CI: 0.45, 0.69), 0.36 (95% CI: 0.24, 0.46), and 0.052 (95% CI: 0.046, 0.058), respectively. This is in agreement with the AQI method. Finally, the impact of reducing the current $PM_{2.5}$ concentrations, the most dominant pollutant in New Delhi was investigated. Results indicated that 39 and 41 premature deaths can be avoided per 100000 by bringing down the yearly averaged concentrations of $PM_{2.5}$ to the levels suggested by INAAQS and WHO, respectively.

4.4.2 Risk function to estimate health risk

Linearly increasing risk function resulted in implausible health risk estimates at high $PM_{2.5}$ concentrations mostly because risk estimates from developing countries were not taken into account while formulating the risk function. Cardiovascular disease risk estimates were observed to be increasing steeply with $PM_{2.5}$ concentrations initially and then flattens out at high $PM_{2.5}$ concentrations while Lung cancer risk estimates were observed to be monotonically increasing throughout the exposure range. IER was observed as the best model to predict risk estimates at high $PM_{2.5}$ concentrations and was found to only slightly over predict mortality by 2.14% per 100000 population as compared to time series study in Delhi. Risk due to Stroke in northern and eastern cities during winter and post-monsoon were observed to be 1.17 and 1.41 times to that of in southern cities. COPD (1.24) and IHD (1.32) risks were observed to be similar in all cities. Delhi, Patna and Lucknow were observed to have similar risks (COPD=1.60, LC=1.90, IHD=1.49, STROKE=2.15) during winter. Risk due to Stroke was observed to be highest (North= 1.37-1.52, South =1.20-1.31, East=1.40-1.52 and West=1.24-1.35) as compared to other diseases.

4.4.3 Geographical source region contribution to mortality in cities

Analysis of air mass back trajectories was done to identify the regions from which trajectories with high $PM_{2.5}$ concentration and associated health risks originated in different seasons in 8 cities across India. $PM_{2.5}$ concentrations were the highest during winter in all cities except Chennai (<60 $\mu g/m^3$). The higher concentrations in Chennai during monsoon (51 $\mu g/m^3$ average) may be due to higher percentage of trajectories originating from landmass (33%) as compared to in other season. Apart from Delhi, other cities in northern and eastern India had high contribution of trajectories from their home state with highest contribution in winter (>32%) and lowest in monsoon (>10.75%). All the cities in Indo-Gangetic plain had higher $PM_{2.5}$ concentrations (>80 $\mu g/m^3$) and associated health risks as compared to other cities. Kolkata and Mumbai were observed to have $PM_{2.5}$ concentrations below the NAAQS limit during monsoon (17.23 $\mu g/m^3$ and 38 $\mu g/m^3$ respectively) and pre-monsoon (49.44 $\mu g/m^3$ and 31 $\mu g/m^3$ respectively), due to higher percentage of trajectories from Indian Ocean (40 and 90% on average respectively) as compared to in other seasons. Chennai, Hyderabad and Bangalore were the cities which were majorly affected by marine sources with bulk of trajectories (at least 27%) from Indian Ocean in three seasons (pre-monsoon, monsoon, post-monsoon) and hence experience lower concentrations compared to cities in other parts of country. Uttar Pradesh was observed to be one of the significant source of $PM_{2.5}$ especially during winter and post-monsoon in Delhi (24.1% and 31.38%), Lucknow (58.51% and 70%) and Patna (44.23% and 54.21%). Lower $PM_{2.5}$ concentration during monsoon apart from wet deposition is also due to fact that higher percentage of trajectories in the season originate from Indian Ocean. Punjab, Haryana, Uttar Pradesh and Pakistan were observed to contribute to 75% of total premature mortality in Delhi during winter. A recent report by TERI (http://www.teriin.org/sites/default/files/2018-08/AQM-SA_0.pdf) has estimated contribution of

$PM_{2.5}$ from regional sources to total $PM_{2.5}$ in Delhi to be 64%. The current study estimates it to be 94%, a coarse grid resolution of 2.5 degree in the current study as opposed to the 4km grid resolution in the study by TERI might be the reason for over-estimation of regional contribution. Uttar Pradesh was observed to be a major contributor to premature mortality in Delhi, Lucknow and Patna accounting for 30%, 71% and 42% of total premature death due to high $PM_{2.5}$ concentration during winter. High $PM_{2.5}$ concentration from West Bengal and Bangladesh were responsible for 52% of total premature mortality in Kolkata. Indian Ocean was a major contributor to premature mortality due to $PM_{2.5}$ concentration in western and southern cities of Mumbai, Hyderabad, Bangalore and Chennai accounting for 18.5%, 32% 54% and 99% of total premature mortality during winter respectively.

CHAPTER 5

CONCLUSION AND FUTURE SCOPE

5.1 GENERAL

This chapter summarizes the major conclusions and key findings of the research work and provides future scope for the research.

5.2 MAJOR CONCLUSIONS

- $PM_{2.5}$ was observed to be the most dominant pollutant in Delhi, during all the seasons, with dominant days being 24% higher in winter and pre-monsoon, than monsoon and post-monsoon.
- No significant differences in $PM_{2.5}$ concentration was observed during weekdays and weekends with IND-AQI category of very poor and severe being 50% of weekends, in comparison to 52% of weekdays.
- Time series epidemiological study conducted in Delhi estimated increase in all-cause mortality in Delhi per 10 $\mu g/m^3$ increase in $PM_{2.5}$ concentration to be 0.69%(95% CI: 0.17%, 1.21%). The excessive risk per $\mu g/m^3$ increase in $PM_{2.5}$ concentration was evaluated to be 0.57.
- 39 and 51 premature deaths can be avoided in Delhi per 100000 population by bringing down the yearly averaged concentrations of $PM_{2.5}$ from current levels (133 $\mu g/m^3$) to the levels suggested by INAAQS (40 $\mu g/m^3$) and WHO (10 $\mu g/m^3$) respectively.
- The cities in Indo-Gangetic plain were observed to have higher annual average $PM_{2.5}$ concentrations (>80 $\mu g/m^3$) as compared to other cities in India. Large-scale seasonal

variations in $PM_{2.5}$ concentration was observed with winter to be the season with highest $PM_{2.5}$ (126.61 µg/m^3) concentration followed by post-monsoon (86 µg/m^3) while lowest $PM_{2.5}$ concentration (38.29 µg/m^3) was observed during monsoon.

- Integrated Exposure Risk function (IER) was found to slightly over predict mortality in Delhi by 2.14% per 100000 population as compared to the risk estimates obtained from the time series epidemiological study in Delhi.
- Health risk in eight cities in different seasons indicated higher COPD, LC, IHD and Stroke risks in the Northern (COPD=1.35, LC=1.50, IHD=1.39, Stroke=2.06) and Eastern cities (COPD=1.27, LC=1.38, IHD=1.35, Stroke=1.93) as compared to in Southern or Western cities.
- Risk of Stroke was observed to be the highest: North= 1.37-1.52, South =1.20-1.31, East=1.40-1.52 and West=1.24-1.35 times to that of other diseases.
- The risk estimates of COPD (1.40) and IHD (1.39) were observed to be similar across the 8 Indian cities.
- Punjab, Haryana, Uttar Pradesh and Pakistan were observed to contribute majorly to $PM_{2.5}$ concentration in Delhi accounting for 75% of total premature mortality due to $PM_{2.5}$ in the city.
- Uttar Pradesh was observed to be a major contributor to $PM_{2.5}$ concentration and associated premature mortality in Delhi (30%), Lucknow (71%) and Patna (42%) during winter.
- West Bengal and Bangladesh were responsible for 52% of total premature mortality in Kolkata.

- Chennai, Hyderabad and Bangalore were majorly affected by marine sources with bulk of trajectories (at least 27%) from Indian Ocean in three seasons (pre-monsoon, monsoon, post-monsoon) and hence experience lower $PM_{2.5}$ concentrations compared to other cities.
- Reduction of both local and regional pollution are required to yield significant reduction in premature mortality due to $PM_{2.5}$ concentration of all cities except Delhi and Lucknow where regional and local sources respectively are dominant.

5.3 FUTURE SCOPE

- Cohort studies are required to be carried out in India to check whether flattening of the health risk vs concentration curve in case of cardiovascular studies takes place at high $PM_{2.5}$ concentration as depicted by IER.
- More epidemiological studies are required to be carried out in various cities across India to reduce the uncertainties in Risk functions.
- Variable baseline mortality rates for states based on difference in per capita expenditure on health must be calculated to obtain better estimation of premature mortality.
- Risk functions similar to that for $PM_{2.5}$ are required to be made available for all other pollutants as well.
- Emission inventories are required to be developed in order to evaluate the pollutant concentrations using Eulerian models.

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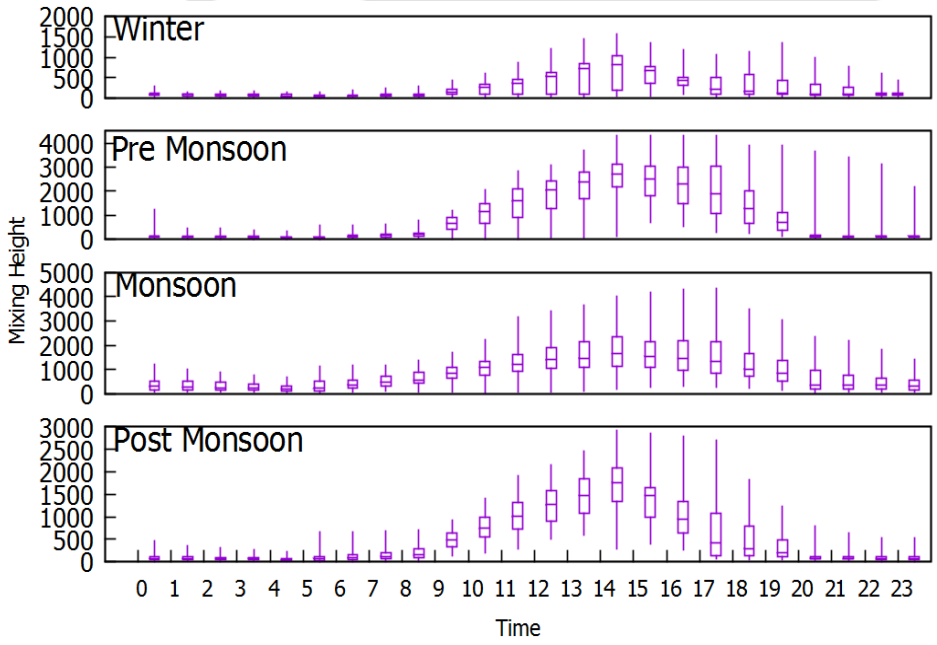
APPENDIX-I

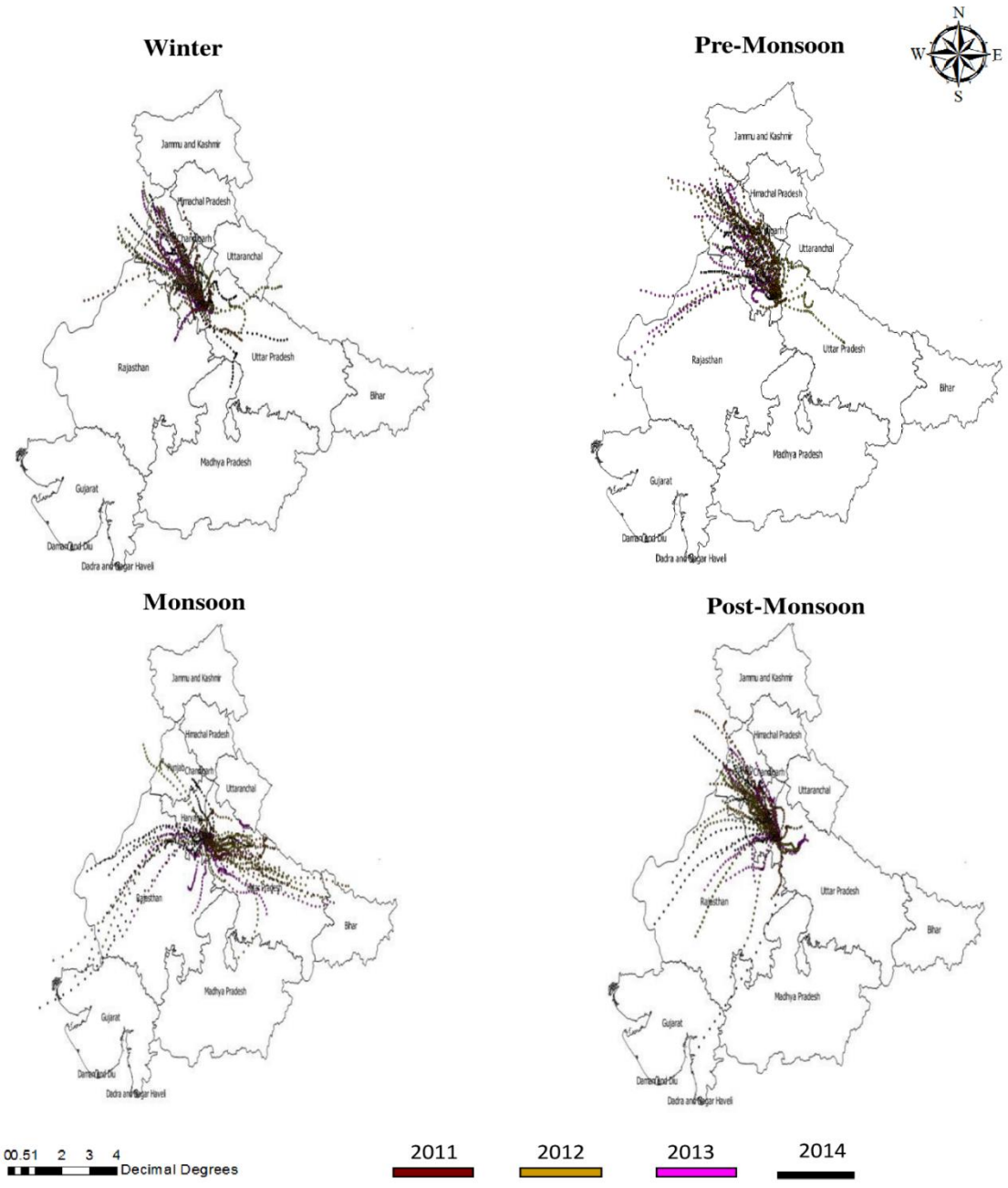
Analysis indicated that mixing layer heights peak in the afternoon, around 2 to 3 PM, with the least daily maximum mixing layer heights during winter.



Mixing Height vs Time of day in different seasons.

Results of back trajectory analysis show a clear seasonal pattern in air mass back trajectories in New Delhi. During winter and pre-monsoon, when more very poor and severe days are observed,

air parcels might have originated in Punjab and Haryana, home of many coal-based power plants and industries. In contrary, air parcels could have originated in Rajasthan and Uttar Pradesh during monsoon, and Rajasthan and Punjab in post-monsoon. Thus, the seasonal differences and the negligible weekday-weekend difference observed in this study could be due to long range transport of air pollutants. Similar conclusions were arrived from a recent study (Saikat Ghosh et al., 2015), which predicted significant influence of neighboring states in $PM_{2.5}$ concentrations, during 2008 to 2010, in New Delhi.

