AMBIENT AIR POLLUTION AND ITS IMPACT ON HUMAN HEALTH: DECODING INDIAN TIER-2 CITIES SCENARIO

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ABSTRACT

This study investigates the relationship between ambient air pollution and public health outcomes across selected Indian cities, with a special emphasis on rapidly urbanizing Tier 2 cities. Using panel data from 2011 to 2020, the analysis evaluates how major ambient air pollutants namely PM_{10}, NO_2, and SO_2 along with meteorological and demographic variables, influence two key health indicators: Medically Certified Cause of Death (MCCD) and Acute Respiratory Infections (ARI). Employing both Fixed Effects and Random Effects panel data models, the study finds that PM_{10} consistently exhibits a significant positive association with ARI, while the effects of NO_2 and SO_2 are mixed and context-dependent. Rainfall and temperature also emerge as influential control variables. Population density is strongly linked with increased MCCD, suggesting urban stress on health infrastructure. The results underscore the urgency of targeted pollution control policies in Tier 2 cities, which are poised to face growing health burdens amid urban expansion.

Keywords: Ambient Air Pollution, Acute Respiratory Infection, Medically Certified Cause of Death, Panel Data Analysis, Tier 2 cities

JEL Codes: C23, I18, R11, Q53

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NO_2 – Nitrogen Dioxide

SO_2 – Sulphur Dioxide

PM — Particulate Matter

PM_{10} – Particulate Matter with size less than 10μ (microns)

$PM_{2.5}$ – Particulate Matter with size less than 2.5μ (microns)

O_3 – Ozone

CO – Carbon Monoxide

VOCs – Volatile Organic Compounds

CPCB – Central Pollution Control Board

NAMP – National Ambient Monitoring Programme

NAAQS – National Ambient Air Quality Standards

CAAQMS – Continous Ambient Air Quality Monitoring Stations

ARI – Acute Respiratory Infection

MCCD – Medically Certified Cause of Death

CD – Circulatory Diseases

RD – Respiratory Diseases

IMD – Indian Meteorological Department

WHO – World Health Organization

FE – Fixed Effects

RE – Random Effects

CHAPTER 1 - Introduction:

According to a report by the United Nations, the world population is estimated to increase to 9.6 billion by 2050. This rapid growth in the population is mostly, not just a single incidence but a combination of many other factors. Growth in population brings an increase in the requirement of land, resources, energy, food, and also a major destruction of environmental assets. Present mega cities in the world cannot hold on to the ever-growing population all long. This pressure is then transferred to Tier 2 cities, which are rapidly developing to hold the growth on the path of becoming megacities. This means that the health burden of people in Tier 2 cities is to increase drastically from the paced-up development activities. Urban development, even though is an important requirement for economic growth brings even more growth in negative consequences like waste management, water table conservations, forest cover loss, pollution, etc.

India is no more different than the conditions faced in other developed countries. In fact, the situations here in India are very detrimental. Ranking 5th in the global air polluted countries, 15 out of 20 most polluted cities are from India. The list of non-attainment cities prescribed by the Central Pollution Control Board (CPCB) has almost 131 cities which is majorly covered by tier 2 cities. The non-attainment cities are those cities that have fallen short of the National Ambient Air Quality Standards (NAAQS) for five consecutive years. India's population growing exponentially, surpassing China to become the world's most populous country with around 140 billion people. The health burden of population is clearly visible through the number of cities present in the list of non-attainment cities.

Air pollution is very much associated with a diverse spectrum of health effects and as well scientific evidence has been well established that exposure to pollution both indoor and ambient air pollution results in several diseases. Exposure to pollution is at times beyond the control of the individual, resulting in higher health burden of the population.

While Air pollution is a broader term which constitutes of indoor, outdoor, occupational settings of pollution, ambient air pollution explicitly focuses on exposure to pollution outdoors.

The sources of outdoor air pollution are vast and some of the major sources are as follows:

1. Vehicular emissions
2. Industrial emissions
3. Stationary power generation
4. Residential heating requirements
5. Agricultural emissions
6. Manufacturing, distribution and use of chemicals
7. Natural processes
8. Construction activities
9. Mining activities
10. Soil and road dust
11. And the list goes on…

1.1.Major Pollutants:

1.1.1. Particulate Matter (PM)

Atmospheric particles are categorized by size, which reflects their source and atmospheric behaviour:

- **Coarse PM (2.5–10 µm):** Generated from mechanical processes like soil resuspension, road dust, sea spray, and vehicle abrasion.
- **Accumulation Mode (0.2–2.5 µm):** Arises from secondary inorganic/organic condensation and coagulated nuclei-mode particles.
- **Nuclei Mode (<0.2 µm):** Originates from combustion and atmospheric nucleation, enriched in carbonaceous aerosols and metals.

$PM_{2.5}$ includes both nuclei and accumulation mode particles, while PM_{10} encompasses all three modes. PM chemical composition includes sulphates, nitrates, ammonium, elemental carbon (EC), and crustal material. Major sources include fuel combustion, vehicle emissions, biomass burning, and industrial processes. The contribution of sea salt and dust can also be significant depending on geography.

1.1.2. Sulphur Dioxide (SO_2)

SO_2 is primarily emitted through the combustion of sulphur-containing fossil fuels and metal smelting. While natural sources include microbial and volcanic activity, anthropogenic sources dominate in urban and industrial regions. Countries with poor regulation and high sulphur fuel content exhibit higher emissions. SO_2 is also a precursor to fine particulate sulphate through atmospheric oxidation processes.

1.1.3. Nitrogen Oxides (NOx)

The main sources of NOx (NO and NO_2) are fossil fuel combustion (vehicles, power plants), soil microbial activity, and biomass burning. Urban emissions are dominated by mobile and stationary combustion, while rural emissions stem more from soil and residential solid fuel use. NOx plays a critical role in photochemical smog formation through reactions with ozone. Vehicle aftertreatment systems may increase NO_2 near roadways. Ammonia, while not a NOx, contributes to secondary PM formation and is primarily from agriculture and vehicles.

1.1.4. Ozone (O_3)

O_3 is a secondary pollutant that is not directly emitted but formed in the atmosphere through photochemical reactions involving nitrogen oxides (NOx) and volatile organic compounds (VOCs) in the presence of sunlight. Tropospheric ozone or bad ozone forms near the ground and is a major component of photochemical smog. In contrast, stratospheric ozone or the good ozone protects Earth from harmful ultraviolet radiation.

Ozone levels typically peak during sunny afternoons in urban areas due to active photochemical reactions. Rural downwind regions may also experience high ozone due to transport from urban sources. Ozone is a strong oxidant and respiratory irritant, affecting lung function, exacerbating asthma, and increasing hospital admissions.

1.1.5. Carbon Monoxide (CO)

CO is a colourless, odourless gas formed primarily from incomplete combustion of carbon-containing fuels. Carbon Monoxides form majorly from road transport (especially petrol vehicles), residential heating (wood, coal, biomass), industrial processes, and wildfires. CO impairs oxygen delivery by binding to haemoglobin, reducing the blood's oxygen-carrying capacity. High exposures can cause dizziness, unconsciousness, or death.

1.2. Pollution Standards

Table 1: WHO Recommended 2021 AQG levels compared to 2005 air quality guidelines

Pollutant	Averaging Time	2005 AQGs	2021 AQGs
$PM_{2.5}$, $\mu g/m^3$	Annual	10	5
	24-hour[a]	25	15
PM_{10}, $\mu g/m^3$	Annual	20	15
	24-hour[a]	50	45
O_3, $\mu g/m^3$	Peak season[b]	-	60
	8-hour[a]	100	100
NO_2, $\mu g/m^3$	Annual	40	10
	24-hour[a]	-	25
SO_2, $\mu g/m^3$	24-hour[a]	20	40
CO, mg/m^3	24-hour[a]	-	4

(Source: WHO Air Quality Guidelines 2021)

Table 2: National Ambient Air Quality Standards (NAAQMS)

Pollutants	Time weighted average	Concentration in ambient air		
		Industrial Area	Residential, Rural & other Areas	Sensitive Area
1	2	3	4	5
Sulphur Dioxide (SO2)	Annual Average	80 µg/m3	60 µg/m3	15 µg/m3
	24 hours	120 µg/m3	80 µg/m3	30 µg/m3
Oxides of Nitrogen as NO2	Annual Average	80 µg/m3	60 µg/m3	15 µg/m3
	24 hours	120 µg/m3	80 µg/m3	30 µg/m3
Suspended Particulate Matter (SPM)	Annual Average	360 µg/m3	140 µg/m3	70 µg/m3
	24 hours	500 µg/m3	200 µg/m3	100 µg/m3
Respirable Particulate Matter (RPM) (size less than 10 microns)	Annual Average	120 µg/m3	60 µg/m3	50 µg/m3
	24 hours	150 µg/m3	100 µg/m3	75 µg/m3
Carbon Monoxides	8 hours	5.0 mg/m3	2.0 mg/m3	1.0 mg/m3
	1 hours	10.0 mg/m3	4.0 mg/m3	2.0 mg/m3

(Source: Central Pollution Control Board (CPCB), Ministry of Environment, Forest, Climate Change, Government of India)

1.2.Diseases caused from air pollution

Short-term effects of exposure to ambient air pollution:

- Coughing and wheezing
- Eye, nose, and throat irritation
- Shortness of breath
- Aggravation of asthma and bronchitis
- Acute Respiratory Infection

Long-term effects of prolonged exposure:

- Chronic respiratory diseases (e.g., asthma, COPD)
- Cardiovascular diseases (e.g., heart attacks, strokes)
- Lung cancer
- Impaired lung development in children
- Increased risk of premature mortality

Particle size	Penetration degree in human respiratory system
>11 μm	Passage into nostrils and upper respiratory tract
7–11 μm	Passage into nasal cavity
4.7–7 μm	Passage into larynx
3.3–4.7 μm	Passage into trachea-bronchial area
2.1–3.3 μm	Secondary bronchial area passage
1.1–2.1 μm	Terminal bronchial area passage
0.65–1.1 μm	Bronchioles penetrability
0.43–0.65 μm	Alveolar penetrability