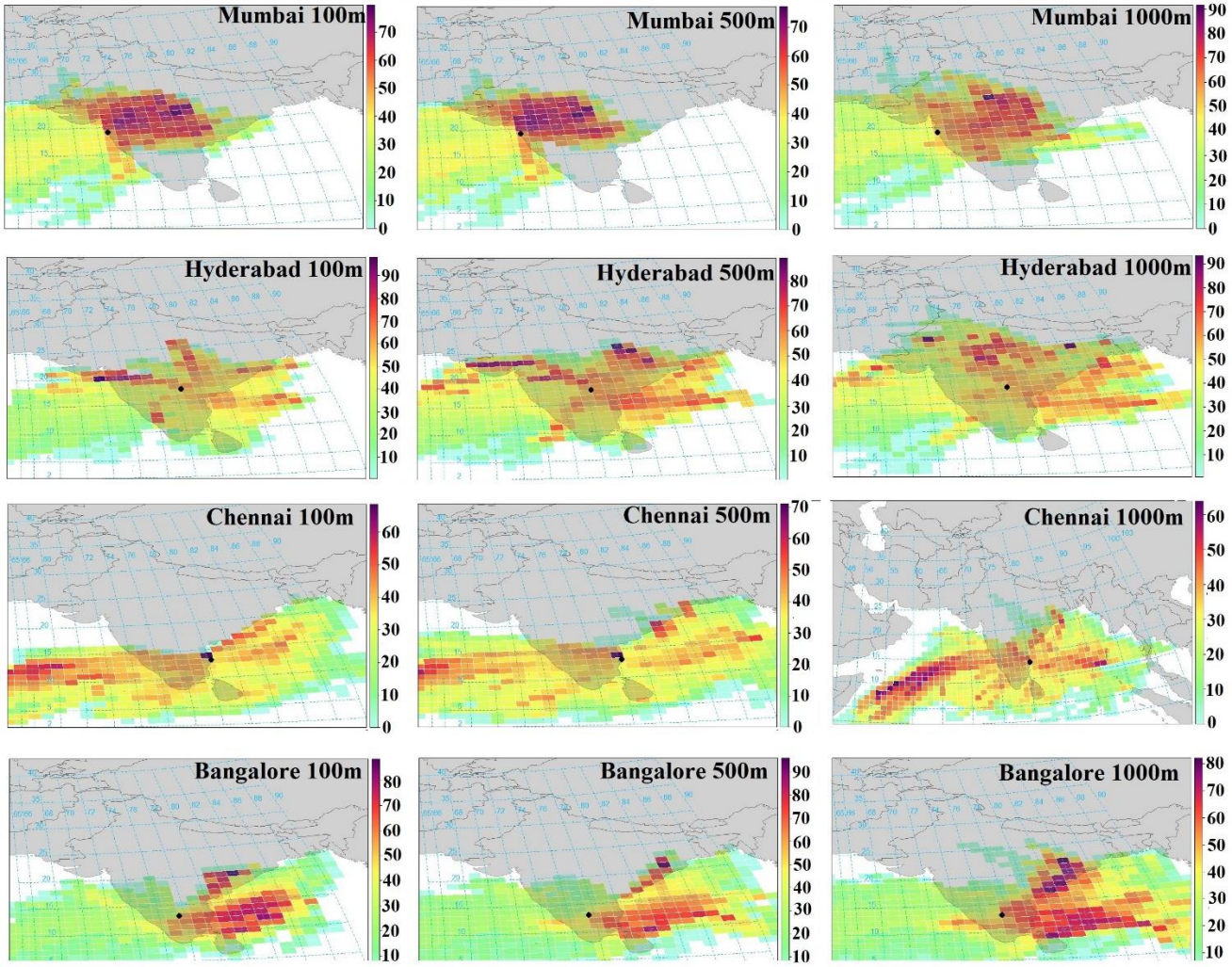


Back Trajectory plots showing long range transport of pollutants from different states to Delhi in different seasons.

APPENDIX-II

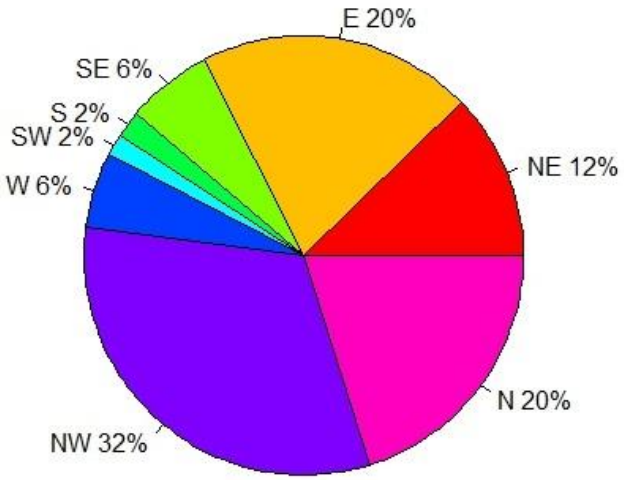
Table indicating the monitoring location name in each city and it's type in the 8 Indian cities

Region	City	Station Name	Location Type
North	Delhi	RK Puram	Residential
	Lucknow	Central School	Commercial
East	Patna	IGSC Planetarium Complex	Commercial
	Kolkata	US Consulate	Commercial
West	Mumbai	Bandra	Residential
	Hyderabad	Hyderabad	Commercial
South	Tamil Nadu	Alandur	Commercial
	Karnataka	Peenya	Industrial

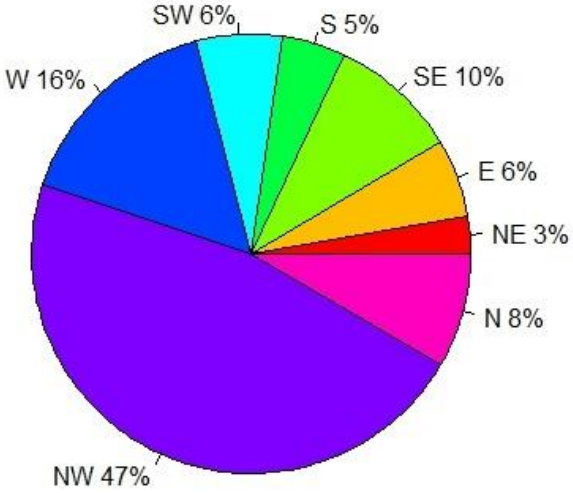


CWT plots of western and southern cities at different heights

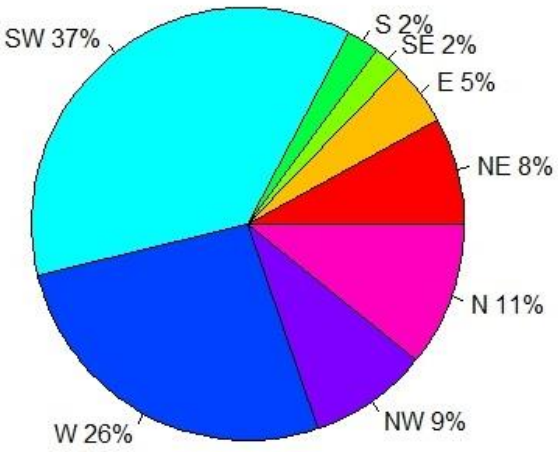
Delhi winter Reanalysis



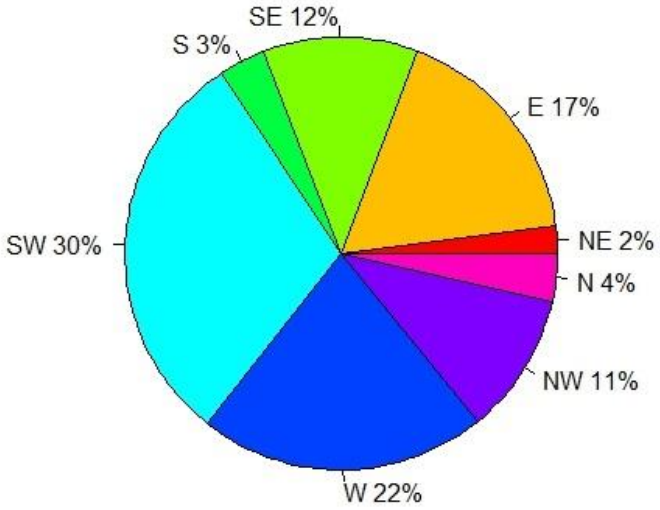
Delhi Winter GDAS0P5



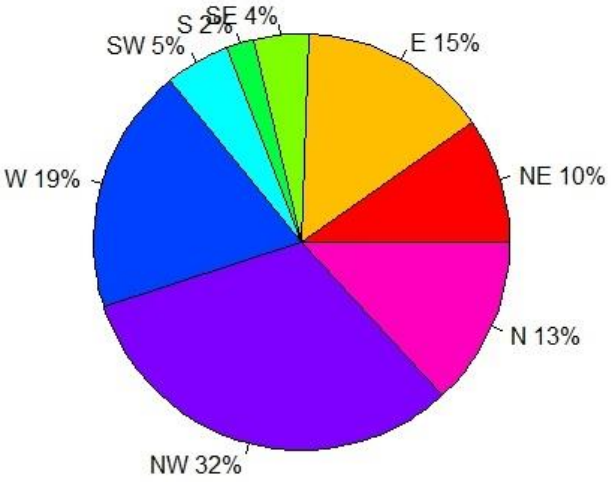
Delhi Monsoon Reanalysis



Delhi Monsoon GDAS0P5



Delhi Post-Monsoon Reanalysis



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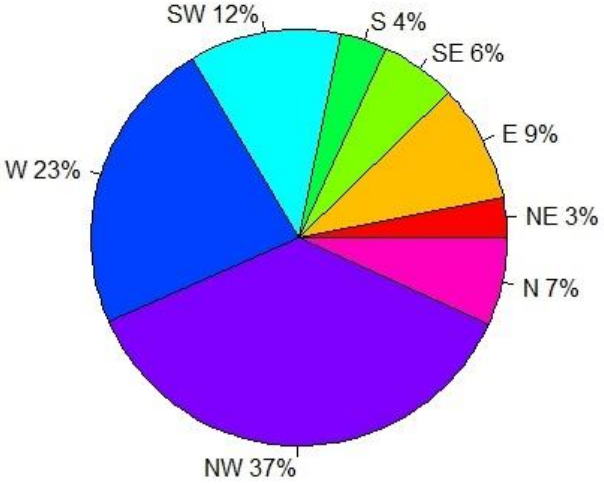
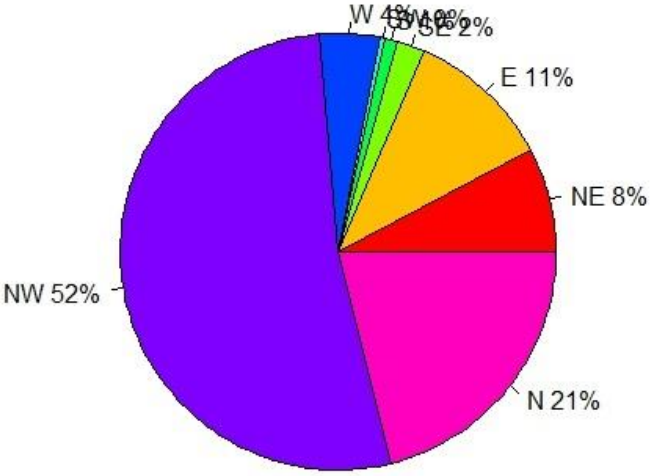


Lucknow Winter Reanalysis



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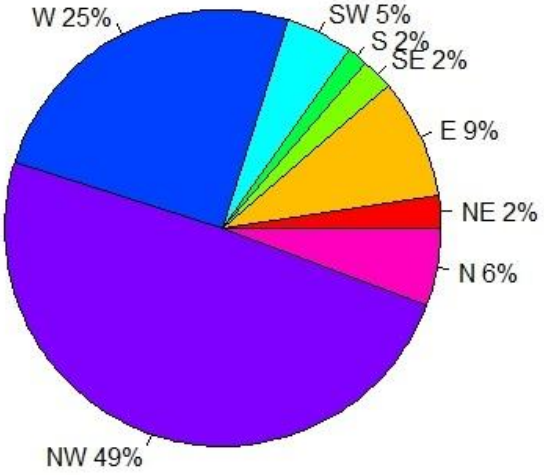

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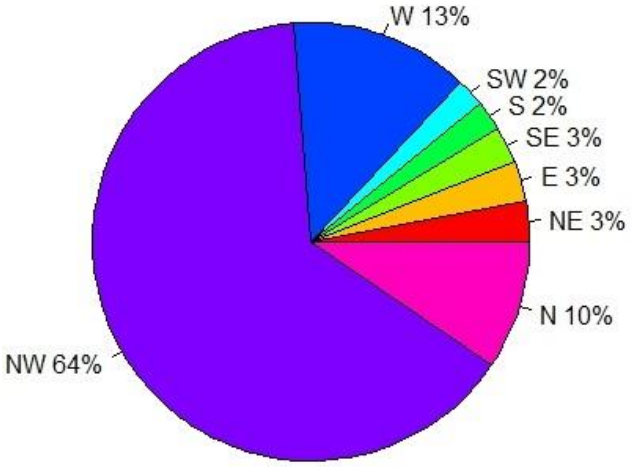

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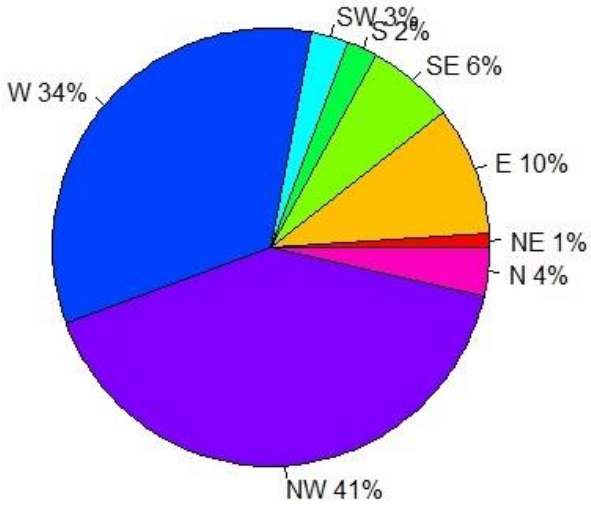

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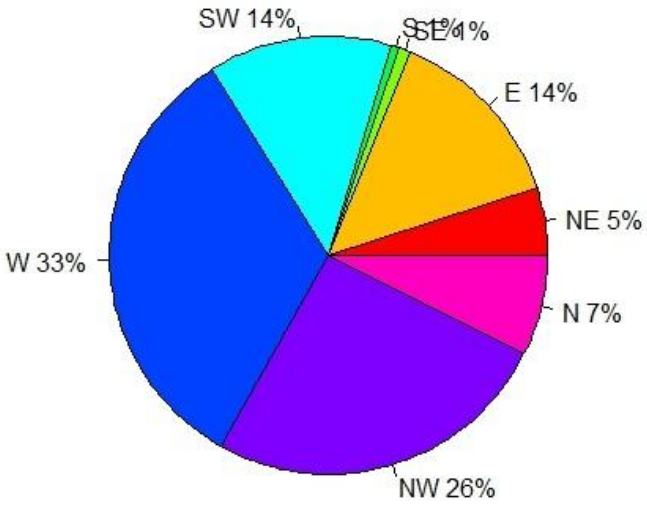

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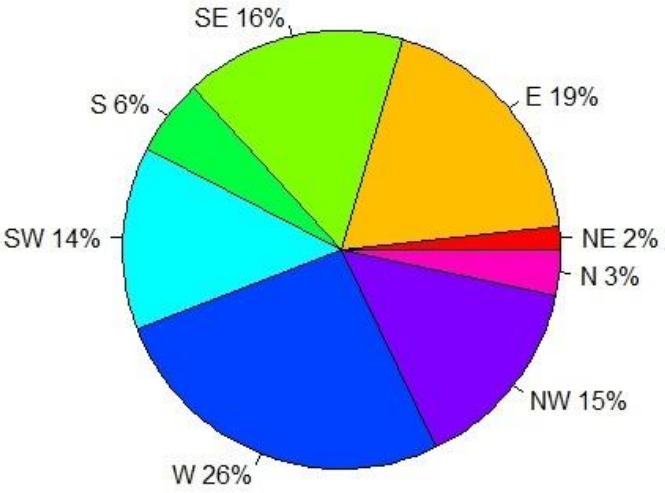