Table 3: Penetrability according to particle size (source: Manisalidis, I., Stavropoulou, E., Stavropoulos, A., & Bezirtzoglou, E. (2020)

CHAPTER 2 - LITERATURE REVIEW:

2.1.Air pollution

Exponentially increasing population and production activities has put a lot of burden on our planet. The Health Effects Institute state that air pollution has become the fifth leading risk factor for mortality worldwide and is responsible for more deaths than many other risk factors. The National Capital Region in India is well known to be the most polluted capital city in the world. With majority of the pollution covering the city of Delhi with a thick blanket towards the end of the year is majorly caused by spillover effects of crop burning from adjacent states and ever-increasing transport figures. Chowdhury et al., 2023, has covered extensively in his research about the economic impacts of air pollution by assessing two cities, viz., Delhi and Narnaul in Haryana. The study measures the economic cost using three approaches, which are, cost of illness, productivity based and by undertaking a contingent valuation exercise. The determined economic costs in terms of health expenditure are quoted at a whopping Rs.4.8 billion. To improve air quality and enhance economic and social development, India has issued a National Ambient Air Quality Standard (NAAQS) for annual PM2.5 concentrations of 40μg/m3 (CPCB, 2009). But the records show that these numbers would be a longing dream. As per the State of Global Air Report, India is far beyond the NAAQS or the WHO guidelines for PM2.5 by registering at levels of 91μg/m3. IEA, 2016 states that about 1% of the Indian population are exposed to the safer limits of PM2.5 levels as stated by the guideline from WHO. An estimate from the OECD suggests that ambient air pollution alone may cost India more than 0.5trillion dollars per year (OECD, 2014). Recent studies indicate that probably less than 10-25% of global dust emissions originate from agricultural soils.

2.2.Tail Pipe Emissions

The average vehicle age is about 6 years for motorized two & three wheelers, between 6 & 7 years for cars, 7.5 – 8 for buses and heavy-duty trucks and lorries (S. Baidya & J. Borken Kleefeld,2009). Road transport includes variety of vehicles ranging from light-duty to heavy duty vehicles, which are mostly powered by internal combustion (IC) engines. As IC engines fuelled by fossil fuels emit harmful gaseous pollutants as well as particulate, road transport sector is largely blamed for environmental and health hazards. High population growth, economic development and rapid urbanization have led to exponential growth in automotive population, hence associated health risks have also increased proportionately. Projection shows that the total number of road vehicles would reach between 2 and 3 billion by 2050. A vehicle's tailpipe plume also includes numerous semi-volatile and volatile organic compounds (VOCs) (e.g., polycyclic aromatic hydrocarbons (PAHs), benzene, ethene, ethylene and toluene), which are highly toxic. Therefore, vehicular emissions form a major fraction of air pollutants, particularly in urban areas, which has now become a serious concern due to their toxicological effects on human health and ecosystem. Experimental investigations have demonstrated that gaseous organic compounds emitted in the vehicular exhaust (both gasoline and diesel) lead to formation of secondary organic aerosols (SOAs), photo chemical smog, and ground level ozone (O3). Together, photo chemical smog and SOAs lead to haze formation, which is evolving as a serious issue in developing countries like China and India. PM from the vehicular exhaust is usually composed of solid carbonaceous particles and organic compounds and can remain as suspended particles in the atmosphere or may deposit on the earth's surfaces. Because of the tiny sizes (μm-nm), PM is inhalable and gets easily transported through the human respiratory system. Transport sector is said to be responsible for approximately 18% of the total GHG emissions globally in 2016.

2.3.Emission Standards

The need for emission standards was brought up by A.J. Haagen Smit in 1952 when he found and a group of scientists recognized that the peculiar combination of warm climate, bounded landscape and rising population in Los Angeles, the State of California was the prevailing reason that the formation of large photochemical smog was composed of hydrocarbons and nitrogen oxides emanating from automobile exhaust emissions. Many nations came forward to put forth the needs of Emission standards and regulate vehicular emissions. An inversion of temperature coupled with coal fires that were used to power London's residential and industries, led to a toxic smog that resulted in more than 3,000 in 3 weeks after the event. Even though short-term health effects were immediately recognised, there were chronic health effects too that persistently increased mortality rates in the months after the event. In an analysis conducted by Bell and Davi, the scale of excess deaths caused by the toxic smog in London was likely closer to 12,000 in excess deaths in the year after. The sustained rise in mortality following the pollution event indicates it wasn't just a case of mortality displacement (where only the most fragile died slightly earlier). Instead, it affected a broader segment of the population, including relatively healthier individuals—highlighting that air pollution poses risks to a wider range of people than previously assumed.

S. Singh et al., 2022, discusses in detail about how the emission standards were rolled out in countries like the United States, Australia, Japan, Europe and India. Focusing particularly on India, the Clean Air Act in 1970, intended to reduce 90% of carbon monoxide, hydrocarbons and nitrogen oxide emissions from automobiles. This was later reformed into the Energy and Environmental Coordination Act in 1974 and the Clean Air Act Amendment in 1977. Emission norms in India for petrol and diesel distillates date back to 1991, 1992 emphasizing the needs for catalytic convertors and unleaded petrol. On April 2000, Euro II norms were made important in the National Capital Region. The Mashelkar committee's report in 2002 was accepted by the Government to introduce the Indianized version of emission standards. This was the birth of the 'Bharat Stage Emission Standards' (BSES). This restricted the intensity of air pollutants emitted from vehicles and targeted mainly at bringing changes in the fuel efficiency and engine design. BS II was released in 2001 followed by BS III in 2005. BS IV came into existence in

April 2010 by implementing in major cities and later enforced throughout India by April 2017. The delay in this process was equalled by leapfrogging to BS VI from BS IV directly. Because of high health risks associated with vehicular emissions, most countries have imposed increasingly stringent emission legislations namely: Euro-VI in European Union (EU); Tier-3 and California standard in USA; China-6 in China; and BS-VI in India. Table 1 and Table 2 shows the comparison between BSVI and emission standards of other nations. This makes it clear that the introduced BS VI emission standards are of international quality and is at a competence level.

Table 4: Comparison of fuel specifications for select gasoline parameters

Fuel parameter	BS VI	Euro 6	EPA RFG average (2005)	EPA convetional gasoline average (2005)	Japan	South Korea	Worldwide Fuel Charter (Category 4)
Sulfur, ppm, max.	10	10	30 ppm (Tier 2) 10 ppm (Tier 3)	30 ppm (Tier 2) 10 ppm (Tier 3)	10	10	10
Research Octane (RON), min.	91/95	95[a]	NS	NS	89/96	91/94	91/95/98
Motor Octane (MON), min.	81/85	85[a]	NS	NS	NS	NS	82.5/85/88
Anti-Knock Index (AKI), min.	NS	NS	87/87/91	87/87/91	NS	NS	NS
Olefins, vol%, max.	21/18	18	11.2-11.9	11.6-12.0	NS	16-19[b]	10

NS = Not specified; / used to separate specifications for different gasoline grades; AKI = (RON+MON)/2
[a]Member states are permitted to allow regular grade gasoline with MON of 81 and RON of 91.
[b]Either aromatics 24 vol.% max and olefins 16 vol.% max or aromatics 21 vol.% max and olefins 19 vol.% max.

Source: ICCT WORKING PAPER 2016-9 Technical Background on India BS VI Fuel Specifications

Table 5: Comparison of fuel specifications for select diesel parameters

Fuel parameter	BS VI	Euro VI	EPA coventional diesel	CARB designated equivalent limit	Japan	South Korea	Worldwide Fuel Charter (Category 4)
Sulfur, ppm, max.	10	10	15	15	10	10	10
Cetane Number (CN), min	51	51	Cetane index ≥ 40 or aromatics ≤ 35%	53	45	52[a]	55
Density @ 15°C, kg/m³	820-860	845 (max)	NS	NS	NS	815-835	820-840
95% Distillation Boiling Point (T_{95}), °C, max.	370	360	NS	NS	360[b]	360[b]	340
Polycyclic aromatic hydrocarbons (PAH), mass %, max.	11	8	NS	3.5	NS	5	2
Flash Point, Abel, °C, min.	35	55	NS	NS	45	40	55

NS = Not specified
[a]48 from November 15 to February 18
[b]Maximum 90% distillation boiling point (T_{90}) specified in Japanese and South Korean standards

Source: ICCT WORKING PAPER 2016-9 Technical Background on India BS VI Fuel Specifications

2.4.Health Effects of Air pollution

Indian cities rank high in global air pollution for PM2.5 which is associated with severe health impacts. While for India their exact quantification remains uncertain, the scientific literature estimates large numbers, ranging between 483,000 and 1,267,000 cases of pre mature deaths annually from outdoor pollution, and 748,000 – 1,254,000 cases from indoor pollution. In 2017, over 4.9 million deaths were associated with air pollution and about 147 million healthy lives lost. Compared with other major urban areas in India, New Delhi had the highest number of deaths attributable to transportation emissions in 2015 and the highest mortality rate—9 deaths per 100,000 population. New Delhi accounted for 2.5% of transportation-attributable deaths from PM2.5 and ozone pollution in India in 2015. An estimated 74,000 premature deaths were attributable to transportation emissions in India 2015. This represents a 28% increase in annual transportation-attributable deaths in India compared with 2010. Chronic exposure to ambient air pollution poses serious health issues related to heart and lungs. Some of the hazards include chronic obstructive pulmonary disease. Ischemic heart disease, stroke and lung cancer for adults and acute respiratory infection for children (Jain et al., 2017; Burnett et al.,2014)

Guarnieri M & Balmes JR., 2014 have studied about the link between outdoor air pollution and Asthma. The Health Effects institute have also concluded the during peak episodes of air pollution, a causal relationship has been established with childhood asthma exacerbations, by characterising emergency room visits, hospitalizations, and an increase in medication use which has been observed both in children and adults.

Li J., et al. 2016, in his systematic review of major air pollutants and risk of COPD exacerbations, he has identified that exposure in short term with respect to major pollutants like particulate matters of sizes 2.5 microns and 10 microns, nitrogen dioxide, sulphur dioxide, carbon monoxide and ozone are all associated with exacerbation frequency. In their study, particulate matter and NO_2 have shown the strongest effect.