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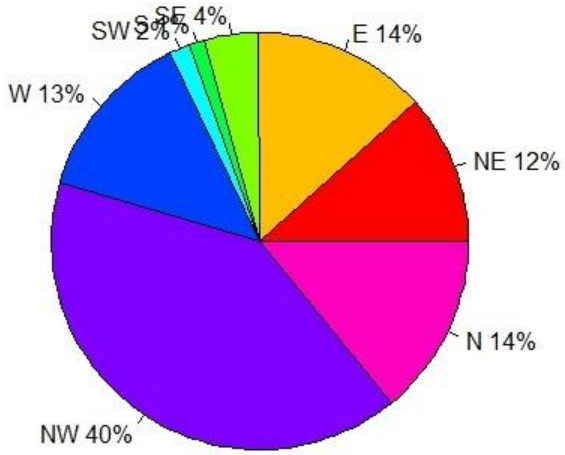

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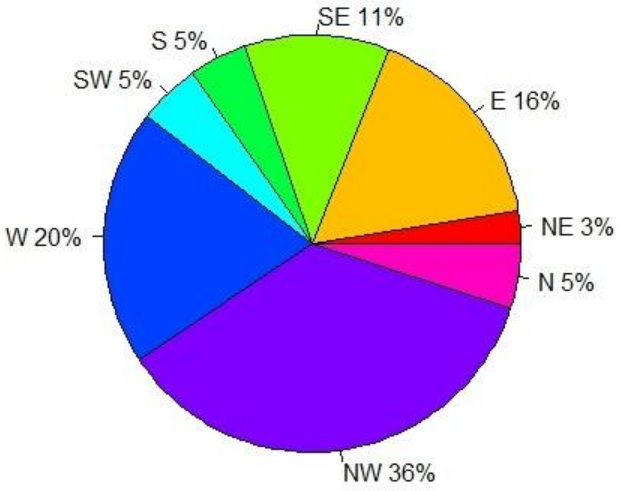
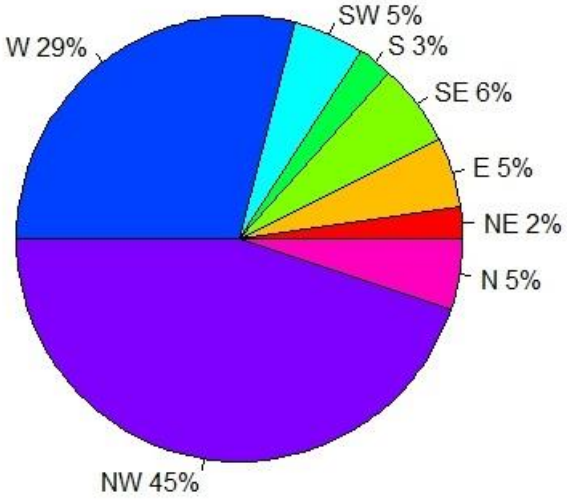


Patna Winter Reanalysis



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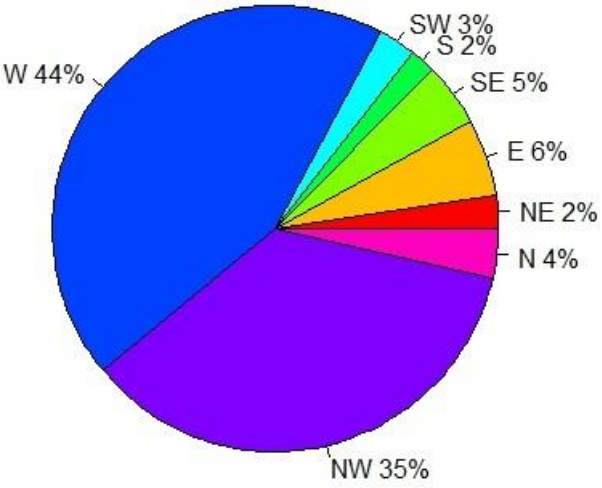

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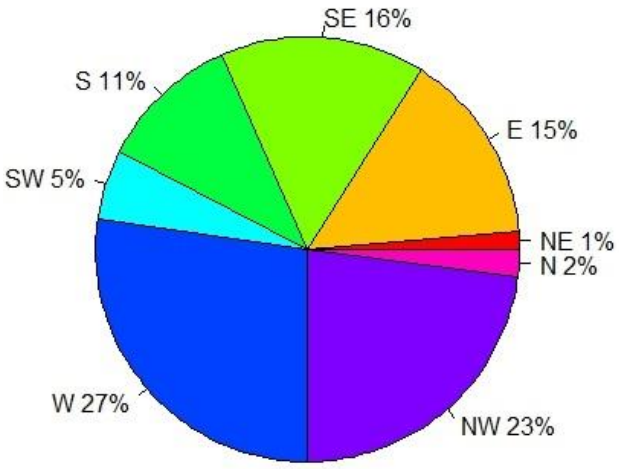

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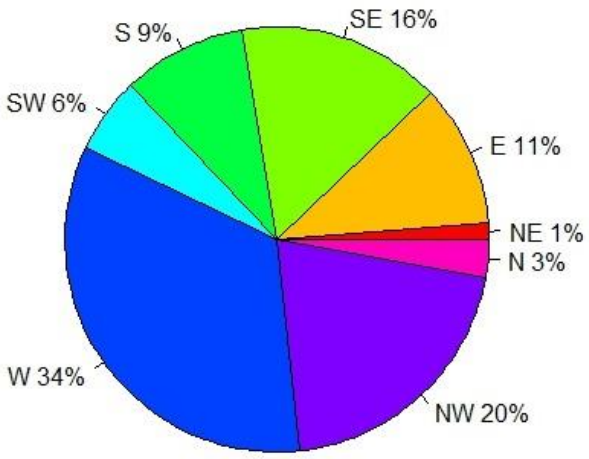

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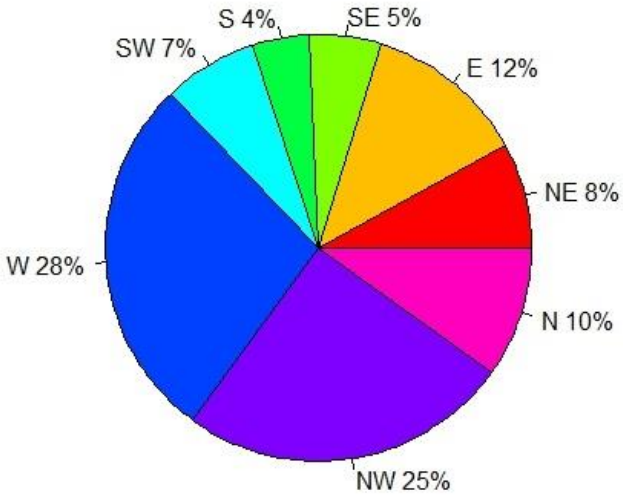

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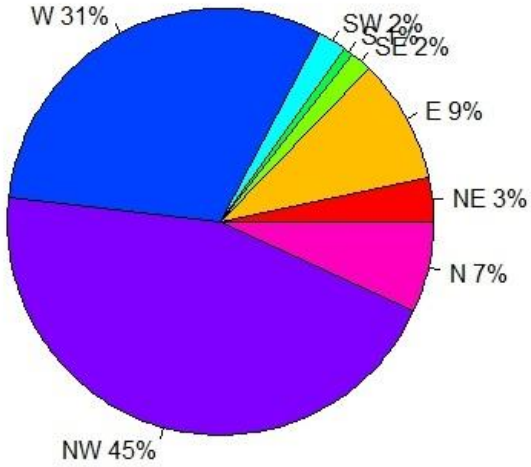
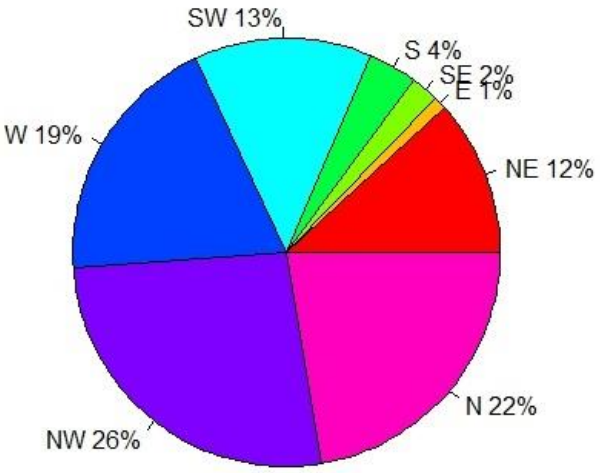


Kolkata Winter Reanalysis



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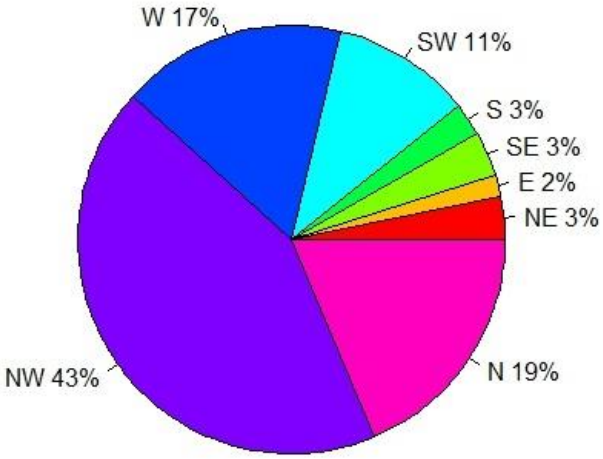
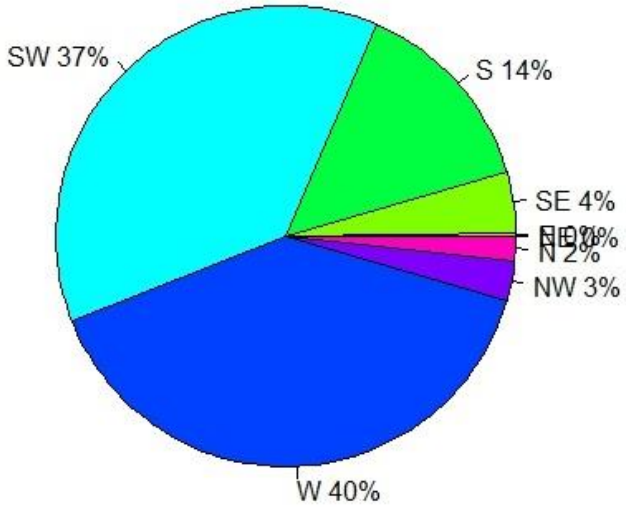


Kolkata Monsoon Reanalysis



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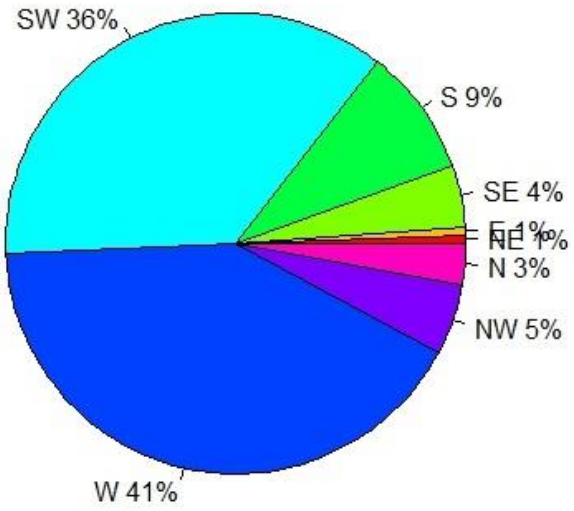
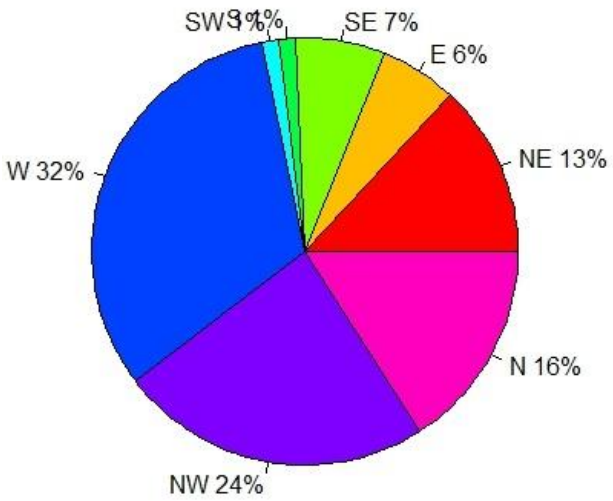
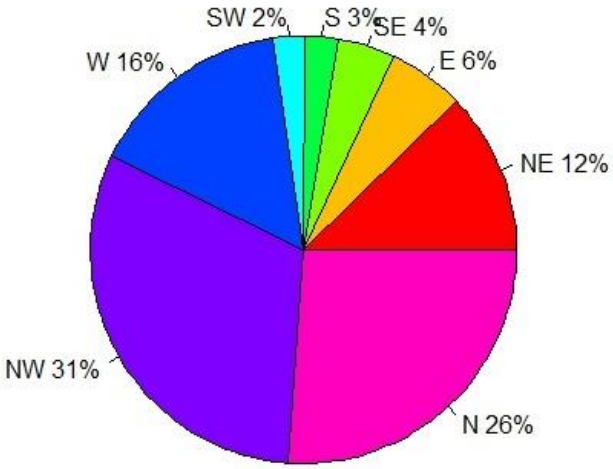


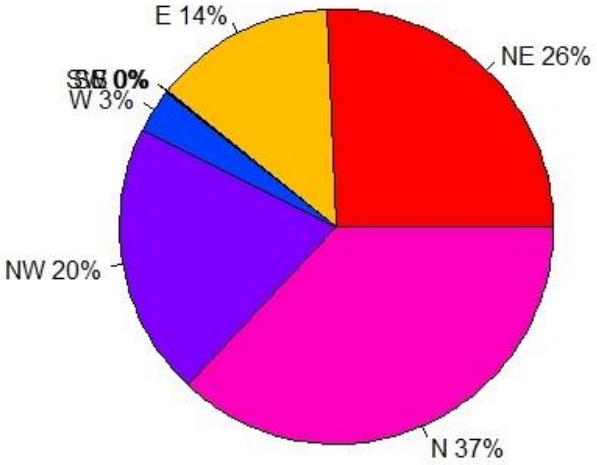
Kolkata Post-Monsoon Reanalysis



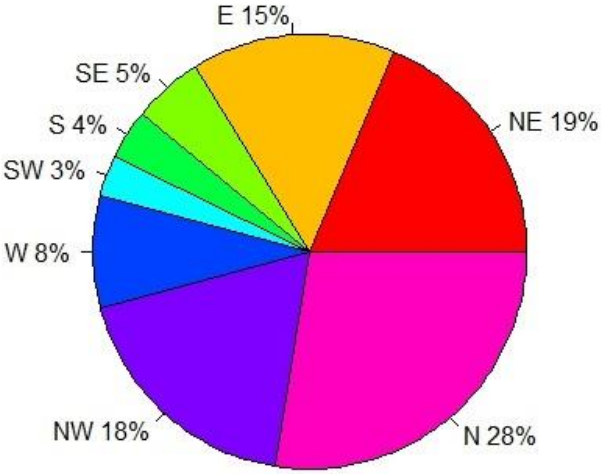
Kolkata Post-Monsoon GDAS0P5



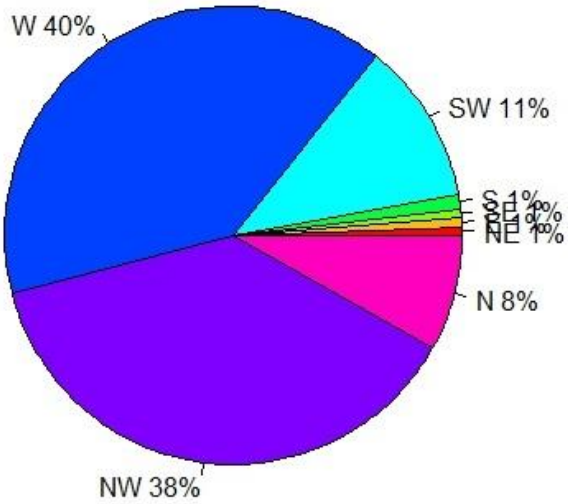
Mumbai Winter Reanalysis



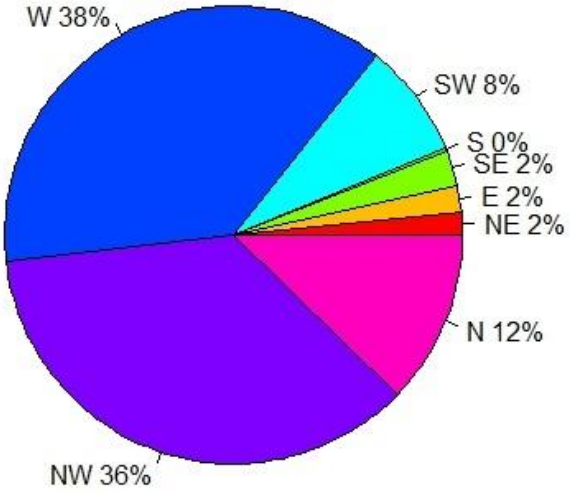
Mumbai Winter GDAS0P5



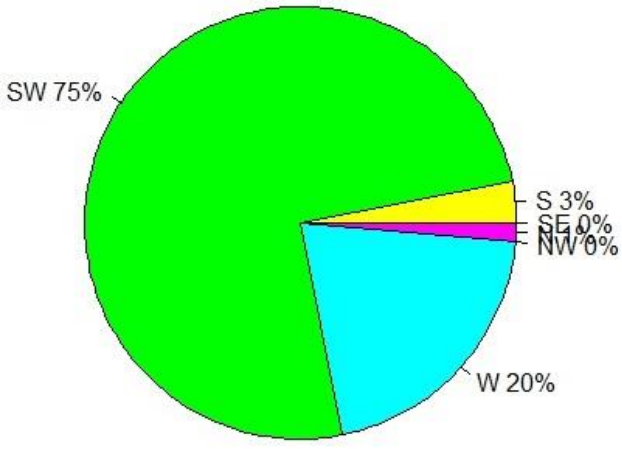
Mumbai Pre-monsoon Reanalysis



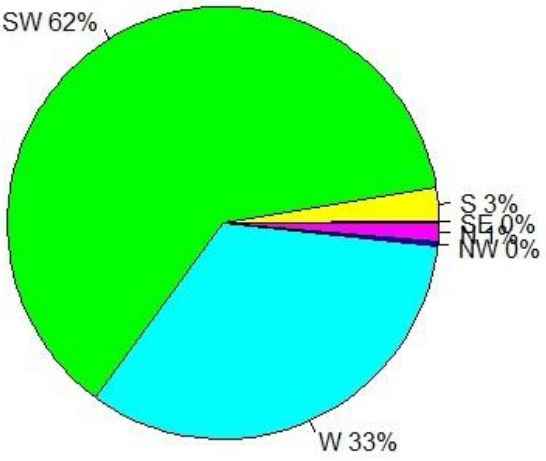
Mumbai Pre-monsoon GDAS0P5



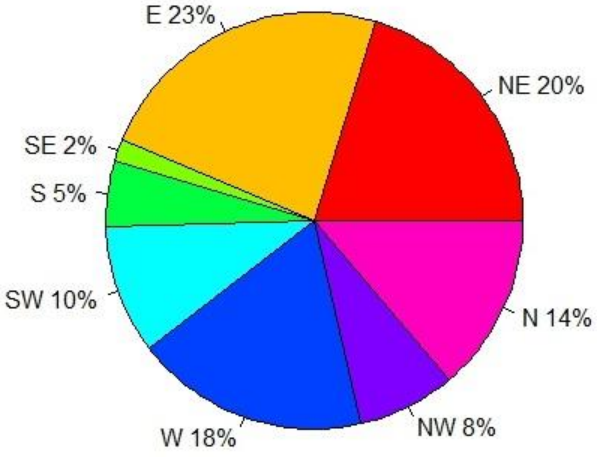
Mumbai Monsoon Reanalysis



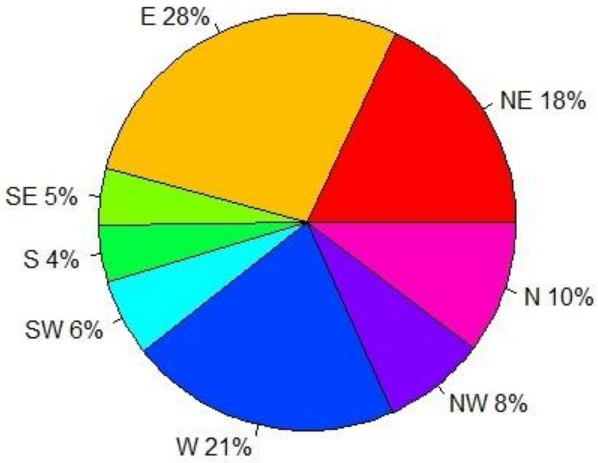
Mumbai Monsoon GDAS0P5



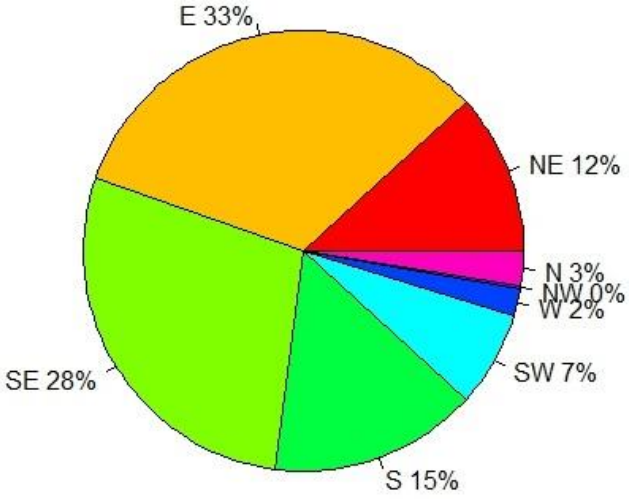
Mumbai Post-Monsoon Reanalysis



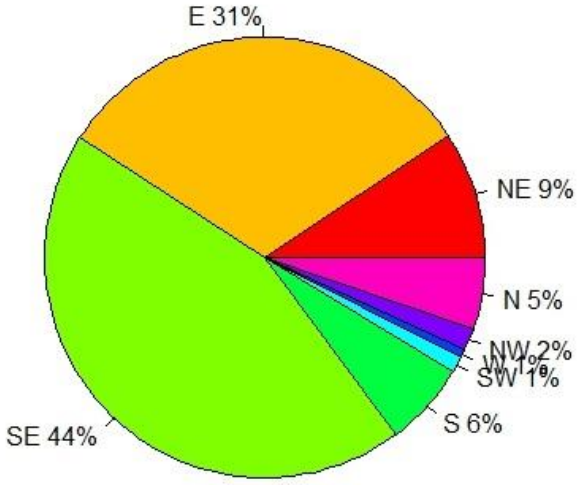
Mumbai Post-Monsoon GDAS0P5



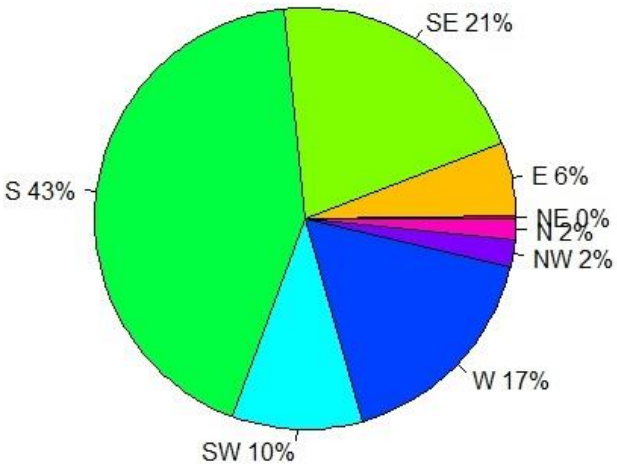
Hyderabad Winter Reanalysis



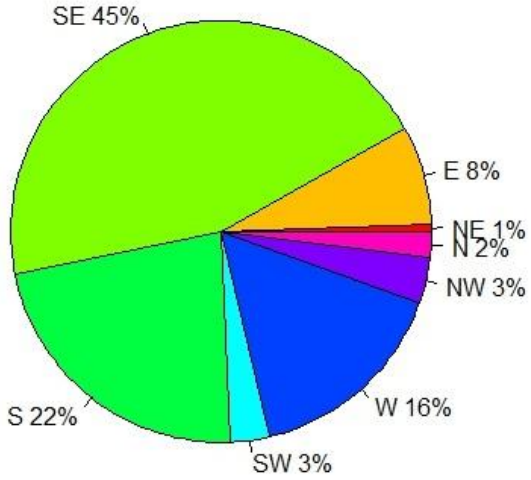
Hyderabad Winter GDAS0P5



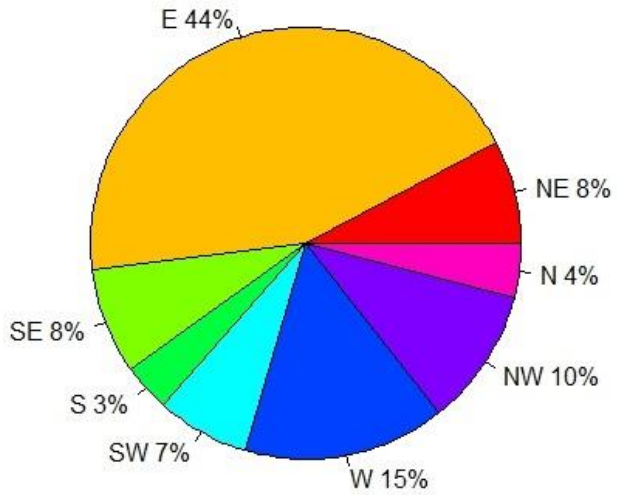
Hyderabad Pre-Monsoon Reanalysis



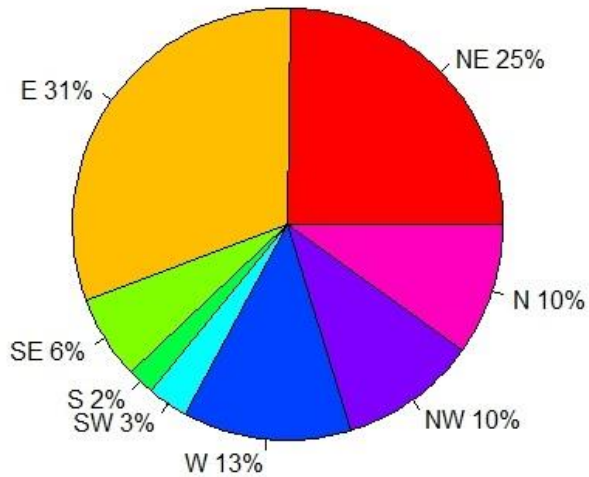
Hyderabad Pre-Monsoon GDAS0P5



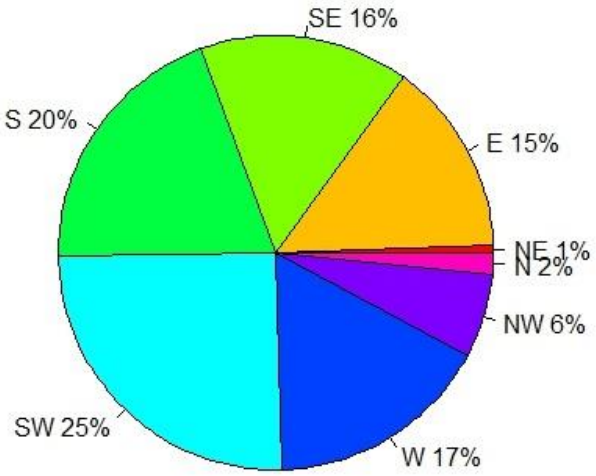
Hyderabad Post-Monsoon Reanalysis



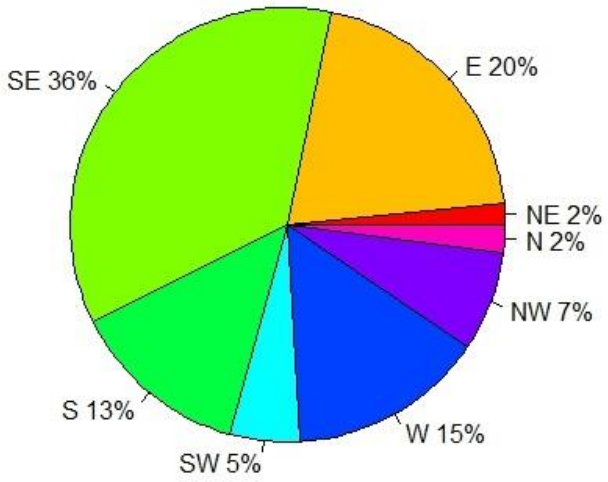
Hyderabad Post-Monsoon GDAS0P5



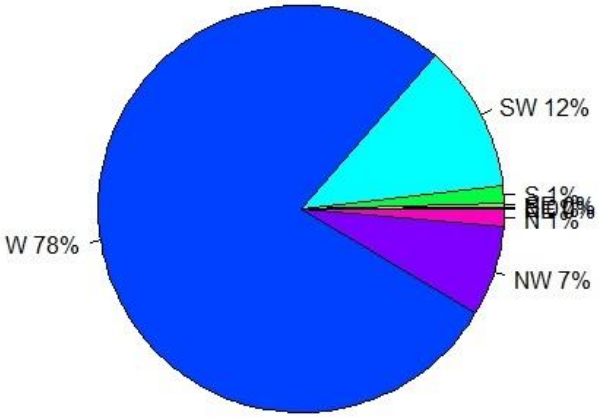
Bangalore Pre-Monsoon Reanalysis



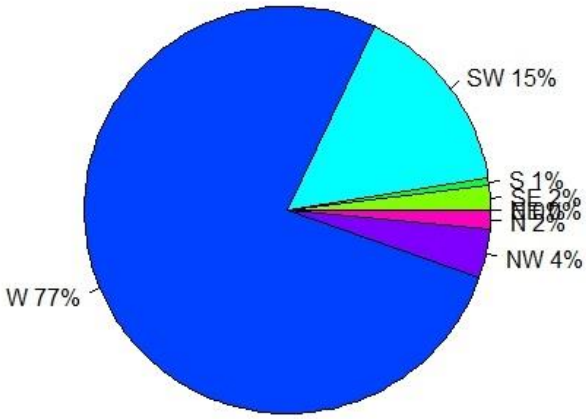
Bangalore Pre-Monsoon GDAS0P5



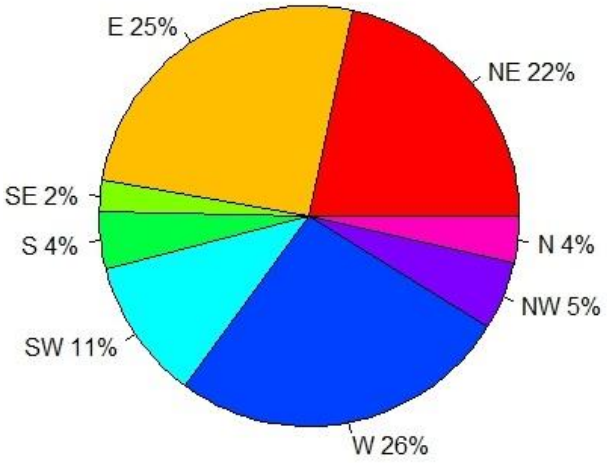
Bangalore Monsoon Reanalysis



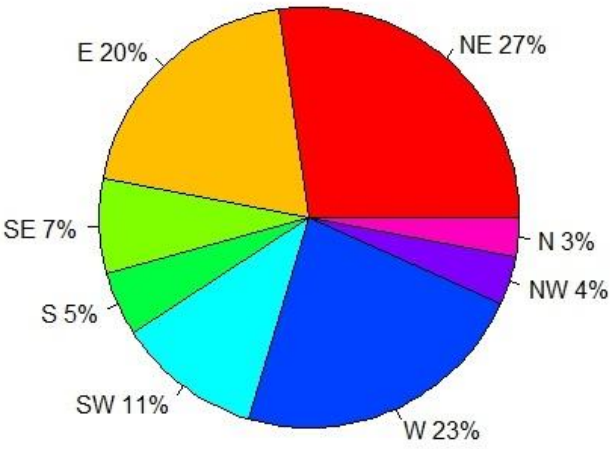
Bangalore Monsoon GDAS0P5



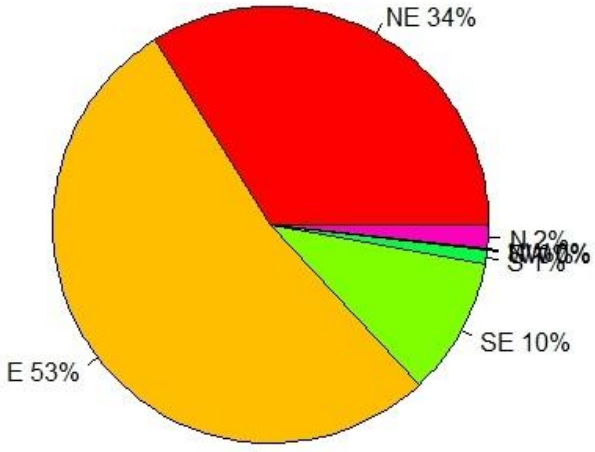
Bangalore Post-Monsoon Reanalysis



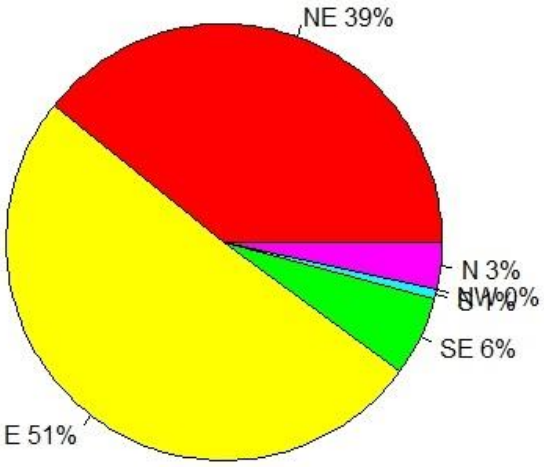
Bangalore Post-Monsoon GDAS0P5



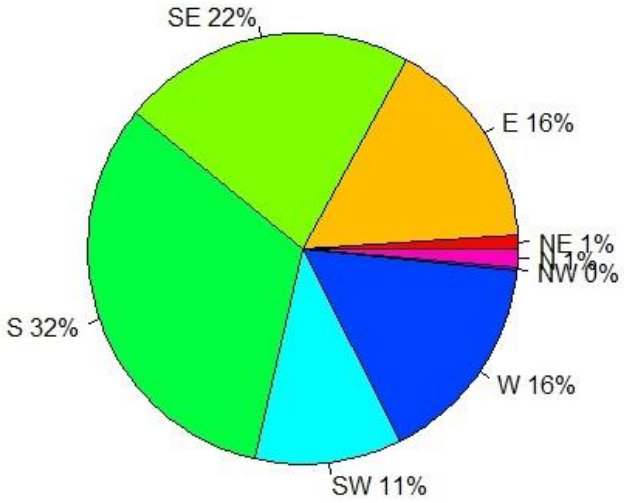
Chennai Winter Reanalysis



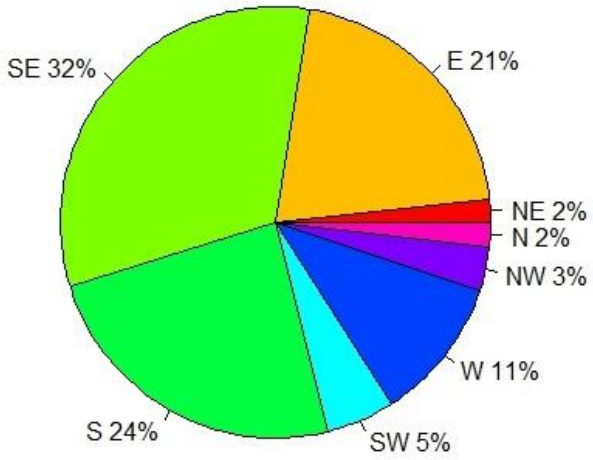
Chennai Winter GDAS0P5



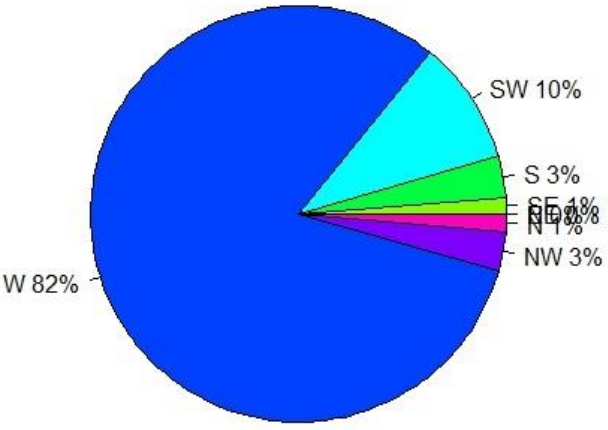
Chennai Pre-Monsoon Reanalysis



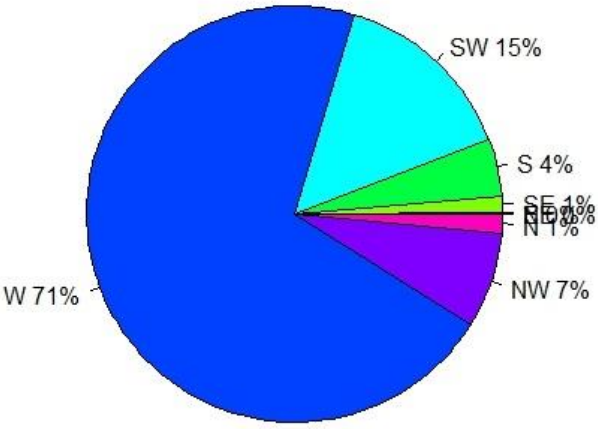
Chennai Pre-Monsoon GDAS0P5

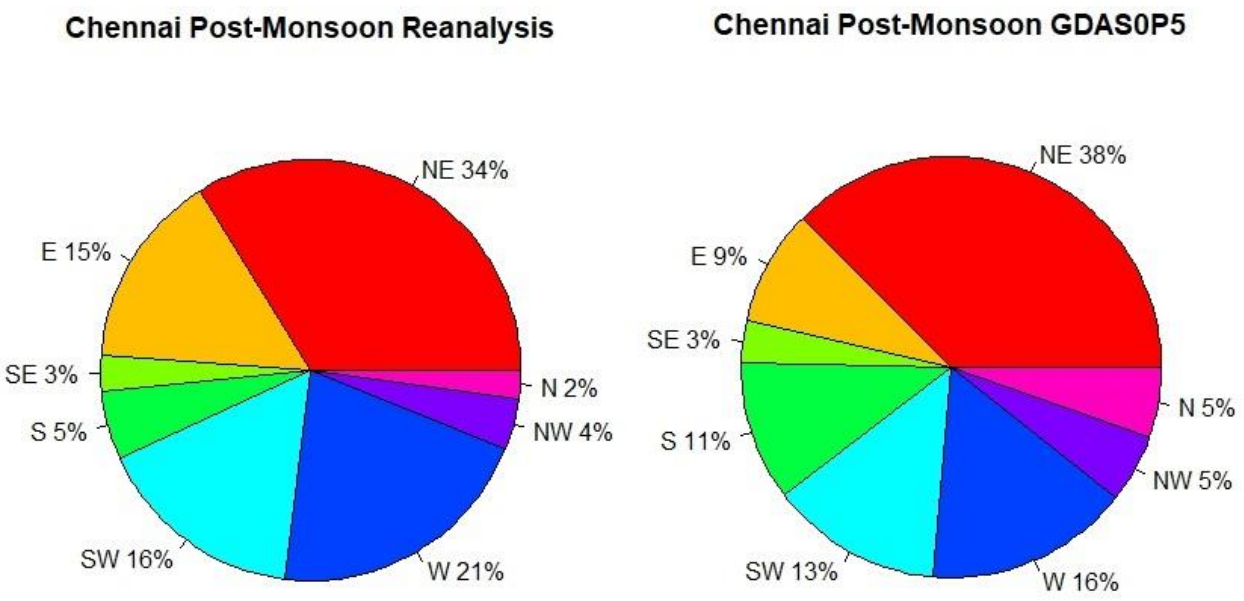


Chennai Monsoon Reanalysis

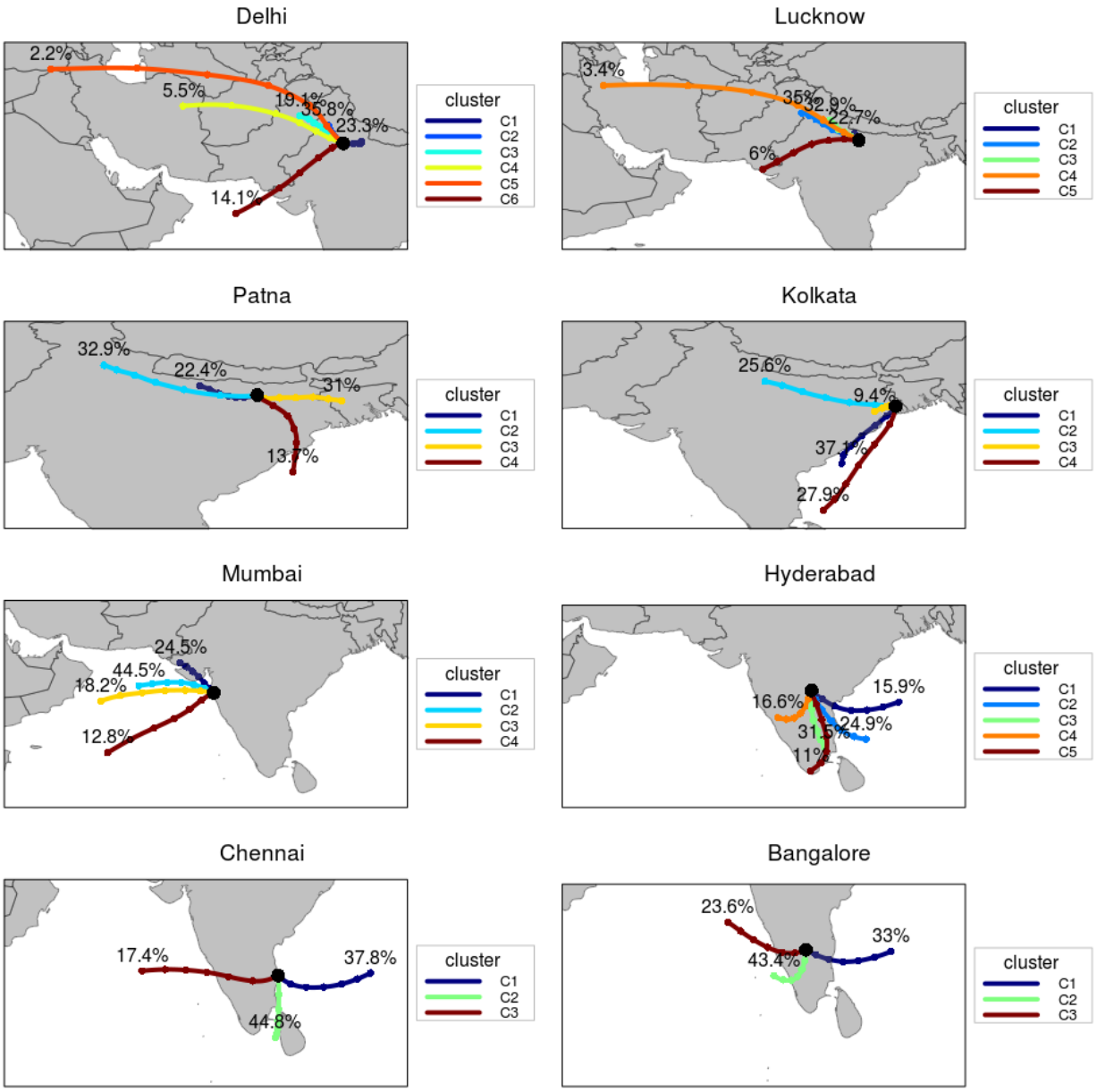


Chennai Monsoon GDAS0P5

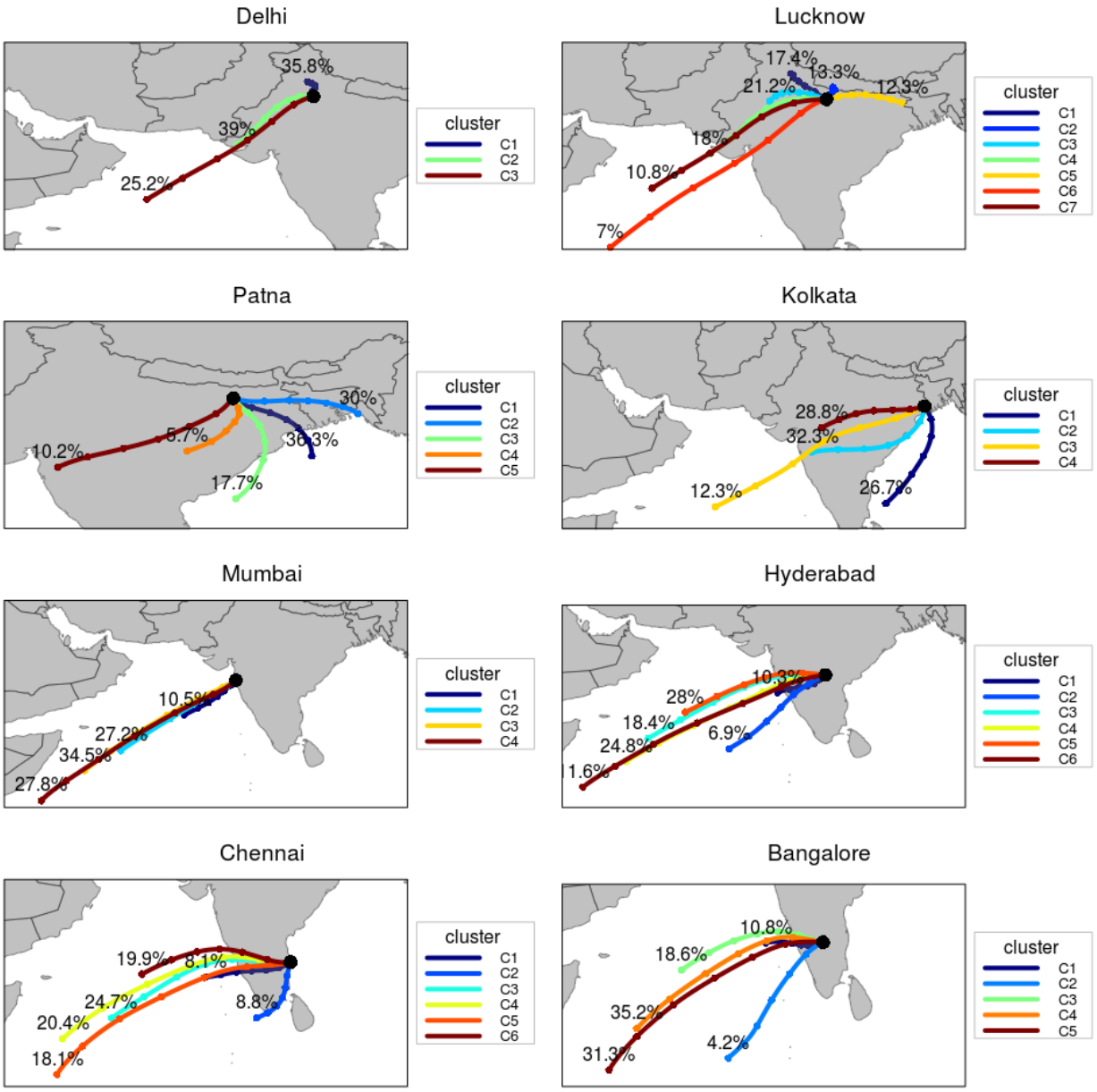




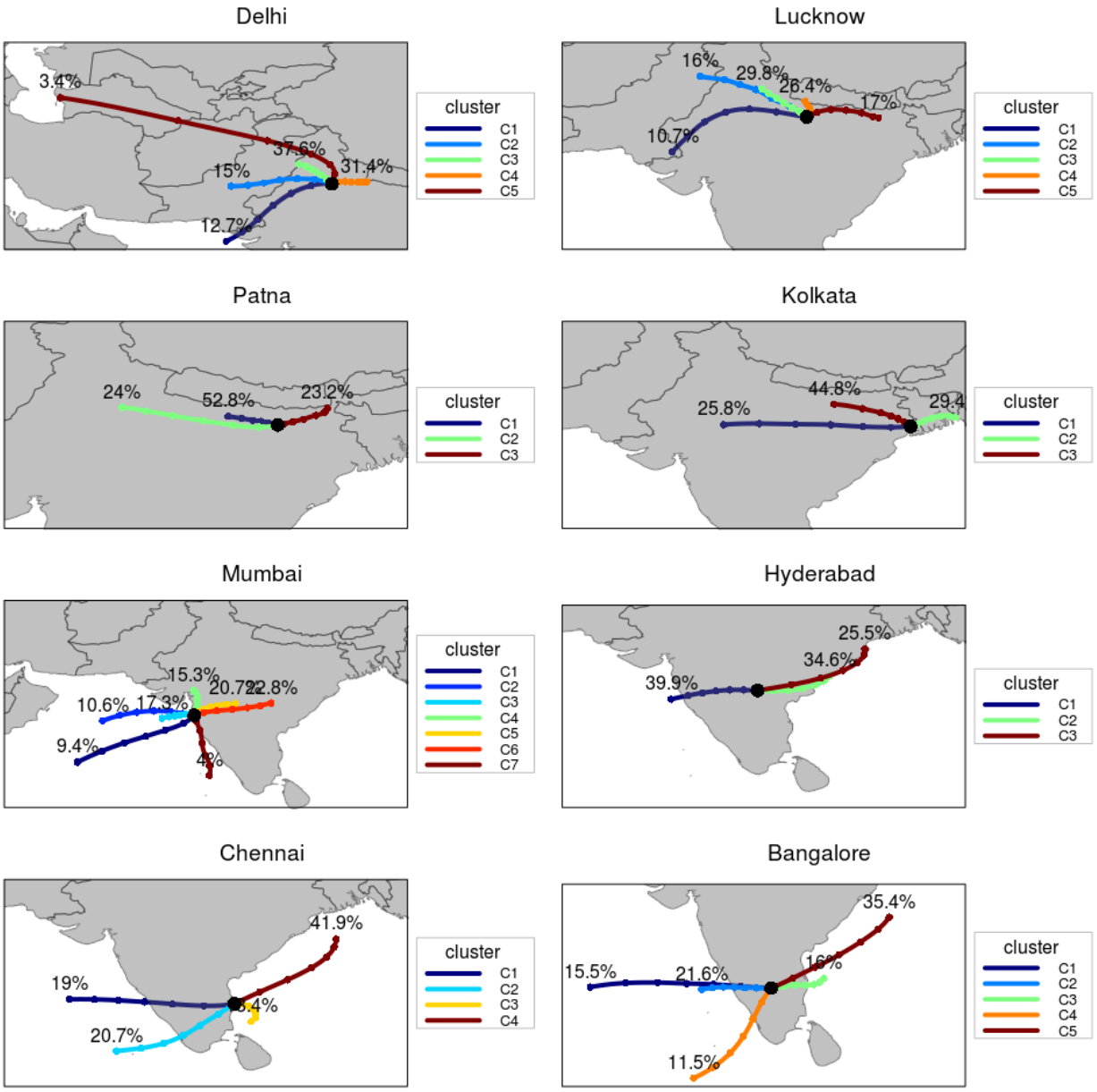
% Wind direction in each sector of HYSPLIT outputs using NCEP/NCAR Reanalysis and GDAS0.5° meteorological data