Psoter KJ, et al. 2015 and Goss CH, et al., 2004 have done studies on the effect of ambient air pollution on cystic fibrosis. Cystic fibrosis is a genetic disorder that causes the body to produce abnormally thick and sticky mucus, primarily affecting the lungs, pancreas,

and other organs. This thick mucus can block airways, digestive ducts, and other passages, leading to a range of health problems. Fine particulate matter of size 2.5 microns may trigger the infection more and lead to a progressive loss of lung function over time.

Lung transplant recipients are especially vulnerable to air pollution due to direct exposure of the transplanted lungs. Given their varied health backgrounds and immunosuppression levels, this group faces potential long-term risks. Recent studies link pollution to poorer outcomes, such as reduced lung function and higher mortality from chronic allograft dysfunction. While more evidence is needed, protecting transplanted lungs from environmental harm remains a critical priority.

2.4.1. SHORT TERM HEALTH EFFECTS:

Szyszkowicz 2018 has stated that the short-term exposure to ambient air pollution is very important in terms of environmental epidemiology.

Rajak, R., & Chattopadhyay, A. 2019, in their study titled 'Short- and Long-Term Exposure to Ambient Air Pollution and Impact on Health in India: A Systematic Review', have looked into a vast detail of epidemiological studies reporting health effects in India caused by Ambient Air Pollution. Most of the studies taken for analysis have attempted in establishing the prevalence of respiratory morbidity. Some of them use hospital admission or visits for treatment of air-related diseases like asthma, wheezing cough and other respiratory illnesses.

Most of the studies taken into consideration for their review have largely focused on major mega cities like Delhi, Mumbai, Chennai, Kolkata, etc and all state that elevated levels of pollution levels are the major causes creating incidence of diminished lung function, acute and chronic respiratory illnesses, wheezing, asthma in both children and adults.

An interesting note is that the people that are very sick or succumb to their illness are not the only ones who have short-term exposure brought on by sudden rises in PM2.5 and PM10. In reality, one or more risk factors account for a large portion of the morbidity and mortality that occur among active adults. About 11,394 deaths (total mortality), 3912

deaths (cardiovascular mortality), 1697 deaths (respiratory mortality), and 16,253 deaths (hospital admission of COPD) were recorded for the entire NCT Delhi in 2000, according to Nagpure et al. (2014). But in just ten years, in 2010, their numbers rose to 18,229, 6374, 2701, and 26,525 correspondingly. In 2015, 29,609.6 thousand (25,923.3–33 562.7) disability-adjusted life-years (DALYs) were caused by PM2.5 exposure in India, according to Cohen et al. (2017).

From their systematic review, it is clear that if ambient air pollution exceeds their normal or prescribed limits, increased results on morbidity, mortality and cases are observed.

2.4.2. LONG TERM HEALTH EFFECTS

Long-term effects of Ambient Air Pollution have been associated with chronic bronchitis, markers of atherosclerosis, respiratory impairment, lung cancer and mortality (Public Health and Air Pollution in Asia [PAPA], 2011; Balakrishnan et al. 2013; Ghosh & Mukherjee 2014; Tobollik et al. 2015; Gawande et al. 2016; & Maji et al. 2016). Studies support the positive associations between ambient pollutants (PM_{10}, NO_2 & SO_2) and high risk of mortality. Evidence state that long-term exposure to Ambient Air Pollution in India is associated with asthma cases, reduced lung function, lung cancer, non-trauma death, cardiovascular deaths, respiratory deaths and premature death. The pooled effect estimates that the excess risk per 10 μg/m3 increase in PM_{10} exposure was 1.004 (1.002 to 1.007) for all-cause mortality in Tamil Nadu, and for Delhi, it was 1.0015 (1.0007 to 1.0023) [PAPA, 2011]. Another study suggests that long-term exposure to outdoor particulate air pollutant (PM_{10}) significantly increased the risk of pneumonia in children, i.e. 1.26 OR (CI: 1.00–1.57) for 6 μg/m3 increase in the concentration (Jiang et al. 2018). Also, ambient NO2 was a consistent risk factor for doctor-diagnosed asthma (OR 1.19 per 10 $\mu g/m^3$), doctor diagnosed rhinitis (OR 1.28 per 10 $\mu g/ m^3$), lifetime eczema (OR 1.16 per 10 $\mu g/m^3$) and current wheeze (OR 1.13 per 10 $\mu g/m^3$) among pre-school children (Norback et al. 2019). A prospective cohort study indicated that the risk of childhood allergic rhinitis was associated with traffic-related air pollutant, NO_2 during entire pregnancy with OR 1.38 (CI: 1.03–1.84) in single-pollutant model and OR 1.69 (CI: 1.03–2.77) in multi-pollutant model (Deng et al. 2016). In addition, case fatality also

depends on age, preexisting cardiovascular disease, obesity, low socioeconomic status smoking and other individual factors. Individual susceptibility to the health effects of air pollution may differ, due to either biological differences or behavioural differences affecting exposure (Stockfelt 2017).

WHO, 2024, has stated that ambient (outdoor) air pollution in both cities and rural areas was estimated to cause 4.2 million premature deaths worldwide per year in 2019 and this mortality was caused due to exposure to fine particulate matter, which causes cardiovascular and respiratory disease, and cancers.

WHO estimates that in 2019, some 68% of outdoor air pollution related premature deaths were due to ischaemic heart disease and stroke, 14% were due to chronic obstructive pulmonary disease, 14% were due to acute lower respiratory infections, and 4% of deaths were due to lung cancers.

People living in low- and middle-income countries disproportionately experience the burden of outdoor air pollution with 89% (of the 4.2 million premature deaths) occurring in these areas. The greatest burden is found in the WHO South-East Asia and Western Pacific Regions. The latest burden estimates reflect the significant role air pollution plays in cardiovascular illness and death.

CHAPTER 3 – OBJECTIVES:

The primary objective of this study is to examine the relationship between ambient air pollution and adverse health outcomes across selected Indian cities, with a specific focus on Tier 2 urban centres that are often underrepresented in pollution-health research. The study aims to quantify the effects of major ambient air pollutants namely PM_{10}, NO_2, and SO_2 on two key health indicators: medically certified cause-of-death (MCCD) data and acute respiratory infections (ARI). Additionally, the analysis investigates the role of weather variables such as rainfall and temperature, along with demographic factors like population density. By employing panel data econometric models, the study seeks to evaluate the magnitude and direction of these effects over time, identify pollutant-specific risks, and determine whether Tier 2 cities exhibit distinct vulnerability patterns compared to larger urban centres.

Therefore, the objectives of the study will be:

1. To understand and estimate the health effects caused by ambient air pollution on morbidity and mortality numbers.
2. To understand the role of weather in aggravating or dissipating pollution
3. Finally, to evaluate the health damages that could be observed by people in Tier 2 cities.

CHAPTER 4 - METHODOLOGY:

4.1.Study area:

India being the most populous country in the world with over 140 billion people, racing against all nations with USD 4.19 trillion GDP and ranking 4th in the world, there is a lot of economic activity going on is what can be told for sure. The country is vast with a total land area of 3.287 million sq km, the 7th largest country in the world/ The country's unique geographical settings with Himalayas in the north and peninsular surrounded by sea at the other 3 sides, the climate is different in each and every city and state. Lying entirely in the northern hemisphere, the mainland extends between latitudes 8° 4' and 37° 6' north, longitudes 68° 7' and 97° 25' east and measures about 3,214 km from north to south between the extreme latitudes and about 2,933 km from east to west between the extreme longitudes. It has a land frontier of about 15,200 km. The total length of the coastline of the mainland, Lakshadweep Islands and Andaman & Nicobar Islands is 7,516.6 km.

This study includes 5 mega cities (green pins) and 9 tier-2 cities (red pins) which are growing at an exponential rate hoping to turn into a mega city soon.

Fig 1: Area of the study (Green – Megacities, Red – Tier 2 cities) Credits: Google Earth

4.2.Data Description:

4.2.1. Medically Certified Cause of Death (MCCD)

Humans as mortals, enter earth at birth, live their life and henceforth, have an exit from earth after their period. Recording the events of birth and death, helps in statistical analyses on problems that may occur during or between the events. Death in here plays a major role in understanding the gravity of concepts like morbidity and mortality. These statistics play a major role in the policy and planning of national health.

In India, it is made mandatory by the passing of the Act – Registrations of Births and Deaths Act in 1969. The Registrar General of India is the highest official who receives the information from Registrars, Sub-Registrars who take note of the births and deaths at district, Corporation and municipality levels. As stated by Kotabagi, R., Chaturvedi, R., & Banerjee, A. (2004), "Registration of not only the occurrence of death, but also its cause is equally important.". They have discussed the details of the procedure to fill and the role of the medical officer in the process of registering the death.

The MCCD data is available from the 'Report of Medical Certification of Cause of Death' provided by the Office of the Registrar General, Ministry of Home Affairs, Government of India. The MCCD data are classified basis on various parameters like age group, sex, and the cause of death. The cause of deaths is coded as per the *International Classification of Diseases, Revision 10* (ICD-10). For this particular study, as we are dealing with the health effects of pollution, we would be considering 2 categories, viz, diseases of the circulatory system (I00-I99) and Diseases of the respiratory system(J00-J98). These are broad categories, and have several sub categories of diseases which are listed below. However, we will be dealing them as a whole and not individually with the sub-categories.