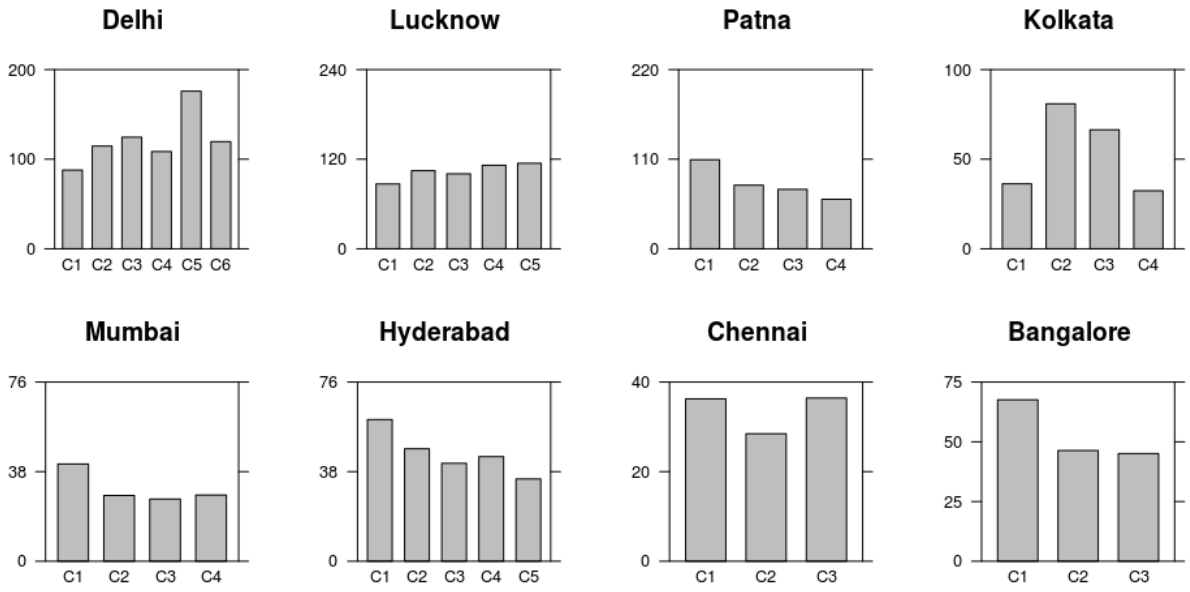
Distribution of trajectories into clusters to identify those associated with high $PM_{2.5}$ concentration during pre-monsoon



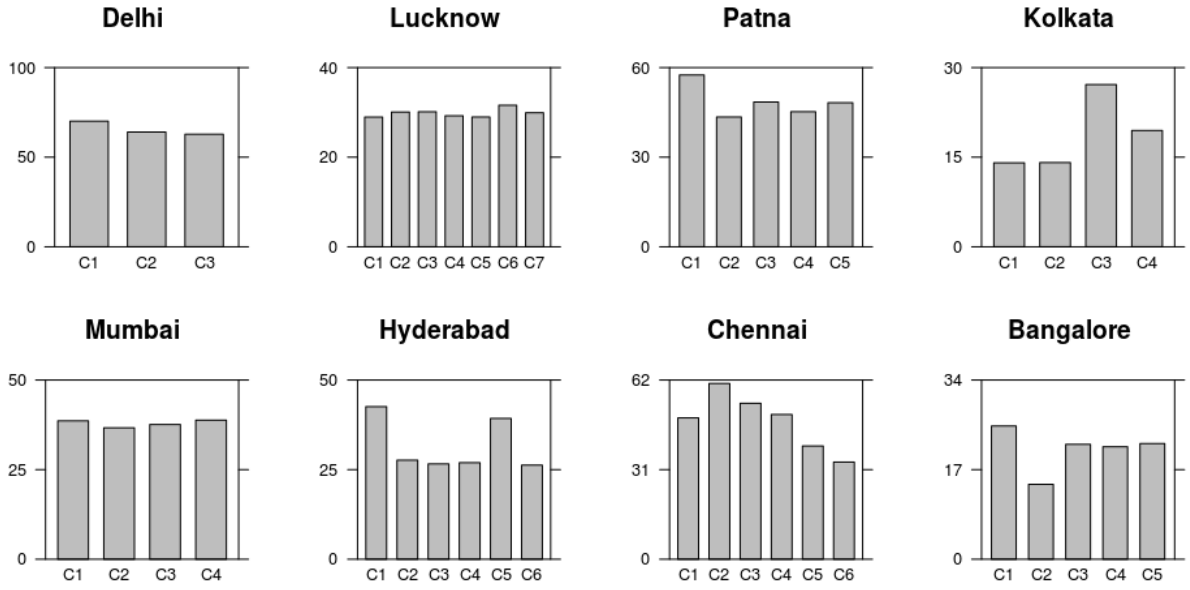
Distribution of trajectories into clusters to identify those associated with high $PM_{2.5}$ concentration during monsoon



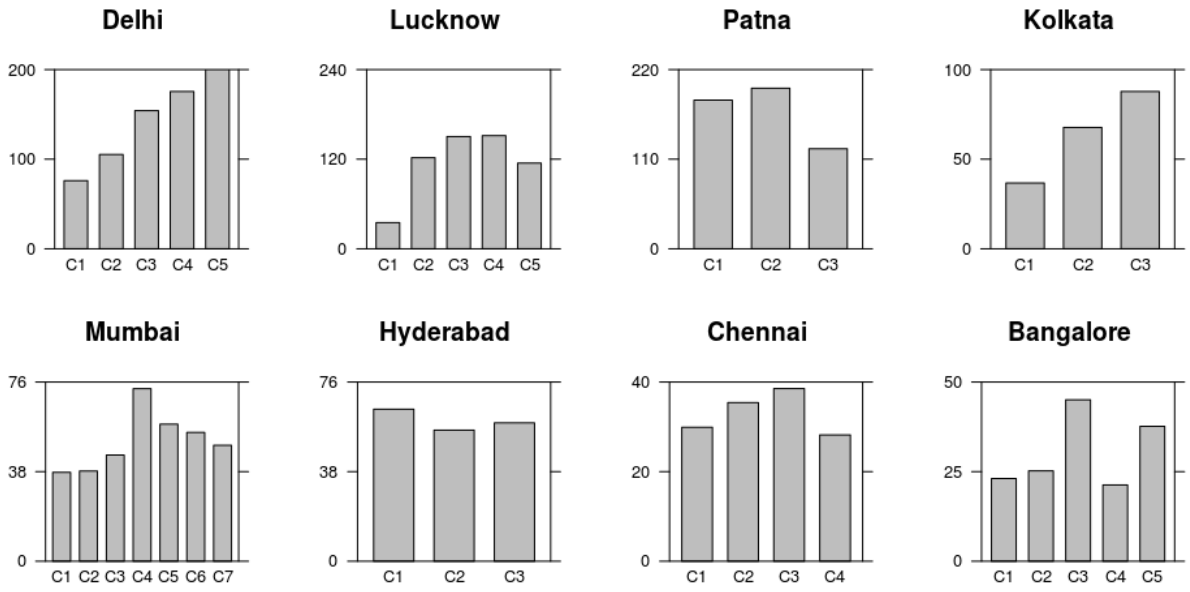
Distribution of trajectories into clusters to identify those associated with high $PM_{2.5}$ concentration during post-monsoon



Average $PM_{2.5}$ concentrations ($\mu g/m^3$) associated with each cluster for each city in pre-monsoon.



Average $PM_{2.5}$ concentrations ($\mu g/m^3$) associated with each cluster for each city in monsoon.



Average $PM_{2.5}$ concentrations ($\mu g/m^3$) associated with each cluster for each city in post-monsoon.

% $PM_{2.5}$ concentration contributed to the cities from neighbouring states and countries during pre-monsoon, monsoon and post-monsoon

Delhi						
Pre-monsoon	Haryana	Punjab	Uttar Pradesh	Pakistan	Rajasthan	Ocean
% Contribution	24.405	20.904	16.872	11.568	10.488	4.093
Monsoon	Rajasthan	Haryana	Ocean	Uttar Pradesh	Gujarat	Pakistan
% Contribution	30.445	18.946	12.367	9.330	6.814	5.621
Post-Monsoon	Uttar Pradesh	Haryana	Punjab	Pakistan	Rajasthan	Uttarakhand
% Contribution	24.169	21.173	15.393	14.033	9.307	4.415
Lucknow						
Pre-monsoon	Uttar Pradesh	Uttarakhand	Punjab	Himachal Pradesh	Haryana	Pakistan
% Contribution	60.498	9.777	6.631	5.941	5.465	3.936
Monsoon	Uttar Pradesh	Rajasthan	Ocean	Gujarat	Nepal	Bihar
% Contribution	43.229	15.353	9.345	5.155	5.117	4.741
Post-Monsoon	Uttar Pradesh	Nepal	Uttarakhand	Rajasthan	Bihar	Haryana
% Contribution	58.512	11.275	7.021	5.318	4.101	3.704
Patna						
Pre-monsoon	Uttar Pradesh	Bihar	Jharkhand	Bangladesh	West Bengal	Uttarakhand
% Contribution	32.596	30.810	7.322	6.751	5.418	4.379
Monsoon	Ocean	Bihar	Jharkhand	Bangladesh	West Bengal	Odisha
% Contribution	18.122	17.386	15.721	15.573	14.812	5.728
Post-monsoon	Bihar	Uttar Pradesh	Nepal	Jharkhand	Madhya Pradesh	Bangladesh
% Contribution	54.217	29.938	5.402	2.733	2.214	1.151
Kolkata						
Pre-monsoon	Ocean	West Bengal	Odisha	Uttar Pradesh	Jharkhand	Andhra Pradesh
% Contribution	43.156	19.323	10.169	9.669	5.338	3.226
Monsoon	Ocean	Madhya Pradesh	West Bengal	Odisha	Maharashtra	Chhattisgarh
% Contribution	37.569	10.906	10.756	9.511	8.705	5.859
Post-monsoon	West Bengal	Bangladesh	Jharkhand	Madhya Pradesh	Ocean	Bihar
% Contribution	38.604	17.565	12.781	8.263	7.241	4.814
Mumbai						
Pre-**monsoon**	Ocean	Gujarat	Maharashtra	Pakistan	Rajasthan	Karnataka
% Contribution	82.553	11.316	4.565	0.786	0.669	0.008
Monsoon	Ocean	Maharashtra				
% Contribution	99.473	0.496				
Post-monsoon	Maharashtra	Ocean	Gujarat	Madhya Pradesh	Telangana	Chhattisgarh
% Contribution	41.889	41.654	6.784	3.805	3.271	0.673

Hyderabad						
Pre-Monsoon	Andhra Pradesh	Ocean	Telangana	Tamil Nadu	Karnataka	Kerala
% Contribution	26.976	25.312	21.265	13.805	10.941	0.658
Monsoon	Ocean	Maharashtra	Karnataka	Telangana	Andhra Pradesh	Tamil Nadu
% Contribution	65.416	15.645	10.384	6.771	1.259	0.249
Post-monsoon	Ocean	Telangana	Andhra Pradesh	Maharashtra	Karnataka	Odisha
% Contribution	31.072	26.689	14.855	10.311	5.458	4.710
Chennai						
Pre-monsoon	Ocean	Tamil Nadu	Karnataka	Srilanka	Kerala	Andhra Pradesh
% Contribution	78.449	10.407	4.966	4.434	0.779	0.733
Monsoon	Ocean	Karnataka	Tamil Nadu	Andhra Pradesh	Kerala	Srilanka
% Contribution	66.613	15.686	10.156	5.676	0.749	0.378
Post-monsoon	Ocean	Tamil Nadu	Karnataka	Andhra Pradesh	Kerala	Sri Lanka
% Contribution	74.127	12.812	6.682	3.310	1.600	0.859
Bangalore						
Pre-**monsoon**	Ocean	Tamil Nadu	Karnataka	Kerala	Andhra Pradesh	Maharashtra
% Contribution	44.688	22.669	21.646	8.471	0.985	0.603
Monsoon	Ocean	Karnataka	Tamil Nadu	Kerala	Maharashtra	Andhra Pradesh
% Contribution	79.779	18.427	1.207	0.444	0.023	0.019
Post-monsoon	Ocean	Karnataka	Andhra Pradesh	Tamil Nadu	Kerala	Maharashtra

APPENDIX-III

A short discussion on polar plots

K. Yu et al. (2004) used wind speed, wind direction and pollutant concentrations to identify sources responsible for air pollution at a site. To generate polar plots for this study, data was portioned into different wind speed and wind direction categories (bins) and the mean $PM_{2.5}$ concentration in each bin was calculated. Later a generalized additive model was used to fit a framework of concentrations as a function of wind speed components (Uria-Tellaetxe & Carslaw, 2014).