1. Diseases of the circulatory system (I00-I99)
    1.1.Acute rheumatic fever and chronic rheumatic heart diseases (I00-I09)
        1.1.1. Acute rheumatic fever (I00-102)
        1.1.2. Chronic rheumatic heart diseases (I05-I09)
    1.2.Hypertensive diseases (I05-I15)
        1.2.1. Hypertensive heart disease (I11)
        1.2.2. All other hypertensive diseases (I10, I12-I15)

1.3.Ischemic heart diseases (I20-I25)

1.3.1. Acute myocardial infarction (I21-I22)

1.3.2. All other ischaemic heart diseases (I20 & I23-I25)

1.4.Diseases of pulmonary circulation and other forms of heart disease (I26-I51)

1.4.1. Pulmonary heart disease and diseases of pulmonary circulation (I26-I28)

1.4.2. Other forms of heart diseases (I30-I51)

1.5.Cerebrovascular diseases (I60-I69)

1.6.Other diseases of the circulatory system (I70-I99)

1.6.1. Atherosclerosis (I70)

1.6.2. Arterial embolism and thrombosis (I74)

1.6.3. Other diseases of arteries, arterioles & capillaries (I71-I73 & I77-I78)

1.6.4. Phlebitis, thrombophlebitis, venous embolism and thrombosis (I80-I82)

1.6.5. All other diseases of the circulatory system (I83-I99)

2. Diseases of the respiratory system(J00-J98)

2.1.Diseases of the upper respiratory tract (J00-J06 & J30-J39)

2.1.1. Acute pharyngitis and acute tonsillitis (J02-J03)

2.1.2. Acute laryngitis and tracheitis (J04)

2.1.3. Other acute upper respiratory infections (J00-J01 & J05-J06)

2.1.4. All other diseases of upper respiratory tract (J30-J39)

2.2.Lower respiratory diseases (J20-J22 & J40-J47)

2.2.1. Acute bronchitis and acute bronchiolitis (J20-J21)

2.2.2. Bronchitis, chronic and unspecified, emphysema (J40-J43)

2.2.3. Asthma (J45-J46)

2.2.4. Other lower respiratory disorders (J22, J44 & J47)

2.3.Other diseases of the respiratory system (J10-J18, J60-J98)

2.3.1. Influenza (J10-J11)

2.3.2. Pneumonia (J12-J18)

2.3.3. Pleurisy (J90)

2.3.4. All other diseases of the respiratory system (J60-J86, J92-J98.)

4.2.2. Down-Scaling State level data to City level

4.2.2.1.$MCCD_{city}$

The MCCD data for our study will be an aggregate of the **Circulatory Diseases (CD)** and **Respiratory Diseases (RD)** which are in state level terms. As our study is based on city level, we will be reducing (or) down-scaling the state level to proxy for city level by using weights. One weight will be based on the number of vehicle registrations in the city to the number of vehicle registrations in their respective state. The numbers are taken from the Road Transport Year book provided by The Ministry of Road, Transport and Highways, Government of India. Another weight will be based on the area which is taken from each city's corporation or municipality website to find out the area under the city limits. State area is taken from geographical data provided by indiastats.com.

Vehicle numbers are used here as a proxy to signify that people with vehicle are exposed to outdoor air pollution, considering them as the attributable population. Area on the other hand will be to signify the area downscaled from state to city. Missing values in vehicle numbers and MCCD were carefully averaged from future years for the missing years.

$$MCCD_{state} = CD + RD$$

$$MCCD_{city} = MCCD_{state}\left[50\%\left(\frac{vehicle_{city}}{vehicle_{state}}\right) + 50\%\left(\frac{area_{city}}{area_{state}}\right)\right]$$

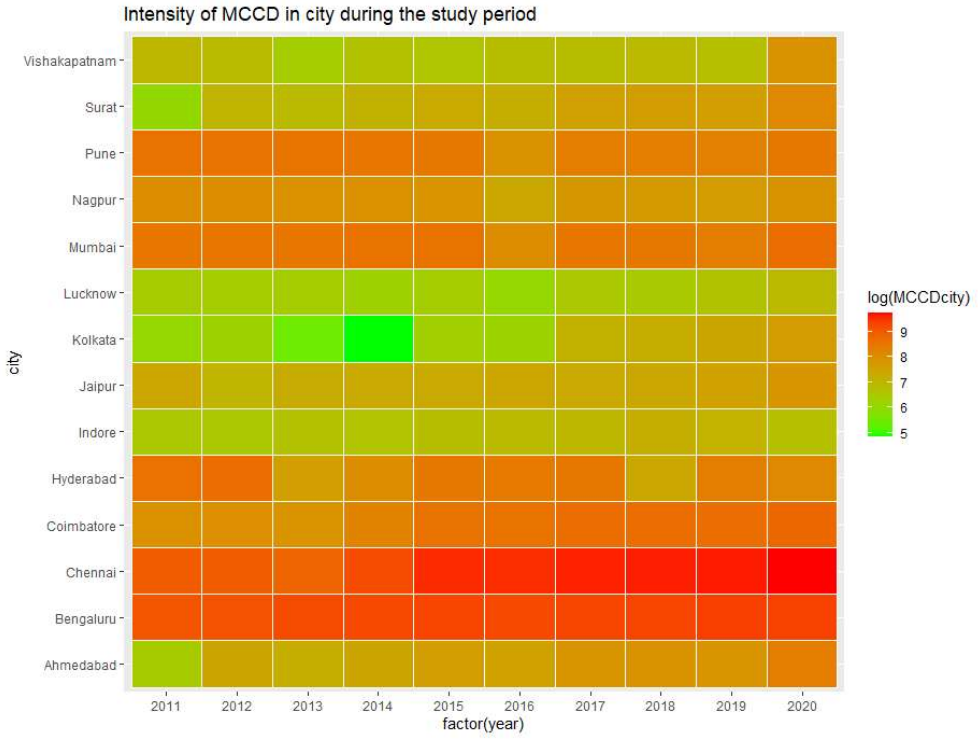

Fig 2: Intensity of MCCD deaths in cities during the study period
(source: MCCD report and author calculations)

4.2.2.2.Acute Respiratory Illness

Higher short-term exposure to Ambient Air Pollutants are associated with increased risk of symptomatic acute respiratory infections among adults. The data is collected from the National Health Profile from the Central Bureau of Health Intelligence under the Directorate General of Health Services, Ministry of Health & Family Welfare, Govt. of India. The number of cases registered per year for Acute Respiratory Infection is taken in for this study. This will be our dependent variable for the 2nd model and will be examined to understand the health effect on the cases rather than the mortality data. Just like MCCD the number of cases registered for ARI is in state level. Therefore, we will be downscaling in the same method followed for MCCD before.

$$ARI_{city} = ARI_{state}\left[50\%\left(\frac{vehicle_{city}}{vehicle_{state}}\right) + 50\%\left(\frac{area_{city}}{area_{state}}\right)\right]$$

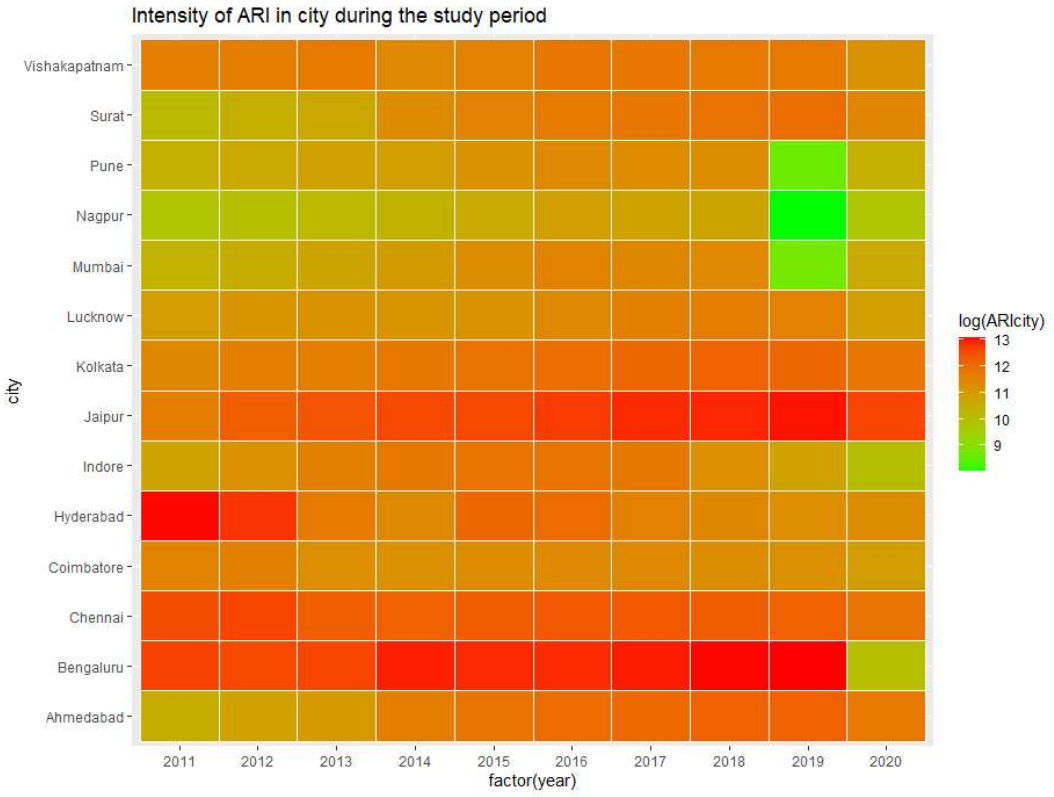

Fig 3: Intensity of ARI cases in cities during the study period
(source: MCCD report and author calculations)

4.2.3. Pollution data:

Yearly air pollution data for NO_2, SO_2 and PM_{10} ranges from 2011 to 2020, a period of 10 years, obtained from the Central Pollution Control Board (CPCB). The **CPCB**, a statutory organisation, was constituted in September, 1974 under the Water (Prevention and Control of Pollution) Act, 1974. Further, CPCB was entrusted with the powers and functions under the Air (Prevention and Control of Pollution) Act, 1981.

It serves as a field formation and also provides technical services to the Ministry of Environment and Forests of the provisions of the Environment (Protection) Act, 1986. Principal Functions of the CPCB, as spelt out in the Water (Prevention and Control of Pollution) Act, 1974, and the Air (Prevention and Control of Pollution) Act, 1981, (i) to promote cleanliness of streams and wells in different areas of the States by prevention,

control and abatement of water pollution, and (ii) to improve the quality of air and to prevent, control or abate air pollution in the country.

Air Quality Monitoring is an important part of the air quality management. The **National Air Monitoring Programme (NAMP)** has been established with objectives to determine the present air quality status and trends and to control and regulate pollution from industries and other source to meet the air quality standards. It also provides background air quality data needed for industrial siting and towns planning.

The National Air Quality Monitoring Programme (NAMP) is a nation-wide programme of ambient air quality monitoring. The network consists of **966** operating stations in **419** cities/towns in 28 states and 7 Union Territories of the country as on 19.11.2024.

The objectives of the NAMP are to determine status and trends of ambient air quality; to ascertain whether the prescribed ambient air quality standards are violated; to Identify Non-attainment Cities; to obtain the knowledge and understanding necessary for developing preventive and corrective measures and to understand the natural cleansing process undergoing in the environment through pollution dilution, dispersion, wind based movement, dry deposition, precipitation and chemical transformation of pollutants generated.

Under NAMP, four air pollutants viz., Sulphur Dioxide (SO2), Oxides of Nitrogen as NO2, Respirable Suspended Particulate Matter (RSPM / PM10) and Fine Particulate Matter (PM2.5) have been identified for regular monitoring at all the locations. The monitoring of meteorological parameters such as wind speed and wind direction, relative humidity and temperature were also integrated with the monitoring of air quality.

The monitoring of pollutants is carried out for 24 hours (4-hourly sampling for gaseous pollutants, 8-hourly sampling for particulate matter and 24-hourly sampling for PM2.5) with a frequency of twice a week, to have one hundred and four (104) observations in a year. The monitoring is being carried out with the help of Central Pollution Control Board; State Pollution Control Boards; Pollution Control Committees and other agencies. CPCB co-ordinates with these agencies to ensure the uniformity, consistency of air quality data and provides technical and financial support to them for operating the monitoring stations. NAMP is being operated through various monitoring agencies. Large number of

personnel and equipment are involved in the sampling, chemical analyses, data reporting etc. It increases the probability of variation and personnel biases reflecting in the data; hence it is pertinent to mention that these data be treated as indicative rather than absolute.

We will be using **Annual Averages of NO_2, SO_2 and PM_{10}** data in this study as it was fully available in the years taken for our study. $PM_{2.5}$, even though an important part in the study of estimating health effects from ambient air pollution, data remains uneven and erratic in nature causing us to omit for this study. Missing values were carefully averaged from nearby cities as a proxy.

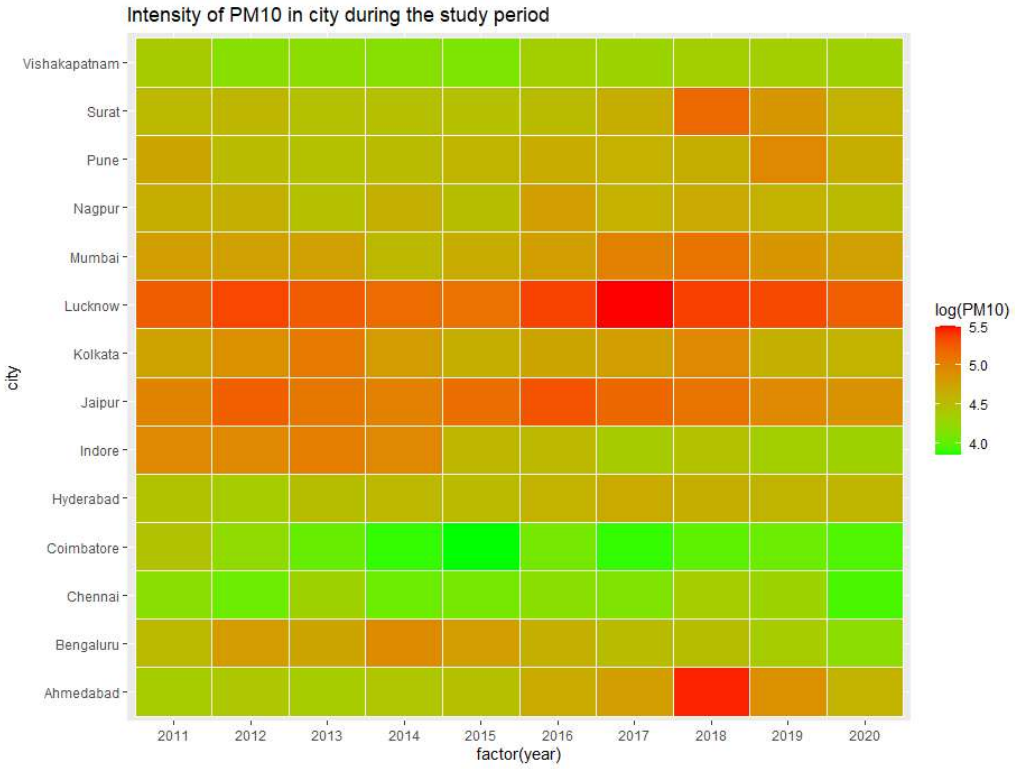

Fig 4: Intensity of PM_{10} in cities during the study period
(source: NAMP data and author calculations)

Fig 5: Intensity of NO_2 cases in cities during the study period
(source: NAMP data and author calculations)

Fig 6: Intensity of SO_2 cases in cities during the study period
(source: NAMP data and author calculations)

4.2.4. Weather data:

The weather patterns in India are quite random in nature but the seasons are not. South western monsoons at the end of the year and the retreating monsoons in July to august are the major rainy seasons which largely impacts the impact of summer. Huge temperature variations across the cities are observed on basis of their location and its surrounding geographies. Weather patterns have a major control over the amount of pollution suspended in the air. Several investigations have suggested that the influence of weather over air pollution concentrations have a significantly time and spatial variability due to the different types of ambient air pollutants and the complex process related to their formation and removal. High temperatures can also increase oxidation and production of sulphate particles in the atmosphere. In addition, other studies have shown that fine particulate matter ($PM_{2.5}$) is correlated with wind speed (advection and turbulence favour $PM_{2.5}$ dispersion) and precipitation (wet deposition and removal favour $PM_{2.5}$ decrease). (Castelhano, F. J., & Réquia, W. J.,2024)

The control variables of weather included in this study are as follows:

1. **Average Annual Rainfall** which are in district level obtained from the rainfall statistics of India, Indian Meteorological Department (IMD), which will be used as a proxy for the selected cities. Rainfall for the year 2011 was interpolated using averages of the next 2 years.
2. **Rainmax** which is the maximum rainfall observed in a day of the year, are sourced from IMD compiled by indiastat.com
3. **Tmin and Tmax** which are minimum and maximum temperatures recorded in a year also sourced from indiastat.com

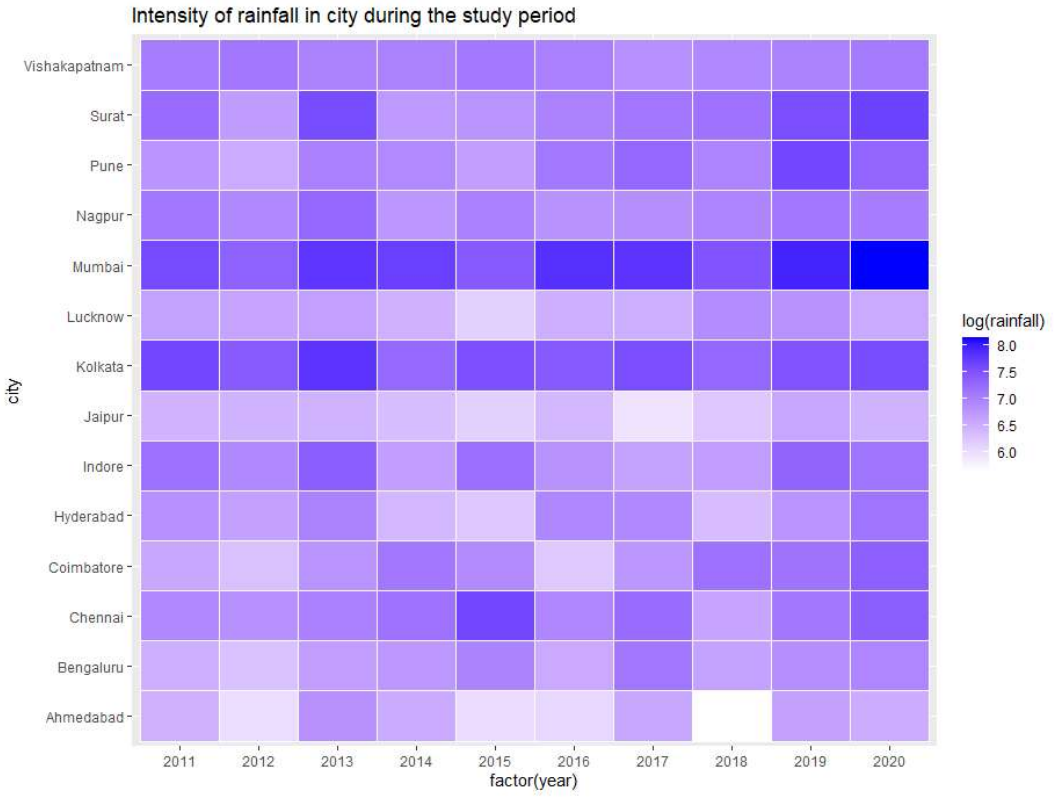

Fig 7: Intensity of Rainfall in cities during the study period
(source: Rainfall Statistics of India and author calculations)

4.2.5. Demographic data

Under the Ministry of Health & Family Welfare, the National Commission on Population have released the Report of the Technical Group on Population Projections in the month of November, 2019. This report contains the population projections for India and its states for the years 2011-2036. Using this report, the population density variable has been estimated with the help population projection estimates. As the data in the report is in state level terms, we first find out the growth proportion of urban population in each selected states taken for our study. The population for city is taken from the 'population finder' tool in the censusindia.gov.in website. The urban population of the said district is taken as a proxy to represent the population as of the city.

The area that is to be divided from the population is taken from each city's corporation or municipality website to find out the area under the city limits.