In order to ascertain and identify the $PM_{2.5}$ concentration hotspots, polar plots were obtained. Polar plots provide concentration hotspots based on wind speed and direction and also help to differentiate between local and regional sources based on the wind speed of the sector in which high $PM_{2.5}$ concentration occurs.

Seasonal polar plots for different cities have been provided below. While, few hot spots are observed in and around Delhi, regional sources in north-west direction are more predominant. Biomass burning in these regions in winter could be a potential contributor to high $PM_{2.5}$ concentration (S. Ghosh et al., 2015). Similar analysis in pre-monsoon, indicated the dominance of sources in Punjab, Haryana, and Rajasthan. In monsoon, in addition to local sources, the dominant sources were from neighbouring Punjab and Haryana. In post-monsoon, while influence of local sources, Uttar Pradesh, Punjab and Haryana was observed, long-range transport from Pakistan and Afghanistan also affected the city. Although, trends in winter and post-monsoon were very similar, the dominance of Uttar Pradesh and local sources was more obvious in post-monsoon than in winter. Polar plots for Lucknow in winter suggest the influence of both local and regional sources. The local sources were located in south, southeast and north-east and the regional sources

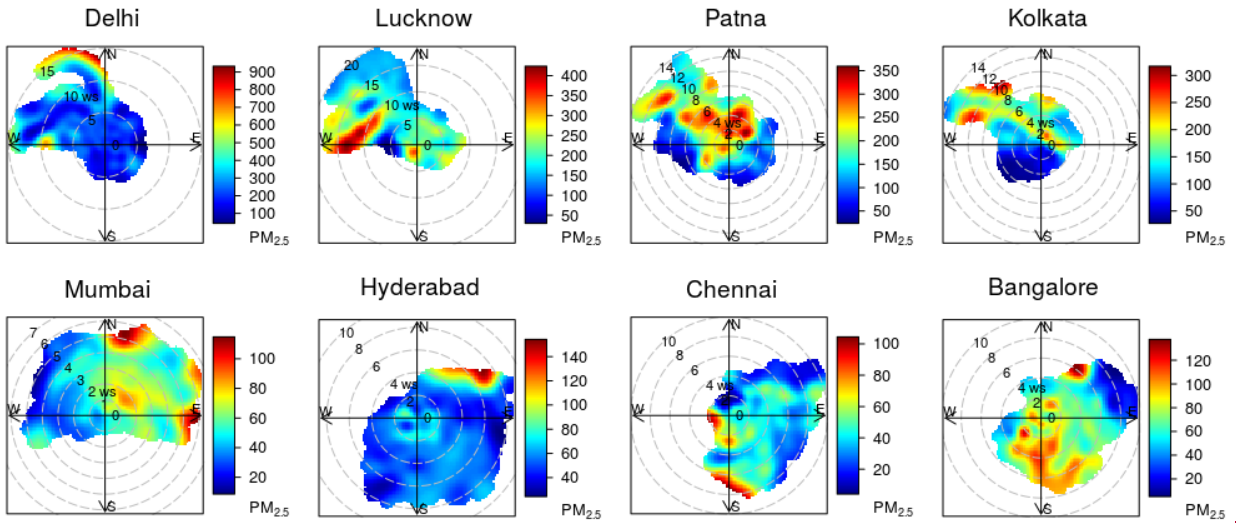
were located in north-west. In pre-monsoon, in addition to local sources, significant contributions of $PM_{2.5}$ from Punjab, Haryana, Rajasthan and Gujarat was observed. In monsoon, local as well as regional sources in Rajasthan and some parts of Nepal and Bihar dominate. In post-monsoon, local sources dominate. This indicates that in post-monsoon, $PM_{2.5}$ in Delhi and Lucknow was more likely due to sources in Uttar Pradesh.

In winter, local sources in northeast and northwest were chief contributors to $PM_{2.5}$ in Patna. In pre-monsoon, regional sources from north-west were more dominant. In monsoon, parts of West-Bengal and Bangladesh were found to be the dominating regions. In post-monsoon, apart from local sources, parts of Madhya Pradesh and Uttar Pradesh seem to be contributing to high $PM_{2.5}$ concentration in the city. Overall, the contributions of local sources was significant on high concentrations days in this city. Similarly, Kolkata in winter was affected by local sources from northeast and northwest and by regional sources from parts of Nepal, Bangladesh, Bihar, U.P. and Jharkhand. In pre-monsoon, sources from north-west affected the city, while regional sources from west were found to be the major contributors of $PM_{2.5}$ pollution in the city in monsoon. In post-monsoon, high $PM_{2.5}$ concentration form north-west suggested presence of a local source in the region. Overall, in addition to local sources, contribution of Bangladesh, Bihar and Jharkhand were also significant on highly polluted days in this city.

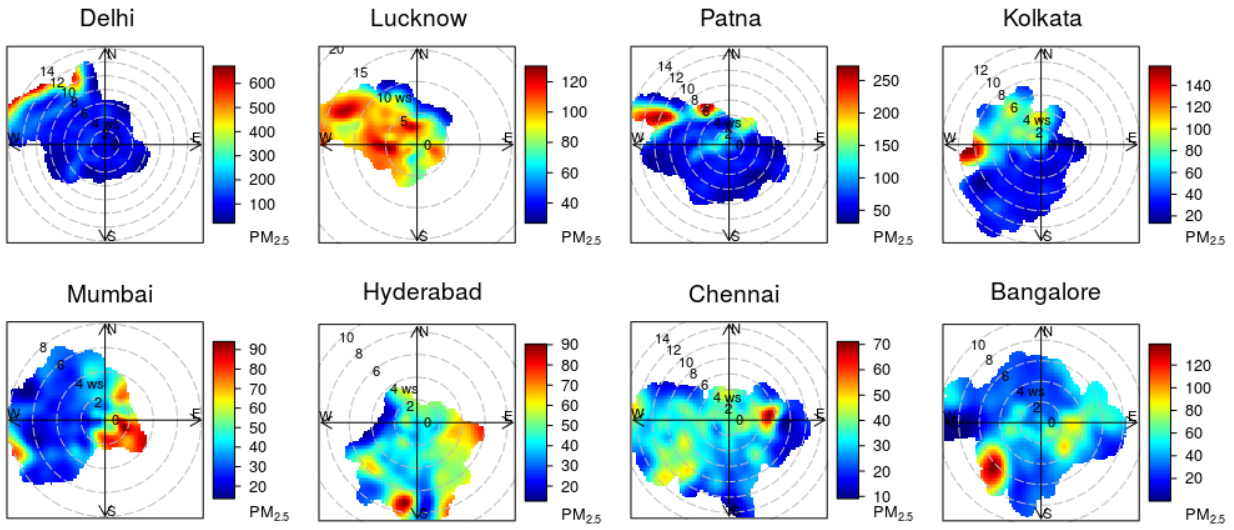
Mumbai on the other hand was effected by local sources in east and northeast in winter and by local sources in northeast in post-monsoon. While marine sources dominated monsoon $PM_{2.5}$, local sources in southeast were chief contributors during pre-monsoon.

In coastal southern city Chennai, marine sources played a dominant role in winter and pre-monsoon, while in monsoon and post monsoon local sources were dominant. This could be reason

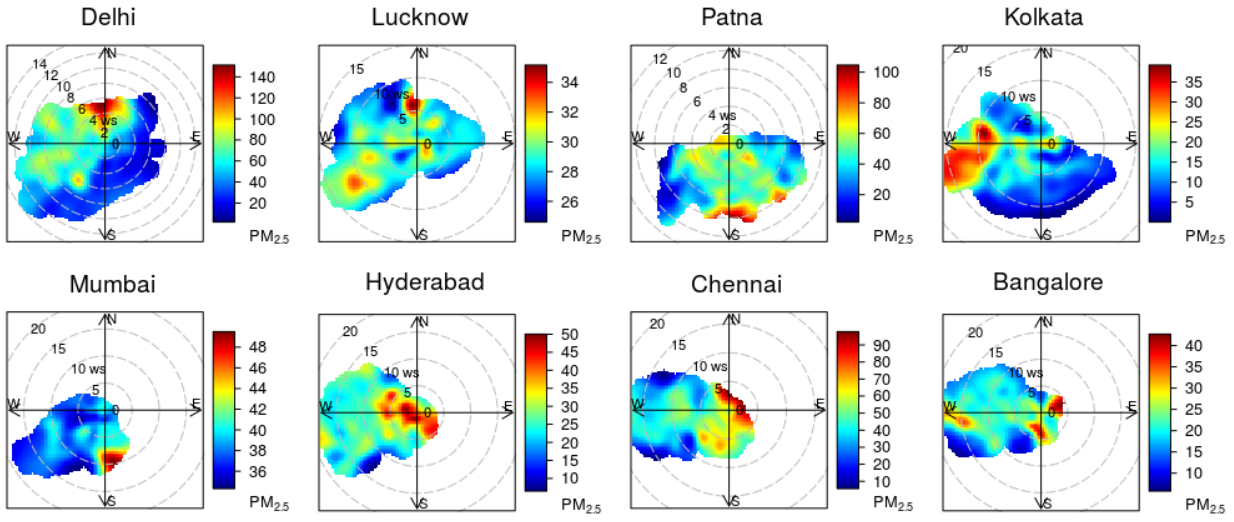
for higher $PM_{2.5}$ concentrations in monsoon in this city. Hyderabad was affected by local sources in north-west and northeast during winter, east and south-west in pre-monsoon, local sources in monsoon and by regional sources in north-west and south-west in post-monsoon. Bangalore was affected by local sources in south and southeast during winter. In monsoon, local sources in east and south dominated, while sources in south-west dominated in pre monsoon. Overall, in eastern, southern and western cities controlling emissions in their respective states could yield favourable results.



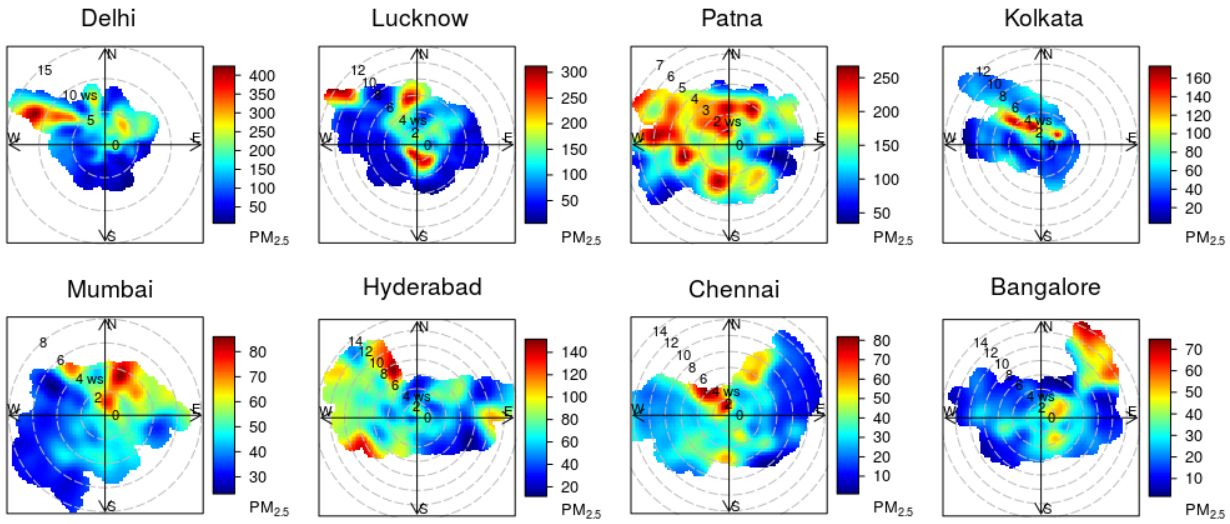
Distribution of $PM_{2.5}$ (µg/m3) concentrations in each wind speed, wind direction bins for each city in winter.



Distribution of $PM_{2.5}$ (µg/m3) concentrations in each wind speed, wind direction bins for each city in pre-monsoon.



Distribution of $PM_{2.5}$ concentrations (µg/m^3) in each wind speed, wind direction bins for each city in monsoon.



Distribution of $PM_{2.5}$ concentrations ($\mu g/m^3$) in each wind speed, wind direction bins for each city in post-monsoon.

A short discussion on correlation analysis between pollutants and meteorology

Seasonal correlation analysis between $PM_{2.5}$ and pollutants such as NO_2, CO, SO_2 and meteorological parameters such as wind speed, wind direction, temperature and RH is shown below. Correlation plots show Pearson correlation coefficient r in percentage with the shape and colour indicating the extent of relationship. In Delhi, $PM_{2.5}$ had negative correlation with wind speed (-0.59) and temperature (-0.34) while it had positive correlation with CO (0.66), NO_2 (0.24) and SO_2 (0.25). Correlation plot for pre-monsoon depicted positive correlation of $PM_{2.5}$with CO (0.68) and NO_2 (0.73), negative correlation with RH and wind speed. In monsoon, $PM_{2.5}$ had positive correlation with CO (0.59) and NO_2 (0.47), while it had negative correlation with RH (-0.50). Correlation plots for post-monsoon depicted positive correlation between $PM_{2.5}$ and NO_2 (0.54), CO (0.72) and SO_2 (0.82) and negative correlation with temperature (-0.76) and wind speed (-0.62). Probable sources of $PM_{2.5}$ include manufacturing and construction, household combustion, vehicles and thermal power stations (Sarath K. Guttikunda & Calori, 2013). In Lucknow, positive correlation between $PM_{2.5}$ and wind speed (0.43) during winter suggests non-local sources. In pre-monsoon, positive correlation existed between $PM_{2.5}$ and CO (0.35) and RH (0.24), and wind speed had negative correlation with $PM_{2.5}$ (-0.21). During monsoon correlation plots depict positive correlation of $PM_{2.5}$ with NO_2 (0.68) and SO_2 (0.69). In post monsoon CO, NO_2 and wind speed had positive correlation with $PM_{2.5}$. Vehicles, industrial emissions and construction activities may be the possible major sources of $PM_{2.5}$ (Verma A. K., 2016).

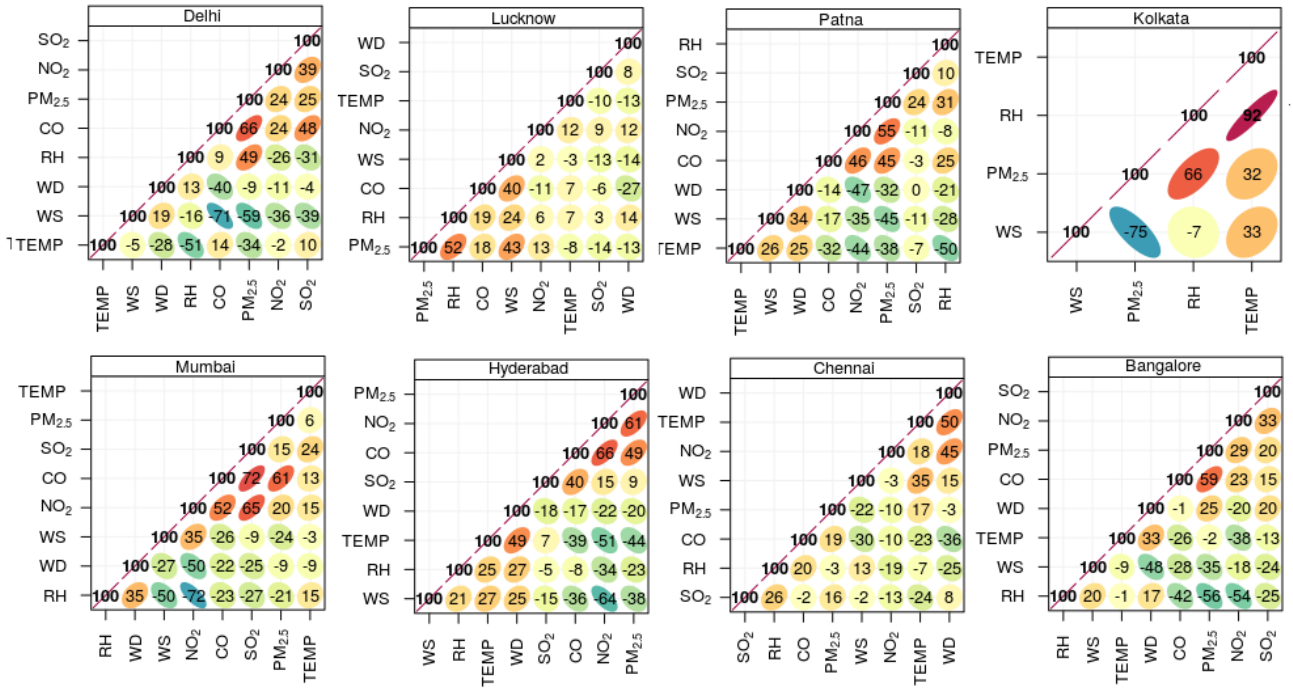
In Patna during winter, negative correlation of $PM_{2.5}$ with wind speed (-0.45) and temperature (-0.38) suggests local sources. Positive correlation were observed with NO_2 (0.55), CO (0.45) and SO_2 (0.24). In pre-monsoon $PM_{2.5}$ had positive correlation with CO (0.23) and

negative correlation with temperature (-0.26). During post-monsoon positive correlation between $PM_{2.5}$ and CO (0.66) and NO_2 (0.57), and negative correlation with wind speed (-0.52) was observed. This indicates that brick kilns, vehicles, thermal power plants, domestic cooking could be the major sources of $PM_{2.5}$ in the city (UrbanEmissions.info, 2014). Data for pollutants other than $PM_{2.5}$ was not available for Kolkata, but the negative correlation between wind speed and $PM_{2.5}$ observed during all seasons suggest that local sources were dominant.

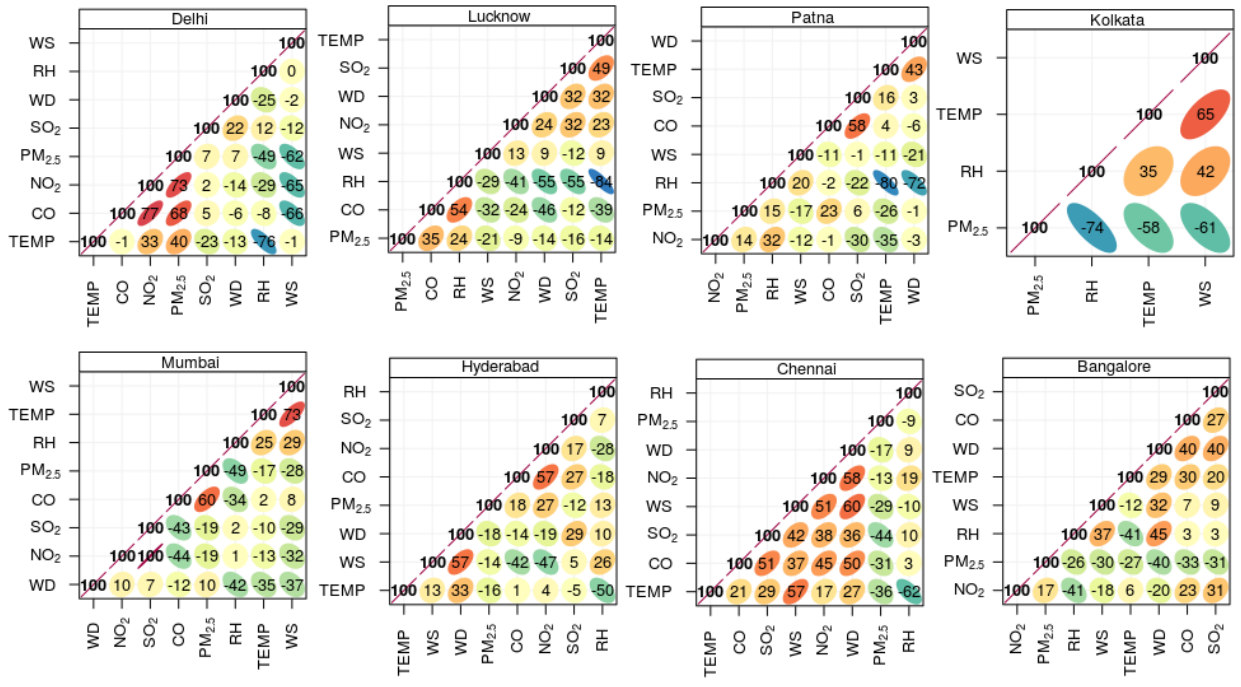
In Mumbai during winter, negative correlation was observed between $PM_{2.5}$ and wind speed (-0.24) while positive correlation was observed between $PM_{2.5}$ and CO (0.61), NO_2 (0.20) and SO_2 (0.15). During premonsooon positive correlation of $PM_{2.5}$ was obtained with CO (0.60), and negative correlation was observed with RH (-0.49) and wind speed (-0.28). In monsoon positive correlation between $PM_{2.5}$ and wind speed (0.33) suggested regional sources to be dominant. In post-monsoon $PM_{2.5}$ depicted positive correlation with NO_2 (0.61), SO_2 (0.45) and temperature (0.58), while it depicted negative correlation with wind speed (-0.41) and RH (-0.44). Primary sources of $PM_{2.5}$ included waste burning, vehicles, industries, household emissions, thermal power plants and marine sources (Sarath K Guttikunda et al., 2014; NEERI, 2010; Shah & Nagpal, 1997).

In Hyderabad, during winter, negative correlation was observed between $PM_{2.5}$ with wind speed (-0.38), temperature (-0.44) and RH (-0.23) indicating that low wind speed and temperature would result in high $PM_{2.5}$ concentrations. Additionally, strong positive correlation was observed with CO (0.49) and NO_2 (0.61). In pre-monsoon $PM_{2.5}$ was found to have positive correlation with CO (0.18) and NO_2 (0.27) which is similar to that in winter. In monsoon correlation plots depicted a positive relationship between $PM_{2.5}$ and NO_2 (0.54) and a negative relationship between wind

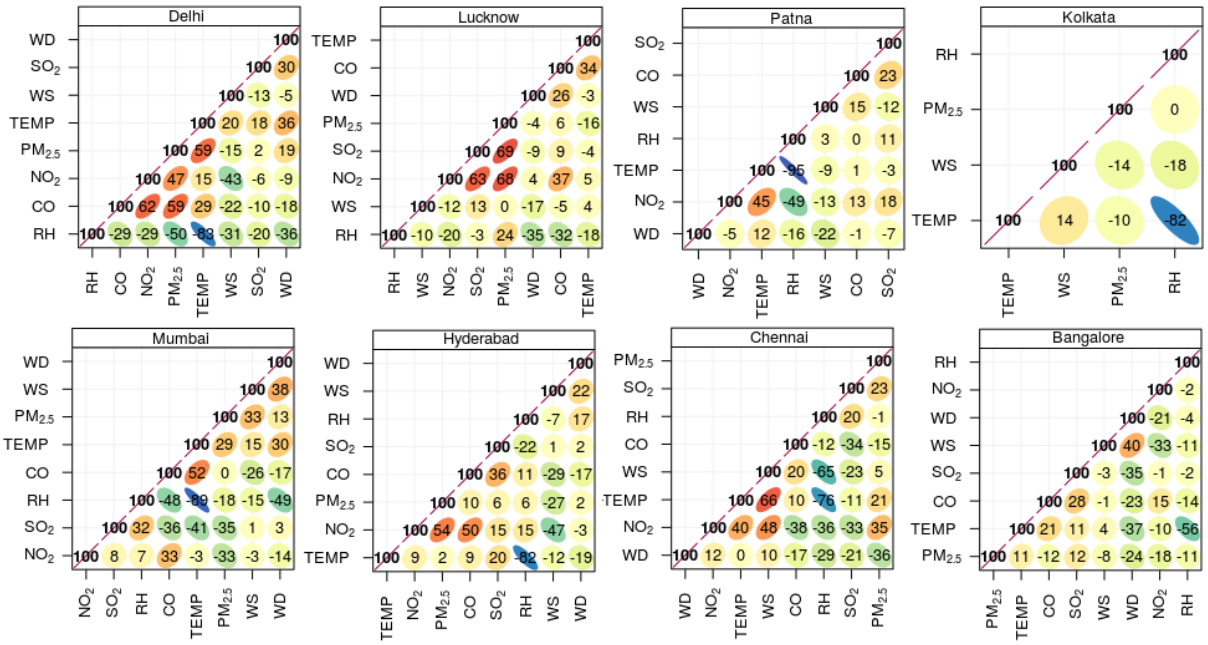
speed and $PM_{2.5}$ (-0.27). No significant correlation of $PM_{2.5}$ with any other parameters was observed during post-monsoon. Potential sources of $PM_{2.5}$ could be vehicles, industries and waste burning (Sarath K Guttikunda et al., 2014). In Bangalore, during winter $PM_{2.5}$ had negative correlation with RH (-0.56) and wind speed (-0.35), and positive correlation with CO (0.59), NO_2 (0.23) and SO_2 (0.15). In pre-monsoon $PM_{2.5}$ depicted a negative correlation with wind speed (-0.30). During monsoon, $PM_{2.5}$ did not show any significant correlation with any other pollutant or meteorological parameter, while during post-monsoon negative correlation with wind speed (-0.29) was observed. Potential sources of $PM_{2.5}$ were suggested to be vehicles, industries, residential sector and construction activities (TERI, 2010). In Chennai, during winter, $PM_{2.5}$ and wind speed had a negative correlation (-0.22) indicating high $PM_{2.5}$ concentrations occurred at low wind speeds in the city. Correlation coefficients of CO and NO_2 with $PM_{2.5}$ were found to be 0.19 and 0.16, respectively. During pre-monsoon $PM_{2.5}$ depicts a negative correlation with wind speed (-0.29). Positive correlation was observed between $PM_{2.5}$ and NO_2 (0.35) as well as SO_2 (0.23) during monsoon. $PM_{2.5}$ however did not show any correlation with any of the parameters in post-monsoon. Prominent sources of $PM_{2.5}$ in the city could be household emissions, vehicles, construction activities, diesel generator sets and marine sources (Sarath K. Guttikunda & Jawahar, 2012).



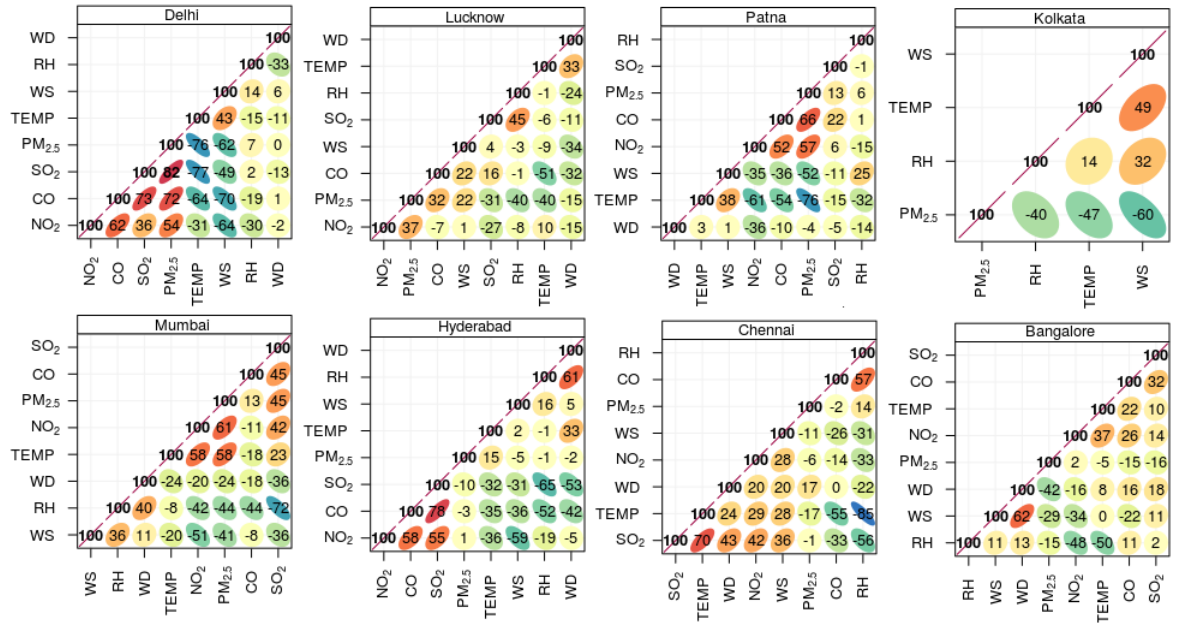
Correlation between $PM_{2.5}$ concentrations ($\mu g/m^3$) with meteorology and other pollutants in each city in winter.



Correlation between $PM_{2.5}$ concentration with meteorology and other pollutants in each city in pre-monsoon.



Correlation between $PM_{2.5}$ concentration with meteorology and other pollutants in each city in monsoon.



Correlation between $PM_{2.5}$ concentration with meteorology and other pollutants in each city in post-monsoon.

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- Shovan Kumar Sahu, Hongliang Zhang, Hao Guo, Jianlin Hu, Qi Ying and Sri Harsha Kota Health risk associated with potential source regions of $PM_{2.5}$ in Indian cities. Air Quality, Atmosphere & Health (under consideration).
- Shovan Kumar Sahu, Hao guo, Hongliang Zhang and Sri Harsha Kota. Impact of power generation on $PM_{2.5}$ concentration and associated health risks in India: current and future scenarios (in preparation).
- Hao guo, Shovan Kumar Sahu, Sri Harsha Kota and Hongliang Zhang. Regional Source Apportionment of $PM_{2.5}$ in North India Using a Source-oriented Regional Air Quality Model (in preparation).

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- Garaga, R., Sahu, S.K., Kota, S.H., Chemical Composition and Sources to Rain Water in North-east India. AOGS 2018, Hawaii, USA.