And therefore,

$$state\ urban\ popln\ growth\ prop_n = \frac{state\ urban\ popln_n - state\ urban\ popln_{n-1}}{state\ urban\ popln_{n-1}}$$

$$popln_{n+1} = city\ urban\ popln_{2011} + (city\ urban\ popln_{2011} * state\ urban\ popln\ growth\ prop)$$

$$popdens = \frac{population_{city}}{area_{city}}$$

4.2.6. Dummy variable

To observe the individual effects in Tier 2 cities, one dummy variable has been added.

1 if city is a Tier 2 city

0 otherwise.

4.3.Summary statistics of the data

Table 6: Summary Statistics

Variable	Min	1st Quartile	Median	Mean	3rd Quartile	Max
CD	1,598	12,213	23,629	42,275	70,816	1,43,453
RD	901	5,094	8,398	11,294	19,486	27,878
MCCD	2,837	17,119	31,185	53,568	93,319	1,71,331
vmnos	4,44,718	14,62,532	21,86,054	25,11,917	31,68,510	96,38,362
vstnos	43,61,786	1,09,52,890	1,74,34,099	1,83,96,841	2,44,52,782	3,77,86,256
pop	20,35,922	31,79,972	40,73,541	46,02,387	54,41,309	1,05,55,041
cityarea	205	426	527	479.4	603	709
statearea	88,752	1,30,060	1,96,244	2,15,911	3,07,713	3,42,239
popdens	3,702	5,758	11,051	10,721	13,489	26,130
MCCDcity	129.3	1,084.80	2,560.60	3,807.70	5,028.60	17,234.40
ARI	1,19,674	11,46,185	18,08,545	18,26,523	22,61,725	53,46,060
ARIcity	3,154	66,191	1,08,716	1,38,374	1,75,045	4,73,358
NO2	13	20	26	30.04	32	87
SO2	2	6	9	10.4	13	37
PM10	47	79.75	100.5	109.46	128.25	246
rainfall	285.9	767.4	1,023.40	1,138.10	1,320.50	3,444.10
Tmin	-0.7	5.7	10.45	9.941	13.8	18.5
Tmax	35.2	39.6	42.5	42.06	44.4	48
rainmax	38	83.35	108.4	121.73	143.78	345.1
tier2city	0	0	1	0.6429	1	1

4.4.MODELS

In panel data analysis, where observations span across multiple entities (e.g., individuals, cities, firms) over time, unobserved heterogeneity can bias estimates if not appropriately addressed. Two common approaches to control for this unobserved heterogeneity are **Fixed Effects (FE)** and **Random Effects (RE)** models.

4.4.1. Fixed Effects (FE) Model

The Fixed Effects model accounts for time-invariant characteristics of the entities by allowing each entity to have its own intercept. It effectively removes all unobserved heterogeneity that does not vary over time.

Econometric Equation:

$$Y_{it} = \alpha_i + \beta X_{it} + \varepsilon_{it}$$

Where,

- Y_{it} is the dependent variable for entity *i* at time *t*
- α_i captures entity specific intercepts (fixed effects)
- X_{it} is a vector of explanatory variables
- B is a vector of coefficients
- ε_{it} is the idiosyncratic error term

Fixed Effects Models are useful when the omitted variables could possibly correlate with the regressors.

4.4.2. Random Effects (RE) Model

The Random Effects model assumes that individual-specific effects are random and uncorrelated with the explanatory variables. It models unobserved heterogeneity as part of the error term.

Econometric Equation:

$$Y_{it} = \alpha + \beta X_{it} + u_i + \varepsilon_{it}$$

Where,

- α is a common intercept
- u_i is the random effect (entity-specific component of the error term)
- Y_{it} is the dependent variable for entity i at time t
- X_{it} is a vector of explanatory variables
- β is a vector of coefficients
- ε_{it} is the idiosyncratic error term

Random Effects model is more efficient than Fixed Effects Model if the assumption that $\text{Cov}(X_{it},U_i) = 0$ holds. However, this is a strong assumption and often tested using the Hausman test.

4.4.3. Hausman Tests:

Hausman test is a statistical test used in panel data analysis to decide between the **Fixed Effects (FE)** and **Random Effects (RE)** models. It tests whether the unique errors (unobserved effects) are correlated with the regressors.

- **Null Hypothesis (H_0):** Random Effects model is appropriate (i.e., no correlation between U_i and X_{it})
- **Alternative Hypothesis (H_1):** Fixed Effects model is appropriate (i.e., correlation exists)

If the RE estimator is consistent and efficient under the null, and FE is consistent under both null and alternative, then a significant difference in the coefficients implies violation of the RE assumption.

Decision Rule:

- If the **p-value < 0.05**, reject H_0 → use **Fixed Effects**
- If the **p-value ≥ 0.05**, fail to reject H_0 → use **Random Effects**

4.4.4. Econometric Models

Model 1: 2 dependent variables MCCDcity & ARIcity (without dummy variable)

Model 1.1

$$\log(MCCD_{city}) \sim \beta_0 + \beta_1 NO2_{it} + \beta_2 SO2_{it} + \beta_3 PM10_{it} + \beta_4 \log(rainfall_{it}) + \beta_5 Tmin_{it} + \beta_6 Tmax_{it} + \beta_7 \log(popdens_{it}) + V_{it}$$

Where, $V_{it} = a_i + u_{it}$

Model 1.2

$$\log(ARI_{city}) \sim \beta_0 + \beta_1 NO2_{it} + \beta_2 SO2_{it} + \beta_3 PM10_{it} + \beta_4 \log(rainfall_{it}) + \beta_5 Tmin_{it} + \beta_6 Tmax_{it} + \beta_7 \log(popdens_{it}) + V_{it}$$

Where, $V_{it} = a_i + u_{it}$

Model 2: 2 dependent variables MCCDcity & ARIcity with a dummy variable specifying Tier 2 cities

Model 2.1

$$\log(MCCD_{city}) \sim \beta_0 + \beta_1 NO2_{it} + \beta_2 SO2_{it} + \beta_3 PM10_{it} + \beta_4 \log(rainfall_{it}) + \beta_5 Tmin_{it} + \beta_6 Tmax_{it} + \beta_7 \log(popdens_{it}) + \delta_1 tier2city_i + V_{it}$$

Where, $V_{it} = a_i + u_{it}$

Model 2.2

$$\log(ARI_{city}) \sim \beta_0 + \beta_1 NO2_{it} + \beta_2 SO2_{it} + \beta_3 PM10_{it} + \beta_4 \log(rainfall_{it}) + \beta_5 Tmin_{it} + \beta_6 Tmax_{it} + \beta_7 \log(popdens_{it}) + \delta_1 tier2city_i + V_{it}$$

Where, $V_{it} = a_i + u_{it}$

CHAPTER 5: RESULTS & DISCUSSIONS

5.1.Model Validation Tests:

5.1.1. Correlation Matrix

There is no perfect correlation between the variables taken for the study in the models. Correlation values range from -0.643 and 0.606 and higher values arise under the same groups of variables. *(See APPENDIX – I)*

5.1.2. Variance Inflation Factor

VIF values for all the variables that were included in the models were checked and the values were found to be approximately around 1. This indicates that there is a very low degree of multicollinearity. Therefore, the model results are not biased due to multicollinearity, supporting the robustness of the estimated effects. *(See APPENDIX – II)*

5.1.3. Hausman Tests

Hausman tests reveal that Random Effects Models explain better for all the models except for Model 2.1 suggesting Fixed Effects Model as the best.
(See APPENDIX – III)

5.2.Model Results:

Table 7: Model 1

Panel Models: MCCD and ARI (Without Dummy)

Dependent Variable		log(MCCDcity)		log(ARIcity)	
		MCCD FE	MCCD RE	ARI FE	ARI RE
Pollution					
	NO2	-0.025***	-0.025***	-0.018**	-0.014*
		(-0.005)	(-0.005)	(-0.008)	(-0.007)
	SO2	0.022**	0.021**	0.012	0.007
		(-0.01)	(-0.01)	(-0.017)	(-0.015)
	PM10	-0.0001	0.0001	0.006**	0.006***
Weather		(-0.001)	(-0.001)	(-0.002)	(-0.002)
	log(rainfall)	0.056	0.088	-0.404**	-0.483***
		(-0.117)	(-0.115)	(-0.192)	(-0.177)
	Tmin	-0.004	0.015	0.073	0.076**
		(-0.027)	(-0.024)	(-0.045)	(-0.03)
	Tmax	-0.038	-0.042*	0.044	0.028
Demographic		(-0.025)	(-0.024)	(-0.041)	(-0.035)
	log(popdens)	2.188***	1.622***	-0.09	0.184
		(-0.4)	(-0.325)	(-0.657)	(-0.33)
	Constant		-5.525*		10.924***
			(-3.067)		(-3.506)
	Observations	140	140	140	140
	R2	0.348	0.312	0.156	0.158
	Adjusted R2	0.239	0.276	0.015	0.113

Note: *p<0.1; **p<0.05; ***p<0.01; Standard Error in Parentheses

For model 1, Hausman tests suggests to choose RE model over FE model. Model 1 is to understand how pollution, weather and demographic factors decide on mortality (MCCD) and morbidity (ARI) values.

For the model with **log(MCCDcity)** as the dependent variable, SO_2 exhibits a statistically significant positive association, suggesting that a 1-unit increase in **SO_2** levels is associated with a **2.1% increase in medically certified deaths**. Interestingly, NO_2 displays a significant **negative** coefficient, indicating a 2.5% reduction in deaths per unit increase, which may reflect unobserved heterogeneity or measurement issues, as this result contradicts established literature. PM_{10} and rainfall are statistically insignificant in this model, while maximum temperature (Tmax) shows a marginally significant negative effect, potentially linked to seasonal changes. Population density has a strong and highly significant positive association with MCCD, highlighting the urban mortality burden.

In the second model with **log(ARIcity)** as the dependent variable, the results indicate that PM_{10} is positively and significantly associated with ARI cases, reinforcing its well-known respiratory health impacts. Rainfall, on the other hand, is negatively associated with ARI, and the relationship is highly significant, likely due to pollutant washout during rainy periods. Minimum temperature (Tmin) also shows a significant positive relationship with ARI, suggesting that warmer nights might influence the spread or reporting of infections. As with the MCCD model, NO_2 again shows a small but significant negative effect. Other predictors such as SO_2, Tmax, and population density are not statistically significant in explaining ARI variation.

Concerns are with the negative estimate arising with NO_2 pollution. Literature has very well-established detrimental health outcomes arising due to exposure to the pollutant in both short term as well as long term periods.

Table 8: Model 2

Panel Models: MCCD and ARI (With Dummy)				
Dependent Variable	log(MCCDcity)		log(ARIcity)	
	MCCD FE	MCCD RE	ARI FE	ARI RE
NO2	-0.025***	-0.026***	-0.018**	-0.015**
	(-0.005)	(-0.005)	(-0.008)	(-0.007)
SO2	0.022**	0.022**	0.012	0.011
	(-0.01)	(-0.01)	(-0.017)	(-0.015)
PM10	-0.0001	0.0001	0.006**	0.006***
	(-0.001)	(-0.001)	(-0.002)	(-0.002)
log(rainfall)	0.056	0.085	-0.404**	-0.506***
	(-0.117)	(-0.115)	(-0.192)	(-0.177)
Tmin	-0.004	0.011	0.073	0.059*
	(-0.027)	(-0.025)	(-0.045)	(-0.032)
Tmax	-0.038	-0.039	0.044	0.039
	(-0.025)	(-0.024)	(-0.041)	(-0.035)
tier2city		-0.561		-0.654
		(-0.595)		(-0.439)
log(popdens)	2.188***	1.584***	-0.09	0.141
	(-0.4)	(-0.323)	(-0.657)	(-0.324)
Constant		-4.895		11.618***
		(-3.084)		(-3.479)
Observations	140	140	140	140
R2	0.348	0.316	0.156	0.172
Adjusted R^2	0.239	0.274	0.015	0.122

Note: *p<0.1; **p<0.05; ***p<0.01; Standard Error in Parentheses

For Model 2, Hausman tests suggest FE model for MCCD and RE model for ARI dependent variables. Model 2 is to specifically understand how Tier 2 cities perform using the 'tier2city' dummy variable.

In the MCCD model, the results indicate that **SO_2** is positively and significantly associated with medically certified deaths: a one-unit increase in SO_2 levels is linked to approximately a **2.2% increase** in MCCD. In contrast, **NO_2** shows a statistically significant negative association, with each unit increase in NO_2 corresponding to about a **2.5% decrease** in MCCD. Although it is not what we expected, this may be due to

confounding factors, measurement issues, or city-specific characteristics captured in the fixed effects. PM_{10}, rainfall, and temperature variables do not show any significant effect in this model. However, **population density** continues to have a strong and significant impact, with more densely populated cities experiencing higher MCCD levels, underscoring the role of urban crowding and infrastructure stress.

For the ARI model, under the Random Effects specification, the findings show that **PM_{10}** is a significant risk factor for respiratory infections. A one-unit increase in PM_{10} levels is associated with a **0.6% rise** in ARI cases. Conversely, **rainfall** continues to exhibit a protective effect, with higher rainfall linked to a **roughly 50% reduction** in ARI incidence, likely due to the washing out of pollutants from the air. **Minimum temperature** also has a positive effect—higher night-time temperatures are associated with an **increase in ARI cases**, potentially due to seasonal shifts in patterns or altered pollutant chemistry. NO_2 again shows a small but statistically significant **decrease in ARI cases** with rising levels, though the effect is modest and may reflect unmeasured urban factors. The dummy variable for **Tier 2 cities** is negative but statistically insignificant, suggesting no clear difference in ARI outcomes between Tier 2 and mega cities after accounting for pollution and weather variables. Population density and SO_2 do not show any statistically meaningful effect in this model.

Model 3

A third model was done to understand if having single pollutant as the independent variable would change things. Here, we have 3 separate models for the 3 different pollution parameters – NO_2, SO_2, PM_{10}. RE was used to understand the models. The outputs are given in '*APPENDIX – IV'*

In the **NO_2-only model**, a 1-unit increase in NO_2 is significantly associated with a **2.2% decrease** in MCCD and a **0.8% decrease** in ARI. Although statistically significant, this inverse relationship contradicts epidemiological expectations and may be attributed to multicollinearity with other excluded variables, differential exposure patterns, or reporting issues in the health data.

In the **SO_2-only model**, SO_2 does not show a statistically significant relationship with either MCCD or ARI. This result suggests that, when SO_2 is considered in isolation, its effect on health outcomes may be muted or overshadowed by co-occurring pollutants or omitted confounders.

In contrast, the **PM_{10}-only model** reveals a significant association with ARI: a 1-unit rise in PM_{10} corresponds to a **0.5% increase** in ARI cases, confirming the established link between particulate pollution and respiratory health. However, no significant relationship is observed between PM_{10} and MCCD in this isolated specification.

Across all three pollutant-specific models, **rainfall consistently shows a large and statistically significant negative effect** on ARI, suggesting that higher precipitation levels help mitigate air pollution concentrations and related respiratory infections. Specifically, each logged unit increase in rainfall is associated with a **roughly 50% reduction** in ARI cases across models. **Minimum temperature (Tmin)** is also positively associated with ARI, indicating that warmer night-time temperatures may exacerbate respiratory infection burdens, possibly by influencing pathogen survival or human exposure. The effect of **maximum temperature (Tmax)** is generally insignificant or weakly negative for MCCD.

Population density remains a strong and significant positive predictor for MCCD across all pollutant-specific models, reinforcing the urban mortality burden driven by crowding and infrastructure stress. However, its effect on ARI is small and statistically insignificant, possibly due to differences in health-seeking behaviour or exposure-response lags.

Overall, Model 3 confirms that PM_{10} is the most consistent pollutant associated with adverse respiratory outcomes, while the effects of NO_2 and SO_2 are either inconsistent or counterintuitive when modelled in isolation. These results underscore the importance of multi-pollutant models and the role of environmental conditions in shaping pollution-health relationships.

CHAPTER 6: CONCLUSION & PROSPECTS

6.1.Conclusion

This study examines the impact of ambient air pollution on public health outcomes across cities in India, having a particular focus in Tier 2 cities. We had analysed the effects by structuring a panel data from 2011 to 2020, a period of 10 years with wide range variables of pollution, weather, demographic factors. The analysis employed both Fixed and Random Effect models to explore the relationship between pollution and 2 health indicators: Medically Certified Cause of Death (MCCD) and Acute Respiratory Infection (ARI) cases.

The results show that among the major pollutants, **particulate matter (PM_{10})** has the most consistent and statistically significant association with increased ARI cases. This reaffirms PM_{10}'s critical role in aggravating respiratory conditions. In contrast, **nitrogen dioxide (NO_2)** showed an inverse relationship with health outcomes in several models, a result that, while statistically significant, may be influenced by data limitations, omitted variable bias, or underlying structural dynamics in urban environments. **Sulphur dioxide (SO_2)** was positively associated with MCCD in the full model but showed weaker or non-significant effects when modelled independently.

The inclusion of the dummy variable did not lead to significant differences in pollution-health relationships, suggesting that once factors like pollution, temperature, and rainfall are accounted for, Tier 2 cities do not systematically differ from larger urban centres in health vulnerability. Nonetheless, population density emerged as a strong and consistent predictor of MCCD, highlighting the challenges of public health management in densely populated areas. Weather variables, especially rainfall and minimum temperature, also played an important role. Higher rainfall was associated with lower ARI cases, likely due to its role in washing out air pollutants, while warmer night temperatures were associated with increased respiratory illness.

Overall, the study contributes to the growing evidence that ambient air pollution poses a serious public health risk in Indian cities that are often underrepresented in environmental health research. The findings underscore the need for pollution control policies that target

not only large metropolitan areas but also the rapidly growing Tier 2 cities, where exposure levels may be high and healthcare infrastructure more limited.

6.2.Prospects

While the panel approach helps in controlling for unobserved heterogeneity across cities, there are major limitations in this study. A part of the data, even when derived from trustable sources which majorly included government reports and publications had missing or data not reported or were very ambiguous to add in our dataset. A proper accountable data if monitored properly would have a major impact in the analysis. Majorly for city level, not much of data has been reported or has been collected. Some data in our study had to be scaled down from state level to represent values from cities. And only yearly data were mostly available, shorter time periods like months, weeks, days seem to be inaccurate.

During the initial phases of this research, pollution studies majorly focused on 2 parts. One, was to use the AirQ+ software given by the WHO, which is used to derive short-term and long-term health effects by inputting pollution data and attributable proportion of population. The model evaluates based Impact Assessment Evaluation and Life tables by calculating Relative Risk and provides results on disease specific terms.

Two, was to forecast the pollution using Long Short-Term Memory or LSTM model. A part of Deep Learning and Neural Networks specifically Recurrent Neural Networks. Studies suggested that this model was accurate in forecasting with several hidden layers on which the model is built. Studies had also included weather patterns like humidity, rainfall, temperature, wind speed into this model.

Future works in this topic could try to combine future forecasts of pollution parameters along with predicting possible health effects that could arise from the future pollution. This could deeply mending policies accordingly and save people from morbidities and mortalities from ambient air pollution.

APPENDICES

APPENDIX – I

Correlation Matrix

	MCCDcity	ARIcity	PM10	NO2	SO2	rainfall	Tmin	Tmax	popdens
MCCDcity	1								
ARIcity	0.401	1							
PM10	-0.384	0.078	1						
NO2	-0.166	-0.063	0.264	1					
SO2	-0.085	-0.19	0.02	0.444	1				
rainfall	0.07	-0.288	-0.108	0.156	-0.026	1			
Tmin	0.559	0.091	-0.643	-0.298	-0.227	0.396	1		
Tmax	-0.395	-0.086	0.269	-0.088	0.141	-0.427	-0.606	1	
popdens	0.099	0.071	-0.101	0.443	0.192	0.141	0.049	-0.108	1

APPENDIX – II

Variance Inflation Factor (VIF)

vif(randommodelMCCD)

NO2	SO2	PM10	log(rainfall)	Tmin	Tmax	log(popdens)
1.245831	1.257014	1.162352	1.078721	1.092188	1.017396	1.085953

vif(randommodelARI)

NO2	SO2	PM10	log(rainfall)	Tmin	Tmax	log(popdens)
1.305967	1.23632	1.177264	1.078541	1.163041	1.044468	1.060053

vif(randomMCCD)

NO2	SO2	PM10	log(rainfall)	Tmin	Tmax	tier2city	log(popdens)
1.251359	1.260441	1.161926	1.079239	1.146133	1.041126	1.091014	1.086413

vif(randomARI)

NO2	SO2	PM10	log(rainfall)	Tmin	Tmax	tier2city	log(popdens)
1.332264	1.274495	1.186022	1.085575	1.342633	1.105066	1.339223	1.071687

APPENDIX - II

Hausman tests:

1. **Model 1 – Without Dummy**

   1.1.MCCD

   phtest(fixedmodelMCCD,randommodelMCCD)

   chisq = 11.543, df = 7, p-value = 0.1166

   alternative hypothesis: one model is inconsistent

   1.2.ARI

   phtest(fixedmodelARI,randommodelARI)

   chisq = 3.7711, df = 7, p-value = 0.8057

   alternative hypothesis: one model is inconsistent

2. **Model 2 – With Dummy**

   2.1.MCCD

   phtest(fixedMCCD,randomMCCD)

   chisq = 19.772, df = 7, p-value = 0.006083

   alternative hypothesis: one model is inconsistent

   2.2.ARI

   phtest(fixedARI,randomARI)

   chisq = 3.1885, df = 7, p-value = 0.867

   alternative hypothesis: one model is inconsistent

APPENDIX – IV

Model 3 – Taking independent pollution parameters

	NO2		SO2		PM10	
Dependent Variable	log(MCCDcity)	log(ARIcity)	log(MCCDcity)	log(ARIcity)	log(MCCDcity)	log(ARIcity)
NO_2	-0.022***	-0.008				
	(-0.005)	(-0.007)				
SO_2			0.001	0.004		
			(-0.01)	(-0.014)		
PM_{10}					-0.001	0.005**
					(-0.002)	(-0.002)
log(rainfall)	0.095	-0.545***	0.026	-0.579***	0.018	-0.548***
	(-0.116)	(-0.18)	(-0.125)	(-0.18)	(-0.125)	(-0.176)
Tmin	0.014	0.058*	0.035	0.067**	0.032	0.084***
	(-0.024)	(-0.03)	(-0.026)	(-0.03)	(-0.026)	(-0.029)
Tmax	-0.04	0.032	-0.048*	0.031	-0.047*	0.028
	(-0.024)	(-0.035)	(-0.026)	(-0.036)	(-0.026)	(-0.034)
log(popdens)	1.501***	0.16	1.354***	0.084	1.374***	0.087
	(-0.315)	(-0.329)	(-0.331)	(-0.324)	(-0.332)	(-0.303)
Constant	-4.453	12.130***	-3.136	12.739***	-3.152	11.918***
	(-3.004)	(-3.503)	(-3.179)	(-3.482)	(-3.177)	(-3.329)
Observations	140	140	140	140	140	140
R^2	0.283	0.105	0.159	0.097	0.162	0.138
Adjusted R^2	0.256	0.072	0.128	0.064	0.131	0.106

Note: *p<0.1; **p<0.05; ***p<0.01; Standard Error in Parentheses

APPENDIX – V

```
#R code using RStudio
#loading libraries and dataset
 library(readxl)
 library(plm)
 library(ggplot2)
 library(car)
 library(stargazer)
 options(scipen = 999)
 d <- read_xlsx("C:/Users/Dilip/Desktop/Dis data.xlsx", sheet = "Sheet1")
 df <- pdata.frame(d)
 #models
 #MCCD ~ without dummy
 fixedmodelMCCD <- plm(data=df, log(MCCDcity) ~ NO2 + SO2 + PM10 +
 log(rainfall) + Tmin + Tmax + log(popdens))
 randommodelMCCD <- plm(data=df, log(MCCDcity) ~ NO2 + SO2 + PM10 +
 log(rainfall) + Tmin + Tmax + log(popdens), model = "random")
 summary(fixedmodelMCCD)
 summary(randommodelMCCD)
 phtest(fixedmodelMCCD,randommodelMCCD)
 #ARI ~ without dummy
 fixedmodelARI <- plm(data=df, log(ARIcity) ~ NO2 + SO2 + PM10 + log(rainfall)
 + Tmin + Tmax + log(popdens))
 randommodelARI <- plm(data=df, log(ARIcity) ~ NO2 + SO2 + PM10 +
log(rainfall)
 + Tmin + Tmax + log(popdens), model = "random")
 summary(fixedmodelARI)
 summary(randommodelARI)
 phtest(fixedmodelARI,randommodelARI)
 #MCCD ~ all other vars
 fixedMCCD <- plm(data=df, log(MCCDcity) ~ NO2 + SO2 + PM10 + log(rainfall) +
 Tmin + Tmax + tier2city + log(popdens))
 randomMCCD <- plm(data=df, log(MCCDcity) ~ NO2 + SO2 + PM10 + log(rainfall) +
 Tmin + Tmax + tier2city + log(popdens), model = "random")
 summary(fixedMCCD)
 summary(randomMCCD)
```

```
phtest(fixedMCCD,randomMCCD)
#ARI ~ all other vars
fixedARI <- plm(data=df, log(ARIcity) ~ NO2 + SO2 + PM10 + log(rainfall) +
Tmin + Tmax + tier2city + log(popdens))
randomARI <- plm(data=df, log(ARIcity) ~ NO2 + SO2 + PM10 + log(rainfall) +
Tmin + Tmax + tier2city + log(popdens), model = "random")
summary(fixedARI)
summary(randomARI)
phtest(fixedARI,randomARI)
#pollution specific models
#NO2 - random
NO2MCCD <- plm(data=df, log(MCCDcity) ~ NO2 + log(rainfall) + Tmin + Tmax +
log(popdens), model = "random")
NO2ARI <- plm(data=df, log(ARIcity) ~ NO2 + log(rainfall) + Tmin + Tmax +
log(popdens), model = "random")
summary(NO2MCCD)
summary(NO2ARI)
#SO2 - random
SO2MCCD <- plm(data=df, log(MCCDcity) ~ SO2 + log(rainfall) + Tmin + Tmax +
log(popdens), model = "random")
SO2ARI <- plm(data=df, log(ARIcity) ~ SO2 + log(rainfall) + Tmin + Tmax +
log(popdens), model = "random")
summary(SO2MCCD)
summary(SO2ARI)
#PM10 - random
PM10MCCD <- plm(data=df, log(MCCDcity) ~ PM10 + log(rainfall) + Tmin + Tmax +
log(popdens), model = "random")
PM10ARI <- plm(data=df, log(ARIcity) ~ PM10 + log(rainfall) + Tmin + Tmax +
log(popdens), model = "random")
summary(PM10MCCD)
summary(PM10ARI)
#vif
vif(randommodelMCCD)
vif(randommodelARI)
vif(randomMCCD)
vif(randomARI)
```

```
#corr matrix
vars <- df[, c("MCCDcity", "ARIcity", "PM10", "NO2", "SO2", "rainfall",
"Tmin", "Tmax", "popdens")]
cor_matrix <- cor(vars, use = "pairwise.complete.obs")
print(round(cor_matrix, 3))
#stargazer outputs
#1
stargazer(fixedmodelMCCD, randommodelMCCD, fixedmodelARI, randommodelARI,
          type = "text",  # use "latex" or "html" for export
          title = "Panel Models: MCCD and ARI (Without Dummy)",
          column.labels = c("MCCD FE", "MCCD RE", "ARI FE", "ARI RE"),
          dep.var.labels = c("log(MCCDcity)", "log(ARIcity)"),
          model.numbers = FALSE,
          digits = 3,
          omit.stat = c("f", "ser"))
#2
stargazer(fixedMCCD, randomMCCD, fixedARI, randomARI,
          type = "text",
          title = "Panel Models: MCCD and ARI (With Tier2 Dummy)",
          column.labels = c("MCCD FE", "MCCD RE", "ARI FE", "ARI RE"),
          dep.var.labels = c("log(MCCDcity)", "log(ARIcity)"),
          model.numbers = FALSE,
          digits = 3,
          omit.stat = c("f", "ser"))
#3
stargazer(NO2MCCD, NO2ARI, SO2MCCD, SO2ARI, PM10MCCD, PM10ARI,
          type = "text",
          title = "Random Effects Models by Pollutant: MCCD vs ARI",
          column.labels = c("NO2-MCCD", "NO2-ARI", "SO2-MCCD", "SO2-ARI",
"PM10-MCCD", "PM10-ARI"),
          dep.var.labels = c("log(MCCDcity)", "log(ARIcity)"),
          model.numbers = FALSE,
          digits = 3,
          omit.stat = c("f", "ser"))
#heatmaps
ggplot(df, aes(x = factor(year), y = city, fill = log(MCCDcity))) +
```

```
 geom_tile(color = "white") +
 scale_fill_gradient(low = "white", high = "black") +
 labs(title="intensity of MCCD in city during the study period")
labs(x = "Year", y = "City", fill = "log(MCCDcity)") +
 theme_minimal()
ggplot(df, aes(x = factor(year), y = city, fill = log(ARIcity))) +
 geom_tile(color = "white") +
 scale_fill_gradient(low = "white", high = "black") +
 labs(title="intensity of ARI in city during the study period")
labs(x = "Year", y = "City", fill = "log(MCCDcity)") +
 theme_minimal()
ggplot(df, aes(x = factor(year), y = city, fill = log(rainfall))) +
 geom_tile(color = "white") +
 scale_fill_gradient(low = "white", high = "black") +
 labs(title="intensity of rainfall in city during the study period")
labs(x = "Year", y = "City", fill = "log(MCCDcity)") +
 theme_minimal()
ggplot(df, aes(x = factor(year), y = city, fill = log(popdens))) +
 geom_tile(color = "white") +
 scale_fill_gradient(low = "white", high = "black") +
 labs(title="population density in city during the study period")
 labs(x = "Year", y = "City", fill = "log(MCCDcity)") +
 theme_minimal()
```

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